Plant Poisoning of Small Ruminants

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Sheep, goats, lamas and alpacas are perennially affected by poisonous plants as a result of drought, over grazing and because the owners lack familiarity of the potential toxic plants in their pastures or the hay they feed their animals. Contrary to popular belief goats are susceptible to plant poisoning.

Many factors determine whether animals are poisoned by plants including the quantity and rate of the plant eaten; the stage of growth of the plant; the growing conditions of the plant; and whether or not the plants have been fertilized or treated with herbicides. The species of the animal, age, sex and general body condition of the animal also determine the outcome of plant poisoning. Lastly the nature of the toxin(s) in the plant in question will have profound effects on the animal eating it. The objective of this presentation is to review plant poisoning in small ruminants based upon the primary presenting clinical signs. The major, generally non-ornamental poisonous plants affecting small ruminants in North America are summarized in Table 1. The list is not intended to be exhaustive, and additional information sources on poisonous plants affecting animals are listed in the references. A listing of toxic ornamental garden plants that should not be accessible to sheep, goats and camelids is given in Table 2

Plants Causing Sudden Death
Cyanogenic Plants

Over 2500 species of plants especially in the plant families Asteraceae, Fabaceae, Linaceae, Rosaceae, and Poaceae contain cyanogenic glycosides capable of causing hydrogen cyanide (HCN, Prussic acid) poisoning in animals. The Rosaceae (Choke cherry, service berry, peach, apple etc.), and the Poaceae (Johnson grass, Sudan grass and many other grasses) have the greatest number of important cyanogenic plants affecting animals.

The quantity of cyanogenic glycosides in plants varies with the stage of growth, time of year, soil mineral and moisture content, and time of day. Cyanogenic glycosides in plants are catabolized by two enzymes – Beta-glycosidase and Hydroxynitrile lyase, to produce HCN.
When the plant tissues are chewed, the enzymes and glycosides are exposed to each other resulting in the formation of hydrogen cyanide. The enzyme Beta-glycosidase is also produced by rumen microorganisms, which in the optimum neutral pH of the rumen for enzyme activity, causes rapid formation of HCN. Ruminants on a high grain diet and consequently with an acidic rumen are also less susceptible to HCN poisoning. In general plant material containing more than 200ppm (20mg/100g of plant) has high potential for poisoning. Johnson or Sudan grass (Sorghum spp.) with levels > 500ppm HCN are highly toxic to ruminants.

Once absorbed HCN reacts with ferric iron (Fe^{+++}) in cytochrome oxidase in hemoglobin to stop cellular respiration. The formation of the cyanide-cytochrome oxidase complex prevents the release of oxygen from oxyhemoglobin thus causing cellular anoxia. Clinical signs of HCN poisoning in ruminants can begin within minutes to hours of eating large amounts of cyanogenic plants. The onset of signs can be accelerated if the animal drinks water after eating the plants as hydrolysis speeds up the liberation of HCN from the glycosides in the rumen. Sudden death of the animal is often the only observed sign. If observed early enough, apprehension, dyspnea, open mouth breathing, ataxia, frequent urination, and mydriasis, may be observed prior to death. Any stress on the animal exacerbates the signs and hastens death. The mucous membranes and venous blood may be bright cherry-red in color, but as the animal becomes anoxic, cyanosis is likely to develop. A syndrome of posterior ataxia and urinary incontinence can develop in horses, cattle and has been reported in sheep that have been fed sorghum hay for a period of weeks.

Timely treatment with a combination of sodium nitrite (10 to 20 mg/kg) and sodium thiosulfate (500 mg/kg) given intravenously and repeated as necessary has been successful in treating HCN poisoning. The sodium nitrite forms methemoglobin which has a greater affinity for HCN thereby breaking the cyanide-cytochrome complex. The thiosulfate then reacts with the cyanide via the enzyme rhodanase forming thiocyanate which is readily excreted in the urine. Research has shown the sodium thiosulfate alone given intravenously to sheep is as effective as the combination of sodium nitrite and sodium thiosulfate (40% solution of sodium thiosulfate given at the rate 25-50g/100kg body weight)

