[[NOTES:

- 1. The text below was pulled from the online database version of this publication that was hosted on the Veterinary Medicine server for many years.
- 2. The original print edition contains additional material not included in this database, including an introduction and acknowledgements. I've tried to note missing parts as where they would have appeared.
- 3. The online database often includes lengthier plant descriptions that those found in the print edition.
- 4. Both the print and online versions use line art for all the plant species. I have not indicated those in the text.
- 5. The print version includes some indices.
- 6. The online version includes some plants not mentioned in the print edition. Those are provided at the end of this file.]]

Indiana Plants Poisonous to Livestock and Pets

Cooperative Extension Service, Purdue University

West Lafayette, IN 47907 Rebecca J. Goetz, writer, extension assistant Thomas N. Jordan, extension weed scientist John W. McCain, extension weed scientist Nancy Y. Su, assistant

Sources and Additional Readings

Botanical texts: Kingsbury, Eshleman, Meuncher, Arena, and Radcliff

Veterinary texts: The Merck Veterinary Manual, poisonous plant booklets for IL, KY and NY, Current Veterinary Therapy for Small Animals, Large Animals and Equine

Veterinary Journals: Veterinary Medicine, Veterinary Record, J. of the American Veterinary Medical Association, J. of Range Management, Modern Veterinary Practice, Compendium for the Practicing Veterinarian, J. of the American Animal Hospital Association, and others

Disclaimer! This material is for informational purposes only, not as a guide to home treatment. Contact a veterinarian or

poison control center in any case of suspected poisoning! This is a work in development. We would appreciate any feedback or suggestions you might have. Please contact the <u>School of Veterinary Medicine Webmaster.</u>

[[The print edition includes an overall introduction to the publication, an acknowledgements section, and an introduction to the "Injurious Plants" section that do not appear in the online database.]]

1. FOXTAIL BARLEY, SQUIRRELTAIL BARLEY, WILD BARLEY

Hordeum jubatum

(grass family)

TOXICITY RATING: Moderate. Irritation from the awns is likely, but serious illness and death are rare.

ANIMALS AFFECTED: All animals can be affected. Grazing animals and outdoor dogs are especially at risk.

DANGEROUS PARTS OF PLANT: Seedheads (awns).

CLASS OF SIGNS: Skin, mouth, ear, nose, and eye irritation; stomach irritation, abscesses.

PLANT DESCRIPTION: This perennial weedy grass (fig. 1) of fields, waste places, and roadsides gets its name from the long bushy flower spikes. Each long, slender, wiry bristle bears small teeth or barbs that point backwards like tiny fishhooks.

SIGNS: The seed heads (especially the awns) of this and several other types of grasses can cause mechanical irritation to the skin, eyes, ears, nose, mouth, and stomach of animals. The grasses can cause problems in pasture as well as in prepared feeds like hay. Embedded seed heads can cause local irritation and infections, or become more deeply embedded in tissues and migrate in the body. Irritation and infection often develop, necessitating removal of the plant matter. Signs depend on location of the seed awn, and can include head shaking; sneezing; nasal or ocular discharge; rubbing at the ears, eyes, or mouth; difficulty in chewing or swallowing; or signs of digestive disturbance. Yellow or green foxtail, rye and millet can cause similar problems.

FIRST AID: For minor irritation, supportive care is all that is required. Minor irritations will resolve in about a day. If any of the following are noted, a veterinarian needs to be contacted: if signs are severe, if signs of irritation do not resolve, if the eyes or ears are involved, or if the animal cannot eat. Infections and abscesses require veterinary care, and some awns may need surgical removal.

SAFETY IN PREPARED FEEDS: Small amounts incorporated in hay and other feeds may not cause much harm, most animals will avoid eating these plants if better quality feed is available (good hay and feeds do not contain many seed heads of these grasses). Hay and other feeds containing large quantities of these seed heads are not safe for consumption, especially if animals are forced to eat these feeds.

PREVENTION: These plants are common weeds in pastures and along roadsides. If problems occur with these plants, consider mowing to reduce seedhead formation or otherwise removing them. The foxtail that commonly grows in the western U.S. (*Setaria*) is especially problematic and animals should have little or no contact with this grass.

2. COMMON BURDOCK

Arctium minus

(daisy family)

TOXICITY RATING: Low. Burs cause local irritation and possibly intestinal hairballs, and most animals avoid ingesting these plants. Serious illness and death are rare.

ANIMALS AFFECTED: All animals may be affected. Pastured animals and outdoor dogs are particularly at risk. Poultry may get burs lodged in the esophagus.

DANGEROUS PARTS OF PLANT: Burs.

CLASS OF SIGNS: Skin, eye, mouth, nose, and ear irritation; stomach irritation; hairballs.

PLANT DESCRIPTION: This familiar biennial weed (fig. 2) of waste places and roadsides grows from a fleshy taproot and produces large, heart-shaped, hairy leaves and red-violet flower heads surrounded by numerous hooked bracts that form a bur-like cup.

Burdock is often confused with cocklebur (a far more dangerous plant). Burdock burs are rounder and have softer, more Velcro-like hairs than cocklebur. Cocklebur burs are oblong and have hooked spines on the bur, and have, on the end of the bur, two spines which are larger and not as strongly hooked.

SIGNS: These burs, and those of cocklebur, crimson clover, rabbitfoot clover, sandbur, and buffalobur may lodge in the skin, eyes, ears, mouth, throat, or stomachs of grazing animals (and sometimes pets). Direct irritation may result, with swelling and pain, or the bur may form the center of a "hairball", a mass of plant matter, hair, and debris that can cause digestive tract irritation and possible obstruction. These burs also tend to lodge in the fur, manes, and tails of animals.

FIRST AID: Remove all visible burs (some may need to be cut out of the hair). If the irritation is minor, provide supportive care. If the eyes, ears, nose, mouth, feet, or hooves are involved, or in cases of digestive upset or inability to eat, a veterinarian needs to be contacted. Burs and hairballs may require surgical removal.

SAFETY IN PREPARED FEEDS: Only poor quality hay and feeds would contain these burs. If the number of burs is small or only in certain sections of the hay (which should be discarded), few problems may result. If there are many burs, especially if the animals are forced to eat these feeds, problems are likely to develop.

PREVENTION: Prevention consists of removing these plants from the pasture. Mowing is effective in reducing the number of flowers and burs that will develop.

[[SECTION: House and Garden Plants]]

3. THE AROID FAMILY

Aglaonema: CHINESE EVERGREEN

Anthurium: ANTHURIUM

Arisaema: JACK-IN-THE-PULPIT, GREEN DRAGON

Caladium: ELEPHANT EARS

Colocasia: TARO

Dieffenbachia: DUMBCANE

Monstera: CUT-LEAF PHILODENDRON, CERIMAN, MEXICAN BREADFRUIT

Philodendron: PHILODENDRON

Scindapsus: DEVIL'S-IVY, POTHOS

Symplocarpus foetidus: SKUNKCABBAGE

Syngonium: TRI-LEAF WONDER, ARROWHEAD VINE, NEPTHYTIS

TOXICITY RATING: Moderate. Pets may sample these commonly available plants with a nibble or two, but rarely ingest any quantity sufficient to cause serious problems or death. Risk increases with hungry or bored animals housed in close proximity to these plants.

ANIMALS AFFECTED: Any animal that chews or ingests the leaves will be affected. In Indiana, nearly all of these plants would be grown as houseplants, therefore pets (including birds and reptiles) are primarily at risk.

DANGEROUS PARTS OF PLANT: Roots, leaves, stems.

CLASS OF SIGNS: Mouth and throat irritation, salivating, possibly stomach irritation, diarrhea (rarely).

PLANT DESCRIPTION: All 2,000 species of this family of plants should be treated as potentially toxic. A few are eaten, such as poi and taro (*Colocasia esculenta*) in Hawaii, but only after the poison is eliminated by cooking. Seven species of aroids occur naturally in Indiana, mostly in wet areas. Jack-in-the-pulpit and skunkcabbage are the most common and best known of these. Dumbcane (fig. 3), pothos, and philodendron are potted plants of offices, restaurant lobbies, and homes.

Aroids are perennials, many arising from corms or rhizomes. Some may be vines. The large netveined leaves, which may have white or colored spots, are borne on leaf stalks that sheathe the stem. Most of these plants have simple leaves, but jack-in-the-pulpit has three-parted foliage (fig. 19). The aroid flower is a fleshy green, white, or yellow spike (spadix) borne inside a wraparound hood or bract (spathe). The fruits are brightly colored berries, borne in tight clusters, not often produced by the house plant species.

SIGNS: The plant cells contain needle-like crystal of insoluble calcium oxalate which penetrate the skin and mouth causing discomfort. In addition, the plants contain proteolytic enzymes which release histamine and kinins, causing swelling and an itching or burning sensation. Affected animals will shake their head, paw or rub the face and mouth, may salivate or foam at the mouth, may seek water, or may have visible swelling. Very severely affected animals may experience oral swelling to the point that swallowing and breathing become impaired. Typically, animals are not severely affected, since a few bites of the plants are often a sufficient deterrent to further consumption. Occasional reports of these plants causing kidney failure in cats have not been well-verified. Effects in cats appear to be limited to the signs described above. Some of these plants have been used with humans to prevent individuals from talking by causing excessive tongue swelling, hence the name "dumbcane".

FIRST AID: For minor irritation, provide supportive care and prevent further exposure. For more severe signs, if the animal does not improve within a few minutes, or if swallowing or breathing is impaired, consult a veterinarian immediately.

SAFETY IN PREPARED FEEDS: These plants are not likely to be incorporated into hay or other feeds, but if so, the toxins are likely to remain.

PREVENTION: Animals should not be allowed to consume these plants. Offer small amounts of fresh grass or other safe plant material (depending on the species), or remove the plant from the pet's environment. Some pets do not "learn their lesson" and may return to chew on these plants. In these situations, it is best to remove the plant from the pet's environment.

4. BULB-BEARING PLANTS OF HOUSE AND GARDEN

TOXICITY RATING: Moderate. Incidents of poisoning by these plants is rare, but special care must be taken, especially with lily of the valley.

ANIMALS AFFECTED: Pets are primarily at risk, cattle have been poisoned by iris.

DANGEROUS PARTS OF PLANTS: Bulbs, rootstocks, whole plants.

CLASS OF SIGNS: Stomach upset, vomiting, diarrhea. For lily of the valley, additional signs may include irregular heartbeat, convulsions, and death.

PLANT DESCRIPTIONS:

1. *Convallaria majalis* (lily-of-the-valley, lily family). A familiar low-growing garden perennial, forms dense clumps from slender rootstocks called "pips." The lily-like leaves are parallel-veined (fig. 4A), and from them rise flower stalks that bear small white blossoms all on one side. The flowers are bell-like and fragrant and rarely develop into the red to orange-red berries.

2. *Hippeastrum vittata* (amaryllis, amaryllid family). A houseplant (fig. 4B) that blooms in only a few weeks after forcing in the wintertime. At first one or two 1 to 3 feet tall, naked stalks appear, each bearing from one to four large, six-petaled, red, blue, white, or bicolored flowers. Later several sword-shaped, fleshy leaves develop from the base of the plant.

3. *Hyacinthus* spp. (hyacinth, lily family). These potted or garden plants grow from a 1 to 2 inch diameter bulb (fig. 4C). The 8 to 12 inch long leaves are narrow, somewhat trough-shaped, and fleshy. The small fragrant, white, pink, or blue lily-like flowers are borne on a leafless stalk that is taller than the leaves.

4. *Iris* spp. (iris or blue flag, iris family). These commonly grown garden perennials also occur wild in wet

meadows, marshes, roadsides, lakeshores, and stream banks. Branching, fleshy rootstocks bear clusters of long, sword-like leaves in which the base of each leaf is folded over the base of the next higher leaf (fig. 4D). The flowers, blue with a yellow heart in our wild species but purple, blue, yellow, or reddish-brown in cultivated varieties, have three upright "standard" petals and three pendant "fall" petals. The fruit is a dry capsule.

