



Guide to Plants of Kentucky Potentially Poisonous to Livestock

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Poisonous plants are responsible for considerable losses to farmers and livestock producers in Kentucky. Many cases of plant poisoning are never diagnosed or even suspected. There are nearly 100 different species of plants growing in Kentucky that under certain conditions may be poisonous to livestock, although only one third of these are likely to cause serious trouble. The primary purpose of this publication is to enable individuals to recognize some plants that are known to be dangerously poisonous and to have knowledge of those additional plants that, under certain conditions, may cause trouble.

Recognition of Plant Poisoning

Plant poisoning is often difficult to diagnose, since the signs can mimic infectious diseases or poisoning from nonplant sources. Plant poisoning may be suspected when there is a sudden onset of unexplained illness, acute disorders of the nervous system or the digestive tract, loss of weight, difficulty in breathing, weakness, coma, and collapse. Plant poisoning may also be suggested if animals have recently been turned out into a

new pasture or the forage source has changed, including a new batch from the same source. Poisonings are also common if animals are not provided sufficient feed/forage or a severe weather event has prevented animals from accessing normal sources. There is usually no fever, except with secondary infections in cases of bracken fern poisoning, or with plants that cause muscle tremors. If the animal dies, identifiable plant parts found in the animal's digestive tract at postmortem examination may lead to a presumptive diagnosis.

Call a Veterinarian

When plant poisoning in livestock is recognized or suspected, the first thing to do is call a veterinarian, since diagnosis and treatment of a poisoned animal must be done early if the animal is to be saved. Some first aid can be given before the veterinarian arrives. Place the affected animal where it is quiet and comfortable and where diagnosis can be made and treatment given. Treatment will be determined by observing the clinical signs and determining what poisonous plants could have been ingested. Other livestock should not be allowed in the pasture where the poisoning occurred nor fed questionable silage or hay until the cause of the poisoning has been determined.

Preventing Plant Poisoning

- Provide plenty of quality forage and avoid overgrazing. Most cases of plant poisoning are closely connected with a lack of suitable forage. When plenty of grass or hay is available, animals will usually avoid poisonous plants, which are often tough and unpalatable.
- Learn to recognize poisonous plants. Study the illustrations and descriptions in this publication until you are familiar with them. Make a thorough survey of your property, wherever stock is turned out to graze. Poisonous plants sometimes occur in the open pasture, but more frequently along fence rows, banks of streams and ponds, and in woodlands. Learn to recognize these plants in their early stages of growth. Cockleburs and some others are poisonous only as seedlings. Your local County Extension office can assist with plant identification.

- Avoid harvesting poisonous plants in hay. The field should be carefully examined before hay is cut.
- Never throw plant clippings or trimmings into livestock pastures. Although this publication focuses on plants typically found in pastures and wooded areas, many ornamental plants can also be poisonous to livestock.

Suggestions for Sending Plant Specimens for Identification

- Unknown plants suspected of being poisonous may be sent to your local County Extension office. When possible, submit the entire plant, including leaves, flowers, roots, fruits, and seeds.
- State the general structure or size of the plant and provide details about the specific site where it was collected, including whether the plant appears to be an herb, shrub, tree or vine and other characteristics, such as color of flowers, that can be useful for proper identification.
- If two or more kinds of plants are sent at the same time, each plant should have a separate identification tag attached to it.
- If plants cannot be sent in fresh condition, they should be pressed out flat and packed between pieces of cardboard before sending.

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Bracken Fern Family - *Dennstaedtiaceae*

Bracken Fern (*Pteridium aquilinum*)

Figures: 1, 2
Also called: Bracken, Brackenfern
Former names: *Pteridium latiusculum*,
Pteris aquiline

Description. This coarse-growing perennial fern has a stout, black, horizontal rootstock. The frond or leaf is broad, triangular, and divided into three main parts, each of which is twice subdivided. Reproduction occurs by spores borne in late summer on the lower edges of the mature fronds. It is common in open acid woodlands and high pastures of the state.

Conditions of Poisoning. Bracken fern contains several toxins, including ptaquiloside and thiaminase. Ptaquiloside concentrations are highest in the young fronds (fiddleheads), while thiaminase concentrations are highest in the rhizomes. Both green and dried plants are toxic. Ingestion of sufficient quantities of bracken fern, usually for several weeks or longer, are necessary to cause disease.

Signs in Ruminants. In ruminants, ptaquiloside is the primary toxin. Cattle that ingest large amounts of ptaquiloside over weeks to months can develop bone marrow suppression, platelet deficiency, coagulation problems, and hemorrhage (excessive bleeding). Animals may bleed excessively from minor scratches or insect bites. More severe cases can develop bleeding from the nose and mouth. Blood may also be observed in feces or urine. Anemic animals can develop weight loss, weakness, rapid breathing, and pale mucous membranes. Sheep are much less susceptible than cattle.

Ruminants ingesting smaller amounts of ptaquiloside over months to years are more likely to develop neoplasia (cancer). In cattle, tumors most often develop in the urinary tract, particularly the bladder. Bleeding tumors can cause bloody urine (enzootic hematuria, also called "red water"). Cattle also occasionally develop tumors in the upper digestive tract, and less commonly in the

rumen. Sheep often develop tumors of the jaw and digestive tract. Once symptoms of bleeding or neoplasia develop, ptaquiloside poisoning is generally fatal.

Sheep with bracken fern poisoning may also develop "bright blindness," a disorder characterized by progressive retinal degeneration, abnormal pupillary light response, and vision loss. Affected sheep may develop a wide-eyed, alert expression.

Signs in Monogastrics. The primary bracken fern toxin in monogastrics (e.g., horses and swine) is thiaminase. Thiaminase breaks down thiamine (vitamin B₁), which is required for normal functioning of the nervous system. Horses exposed to thiaminase may develop anorexia, weight loss, depression, constipation, incoordination, paralysis, and a crouching stance with splayed feet. When forced to move, animals may tremble. Severely affected animals can develop cardiac arrhythmias and convulsions, and they usually die. Pigs are much more resistant, only developing signs when forced to subsist primarily on bracken fern. Signs in pigs are less distinct, often presenting as sudden death. Other signs in pigs can include weakness, weight loss, sudden onset of difficulty in breathing, and recumbency (inability to rise).

Treatment. Repeated administration of thiamine can be effective in thiamine-deficient horses if an early diagnosis is made. Disease can develop days or weeks after removal from the bracken fern, therefore all exposed animals should be treated, even if asymptomatic. Bracken fern poisoning is controlled by preventing access to bracken fern in pastures and checking hay for bracken fern contamination.

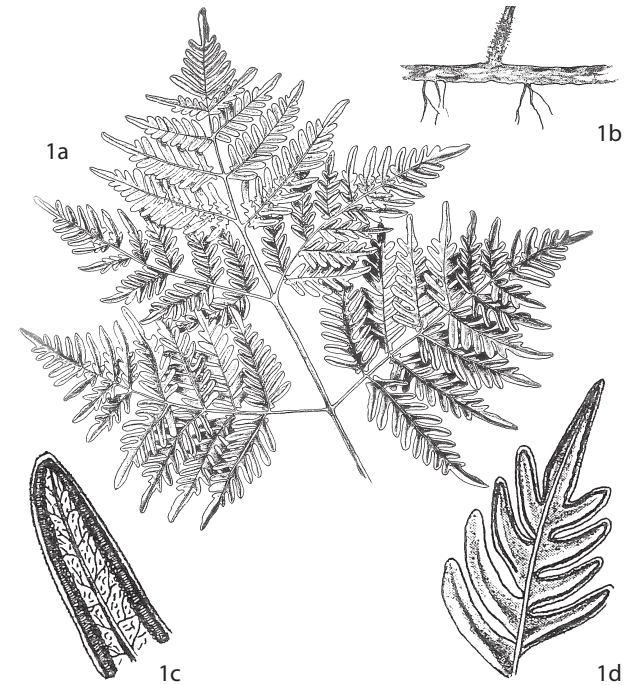


Figure 1. Leaf (a) and rootstock (b). Detail showing spore case along margin on underside of leaf (c, d).



Figure 2.

Horsetail Family - *Equisetaceae*

Horsetail (*Equisetum arvense*)

Figures: 3, 4
Also called: Field Horsetail, Scouring Rush

Description. Shoots are of two types. Both are round, hollow, and jointed, arising from a perennial, creeping, underground rootstock. One type of shoot is tan and appears in early spring. It bears the reproductive spores in a terminal, cone-like structure. The other type is sterile and appears later, bearing whorls of pine-needlelike branches. Small, toothlike leaves are arranged in whorls around the stem at the joint.

Horsetail is common on sandy, moist soil; in meadows; and along roadside ditches, stream banks and railroad embankments.

Conditions of Poisoning, Signs, and Treatment. See *Scouring Rush* on page 6.

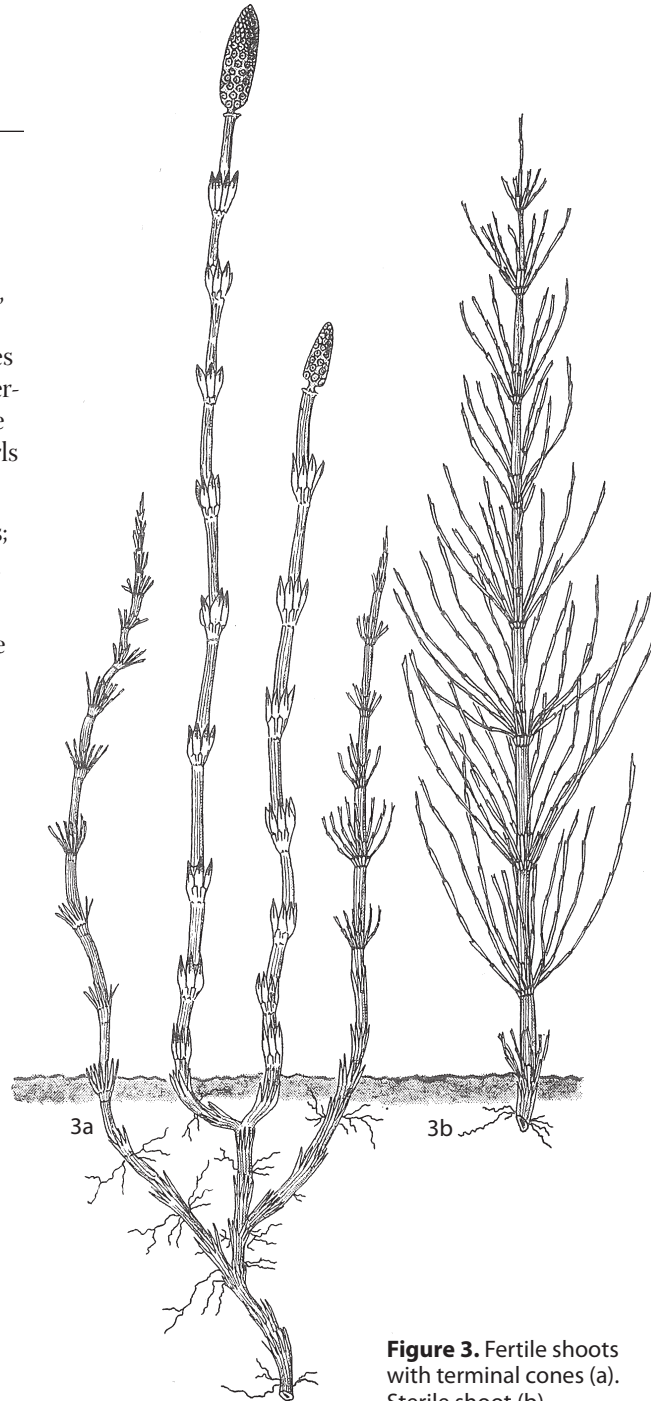


Figure 3. Fertile shoots with terminal cones (a). Sterile shoot (b).



Figure 4.

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Horsetail Family - *Equisetaceae*

Scouring Rush (*Equisetum hyemale*)

Figures: 5, 6
Also called: Rough Horsetail

Description. Scouring rush shoots are similar to horsetail shoots, but form long, tapering, cane-like stalks, one to six feet high. Stems are stiff and evergreen, with terminal spore-bearing cones. Small, toothlike leaves are arranged in whorls around the stem at the joints. Scouring rush is common in wet localities and thickets; along streams, roadside ditches, and borders of swamps; and in mountain sections of the state.

Conditions of Poisoning. Horsetails and scouring rushes contain thiaminase (see *Bracken Fern* on page 4). Fresh *Equisetum* species are not palatable, so poisoning is unlikely from fresh plants unless animals are starving. Poisoning occurs more often in horses that ingest hay contaminated with 20 percent or more of *Equisetum*. Animals must typically ingest *Equisetum* for three to four weeks before developing clinical signs. Cattle are rarely affected.

Signs. Horses develop anorexia, weight loss, incoordination, and muscle tremors. Signs may worsen over several days to weeks and can include paralysis of the rear legs, inability to rise, terminal convulsions, and death.

Treatment. Repeated administration of thiamine is effective. Full recovery is expected except in the most advanced cases.

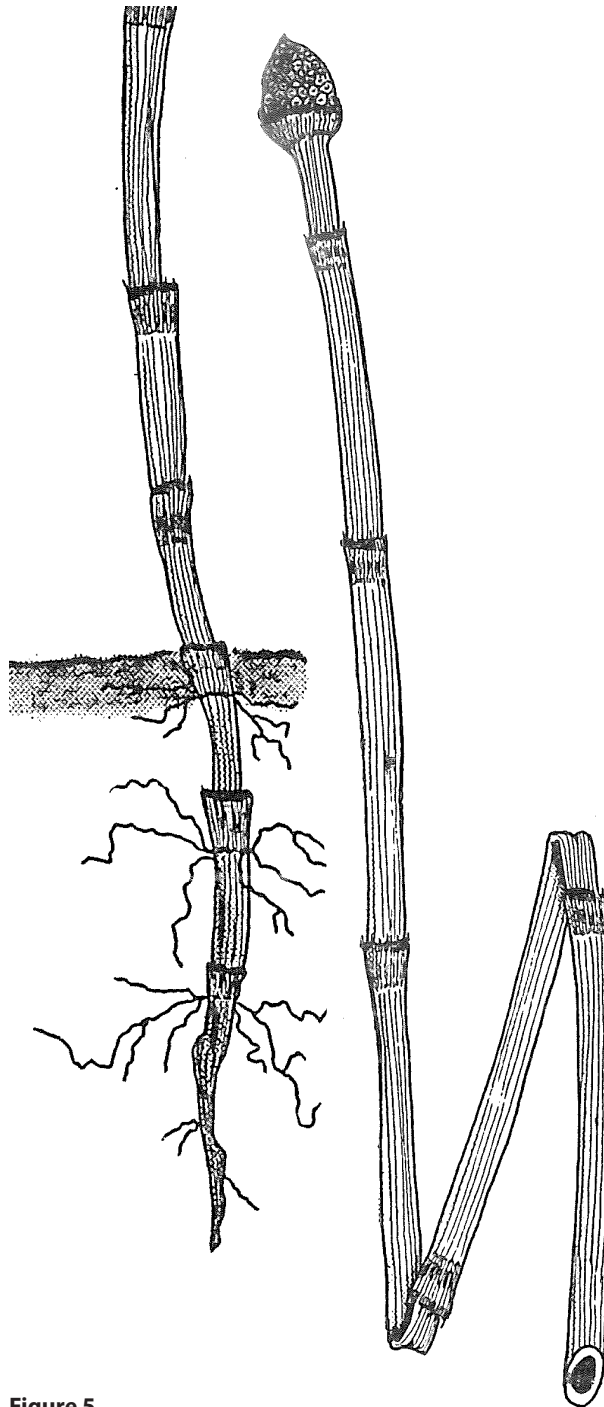


Figure 5.



Figure 6.

Lily Family - *Liliaceae*

Star-of-Bethlehem (*Ornithogalum umbellatum*)

Figures: 7, 8

Also called: Snowdrop, Nap-at-Noon

Description. Star-of-Bethlehem is a perennial that reproduces mostly by bulbs and rarely by seed. It may be confused with wild garlic, which belongs within the same plant family. However, Star-of-Bethlehem does not have the strong scent of onion or garlic. The leaves are about as long as the stem and have a light green midrib. The stems develop from small bulbs that usually grow in clumps. Plants grow to a height of four to 12 inches, and bear several white, star-shaped flowers.

An escaped ornamental that can crowd out desirable grasses and other desirable vegetation, Star-of-Bethlehem shows new growth starting in late January or early February in Kentucky. The plant grows to maturity and develops flowers in May or early June. The tops then die back, leaving brown areas.

Conditions of Poisoning. All parts of the plant contain a variety of cardenolides, with the highest concentrations in the bulbs. Plants are poisonous both when fresh and in hay. Poisonings, which are rare in North America, are most common in sheep.

Signs. Clinical signs relate primarily to the digestive system. Affected animals often develop severe diarrhea that may be fetid, watery, and bloody. Additional signs can include inappetence, depression, colic, and increased heart and respiratory rates. Death can occur rapidly, especially if bulbs are ingested.

Treatment. Activated charcoal may help decrease absorption of the toxin. Animals should be kept calm and quiet, and stress should be minimized.

Figure 7. Bulbs (a), leaf showing light green midrib (b), and stem with star-shaped flower (c).

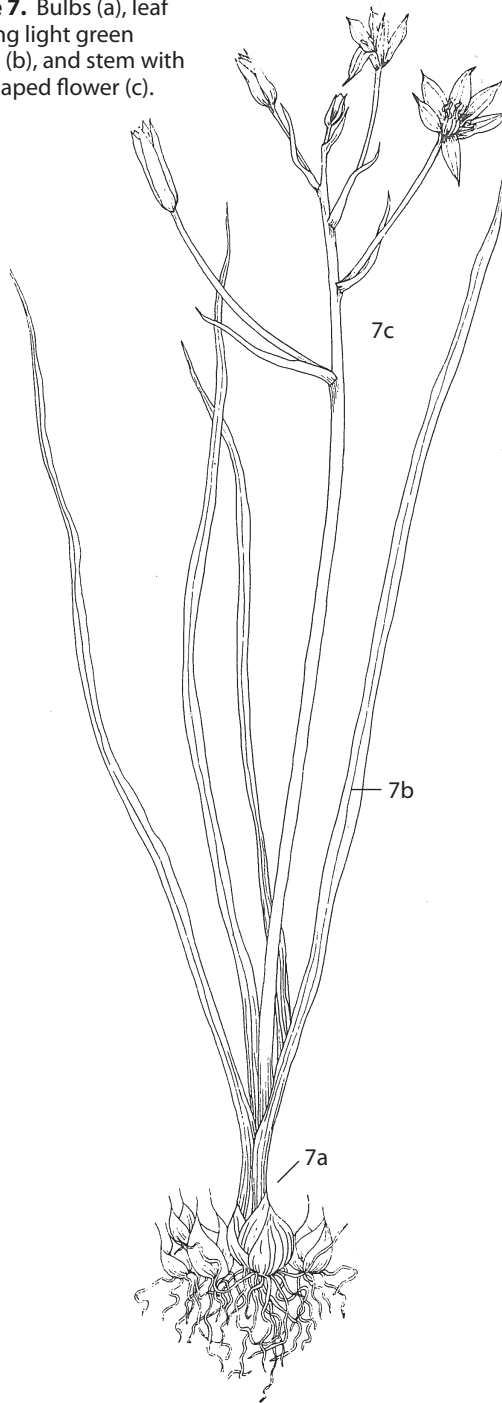


Figure 8.

Grass Family - *Poaceae*/*Gramineae*

Johnsongrass (*Sorghum halepense*)

Figures: 9, 10

Description. A warm-season, perennial grass, Johnsongrass can grow three to six feet high, with scaly, creeping rhizomes. Seed heads form long, open terminal panicles.

All three *Sorghum* species listed in this guide can be cultivated as forage crops, but Johnsongrass is primarily considered a weed in Kentucky, since it can spread rapidly into cultivated fields. Once established, it is hard to eradicate because of its rhizomes (underground stems) and long-lived seed.

Conditions of Poisoning. See *Sudan Grass* on page 10.

Signs and Treatment. See *Cyanide Poisoning* on page 55 and *Nitrate Poisoning* on page 54.