Nitrate Poisoning

Many plants including barley, wheat, rye, corn, sorghum and Sudan grasses, and common annual weeds such as pig weed, kochia weed, Russian thistle, bugseed can all accumulate toxic levels of nitrates. Application of nitrate fertilizers or drainage from fertilized fields and contaminated water sources can be as a source for nitrate. Drought conditions also promote accumulation of nitrate in plants. Nitrate itself is not overly toxic, but when however, when rumen microflora convert it to nitrite it becomes highly toxic. Sheep and goats and occasionally alpacas are susceptible to nitrate poisoning and have been lethally poisoned by eating oat hay containing 3.2% nitrate. (McKenzie et al. Australian Vet J 2009 87:113-115). Low energy diets increase a ruminants susceptibility to nitrate poisoning.

Cattle, sheep and goats are the species most commonly affected by nitrate poisoning, with the fetus in utero being highly susceptible. Nitrite ions rapidly oxidize hemoglobin in red blood cells, forming methemoglobin, which cannot transport oxygen. When over 40% of hemoglobin is oxidised to methemoglobin, clinical signs of poisoning develop. Death occurs as methemoglobin levels reach 80%. Sudden deaths are common but if observed early enough sheep and goats may exhibit a rapid weak pulse, muscle tremors, tachypnea, brown mucous membranes, excessive salivation, staggering gait, disorientation, and frequent urination prior to coma and death.

The preferred treatment for nitrate poisoning is methylene blue solution administered intravenously. As a reducing agent it converts methemoglobinto hemoglobin thereby restoring normal oxygen transport by the red blood cells.The recommended dose for methylene blue is 4 -15 mg/kg body weight administered as a 2-4% solution. In sheep, the half-life of methylene blue is about 2hours, indicating that small doses of the drug can be repeated as needed every few minutes to reduce methemoglobinemia and allow removal of further nitrate from the rumen. Cold water with added oral broad-spectrum antibiotics can help decrease nitrate reduction to nitrite by rumen microorganisms. Diluted vinegar given orally via stomach tube has similar beneficial effects.

Nitrate levels in aqueous humor of 20-40 ppm should be considered suspect, and over 40 mg/L (40ppm) could be considered diagnostic of nitrate poisoning. As a general rule, levels of nitrate over 0.5% in forages and water levels exceeding 200 ppm are potentially hazardous to pregnant animals especially if fed continuously. Forages containing in excess of 1% nitrate dry matter should be considered toxic.

Cardiotoxic Plants

Avocado (Persea Americana):- Leaves and possible all parts of the Guatemalan avocado are toxic to goats causing a non-infectious mastitis in lactating does, and myocarditis if sufficient amounts of the avocado leaves are consumed. The toxin, persin, apparently causes destruction of the lacteal cells and milk production does not recover once the doe is removed from the avocado. Goats fed as little as 31 g/kg body weight of avocado leaves showed marked decrease in milk production and

developed hard swollen udders 24 hours. The milk was of a cheesy consistency and contained clots, and the somatic cell counts are markedly elevated.

Oleander (*Nerium oleander)*: Livestock are usually poisoned when they browse on oleander or when trimmings are carelessly thrown into animal enclosures. Potent cardiac glycosides (cardenolides) are present in all parts of the plant. Oleander leaves remain toxic when dry. The lethal dose of the green oleander leaves for cattle and horses is 0.005 percent of the animal's body weight. Yellow oleander (*Thevetia* spp.) are equally as toxic.
Cardiac dysrhythmias and heart block may be observed prior to death.

Yew (*Taxus* spp.): Yews contain a group of 10 or more toxic alkaloids, the most toxic of which are taxine A and B. Taxine inhibits normal sodium and calcium exchange across the myocardial cells, preventing depolarization and causing arrhythmias. All parts of the plant, green or dried, except the fleshy part of the aril surrounding the seed are toxic. Livestock are frequently poisonedwhen fed clippings from cultivated yews. The highest concentration of the alkaloidsis generally found in the leaves in winter time. Dried leaves remain toxic. Adult cattle and horses have been fatally poisoned with as little as 8-16 oz of yew leaves or 0.1 to 0.5% of their body weight.