5. *Narcissus* spp. (daffodil, jonquil, narcissus; amaryllid family). Springtime in Indiana is officially proclaimed by the yellow trumpets of daffodils (fig. 4E). These perennials produce

lily-like leaves and slender stalks that each bear a conspicuous orange, white, or yellow flower with six petals, parts of which fuse to form the trumpet.

SIGNS: This group of plants is divided into two sections. The first section includes lily of the valley, and the second section includes the other bulbs.

Lily of the valley is the far more dangerous plant, producing a mixture of many cardiac glycosides, especially convallatoxin. Toxic signs in pets after they chew on the plant would include stomach upset, vomiting, irregular heartbeat, convulsions, and death if sufficient quantities are consumed. The toxin in lily of the valley acts in a similar manner to the toxin in foxglove, a plant from which digitalis, a powerful cardiac medication, is derived.

The bulbs or corms of tulip, daffodil, jonquil, narcissus, amaryllis, and iris produce primarily gastrointestinal signs (vomiting and diarrhea) after consumption, and are not as toxic as lily of the valley. Pets are more likely to come into contact with these plants than are horses or livestock, however, livestock (cattle) have been poisoned when grazing wild-growing iris and eating the rootstocks.

Concerning pets, dogs are the most likely to consume the bulbs, and the usual time for poisoning is when the bulbs are removed from the ground or stored prior to planting. This is not a common poisoning, but some animals, once they find the stored bulbs, will consume them. While unlikely, some animals will dig the bulbs up in the yard and eat them. The toxic components in these bulbs is not well identified, but may be a mixture of alkaloids, having an irritant action on the gastrointestinal tract.

FIRST AID: For lily of the valley, contact a veterinarian immediately. If the animal is vomiting, allow it to continue to do so, since this will remove the toxin from the gastrointestinal tract. If the vomiting is severe or persists, a veterinarian needs to be contacted. In cases where the pet has eaten a large quantity, emergency treatment is imperative. For the other bulbs and for iris, vomiting and diarrhea should resolve in a few hours. If the signs continue, or if the pet is weak, sick, pregnant, nursing ,or old, a veterinarian can provide supportive care until the toxin is eliminated.

SAFETY IN PREPARED FEEDS: It is unlikely that these plants would be incorporated into animal feeds. On the rare event that this occurs, the toxins would remain, and the feed is then unsafe for consumption.

PREVENTION: Do not allow livestock to graze where iris grows, especially if there is insufficient forage available. For pets, do not allow access to stored bulbs, and restrict access to bulb beds and newly planted bulbs. Never pen or place an animal within reach of these plants, especially lily of the valley, since the pets (including birds) may nibble them out of boredom.

5. ENGLISH IVY

Hedera helix

(ginseng family)

TOXICITY RATING: Low. Although reported to be very toxic in a few scattered reports, this is not a frequently encountered toxicosis.

ANIMALS AFFECTED: Cats, dogs, birds, other pets.

DANGEROUS PARTS OF PLANT: Leaves, berries.

CLASS OF SIGNS: Oral irritation, stomach irritation, diarrhea, breathing problems, coma, death.

PLANT DESCRIPTION: The vines that give many colleges their "halls of ivy" appearance may be poisonous. These woody vines, also used as groundcovers, have alternate, palmately-lobed, dark green leaves with lighter green veins (fig. 5). Flowers are uncommon but are borne in small umbrella-like clusters and produce small black berries.

SIGNS: A saponic glycoside, the aglycone hederagenin, is found in the leaves and berries. This is an uncommon poisoning, but incidents have been reported in dogs, cats, and pet monkeys, especially when the green berries were eaten. The most common signs relate to mouth and stomach irritation, but coma and death may occur if large quantities are consumed.

FIRST AID: There is no specific antidote. If animals are observed eating English ivy, contact a veterinarian immediately, especially if a large quantity of the plant was consumed. A bite or two of a leaf is not likely to cause a serious problem, but this should be discouraged.

SAFETY IN PREPARED FEEDS: There are no clear reports on this, but consider English ivy unsafe in prepared feeds.

PREVENTION: Keep animals, especially curious nibblers, away from English ivy. Exercise caution for indoor animals as well, since English ivy grows up the sides of buildings and can grow through window openings.

6. LUPINE, BLUEBONNET, QUAKER-BONNETS

Lupinus perennis (wild)

Lupinus polyphyllus (cultivated)

(pea family)

TOXICITY RATING: Low to moderate in Indiana. In the western rangelands, where lupine grows plentifully, the risk of toxicosis would be high. Different species of lupine have different toxicities. According to reports, *L. leucophyllus* (velvet or wooly-leafed lupine) is the most toxic and should never be grazed since all stages of plant growth are toxic.

ANIMALS AFFECTED: Sheep are primarily affected, but all animals are susceptible.

DANGEROUS PARTS OF PLANT: All parts, especially pods with seeds.

CLASS OF SIGNS: Breathing problems, behavioral changes, trembling, birth defects, coma, death.

PLANT DESCRIPTION: Lupines (fig. 6) are herbaceous perennials grown in gardens or found wild along roadsides, in fields, and in open woods. Wild lupines are common only in the prairie and lake counties of Indiana. In the rangelands of the West, they are a leading cause of livestock poisoning. Several stems often grow from one creeping root and reach 12 to 30 inches in height. The leaves are alternate and pal-

mately compound with 7 to 11 spear-tip-shaped, softly hairy segments. Elongate spikes of blue, purple, white, magenta, or bicolored pea-like flowers in early summer are followed by 1- to 2-inch, fuzzy, pea-like pods.

SIGNS: Toxicity in lupine is believed to result primarily from the alkaloid D-lupanine. The signs of lupine poisoning can develop within an hour or may take as long as a day. The signs are related to the nervous system and resemble the signs seen with excessive consumption of

nicotine (tobacco): twitching, nervousness, depression, difficulty in moving and breathing, and loss of muscular control. If large quantities were consumed, convulsions, coma, and death by respiratory paralysis may occur. In cows that graze lupine, skeletal birth defects in calves can occur, and the syndrome is called "crooked calf".

FIRST AID: There is no antidote. Allow affected livestock to rest quietly, especially if they are unfamiliar with human contact. Handling, trailering, or other stress on the animals after they have been grazing lupine will make the signs worse and can increase losses.

SAFETY IN PREPARED FEEDS: The alkaloids in lupine remain after the plants have dried, so prepared feeds are unsafe for consumption, especially if the feeds contain lupine seed pods.

PREVENTION: Do not allow hungry animals access to lupine, particularly when in the seed stage, if other forage is not available. If lupines are prevalent in the pasture, become familiar with the particular species, since toxicities vary. Do not handle, process, or ship animals that are heavily grazing lupine since this type of stress will increase the number of animals that will become sick and/or die. Livestock can graze lupine without incident as long as excessive ingestion is avoided and animals are not handled or trailered while on lupine pastures (and if the animals are not pregnant). In cattle, to avoid birth defects, do not allow grazing between days 40 and 70 of gestation.

7. CATNIP

Nepeta cataria

(mint family)

TOXICITY RATING: Low

ANIMALS AFFECTED: Cats.

DANGEROUS PARTS OF PLANT: Stems, leaves.

CLASS OF SIGNS: Behavioral changes.

PLANT DESCRIPTION: Catnip (fig. 7) has all the characteristic earmarks of a member of the mint family: stems square in cross-section, leaves opposite and fragrant, and small flowers in tight clusters at the ends of branches. This perennial herb may grow up to 3 feet tall and be highly branched. The gray-green to green leaves are heart-shaped with scalloped edges and are often crowded toward the top of the plant. The flowers are white, dotted with purple, two-lipped, and produce four tiny, dark nutlets per flower.

SIGNS: Only cats are affected, and some cats are affected more than others. Aromatic oils and the monoterpene, nepetalactone, cause the signs. Cats will rub and sometime ingest the plant, and then act "drunk" or "wild" for up to an hour or more. No lasting toxicity is reported. If excessive amounts are ingested, vomiting and diarrhea can result, but the signs are self-limiting. Catnip can be considered as a legal recreational drug for cats!

FIRST AID: None is required.

SAFETY IN PREPARED FEEDS: Not applicable.

PREVENTION: Catnip is safe, however avoid excessive ingestion since vomiting or diarrhea may occur.

8. CHRISTMAS PLANT

Poinsettia pulcherrima (Euphorbia pulcherrima)

(spurge family)

TOXICITY RATING: Low.

ANIMALS AFFECTED: All animals can be affected, but pets are more likely to come into contact with Poinsettia than are livestock.

DANGEROUS PARTS OF PLANT: Leaves and stems primarily, but all parts may be toxic.

CLASS OF SIGNS: Skin, mouth, eye, and stomach irritation.

PLANT DESCRIPTION: People commonly display this potted plant (fig. 8) in houses and offices in the wintertime. These 1 to 4 feet tall plants with yellow stems bear alternate, coarsely toothed, smooth, green leaves. The top leaves turn red. Although many people mistake them for petals, they function as "bracts", calling attention to the true flowers which are tiny, yellow, and clustered at the top of the plant. The inconspicuous fruits are small, green, three-lobed, fleshy capsules.

SIGNS: The milky sap (a latex) is irritating to skin, eyes, and mucus membranes. Once considered extremely poisonous, toxicity is more likely to manifest as irritation, discomfort, rash, and stomach upset. Nausea and vomiting may occur if sufficient quantities are consumed. Typically, animals will show head-shaking, salivation, and pawing or rubbing at the mouth or eyes.

FIRST AID: Wash sap off the animal to prevent further ingestion. Call a veterinarian if the eyes are affected, or if signs do not resolve in a few minutes.

PREVENTION: Poinsettia should not be allowed near curious animals.

9. RHUBARB, PIE PLANT

Rheum rhaponticum

(dock family)

TOXICITY RATING: Low, unless animals are fed the leaves intentionally.

ANIMALS AFFECTED: All animals may be affected, those being fed garden trimmings are particularly at risk (cattle, swine, sheep and goats).

CLASS OF SIGNS: Staggering, trembling, breathing difficulties, weakness, diarrhea, increased drinking and urinating, death.

PLANT DESCRIPTION: This herbaceous garden perennial (fig. 9) develops from a heavy rootstock. Its leaves grow from the base of the plant on stout, shiny, red stalks. Heart-shaped and 1 to 2 feet long by 1/2 to 11/2 feet wide, the leaf blades have a smooth and shiny surface, darker above, with five main veins and wavy margins. The hollow stems end in greenish-white flower clusters in late spring.

SIGNS: The leaves contain oxalic acid, soluble oxalates, and citric acid, although the stems are edible. Some oxalates are insoluble and cause local irritation but the oxalates in rhubarb (and other species, such as sorrel or dock, *Rumex*) are soluble, and cause systemic problems, especially in the kidneys, or they can affect the electrolytes in the body, such as the balance of calcium and magnesium. Poisoning can be acute, when large amounts of oxalates are consumed

quickly, or may be chronic, where smaller amounts are eaten over a longer period of time. Low blood levels of calcium and kidney failure are commonly reported findings in soluble oxalate toxicity.

Affected animals will appear depressed, and may stagger and tremble and be weak. Often, they will drink and urinate more as kidney function declines. Diarrhea may be noted, and affected animals may die if the electrolyte balance is extremely deranged or if the kidneys fail.

FIRST AID: There is no specific antidote for oxalate toxicity. If an animal is observed eating a large quantity of rhubarb or other oxalate plant, call a veterinarian immediately. If the plants were ingested a day or more previously, only supportive care can be given. A veterinarian will be able to provide assistance, although death may result from electrolyte imbalance or from kidney failure.

SAFETY IN PREPARED FEEDS: Rhubarb is not safe in hay, nor is any other oxalatecontaining plant.

PREVENTION: Ruminants have some ability to adapt to oxalate plants if they are introduced to them gradually, since the rumen bacteria will detoxify the oxalates to a certain degree, although feeding these plants is never recommended. Do not allow animals to ingest large amounts of oxalate plants quickly, and it is best to not allow the feeding of oxalate plants at all. Do not incorporate weeds or rhubarb leaves into feed for animals, especially ruminants and swine, since these animals are not finicky eaters. Always make sure that animals have sufficient water, since oxalate toxicity is worsened if animals go thirsty.