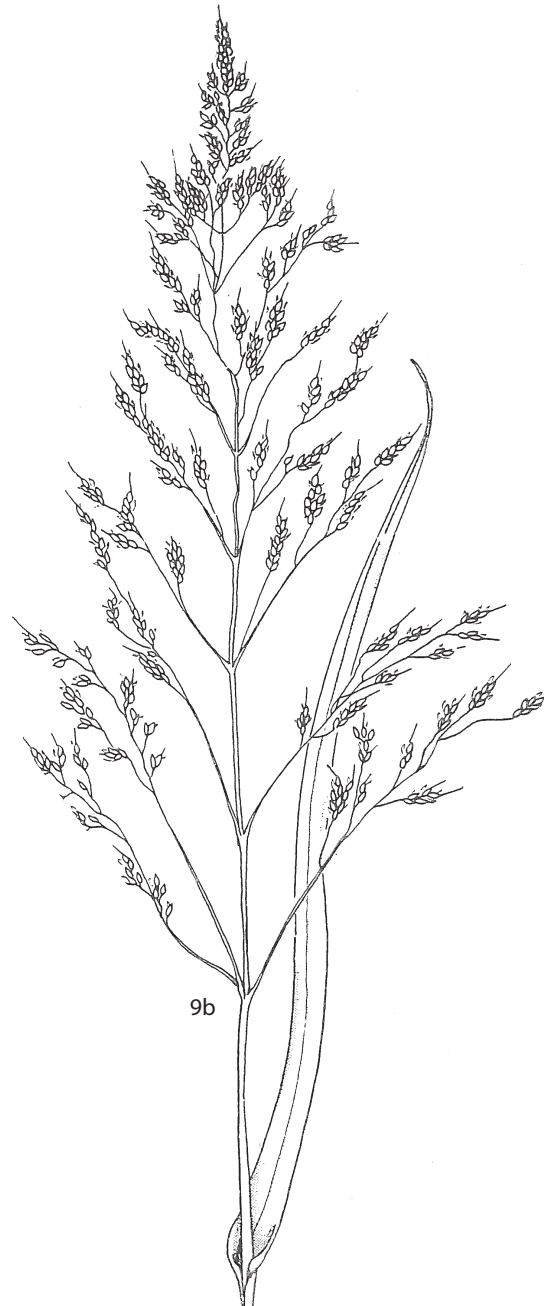
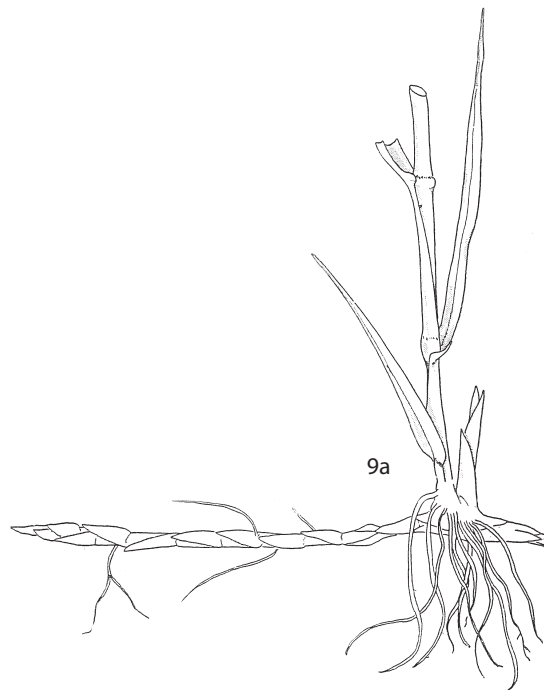


Figure 9. Parts of the plant showing rhizomes (a) and the flowering portion of the plant (b).



Figure 10.

Grass Family - *Poaceae*/*Gramineae*

Sorghum (*Sorghum bicolor*)

Figure: 11
Also called: Shattercane, Wild Cane
Former name: *Sorghum vulgare*

Description. Sorghum is a medium-to-tall, warm-season, annual grass with seed heads that are somewhat similar in appearance to Johnsongrass, but more compact.

Conditions of Poisoning. See *Sudan Grass* on page 10.

Signs and Treatment. See *Cyanide Poisoning* on page 55 and *Nitrate Poisoning* on page 54.



Figure 11.

Grass Family - *Poaceae*/*Gramineae*

Sudan Grass

(*Sorghum bicolor* ssp. *drummondii*)

Figure: 12
Subspecies of: Sorghum
Synonym: *Sorghum vulgare* var. *sudanese*

Description. An annual grass, Sudan grass is similar in appearance to sorghum and johnsongrass. Neither sorghum nor Sudan grass has scaly, creeping rhizomes.

Conditions of Poisoning. *Sorghum* species (e.g., Johnsongrass, sorghum, Sudan grass and hybrid sorghum-Sudan) contain dhurrin, a cyanogenic glycoside. When plant cells are crushed, chewed, wilted, frozen, chopped, or damaged (e.g., by frost, insects, drought, herbicides, and other causes), plant enzymes cleave dhurrin, releasing cyanide gas (HCN). Dhurrin concentrations are highest in the leaves, particularly new growth. Peak concentrations occur in the first week after germination, declining markedly once the plant reaches approximately two feet in height. Regrowth (for example, after a light frost) contains extremely high dhurrin concentrations similar to newly germinated seedlings; it may be several weeks before the risk subsides. Heavy frost periods, in which plants are killed, are typically safe several days after the plants thaw, turn brown and die.

Sorghum species are also prone to accumulating nitrate when heavily fertilized (see *Nitrate Poisoning* on page 54).

Signs and Treatment. See *Cyanide Poisoning* on page 55 and *Nitrate Poisoning* on page 54.



Figure 12.

Tall Fescue (*Lolium arundinaceum*)

Figure: 13
Synonym: *Schedonorus arundinaceus*, *Festuca arundinacea*

Description. A cool-season perennial grass, tall fescue exhibits a bunch-type growth habit with short rhizomes. Plants grow two to four feet tall during the seed head stage of growth in the spring and can also produce vigorous growth in the fall. The shiny, dark green leaves contain prominent veins.

Tall fescue is found throughout Kentucky in pastures, hay fields, turf areas, and most other grassland sites. It can tolerate soil acidity, low fertility, and poor drainage, and it is relatively tolerant to overgrazing and drought conditions.

Conditions of Poisoning. Endophyte-infected tall fescue contains various ergopeptine alkaloids produced by the endophyte. Ergovaline is the primary alkaloid in tall fescue, accounting for approximately 90 percent of the alkaloid content. These alkaloids confer many benefits to the plant (e.g., increased vigor, pest- and drought-resistance, and tolerance to many adverse soil and environmental conditions). The highest concentrations of ergovaline are in the seeds. Hay from infected fields may remain high in ergovaline, although concentrations generally decrease compared to the fresh plant.

Signs in Ruminants. In cattle, ergovaline causes vasoconstriction and decreased blood supply to the skin and extremities. Clinical signs depend on ambient temperatures and are less severe at moderate temperatures. In hot weather, reduced blood flow to the skin's surface limits the animal's ability to cool itself. Affected cattle develop signs of "summer slump," including poor growth or weight loss; decreased feed intake; a dull, rough hair coat; failure to shed the winter hair coat; slobbering; open mouth and/or rapid breathing; and an increased susceptibility to heat stress. Animals avoid grazing during the day, instead seeking shade, ponds, or

mud wallows to find relief from heat. In cold weather, the limited blood flow can cause necrosis (dry gangrene) of the extremities, including the lower limbs, ear tips, and tail tips. The condition first appears as swelling and redness at the coronary band, progressing to shifting hind-limb lameness, weight loss, and finally dry gangrene of the hooves (fescue foot). In severe cases, the affected portions of the hoof slough off.

Fat necrosis has been observed with chronic (long-term) fescue toxicosis. Masses of hardened fat in the abdominal and/or pelvic cavities can obstruct the birth canal and contribute to dystocia (difficult birth) and intestinal blockage. This condition is sometimes diagnosed with rectal palpation.

Ergopeptine alkaloids can cause adverse effects on both male and female reproductive function, including delayed puberty, reduced conception rates, altered sperm motility, ovarian follicle abnormalities, and decreased milk production. Decreased feed consumption may also contribute to poor reproductive function, especially when accompanied by heat stress.

Signs in Horses. Ergovaline poisoning in horses causes primarily reproductive effects, the most catastrophic of which are in late-term (third trimester) pregnant mares. Ergovaline causes prolonged pregnancies during which the foal continues to grow larger. Severe dystocias can ensue, with extensive trauma and death of both the foal and the mare. Premature placental separation ("red bag"), dysmature ("dummy") foals, and agalactia (lack of milk production by the dam) are common. In addition to the effects in late-term mares, ergovaline can cause failure of mares to cycle normally, reduced conception rates, and early embryonic death.

Treatment. Remove animals from the infected pasture or hay. Clinical effects can persist for weeks to months after removal of the ergovaline source. In late-term pregnant mares, domperidone is effective at treating agalactia and prolonged pregnancy. Treatment with domperidone is ineffective in early pregnant mares and in cattle, likely because these effects are due to different mechanisms. In those cases, prevention and symptomatic care are key. Provide cattle with shade, fans,

sprinklers, and other heat-stress mitigation measures in hot weather. Keeping pastures mowed helps minimize exposure to the high-concentration seed heads. Intensive grazing can also limit ergovaline production. Long-term measures include diluting pastures with other grasses or legumes, using warm-season grass pastures in late spring and early summer instead of fescue, and replanting endophyte-infected pastures with cultivars infected with novel (friendly) endophyte seed. Although endophyte-free tall fescue cultivars exist, they are quickly outcompeted by endophyte-infected plants due to the benefits conferred by the endophyte alkaloids.



Figure 13.

Buckwheat or Smartweed Family - *Polygonaceae*

Curly Dock (*Rumex crispus*)

Figure: 14
Also called: Yellow Dock

Description. A perennial herb, curly dock reproduces from seed or a taproot that forms a basal rosette during late summer through early spring. Flower stalks, which emerge in the spring, may reach five feet in height. Rosette leaves are dark green, sometimes with a purple tint, with wavy margins, and containing petioles. Stem leaves become progressively smaller up the flowering stalk, arranged alternately along the stem. Where the leaf petioles attach to the stem, a membranous sheath (ocrea) circles the stem. Flowers occur in clusters on the upper portion of elongated stems, consisting of small, greenish sepals that become reddish-brown as they mature. The fleshy taproot can become large and is often yellowish-orange.

Conditions of Poisoning. *Conditions and signs of poisoning for curly dock can also apply to other soluble oxalate-containing plants (e.g., beets, rhubarb, halogeton, lamb's quarter).*

Soluble oxalate concentrations are highest in rapidly growing plants and are higher in the leaves than in seeds and stems. Poisoning is rare but has been reported in sheep, cattle, and horses. Large amounts must be consumed in a short period of time for poisoning to occur. Fresh plants have a tart taste, and they are rarely eaten in sufficient quantities to cause a problem.

Rumex species can also accumulate nitrates. (See *Nitrate Poisoning* on page 54.)

Signs. Soluble oxalates form complexes with calcium (calcium oxalate crystals), causing hypocalcemia (low blood calcium) and kidney damage. Depression, weakness, tremors, excess salivation, bloat, staggering gait, collapse, and labored breathing can develop, usually within 12 hours. If left untreated, death can occur in

hours or up to five days. Digestive tract irritation can also occur from anthraquinone glycosides.

Treatment. Prompt administration of intravenous calcium is necessary to reverse hypocalcemia and related

effects. Repeated administration may be necessary, as relapses may occur. Supportive care, including intravenous fluids, is also beneficial. Chronic kidney failure is possible if the animal sustains severe kidney damage.



Figure 14.

Spiny Amaranth (*Amaranthus spinosus*)

Figure: 15
Also called: Spiny Pigweed

Description. Spiny amaranth is a branching summer annual that grows one to five feet tall. Relatively small (one to two inches in length), ovate leaves with leaf petioles are arranged alternately along the stem. Paired spines can be found at the base of the leaf petioles. The stems are smooth (without hairs) and often red to reddish-purple. Flower-containing seed heads occur at the ends of stems and in small clusters in the leaf axils that contain the spines, where the leaf petioles meet the stem.

Spiny amaranth is more commonly found in pastures and hay fields, particularly in areas grazed or utilized heavily by livestock; it is less often observed in agronomic crops and turf grasses. Occasionally other pigweed species such as smooth pigweed (*Amaranthus hybridus*) may also be found in grazed pasture areas.

Conditions of Poisoning. Amaranth contains unknown toxic compounds in the leaves and grains. Cattle, sheep, and pigs can develop kidney disease after large amounts of fresh, green plants are eaten for five to 10 days. Problems most often occur in late summer and fall, especially if animals are introduced into weedy pasture. Some animals seem predisposed to seek out the weed and consume it. Heart failure can occur in pigs fed large amounts of the grains. Horses are rarely affected.

Pigweeds are soluble oxalate accumulators, although the clinical signs and pathology of amaranth poisoning are not consistent with oxalate poisoning. (See *Curly Dock* on page 12.) Under certain conditions, pigweeds can also accumulate nitrates. (See *Nitrate Poisoning* on page 54.)

Signs. Cattle, sheep, and pigs can develop kidney disease after large amounts of fresh, green plants are eaten

for five to 10 days. Signs can include weakness, trembling, unsteadiness, incoordination, knuckling, diarrhea with or without blood, paralysis and death in as few as one to two days. Surviving animals often develop fibrosis (scarring) and decreased kidney function. Pigs fed large amounts of the seeds in grain can develop acute cardiovascular disease and sudden death.

Treatment. Remove from the source. Intravenous fluids and other supportive care may be necessary to allow recovery of kidney function. Recovery is slow, requiring several weeks or more. Prognosis for full recovery is poor, and prevention relies on avoiding exposure.



Figure 15.

Pokeweed Family - *Phytolaccaceae*

Pokeweed (*Phytolacca americana*)

Figures: 16, 17
Also called: Poke, Pokeberry, American Pokeweed

Description. A tall perennial herb, pokeweed produces a large taproot. Stems are succulent, smooth, and purplish, growing three to 10 feet high. Alternate leaves are pointed and narrowly oblong, with smooth margins. Small, white flowers are borne in long, drooping clusters. Fruits are flattened, spherical, dark-purple berries, usually with 10 seeds.

Pokeweed is common on recently cleared land, in open woods and pastures, and along fencerows.

Conditions of Poisoning. All plant parts, especially the roots and seeds (berries), contain numerous digestive tract irritants. All species of animals are susceptible. Approximately four pounds per head of green plant has caused symptoms in dairy cattle.

Signs. The most common sign of pokeweed poisoning is diarrhea, which can be severe and last 24 hours or longer. Diarrhea typically develops within three to four hours of ingestion in horses and pigs, and slightly longer (six to eight hours) in ruminants. Other signs can include oral irritation and ulceration, excessive salivation, moderate to severe gastroenteritis, vomiting/regurgitation, and colic. Death can occur if very large amounts are consumed. Pigs may dig up and consume the roots of the plant; these animals can develop incoordination, sedation, seizures, paralysis, and death.

Treatment. Activated charcoal may decrease absorption of toxins. Cathartics should not be used in animals with diarrhea. Fluids with electrolytes can be administered orally or intravenously to correct dehydration and electrolyte abnormalities.

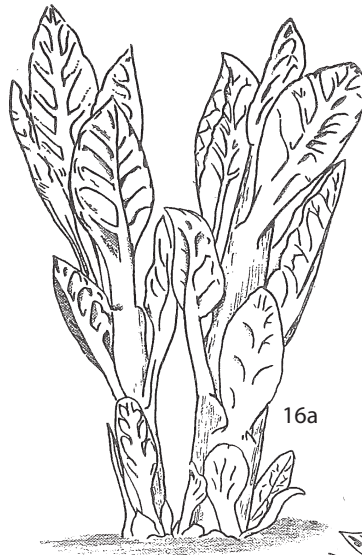


Figure 16. Young shoots (a). Part of mature plant with flowers and fruits (b). Tap root (c).

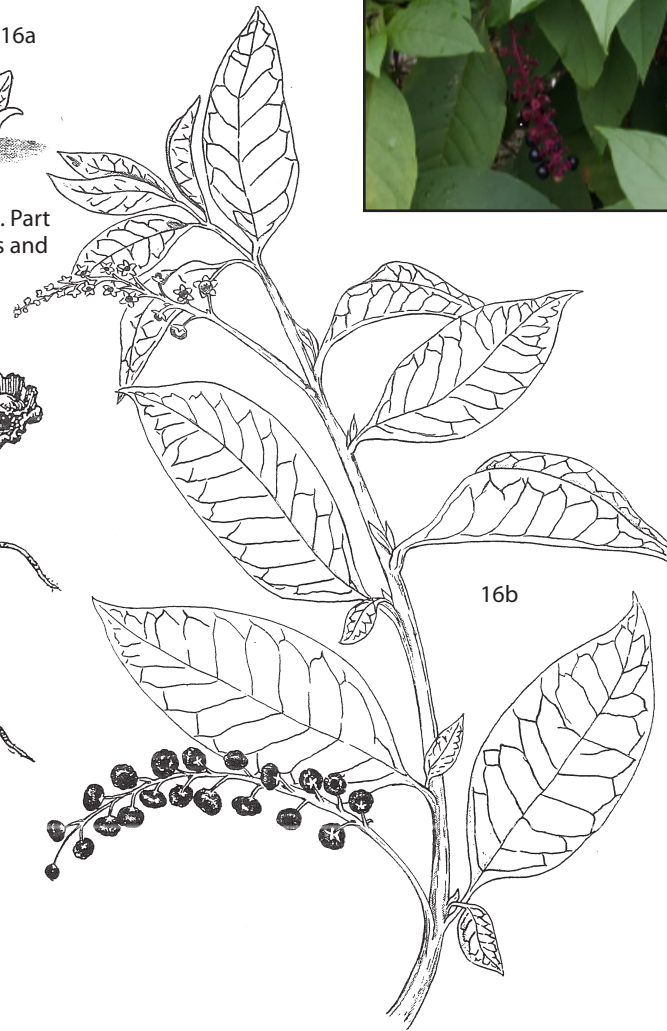
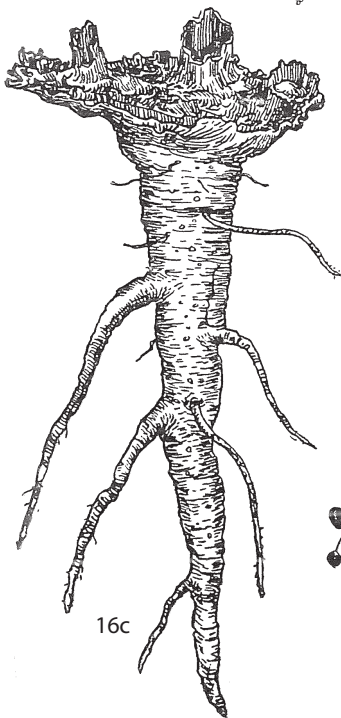


Figure 17.

Crowfoot or Buttercup Family - *Ranunculaceae*

Buttercup (*Ranunculus* spp.)

Figure: 18

Description. Different species of buttercup can be found in Kentucky. Most buttercups can grow four to 10 inches tall and have a fibrous root system. Leaf shapes, which are highly variable, can be slightly lobed to deeply dissected among the different species and can vary on the same plant depending on maturity. Flowers consist primarily of five glossy yellow petals and five green sepals.

Buttercups primarily grow throughout the winter and spring months, producing flowers in May and early June. They are often found in heavily grazed pastures and other areas where vegetative cover is sparse during the fall, winter, and early spring.

Conditions of Poisoning. Fresh buttercup leaves, flowers, and stems have a sharp, pungent taste and are usually avoided by grazing animals. Problems generally occur when other forage is unavailable and animals are forced to consume large quantities of buttercups. Buttercups contain ranunculin, which is converted by plant enzymes to protoanemonin when the plant is chewed or crushed. The toxin is present in stems and leaves, and concentrations are particularly high in flowering plants. Protoanemonin is detoxified upon drying, so hay contaminated with buttercups is of minimal risk. Ensiling may also decrease the toxicity of buttercups, although limited information is available.

Signs. Protoanemonin causes blistering of the skin, mouth, and digestive system on contact. All species are susceptible to the irritant compound, although horses are generally the most sensitive to gastrointestinal effects. Signs can include irritation, reddening, and blistering of the lips, skin, mouth and digestive system; drooling; mouth pain; redness and swelling of the muzzle, lips, and face; diarrhea; gastric ulcers; and abdominal pain. Affected animals may go off feed completely if chewing and swallowing becomes too pain-

ful. Signs can be severe if large quantities are ingested, but the acrid taste and immediate discomfort usually deters further grazing in horses and cattle. Protoanemonin is excreted in milk and may confer to it a bitter taste. Although one report suggested a possible link between buttercup consumption and abortion in cattle and horses, attempts to reproduce abortions by feeding large quantities of buttercups have not been successful in either species. The suggested association remains unconfirmed and highly unlikely.