Milkweeds (*Asclepias* spp.): Many species of milkweed are found in North America, the most poisonous of which are those species with narrow, grass-like leaves. The principle toxins are cardenolides with digitalis-like properties. As little as 0.1 to 0.2% body weight of plant on a dry matter basis of the narrow-leafed species *A. subverticillata*, induced fatal poisoning in sheep. In addition to the cardiotoxic effects of the cardenolides common to most milkweeds, other glycosides and resinoids identified in milkweeds have direct effects on the respiratory, digestive, and nervous systems causing dyspnea, colic and diarrhea, muscle tremors, seizures, and head pressing. Milkweeds remain toxic when dry in hay.

Nephrotoxic Plants
Halogeton- (*Halogeton glomeratus*) is a noxious annual, multibranched herb with red stems and spreadinghorizontal branches that was introduced from Asia. It prefers alkaline soils. Sodium oxalate may comprise 30-40% of the dry matter content of the plant. Poisoning in sheep occurs when 0.3-0.5% of the animal's body weight of plant is consumed over a short period. A lethal dose of halogeton for an adult sheep is about 1.5 lb of green plant. Naïve animals or those on a poor level of nutrition are very susceptible to poisoning. Halogeton remains toxic when dried and is found to be quite palatable by sheep especially after it has been rained upon. Range sheep commonly utilize halogeton as a winter forage when grazing western rangelands. They are able to do this once they are slowly adapted to eating halogeton.
The soluble oxalates are readily absorbed from the rumen and bind with serum calcium and magnesium, causing a sudden decrease in available serum calcium and magnesium. Oxalate poisoning is typified by acute hypocalcemia causing animals to develop muscle tremors and weakness, leading to collapse and eventually death. Oxalates also interfere with cellular energy metabolism that contributesto the acute death of affected animals. In chronic oxalate poisoning, insoluble calcium oxalate causes severe oxalate nephrosis. If animals do not die from the acute effects of hypocalcemia and impaired cellular energy metabolism, death results from oxalate nephrosis.

Plants containing high levels of soluble oxalates

SCIENTIFIC NAME	COMMON NAME	Grasses
Amaranthus spp.	Red-rooted pigweed	*Cenchrus ciliaris* - Buffel grass
Bassia hyssopifolia	Five hooked bassia	*Panicum* spp. - Elephant grass
Beta vulgaris	Sugar beet	*Pennisetum clandestinum* - Kikuyu grass
Chenopodium spp.	Lambs-Quarter	*Setaria sphacelata*- Setaria grass
Halogeton glomeratus	Halogeton	
Kochia scoparia	Kochia, summer cypress	
Oxalis spp .	"Shamrock," sorrel	
Portulaca oleraceae	Purslane	
Phytolacca americana	Poke berry	
Rumex spp.	Sorrel, dock	
Rheum rhaponticum	Rhubarb	
Salsola spp.	Russian thistle, tumbleweed	
Sarcobatus vermiculatus	Greasewood	

Oak (*Quercus* spp.): All species of oak have the potential to poison animals, especially those eating larege quantities of the young leaves. The principal toxins are gallotannins, found in the leaves, bark, and acorns of oaks. Tannic acid is an astringent causing necrosis of the intestinal mucosa and renal tubules. Goats and wild ruminants are apparently better able to detoxify tannic acid than other livestock because they have a tannin-binding protein in their saliva that neutralizes tannic acid. Goats have been used effectively to browse on oaks thereby reducing the spread of the oak and increasing the grazing capacity of the range.
Clinically animals become depressed, anorexic and develop intestinal stasis. Excessive thirst and frequent urination may be observed. The feces are hard and dark initially, but a black tarry diarrhea often occurs later in the course of poisoning. Teeth grinding and a hunched back are often indicative of abdominal pain. Severe liver and kidney damage is detectable by marked elevations in serum liver enzymes, creatinine, and urea nitrogen. Icterus, red-colored urine, and dehydration are further signs of oak poisoning.

Neurotoxic Plants
Locoweeds (*Astragalus* and *Oxytropis* spp.): Locoweeds are the most economically important poisonous plant in North America. Some species of locoweed, but not all contain the indolizidine alkaloid swainsonine produced by the endophyte *Undifilum oxytropis*. Swainsonine inhibits mannosidase key to lysosomal and glycoprotein metabolism. This results in the accumulation of partially metabolized sugars in cells that disrupts protein synthesis and cell function including altered hormones, enzymes, and receptor binding in most cells. locoweeds/milkvetches cause respiratory problems and peripheral nerve degeneration due to nitroglycoside compounds in the plants. A third syndrome is caused by chronic selenium toxicity due to the ingestion of locoweeds or vetches that accumulate selenium.