10. AZALEA, RHODODENDRON

(Rhododendron spp.)

(heath family)

TOXICITY RATING: Moderate. These plants grow wild in the East and cause significant problems there, the danger from these plants in Indiana is much less.

ANIMALS AFFECTED: All animals may be affected.

DANGEROUS PARTS OF PLANT: all parts, especially leaves.

CLASS OF SIGNS: Stomach irritation, abdominal pain, abnormal heart rate and rhythm, convulsions, coma, death.

PLANT DESCRIPTION: These perennial shrubs have tough, glossy, smooth-margined evergreen leaves. The large, showy flowers are in terminal clusters and have five white, pink, or red petals (fig. 10). Some horticultural varieties have yellow or orange petals. Common and local names for these plants include "lambkill" and "calfkill". These plants have been used by people to commit suicide.

SIGNS: These plants, as well as mountain laurel (*Kalmia* spp.) contain grayanotoxins (glycosides) which affect the gastroenteric (stomach and intestines) and cardiovascular systems. The older name for this toxin was andromedotoxin.

In order for toxic signs to manifest, 0.2% by weight of green leaves needs to be ingested. Gastroenteric signs develop first, generally within 6 hours of ingestion, including salivating, vomiting (in capable species), diarrhea, abdominal pain, and tremors. Disturbances in cardiac rate and rhythm may then be noted. If sufficient quantites were consumed, convulsions may occur, followed by coma and death. Not all affected animals will die, and livestock may recover without treatment, depending upon amount ingested.

FIRST AID: Prevent further ingestion and provide supportive care. Veterinary attention is needed if ingestion was recent, or if clinical signs are present.

SAFETY IN PREPARED FEEDS: These plants are not safe in hay nor in any other prepared feed.

PREVENTION: Animals should not be allowed to graze these plants. Keep hungry livestock away from areas where these plants grow. Pets may nibble or taste the leaves out of curiosity or boredom, and this is not advised, but seldom leads to clinical toxicosis. Honey made from the nectar of these flowers is also toxic and should not be consumed, so exercise caution when placing beehives.

11. CASTORBEAN, CASTOR OIL PLANT

Ricinus communis

(spurge family)

TOXICITY RATING: High. Death is likely with consumption of even small amount of castorbean.

ANIMALS AFFECTED: All animals may be affected.

DANGEROUS PARTS OF PLANT: The seeds are the primary source of toxin, but the rest of the plant may be considered to be slightly toxic as well.

CLASS OF SIGNS: Stomach irritation, diarrhea, abdominal pain, increased heart rate, profuse sweating, collapse, convulsions, death.

PLANT DESCRIPTION: This stout, robust shrub-like plant with reddish to purple stems may reach 12 feet in height. A perennial in its native tropics, it is grown as a garden annual in Indiana. The large (4 to 30 inches across), umbrella-like leaves (fig. 11) have 5 to 9 pointed, finger-like lobes. Long purple leaf-stems are attached near the centers of the leaf blades. Greenish-white or reddish-brown flowers are produced in narrow, upright clusters. The fruit is a three-lobed, green or red capsule with a soft, spiny

exterior. One large, mottled, attractive seed (fig. 11A) develops in each lobe. Some people think the seeds look like well-fed ticks.

SIGNS: The phytotoxin ("plant toxin") in castorbean is ricin, a water soluble protein, which is concentrated in the seed. Also present are ricinine, an alkaloid, and an irritant oil. As a side note, commercially prepared castor oil contains none of the toxin. There is great species variability in susceptibility to ricin toxin, with humans and horses being the most at risk (25 grams of castorbean seed is lethal for the horse). Due to the powerful toxin however, it is best to keep all animals (livestock and pets) away from this plant.

The seed is only toxic if the outer shell is broken or chewed open. Seeds swallowed intact usually pass without incident. Signs of toxicity may not manifest for 18 to 24 hours after ingestion. The animal first shows signs of depression and a mild increase in temperature. Later, gastrointestinal signs predominate, including vomiting in humans and other species that can

vomit (horses cannot vomit), profuse diarrhea (which may be bloody), colic and abdominal pain. The affected animal may then go into convulsions, collapse and die, with death generally occurring within 36 hours of consumption. The toxic signs are the result of severe gastrointestinal irritation, anaphylaxis and shock. This toxin is closely related to the toxin in rosary pea (a section on this plant is also included in this book).

FIRST AID: If animals are observed eating castorbean, call a veterinarian immediately, since emergency measures need to be undertaken. If consumption occurred several hours ago, a veterinarian will be able to provide supportive care and treat for shock, but death may still result from this powerful toxin.

SAFETY IN PREPARED FEEDS: The seeds may become incorporated into hay or grains. The seeds are never safe to eat, especially by horses or livestock, since these animals could easily crack the seed coat. After making castor oil, the remaining seed cake is sometimes used for animal feed. This needs to be boiled or heated to remove the toxin.

PREVENTION: Do not allow seeds or any other part of the plant to be incorporated into animal feeds. It is advisable to not let castorbean grow in the vicinity of pastures used for grazing or forage production. Do not allow pets access to this plant. To decrease the chances of intoxication, snip off the flower heads before they develop into seeds; this will protect children as well as pets.

12. COMMON TANSY

Tanacetum vulgare

(daisy family)

TOXICITY RATING: Low.

ANIMALS AFFECTED: All animals may be affected.

DANGEROUS PARTS OF PLANT: Leaves, stems.

CLASS OF SIGNS: Salivating, colic, abdominal pain.

PLANT DESCRIPTION: These old-fashioned garden plants (fig. 12) were introduced from Europe and are now found in old gardens or along roadsides, creek banks, and waste areas. They are perennial herbs from a short, stout rootstock and bear alternate fern-like leaves with saw-toothed margins. The yellow, strong-scented, bitter-tasting flowers are in flat-topped clusters at the tops of the plant.

SIGNS: This is not a commonly reported toxic plant. Animals rarely ingest it, and this plant rarely occupies significant acreage in pastures. Illness and death in humans has been reported due to attempts at medicinal uses of concentrated plant extracts. Toxic signs may include salivating and abdominal pain (colic), with the possibility of convulsions and abortions occurring. The oil tanecetin is believed to cause the clinical signs.

FIRST AID: The risk of serious illness and death is slight with this tansy. Provide adequate and proper forage if the animals are eating this plant, and do not let pets nibble on the leaves or flowers.

SAFETY IN PREPARED FEEDS: Information is lacking on this, however, consider the plant toxic in prepared feeds.

PREVENTION: The risk of toxicosis is low with tansy, however if a pet begins to chew this plant, consider keeping the pet and plant separated (especially if the pet is confined in the plant's vicinity). If tansy occupies a significant amount of a pasture, consider mowing or otherwise eliminating the plant, or keeping the animals and plants separated.

[[SECTION: Forage and Crop Plants]]

13. CULTIVATED OATS

Avena sativa

(grass family)

TOXICITY RATING: Moderate.

ANIMALS AFFECTED: Cattle, swine, sheep, goats, poultry, horses.

DANGEROUS PARTS OF PLANT: All parts.

CLASS OF SIGNS: Breathing difficulty, skin irritation, paralysis, convulsions, death (rarely).

PLANT DESCRIPTION: This widely cultivated annual grass has escaped into waste places. The plants grow up together in small tufts. The fruiting structure is a terminal, loose panicle (fig. 13).

SIGNS: Oats may cause up to three separate problems, depending upon the conditions:

a) Oats can accumulate excessive amounts of nitrates, especially when heavily fertilized. Many grasses and forages can accumulate toxic levels of nitrates depending upon fertilization practices. This is a serious problem, especially in cattle. The signs are then consistent with nitrate toxicosis: the blood turns a brownish color due to the action of nitrate on hemoglobin. The blood is not capable of carrying oxygen efficiently, and animals show signs of hypoxia: increased respiratory rate, anxiety, collapse, convulsions, and death. Nitrate toxicosis is less often reported in non-ruminants (horses, swine, etc), but can occur. The primary signs in these species is stomach and intestinal irritation, although an inability of the blood to carry oxygen may develop and require treatment.

b) Oats can also induce a photosensitivity reaction in animals, although the exact mechanism (direct contact and/or ingestion) is uncertain. The skin becomes increasingly susceptible to damage by ultraviolet radiation, and the typical presentation in affected animals is sunburn on the lightly pigmented parts of the body. Both fresh oats and oat straw can cause a skin reaction.

c) Moldy oats (called smutty oats), which result from the improper curing or storing of oat hay, can cause paralysis, convulsions or death if sufficient quantities are ingested.

FIRST AID: In all three cases of possible toxicoses, the first and most important step is to remove the oats from the animals.

a) For nitrate toxicosis, it is best to call a veterinarian, especially when cattle or other ruminants are affected. There are emergency measures that can be performed, but severely affected animals still may die. Horses, swine, and poultry are less likely to be affected, but if clinical signs develop, they may require treatment as well, although it may be impractical to attempt treatment on swine or poultry.

b) For photosensitization, allow the animal access to shade. The animal may need shade for a week or two until the sunburned areas heal. Animals on pasture can be turned out at night and stabled during the day. Remove oat straw if used for bedding. The sunburn may be severe

enough to prevent normal function, such as seeing, eating, walking, allowing young ones to nurse, etc. In these cases, a veterinarian needs to be called to provide supportive care until the lesions heal. In addition, a veterinarian needs to be called if the sunburned areas are extensive or if they appear infected, or if the animal is depressed and unwilling to eat.

c) For moldy oats, discard affected feed and provide proper forage. If clinical signs develop, call a veterinarian.

SAFETY IN PREPARED FEEDS: Ideally, oat hay with excessive levels of nitrate should not be fed. If feeding this hay cannot be avoided, the animals should receive a well-balanced and adequate diet, since these animal will be less likely to show toxic signs. Nitrate contaminated feed should only make up a small portion of the total ration. Since the cause of oat photosensitivity is unknown, consumption of oat hay or grass may induce the clinical syndrome. Moldy oats are not safe for consumption and should be discarded.

PREVENTION: Ideally, the oat feed should be discarded. If this is impossible, know that properly fed cattle can consume forages higher in nitrate than can poorly fed cattle, therefore, feed cattle an adequate and balanced diet, especially if high nitrate forages are to be included in the diet. Do not over-fertilize oats or any grass forage, to reduce the likelihood of this common toxicosis. Photosensitive individuals may need to have oats permanently removed from their diet. Moldy oats should never be fed.

14. ERGOT

Claviceps purpurea

(fungus)

TOXICITY RATING: Moderate. While ergot itself is extremely dangerous, modern feed production practices are vigilant in not allowing ergot into grain mixtures. Pasture poisoning remains the most likely source for toxicosis.

ANIMALS AFFECTED: Any animal consuming affected grain or grass seed heads: primarily swine, cattle, sheep, and goats. Poultry and horses may also be affected.

DANGEROUS PARTS OF PLANTS: Fungal bodies in the seed heads of grains and grasses.

CLASS OF SIGNS: Behavioral changes, lameness, abortions, convulsions, gangrene, death.

PLANT DESCRIPTION: Ergot (fig. 14) is a fungus parasite of the heads of grasses. In mid- to late-summer, entire grains are replaced by the black, hardened bodies of the fungus. Each body is up to 1 inch long, larger than the normal grain, and protrudes conspicuously from the head. One to a half dozen ergot bodies may develop on one head of grass. Ergots on wheat are straight. On rye they usually are curved. Ergot is found wherever its host plants grow. Rye is the most frequently attacked grain. Other grain, hay, turf, and weedy grasses may be affected, including wheat, oats, wild rice, Kentucky bluegrass, perennial ryegrass, redtop, smooth brome, orchardgrass, quackgrass, reed canarygrass, etc. When grain or hay is harvested, ergot bodies may fall to the ground and be left behind to infect the next season's crop.

