

Toxic Plants

Neurotoxic and Teratogenic

Digestive Tract

Kidneys

Neurotoxic

Skin and liver

Reproductive System



Cardiovascular effects

Blood

Musculoskeletal

Nitrate-accumulators

Plant hazards to pets

Plant	Toxin	Toxic MOA	Intoxication Symptoms	Diagnosis and Treatment	Picture
<p>Poison Hemlock <i>Conium spp.</i></p>	<p>Piperidine and other alkaloids</p> <p>Coniine, γ-coniceine, N-Methyl coniine</p> <p>Fresh, hay and seeds are toxic at 5g/kg bw</p>	<p>Alkaloids act on autonomic nervous system and mimic the action of Ach (nicotinic in nature)</p>	<p>Neurotoxic: Shaking, twitching, paralysis, staggering, convulsions, heavy breathing, coma, death</p> <p>Teratogenic: Immobilization of fetal movement, arthrogryposis, cleft palate, carpal flexure, torticollis, scoliosis</p> <p>Cattle and pigs are most susceptible</p>	<p>Dx: Alkaloids in urine or GI contents</p> <p>Tx: No specific treatment exists</p> <p>Acute poisoning can be addressed with activated charcoal and cathartics, careful monitoring, and limiting stress</p> <p>Prevention is KEY</p>	
<p>Tobacco <i>Nicotiana spp.</i></p>	<p>Piperidine and other alkaloids</p> <p>Nicotine, nornicotine, anabasine</p> <p>Toxic when dry and fresh at 1% bw</p>	<p>Alkaloids act on autonomic nervous system and mimic the action of Ach (nicotinic in nature)</p>	<p>Neurotoxic: Shaking, twitching, paralysis, staggering, convulsions, heavy breathing, coma, death</p> <p>Teratogenic: Immobilization of fetal movement, arthrogryposis, cleft palate, carpal flexure, torticollis, scoliosis</p> <p>Cattle, sheep, goats, and pigs are most susceptible</p>	<p>Dx: Alkaloids in urine or GI contents</p> <p>Tx: No specific treatment exists</p> <p>Acute poisoning can be addressed with activated charcoal and cathartics, careful monitoring, and limiting stress</p> <p>Prevention is KEY</p>	

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<p>Lupine <i>Lupine spp.</i></p>	<p>Piperidine and other alkaloids</p> <p>Lupanine, sparteine, anagyrene, ammodendrine</p> <p>Toxic when dried, fresh, and seeds at 0.25% bw</p>	<p>Alkaloids act on autonomic nervous system and mimic the action of Ach (nicotinic in nature)</p>	<p>Neurotoxic: Shaking, twitching, paralysis, staggering, convulsions, heavy breathing, coma, death</p> <p>Teratogenic: Immobilization of fetal movement, arthrogryposis, cleft palate, carpal flexure, torticollis, scoliosis</p> <p>Sheep and cattle are the most susceptible</p>	<p>Dx: Alkaloids in urine or GI contents</p> <p>Tx: No specific treatment exists</p> <p>Acute poisoning can be addressed with activated charcoal and cathartics, careful monitoring, and limiting stress</p> <p>Prevention is KEY</p>	
<p>Cyanogenic plants Sorghum species <i>Prunus spp.</i> (choke cherry) <i>Trigochin spp.</i> (arrow grass) <i>Malus spp.</i> (crab apple leaves) <i>Eucalyptus cladocalyx</i> (sugar gum) <i>Amelanchier alnifolia</i> (service berry)</p>	<p>Cyanide-containing plants</p> <p>Amygdalin and dhurrin</p>	<p>Hydrolysis by beta-glucosidase yields hydrogen cyanide (occurs through GI tract)</p> <p>Frost, wilting, stunting, and mastication can also cause hydrolysis</p> <p>Free cyanide is poisonous to all animals</p> <p>Absorbed free cyanide binds to iron (Fe 3+) in cytochrome oxidase</p>	<p>Animals are often found dead</p> <p>Death occurs within 1-2 hours after exposure to a lethal amount</p> <p>Labored breathing, ataxia, frothing at the mouth, muscle tremors, convulsions</p> <p>Bright red mucous membranes initially then cyanosis of mucous membranes terminally</p>	<p>Dx: Check color of blood – Cherry Red</p> <p>Collect blood sample for cyanide testing</p> <p>Liver and muscle tissue can also be used for cyanide testing</p> <p>Tx: Sodium nitrite and Sodium thiosulfate</p> <p>1mL of 20% sodium nitrite IV 3mL of 20% sodium thiosulfate IV</p>	 <p>Toyon Plant</p>