Like cattle, sheep and goats with locoweed poisoning show reproductive problems including abortions, infertility, subcutaneous edema in the fetus, and fetal deformities. Cytoplasmic vacuolation identical to that seen in many other organs is also evident in the placental tissues. The placenta is most susceptible to the effects of locoweed during the first 90 days of pregnancy. Normal placentation may be interrupted causing fetal resorbtion, abortion, or hydrops allantois. The latter condition has been observed in sheep and cattle and is commonly referred to as water belly because the uterus becomes greatly distended with fluid. Lambs born to locoweed-poisoned dams may be born alive but weak and often die after a few days. Others may be smaller than normal or have deformities of the limbs or head.

Rams grazing locoweed for prolonged periods undergo testicular atrophy with decreased spermatogenesis.

Lupine (*Lupinus* spp.): Many species of lupine grow throughout North America but relatively few are toxic to livestock. Toxic lupines contain quinolizidine and piperidine alkaloids, the principal teratogenic quinolizidine alkaloid being anagyrine. Arthrogryposis and cleft palate seen in calves whose dams ate lupine in their first trimester is not commonly seen in sheep. More commonly lupines cause an acute fatal neurologic disease in sheep. The toxins responsible for the neurotoxicity are a variety of alkaloids other than those that are teratogenic. Sheep ingesting from 0.25 - 0.5% of their body weight of seeds from certain lupines (include *L. leucophyllus*, *L. argenteus*, *L. leucopsis*, and *L. siricus*) found in the western United States develop acute neurologic disease characterized by muscle tremors, noisy labored breathing, convulsions, coma, and death.

Burrow weed, Jimmy weed (*Isocoma* spp.): Burrow weed (rayless golden rod) is a common native plant of the alkaline soils of drier rangeland in Texas, Arizona, and New Mexico. Tremetone (tremetol) and possibly other compounds in *Isocoma* species is similar to that found in white snakeroot (*Ageratum altissimum*). Sheep, cattle and horses eating the plant develop marked depression, reluctance to move, muscle tremors and will collapse if forced to move. As the disease progresses animals get progressively weaker and die.

Poison or Spotted Hemlock (*Conium maculatum*): Originally introduced from Europe, poison hemlock has become a widely distributed noxious weed in North America. Eight piperidine alkaloids have been found in various parts of the plant. The two predominant toxic alkaloids are coniine (mature plant and seeds), and g-coniceine (young plant). The mechanism of action of the conium alkaloids is complex effectively blocking spinal cord reflexes. Muscle tremors are followed by neuromuscular blockade and paralysis. In sheep, repeated doses of *Conium* at 10 g/kg body weight were lethal. Cyanosis, respiratory paralysis, and coma without convulsions precede death. Goats may recover from hemlock poisoning only to develop a strong craving for the plant, which ultimately proves fata. Pregnant animals that survive the acute toxicity may abort. Lambs born to ewes fed poison hemlock in the 30 – 60th days of gestation develop excessive carpal joint flexure and lateral deviation.

Water Hemlock (*Cicuta* spp.): Native to North America, water hemlock is one of the most poisonous of plants to all animals. All parts of the plant and especially the roots contain Cicutoxin ($C_{17}H_{22}O_2$) that is rapidly absorbed from mucous membranes and acts on the central nervous system to produce rapid onset of ataxia, convulsive seizures and lateral recumbency, dilated pupils and die from respiratory paralysis. Animals consuming a sublethal dose, will recover if not stressed. There is no specific antidote.

Teratogenic Plants

Plants that are teratogenic and capable of causing abortions include lupines, locoweeds, tobacco, poison hemlock, rhododendrons and western false hellebore or skunk cabbage. Does may abort hairless kids with pronounced goiter after eating tansy mustard (*Descurainia pinnata)* in late pregnancy. Unlike cows that abort after eating pine needles (*Pinus ponderosa*), sheep do not abort after being fed a diet of 80% pine needles!