SIGNS: The amines and the alkaloids in ergot (ergotamine being one of the major alkaloids) produce a number of clinical signs relating primarily to vasoconstriction and psychoactive effects. The ergot toxins are very similar structurally to lysergic acid (LSD). Wheat and rye contaminated with ergot has affected humans, and the resulting disorder is called St. Anthony's

Fire. It is also believed that the one of the reasons for the Salem witch trials was due to the abnormal behavior of persons affected with ergot toxicosis from contaminated rye flour. Ergot has been used in herbal medicine to control hemorrhage during childbirth, and is used currently as a drug to help control migraines.

Animals may be affected by ergot from eating small amounts over a long period of time, or eating greater quantities in a short period of time. Chronic toxicity is more common, with signs manifesting within several weeks of ergot consumption, and field exposure to ergot is more common than processed feed or flour exposure.

The clinical signs can vary, but often begins with swelling of and pain in the extremities, especially the hind legs, due to the constriction of blood vessels. Later, sensation is lost in the affected areas, and dry gangrene can develop. Eventually, the affected areas may slough ("fall off"), with typical losses including claws, hooves, teats, ears, or tail. In addition to the vascular effects, neurologic signs are often seen, such as apprehension, nervousness, and convulsions. Some animals may show signs of depression. Death can occur, but only with significant quantities ingested. In sublethal cases, once the source of ergot is removed, recovery from neurologic signs is likely, but recovery from the vascular effects and gangrene is not likely. Ergot poisoning has been implicated in cases of abortion in large animals.

FIRST AID: Remove animals from the source of ergot, whether in feed or pasture. Call a veterinarian if nervous signs are present. In cases of dry gangrene, the affected portion will be likely to spontaneously amputate. A veterinarian can advise and assist. Always exercise caution when working around animals that have nervous and behavioral changes to prevent human injury.

SAFETY IN PREPARED FEEDS: Ergot remains toxic in feed and should never be fed. Most feed producers are aware of ergot, so it is rare to have it contaminate commercially prepared feeds.

PREVENTION: Screen grains carefully prior to feeding or planting. Affected pastures may produce ergot each year under the right environmental circumstances (typically after wet, cool spring growing seasons). Commercially prepared feeds will not contain ergot. Pasture managers need to be able to identify ergot and limit animal access to affected fields. Keeping the pasture grasses mowed to prevent seed head formation will greatly decrease the presence of the ergot fungus. The fungus is likely to be present in the pasture, so infestations may occur in any year with the right environmental conditions.

15. TALL FESCUE

Festuca arundinacea

(grass family)

TOXICITY RATING: Moderate to high, depending upon individual circumstance.

ANIMALS AFFECTED: Horses, cattle, possibly other ruminants.

DANGEROUS PARTS OF THE PLANT: Seed head, stem and leaf sheath.

CLASS OF SIGNS: Reproductive problems, "poor doers", lameness, dry gangrene, fever, death.

PLANT DESCRIPTION: This grass (fig. 15), often cultivated in wet pastures for forage or for turf, is a perennial, 3 to 4 foot tall clump grass with medium-wide leaves that are rough-ribbed

on top. It has no rootstocks (rhizomes). The heads are open and many-branched. Escaped plants may be found along roadsides and in waste areas, especially in the southern half of the state.

SIGNS: Toxicity is the result of an endophytic ("inside the plant") fungus, *Acremonium coenophialum*, which is believed to enable the grass to be more hardy and outcompete other grass species. The grass itself is not toxic. The fungus is passed in the seed, and is not transmitted directly from plant to plant.

In horses, pregnant mares are most at risk when eating fescue, since the alkaloids produced by the fungus inhibit prolactin release. Mares will have an increased risk of prolonged gestation, abortion, stillbirth, dystocia (difficult birth), foal mortality, retained or thickened placenta, no milk, and mare death (in foaling, or from a retained placenta).

In cattle, several syndromes have been reported, including fescue toxicosis (summer slump), fescue foot and abdominal fat necrosis. Summer slump causes slower gains, decreased milk production, poor appetite, retention of winter coat, reproductive problems, and elevated temperature. Diarrhea may also be present. Summer slump tends to occur in the warmer months, but has been noted at any time of year, and is the most common of the three syndromes. Fescue foot tends to develop in the late fall and winter, and the extremities (typically tail, ears, and rear feet) undergo necrosis ("death"). Another name for this type of necrosis is "dry gangrene". Fat necrosis develops when areas of fat inside the abdomen die.

Additional note: Fescue can accumulate nitrates under conditions of overfertilization (see the section on oats for more information on nitrate toxicosis).

FIRST AID: There is only supportive and symptomatic treatment once signs appear. A veterinarian can advise on treatment of more severely affected animals. Pregnant mares will be likely to need assistance when foaling and in the post-foaling period. Foals that survive will require supplemental colostrum. Management and prevention are the best means to minimize losses.

SAFETY IN PREPARED FEEDS: The toxin remains active in hay.

PREVENTION: There are several options, depending on the farm situation. Fungicides do not work, so animal and pasture management are the only viable alternatives. Pastures can be tested for the presence and degree of fungal contamination, and reseeding may be required. If reseeding the pasture is not feasible, keeping the pasture short will prevent seed formation. Feeding other forages, such as other warm season grasses or legumes, will be of benefit. Fescue pastures can also be diluted with legume planting (red or white clover). Heavy fertilization may make the problem worse, especially in cattle. If fescue has to be used for mares, at least avoid feeding fescue hay or pasture during the last 30 to 60 days of gestation to minimize problems. Endophyte-free strains of fescue exist, although they do not grow as well as tall fescue with endophyte.

16. YELLOW SWEETCLOVER WHITE SWEETCLOVER

Melilotus officinalis Melilotus alba

(pea family)

TOXICITY RATING: Moderate.

ANIMALS AFFECTED: All animals that eat affected hay may be poisoned.

DANGEROUS PARTS OF PLANTS: All above-ground parts when present in moldy hay.

CLASS OF SIGNS: Bruising, spontaneous bleeding.

PLANT DESCRIPTION: These coarse biennial herbs (fig. 16) have alternate, three-parted, toothed leaves and bear white or yellow flowers in long, slender, spike-like clusters in the leaf axils. The numerous small, pea-like, white or yellow flowers fall soon after blooming. Pods are small, egg-shaped to round, inflated, and contain 1 to 4 seeds. Sweetclover grows along roadsides, fence rows, and in old fields. It is cultivated as a forage crop and soil builder. The plants favor alkaline or calcareous soils.

SIGNS: Clinical signs are related to the anticoagulant ("prevents blood clotting") activity of dicoumarol (also called dicoumarin). Coumarin, present in sweet clover, is converted to dicoumarin during improper curing of sweet clover hay, or when the plant is excessively stressed (frosts, drought). Fresh, undamaged sweetclover is safe for consumption.

Signs are related to the consumption and inadequate production of vitamin K, responsible for blood clotting, therefore excessive and uncontrolled bruising and bleeding will occur. The bleeding may be noticeable (through the nose, mouth or a wound), or may occur under the skin as large bruises, but can also occur inside the body, making an accurate diagnosis more difficult. The toxin can be passed in the milk, therefore nursing animals may be affected. The moldy hay needs to be consumed for 2 weeks or longer before signs manifest and this toxicosis is most often seen in winter after several weeks of moldy sweetclover has been consumed and is typically a herd problem.

Affected animals are weak, anorexic, may exhibit visible bleeding, have pale mucus membranes, increased respiratory rates, rapid and weak pulses, and may die. Often more than one animal is affected at a time.

Dicoumarin and related drugs are used in human medicine as "blood thinners" and are commonly used in rat and mouse poisons (which if ingested by any other animal can cause excessive bleeding and death if not treated).

FIRST AID: Remove all moldy hay from the animals. A veterinarian needs to be called if animals are bleeding, although once bleeding starts, it may be difficult to save the animal. Treatments can include blood or plasma transfusions (which may not be available or practical with large numbers of livestock) or treatment with vitamin K1. Vitamin K1 therapy requires a minimum of 12 to 24 hours to be effective, therefore severely affected animals may not survive this time period.

PREVENTION: Toxicity can be prevented by properly curing sweet clover hay, especially by allowing the stems to dry before baling. Extra caution must be taken with second year growth, since it contains a higher concentration of toxin. Affected hay may remain toxic for years. Moldy sweet clover hay should not be fed, but if it has to be fed, alternating with a non-moldy quality hay every few days will minimize clinical signs. Moldy sweet clover hay should be withheld for 4 to 6 weeks prior to anticipated parturition ("giving birth") or surgical procedure, to lessen the chances of these animals bleeding to death. Discard milk from affected animals to prevent signs developing in nursing animals.

17. TOBACCO

Nicotiana spp.

(nightshade family)

TOXICITY RATING: Low to moderate, depending on the situation.

ANIMALS AFFECTED: All animals may be affected, but pets in contact with tobacco products are at risk as are pigs allowed to forage on harvested tobacco fields.

DANGEROUS PARTS OF PLANT: Leaves.

CLASS OF SIGNS: Gastrointestinal irritation, trembling, staggering, weakness, breathing problems, heart problems, collapse, birth defects, death.

PLANT DESCRIPTION: These tall annual plants grow from fibrous roots and produce large, hairy leaves and terminal clusters of tubular, 2 inches long, white, red, lavender, or yellow flowers on short stalks. Many-seeded capsules may appear in late summer. The tobacco species with colorful flowers (fig. 17) are grown as garden ornamentals. Those with yellow-green flowers and the largest leaves are an economic crop in the southern counties of the state.

SIGNS: The toxin in tobacco is nicotine, an alkaloid with an irritating effect on the stomach and intestines and also the nervous system. This toxin is related to the toxins in poison hemlock and lupine. Concerning nicotine concentrations, an average cigarette can contain between 20 and 30 mg, and 120 mg for a cigar. One report indicates that for a human unaccustomed to tobacco, 4 mg can cause clinical signs, and 60 mg at one time can cause death.

Pets can easily be poisoned by tobacco products in the home, either accidentally or maliciously. The primary route of poisoning is by ingestion (eating tobacco products or drinking tobaccotainted water), but inhalation of smoke is also possible. Clinical signs nearly always are present, but only rarely is a lethal dose ingested. The initial signs of poisoning can develop within 10 to 15 minutes or may not manifest for several hours. At first, the irritating effect that tobacco has on the stomach and intestines will cause salivating, vomiting (if capable), and diarrhea. Shortly after the digestive signs develop, neurologic signs appear. Initially, nicotine stimulates the nervous system, with depression of the nervous system occurring at a later time. Early signs include nervousness, shaking, trembling, a stiff and uncoordinated gait, weakness and collapse. Cardiac abnormalities may be noted as well as breathing difficulties, to the point of respiratory paralysis (the cause of death in lethal cases). Tobacco is also teratogenic, causing birth defects if the mother animal was pregnant when the toxicosis occurred.

In large animals, pigs are the most likely to suffer tobacco poisoning, since they are allowed to forage on harvested tobacco fields. In addition to the gastric and neurologic signs, birth defects have been well documented when pregnant sows ingest tobacco. The most common defects are limb deformities in the piglets.

FIRST AID: For pigs and other large animals, it is unlikely that a lethal dose would be consumed. Keep the animal quiet and undisturbed and clinical signs should resolve within a few hours, although birth defects may still occur. For more severely affected large animals or in cases where it is known that a large quantity was ingested, call a veterinarian as soon as possible, since emergency evacuation of the gastrointestinal tract may be necessary. For pets, contact a veterinarian if any ingestion has occurred. With their smaller size, pets will show clinical signs more rapidly and more severely than large animals. In addition, the effects of nicotine can come on rapidly.

SAFETY IN PREPARED FEEDS: Tobacco remains toxic when dried, therefore processed feeds containing tobacco are not safe for consumption.