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		preventing normal enzymatic action leading to an inactivation of cellular respiration. Oxygen-saturated hemoglobin cannot release O ₂ leading to cherry red venous blood.			
<p>Yellow Star Thistle <i>C. solstitialis</i> and Russian knapweed <i>C. repens</i></p>	<p>Lactones</p> <p>Fresh and dry plants are toxic</p> <p>Large intakes over weeks to months are needed to result in disease 80-200% bw</p>	<p>Toxins destroy dopaminergic neurons in the substantia nigra and globus pallidus affecting CN V, VII, and XII</p> <p>Only affects horses</p>	<p>Equine nigropallidal encephalomalacia (ENE)</p> <p>“Equine Parkinson’s Disease”</p> <p>“Chewing disease”- continuous chewing movements, frothing of saliva, difficulty prehending food</p> <p>Frequent yawning, open mouth tongue protruding, ulceration of tongue, lips and gingiva</p> <p>Head submersion and then head tipping back while drinking</p>	<p>Dx: Clinical signs</p> <p>Pathognomonic lesions</p> <p>MRI: Bilateral malacia of the Substantia nigra or Globus pallidus</p> <p>Tx: None, prevention is KEY</p>	 <p>Russian knapweed</p>  <p>YST</p>

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<p>Milkweed <i>Asclepias</i> spp.</p>	<p>Neurotoxins: Not clearly identified; glycosides and resinoids are present with direct effect on nervous system</p> <p>Cardiotoxins: Cardiac glycosides (similar to oleandrin)</p>	<p>Inhibition of sodium-potassium pump (Na/K-ATPase) resulting in depletion of intracellular potassium and increased levels of intracellular sodium</p>	<p>Neurotoxins: Colic or abdominal discomfort, incoordination and weakness, convulsions and death due to respiratory failure</p> <p>Cardiotoxins: Depression, weakness, irregular respiration, apparent abdominal discomfort, cardiac arrhythmias, recumbency</p>	<p>Dx: Clinical signs and history of ingestion</p> <p>Tx: Supportive therapy, prevention is key!</p>	 
<p>Locoweeds <i>Astragalus</i> & <i>Oxytropis</i> spp.</p>	<p>Miserotoxin (glycoside containing nitro group)</p> <p>Locoism: indolizidine alkaloids - swainsonine</p> <p>Selenium accumulators</p>	<p>Swainsonine: Inhibition of alpha-D-mannosidase and Golgi mannosidase II</p> <p>Important for the metabolism of saccharides and the formation of glycoproteins</p> <p>Undigested carbohydrates accumulate in</p>	<p>Nitro compounds: Livestock poisoning- Cracker heels or roaring disease</p> <p>“Loco” abnormal neurologic behavior -circling, depression, incoordination, staggering gait, difficulty being handled, unpredictable</p>	<p>Dx: Clinical signs and history of exposure</p> <p>Tx: No specific treatment available, nervous system damage is reversible to a point</p> <p>*Recovering horses should be considered unsound</p> <p>Prevention is key</p>	 <p><i>Astragalus bisulcatus</i></p>