Western false hellebore, corn lily (*Veratrum* spp.): Over 50 complex alkaloids have been identified from *Veratrum* spp. The plant is most toxic when it first emerges in the early spring, becoming unpalatable as it matures. The alkaloids cyclopamine, jervine, and cyclopasine are teratogenic. Pregnant ewes consuming the plant on the 13 – 14th days of gestation produce lambs that have a single eye located in the center of its head. If *Veratrum* is eaten later in gestation (30-35th day) other defects including shortened legs and tracheal agenisis may develop. Embryonic death in lambs

without development of cyclopia may also occur. Sheep, cattle, goats, and llamas are also susceptible to *Veratrum* poisoning.

Plants causing liver disease and secondary photosensitization
Plants containing pyrrolizidine alkaloids such as Senecio, hounds tongue (*Cynoglossum officinale*), rattle pod (*Crotolaria* spp.) and fiddle neck (*Amsinckia* spp.) are the major cause of liver disease and secondary photosensitization. Pyrrolizidine alkaloids absorbed from the digestive tract are bioactivated to toxic pyrroles the liver's mono-oxygenase system. These pyrroles affect the endoplasmic reticulum of the liver cells inhibiting mitosis and the replication of hepatocytes. Over a period of weeks the liver undergoes characteristic pathologic changes of megalocytosis, bile duct hyperplasia, and fibrosis.
Goats, sheep, cattle, and horses are susceptible, but sheep require about 20 times the amount of *Senecio* it takes to poison a cow on an equivalent weight basis. Sheep are able to detoxify the pyrrolizidine alkaloids through specialized rumen bacteria before they are absorbed. Goats are similar to sheep in their relative resistance to poisoning from pyrrolizidine alkaloids but goats have been shown to abort if they consume 1% of their body weight per day of dry *S. jacobaea.*
Photophobia, excessive tearing, and swelling and redness of non-pigmented skin, develop initially before the affected skin becomes necrotic and sloughs. White breeds of sheep often only develop lesions on the ears and face because of the protective fleece covering unless they have been recently sheered. Prior to the development of secondary photosensitization, liver enzymes are elevated, and when the liver is severely affected signs of hepatic encephalopathy may develop.

Cocklebur (*Xanthium strumarium*): As common invasive weeds, cockleburs are poisonous to animals owing to the presence of the potent hepatoxin Carboxyactractyloside. The glycoside is present in high concentration in the seeds and the two-leafed cotyledon stage, but declines by the four-leaf stage and is absent in the mature plant. Acute diffuse central-lobular and paracentral coagulative necrosis are typical of cocklebur poisoning.

Miscellaneous Toxic Plants
Sneeze weed (*Dugaldia hoopesii*): Common native perennial plants in the intermountain and southwest areas of the USA, the sneeze/bitterweeds are a problem to range sheep.Orange sneezeweed and other bitterweeds (*Helenium* spp.) contain sesquiterpene lactones, which are irritating to the nose, eyes, and gastrointestinal tract. The primary toxin is hymenovin (dugaldin), but other lactones contribute to the toxicity. Other than the direct irritant effects on the digestive system,the lactones have a profound effect on metabolism through their ability to bind with sulfhydryl groups. Affected sheep regurgitate rumen contents ('Spewing sickness') and consequently lose weight. Secondary inhalation pneumonia is commonly seen in animals with bitterweed poisoning.

Burr Buttercup (*Ceratocephalus testiculatus*): A non-native invasive weed of drier areas, bur buttercup can be highly toxic to sheep, with a lethal dose being 500g of green plant for an adult sheep. Bur buttercup and some other buttercups (*Ranunculus* spp.) contain significant quantities of the irritating compound ranunculin. Clinical signs of bur buttercup poisoning are weakness, depression, diarrhea, labored breathing, and anorexia. Gross necropsy findings include inflammation and edema of the rumen, congestion of the lungs, liver, and kidneys; excessive fluid in the thoracic and abdominal cavities, and hemorrhages in the left ventricle of the heart.