PREVENTION: Do not allow pets to be in contact with tobacco or tobacco products. Do not let animals drink from puddles or cups that have leached tobacco juice in them (such as when a water-filled cup has been used as an "ashtray", or spittoons), since this water can have extremely high concentrations of nicotine. Forced ingestion or inhalation is inhumane and potentially lethal. Concerning sows, there is disagreement over whether there exists a "danger window" for consumption of tobacco during gestation (currently there exists two thoughts, that on days 10 to 30 of gestation the piglets are susceptible, and another that indicates that anything after 30 days is dangerous). Until more clarity is brought to bear on the issue, do not let pregnant sows graze on tobacco.

18. ALSIKE CLOVER

Trifolium hybridum

(pea family)

TOXICITY RATING: Low.

ANIMALS AFFECTED: All grazing animal may be affected.

DANGEROUS PARTS OF PLANT: All green parts (when dewy).

CLASS OF SIGNS: Gastrointestinal irritation, photodermatitis (sunburn or sunscald).

PLANT DESCRIPTION: These perennial legumes (fig. 18) are commonly grown for pasture or hay and may be found as escapes in fields, roadsides, and waste areas. They have the familiar three-parted clover leaf. The flowers are axillary, not terminal as in red clover, and are pink to white in a clover head.

SIGNS: This is not a commonly reported toxicity, and is usually not serious even if toxicity occurs. It is unknown if the wet clover causes problems by contact or ingestion. The typical signs associated with alsike clover are gastrointestinal distress, including mild colic and diarrhea. Photodermatitis ("sunburn") is also possible, especially on the parts of the body that contact the wet grass (lower legs, mouth). Liver damage has been suggested, but not well-verified. This syndrome, which can be caused by plants in addition to alsike, is sometimes called "dew poisoning" or "trifoliosis".

In rare cases, the sunburn may spread to the entire body, especially in lightly pigmented areas. Newly shorn sheep may be particularly at risk. Large amounts of alsike must be consumed before serious body-wide sunscald develops.

FIRST AID: Remove the animals from the pastures especially in the early morning when the plants are dew-covered. Animals severely affected by sunscald need to be kept out of the sun until recovered (turn them out at night). Care for gastrointestinal and sunburned areas symptomatically. Call a veterinarian if signs are severe or if the animal does not recover in a day or two.

SAFETY IN PREPARED FEEDS: Alsike clover is safe when dry, therefore prepared feeds containing alsike is safe for consumption.

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

NOTE: Red clover (Trifolium pratense, pea family), buckwheat (Fagopyrum esculentum, dock family), and alfalfa (Medicago sativa, pea family) can sometimes cause similar poisoning. However, white clover (Trifolium repens, pea family) poisoning, when it occurs, causes cyanotic or estrogenic (hormone) symptoms, especially in swine. All of these plants sometimes cause bloating, especially when the animals are put out in lush growth that they are not accustomed to eating.

[[SECTION: Woodland Plants]]

19. JACK-IN-THE-PULPIT, INDIAN TURNIP

Arisaema triphyllum

(aroid family)

TOXICITY RATING: Low.

ANIMALS AFFECTED: All animals may be affected.

DANGEROUS PARTS OF PLANT: Bulbs, stems, possibly leaves.

CLASS OF SIGNS: Oral and gastric irritation, mouth and throat swelling on rare occasions may be severe enough to affect breathing.

PLANT DESCRIPTION: These herbaceous perennials (fig. 19) pop up in spring in Indiana woodlands. They grow 1 to 2 feet tall from a tuberous root. The large leaves are three-parted, smooth-margined, and net-veined. Each plant produces one bloom beneath the leaves on a short stalk. The "jack" is a fleshy green spike ("spadix") bearing a number of inconspicuous male and female flowers. The most noticeable

part of the bloom is the "pulpit", a modified leaf ("spathe") that wraps around and hides the spadix. It may be all green or striped with red or reddish-violet. In late summer the spathe falls away, revealing a cluster of bright red berries.

SIGNS and FIRST AID: See the section for the Aroid discussion. Rarely is enough of this plant consumed to cause a problem, but the potential exists, especially in spring when other forages are not readily available and if the livestock have access to a wooded area. Signs are self-limiting, and a veterinarian only needs to be contacted if signs do not resolve or if breathing is affected.

PREVENTION: Jack-in-the-Pulpit grows in wooded, shaded areas in the spring, so limit animal access to these areas when plants begin to emerge.

20. DWARF LARKSPUR, STAGGERWEED, POISON WEED

Delphinium tricorne CULTIVATED LARKSPUR *Delphinium ajacis* (buttercup family) TOXICITY RATING: Moderate for Indiana. These plants are a more serious threat in the western ranges.

ANIMALS AFFECTED: Cattle are the primary animals affected; toxin can also affect other ruminants as well as horses.

DANGEROUS PARTS OF PLANT: All parts, especially seeds and young leaves.

CLASS OF SIGNS: Nervousness, incoordination, staggering, salivating, bloating, abnormal heart beat, breathing difficulty, paralysis, convulsions, death.

PLANT DESCRIPTION: These short annual or perennial herbs (fig. 20), 1/2 to 4 feet high, bear alternate, deeply-lobed ("crowfoot") leaves and elongate clusters of spurred white, blue, or purple flowers in the spring. Roots grow in tuberous clusters. This weed commonly grows in rich open woods, along

streams, in old fields, along roadsides, and on sand hills. Wild larkspur is most common in the southern two-thirds of the state, but the equally poisonous cultivated larkspur may be found in gardens statewide.

SIGNS: Larkspur is primarily a problem in western ranges, especially with cattle. Apparently, the plant is palatable, which increases the risk of clinical toxicosis. The toxicity of larkspur varies, with the highest periods of toxicity occurring during early growth, and when the plant goes to seed. The toxin is a mixture of alkaloids, including ajacine and delphinine, and blocks communication between nerves and muscles (like curare). Signs appear within a few hours of ingestion. Approximately 1/4 pound of larkspur per 100 pounds body weight may be lethal for cattle. A higher dose is needed before sheep and horses show clinical signs.

FIRST AID: There is no antidote for larkspur poisoning, and treatment is supportive. A veterinarian needs to be called if the animals are bloated, or if consumption was very recent (the veterinarian may be able to empty the stomach or rumen). Affected animals should not be handled any more than necessary, since stress will worsen the signs.

SAFETY IN PREPARED FEEDS: Larkspur remains toxic when dry, therefore hay and other prepared feeds will be toxic.

PREVENTION: Do not let animals (especially cattle) graze larkspur, particularly during the early growth and seed stages. Keeping pastures mowed will greatly reduce the number of larkspur plants.

21. DUTCHMAN'S BREECHES, STAGGERWEED

Dicentra cucullaria

SQUIRRELCORN, STAGGERWEED

Dicentra canadensis

CULTIVATED BLEEDING HEART

Dicentra spectabilis

(poppy family)

TOXICITY RATING: Low. The plant is unpalatable and is not eaten when better forages are available. Also, larger quantities need to be consumed to show clinical signs, and only rarely is consumption of *Dicentra* lethal.

ANIMALS AFFECTED: Cattle are primarily affected, horses and sheep also appear susceptible. Due to the nature of the toxic component (morphine-like derivatives), all species are likely to be susceptible at sufficient dosages.

DANGEROUS PARTS OF PLANT: Leaves, stems, roots.

CLASS OF SIGNS: Salivating, breathing difficulty, abdominal pain, collapse, convulsions, neurologic signs (running with head held up).

PLANT DESCRIPTION: These delicate perennials with finely-cut, fern-like leaves bear 1 to 10 showy flowers on slender stalks. The 4 to 10 creamy white flowers of Dutchman's breeches (fig. 21A) have spurs like bloomer legs. The white-flowered squirrel corn (fig. 21) and pink-flowered bleeding heart plants bear heart-shaped petals with teardrop-shaped appendages at the bottom. Small, yellow, pea-like tubers are scattered along the underground stem of squirrelcorn. The wild staggerweeds are common spring wildflowers in established woodlands.

SIGNS: This plant is one of the first to grow in the spring. It is considered to be unpalatable, but may be consumed if no other forages are available. The plant contains isoquinoline alkaloids, such as apomorphine, protoberberine, and protopine. Debate exists over whether the plants cause immediate signs, or if the plant needs to be consumed over several days before signs develop. Clinical signs begin with salivation and difficulty in breathing. Later, colic (abdominal pain) and possibly diarrhea may be noted. The animals may run with held up high, become uncoordinated, and may collapse and exhibit convulsions. Some have reported a loss of milk production in affected animals. Other disorders resembling *Dicentra* poisoning may also occur in the spring, making a diagnosis difficult. Death with *Dicentra* poisoning is rare, and animals tend to recover on their own in a few hours after they are removed from the plant. A plant called *Corydalis* (fitweed) will cause similar signs in animals (similar toxic component).

FIRST AID: Remove all animals from the plant and provide adequate safe forage. Signs tend to resolve in a few hours. For seriously affected animals, or those who are very young or ill, a veterinarian can provide basic emergency care and provide relief.

SAFETY IN PREPARED FEEDS: Precise information could not be located. Consider *Dicentra* unsafe in hay and other prepared feeds.

PREVENTION: Provide adequate feed for pastured animals, especially in the early spring. Animals will avoid *Dicentra* if better feed is available. If animals continue to eat this plant, consider fencing off the area where *Dicentra* grows to limit access.

22. WHITE SNAKEROOT, WHITE SANICLE, RICHWEED

Eupatorium rugosum

(daisy family)

TOXICITY RATING: High. White snakeroot will be eaten, especially in the late summer and fall, and is often lethal.

ANIMALS AFFECTED: Cattle, horse, goat, sheep, swine. All grazing animals can be affected by white snakeroot, and the toxin passes in the milk, so nursing animals and humans are also at risk.

DANGEROUS PARTS OF PLANT: Leaves and stems, possibly flowers. Roots seem to have a lower toxicity.

CLASS OF SIGNS: Trembling, sweating, depression, stiff gait, heart failure, jaundice, toxic milk, death (may be sudden).

PLANT DESCRIPTION: White snakeroot (fig. 22) grows from fibrous, matted roots as a smooth, erect, perennial herb 1 to 3 feet high with opposite, oval, pointed-tipped leaves with sharply-toothed edges. The upper surfaces of the leaves are dull, the lower surfaces shiny with three prominent main veins. Small white flowers in compound terminal clusters are conspicuous in late summer. White snakeroot is found in woods, damp and shady pastures, and occasionally in thickets and clearings (especially at the edges of wooded areas) in all 92 Indiana counties.

SIGNS: Clinical signs include: depression, stiff gait, periods of sweating, normal or subnormal body temperature, labored or shallow respiration, muscle tremors, trembling, partial throat paralysis, jaundice, passage of hard feces, prostration, death (death may be sudden with no prior signs). Onset of signs is typically 2 days to 3 weeks. Death occurs within 1 day to 3 weeks, with horses typically succumbing in 1 to 3 days. Even if the horse does not die from this toxin, it may suffer permanent heart damage and be unsuitable for work or pleasure purposes. The toxic component is tremetol, and the toxic dose of the green plant is approximately 1% to 10% of the body weight of the animal at one time or over several doses. The toxin is cumulative, so one large dose or multiple smaller doses over time can kill. The toxin is excreted in the milk, so lactating animals are slower to show signs of toxicity, but the nursing animals will then be affected by the toxin. Humans who drink raw milk from affected animals can also be poisoned, sometimes fatally (the disorder was called "milk sickness" in colonial times).

The primary danger occurs in late summer throughout the fall, especially in overgrazed pastures or where there is insufficient food. Poisonings in early winter where the animals eat stalks that extend above the snow have also been reported. The edges of woods or thickets are common locations for white snakeroot. Dry years are also associated with more reports of toxicity, perhaps due to inadequate pasture forage.

FIRST AID: Supportive care is required, since there is no specific antidote. Many affected animals will die or be permanently disabled. Remove all animals from the pasture or fence off the wooded areas, especially in the fall through winter. Continue to milk lactating animals, and discard milk. A veterinarian will be able to provide supportive care to animals showing signs, but death is likely once clinical signs develop.