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		<p>lysosomes which increase in size and number leading to lysosomal storage disease</p> <p>*Nervous system and reproductive system are targeted and clinical signs are not seen until several weeks of exposure</p>	<p>Infertility and reproductive failure -abnormal placentation: abortions, infertility, fetal resorption, fetal deformities (crooked legs), effects on spermatogenesis</p> <p>Heart failure in cattle at high altitudes (right sided heart failure “high mountain disease”)</p> <p>Weight loss and poor performance: Difficulty prehending food and a decreased appetite</p>		 <p><i>Astragalus mollissimus</i></p>
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<p>Water hemlock <i>Cicuta spp.</i></p>	<p>Cicutoxin -Long-chain alcohol</p> <p>Toxin is concentrated in the tuberous roots but all parts of the plant are toxic</p> <p>Newly emerging plants in the spring are mostly toxic while mature leaves and dry stems are minimally toxic</p>	<p>Unknown MOA</p>	<p>Lethal dose of fresh root of <i>C. douglasii</i></p> <p>Sheep: 2 oz Cattle: 10-12 oz Horse: 8 oz</p> <p>Death usually occurs within 1-3 hours of exposure</p> <p>Convulsive seizures and lateral recumbency often occur</p> <p>Ataxia, incoordination, salivation, and vigorous chewing movements</p>	<p>Dx: Clinical signs- acute onset of violent, tetanic seizures, death due to hypoxia</p> <p>Post-mortem: chewed hemlock roots are often found in the esophagus and rumen</p> <p>Lesions: Not pathognomonic</p> <p>Microscopic examination of the GI contents to confirm water hemlock ingestion</p> <p>Tx: rarely possible but IV barbiturates may help, prevention is KEY</p>	 
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<p>Larkspurs <i>Delphinium</i> <i>spp.</i></p>	<p>Diterpenoid alkaloids Methyllycaconitine</p> <p>Toxicity varies with the species, season, stage of growth, amount ingested and duration of exposure</p>	<p>Inhibition of Ach at nicotinic postsynaptic receptor sites “curare like action”</p> <p>Loss of motor function especially the diaphragm and esophagus</p> <p>Muscle weakness and paralysis</p>	<p>Cattle are most susceptible but horses and goats are at risk</p> <p>Sheep can tolerate 4-5x the amount that is fatal to cattle</p> <p>Sudden death usually occurs 3-4 hours post exposure</p> <p>Bloat (result of NM blocking on the esophagus)</p> <p>Aspiration pneumonia from inhalation of regurgitated rumen contents</p> <p>Death due to respiratory failure</p>	<p>*Larkspurs cause more fatal poisonings of cattle in the Western US than any other native plant species. It is 2nd to locoweeds in terms of economic loss</p> <p>Dx: clinical signs</p> <p>Post-mortem: no specific lesions</p> <p>Microscopic examination of GI contents to confirm larkspur ingestion</p> <p>Detection of alkaloids in GI contents</p> <p>Tx: only useful if an early diagnosis is made</p> <p>Anticholinesterase drugs: -Physostigmine (0.4-0.8 mg/kg IV) -Neostigmine (0.04 mg/kg) not as effective</p> <p>Avoid stress and excitement Relieve bloat Prevention is key</p>	
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<p>Brackenfern <i>Pteridium aquilinum</i></p>	<p>Thiaminase: Breakdown of vitamin B1 (thiamin)</p> <p>Thiamin is essential for energy metabolism</p> <p>Ptaquiloside: Cardiogenic Bone marrow depressant activity Excreted into milk (risk for humans and calves)</p>	<p>Thiamin deficiency</p> <p>Urinary bladder cancer (enzootic hematuria)</p> <p>Hemorrhaging and bone marrow destruction</p> <p>Retinal degeneration and blindness</p> <p>Digestive tract cancers</p>	<p>Cattle and sheep: Acute hemorrhagic syndrome Sever bone marrow depression, thrombocytopenia, anemia, leukopenia, hemorrhaging from the nose, mouth, vagina, hemorrhagic diarrhea, melena, hematuria, high mortality *After ingestion of plant for 1-2 months</p> <p>Cattle Enzootic hematuria: “Red water disease” Small polyp-like tumors in the bladder</p> <p>Sheep “Bright blindness” Irreversible retinal degeneration Associated with grazing Dilated pupils</p> <p>Horses: Thiamin deficiency Polioencephalomalacia (PEM)</p>	<p>Dx: clinical signs</p> <p>Tx: For thiamin deficiency in horses</p> <p>5mg/kg thiamin IV for several days</p>	
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<p>Oleander <i>Nerium oleander</i></p>	<p>Oleandrin, oleandrogenin, thevetin, neriifolin, peruvoside, ruvoside</p> <p>Cardiac glycoside containing plant</p>	<p>Inhibition of sodium-potassium pump (Na/K-ATPase) resulting in depletion of intracellular potassium and increased levels of intracellular sodium</p> <p>Toxic when fresh and dry Minimum lethal dose is ~ 5 leaves</p>	<p>Diarrhea, depression, anorexia, excessive salivation</p> <p>Cardiac signs: Bradycardia, tachycardia, arrhythmias</p> <p>Sudden death Kidney failure</p>	<p>Dx: Diagnosis of leaves in ingesta</p> <p>Chemical analysis of serum, urine, or ingesta (oleandrin) Elevated serum K⁺</p> <p>Tx: Llamas, cattle, horses – activated charcoal 1-5g/kg bw</p> <p>Bradyarrhythmia: Atropine sulfate</p> <p>Tachyarrhythmias: Propranolol, lidocaine, phenytoin, metoprolol</p> <p>AVOID calcium and potassium containing fluids</p>	