Treatment. Treatment is rarely necessary. In horses, intestinal protectants such as sucralfate may be beneficial if gastrointestinal discomfort is severe. In cattle, laxatives like mineral oil can speed gastrointestinal transit time, but they should not be used in animals with diarrhea. Prevention is based on maintaining appropriate stocking rates. Buttercups proliferate in overgrazed pastures, and problems are typically seen only when other forage is unavailable.



Figure 18.

Dwarf Larkspur (*Delphinium tricorne*)

Figures: 19, 20
Also called: Spring Larkspur

Description. A stout perennial, dwarf larkspur can grow from four inches to three feet high. Alternate leaves are very deeply lobed. The flowers are spurred and blue, or occasionally white. Flowers are arranged in terminal clusters, appearing in spring. The root is a tuberous cluster.

Plants are commonly found in rich, open woods and along streams. The annual larkspur (*Delphinium ajacis*), which often escapes from gardens and establishes itself as a weed in fields and roadsides, is also poisonous.

Conditions of Poisoning. The principal toxins in larkspurs are diterpene alkaloids, which cause paralysis of skeletal, cardiac, and smooth muscles. Dried plants remain toxic. Cattle are most susceptible to larkspur poisoning. Sheep and horses can also be affected, but they appear to require larger amounts of plant material. Toxic doses are difficult to establish, as alkaloid content and palatability can fluctuate dramatically over the growing season.

Signs. Cattle may develop tremors, staggering, falling, bloat, and difficulty in breathing within three to four hours of ingestion. Weakness can be episodic, with animals regaining their feet after a period of rest. Fatal bloat may develop, especially in recumbent animals facing downhill. Death can also occur due to respiratory muscle paralysis. In milder cases, animals can recover with good supportive care.

Treatment. Animals facing downhill and unable to rise should be turned to face uphill to decrease risk of bloat. Supportive care may include bringing feed and water to recumbent animals. Atropine has been suggested as a treatment, but the stress of handling a symptomatic animal may worsen rather than improve signs.



Figure 19. Entire plant, showing flowers (a), lobed leaves (b), and tuberous roots (c).



Figure 20.

Fumitory Family - *Fumariaceae*

Squirrel Corn (*Dicentra canadensis*)

Figures: 21, 22

Description. An herbaceous perennial, squirrel corn can grow up to one foot tall, with finely cut, fernlike leaves. Small, yellow, pea-like tubers are arranged along the underground stem. Slender stalks bear up to 10 creamy white flowers, each with two short, rounded projections.

Conditions of Poisoning, Signs, and Treatment. See *Dutchman's Breeches* on page 18.

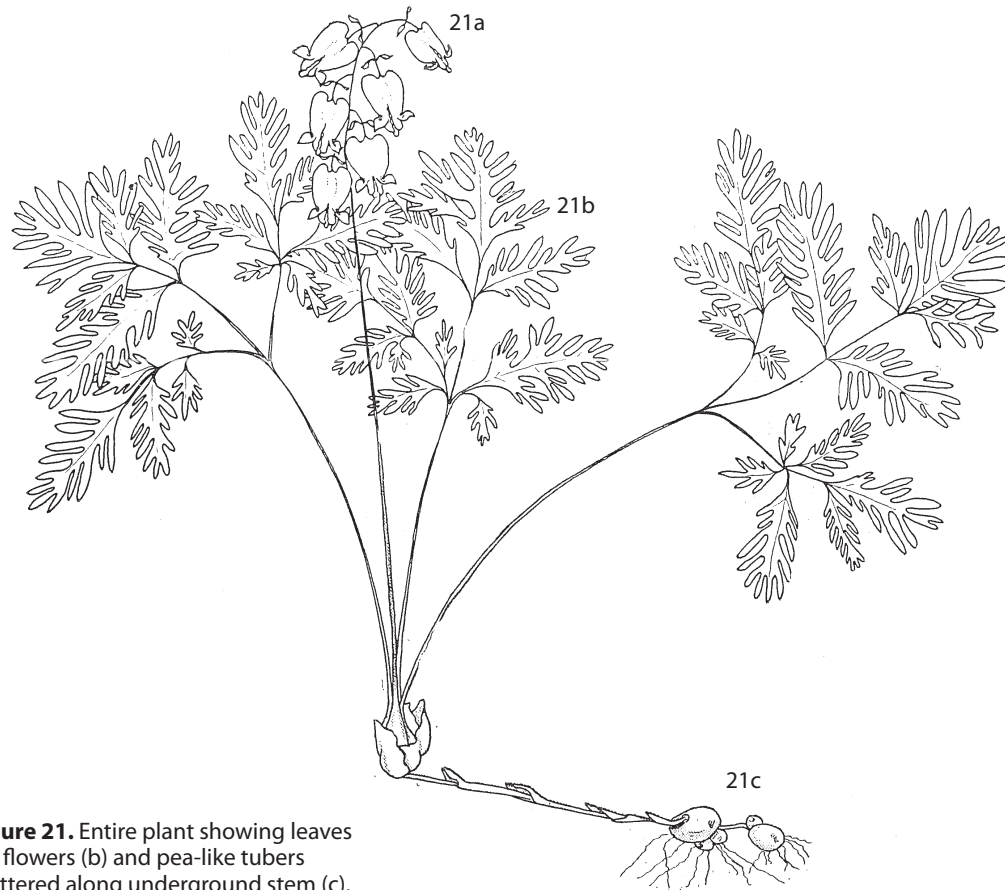


Figure 21. Entire plant showing leaves (a), flowers (b) and pea-like tubers scattered along underground stem (c).



Figure 22.

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Fumitory Family - *Fumariaceae*

Dutchman's Breeches (*Dicentra cucullaria*)

Figures: 23, 24

Description. Closely resembling squirrel corn, Dutchman's breeches plants have small, grain-like tubers clustered at the base of the stem. The flowers have two spur-like projections.

Both species are among the earliest of spring plants, blossoming in April or May. They are common in rich, open woods, often in areas associated with dwarf larkspur.

Conditions of Poisoning. Both *Dicentra* species contain a number of toxic alkaloids, although *D. cucullaria* (Dutchman's breeches) is more poisonous than *D. canadensis* (squirrel corn). Although the plants are palatable and readily eaten, plant populations are seldom sufficiently abundant to cause disease. Poisoning is rare but can occur in early spring (April or May) when animals are grazing in wooded areas. Problems are reported most often in cattle and sheep.

Signs. Affected animals can develop episodes of muscle tremors, staggering (sometimes called spring staggers), frothy salivation, labored breathing, and seizures. The animal often returns to normal between episodes. Several episodes can occur before the animal completely recovers or, in very rare instances, dies.

Treatment. There is no specific treatment except removal from the area. Poisoning is rare, and signs are generally self-limiting.



Figure 24.

Legume or Pea Family - *Fabaceae/Leguminosae*

Hairy Vetch (*Vicia villosa*)

Figure: 25
Also called: Winter Vetch

Description. Hairy vetch, with its vine-like growth, can grow as a trailing winter annual, biennial, or summer annual forming large mats of vegetation. Stems grow up to three feet long. Leaves are compound with five to 10 pairs of oblong leaflets per individual leaf. Tendrils form on the ends of the leaves. Purple flowers form in early summer to midsummer.

Conditions of Poisoning. Hairy vetch has been associated with two distinct clinical syndromes in North America. Ingestion of seeds has been reported to cause neurologic signs in cattle. Grazing vetch foliage has been associated with a sporadic hypersensitivity (allergic type) reaction. Hypersensitivity occurs primarily in mature cattle, and it does appear to have a genetic link. Several weeks of grazing hairy vetch is typically necessary for animals to develop signs, although Holstein and Angus cattle have been reported to develop signs after ingesting vetch for less than two weeks. Signs may also become apparent weeks after cattle have been removed from the vetch pasture. The disease is more severe in cattle over three years old, although it can still be fatal in younger animals. Hairy vetch is frequently consumed by cattle without apparent problems, further supporting the theory of allergic and/or genetic factor involvement. The plant appears less problematic in hay or when ensiled.

Signs. Ingestion of the seeds can cause neurologic signs in cattle similar to rabies. Animals may bellow, develop seizures, and die suddenly. Ingestion of vetch forage can result in a systemic granulomatous disease in cattle and horses. Animals can develop dermatitis with itching, hair loss, oozing, crusting, and/or scaling of affected skin; diarrhea; weight loss; sporadic abortions; and red-tinged urine. Additional signs in horses can include conjunctivitis, corneal ulceration, enlarged lymph

nodes, and dependent edema. The interval between the appearance of signs and death ranges from three days to five weeks, but it is usually between 10 and 20 days. Severely affected animals are unlikely to recover, although they may briefly appear to improve before relapsing. The extensive inflammation can affect multiple body systems, causing cardiovascular failure, kidney failure, or permanent loss of condition and emaciation.

Treatment. Animals should be removed from vetch pastures immediately if skin lesions are observed. Prevention strategies are limited due to the sporadic nature of the disease. Treatment may include pain relief, anti-inflammatory medications, and other symptomatic and supportive care.



Figure 25.

Black Locust (*Robinia pseudoacacia*)

Figures: 26, 27

Description. Black locust is a medium-sized tree, often with two short spines at the base of the leafstalk. The bark is rough, and leaves are alternate and pinnately compound. Individual leaflets are oval without teeth. Creamy white, fragrant flowers are similar to sweet peas and arranged in long, drooping clusters. The fruit is a flat, brown pod, one-half inch wide and two to four inches long, containing four to eight small, kidney-shaped beans.

Black locust grows commonly in woods and thickets. It is often planted as an ornamental and for erosion control, but it has spread widely as a weed tree along highways and in waste places.

Conditions of Poisoning. Black locust trees contain robinine, a toxalbumin. The highest concentrations are in the inner bark, and multiple cases have occurred due to horses stripping the bark from black locust trees. Young sprouts, leaves (especially wilted), pods and seeds can also cause problems if ingested in sufficient quantities. Horses are most susceptible to poisoning, although cattle, sheep, and goats can also be affected.

Signs. Horses can develop gastrointestinal signs, including anorexia, colic, and watery diarrhea, within an hour or two of ingestion. Signs can progress to dehydration, weakness, posterior paralysis, head pressing, absence of pupillary light reflexes, cardiac arrhythmias, and cold extremities. Horses can develop dilated pupils, cardiac arrhythmias, and eventually laminitis. Cattle may become belligerent.

Treatment. Recovery is possible, but it can take several weeks. Intravenous fluids are necessary to correct dehydration and prevent death from hypovolemic shock. Activated charcoal and anti-diarrheal agents may also be helpful if given early.

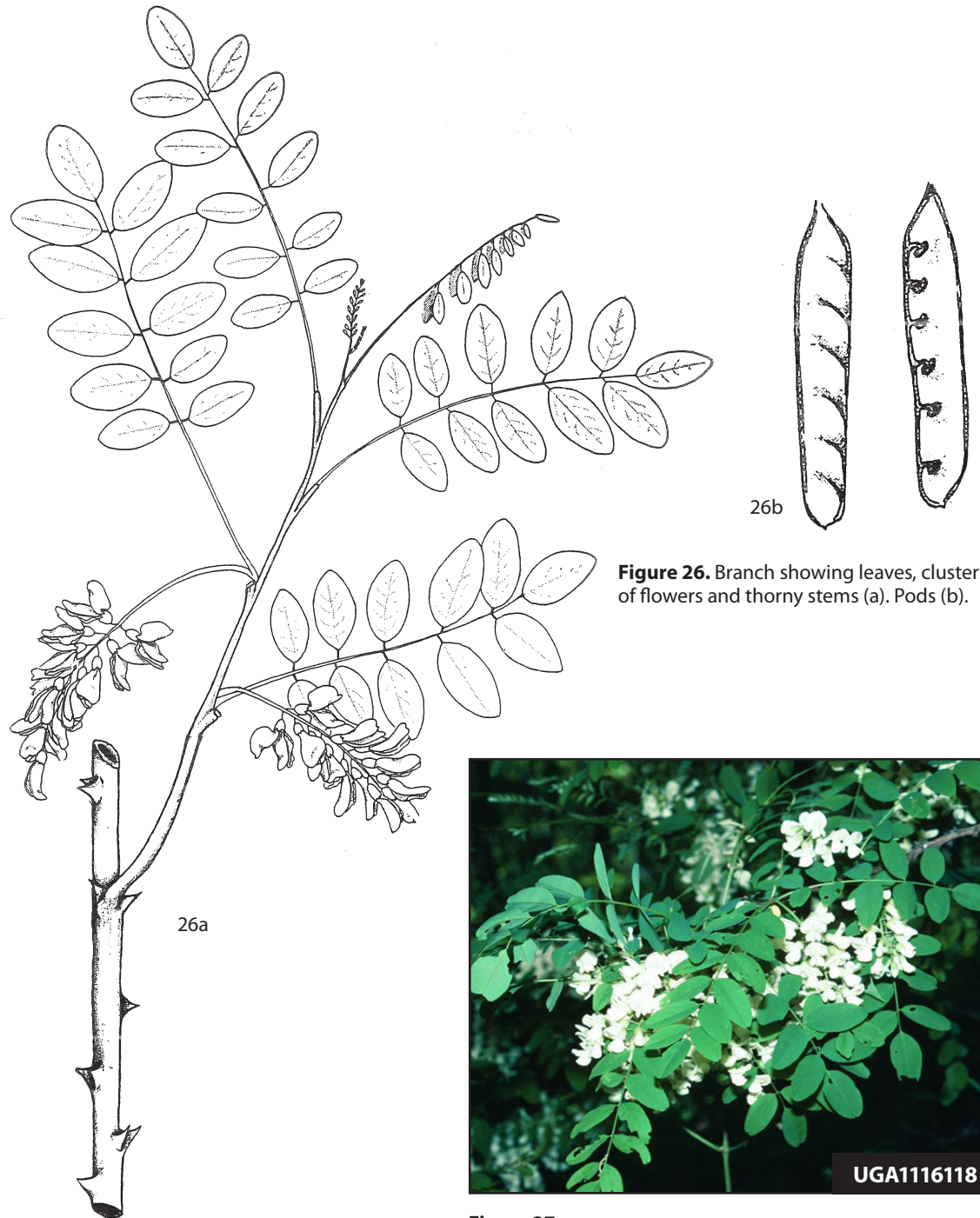


Figure 27.

Kentucky Coffee Tree (*Gymnocladus dioica*)

Figures: 28, 29

Description. The Kentucky coffee tree is a large tree, ranging from 60 to 80 feet in height, with rough bark. The trunk, which is relatively short and one to two feet in diameter, divides into numerous large branches that end in contorted, stout twigs. The leaves are twice pinnately compound, with a hundred or more separate oval leaflets arranged on branches of the rib. Its fruit is a flat, leathery pod, four to six inches in length, containing four to seven hard, flat, dark-brown seeds. Pods often remain on the tree until late winter. The tree usually grows on rich bottom land and along streams, scattered among other trees.

Conditions of Poisoning. The toxin in the Kentucky coffee tree has not been identified, but it appears to be present in leaves, seeds, and pulp surrounding the seeds. Trimmings or sprouts from stumps may present increased risk, as can new foliage in late spring. The toxin is water-soluble, and problems can occur if pods fall into water troughs. Heating appears to destroy the toxin, and roasted seeds are nontoxic. The Kentucky coffee tree may be confused with the honey locust (*Gleditsia triacanthos*), which is not toxic.

Signs. Signs can develop within an hour and can include digestive system irritation, excessive salivation, diarrhea, colic, weakness, and depression. If very large quantities are consumed, hypotension (low blood pressure), decreased heart and respiratory rate, muscle paralysis, convulsions, and death may result.

Treatment. Activated charcoal may limit absorption of the toxin from the digestive tract. Supportive care such as intravenous fluid therapy may also be helpful, particularly in animals with diarrhea. Anticonvulsants (e.g., diazepam and phenobarbital) may be required to control seizures.



Figure 28.



Figure 29.

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Castor Bean (*Ricinus communis*)

Figures: 30, 31

Description. Castor bean is commonly grown as an ornamental, but in many places it has become a weed. Plants are erect and stand two to four feet tall, with star-shaped palmate leaves alternately arranged on reddish stems.

Conditions of Poisoning. All parts of the castor bean plant contain ricin, but the highest concentrations are in the seeds. Castor bean poisoning is often associated with feed contaminated with castor bean seeds. Seeds must be chewed to release the ricin; seeds swallowed whole may pass through the digestive tract intact and without incident. All animal species are susceptible to ricin poisoning, but monogastrics (e.g., horses and pigs) appear to be at higher risk than ruminants. In one report, two grams of ground castor beans per kilogram of body weight was fatal to cattle.

Signs. Ricin can cause profuse, bloody diarrhea; straining, colic, or bloat; weakness, trembling, and incoordination; anorexia; thirst; sweating; difficulty in breathing; progressive central nervous system depression; belligerence; and convulsions. Rapid loss of water and electrolytes results in hypovolemic shock and death. Once severe signs are apparent, prognosis for survival is poor.

Treatment. Animals with diarrhea require aggressive intravenous fluid therapy with supplemental electrolytes and glucose. Activated charcoal may help reduce toxin absorption, but it is of less importance than fluids.

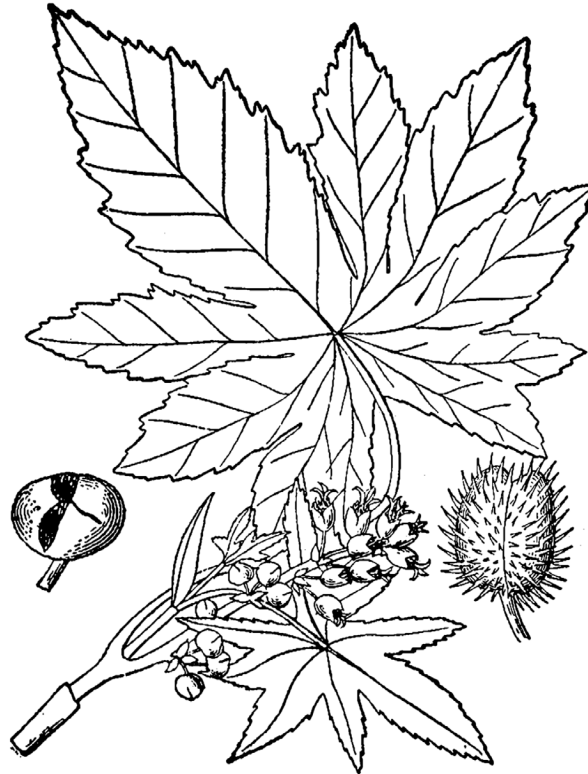


Figure 30.



Figure 31.

St. John's Wort (*Hypericum perforatum*)

Figures: 32, 33
Also called: Klamath Weed, Rosin Rose

Description. St. John's wort grows as an erect, freely branching perennial, reaching one to three feet tall. Opposite leaves are less than one-half inch wide and oblong or linear in shape. Leaves have smooth margins and are covered with transparent dots. The plants have many yellow flowers in somewhat flattened clusters at the top of the stem, with petals bearing black, glandular dots on the margins. The fruit is a three-part capsule containing many small, dark-brown seeds.

The plants grow abundantly in old meadows, pastures, wastelands, and along the roadside.

Conditions of Poisoning. St. John's wort leaves and flowers contain hypericin, a group of photodynamic pigments. Hypericin in the bloodstream interacts with sunlight to produce severe dermatitis in areas of unpigmented skin. Hypericin concentrations are lowest in young shoots, increasing with plant growth to maximal concentrations during flowering. Normally, livestock will not eat mature St. John's wort if other forage is available, although young, tender shoots are more palatable. Contaminated hay and feeds remain toxic. Sheep are most susceptible to poisoning, although cases have also been reported in cattle. Goats are resistant and eat the plant readily with little to no effect.

Buckwheat (*Fagopyrum esculentum*) contains similar toxins (fagopyrin, photofagopyrin, and pseudohypericin) and causes similar clinical signs.

Signs. Photosensitization usually develops two to 21 days after animals ingest St. John's wort. Affected animals develop dermatitis in areas of unpigmented skin such as the teats, udder, escutcheon (skin above the back of the udder), muzzle, ears, eyelids, and other areas of lightly pigmented and less-haired skin. Signs include itching, reddening, swelling, and sloughing of the skin;

thickened skin with a gelatinous appearance; oozing and crusting, especially around the face; and conjunctivitis. Pigmented skin is unaffected. Skin lesions in sheep may be limited to the ears, face, and coronary bands due to the protective effect of wool on other areas. Additional clinical signs can include diarrhea, restlessness, agitation, anorexia, and elevated body temperature and respiratory rate. Signs may last for several weeks, and

subsequent healing may take two months or more due to slow healing of affected areas.