Resources
Poisonous Plants http://southcampus.colostate.edu/poisonous_plants
Livestock-Poisoning Plants of California. http://anrcatalog.ucdavis.edu/pdf/8398.pdf
Plants Poisonous to Livestock. http://www.ansci.cornell.edu/plants/goatlist.html
Toxic Plants of North America. George E. Burrows & Ronald J. Tyrl

Table 1: Native and Exotic Plants Commonly Associated with Small Ruminant Poisoning

Common Name	*Botanical Name*	Toxin	Predominant Clinical Signs	
Plants Containing Cyanogenic Glycosides				
Johnson grass, Sudan grass	*Sorghum spp.*	**HCN**	Dyspnea, Sudden death	●
Arrow grass	*Triglochin spp.*	HCN	Dyspnea, Sudden death	●
Choke cherry	*Prunus spp.*	HCN	Dyspnea, sudden death	●
Christmas berry	*Heteromeles arbutifolia*	HCN	Dyspnea, Sudden death	
Nitrate Poisoning Plants				
Annual weeds		Nitrate	Dyspnea, Sudden death	●
Grasses		Nitrate	Dyspnea, Sudden death	●
Cardiotoxic Plants				
Avocado	*Persea americana*	Persin	Mastitis, cardiac failure	●
Oleander	*Nerium oleander*	Nerin	Cardiac arrhythmias, death	●
Milk weeds	*Asclepias spp*	Cardenolides	Arrhythmias, nervous signs, death	●
Yew	*Taxus spp.*	Taxine	Sudden death	●
Dogbane, Indian hemp	*Apocynum spp.*	Cymarin	Arrhythmias	
Nephrotoxic Plants				
Halogeton	*Halogeton glomeratus*	Oxalates	Renal failure	●
Oak	*Quercus spp.*	Gallotannins	Gastro enteritis, Renal failure	●
Greasewood	*Sarcobatus vermiculatus*	Oxalates	Renal failure	●
Dock	*Rumex spp.*	Oxalates	Renal failure	
Neurotoxic Plants				
Locoweeds	*Astragalus/Oxytropis spp.*	Swainsonine	Neurologic signs, congenital defects	●
Lupine	*Lupinus spp.*	Anagyrine	Nervous signs, teratogenic	●
Poison hemlock	*Conium maculatum*	Coniene	Death, teratogenic	●
Water hemlock	*Cicuta spp.*	Cicutoxin	Respiratory depression,death	●
Burrow weed	*Isocoma spp.*	Tremetol	Muscle tremors, weakness, death	●
White snake root	*Ageratina altissima*	Tremetol	Muscle tremors, weakness, death	

Teratogenic Plants, Abortions				
Locoweeds	*Astragalus/Oxytropis spp.*	Swainsonine	Congenital defects, abortions	●
Lupine	*Lupinus spp.*	Anagyrine	Congenital deformities	●
False hellebore	*Veratrum spp.*	Cyclopamine	Cyclopia, fetal tracheal agenisis	●
Tree tobacco	*Nicotiana glauca*	Anabasine	Cleft palate	●
Rhododendrons	*Rhododendron spp.*	Andromedotoxin	Abortions	●
Photosensitization, Liver disease				
Groundsel	*Senecio spp.*	Pyrrolizidine alkaloids	Liver failure, photosensitization	●
St John's wort	*Hypericum perforatum*	Hypericin	Photosensitization	
Fiddleneck	*Amsinckia spp.*	Pyrrolizidine alkaloids	Liver failure, photosensitization	●
Rattle pod	*Crotolaria spp.*	Pyrrolizidine alkaloids	Liver failure, photosensitization	●
Hound's Tongue	*Cynoglossum officinale*	Pyrrolizidine alkaloids	Liver failure, photosensitization	●
Spring parsley	*Cymopterus watsonii*	Furanocoumarins	Photosensitization	●
Cocklebur	*Xanthium spp.*	Carboxyactractylosid e	Liver failure, death	●
Horsebrush	*Tetradymia spp.*	Unknown	Photosensitization	
Miscellaneous Plants				
Sneezeweed	*Dugaldia, Helenium spp.*	Sesquiterpenes	Vomiting, inhalation pneumonia	●
Burr buttercup	*Ceratocephalus testiculatus*	Ranunculin	Gastroenteritis, diarrhea, death	●

- Plants frequently associated with poisoning in small ruminants

Reference: A Guide to Plant Poisoning of Animals in North America by Anthony P. Knight & Richard G. Walter. Teton New Media. Jackson, Wyoming