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<p>Azalea <i>Rhododendron spp.</i></p>	<p>Grayanotoxanes <i>Ericaceae</i></p> <p>Highest concentration in leaves</p>	<p>Interferes with voltage gated sodium channels located in the cell membrane of neurons</p> <p>Minimum lethal dose in goats is 0.1% bw</p> <p>Goats are most susceptible</p>	<p>“Mad Honey Disease” in humans</p> <p>Vomiting, salivation, colic, depression</p> <p>Tachycardia, tachypnea, recumbency, elevated body temperature, seizures</p>	<p>Dx: Urine, serum, GI contents for grayanotoxanes</p> <p>ID plant material in environment and/or rumen content</p> <p>Tx: No antidote Decontamination- Activated charcoal and cathartics</p> <p>Antiarrhythmics</p> <p>Supportive therapy with fluids</p> <p>Antibiotics if the animal has aspirated</p>	
<p>Avocado <i>Persea americana</i></p>	<p>Persin</p> <p>All above ground parts are toxic. Leaves are especially toxic when dried</p>	<p>Unknown</p>	<p><u>Birds, Rabbits, Goats:</u> Acute death and cardiac signs</p> <p><u>Cattle, horses, goats, rabbits:</u> Mastitis and agalactia</p> <p><u>Horses:</u> Colic, diarrhea, neck edema</p>	<p>Dx: post-mortem lesions</p> <p>Tx: Supportive</p>	

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<p>Yews <i>Taxus spp.</i></p>	<p>Taxine alkaloids Toxic when fresh or dry *Berries are not toxic when ripe</p>	<p>Fatal cardiac conduction disturbance 6-8 oz are lethal for an adult cow or horse Deer are NOT affected</p>	<p>Altered mental status, decreased awareness, cyanosis, breathing difficulty</p>	<p>Dx: Taxine alkaloids in GI content Tx: Activated charcoal and Atropine</p>	
<p>Dumbcane <i>Dieffenbachia sequine</i> Pothos varieties <i>Epipremnum</i> Calla Lily <i>Zantedeschia</i> *Many common houseplants</p>	<p>Insoluble calcium oxalates All parts of the plant are toxic</p>	<p>Calcium oxalates form raphides which release proteolytic enzymes which increase the severity of symptoms</p>	<p>Rapid onset of clinical signs Hypersalivation, head shaking, chewing, pawing at mouth</p>	<p>Dx: Clinical signs Tx: flush mouth with water and remove plant material Symptomatic and supportive care</p>	
<p>Bristlegrass <i>Setaria spp.</i> Yellow Foxtail <i>Setaria glauca</i></p>	<p>N/A</p>	<p>Mechanical damage via barbed bristles or prickly plant parts</p>	<p>Livestock/Horses: Excessive salivation, ulceration, granulation tissue filling the ulcer, anorexia Dogs: Repetitive sneezing, limping,</p>	<p>Dx: Clinical signs Tx: Mechanical removal of the plant material Care for abscesses and infections</p>	

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			continuous licking, head shaking and ear scratching, lacrimation		
Black locust <i>Robinia pseudoacacia</i>	Lectins: abrin, ricin, robinin Bark and seeds are the most toxic 0.04% of bw is toxic to horses	Targets glycoprotein chains A and B B chain: binds to galactoside-containing proteins on cell surface facilitating internalization A chain: Enters the ER and depurinates 28S rRNA causing an inhibition of protein synthesis and cellular death	GI irritation hours to days after exposure Colic Increased heart rate (tachycardia) Hypovolemic shock	Dx: Clinical Signs Tx: Supportive and symptomatic care, GI decontamination, activated charcoal, fluids	
Rosary pea, precatory bean <i>Abrus precatorius</i>	Lectins: abrin , ricin, robinin Lethal dose: 0.00015% of a person's bw	Targets glycoprotein chains A and B B chain: binds to galactoside-containing proteins on cell surface facilitating internalization A chain: Enters the ER and depurinates 28S rRNA causing an inhibition of protein synthesis and cellular death	GI irritation hours to days after exposure Colic Increased heart rate (tachycardia) Hypovolemic shock	Dx: Clinical Signs Tx: Supportive and symptomatic care, GI decontamination, activated charcoal, fluids	