Treatment. Remove the source of the plant. Provide shade for animals to protect them from sunlight. Pain medications and soothing topical medications may also be helpful.

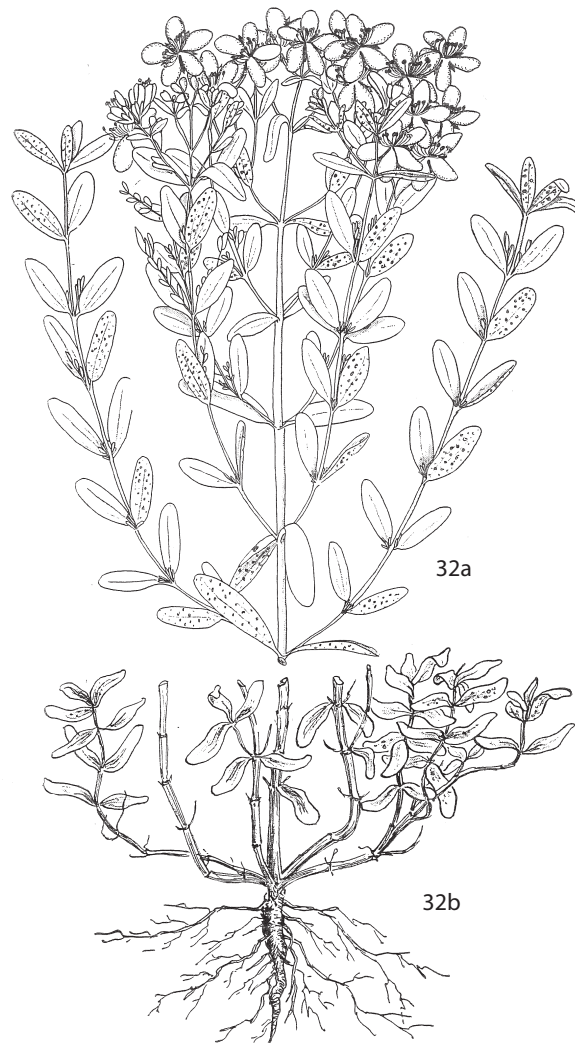


Figure 32. Flowering shoot (a), lower part of plant showing shoots and runners (b).



Figure 33.

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Poison Hemlock (*Conium maculatum*)

Figures: 34, 35, 36
Also called: Spotted Hemlock, Deadly Hemlock

Description. This smooth biennial or winter annual herb stands three to eight feet high, with a purple-spotted stem and finely divided compound leaves. Flowers are small and white, arranged in umbels that blossom in early summer. Leaves have a rank, disagreeable odor. Poison hemlock can be distinguished from water hemlock by more finely divided leaves and a long, parsnip-like root. It is common along roadsides, banks of streams, ditches, in fields, and around farm buildings.

Conditions of Poisoning. Poison hemlock contains several neurotoxic alkaloids. Mature plants contain much higher alkaloid concentrations than young plants, but they are bitter and unpalatable (although some cattle seem to develop a taste for the plants). Cattle kept on dry lots may break through fences in search of green forage and ingest large quantities of poison hemlock despite the bad taste. Contaminated hay or silage is more dangerous, because alkaloid concentrations remain high but palatability is greatly improved. Poison hemlock seeds can also contaminate hay or grain.

Signs. Clinical signs of acute poison hemlock intoxication are similar in all species, often developing within hours of ingestion. Affected animals may develop nervousness, apprehension, muscle tremors, incoordination, weakness, staggering, groaning, colic, abdominal pain, hypersalivation, tearing, and frequent urination. The breath may have a musty, mouse-urine odor. Signs can progress to severe depression, narcosis, paralysis, respiratory depression, collapse, and death due to respiratory failure. In sublethal ingestions, signs can begin to improve within several hours, with full recovery in as few as six to eight hours.

Ingestion of poison hemlock by pregnant livestock can result in birth defects in the offspring, even without clinical signs in the dam. Inhibition of fetal move-

ment during critical developmental periods can cause defects including contracted limbs, joint rigidity, rib cage anomalies, vertebral curvature, and cleft palate. In cattle, the susceptible period of pregnancy is 40 to 100 days, while in swine, sheep and goats, the susceptible period is 30 to 60 days. Sheep are more tolerant of the toxic and teratogenic effects of poison hemlock.

Treatment. Activated charcoal may help bind alkaloids if administered prior to onset of signs. Avoid exciting or stressing symptomatic animals, as that may exacerbate symptoms and result in death. Poisoning is prevented by providing sufficient, good-quality forage and preventing livestock exposure.

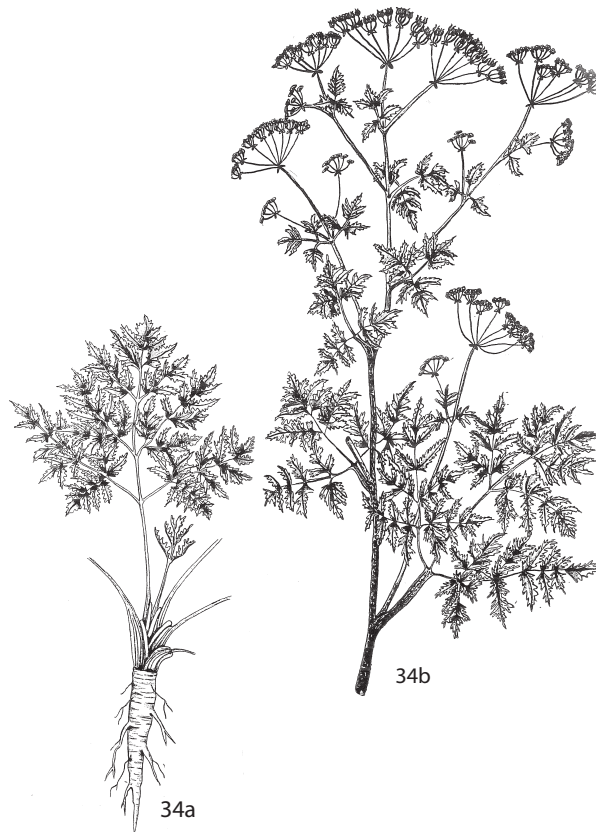


Figure 34. Taproot and young leaves (a). Upper portion of plant with finely divided leaves and umbels of flowers and fruits (b).



Figure 35.



Figure 36.

Water Hemlock (*Cicuta maculata*)

Figures: 37, 38
Also called: Common Water Hemlock, Poison Parsnip, Spotted Cowbane, Spotted Parsley, Spotted Water Hemlock

Description. An herbaceous perennial, water hemlock grows three to seven feet high, with leaves pinnately to tripinnately compound. Sharply toothed leaves up to 15 inches long appear fernlike. The stem is smooth, hollow, and branching, with purplish-green striations. The multichambered tuberous root contains a yellowish, oily liquid. Small, white flowers are arranged in umbels that blossom in the spring or early summer. Water hemlock can be distinguished from poison hemlock by the differences in root and leaf structures. Typically, water hemlock is a wetland plant more common at the edge of waters such as ditches and stream banks.

Conditions of Poisoning. Water hemlock contains cicutoxin, a potent neurotoxin. Early spring leaves contain higher concentrations than mature leaves, and the tuberous roots contain extremely high concentrations. As little as 0.5 percent body weight of fresh plant can cause death, and all species are susceptible.

Signs. Signs can develop within minutes of ingestion. Early signs include uneasiness, muscle tremors, and staggering, followed by jaw champing, teeth grinding, and frenzied activity. Ruminants may bloat. Signs progress to severe convulsive episodes followed by periods of exhaustion with minimal relaxation. Death occurs rapidly, often within a few hours, due to respiratory paralysis.

Treatment. Anticonvulsants can help control seizures; however, approaching symptomatic animals may be dangerous.

Figure 37.

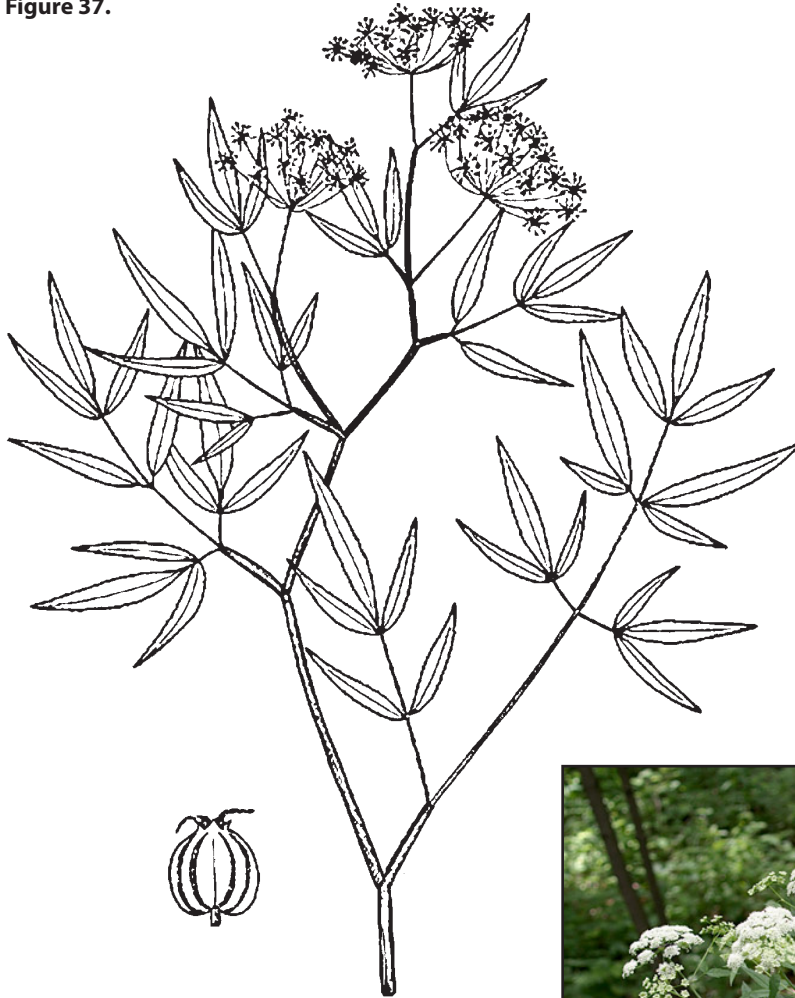


Figure 38.

Dogbane Family - *Apocynaceae*

Hemp Dogbane

(*Apocynum cannabinum*)

Figures: 39, 40

Also called: Indian Hemp

Description. Hemp dogbane is a branching perennial, growing from one to five feet high. The stem contains a milky juice or latex and arises from a vertical underground rootstock. The leaves are opposite and oblong in shape with smooth margins. Greenish-white flowers are borne in clusters at the ends of the stems and branches. The fruit is a long, slender pod containing many seeds that bear flossy tufts.

It grows commonly along roadsides and in fields, meadows and wastelands, often in large colonies.

Conditions of Poisoning. All parts of hemp dogbane contain toxic cardenolides. In large amounts, cardenolides can cause cardiac arrhythmias. Poisoning with fresh plants is rare, as the bitter taste usually discourages ingestion. Intoxication is more common with contaminated hay, as drying improves palatability and plants are willingly eaten in hay. Cases are more often reported in horses than ruminants.

Signs. Clinical signs include slow heart rate, loud heart sounds, jugular pulse, arrhythmias, dilated pupils, diarrhea, profuse sweating, cold extremities, and weakness. Death is unlikely but could occur with very large ingestions.

Treatment. Activated charcoal can help decrease absorption. Digoxin-specific antibodies may be used if available but may be cost prohibitive. Prevent poisoning by examining hay for hemp dogbane contamination.

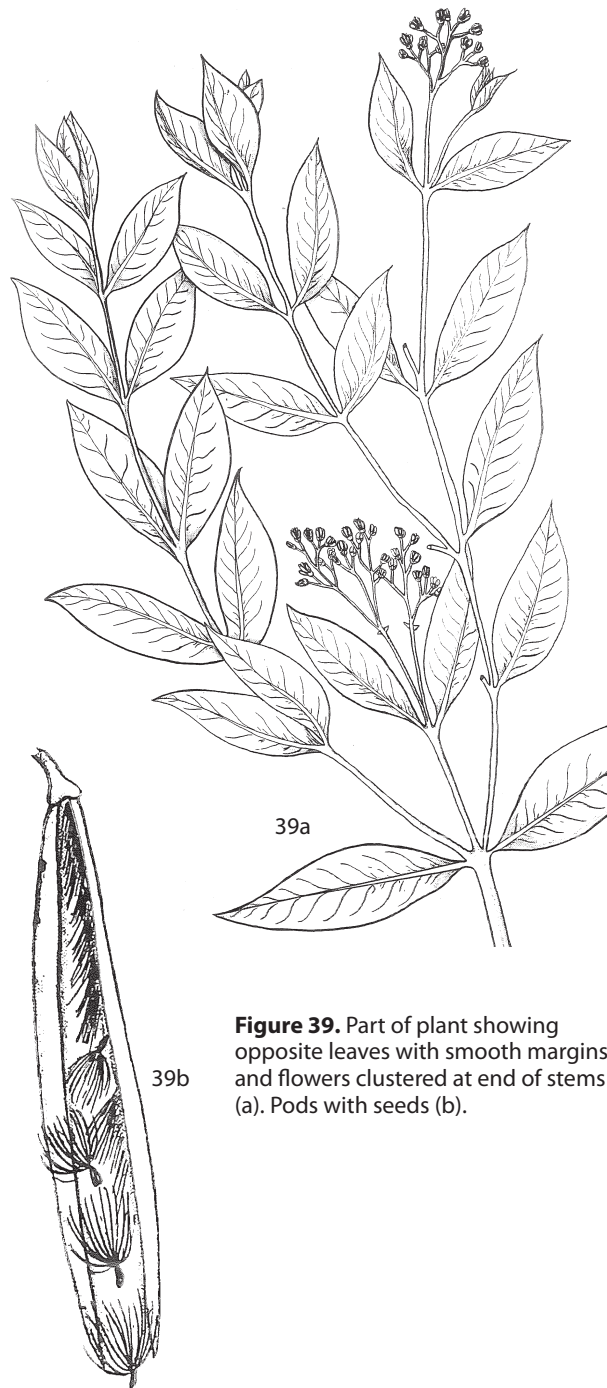


Figure 39. Part of plant showing opposite leaves with smooth margins and flowers clustered at end of stems (a). Pods with seeds (b).

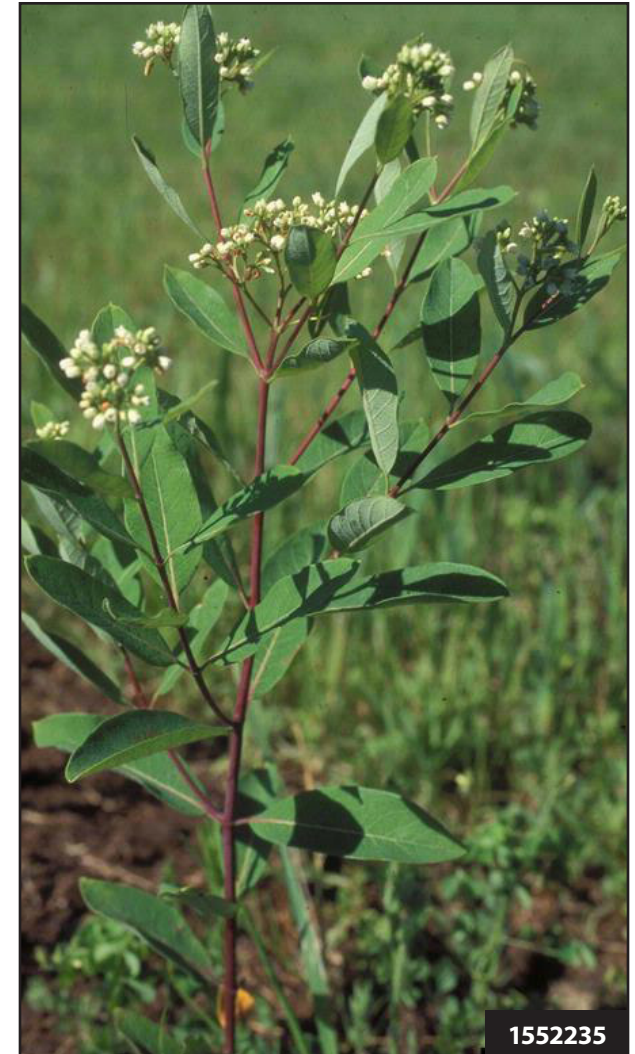


Figure 40.

Milkweed Family - *Asclepiadaceae*

Swamp Milkweed (*Asclepias incarnata*)

Figures: 41, 42

Description. Stems of swamp milkweed can be solitary or clustered, growing one to five feet high. Leaves are opposite. Flowers can be pink to rose-purple and are arranged in umbels. The fruit is a pod containing seeds with flossy tufts.

Conditions of Poisoning, Signs, and Treatment. See *Butterfly Milkweed* on page 29.



Figure 41. Opposite leaves and flower clusters (a). Seedpod (b).



Figure 42.

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Milkweed Family - *Asclepiadaceae*

Common Milkweed (*Asclepias syriaca*)

Figures: 43, 44, 45

Description. Common milkweed grows two to four feet high as an erect perennial. The stem bears broad, opposite leaves and milky sap. Dull-pink flowers are arranged in simple umbels, and the fruit pod contains seeds with flossy tufts.

Conditions of Poisoning, Signs and Treatment. See *Butterfly Milkweed* on page 29.

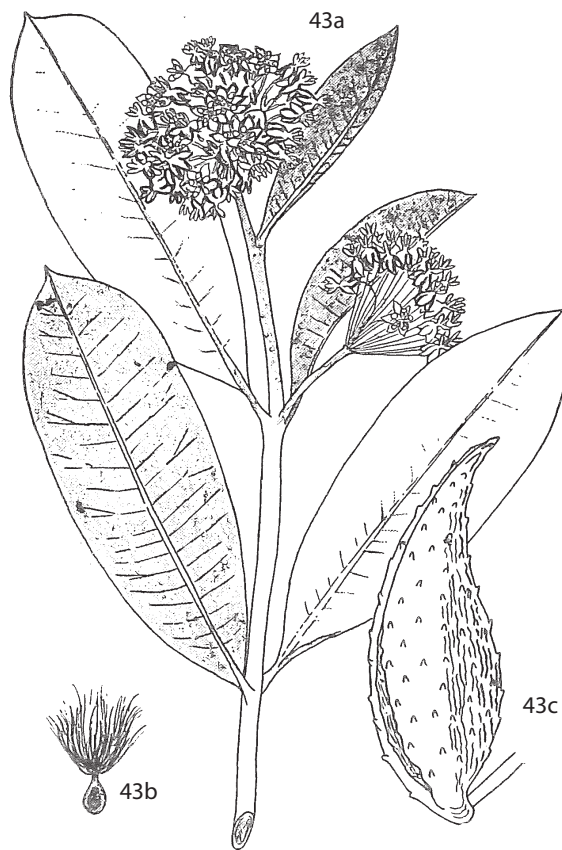


Figure 43. Opposite leaves and flower clusters (a). Detail of tufted seed (b). Seedpod (c).



Figure 44.



Figure 45.

Butterfly Milkweed (*Asclepias tuberosa*)

Figures: 46, 47

Description. Butterfly milkweed is an erect perennial, growing one to three feet high. The stems hold alternate leaves and no milky sap. Its bright-orange flowers are arranged in simple umbels, and the fruit pods contain seeds with flossy tufts.

Conditions of Poisoning. Some broadleaf milkweeds contain cardenolides that affect the heart by slowing electrical conduction through the heart muscle and increasing the force of contractility (see *Hemp Dogbane* on page 26). All plant parts are toxic, but fruits and seeds contain the highest concentrations of cardenolides. Milkweeds are most toxic during rapid growth and remain toxic when dried in hay. Livestock are unlikely to eat fresh broadleaf milkweeds; the leaves are fibrous and unpalatable, and the stems contain a bitter, milky latex. Animals are more likely to be poisoned by contaminated hay. Although all animal species are at risk of poisoning, most reported cases have occurred in sheep and cattle. Depending on the type of milkweed, 1–2 percent of an animal's body weight in green plant can cause clinical signs.