SAFETY IN FEEDS: Drying decreases but does not eliminate the toxin, therefore hay with white snakeroot in it is unsafe for consumption.

PREVENTION: Learn to recognize and avoid white snakeroot. Do not allow animals to graze this plant under any circumstance. To do this, fence off wooded areas, provide supplemental feed (especially in the late fall and winter), or treat the snakeroot with herbicides. Be cautioned that treatment with herbicides may make the plant more palatable, so allow several weeks to pass between spraying and allowing animal access (be sure the plants are completely dead). The

problem may recur the following year, so plan ahead to avoid animal loss. Under no circumstances should raw milk from affected animals be used for animal or human consumption.

23. BRACKENFERN, BRAKE FERN

Pteridium aquilinum

(fern family)

TOXICITY RATING: Moderate.

ANIMALS AFFECTED: Ruminants (especially cattle), horses, sometimes swine. Any grazing animal is susceptible.

DANGEROUS PARTS OF PLANT: All parts, especially the roots.

CLASS OF SIGNS: In horses and swine: Weight loss, weakness, gait abnormalities, abnormal heart rate and/or rhythm, inability to rise, death.

In ruminants: Bleeding disorders (bruising, hemorrhaging, anemia), breathing difficulties, weight loss, death.

PLANT DESCRIPTION: The broad, triangular leaves (fronds) of this perennial fern rise 2-3 feet tall (sometimes to 4 feet) from a thick, brown or black, horizontal rootstock. Each frond divides into three main parts, and each of these is twice subdivided (fig. 23). The edges of the leaves usually turn under. Late in summer the lower edges of mature fronds bear powdery clusters of brown spores (fig. 23A). These ferns are common in open, acid woodlands, burned-over areas, and open pastures in dry, sandy, or gravelly soil. Stands of bracken may be so dense that they crowd out all other plants. Although brackenfern grows statewide, it is more common in the northern prairie and lake regions.

SIGNS: Toxic signs vary between ruminants (cattle, sheep and goats) and monogastric animals (like horses and swine):

Horses: The toxin in brackenfern is thiaminase, an enzyme that destroys thiamine (vitamin B1). The horse then essentially suffers from a vitamin deficiency of thiamine, which causes myelin degeneration of peripheral nerves (a loss of the fatty insulation layer to nerves that primarily control muscles). Poisoning can occur at any time of year, but is more likely in the late summer when other forages are scarce and the level of thiaminase is at its peak. Bracken is not considered palatable, but horses will eat it if no other forage is available, or they will consume it in hay or bedding, where it remains toxic. Some horses are believed to acquire a taste for it, and these horses will consume it even if other forages are available.

Horses need to consume bracken for one to two months prior to manifesting clinical signs. After this time horses may then be fed bracken-free forage and yet still develop clinical signs within 2 to 3 weeks. The first signs in horses is weight loss after a few days on bracken. Later, weakness and gait abnormalities are present, which progress to staggering, hence "bracken staggers". Affected horses may stand with their legs widely placed and their back arched. Muscle tremors and weakness is apparent when the horses are forced to move. Early in the course of the syndrome, a slow heart rate and abnormalities of the heart rhythm may be noted. Near the end of the clinical course, the heart rate and temperature rise, and the animals cannot get up and may have spasms and an upward arching of the head and neck. The syndrome runs its course, with death occurring within 2 to 10 days of the onset of signs, but it can be treated. Swine would show signs similar to those in horses.

Ruminants, especially cattle: Thiaminase does not adversely affect ruminants since the ruminal bacteria degrade the enzyme. However, other toxins in bracken affect ruminants, most notably ptaquiloside, a lactone toxin that affects the bone marrow. The toxin is present in all parts of the plant, but is concentrated in the rhizomes, and is toxic in fresh as well as dried plants. Consumption of bracken results in the depression of bone marrow (and thus red and white blood cell and platelet production), and the plant has a direct or indirect anti-coagulant property. Cattle show signs after grazing bracken for 1 to 2 months, although death may occur within this time frame as well. Affected cattle have an increased temperature, weight loss, and exhibit increased bruising and bleeding. From the excessive bleeding, cattle are anemic, and can die within a week of showing signs. Young cattle may develop swelling in the larynx and have difficulty breathing. Sheep may be poisoned in a similar manner, but are apparently more reluctant to consume bracken.

The plant is also reported to contain carcinogenic substances, but instances of cancer in animals resulting from bracken fern ingestion is not well reported.

FIRST AID: Horses: If horses are observed eating bracken, immediately remove them from the pasture, or in some way prevent access to the plant. Hay with bracken in it should never be fed. If large amounts were consumed, and especially if clinical signs are present, call a veterinarian immediately. The antidote is daily injections of thiamine for up to two weeks. Do not wait until the animal cannot rise, by then it may be too late. Provide similar first aid to swine.

For ruminants: Immediately remove cattle from bracken pastures, or fence off the bracken areas to limit access. Do not feed hay or bed animals on straw that contains bracken. A veterinarian can assist with treatment of affected animals, but this may be cost prohibitive. Evacuation of the rumen and intestinal tract is usually not of value, since the poison accumulates in the system for many days, and there may be little or no toxin remaining in the digestive tract to be removed once clinical signs appear. Treatment is concerned with alleviating the clinical signs and providing supportive care. Blood transfusions may be attempted, but the prognosis is poor for clinically affected animals.

SAFETY IN PREPARED FEEDS: Bracken remains toxic when dry, and is never safe for consumption.

PREVENTION: Grazing animals should not be allowed access to bracken fern, especially if they have developed a taste for it. Provide supplemental feed if the pasture is low in adequate forage. Never use hay or bedding material that contains bracken.

NOTE: Sensitive fern (*Onoclea sensibilis*, fern family), may also be poisonous. Horses reportedly have become nervous and uncoordinated after eating this common fern of marshy areas.

24. GROUNDSEL, RAGWORT

Senecio spp.

(daisy family)

TOXICITY RATING: High. Senecio is very toxic both fresh and in hay, affected animals often die.

ANIMALS AFFECTED: All grazing animals may be affected, but horses and cattle are particularly susceptible. Young, growing animals are more susceptible than mature animals, and fetuses may be affected in utero.

DANGEROUS PARTS OF PLANT: All above ground parts, with higher concentrations in the seeds.

CLASS OF SIGNS: "Poor doer", weight loss, unthriftiness, poor hair coat, anorexia, behavioral changes, sunscald, liver failure, jaundice, death.

PLANT DESCRIPTION: There are several species of ragworts. In Indiana they are perennial herbs about 1 foot tall (rarely to 3 feet tall). Basal leaves are spoon-shaped and stem leaves are alternate and pinnately cut into narrow segments. At the top of the plant are clusters of yellow composite (daisy-like) flowers with yellow ray petals. Most ragworts grow along roadsides, in pastures, and in wet or waste areas. *Senecio aureus* (golden ragwort) blooms in early spring in woodlands or meadows statewide (fig. 24).

SIGNS: Poisoning from ragwort may be chronic or acute, with the chronic form more commonly encountered. The disease is also called "seneciosis" or "pictou" disease. The toxic principle is a mixture of pyrrolizidine alkaloids which adversely affect the liver. The plant grows in late winter through the spring and tends to be incorporated in first cutting hays. Animals may not show signs until the fall or winter after eating this hay for some time. In horses especially, the clinical signs may develop months after ragwort hay is no longer being fed, making an accurate diagnosis difficult.

In the rare acute toxic case (20 or more pounds consumed in a short period of time for a horse), the animals may become very excited and violent, with gastrointestinal signs (colic, diarrhea), dilated pupils and increased heart rate, and sudden death. Chronic toxicity is far more likely to be encountered. The animals must consume the plants fresh or in hay for weeks to months before clinical signs appear. The toxin is cumulative, and continues to damage the liver until enough of the liver is affected to cause clinical signs. Approximately 50 to 150 pounds of ragwort needs to be consumed by a horse before signs appear. Cattle are more resistant to the toxic effects than horses and reports have indicated that cattle may be fed small amounts of ragwort hay without ill effects. There are reports that sheep are relatively resistant to this toxin.

In chronic cases, the first clinical signs are weight loss, "poor doer", unthriftiness, and a decreasing appetite. These and all clinical signs are reflective of the gradual loss of liver function. Later, nearly total anorexia, jaundice, behavioral changes (depression, drowsiness, confusion, aimless wandering, "sleepy staggers") and an uncoordinated gait may manifest. Some animals will head press, where the animals lower their head and press it and/or their neck against a sturdy object. Some animals have been noted to stumble over objects and to appear blind. Since the liver is responsible for degrading diet-derived plant pigments, these pigments are not destroyed, and enter into the circulation. The pigments react with light and can cause photosensitization and sunscald, especially to the more lightly pigmented areas. Near the end of the course of the disease, abdominal fluid may build up ("ascites"), and fluid may build up under the skin in the "lower" parts of the body (throat latch, under the abdomen, legs), and the animal may develop diarrhea. Loss of blood's ability to clot (another important liver function) may be present as well, with bruising and hemorrhaging noted (which may occur in the urinary tract and present as red to black colored urine). Just prior to death, the animal may collapse and go into convulsions. Once clinical signs become apparent, the chances that the animal will recover are very unlikely.

In cattle, other signs can be noted in addition to those listed above. The muzzle may become dry and scaly, and animals may strain to defecate, causing the rectum to prolapse. Affected animals will produce milk with an unusual odor. It is not known, but it is suspected, that this milk may be toxic.

FIRST AID: Once clinical signs are present, recovery is less likely. Newer and experimental treatments exist, but the prognosis is poor for survival. Remove all ragwort hay from animals and provide proper feed. A veterinarian may be able to provide some treatment or referral to an animal hospital for care of valuable animals, but even this treatment option has a low rate of success, especially for severely affected animals.

SAFETY IN PREPARED FEEDS: All plants that contain pyrrolizidine alkaloids remain toxic in hay, silage and other feeds. Horses should never be allowed to consume any feed containing ragwort. Ruminant (cattle primarily) may be able to tolerate small amounts of ragwort hay if it is fed intermittently and enough good forage is made available. The recommendation for cattle is that ragwort hay be fed at 10% or less of the body weight over the course of a year. This would amount to 50 pounds per year for a 500 pound animal. If at all possible, the ragwort hay should never be fed, even to cattle.

PREVENTION: Learn to recognize ragwort both fresh and in hay, and only deal with reputable feed dealers. Never feed ragwort to horses, and it is advisable not to feed it to cattle. If ragwort hay must be fed to cattle, follow the guidelines listed above.

NOTE: Plants that also may cause similar signs due to the presence of pyrrolizidine alkaloids: Stinking willie (*Senecio jacobea*), common groundsel (*S. vulgaris*), fiddleneck and tarweed (*Amsinckia* spp), rattlebox (*Crotalaria*), Hound's tongue (*Cynoglossum officinale*), heliotropum (*Heliotropium* spp) and salivation Jane (*Echium lycopsis*). Ragwort poisoning is a major problem in the western United States and may be increasing in the East. The greatest risk in Indiana seems to be in the southwestern counties where cressleaf groundsel (*Senecio glabellus*) grows. Another species, prairie ragwort (*Senecio plattensis*), occurs in the northern half of the state. Ragwort growing in alfalfa fields has been reported.

25. GREEN FALSEHELLEBORE, WHITE HELLEBORE, INDIAN POKE

Veratrum woodii

(lily family)

TOXICITY RATING: Moderate to high, depending on individual circumstance.

ANIMALS AFFECTED: Sheep are affected primarily, but chickens and cattle may also be at risk.

DANGEROUS PARTS OF PLANT: All parts, especially roots.

CLASS OF SIGNS: Gastrointestinal irritation, salivating, weakness, trembling, heart problems,

breathing difficulties, birth defects.

PLANT DESCRIPTION: These perennial herbaceous plants (fig. 25) have stout, erect, unbranched, 1-8 feet tall stems arising from short, thick rootstocks. There are clusters of large, broad, alternate leaves that to some people resemble garden cabbage or skunk cabbage. These leaves are parallel-veined and pleated like a skirt. Green to greenish-white, inconspicuous

flowers occur in large terminal clusters. *Veratrum woodii* grows in woods or on hillsides and bluffs, most commonly in the central and southern parts of the

state.