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<p>Castor bean <i>Ricinus communis</i></p>	<p>Lectins: abrin, ricin, robinin</p> <p>Seeds must be broken or crushed to release the toxin</p> <p>Toxalbumin: Protein with affinity for sugar molecules, one of the most toxic compounds of plant origin *Castor oil does not contain ricin</p> <p>60 seeds can kill a horse 3-4 seeds can kill a duck 2-20 seeds can kill a person</p> <p>0.2% bw may cause toxicosis</p>	<p>Targets glycoprotein chains A and B</p> <p>B chain: binds to galactoside-containing proteins on cell surface facilitating internalization</p> <p>A chain: Enters the ER and depurinates 28S rRNA causing an inhibition of protein synthesis and cellular death</p>	<p>Lag period: a few hours to days</p> <p>Vomiting with blood</p> <p>Diarrhea- often bloody with tenesmus and abdominal pain</p> <p>Lesions: Catarrhal to hemorrhagic gastroenteritis, petechial hemorrhages on serosal surfaces, necrotizing enteritis, edematous mesenteric lymph nodes</p>	<p>Dx: hx of exposure, presence of seeds in excreta Leukocytosis Increased Alt Detection of alkaloids (ricinine in gastric contents)</p> <p>Tx: Supportive and symptomatic care, no specific antidote</p> <p>Poor prognosis if well-masticated / large quantity consumed</p> <p>Prevention is key *most heat destroys ricin</p>	
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<p>Angel's trumpet, Thorn apple (jimsonweed) <i>Datura spp,</i> <i>Brugmansia spp</i></p>	<p>Tropane alkaloids *think atropine* Hyoscine, hyoscyamine Highest concentration in seeds and leaves</p>	<p>Competitive blockade of acetylcholine at muscarinic receptors both centrally and peripherally as well as at end organ sites of the parasympathetic nervous system</p>	<p>Anticholinergic toxidrome Increased respiratory and heart rate, dry mouth, incoordination Dilation of pupils, decreased GI motility Positive drug testing result in race horses</p>	<p>Dx: Clinical signs and hx Tx: Activated charcoal, supportive and symptomatic treatment</p>	
<p>St. John's wort <i>Hypericum perforatum</i></p>	<p>Hypericin Primary photosensitization</p>	<p>Presence of photodynamic agent in the bloodstream (hypericin) and its excitement by UV light</p>	<p>Increase in sensitivity to UV radiation Reaction most severe in nonpigmented skin: Erythema and edema, pruritis Photophobia, hyperesthesia,</p>		

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<p>Common groundsel <i>Senecio vulgaris</i></p> <p>Tansy ragwort <i>Senico jacobaea</i></p>	<p>Pyrrolizidine-alkaloid containing plant Seneciphylline, senecionine, jacobine, jaconine, retrorsine, riddelline Monocrotaline, spectabiline, retusine</p> <p>Phylloerythrin is the photosensitizing compound</p> <p>Secondary photosensitization (hepatogenous)</p>	<p>Hematogenous photosensitization</p> <p>Liver function imparinment</p> <p>Phylloerythrin is a breakdown product of chlorophyll, usually removed by the liver but due to the liver damage it accumulates in the circulation</p> <p>Liver activation of PA to toxic pyrroles leads to hepatic disease</p> <p>Consumption of 5-10% bw over a few days can cause acute liver disease in horses and cattle</p> <p>Chronic liver disease occurs when small amounts are consumed over several months (25-50%) bw</p>	<p>Affects cattle and horses Young animals more susceptible and it is generally only consumed during drought conditions because it is unpalatable</p> <p>Sheep are more resistant (but not completely immune)</p> <p>*Head pressing in horses due to the buildup of ammonia in the CNS since it cannot be cleared from circulation</p>	<p>Dx: Gross lesions</p> <p>Acute- signs of liver failure, icterus, edema</p> <p>Chronic-Firm nodular liver, cirrhosis, icterus +/- photosensitivity</p> <p>Microscopic lesions: hepatocytomegaly, atypical nuclei or karyomegaly</p> <p>Bridging periportal fibrosis Bile duct proliferation</p> <p>Tx: No specific treatment supportive care for liver failure</p>	<p> Common groundsel</p> <p> Tansy ragwort</p>