Signs. Signs of poisoning generally begin within 8 to 10 hours of ingestion and may include slowed heart rate, loud heart sounds, a visible jugular pulse, depression, stupor, reluctance to stand, diarrhea (sometimes bloody), weakness, labored respiration, and sometimes a distinct groaning sound. In more severe cases, the pulse may become weak and rapid, and cardiac arrhythmias can develop. Death may occur without signs of struggle. Animals consuming a sublethal dose should recover over several days but may be very weak during recovery.

Treatment. Activated charcoal can decrease toxin absorption. In animals with very low heart rates, atropine may be indicated, although extreme caution should be used in horses. Atropine poisoning in horses can cause fatal gastrointestinal ileus.



Figure 46. Alternate leaves, flower clusters (a). Seedpod (b).



Figure 47.

Whorled Milkweed (*Asclepias verticillata*)

Figures: 48, 49

Description. A slender perennial standing one to three feet high, whorled milkweed has very narrow leaves with curled margins that are arranged in whorls. The flowers are greenish-white and arranged in small umbels. The fruit is a pod containing seeds with flossy tufts.

Conditions of Poisoning. In contrast to the cardiotoxic compounds found in broadleaf milkweeds, whorled (verticillate-leaved) milkweeds contain neurotoxic compounds. Whorled milkweeds pose higher risk than broadleaf milkweeds for several reasons. Whorled milkweeds are more palatable than broadleaf species, especially in hay. The leaves are also finer in texture and harder to avoid in hay than the leaves of broadleaf milkweeds. Whorled milkweed at 0.4–1.0 percent of body weight in green plant can be toxic. Most cases are seen in horses, but they have also been reported in sheep and cattle.

Signs. Horses begin showing signs very quickly after ingestion, but signs can be delayed for more than 12 hours in ruminants. Signs are similar regardless of species and can include colic, uneasiness, depression, weakness, trembling, incoordination, stumbling, salivation, and profuse sweating. Ruminants may also bloat. Severe cases can develop seizures. Death appears to be due to respiratory failure.

Treatment. Anticonvulsants may be necessary to control seizures. Recovery can occur, even in cases that develop violent seizures, provided the animal is prevented from injuring itself.



Figure 48. Whorled leaves and flower clusters (a). Seedpod (b).



Figure 49.

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Mint Family - *Lamiaceae*

Perilla Mint (*Perilla frutescens*)

Figures: 50, 51
Also called: Perilla, Purple Mint, Mint Weed, Beefsteak Plant, Wild Coleus

Description. Perilla mint is an erect summer annual, growing up to two feet in height. The leaves are oval to oblong with toothed margins, oppositely arranged along square stems. The coloring of leaves and stems can range from green or dark green to purple. The leaves emit a distinctive minty odor when crushed, especially when mature. Imperfect flowers, which are white to whitish purple, occur in terminal clusters. Perilla mint is found in pastures, hay fields, fencerows, roadsides, and other waste places, often in partially shaded areas.

Conditions of Poisoning. Perilla mint leaves and seeds contain toxic perilla ketones. Ketone concentrations can vary considerably between varieties of perilla mint and between stages of maturity. Concentrations are minimal in immature plants and much higher in flowering plants. The highest concentrations are in the seeds themselves. Perilla mint thrives in shaded areas in late summer, often reaching the highly toxic seed stage when desirable pasture grasses are scarce. Concurrent hot weather can further entice cattle into shaded areas, where they graze plants they would normally avoid. Mature cattle are most often affected, although poisoning can also occur in yearlings and calves. Dried hay is less toxic than fresh plants, but it can still pose a risk.

Signs. Perilla mint poisoning in ruminants causes acute respiratory distress syndrome (ARDS). Clinical signs include labored, open-mouthed breathing, often with a loud expiratory grunt; dilated nostrils; excessive/foamy salivation; protrusion of the tongue; and extension of the head and neck. In extreme cases, subcutaneous emphysema (air under the skin) may be felt over the upper portions of the neck, shoulders and back. Even mild stress or exercise may cause the animal to collapse and die. Severely affected animals usually die quickly once

signs develop. Animals that live past 48 hours typically survive the acute syndrome but may develop chronic lung problems or heart failure. Affected animals may be found dead with no signs observed.

Treatment. Treatment is generally of limited value once clinical signs develop, but may include diuretics, nonsteroidal anti-inflammatory medications, and corticosteroids. The stress of handling cattle can cause prompt death, so treatment, if attempted, must be handled very cautiously. A dart gun may be necessary to avoid moving the animal to a treatment facility. Prevention involves implementing effective weed control and providing plenty of good-quality supplemental forage or feed when pasture is limited.

See *Acute Respiratory Distress Syndrome (ARDS)* on page 55 for more information.

Figure 50.

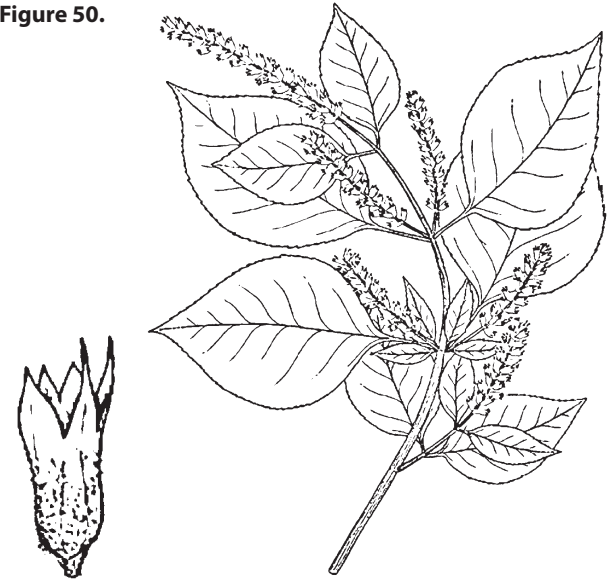


Figure 51.

Jimsonweed (*Datura stramonium*)

Figures: 52, 53
Also called: Thorn Apple, Jamestown Weed

Description. Jimsonweed is a stout, coarse annual, growing two to five feet tall, with spreading branches. Green or purplish leaves are alternate, coarsely toothed, and strongly scented. The large, trumpet-shaped flowers are white or purplish. Fruit is a hard, spiny capsule that splits into four valves at maturity.

Jimsonweed is common in cultivated fields and waste places, and it is often abundant in barnyards and abandoned pastures.

Conditions of Poisoning. Jimsonweed contains several tropane alkaloids similar in structure to atropine, the most abundant of which are hyoscyamine and scopolamine. Alkaloid concentrations are highest in the seeds. Fresh plants are unpalatable and are therefore of limited concern. Many cases occur when seeds contaminate animal feeds. The toxins are heat-stable, thus even cooked or heat-treated feeds can be a risk.

Signs. Hyoscyamine and scopolamine cause anticholinergic effects, including intermittent depression, weakness, reduced gut motility, decreased appetite, colic, and bloat in ruminants. Higher doses can cause excessive thirst, dilated pupils, blindness, dry mucous membranes, increased heart and respiratory rate, wandering, bizarre behavior, and constipation. With very large ingestions, animals can develop incoordination, delirium (hallucinations), extreme weakness, respiratory paralysis, and death. In horses, severe colic can occur due to reduced gut motility. Onset of disease can range from minutes to hours and can last a day or more.

Treatment. Activated charcoal and cathartics may reduce toxin absorption, although the inhibitory effects of tropane alkaloids on gut motility may limit the effectiveness. Anticonvulsants may be indicated to control seizures. Physostigmine is antidotal and can be used in severe cases.



Figure 52. Branch showing coarsely toothed leaves, trumpetlike flower and capsular fruit.



Figure 53.

Nightshade Family - *Solanaceae*

Eastern Black Nightshade (*Solanum ptychanthum*)

Figures: 54, 55

Description. Eastern black nightshade is an erect summer annual, growing up to 1.5 feet tall with branched stems. The leaves are simple, alternate, and ovate or ovate-lanceolate in shape. Leaf margins may be entire or with blunt teeth. Leaves are often tinted purple on the undersurface, particularly in the younger vegetative growth stages. Flowers are star-shaped and white with a yellow, cone-shaped center. Berries are 0.25 to 0.5 inches in diameter, growing in clusters of five to seven; they are initially green and turn purplish-black at maturity. It is a weed primarily found in agronomic grain crops.

Conditions of Poisoning, Signs, and Treatment. See *Carolina Horsenettle* on page 34.



5437066

Figure 54.



1558030

Figure 55.

Nightshade Family - *Solanaceae*

Carolina Horsenettle (*Solanum carolinense*)

Figure: 56

Description. Carolina horse nettle is a branched perennial that typically grows approximately one foot tall, but it may reach up to three feet in height. It propagates by seed and vegetatively via its network of deep, creeping roots. Leaves are simple, alternate, and oval to elliptic or oblong in shape. The stems become woody as plants mature with age. Both leaves and stems have prickles and are covered with star-shaped hairs. Flowers are star-shaped with five petals, white to violet, with a yellow, cone-shaped center. The fruit is a berry, 0.5 to 0.75 inches in diameter; berries are green when immature and turn yellow with maturity. It is commonly found in agronomic crops, pastures and hay fields.

Conditions of Poisoning. *Solanum* species contain a diverse group of toxic alkaloids including solanine, a potent irritant of the digestive tract. Solanine concentrations are highest in the berries and can poison both livestock and humans.

Signs. Poisoned animals can develop loss of appetite; increased salivation and slobbering; abdominal pain/colic; teeth grinding; diarrhea; dilation of pupils; dullness; depression; weakness; incoordination; progressive paralysis; and occasionally death. Pigs may vomit and goats may develop symptoms of regurgitation, respiratory difficulty and neurologic signs. Horses may develop colic from eating large numbers of berries. Rarely, severely affected animals may develop muscle twitching, collapse, seizures, and death.

Treatment. Animals usually recover completely once the source is removed. Symptomatic and supportive care may be necessary in more severe cases.



Figure 56.

Bellflower or Bluebell Family - *Campanulaceae*

Indian Tobacco (*Lobelia inflata*)

Figures: 57, 58, 59

Description. Indian tobacco is a hairy annual with a leafy branched stem, growing one to two feet high. The leaves are thin and oval or oblong, with toothed margins. The pale-blue flowers are two-lipped and inconspicuous, borne in the axils of the upper leaves. The fruit is a capsule covered by the swollen, inflated calyx.

Indian tobacco commonly grows in meadows, pastures and cultivated fields.

Conditions of Poisoning, Signs, and Treatment. See *Great Lobelia* on page 36.



Figure 57. Entire plant showing alternate leaves, inflated fruits, and fibrous roots.



Figure 58.



Figure 59.

Great Lobelia (*Lobelia siphilitica*)

Figures: 60, 61
Also called: Great Blue Lobelia, Louisiana Lobelia

Description. Great lobelia is a perennial herb that grows to a height of three feet. The stem is leafy, rather stout, and usually unbranched. Its deep-blue flowers are about three-quarters of an inch long, arranged in a dense terminal spike.

Great lobelia is commonly found in roadside ditches, swampy areas, wet pastures, and along the edges of streams and ponds.

Conditions of Poisoning. Leaves, stems, and fruits of *Lobelia* species contain various pyridine alkaloids similar to nicotine, the most abundant of which is lobeline. Lobeline concentrations are highest before and during flowering. *Lobelia* species rarely cause problems, with the exception of *L. berlandieri* in southern Texas and northern Mexico. Poisoning can occur in heavily infested pastures, or when other green forage is scarce (e.g., during dry seasons) and the plant is eaten in large quantities for several days. Ruminants are most often affected.

Signs. Initial signs can include excitation, rapid breathing, diarrhea, ocular and nasal discharge, drooping ears, and extension of the neck, followed by depression, loss of appetite, salivation, regurgitation or vomiting, dilated pupils, staggering, labored breathing, incoordination, and collapse. In severe cases, the animal may collapse and die of respiratory failure.

Treatment. Remove the source. Administration of activated charcoal may help reduce absorption of the alkaloids.



Figure 60. Upper portion of plant showing leaves and flowers.



11479195

Figure 61.

White Snakeroot (*Ageratina altissima*)

Figures: 62, 63
Also called: White Sanicle, Boneset
Former name: *Eupatorium rugosum*

Description. White snakeroot is a smooth, erect, perennial herb that stands one to five feet high. Its opposite, oval leaves have pointed tips and sharp-toothed edges. The upper surface of the leaf is dull, and the lower surface is shiny. Each leaf has three prominent main veins on the underside. Small white flowers appear in compound clusters in late summer. It can be confused with late boneset (*Eupatorium serotinum*), which is more common across Kentucky.

White snakeroot is found in woods and damp, shady pastures, and occasionally in thickets and clearings.

Conditions of Poisoning. White snakeroot contains a complex mixture of toxic compounds. Fresh plants are unpalatable; poisoning typically occurs during the late summer when other forages have been consumed or during inclement weather when animals take shelter in the woods. White snakeroot remains toxic when dried in hay, although the risk decreases somewhat. Cases have been reported in nearly all livestock species. Toxins and metabolites are excreted in the milk, and nursing animals can develop clinical signs even if the dam remains asymptomatic. Humans consuming milk from exposed animals are also at risk of developing "milk sickness." Meat from intoxicated animals may also pose a risk to humans who consume it.

Signs. Signs generally develop five to seven days after ingestion of the plant. Initially, affected animals are depressed, weak, stiff, reluctant to move, slow, and sluggish. Signs can progress to include fine-muscle tremors of the nose, flanks, and legs, especially with exercise. Animals may also develop constipation, weight loss, dark urine, and an acetone-like odor to the breath and urine. Horses can develop excessive salivation; difficulty in swallowing; labored breathing; choking; and profuse,

patchy sweating. Tremors are less prominent in horses than in cattle. Later signs include a rapid, irregular heart rate; a stiff, stumbling gait; rapid breathing; terminal collapse; and coma. Cardiac effects can be pronounced in horses, including arrhythmias, jugular pulses, and ventral edema. Death can occur within several days. Goats ingesting large amounts of white snakeroot can develop loss of appetite, depression, head-pressing, paddling seizures, and photosensitization.

Treatment. Repeated administration of activated charcoal may help decrease absorption and re-absorption of toxins. Otherwise, treatment is symptomatic and supportive. Nutritional support is particularly important, especially in recumbent animals. Parenteral administration of glucose may be necessary to treat and/or prevent ketoacidosis. Recovery can occur after a prolonged period of muscle weakness. Horses have a favorable prognosis for recovery if cardiac effects and muscle tremors are not severe. The aftereffects of poisoning are largely unknown, but extensive skeletal or cardiac muscle damage could permanently affect the athletic ability of poisoned animals.



Figure 62.

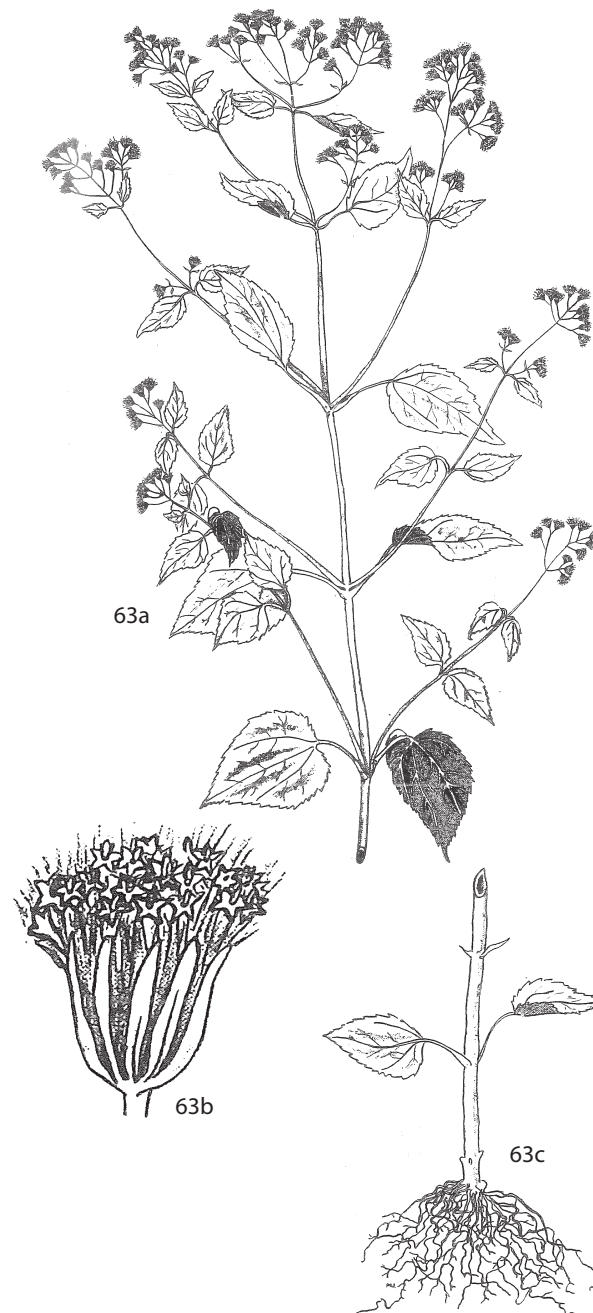


Figure 63. Top of plant showing flower clusters and opposite leaves with three prominent veins (a). Detail of simple flower cluster or head (b). Fibrous roots (c).

Aster Family - Asteraceae/Compositae

Common Sneezeweed (*Helenium autumnale*)

Figures: 64, 65
Also called: Staggerwort, False or Swamp Sunflower,
Bitterweed

Description. Common sneezeweed is an erect-growing, coarse perennial, two to six feet tall, with a narrowly winged stem. Leaves are alternate, lance-shaped and coarsely toothed. The yellow flowers resemble small sunflowers. Toothed, ray flowers are characteristically turned downward. Another species, the purple-headed sneezeweed (*Helenium nudiflorum*), is less common in the state.

Common sneezeweed is often found around water holes, streams, and ditches, and in swampy pastures and meadows.

Conditions of Poisoning, Signs, and Treatment. See *Bitter Sneezeweed* on page 39.



Figure 64. Top of plant showing alternate leaves, heads of flowers and winged stems.



Figure 65.

Bitter Sneezweed (*Helenium amarum*)

Figures: 66, 67
Also called: Bitterweed, Sneezweed, Narrow-Leaved Sneezweed, Yellow Dog Fennel

Description. Bitter sneezweed is an erect summer annual that stands one to two feet high, with fine, narrow leaves. The flowers are yellow and similar in appearance to common sneezweed, but the plants differ in leaf shape.

Bitter sneezweed is usually found in fields, pastures, cultivated areas, and around farm buildings. It is more common in western Kentucky.

Conditions of Poisoning. Bitterweeds and sneezweeds contain sesquiterpene lactones. Effects are cumulative, and disease typically occurs after the plants are grazed for days or weeks. Animals are most often poisoned in late summer and early fall when the plants come into bloom. The plants are poisonous when fresh or cured in hay. Sheep, cattle, and horses are susceptible. Plants are unpalatable and rarely eaten when other forage is available. Most livestock avoid these plants, but individual animals may eat sufficient quantities to cause death. Consumption of *H. amarum*, which contains a very bitter lactone called tenulin, causes milk and meat to have a bitter flavor.

Signs. Lactones in sneezweeds and bitterweeds can affect the digestive tract, the nervous system, and the heart. Signs typically develop after several weeks or longer of ingesting the plants, and they can include depression, inappetence, coughing, regurgitation and bloat. Projectile vomiting (spewing sickness) may be seen in sheep. Severely affected animals may develop tremors, difficulty in breathing, head pressing, convulsions, and death in 24–48 hours. With smaller ingestions over prolonged time periods, signs are primarily limited to gradual deterioration, weight loss, and dehydration. Recovery is unlikely once clinical signs of disease are observed.

Treatment. Remove all animals from the contaminated pasture. Activated charcoal mixed in a protein supplement may prevent signs from developing, but it is not recommended in vomiting animals due to the risk of aspiration. Supplemental methionine may be of benefit in asymptomatic animals. Otherwise, general supportive care, including nutritional support, is indicated for affected animals.