SIGNS: False hellebore can cause toxicity in grazing animals or more commonly, cause birth defects. Both of these syndromes are more common in sheep than in other species. It is possible that the toxins causing birth defects are not the same toxins that affect the grazing animals. Since toxicity of grazing animals is exceedingly rare and usually not lethal, this section will briefly cover the grazing animal toxicosis, and will focus on the teratogenic effects of false hellebore.

The toxic component in false hellebore is a mixture of alkaloids (primarily jervine, cyclopamine, and cycloposine). In grazing animals that consume a toxic dose, salivation, gastrointestinal irritation, weakness, incoordination, decreased heart rate, and breathing difficulties may be noted. Rarely, animals may convulse and die.

More important are the effects that false hellebore has on fetuses. The toxins are known teratogens, causing developmental problems with lambs in utero. Specifically, if a pregnant ewe eats false hellebore on the 14th day of gestation, the lamb may die or have severe developmental problems. The problems in the lamb affect mostly the brain, skull and face, and the lambs can be born with a "monkey-face", or with the eyes in the center of the face ("cyclops") or hydrocephalus, or failure of the head to develop. These lambs are usually born dead or tend to die shortly after birth. In some cases, the ewes gestation is prolonged and the lamb grows too large, necessitating assistance at delivery or a C-section. It is possible that only one of a pair of twin lambs will be affected.

In addition to the well researched aspects in lambs, false hellebore, when ingested at any time prior to the 32nd day of gestation can cause many birth defects and death of the fetuses.

FIRST AID: For grazing animals, treatment is symptomatic. Call a veterinarian if signs are prolonged or severe. Nearly all animals will recover once removed from the plants. For affected fetuses, there is little that can be done other than to assist the ewe in delivery, since some of the lambs are large and/or malformed.

SAFETY IN PREPARED FEEDS: False hellebore reportedly remains toxic when dry, therefore feeds containing this plant should not be fed, especially to sheep.

PREVENTION: False hellebore is a big problem in western ranges, but can affect animals in Indiana. The danger is particularly high with sheep. It would be best to keep all pregnant ewes away from false hellebore until after their 33rd day of gestation. In addition, plants are more toxic in the spring, and toxicity decreases through the growing season. The roots and rhizomes are considered to be more toxic (lethal), with the leaves containing more of the teratogenic (birth defect) compounds. Therefore, be cautious with animals who appear to be eating this plant, and attempt to limit all access to false hellebore as much as possible.

[[SECTION: Marsh and Streambank Plants]]

26. MILKWEEDS Asclepias spp. (milkweed family) TOXICITY RATING: Low to moderate. Milkweeds are unpalatable, and have variable toxicities. Death is not likely unless large quantities are consumed.

ANIMALS AFFECTED: All animals may be affected. Sheep are most at risk, but cattle, goats, horses, poultry, and pets are also at risk.

DANGEROUS PARTS OF PLANT: Stems, leaves, roots.

CLASS OF SIGNS: Gastrointestinal irritation (primarily vomiting and diarrhea), incoordination, tremors, heart problems, respiratory difficulty, death.

PLANT DESCRIPTION: Milkweeds, such as common milkweed, *Asclepias syriaca* (fig. 26), get their name from the thick, sticky, milky sap that oozes out of cut or torn leaves, stems, and fresh pods. The usually solitary stems of milkweed grow 1 to 5 feet tall and bear opposite (sometimes whorled), sometimes fleshy leaves with entire margins. Flowers emerge in umbrella-like clusters and range in color from pink to rose-purple to orange or white. The fruit (fig. 26A) is a pod with "tufted" seeds. A dozen species of milkweeds grow in Indiana woods and swamps, but most commonly in dry soils of fields and road-

sides. Dogbanes (*Apocynum* spp.), which are easily confused with milkweeds, are found in the same habitats and may cause similar poisoning.

SIGNS: There are several different types of milkweeds with varying degrees of toxicity, with the whorled milkweeds being the most toxic. Milkweed plants are considered unpalatable and are eaten only when other forages are not available, and may also be found in hay and processed feeds. The primary toxicants are cardiac glycosides that cause gastrointestinal, cardiac and respiratory problems and can cause death if enough is consumed. Resins (especially galitoxin) in the milky sap may also contribute to the toxicity of milkweed. In ruminants, the first signs are incoordination, muscle tremors and spasms, bloat, increased heart rate, breathing problems, and occasionally death. Horses are very reluctant to eat this plant, and its toxicity is only rarely reported: colic, diarrhea, abnormal heart rate and rhythm, rarely death. In animals that are capable of vomiting (pigs, dogs, cats, humans), this is the first sign to develop and is beneficial in that further absorption of the toxin is lessened. Horses cannot vomit, and vomiting is not generally observable in ruminants (if vomiting occurs, the contents still remain in the rumen), therefore toxic signs will be worse in these species.

FIRST AID: There is no antidote if an animal consumes milkweed. It is important to limit further ingestion of the plants or contaminated feed. If the animal recently consumed a large amount of the plant, a veterinarian should be called so that the gastrointestinal tract can be emptied and supportive care provided. Small tastes of the plants tend to result in minor oral irritation, and serve as a deterrent to further consumption, and these little nibbles typically do not require treatment.

SAFETY IN PREPARED FEEDS: Milkweed is toxic both fresh and dried, therefore hay, silage, green chop, and processed feeds that contain milkweed are never safe for consumption.

PREVENTION: Animals will avoid milkweed as long as there is sufficient forage available. Care must be taken to avoid incorporation of milkweed into prepared feeds and hay, and these feeds should be discarded.

27. SPOTTED WATER-HEMLOCK, WATER-HEMLOCK, SPOTTED COWBANE

Cicuta maculata

(parsnip family)

TOXICITY RATING: High. This is one of the most toxic plants in the U.S.

ANIMALS AFFECTED: All animals can be affected, but cattle are especially at risk.

DANGEROUS PARTS OF PLANT: The roots contain the highest concentration of toxin, but all parts are toxic.

CLASS OF SIGNS: Nervousness, breathing difficulties, muscle tremors, collapse, convulsions (seizures), death (which may be sudden).

PLANT DESCRIPTION: The perennial stem of water-hemlock (fig. 27) may grow to 7 feet from its cluster of 2 to 8 fleshy or tuberous roots. Stems are smooth, branching, swollen at the base, purple-striped or mottled, and hollow except for partitions at the junction of the root and stem (fig. 27A). A yellow, oily liquid smelling like parsnips exudes from cut stems and roots. Leaves are alternate, two to three times pinnately compound, and toothed, with the leaf veins extending to the leaf notches. Leaf petioles partially sheath the stems. The small white flowers are borne in flat-topped, umbrella-like clusters at the tips of stems and branches. Seed pods are small and dry with rounded, prominent ribs. Water-hemlock is found in swampy areas and marshes, wet meadows and pastures, and along streambanks and low roadsides.

SIGNS: This plant is considered to be one of the most toxic plants in the United States since so little of it needs to be consumed to cause death. Humans have been killed after only one or two bites of what they thought were "parsnips" (water hemlock root resembles a parsnip). Cattle are the primary species affected, hence the name "cowbane", especially in early spring when the plants are smaller and apparently more palatable and the roots are easily pulled up. Animals may also be poisoned if water hemlock is plowed under or if ground is reclaimed, since this may expose the root. Toxicity decreases through the growing season, and the toxicity of aboveground parts may be negligible when dry. The roots however are toxic at all times, even when dry. Animals have been poisoned by drinking water that had been contaminated with trampled water hemlock roots.

The toxin is cicutoxin, a yellow, viscous resin with a carrot-like odor, which affects the central nervous system. The toxic dose (the dose needed to cause clinical signs) and the lethal dose are nearly the same, with a little more than 1 gram of water hemlock per kilogram of body weight able to kill sheep, and 8 ounces (approximately 230 grams) will kill a horse.

Once the animal has ingested even a small amount of the plant, signs will develop within an hour, and as soon as 10 to 15 minutes. The syndrome is typically very violent. Stimulation of the central nervous system begins with nervousness, and dilated pupils. Later, muscle tremors occur, the animal has difficulty breathing, falls down and goes into convulsions. Death, from respiratory paralysis and terminal convulsions, is a typical outcome, occurring within 30 minutes of the onset of signs. If a sublethal dose is consumed, and the animal survives for 4 to 6 hours (or in one report, over 2 hours), the animal may recover, but may suffer from temporary or permanent damage to heart and/or skeletal muscle.

FIRST AID: If animals are seen eating water hemlock, especially the roots, get all animals away from the plant, and call a veterinarian immediately. Emergency measures (emptying the stomach or rumen and the use of medications to control seizures) may be tried, but death may still occur. The seizures cause severe damage to the heart and skeletal muscle, and this damage can be

avoided if the seizures are controlled. However, this is rarely possible under farm and field conditions since the toxin acts so quickly.

SAFETY IN PREPARED FEEDS: The above-ground parts of water hemlock decrease in toxicity over the growing season and lose additional toxin with drying. Therefore, the hay may not cause clinical toxicosis, but it is still advisable not to feed hay, or other prepared feeds, that contain water hemlock. The root is never safe, and remains toxic when dry, and should never be fed.

PREVENTION: Do not allow livestock (especially cattle) to graze in areas containing water hemlock. Prevent access to these areas or completely remove the plant (most importantly the roots) prior to introducing livestock, especially in the spring or when the roots may be exposed due to plowing, ditch maintenance, or other such activity. Never allow water hemlock to be incorporated into hay or other prepared feeds.

28. POISON HEMLOCK

Conium maculatum

(parsnip family)

TOXICITY RATINGS: Moderate to high.

ANIMALS AFFECTED: All animals may be affected. Grazing animals, swine and animals that may eat the seeds (especially poultry) are more at risk than pets.

DANGEROUS PARTS OF PLANT: All parts, especially young leaves and seeds.

CLASS OF SIGNS: Nervousness, trembling, incoordination, depression, coma, death, birth defects.

PLANT DESCRIPTION: This biennial herb (fig. 28) grows 3 to 8 feet tall and has a smooth purple-spotted stem and triangular, finely divided leaves with bases that sheather the stem. Fresh leaves and roots have a rank, disagreeable, parsnip-like odor. The small but attractive white flowers, arranged in umbrella-like clusters, open in early summer. The fruit is tiny, flattened, and ridged. Underground is a fleshy, unbranched white taproot (fig. 28A). Unlike wild carrot (*Daucus carota*, parsnip family), there are no hairs on the stems or leaves of poison-hemlock and no branching, feathery bracts beneath the flower clusters. These plants are commonly found along roadsides, edges of cultivated fields, railroad tracks, irrigation ditches, stream banks, and in waste areas.

SIGNS: The toxic components include the volatile alkaloids coniine and gamma-conicine. A lethal dose for a horse is 4 to 5 pounds of leaves, cattle may be poisoned with 1 to 2 pounds, and sheep with a half pound or less . Humans are often poisoned, mistaking the roots for parsnips, the leaves for parsley, or the seeds for anise.

Affected animals show signs within 2 hours of eating the plant, and tend to become nervous, and will tremble and become uncoordinated. After the excitement phase, the animal becomes depressed. The heart and respiratory rates slow down, the legs, ears and other extremities become cold, colic and/or bloating may occur. Even at this stage, the animal may not die, but may remain like this for several hours to days, and then recover. In lethal cases, the animals tend to die within 5 to 10 hours after the onset of the clinical signs, typically from respiratory failure

(in which case the mucus membranes will appear blue). A mousy odor has been reported to emanate from affected animals.

The primary time of year for poison hemlock is spring, often when there is insufficient forage available. At this time, the plant may also be more palatable. The toxicity increases throughout the growing season, and the roots become toxic only later in the year. Once dried, the toxicity is considered to be reduced but not eliminated.