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<p>Red maple <i>Acer rubrum</i></p>	<p>Unidentified toxin in wilted and dried leaves (for about 1 month)</p> <p>Green leaves are not toxic</p> <p>Horses, ponies, zebras, and alpacas are affected</p>	<p>Oxidative damage to RBC leading to hemolytic anemia</p> <p>*Heinz bodies on cytology</p> <p>Ingestion of 1.5g/kg bw is lethal in ponies</p>	<p>Signs appear several days after exposure</p> <ul style="list-style-type: none"> -Acute hemolytic anemia -Red-brown urine, oliguria, anuria -Weakness, tachypnea, depression -Cyanosis, icterus 	<p>Dx: Bloodwork showing a low PCV, Heinz bodies, hyperbilirubinemia, hemoglobinuria and proteinuria</p> <p>Lesions: Generalized icterus, splenomegaly, severe diffuse congestion of kidneys</p> <p>Tx: Symptomatic and supportive care, activated charcoal, dexamethasone, ascorbic acid, blood transfusion, fluids to maintain kidney function, hemoglobin glutamer (oxyglobin)</p>	
<p>Onion <i>Allium spp</i> <i>-cepa (onion)</i> <i>-sativum (garlic)</i></p>	<p>n-Propyl disulfides</p> <p>Present in raw, cooked, and dried onions</p> <p>*dogs, cats, and cattle are most susceptible</p> <p>** Increased sulfide groups in cats makes them more susceptible</p> <p>Dogs: 11-15g/kg bw of raw onions are toxic</p>	<p>Increased free radical formation</p> <p>Direct erythrocyte membrane damage and denatured hemoglobin</p> <p>Heinz body formation and acute hemolysis</p>	<p>Inappetence, lethargy, tachycardia, tachypnea</p> <p>Onion odor to the breath, pale mucous membranes</p> <p>Abortions are possible</p>	<p>Dx: Lab findings; hemolytic anemia, Heinz body formation (obvious with new methylene blue stain)</p> <p>Eccentrocytes-ragged fringe of cytoplasm along one side of the cell, occurs secondary to oxidative stress</p> <p>Tx: Avoid stress, blood transfusions</p>	 <p><small>http://en.wikipedia.org/wiki/File:Onion_whitebackground.jpg</small></p>

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<p>Redroot pigweed <i>Amaranthus retroflexus</i></p>	<p>Unknown toxic principal but it causes an accumulation of nitrates and soluble oxalates</p> <p>Cattle, sheep and goats are most susceptible</p>	<p>Soluble oxalates are absorbed into the blood where it interacts with calcium. Calcium and magnesium oxalates are secreted and blocks nephron tubules</p> <p>Highest concentration of soluble oxalates (sodium and potassium oxalates) are in the leaves</p>	<p>Posterior weakness, incoordination and sternal recumbency</p> <p>Death may occur up to 10 days after removal from plants</p> <p>Gross lesions: Perirenal edema +/- hemorrhage Ascites</p> <p>Histologic lesions: Acute tubular necrosis affecting both proximal and distal tubules</p>	<p>Dx: Clinical signs and lesions post-mortem</p> <p>Hypocalcemia</p> <p>Crystallization of calcium oxalates in the kidneys</p> 	
<p>Oak <i>Quercus spp.</i></p>	<p>Hydrolysable tannins = Polyphenolic complexes</p> <p>Phenolic acids: Gallic acid, pyrogallol, resorcinol</p> <p>*GI and renal consequences</p>	<p>The phenolic acids have an astringent effect on the gut mucosa leading to GI irritation, they react with cell proteins causing denaturation and cell death, and they also lead to tissue destruction (kidney and liver) *kidney damage is severe especially in cattle</p> <p>**Need to be consumed in large</p>	<p>Cattle: Abrupt onset, diarrhea or constipation with bloody or mucoid feces, anorexia, listlessness, rumen stasis, oliguria, weakness and recumbency</p> <p>*If BUN is highly elevated the prognosis is guarded</p> <p>Goats/Deer: Can browse oak effectively,</p>	<p>Dx: Gross lesions Ascites, hydrothorax, perirenal blood-tinged edema, hemorrhagic and ulcerative gastroenteritis, acorns in rumen</p> <p>Histologic lesions: Coagulation necrosis of proximal convoluted tubules, *regenerative if basement membrane is intact</p> <p>Tx: Remove from access to oak; if unavoidable, grain</p>	