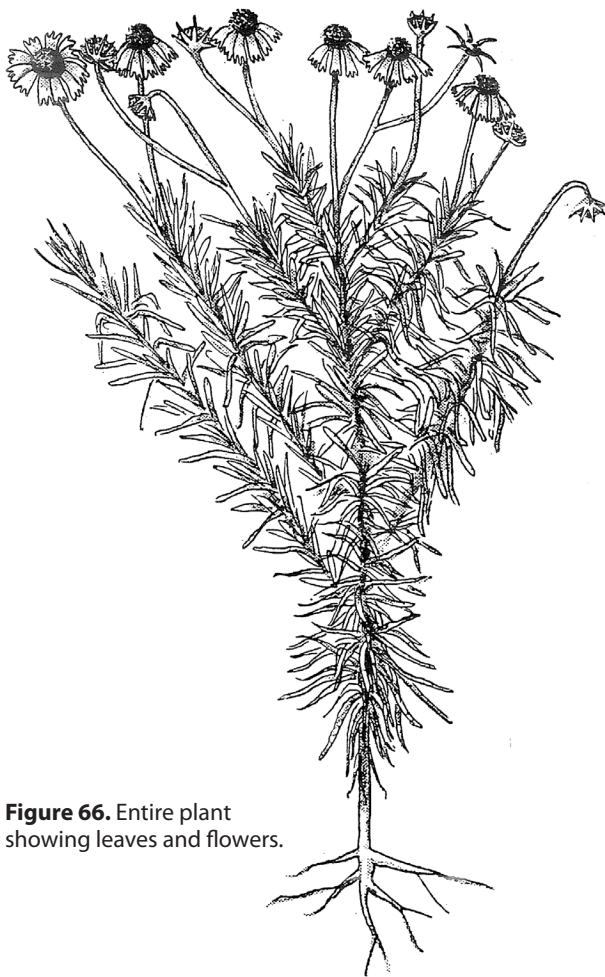


Figure 66. Entire plant showing leaves and flowers.



Figure 67.

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Aster Family - *Asteraceae/Compositae*

Cocklebur

(*Xanthium strumarium*)

Figure: 68
Also called: Burweed, Sheep Bur

Description. A branching summer annual measuring one to three feet in height, cocklebur has stems that are rounded to angled, sometimes with red spots. The leaves are alternate, hairy, rough in texture, somewhat heart-shaped, and variously toothed and lobed. The flowers are inconspicuous and grow as both terminal spikes, which produce only pollen, and clusters in the axils of the leaves, which produce the seed. The fruit is a hard, oval, prickly bur, about three-quarters of an inch long, containing two seeds. Seeds sprout from the buried burs. The younger seedlings have small, strap-shaped cotyledon leaves, one-quarter inch wide and 1¼ inches long; as the plant matures, it produces the more characteristic leaves.

Cocklebur grows commonly in waste places, cultivated fields, along roadsides, in beds of dry ponds, and on overflowed land along streams.

Conditions of Poisoning. Cocklebur seeds and two-leaf seedlings (cotyledon stage) contain carboxyatractyloside; the toxin is not present in more mature plants. Seedlings are often a hazard in the summer when large numbers germinate simultaneously. Seeds can contaminate feed, hay, or silage. Cocklebur poisoning can occur in cattle, sheep, horses, and swine, but calves and pigs are most frequently affected.

Signs. Carboxyatractyloside causes rapid liver damage. Pigs can develop signs within a few hours, including depression, loss of appetite, weakness, vomiting, incoordination, abdominal pain, and a peculiar gait with the head held high. Signs are similar in young calves, but they are usually delayed 12 hours or more. Signs can include excess salivation, muscle tremors, and seizures. In older cattle with functional rumens, signs can be delayed for a day or longer, especially if intact burs are ingested. Mechanical trauma from intact burs can

cause mouth pain and excessive salivation. In horses, colic, rolling, and difficulty in breathing are the most prominent signs.

Treatment. No specific treatment exists. Activated charcoal and cathartics such as magnesium sulfate may

reduce absorption of toxin. Symptomatic and supportive treatment may include fluid and electrolyte administration and sedation in animals with muscle spasms or excitation. Prognosis for recovery is poor.



Figure 68.

Common Groundsel (*Senecio vulgaris*)

Figures: 69, 70
Also called: Ragwort

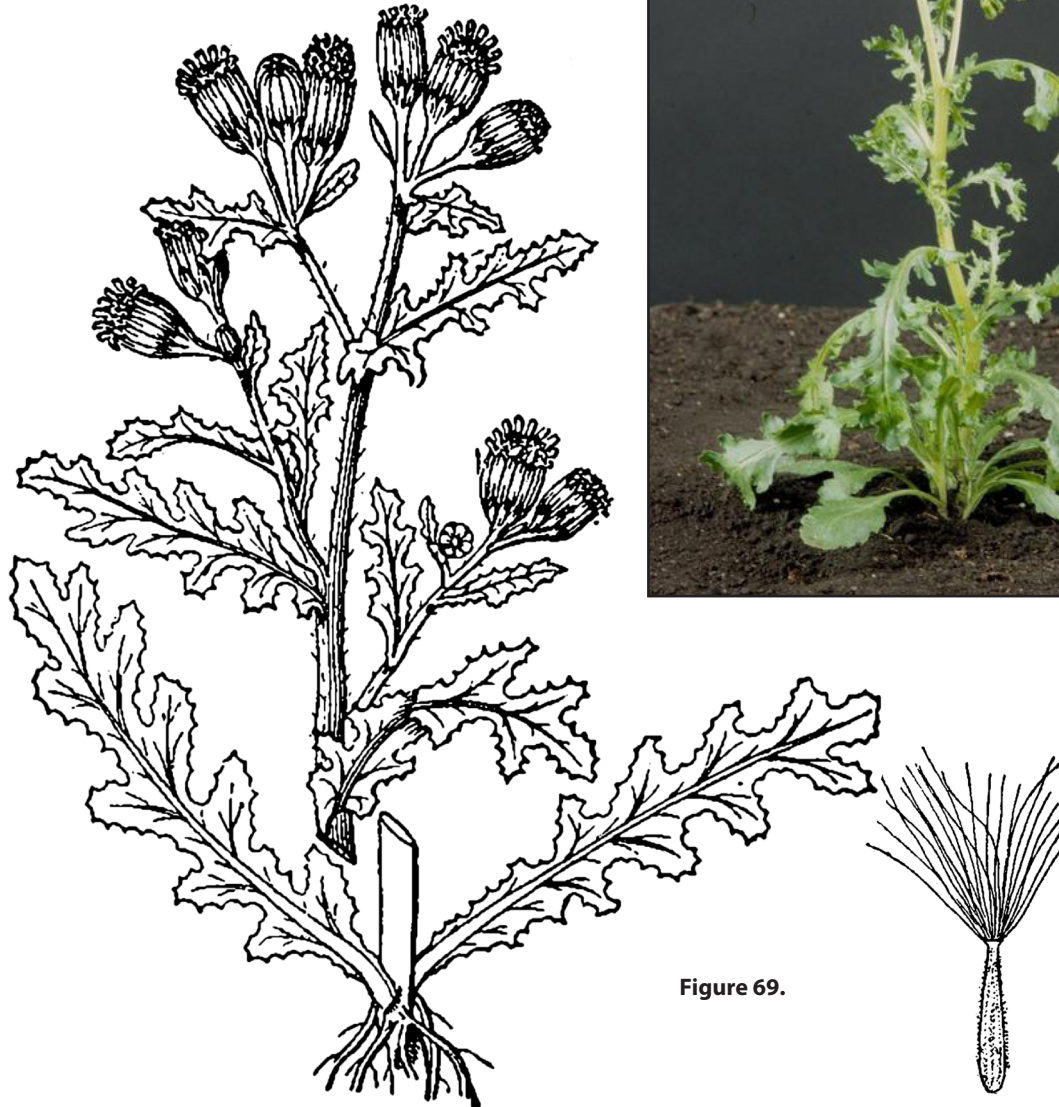
Description. Common groundsel is an erect winter annual that grows to a height between six inches and two feet, with fleshy, ribbed stems that are often red or purplish. The stems are hollow, and the leaves are arranged alternately along the stem. Mature leaves are sparsely hairy, somewhat fleshy and lobed. Flowers form bright yellow, daisy-like petals that emerge from vase-shaped, green sepals. A prolific seed producer, mature flowers form a smaller, dandelion-like, globe-shaped seed head.

Common groundsel grows best in moist, fertile soil, but it can also be found in other environments such as grain crop fields, along roadsides, and in other less-managed areas.

Conditions of Poisoning. Many *Senecio* species contain pyrrolizidine alkaloids (PAs), which can cause liver failure. Horses and cattle are most often affected; sheep and goats are relatively resistant. Most PA-containing plants are not very palatable, and are only problematic when animals are forced to eat them because alternative forages are limited. Poisoning is more often from contaminated feed and hay.

Signs. PA-poisoned animals often develop chronic liver failure after weeks to months of ingestion. Depression, inappetence, and weight loss are typical in the early stages in all species. Horses develop acute onset of head pressing, pacing, chewing, yawning, intermittent drowsiness, rectal straining, and constipation or diarrhea. Onset in cattle is often slower and includes weight loss, weakness, and rectal straining, with less pronounced neurologic signs. Other signs can include jaundice (yellowing of the mucous membranes), photosensitivity, edema (swelling), and fluid accumulation in the abdomen. Extremely high doses can result in acute liver failure, but this is less common.

Treatment. Treatment is generally unrewarding. A degree of recovery is possible with supportive care, including fluids and nutritional support, but fibrosis (scarring) of the liver is irreversible, and affected animals may decompensate when stressed. Efforts should be focused on preventing exposure.



1617552

Figure 70.

Figure 69.

Yew Family - *Taxaceae*

Yew
(*Taxus* spp.)

Figures: 71, 72
Examples: Canada Yew
(*Taxus canadensis*)
Japanese Yew
(*Taxus cuspidata*)

Description. Yews are evergreen shrubs with alternate, stiff, needlelike leaves that are one-half to one inch long. Leaves are dark green and glossy above and yellow-green on the lower surface. Seeds are solitary, borne in a cup-shaped, fleshy, bright red fruit similar in appearance to a berry.

Conditions of Poisoning. Nearly all parts of *Taxus* plants contain highly toxic taxine alkaloids. The only exception is the fleshy red aril ("berry") around the seed. Taxine alkaloids cause cardiac conduction abnormalities and arrhythmias. *Taxus* remains toxic even when dried. Livestock are frequently poisoned by discarded clippings placed where animals can reach them. Animals turned out to graze old homesteads overgrown with *Taxus* are also commonly affected. All species, including cattle, sheep, goats, pigs, and horses, may be affected. The lethal dose varies by species. Horses are very sensitive, with an estimated minimum lethal dose of 0.2–0.4 grams of leaves per kilogram of body weight. In cattle, 2.0 grams per kilogram of body weight is fatal. Goats are more resistant, with a minimum lethal dose of 12 grams per kilogram of body weight.

Signs. *Taxus* poisoning often results in sudden death without any observed signs. Horses may collapse and die within minutes of ingestion, or they may briefly tremble and quiver prior to death. Onset of signs can be delayed for days to weeks in ruminants. Animals may develop depression, weakness, nervousness, muscle tremors, staggering, difficulty in breathing, grunting, diarrhea, and convulsions. Animals surviving the initial episode can die of chronic heart failure long after exposure.

Treatment. Several treatments are possible if clinical signs have not yet developed. In smaller animals (e.g., calves, goats, and sheep), a rumenotomy can be attempted to remove plant material. Oral administration of activated charcoal and a cathartic such as magnesium sulfate may help decrease absorption of the toxin. Once signs occur, the stress of treatment will generally outweigh any potential benefit. In these cases, the animals should be kept quiet and stress minimized to avoid triggering an arrhythmia and possible death.

Figure 71.

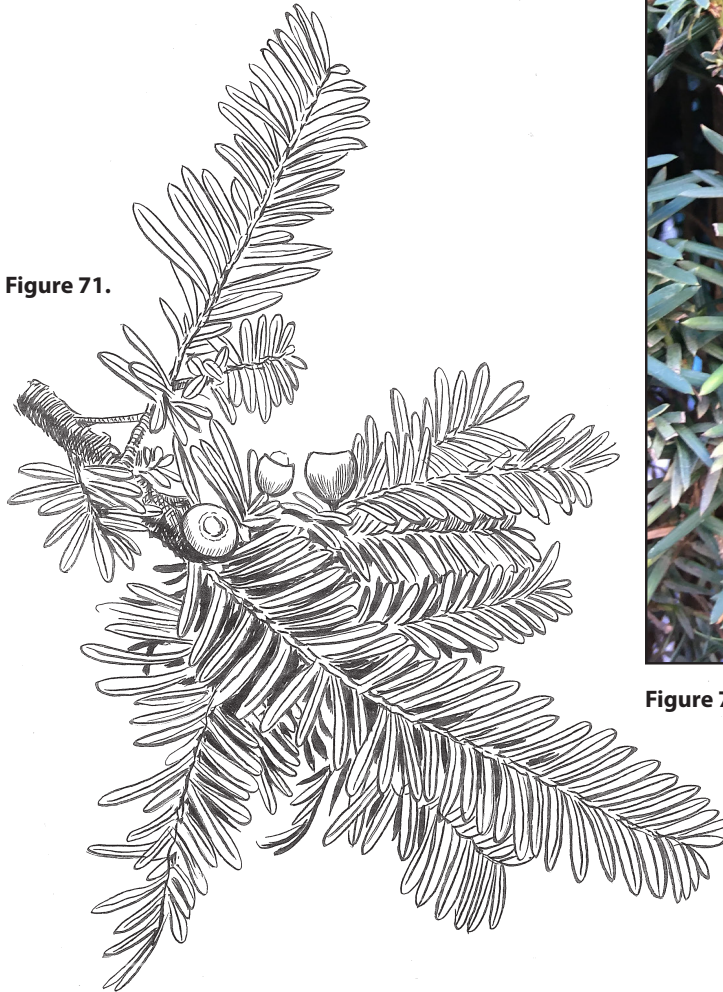


Figure 72.

Heath Family - *Ericaceae*

Mountain Laurel (*Kalmia latifolia*)

Figures: 73, 74
Also called: Calico Bush, Poison Laurel

Description. Mountain laurel grows as an evergreen shrub, three to nine feet high. Leaves up to five inches long can be alternate or irregular. The leaves are leathery, shiny, and light green on their lower surfaces. Cup-shaped flowers, which are borne in spring, are pink or white. Mountain laurel is common in upland woods and hilly pastures, and on acid soil.

Conditions of Poisoning, Signs, and Treatment.
See *Rhododendron* on page 44.

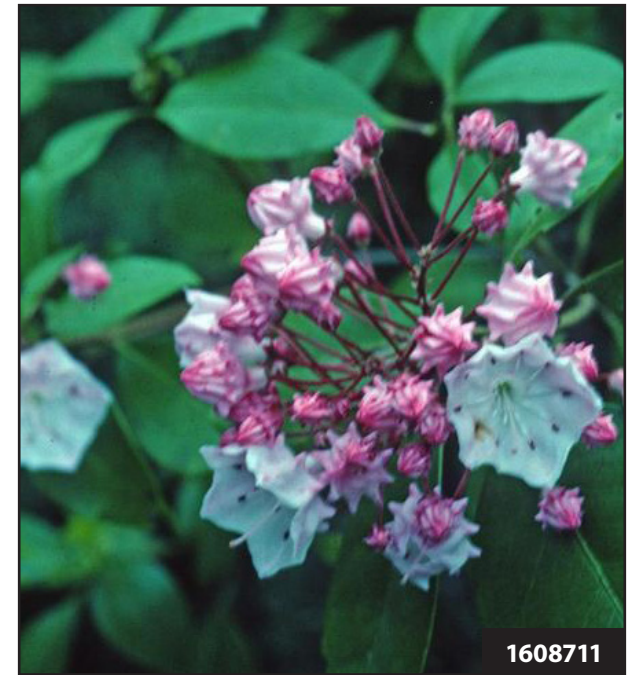
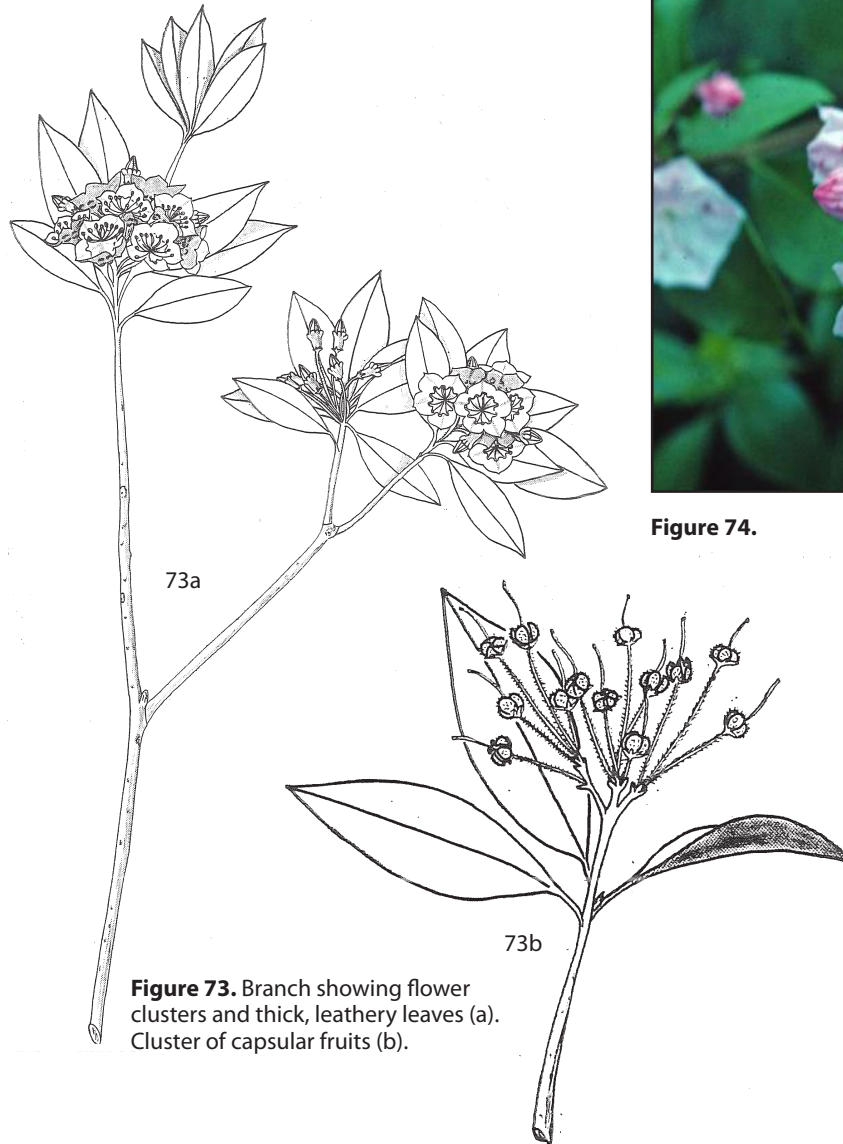


Figure 74.

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Heath Family - *Ericaceae*

Rhododendron (*Rhododendron* spp.)

Figure: 75

Description. Rhododendron is an evergreen shrub or small tree with alternate, leathery leaves. Leaves can appear whitish or rusty on lower surfaces. Flowers are bell-shaped and rose-pink to white. It is common in damp woods, swamps, and upland areas.

Conditions of Poisoning. Several members of the heath family, including *Rhododendron* spp. (including azaleas), *Kalmia* spp. (laurel) and *Pieris japonica*, contain grayanotoxins. Grayanotoxins are present in all parts of the plant, including nectar, flowers, and stems, but the highest concentrations are in the leaves. Some of these plants are evergreen and especially dangerous in winter and early spring, when little other forage is available and hungry, cold-stressed animals enter areas containing the plants. Cases are most often reported in goats and sheep, and less often in cattle and horses.

Signs. Clinical signs usually develop within several hours but can be delayed in cattle. Initial signs include inappetence, bloat, excessive frothy salivation, abdominal pain, and regurgitation of rumen contents (sometimes appearing as green nasal discharge). Goats may have projectile vomiting and vocalization. Neurologic signs often follow, including depression, restlessness, incoordination, muscle tremors, and dilated pupils. Severe intoxications can progress to difficult breathing, cardiac arrhythmias, weakness, paralysis, coma, convulsions, and death. In severe cases, death can occur within hours. Aspiration pneumonia due to vomiting/regurgitation is not uncommon. Prognosis is good unless aspiration pneumonia develops. Acute effects generally resolve within 24 hours, although weakness and other neurologic abnormalities can last three to five days.