Table 2: Poisonous Ornamental Plants Potentially Harmful to Small Ruminants

Alkaloids

Botanical Name	Common Name	Predominant Clinical Signs
Aconitum spp.	Monk's hood, aconite	Vomiting, death
Albizia spp.	Mimosa tree	Neurologic signs
Aleurites spp.	Tung nut	Gastroenteritis
Amaryllis belladonna	Amaryllis, naked lady	Vomiting, diarrhea
Atropa belladonna	Deadly nightshade	Tachycardia, mydriasis
Brugmansia spp.	Angel's trumpet	Tachycardia, respiratory failure
Brunfelsia spp.	Yesterday-today-tomorrow	Seizures, vomiting
Buxus spp.	Boxwood	Vomiting, diarrhea
Calycanthus spp.	Sweet shrub, allspice	Muscle tremors, seizures
Cassia spp.	Golden shower tree	Gastroenteritis
Catharanthus spp.	Periwinkle	Neurotoxic, hypotension
Celastrus spp.	American bittersweet	Vomiting, diarrhea
Cestrum spp.	Cestrum, jasmine	Hepatotoxicity, calcinosis
Datura spp.	Jimson weed	Respiratory failure, hallucinations
Erythrina spp.	Coral bean	Muscle paralysis
Euonymus spp.	Burning bush	Gastroenteritis, cardiac dysrhythmias
Gelsemium spp.	Carolina jessamine	Neurotoxic, death
Gloriosa superba	Glory lily	Gastroenteritis, shock
Hymenocallis spp.	Spider lily	Gastroenteritis
Hyoscyamus spp.	Black henbane	Tachycardia, respiratory failure
Ilex spp.	Holly	Gastroenteritis
Ipomoea spp.	Morning glory	Neurologic abnormalities
Laburnum spp.	Golden chain tree	Vomiting, tachycardia
Nandina domestica	Heavenly bamboo	Seizures, respiratory failure
Narcissus spp.	Daffodils, narcissus	Vomiting, diarrhea
Nerine spp.	Nerine, or spider lily	Vomiting, diarrhea
Nicotiana spp.	Tobacco	Seizures, respiratory failure
Papaver spp.	Poppy	Excitement, depression
Physalis spp.	Ground cherry	Vomiting, colic
Solanum pseodocapsicum	Jerusalem cherry	Vomiting, respiratory failure
Sophora spp.	Mescal bean	Neurologic signs, ataxia
Zephyranthes spp.	Atamasco or rain lily	Vomiting, gastroenteritis

Toxalbumins (Lectins)

Abrus precatorius	Rosary pea	Enteritis, shock, death
Blighia sapida	Akee	Hypoglycemia, seizures
Hyacinthus spp.	Hyacinth	Gastroenteritis, death
Jatropha spp.	Physic nut, coral plant	Gastrointestinal irritation
Phoradendron spp.	Mistletoe	Gastrointestinal irritation
Ricinus communis	Castor bean	Gastroenteritis, shock, death
Robinia spp.	Black locust	Gastroenteritis, shock
Wisteria spp.	Wisteria	Gastrointestinal irritation