Toxic Plants

Neurotoxic and Teratogenic

Digestive Tract

Kidneys

Neurotoxic

Skin and liver

Reproductive System



Cardiovascular effects

Blood

Musculoskeletal

Nitrate-accumulators

Plant hazards to pets

		<p>amounts and preceding a period of feed restriction to cause toxicity</p>	<p>tannin-binding proteins in saliva and GI tract</p> <p>Horses: Diarrhea, colic, tenesmus, fewer renal effects</p>	<p>mix containing 10% calcium hydroxide may be effective in binding tannins</p> <p>Activated charcoal or mineral oil and fluids to correct dehydration/acidosis</p>	
<p>Skunk cabbage, corn lily <i>Veratrum californicum</i></p>	<p>Cevanine alkaloids -Neurotoxic effect</p> <p>Jervanine alkaloids -Teratogenic effects -Cyclopamine, cycloposine and jervine</p> <p>*Cyclopamine is believed to be the most important due to its greater concentration in plants</p> <p>All species are affected, rarely enough ingested to cause neurotoxicity, mostly teratogenic effects occur</p> <p>Frost results in loss of toxicity</p>	<p>Cevanine: Bind open voltage-selective Na⁺ channels</p> <p>Cyclopamine interferes with intercellular signaling and patterning during embryogenesis and organogenesis</p>	<p>Sheep: Day of gestation</p> <p>14th day: Cyclops</p> <p>19-21 day: Embryonic death</p> <p>28-32 days: Limb defects</p> <p>31-33 days: tracheal stenosis</p>	<p>Dx: Visual appearance of animal</p> <p>Tx: none</p> 	

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<p>Ponderosa pine <i>Pinus ponderosa</i> <i>Pinus contorta</i> <i>Juniperus communis</i></p>	<p>Isocupressic acid *2.2-2.7 kg of pine needles per day for > 3 days causes abortion</p>	<p>Reduction in uterine blood flow resulting in reduction in nutrients and oxygen to the fetus which stimulates release of fetal cortisol leading to abortion</p>	<p>Abortion 2-21 days after exposure Greatest risk in the last three months of pregnancy (3rd trimester)</p>	<p>Dx: Visual appearance and abortion Tx: none, prevention is key!</p>	 <p>Ponderosa pine</p>  <p>Common Juniper</p>
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<p>Black Walnut <i>Juglans nigra</i></p>	<p>Exact toxin is unknown *Horses are the primary affected species</p>	<p>Unknown MOA Wet bedding is the most dangerous Walnut shavings are dark brown whereas cedar and pine shavings are pale Bedding needs to dry in the sun for 2+ months before use</p>	<p>May occur in “outbreaks” as large groups of horses are exposed at once to new bedding Reluctance to move within 24 hours of exposure Depression Increased temperature, heart and respiration rate, digital pulses, hoof temperature Lower limb edema Severe laminitis with continued exposure -P3 rotation and separation</p>	<p>Dx: History of recent exposure and acute clinical signs Tx: Nonfatal disease if addressed early Removal of offensive bedding Oral detoxification -Mineral oil -Activated charcoal -Cathartic Treat limb edema and laminitis as indicated</p>	
<p>Day-blooming Jessamine <i>Cestrum diurnum</i></p>	<p>glycoside of 1,25-dihydroxycholecalciferol</p>	<p>Contains an active metabolite of vitamin D; Increased levels of D3 result in the excess accumulation of calcium in the tissues leading to calcification of muscles, tendons and ligaments</p>	<p>Lameness, stiffness, recumbency, heart murmurs Decreased appetite and weight loss</p>	<p>Dx: history of exposure and clinical signs Typically chronic exposure with no recovery</p>	