Treatment. Activated charcoal is contraindicated in vomiting/regurgitating animals due to the risk of aspiration. In animals that are not vomiting/regurgitating, administration of activated charcoal or magnesium

sulfate by a veterinarian may help reduce toxin absorption. Surgery to remove leaves may be possible in smaller animals prior to onset of signs. One report of intravenous lipid emulsion therapy in goats that ingested *Pieris* leaves appeared promising, although controlled studies are lacking and the treatment may be cost prohibitive in many cases. Additional supportive care measures include pain relief, bloat treatment, and antibiotics if aspiration is likely.



5541788

Figure 75.

Walnut Family - *Juglandaceae*

Black Walnut (*Juglans nigra*)

Figure: 76

Description. Black walnut trees can grow 60 to 100 feet tall, with a trunk two to three feet in diameter. The bark is deeply furrowed and either dark brown or grayish black. Leaves are compound, with 15 to 23 leaflets borne on short petioles about one to two feet long. The edible fruit (nut) is contained within an outer, hairy, yellow-green husk. The roots of the tree exude a chemical called juglone that can inhibit growth of other plants. A common tree, black walnut is found along roadsides, fields, and forest edges throughout the eastern United States.

Conditions of Poisoning. Fresh black walnut shavings from new or old wood are associated with poisoning in horses. The exact compounds and mechanisms are not known, although the toxic principle appears to be present in aqueous extracts of black walnut heartwood. Initial investigations into juglone as a possible culprit were promising, but administration of purified juglone failed to produce clinical signs in controlled studies. Well-cured (one month or more) walnut shavings appear to pose minimal risk. Animals can also ingest large numbers of walnuts if other food is scarce.

Signs. Black walnut poisoning in horses can cause acute laminitis (primarily in the front limbs) and lower-limb edema (primarily in the hind limbs). Affected horses develop warm, sensitive hooves and are reluctant to move. If forced to do so, they walk with short, stiff steps and attempt to bear most of their weight on their hind limbs. Body temperature, pulse, and respiration often increase.

Treatment. Acute episodes of laminitis resolve rapidly if horses are promptly removed from the source of black walnut. If horses are left in contact with the shavings, signs can progress, eventually causing chronic laminitis and rotation of the coffin bone (third phalanx). Cold hosing the legs of affected animals can aid in topical

decontamination, provide pain relief, and reduce heat and swelling. In more severe cases, anti-inflammatory medications may be necessary to provide additional relief. Prognosis for full recovery is excellent with prompt treatment.



Figure 76.

Beech Family - *Fagaceae*

Oak
(*Quercus* spp.)

Figures: 77, 78

Description. Oaks are easily recognized by their fruit (acorns). Nearly all oaks have a leaf that is broadly bladed and deeply cut along the margin, forming sections or lobes. However, there are a few species that have leaves resembling willow leaves. In wooded areas, young oak trees may occur in large numbers.

Conditions of Poisoning. Acorns, oak seedlings, and oak buds can cause problems for livestock when eaten in very large amounts. Most cases occur in early spring or fall, or during severe weather events when acorns or buds are plentiful but little other forage is available. All oak species contain tannins and phenols, which can be toxic if ingested in large enough amounts; tannin content varies with oak species and leaf age. Cattle are most often affected, especially young animals (one to three years old). Sheep are also susceptible. Goats are much more resistant to oak poisoning due to tannin-binding proteins in the saliva and digestive tract. Goats are effective browsers, and they have been used to control oak, once adapted to eating it. Horses and pigs are rarely affected by oak poisoning.

Signs. Tannins and phenols cause digestive tract irritation and kidney damage. Illness typically develops a week or more after consumption of large quantities of oak. Digestive tract signs can include inappetence; lethargy; bloat; constipation; straining; and hard, dark feces progressing to bloody or black, tarry diarrhea. Animals may grind their teeth and adopt a hunched posture due to abdominal pain. Signs of kidney disease include excessive thirst; increased urination; brown or red urine; and swelling of the neck, brisket, abdomen, and perineal areas. Death due to kidney failure can occur within five to seven days. Animals that survive acute oak poisoning may suffer chronic kidney failure. In horses, kidney effects are less pronounced, and gastrointestinal signs are the most consistent and severe. Signs in horses include

diarrhea with or without blood; colic; and straining. Prognosis in horses depends on the severity of digestive tract problems.

Calves born to cows that consumed a large number of acorns during pregnancy (“acorn calves”) may be born with joint laxity, short legs, deformed hooves and skull abnormalities. This syndrome is not attributed directly to the oak tannins and phenols, but it may be due to malnutrition associated with animals forced to subsist solely on acorns.

Treatment. Remove animals from the source of oak and provide good-quality forage. Oral calcium hydroxide can neutralize tannins in the rumen. Intravenous fluids may be necessary to restore volume and support kidney function. Marked weight loss may occur during recovery, but cattle that survive appear to have compensatory weight gains. Poisoning can be prevented by limiting oak consumption. Ingestion can be curtailed by ensuring that animals have plenty of quality forage; fencing off oak trees during budding, early leaf growth, and acorn drop; and providing supplemental feed when necessary.



Figure 77.

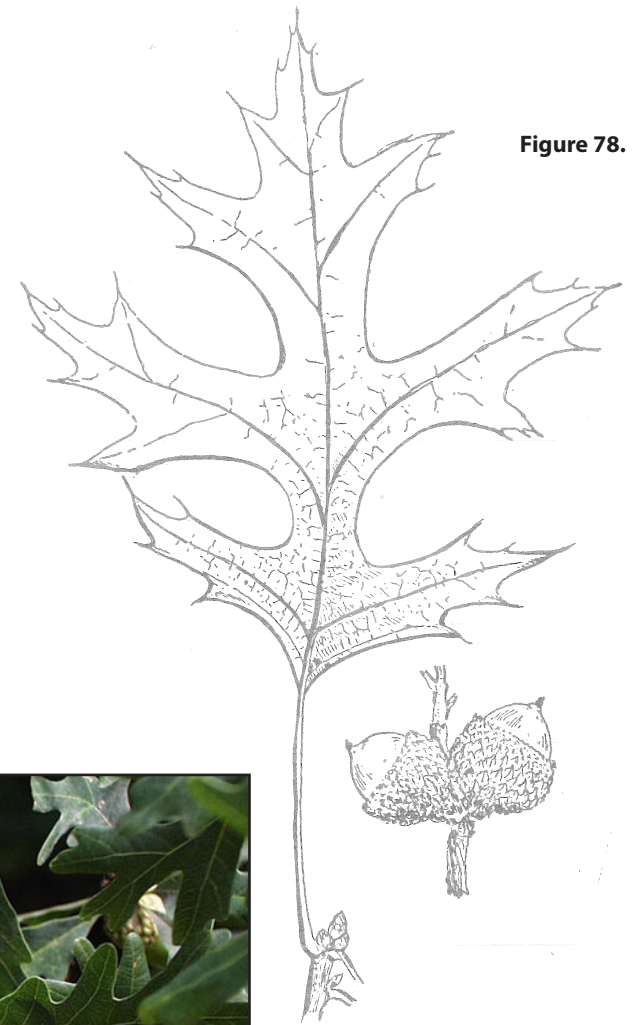


Figure 78.

Rose Family - *Rosaceae*

Wild Black Cherry (*Prunus serotina*)

Figures: 79, 80

Description. Wild black cherry is a tree or shrub with slender, horizontal branches. Bark of young branches and twigs is reddish-brown with prominent white lenticels (pores). Leaves are alternate, simple, elliptical, and pointed, with finely toothed margins. The leaves are also leathery in texture and usually have a row of hairs on the lower surface along both sides of the midrib. Small, white flowers form in drooping clusters and produce dark red to black cherry fruits.

Wild black cherry commonly grows along fencerows, in roadside thickets, and in rich, open woods.

Conditions of Poisoning. See *Chokecherry* on page 48.

Signs and Treatment. See *Cyanide Poisoning* on page 55.

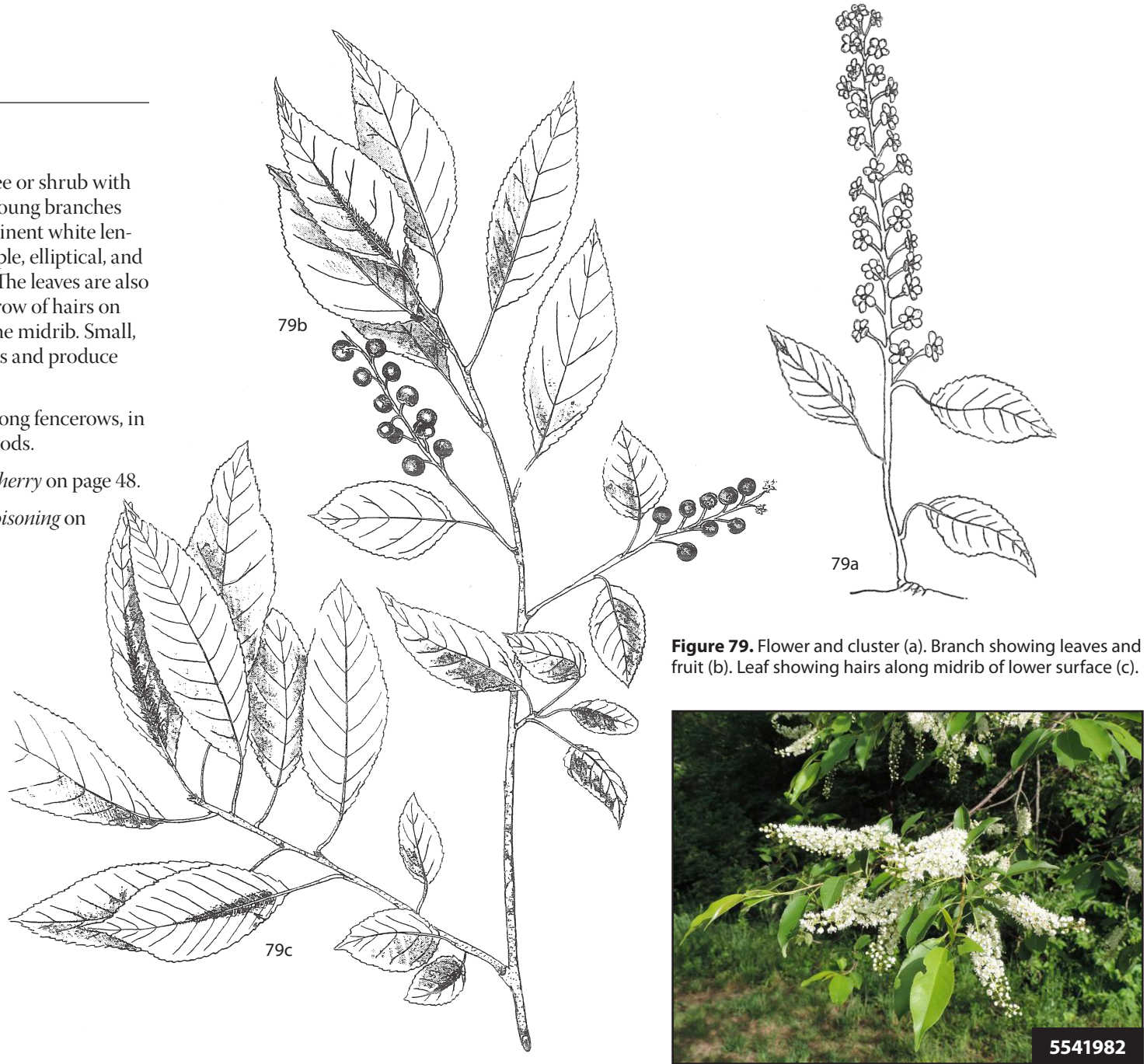


Figure 79. Flower and cluster (a). Branch showing leaves and fruit (b). Leaf showing hairs along midrib of lower surface (c).



Figure 80.

Rose Family - *Rosaceae*

Chokecherry (*Prunus virginiana*)

Figure: 81
Also called: Common Chokecherry

Description. Chokecherry is a small tree or thicket-forming shrub of limited distribution in Kentucky. Alternate leaves are oval to elliptical with finely serrated margins, and their color is shiny, dark green above and lighter green underneath. Flowers are white and grow in drooping clusters. Fruits (berries) are bright red to black.

Conditions of Poisoning. *Prunus* spp. contain cyanogenic glycosides—namely, amygdalin in the seeds and prunasin in the leaves, bark and shoots. Poisoning most frequently occurs when ruminants consume wilted leaves (e.g., after storm damage), new growth, and seeds. The lethal dose in cattle is in the range of 2 to 2.5 milligrams of cyanide per kilogram of body weight. Forage cyanide concentrations exceeding 200 ppm are considered high enough to deliver a lethal dose of 2 milligrams of cyanide per kilogram.

Signs and Treatment. See *Cyanide Poisoning* on page 55.



Figure 81.

Soapberry or Maple Family - *Aceraceae*

Red Maple (*Acer rubrum*)

Figures: 82, 83

Description. Red maple is a medium-sized tree growing 40 to 70 feet tall. Simple alternate leaves are two to five inches wide and shaped with three to five lobes that have toothed margins. The small, winged fruit, called a samara, grows in pairs. Red maple is named for its red twigs and flowers, and it is known for its brilliant fall foliage.

Red maple is a common tree throughout its native range of eastern North America.

Conditions of Poisoning. *Acer rubrum* leaves contain gallotannins and gallic acid, which are converted to pyrogallol by intestinal bacteria in equines (e.g., horses, donkeys, mules, and zebras) and alpacas. Pyrogallol causes hemolysis (destruction of red blood cells). Laboratory experiments incubating equine red blood cells with silver (*Acer saccharinum*) and sugar (*Acer saccharum*) maple leaf extracts also showed significant hemolysis. Therefore, all maples, particularly red maple hybrids, should be considered toxic until proven otherwise. Wilted leaves are the most dangerous, and most poisoning cases result from downed limbs after storms, or when tree trimmers allow branches to fall into paddocks. Doses of 1.5 grams of leaves or less per kilogram of body weight may be poisonous.

Signs. Onset of clinical signs often occurs a day following ingestion of wilted or partially dried leaves. Signs typically progress over one to two days, and they can include anorexia, depression, lethargy, dark-brown urine, severe anemia, dark or bluish mucous membranes, colic, laminitis, coma, and death.

Treatment. Intravenous fluid therapy is necessary to restore volume and maintain oxygen delivery to vital organs. Blood transfusions may be necessary in severely anemic animals. Oxygen therapy has been suggested, but it is of limited use in severely anemic animals. Antioxidants, such as ascorbic acid and vitamin E, may also be helpful early on in limiting oxidative damage. Activated charcoal may be useful in the early stages, but it is of limited use once anemia has developed.

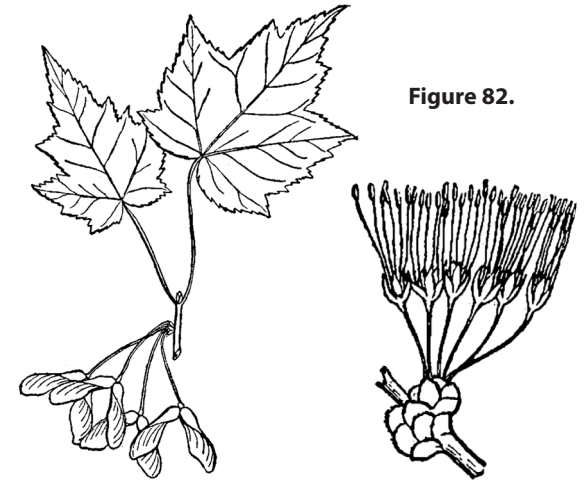


Figure 82.



Figure 83.

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Soapberry or Maple Family - *Aceraceae*

Boxelder (*Acer negundo*)

Figures: 84, 85

Description. Boxelder is a medium-sized tree typically growing 30 to 50 feet tall. The leaves differ from most maples by having pinnately compound leaves consisting of three to five toothed leaflets (three leaflets are most common). Leaves closely resemble poison ivy but have an opposite leaf arrangement. The small, winged fruit (called a samara) grows in pairs, similar to other *Acer* species. Leaves usually produce yellow foliage in the fall.

Boxelder is common throughout its native range of eastern North America.

Conditions of Poisoning. Boxelder seeds and seedlings contain hypoglycin A, the putative toxin responsible for equine atypical myopathy (EAM, also known as seasonal pasture myopathy) in grazing horses. EAM is an acute, degenerative muscular disorder primarily affecting the postural, cardiac, and respiratory muscles. Poisoning is most common in fall, when samaras drop, and spring, when large numbers of seedlings may be present in pastures. Occurrence is sporadic, often occurring in some horses while others grazing the same pasture remain unaffected. Sycamore maple (*Acer pseudoplatanum*) also contains hypoglycin A but is uncommon in Kentucky.

Signs. Affected horses develop a stiff gait and become unwilling to walk. They may sweat profusely and develop congested mucous membranes and dark urine. Signs often progress to severe weakness, inability to stand, and difficulty in breathing.

Treatment. Intravenous fluid therapy is essential to correct dehydration and acid-base imbalances and restore blood flow to the kidneys. Glucose administration can provide energy to affected muscles. Blood glucose levels should be closely monitored, and insulin should be administered as needed to prevent severe hyperglycemia. Carnitine may help stimulate glucose metabolism

while also detoxifying active metabolites and speeding excretion in the urine. Additional treatments may include vitamin E and selenium supplementation, pain relief such as NSAIDs, and muscle relaxants. Activated charcoal or laxatives may decrease toxin uptake but are most effective if given soon after exposure. Other supportive measures include urinary catheterization and keeping affected animals warm and quiet.

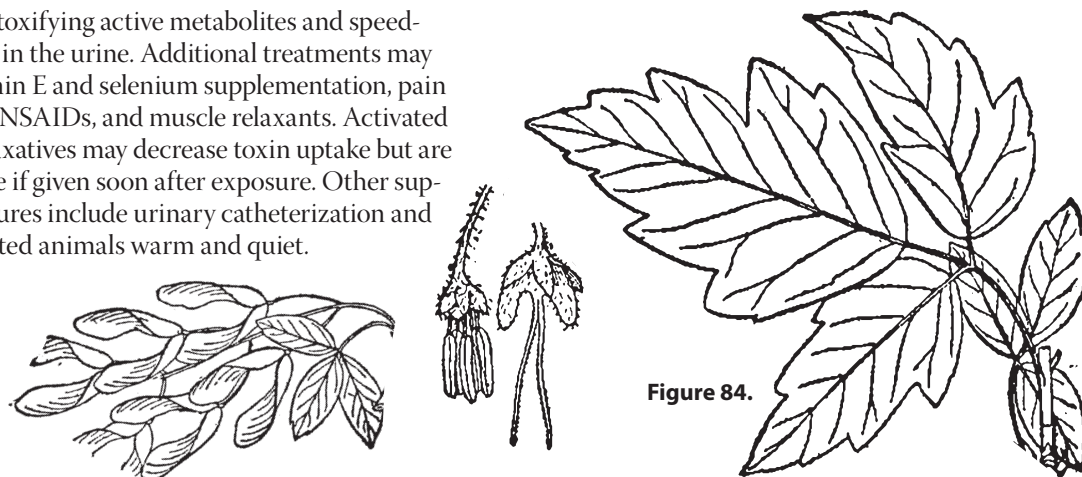


Figure 85.

Buckeye or Horse Chestnut Family - *Hippocastanaceae*

Ohio Buckeye (*Aesculus glabra*)

Figures: 86, 87, 88

Description. The Ohio buckeye is a tree or shrub with opposite leaves that are palmately compound. Leaves usually have five leaflets arranged like the fingers of a hand. The flowers are yellowish, growing in large clusters at the ends of branches. The fruit is a prickly capsule (at least when young) with a leathery texture. Fruits hold one to three seeds, which are glossy brown with a pale scar—hence the common name buckeye. The bark has a strong, offensive odor.

Ohio buckeye is commonly found in rich, moist woods and along riverbanks.

Conditions of Poisoning, Signs, and Treatment. See *Sweet Buckeye* on page 53.



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Figure 87.