Genus	Common Name	Clinical Signs

Oxalates

Aglaonema spp.	Chinese evergreen	Salivation, oral edema
Alocasia spp.	Elephant's ears	Salivation, oral edema
Anthurium spp.	Flamingo flower	Salivation, oral edema
Caryota spp.	Fish tail palm	Salivation, oral edema
Colocasia spp.	Elephant's ears, taro	Salivation, oral edema
Dieffenbachia spp.	Dumb cane	Salivation, oral edema
Epipremnum spp.	Pothos	Salivation, oral edema
Monstera spp.	Ceriman	Salivation, oral edema
Oxalis spp.	Oxalis, sorrel	Salivation, hypocalcemia
Parthenocissus spp.	Virginia creeper	Vomiting, diarrhea
Philodendron spp.	Philodendron	Salivation, oral edema
Phytolacca american	Pokeberry	Gastroenteritis
Spathiphyllum spp	Peace lily	Salivation, oral edema
Zantedeschia spp.	Calla, arum lily	Calcium oxalate raphides
Glycosides		
Acocanthera spp.	Bushman's poison	Dysrhythmias, death
Actea spp.	Bane berry	Gastroenteritis
Adenium obesum	Desert rose	Cardiac dysrhythmias
Adonis spp.	Adonis, pheasant's eye	Dysrhythmias, enteritis
Aesculus spp.	Buckeye, horse chestnut	Vomiting, diarrhea, neurologic signs
Aloe spp.	Aloe	Diarrhea
Glycosides		
Clematis spp.	Clematis	Gastroenteritis, diarrhea
Convallaria majalis	Lily of the valley	Dysrhythmias, enteritis
Cryptostegia spp.	Rubber vine	Gastroenteritis
Cycas spp.	Cycad, sago palm	Liver necrosis, neurotoxicity
Digitalis spp.	Fox glove	Dysrhythmias, gastroenteritis
Eriobotrya spp.	Loquat	Cyanosis, respiratory failure, death
Euonymus spp.	Burning bush	Gastroenteritis, cardiac dysrhythmias
Hydrangea spp.	Hydrangea	Cyanosis, respiratory failure, death
Kalanchoe spp.	Kalanchoe	Dysrhythmias, gastroenteritis
Nandina domestica	Heavenly bamboo	Cyanosis, respiratory failure, death
Nerium oleander	Oleander	Cardiac dysrhythmias, death
Ornithogallum spp.	Star of Bethlehem	Cardiac dysrhythmias
Osteospermum ecklonis	African daisy	Dyspnea death from cyanide
Podophylum spp.	May apple	Gastroenteritis, neurologic signs
Prunus spp.	Choke cherry	Cyanosis, respiratory failure, death
Pyracantha coccinea	Pyracantha	Cyanosis, respiratory failure
Thevetia spp.	Yellow oleander	Cardiac dysrhythmias, death
Zamia spp.	Sago palm	Liver necrosis

Genus	Common Name	Clinical Signs
Saponins/Sapogenins		
Aesculus spp.	Buckeye, horse chestnut	Gastroenteritis
Agave lecheguilla	Agave	Cholecystitis, biliary obstruction
Gymnocladus spp.	Kentucky coffee tree	Gastroenteritis
Phytolacca american	Pokeberry	Gastroenteritis
Pittosporum spp.	Pittosporum	Gastroenteritis
Schefflera spp.	Umbrella tree	Gastroenteritis
Sesbania spp.	Rattlebush, coffee bean	Gastroenteritis
Yucca spp.	Yucca	Cholecystitis, biliary obstruction
Terpenoids		
Achillea spp.	Yarrow	Gastroenteritis
Allamanda spp.	Allamanda	Gastrointestinal irritation
Caesalpinia spp.	Peacock flower	Gastrointestinal irritation
Capsicum spp.	Peppers	Gastrointestinal irritation
Euphorbia spp.	Euphorbias	Stomatitis, vomiting
Hedera spp.	Ivy	Gastrointestinal irritation
Iris spp.	Iris	Gastrointestinal irritation
Jatropha spp.	Physic nut, coral plant	Gastrointestinal irritation
Kalmia spp.	Laurel	Vomiting, cardiac dysrhythmias
Lantana spp.	Lantana	Cholecystitis/stasis
Leucothoe spp.	Fetterbush	Vomiting, cardiac dysrhythmias
Ligustrum spp.	Privet	Vomiting, diarrhea, ataxia
Lonicera spp.	Honeysuckle	Vomiting, diarrhea
Lyonia spp.	Staggerbush, maleberry	Vomiting, cardiac dysrhythmias
Melaleuca spp.	Melaleuca, tea tree	Neurologic signs
Melia azadarach	Chinaberry tree	Vomiting, diarrhea, ataxia
Pieris spp.	Pieris	Vomiting, cardiac dysrhythmias
Plumeria spp.	Plumeria	Vomiting, diarrhea
Sambuccus spp.	Elderberry	Gastrointestinal irritation
Sapindus spp.	Soapberry	Gastrointestinal irritation
Schefflera spp.	Umbrella tree	Saponins, terpenoids, oxalates
Schinus spp.	Pepper tree	Dermatitis, vomiting
Sulfoxides and other Oxidants		
Allium species	Onions	Heinz body anemia

Adapted from: A Guide to Poisonous House and Garden Plants by Anthony P. Knight. Publisher Teton New Media, Jackson Wyoming