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<p>False dandelion <i>Hypochaeris radicata</i></p>	<p>Unknown toxin</p>	<p>Unknown MOA</p>	<p>Horses develop a lameness called "stringhalt"</p> <p>Hypermetria and hyperflexion of the hind legs</p> <p>Difficulty stepping backwards</p>	<p>Dx: Clinical signs and history of exposure</p> <p>Tx: Horses typically recover over a period of months after exposure stops</p>	
<p>Sudan grass, Johnson grass <i>Sorghum spp.</i></p> <p>Pigweed <i>Amaranthus retroflexus</i></p> <p>Lamb's-quarters <i>Chenopodium album</i></p> <p>Alfalfa, oat, corn nightshades <i>Solanum</i></p>	<p>Nitrate</p> <p>*Only ruminants and pseudoruminants are susceptible</p>	<p>The nitrate in plants is converted into toxic nitrite which is then converted into methemoglobin. Methemoglobin causes oxygen to be incapable of transporting leading to anoxia</p>	<p>Acute syndrome: Onset ½ - 4 hours after feeding</p> <p>GI irritation, difficulty breathing, tremors, ataxia</p> <p>Rapid, weak heart beat</p> <p>Convulsions, death in 6-24 hours</p> <p>Abortion *especially in the last trimester</p> <p>Chronic syndrome: Results of field observations, abortions often 3-5 days after exposure</p>	<p>Dx: Clinical history and chemical analysis</p> <ul style="list-style-type: none"> -lots of deaths -change in feed / new hay -hungry animals <p>Samples</p> <ul style="list-style-type: none"> -multiple forage samples -eyeballs (intact and frozen) -serum or blood <p>Ddx: Pesticide poisoning (OP/carbamate insecticides)</p> <p>Cyanide, copper, red maple, ionophore poisoning, hypophosphatemia</p> <p>Tx: Methylene blue</p> <p>Dosage: 4-22 mg/kg IV in a 1-2% solution</p>	

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			Brown discoloration of blood and tissues	*Withdrawal times for milk and meat are required	 <p>Pigweed Sudan grass</p>
Macadamia Nuts	Unknown toxin	<p>Unknown MOA</p> <p>Only reported in dogs Toxic dose: 2.4g of nuts/kg bw</p> <p>5-40 nuts/dog leads to toxicosis</p>	<p>Within 12 hours of ingestion</p> <p>Weakness, especially of the hind limbs, tremors, stiffness</p> <p>Depression, vomiting</p>	<p>Dx: Clinical signs</p> <p>Tx: induce emesis; activated charcoal for larger ingestions</p> <p>Many dogs recover without specific treatment</p>	
<p>Lilies</p> <p><i>Lilium</i></p> <p>Tiger Lily</p> <p>Asiatic Lily</p> <p>Easter Lily</p> <p>Daylily</p>	Specific toxin unknown	<p>Specific MOA unknown but renal tubular epithelial cells are damaged</p> <p>Cats are believed to create a toxic metabolite when lilies are ingested leading to severe kidney injury</p> <p>Dogs experience GI upset</p>	<p>Salivation</p> <p>Vomiting</p> <p>Anorexia</p> <p>Depression</p> <p>Renal failure with initial polyuria then anuria within 2-3 days</p>	<p>Dx: Clinical signs, finding a chewed piece of the plant or parts of the plant present in vomit</p> <p>Evaluate kidney function via blood and urine</p> <p>Tx: Induce emesis, activated charcoal, IVF, dialysis in severe cases</p>	

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<p>Raisins and Grapes <i>Vitis vinifera</i></p>	<p>Unknown toxin</p>	<p>Acute renal failure due to unknown MOA</p> <p>Toxicity ~ 10g/kg</p>	<p>Initial signs: Vomiting (within 2 hours after exposure)</p> <p>Diarrhea, lethargy, polydipsia (within 5 hours)</p> <p>Acute Renal Failure (within 24 hours) anorexia, depression, tremors, hypovolemia, diarrhea, vomiting</p>	<p>Dx: clinical signs and lab work</p> <p>Tx: Decontamination (if recent ingestion)</p> <p>Fluid diuresis</p> <p>Hemodialysis or peritoneal dialysis</p>	
<p>Cycad Palms Sago palms <i>Cycas spp.</i> <i>Zamia spp.</i> <i>Macrozamia spp.</i></p>	<p>Cycasin</p> <p>Leaves, seeds, and roots are toxic (seeds are the most toxic)</p>	<p>Irritation of GI tract and hepatic necrosis in dogs and sheep</p> <p>Dogs: Ingestion of 1-2 seeds can be lethal, signs within 24 hours</p>	<p>Dogs: vomiting, diarrhea, depression, anorexia, liver failure</p> <p>Sheep: GI irritation, lethargy, anorexia, weight loss, death</p> <p>Cattle: ataxia, paresis, hepatic damage possible (neurotoxic amino acid)</p>	<p>Dx: Clinical signs and history of ingestion</p> <p>Tx: GI decontamination, activated charcoal</p> <p>Supportive and symptomatic care: GI protectants, fluid</p>	