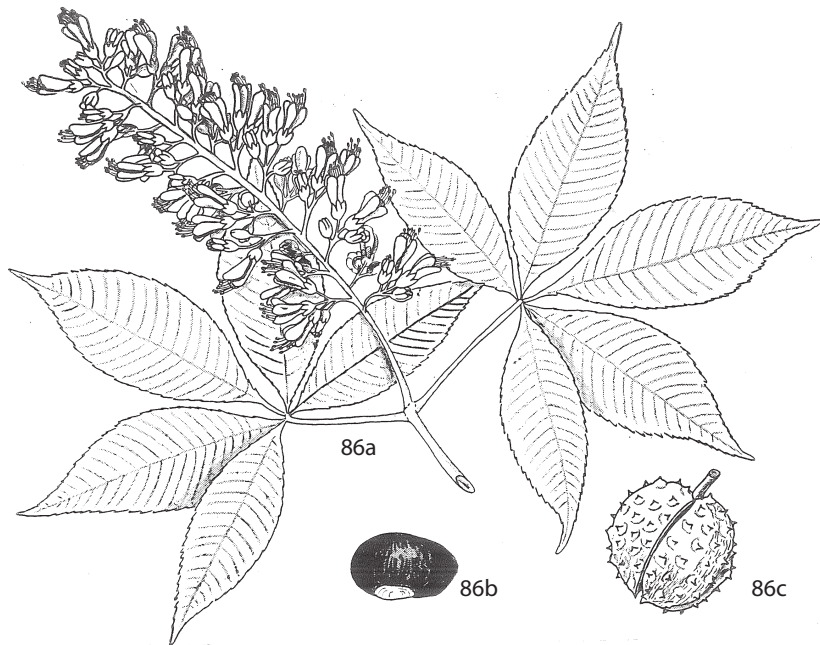


Figure 86. Branch showing leaves and flower cluster (a). Seed (b). Spiny fruit (c).



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Figure 88.

Buckeye or Horse Chestnut Family - *Hippocastanaceae*

Horse Chestnut

(*Aesculus hippocastanum*)

Figures: 89, 90

Description. Horse chestnut is similar to Ohio buckeye, but the leaves usually have seven leaflets and the flowers are white.

The cultivated species was introduced from Europe and is widely planted as an ornamental shade tree.

Conditions of Poisoning, Signs, and Treatment. See *Sweet Buckeye* on page 53.

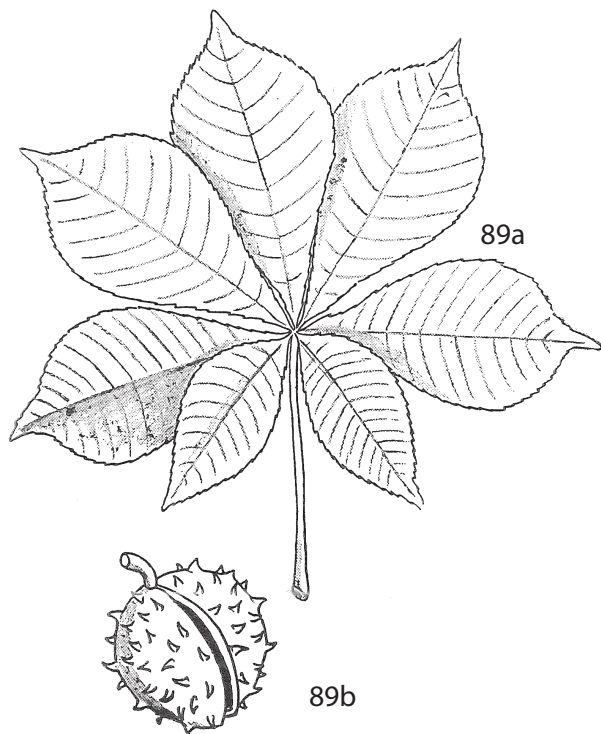


Figure 89. Leaf (a), and spiny fruit (b).



Figure 90.

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Buckeye or Horse Chestnut Family - *Hippocastanaceae*

Sweet Buckeye (*Aesculus flava*)

Figures: 91, 92
Also called: Yellow Buckeye
Former name: *Aesculus octandra* Marshall

Description. Sweet buckeye is similar to Ohio buckeye, but the bark has only a slight odor and the fruits are not prickly. It is common in rich, moist woods and along riverbanks.

Conditions of Poisoning. *Aesculus* species contain mixtures of various toxic compounds, including glycosides and saponins. Problems are often seen in the fall when the fruits fall from the trees and are eaten in large numbers off the ground. Some of the toxins are water-soluble, and fallen fruits can contaminate water troughs. In the spring, poisoning can occur if large amounts of new growth (leaves, buds, or bark) are ingested. *A. hippocastanum* is more toxic than other *Aesculus* species. In calves, ground nuts of *A. flava* at 0.5 percent of body weight produced severe signs; 1 percent of body weight was lethal.

Signs in Ruminants. In ruminants, glycosides are converted by rumen microbes to neurotoxic aglycones. Neurologic signs begin approximately 16 hours after ingestion of a toxic dose. Affected animals become reluctant to move and develop a stiff-legged or “sawhorse” stance. When made to move, animals may stumble, stagger, and move with a distinctive, bunny-hopping gait. Occasionally, cattle with buckeye poisoning may even do somersaults. Other signs may include muscle tremors, weakness, a stargazing posture, depression, colic, collapse, and death. Most animals survive, although the prognosis is worse with severe neurologic signs.

Signs in Monogastrics. Horses, pigs, and other monogastrics are primarily affected by the saponins, which cause irritation and inflammation in the digestive tract. Inflammation further increases toxin absorption.

Affected animals may develop colic, inappetence, and diarrhea.

Treatment. Animals should be removed from the source, using caution with neurologic animals. Activated charcoal and cathartics may decrease toxin absorption, although cathartics are contraindicated in animals with diarrhea, and approaching severely neurologic animals may not be safe. Supportive care, including intravenous fluid therapy, nutritional support, and repositioning recumbent animals, is often necessary. Anticonvulsants may be indicated to control seizures. Once animals become recumbent, prognosis is worse, although they can still recover with aggressive supportive care. Surviving animals typically recover in 72 to 96 hours, but in severe cases, neurologic problems may persist for several weeks.



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Figure 92.

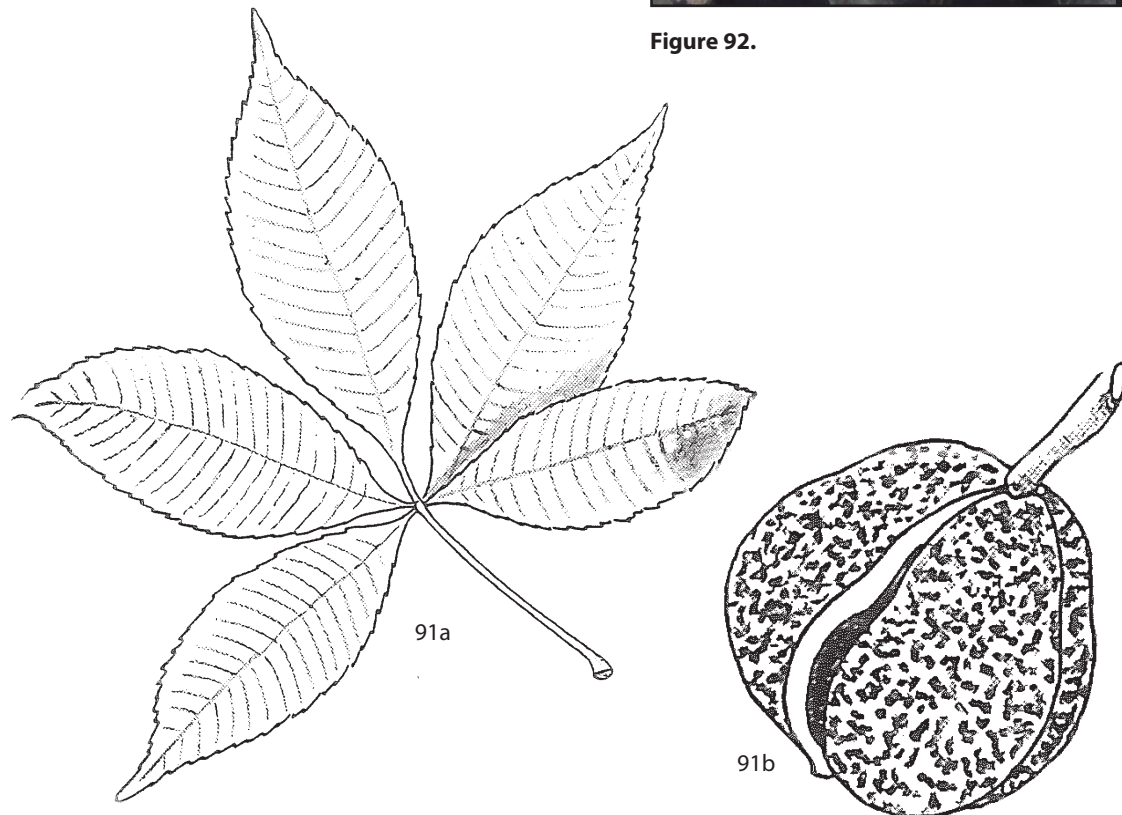


Figure 91. Leaf (a) and fruit (b).

Nitrate Poisoning

Conditions of Poisoning. Certain heavily fertilized crops can accumulate nitrate in concentrations toxic to ruminants. In Kentucky, corn, sorghum, Sudan grass, and sorghum-Sudan hybrids are most commonly associated with poisoning, although other heavily fertilized crops, including oats, wheat, ryegrass, barley, alfalfa, pearl millet, soybeans, beets, and *Brassica* spp. (rape, kale, turnips) can also pose a risk. Nitrate-accumulating weeds include Johnsongrass, ragweed, pigweed, thistle, bindweed, dock, nightshades, and jimsonweed. Many of these weeds invade fields where they can contaminate forages, especially in newly planted hay fields.

Heavy fertilization provides large amounts of nitrate for uptake. Under normal conditions, plants take up nitrate through their roots and transport it to the leaves for use in photosynthesis. Photosynthesis slows under adverse environmental conditions, such as drought, leaf damage (e.g., due to disease, hail, frost, insects, or herbicides), a sudden change from warm and sunny to cool and cloudy weather, or other plant stressors. When this occurs, nitrate is no longer transported to the leaves, but it remains in the lower stalks and stems in potentially toxic concentrations.

Nitrate concentrations are not decreased by drying for hay. Proper ensiling for at least 30 days can reduce nitrate concentrations by approximately one third. Hungry or cold animals can consume large amounts of forage in a short period of time and are at greater risk. Low-energy diets also increase susceptibility to nitrate poisoning. Problems can occur very quickly or can be delayed for several days after introducing high-nitrate forages; as rumen microbes adapt to utilizing nitrate, nitrate-to-nitrite conversion initially occurs more rapidly than nitrite-to-ammonia conversion, causing nitrite to accumulate.

Dietary nitrate is reduced by rumen microbes in a series of steps from nitrate to nitrite to ammonia, eventually becoming incorporated into microbial proteins. Intake of excessive nitrate results in the rapid formation of large quantities of nitrite (NO_2^-). Nitrite ions oxidize

heme iron within red blood cells, forming methemoglobin. Methemoglobin cannot transport oxygen, and animals die of tissue hypoxia (lack of oxygen).

Signs. Affected animals typically show signs of nitrate poisoning within one to four hours of ingestion. Production of methemoglobin lends the blood a chocolate-brown color. As methemoglobin concentrations increase, the mucous membranes can become dark or muddy, and animals may develop weakness; trembling; rapid breathing; severe incoordination and staggering; rapid, shallow breathing; weak pulses; apprehension; and belligerence. Death can occur within hours of ingestion. The oxidation reaction is reversible, and methemoglobin will eventually reduce back to hemoglobin. Animals generally die or recover within 24 hours. Abortions can occur in pregnant animals at any stage of gestation due to the combined effects of decreased oxygen to the fetus and the limited ability of the fetus to metabolize nitrite. Abortions typically occur within a week of exposure, but they can be delayed in some cases.

Treatment. In most cases the animals die quickly, before treatment can be started. Nitrate sources should be identified and removed. Vinegar administered via stomach tube can lower rumen pH, slowing nitrate reduction; however, stress of handling must be minimized to avoid causing sudden death. Administration of methylene blue intravenously by the veterinarian can speed the reduction of methemoglobin back to hemoglobin, but withdrawal guidelines for food animals must be followed and are subject to change.

Prevention. The amount of nitrate that can be safely consumed in forages is approximately 45 grams of nitrate per 100 pounds of body weight. All potential sources of nitrate, including water and feed, must be considered. If testing for nitrates, be aware that nitrate levels can be reported in a variety of ways, and the units can differ between laboratories. Nitrate can be reported as nitrate (NO_3), nitrate-nitrogen ($\text{NO}_3\text{-N}$), or potassium nitrate (KNO_3). These numbers are not equivalent, as they represent different chemical structures. Make sure the feeding guidelines used for a particular result match the type of analysis performed.

Plants or hay containing more than 1.7 percent nitrate (>17,000 ppm) dry matter are potentially fatal and should not be fed. Plants containing more than 0.5 percent nitrate (>5,000 ppm) should be considered potentially toxic to pregnant animals. Forages containing more than 0.5 percent nitrate should be mixed and diluted with low-nitrate forage or fed to less susceptible species. Sheep and goats are less susceptible to nitrate poisoning than cattle. Horses are very resistant; compared to rumen microflora, hindgut microbes in horses convert only very small amounts of nitrates to nitrites.

Poisoning is best prevented by recognizing plants that are likely to accumulate nitrates and testing suspect forages before feeding. If high-nitrate forages must be utilized, the following suggestions will reduce the risk of nitrate poisoning*:

1. Splitting forage feeding to twice a day is suggested. The diet should contain less than 5,000 ppm NO_3 to avoid reproductive impacts. Non-pregnant animals could be fed slightly higher levels after being acclimated.
2. Introduce the nitrate forage slowly. Give half the final amount of high-nitrate forage for the first two to three weeks. Cattle have the ability to increase their tolerance to nitrates in their diet with time, as this allows for the rumen bug population to gear up to utilize the nitrates more efficiently.
3. Feeding three to four pounds of corn per head per day to mature cattle can help the rumen bugs convert the nitrate-nitrite to microbial protein faster. Low-energy diets are known to increase an animal's susceptibility to nitrite poisoning.
4. The best method to feed high-nitrate forage is grinding and mixing it in a total mixed ration (TMR). This can help to minimize sorting, or the boss cows eating more of a low-nitrate hay and leaving other cows to eat greater amounts of high-nitrate forage.
5. Offer excessive amounts of hay, so cattle will eat leafy portions and leave the high-nitrate stems behind.
6. There is no assurance that the forage samples submitted for testing are representative, and some bales may test even higher than reported. Thus, err on the side of caution, especially when feeding pregnant cattle.

7. There are propionibacterium products available in bolus or powder form that are reported to reduce nitrate and nitrite levels in the rumen by approximately 40 percent. These products must be established in the rumen for at least 10 days before allowing cattle to consume high-nitrate feedstuffs.

*Courtesy of Dr. Jeff Lehmkuhler, UK Extension beef cattle nutrition specialist

Cyanide Poisoning

Prussic Acid

Conditions of Poisoning. The primary cause of cyanide poisoning in ruminants is the ingestion of plants containing cyanogenic glycosides (e.g., dhurrin, amygdalin, and prunasin). When plant cells are crushed, chewed, wilted, frozen, chopped or otherwise damaged, plant enzymes release cyanide gas (HCN). Cyanide gas is rapidly absorbed into the bloodstream. Rumen microflora also contain enzymes capable of releasing HCN from the glycosides. Cyanide prevents oxygen release from hemoglobin, and the animal dies of tissue hypoxia (lack of oxygen). Oxygen saturation of venous blood is responsible for the cherry-red color of blood associated with cyanide poisoning.

The cyanogenic potential of plants varies by the species and variety of the plant, weather, soil fertility and stage of plant growth. Cyanide poisoning of livestock has been associated with *Sorghum* species, including Johnsongrass, sorghum-Sudan grass, and other forage sorghum; *Prunus* species (e.g., wild cherry, black cherry, and chokecherry); elderberry (*Sambucus* spp.); serviceberry (*Amelanchier alnifolia*); and less frequently, arrow grass (*Triglochin* spp.), white clover (*Trifolium repens*), bird's-foot trefoil (*Lotus* spp.), and many others.

Young plants, new shoots, and regrowth of plants after cutting contain the highest levels of cyanogenic glycosides. Concentrations decrease as plants mature. Leaf blades contain higher concentrations than leaf sheaths or stems, younger (upper) leaves contain higher concentrations than older (lower) leaves, and seed heads contain low concentrations. Damage to the plant (e.g.,

from frost, drought, insects, storms, treatment with herbicides such as 2,4-D, or other causes) increases the danger by allowing plant enzymes to begin releasing HCN even before the plant is chewed. Drying decreases the cyanogenic potential over time, so adequately cured hay is rarely hazardous. Ensiling will significantly reduce the cyanogenic potential, although to a lesser degree than curing for hay.

The lethal dose of cyanide is in the range of 2 to 2.5 milligrams per kilogram of body weight. Forage cyanide concentrations exceeding 200 ppm are considered high enough to deliver a toxic dose.

Signs. Animals rarely survive more than one to two hours after consuming lethal quantities of cyanogenic plants, and usually die within five to 15 minutes. Signs may include apprehension; rapid, labored breathing; irregular pulse; frothing at the mouth; dilated pupils; muscle tremors; and staggering, followed by collapse and death.

Treatment. Treatment of mildly affected animals is possible, but severely affected animals are often found dead. Contact a veterinarian immediately if cyanide poisoning is suspected. Intravenous sodium thiosulfate is an effective treatment if instituted early. Repeated doses may be necessary if the animal does not respond quickly. Administering 0.5–1.0 liter of a diluted vinegar solution (one gallon of vinegar diluted in three to five gallons of water) via stomach tube can lower rumen pH, reducing the production of hydrogen cyanide; however, stress of handling may exacerbate signs and possibly lead to the animal's death. Most animals that survive treatment recover fully.

Prevention. The risk from potentially dangerous plants and forages may be reduced by following these management practices:

1. Graze sorghum, sorghum hybrids, or Johnsongrass only when the plants are more than 18–24 inches tall. Do not graze plants with young tillers.
2. Do not graze plants during drought periods when growth is severely reduced or the plant is wilted. Do not graze until growth has resumed for four to five days after a rainfall.

3. Do not graze potentially hazardous forages when frost is likely. It may be several weeks after a light frost (in which the plants survive and regrow) before concentrations decrease. Do not graze after a killing frost until the plant material is completely dry and brown (usually within 72 hours).
4. Prevent access to wild cherry leaves. After storms or before turnout to a new pasture, always check for and remove fallen cherry tree limbs and leaves.
5. Do not feed forages suspected of containing high cyanide concentrations as green chop. If cutting for hay, allow plants to dry completely before baling. Delay feeding silage for six to eight weeks following ensiling.
6. Forage species and varieties may be selected for low cyanide potential. There are wide differences among plant varieties. Some of the Sudan grasses, such as Piper, are low in cyanide.
7. Test any suspect forages before allowing animal access. A rapid field test is available that can provide on-site results. Contact your county agricultural extension agent for further information.

Acute Respiratory Distress Syndrome (ARDS)

Conditions of Poisoning. Acute respiratory distress syndrome (ARDS) can be caused by exposure to perilla ketones (perilla mint), 4-ipomeanol (produced by sweet potatoes, scarlet runner beans, peanut hay, and other plants infected with specific mold species), 3-methylindole (a metabolite of tryptophan, which is found in high concentrations in *Brassica* species and in lush pastures of any forage), and other related compounds.

Signs. Clinical signs include sudden onset of difficulty in breathing, with a loud expiratory grunt and open-mouthed breathing or panting. Although mature cattle are most often affected, ARDS can also occur in yearlings and calves. Prognosis is guarded and treatment is ineffective. Atypical interstitial pneumonia (AIP) describes the characteristic pattern of lung damage seen at postmortem examination.

Tryptophan-induced ARDS (fog fever) was first reported more than 200 years ago. Many cases occur in early spring, when cattle that have been fed dry hay over the winter are turned out onto lush, new spring pastures. Certain *Brassica* species (e.g., kale, rape, and turnip tops) also have particularly high tryptophan concentrations and can pose a risk if abruptly introduced into the diet. Signs typically develop several days to weeks after the diet change.

Prevention. Prevention of fog fever is based on management strategies that incorporate gradual forage change and the addition of ionophores in the diet. Monensin can reduce the conversion of tryptophan to 3-MI in the rumen by as much as 90 percent. For monensin to be effective, it must be present in the rumen at the time of exposure and should be fed continuously for at least 10 days after turnout. If cattle have been on lower-quality

forage, gradually introduce them to lush pastures (or *Brassica* spp.) by allowing access for only a few hours per day for the first five to seven days. This is best accomplished by strip grazing pasture utilizing portable electric fencing. Gradually increase grazing time over a 10- to 12-day period.

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Figure 82. N.L. Britton and A. Brown, 1913, *An Illustrated Flora of the Northern United States, Canada and the British Possessions*. Kentucky Native Plant Society, New York, via USDA-NRCS PLANTS database.

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