Poison hemlock can also cause birth defects in ruminants and swine, with cattle and swine more susceptible than sheep and goats. The most often reported birth defects are cleft palate and spinal abnormalities. The gestational ages that have been associated with birth defects are: for goats, days 30 through 60; for cattle, days 40 through 70; for pigs, days 30 through 60. The birth defects resemble those seen with lupine, with lupine being the more dangerous plant.

FIRST AID: If animals are seen ingesting poison hemlock, call a veterinarian immediately. Treatment consists of eliminating the toxin from the gastrointestinal tract and providing supportive care. If the animals become comatose but do not die, they will require intense nursing care until they recover.

SAFETY IN PREPARED FEEDS: Reportedly, poison hemlock has a decreased toxicity when dry, but hay that contains a large proportion of poison hemlock may still cause problems (besides, it would have a low nutritional value). Recommendations are to feed little or no hay that contains poison hemlock. Seeds may contaminate grain-type feeds, making these feeds unsafe for consumption.

PREVENTION: Most animals will avoid poison hemlock if other forages are available. Tiny amounts may be consumed with little problems resulting, but significant consumption, especially of the seeds, will produce clinical signs and possibly death. Eliminating the plant from the pasture is the best solution; treatment with herbicides may be tried. Be sure all plants are dead prior to re-introduction of animals, since treated plants may be more palatable. Poison hemlock may be difficult to eradicate.

29. FIELD HORSETAIL SCOURINGRUSH

Equisetum arvense Equisetum hyemale

(horsetail family)

TOXICITY RATING: High for horses, moderate for other species.

ANIMALS AFFECTED: Horses are the species most affected, cattle and sheep may be affected, but this occurs rarely.

DANGEROUS PARTS OF PLANT: All parts, both fresh and dried.

CLASS OF SIGNS: Weight loss, weakness, gait abnormalities, abnormal heart rate and/or rhythm, inability to rise, death.

PLANT DESCRIPTION: Two types of shoots 1 to 3 feet tall merge from horsetail's underground rootstock. Both types are round, hollow, stiff, and jointed. The stem sections easily pull apart. The first type of shoot (fig. 29A) is tan, appears early in spring, and ends in a terminal, cone-like structure. The later, green, sterile shoot (fig. 29A) bears whorls of pine-needle-like branches and looks like a horse's tail. Scouringrush sends up long, tapering, cane-like shoots 1-6 feet tall. These stiff, evergreen shoots terminate in spore-producing cones. Leaves are reduced to teeth-

like scales arranged in whorls around the joints of the stems (fig. 29B). The plants commonly grow on shaded, moist soil in meadows, along roadsides, in ditches and thickets, along stream banks, at the borders of swamps, and on railroad embankments.

SIGNS: The toxic signs associated with horsetail are essentially the same as for bracken fern, since the toxin is the same: thiaminase. Horsetail does not contain the bone marrow toxin found in brackenfern. See the section on bracken fern (horses) for more details.

FIRST AID: A veterinarian needs to be called to assist in the treatment of thiamine deficiency. For more discussion, see brackenfern.

SAFETY IN PREPARED FEEDS: Consumption of horsetail in feeds is the most likely route of poisoning, but fresh plants are also toxic (but considered unpalatable).

PREVENTION: Do not allow horsetail to be incorporated into feeds, especially if these feeds are intended for horses. Do not feed contaminated hay.

30. BUTTERCUPS

Ranunculus spp.

(buttercup family)

TOXICITY RATING: Low. Most animals avoid buttercups, and seldom ingest enough to cause any serious toxicity.

ANIMALS AFFECTED: All animals that chew on or ingest the plant can be affected.

DANGEROUS PARTS OF PLANT: Fresh leaves and stems.

CLASS OF SIGNS: Oral and gastrointestinal irritation are the primary signs (oral irritation, salivation, abdominal pain, diarrhea which may be bloody).

PLANT DESCRIPTION: Buttercups arise from fibrous roots, thickened rootstocks, or bulbs to form a rosette of basal leaves and often a low stem with alternate and divided (three-parted) leaves. The axillary, solitary flowers have five green sepals, five glossy yellow petals, and numerous reproductive parts and seeds. Buttercups usually are found in moist woods, meadows, fields, pastures, and sometimes along roadsides and in drier sites. Bristly buttercup (*Ranunculus hispidus*, fig. 30A) is typical of the wet site species; celeryleaf buttercup (cursed crowfoot, *Ranunculus sceleratus*, fig. 30B) is typical of the small-flowered, dry-site species.

SIGNS: The toxin in buttercup is protoanemonin, a volatile yellow oil, which causes intense oral irritation and gastrointestinal irritation. Problems in livestock tend to occur most often in the spring, herbivorous pets may be poisoned at any time if they have access to the plant. The plant is not palatable, and causes almost immediate oral irritation, so animals tend to avoid it. The toxicity of buttercup varies greatly among the different species and during the course of the growing season. Seldom is buttercup reported as a significant threat to animals. In experimental feeding trials with greater quantities of buttercup, prostration, coma and death have been reported, but these signs are rarely reported under field conditions.

FIRST AID: No treatment is necessary unless severe gastrointestinal signs are present (colic, bloody diarrhea) or if a large quantity was observed to be eaten. Minor oral irritation will resolve on its own.

SAFETY IN PREPARED FEEDS: Reportedly, the toxin is volatilized ("evaporates off"), so processed feeds and hay may be safe for consumption. However, consumption of any significant quantity of contaminated feeds is not recommended.

PREVENTION: Buttercup is a common pasture contaminant, and tends to not cause problems as long as there is sufficient quantities of good forage available. Eradication is not usually necessary, since animals tend to avoid this plant on their own. If it has become a problem, mowing or eradication are recommended.

NOTE: Marsh marigold (*Caltha palustris*), a closely related plant found in similar habitats, also contains protoanemonin and causes mouth and stomach irritation as well as generalized distress when cattle, sheep, or horses eat the fresh tops. Hay has been considered safe for consumption. First aid is the same as for buttercups.

31. STINGING NETTLE WOOD (BULL) NETTLE

Urtica dioica Laportea canadensis

(nettle family)

TOXICITY RATING: Low. Local irritation is the most common sign which shortly resolves on its own.

ANIMALS AFFECTED: Any animal that brushes against or consumes the plant can be affected. Short-haired hunting dogs and other dogs that run through the underbrush are more likely to encounter this plant.

DANGEROUS PARTS OF PLANT: Stems, leaves.

CLASS OF SIGNS: Facial, skin and oral irritation, salivation, pawing at the mouth, possible ocular irritation.

PLANT DESCRIPTION: These herbaceous perennials are common on moist ground in flood plains, woodlands, and along stream and river banks. They often occur in colonies so large that they are the only herbaceous plant present. The tough unbranched stems grow 2 to 5 feet tall from fibrous roots and are covered with stinging bristles. The leaves are opposite, thin, egg-shaped, toothed, and tapered at the tip. They measure 2 to 6 inches by 1 to 2 inches in stinging nettle and 3 to 8 inches by 3 to 5 inches in wood nettle. The 3 to 5 main veins from the base make the leaf (especially in wood nettle) strongly resemble the leaves of white snakeroot. However, the stinging hairs on the lower surface of the leaves prove the plant's identity. The tiny, green or greenish-white flowers droop in axillary clusters in stinging nettle (fig. 31) and stand upright in branching clusters at the top of the stem in wood nettles.

SIGNS: The small, hollow hairs in stinging nettle contain several irritating substances such as histamine (the mediator of some allergic reactions), serotonin, acetylcholine and formic acid (ants contain a high concentration of formic acid). These substances, coupled with the hairs ability to scratch the skin and mucus membranes, results in almost immediate burning, itching and irritation. Typically, signs are present for a few minutes to a few hours, and resolve on their own. If oral contact was made, the animal may shake its head, salivate, and rub its mouth. Skin irritation is possible, especially with short-coated dogs, and ocular (eye) irritation is also possible. It is possible that the animals attempts to comfort itself and relieve the irritation may cause more damage than the plant itself. On very rare occasions (and only after significant amounts were

consumed or contacted) will more severe systemic signs manifest (trembling, weakness, disturbances in heart rate). Nearly all animals (including humans) learn to stay away from nettle.

FIRST AID: Limit further exposure as much as possible. Remove affected animals from the areas where the plants are located, and monitor the animals so they don't self-traumatize. Recovery should occur within a few minutes to hours. If animals continue to have difficulty, or if more serious signs develop, or at any time if the eyes are affected, contact a veterinarian.

SAFETY IN PREPARED FEEDS: There is little information on the safety of nettle in feeds. However, due to its irritant potential, inclusion of this plant into hay or feeds is not recommended.

PREVENTION: Use caution in marshy or wooded areas where dense stands of nettle may be located. Prevent animals from contacting the plants as much as possible.

32. COMMON COCKLEBUR

Xanthium strumarium

(daisy family)

TOXICITY RATING: High.

ANIMALS AFFECTED: All animals may be affected. Cattle, swine, sheep, and poultry are more at risk than horses and pets.

DANGEROUS PARTS OF PLANT: The seeds and seedlings contain the highest quantity of toxin, yet the whole plant can be considered toxic. The seed burs can cause mechanical damage.

CLASS OF SIGNS: Gastrointestinal irritation, weakness, breathing difficulty, behavioral changes, cardiac abnormalities, death.

PLANT DESCRIPTION: The angled, sometimes red- or black-spotted stems of cocklebur grow 1 to 3 feet high. Leaves of this many-branched annual are alternate, hairy, rough-textured (sandpaper-like), somewhat heart-shaped, toothed, and lobed (fig. 32). Flowers are inconspicuous with male flowers in terminal spikes, female flowers in clusters in the leaf axils. The fruit is a hard, oval, prickly bur about 3/4 inch long containing two seeds (fig. 32A). Because seeds germinate best after being soaked in water, the plants are usually found along the shores of ponds where water has receded. The edges of farm ponds may be lush with young cockleburs. Seedlings have small strap-shaped leaves 1/4 inch wide by 11/4 inches long (fig. 32B). They also pop up in gardens, fields, roadsides, and other areas of nearly full sunlight.

SIGNS: There are several types of toxins in cocklebur, one being a glycoside, carboxyatractyloside, which causes hypoglycemia (low blood sugar) and a group of sesquiterpene lactones which can cause vomiting (in those species capable of vomiting), weakness, tremors, weak pulse, a loss of appetite and convulsions. Liver damage may also result from these toxins, and death is likely if a sufficient dose was consumed (ingestion of green plant at approximately 0.75% of body weight). The seed bur can cause mechanical damage (please refer to the section on Foxtail barley). The most common time for toxic signs is late spring and early summer for the seedlings, later in the summer for the burs, and at any time seedcontaminated prepared feeds are ingested. As the cocklebur plant matures, the toxicity decreases (except for the seeds). The seedlings are extremely dangerous, and typically sprout in wet areas, such as alongside streams, at the edges of ponds, in receding floodplains, etc. Animals will tend to avoid cocklebur, even the seedlings, as long as there is sufficient forage available. Species that are less discriminant eaters (cattle, swine) are particularly at risk where cocklebur grows. Once toxic signs develop, death may follow within 48 hours. Some animals may recover, but this may take some time (weeks).

Other conditions of toxicity can occur when seeds are incorporated into prepared feeds. Feeds that may become contaminated include hay (especially if cut later in the growing season when cocklebur has gone to seed), silage, or grains (corn, beans). If the feeds (especially the grains) are highly processed, it may be difficult to arrive at an accurate diagnosis of cocklebur toxicosis. The seed is highly toxic, and any species may become poisoned upon consumption.

FIRST AID: If animals are observed eating cocklebur, contact a veterinarian immediately. In the meantime, prevent further consumption of the plant by all animals. Keep animals quiet until the veterinarian arrives.

SAFETY IN PREPARED FEEDS: The seedling, when dried, retains its toxicity, therefore feeds with dried seedlings are not safe for consumption. Mature plants are less toxic, but consumption is still not advised. Feeds that contain burs and/or seeds are a mechanical threat as well as a toxic one, therefore these feeds are also not safe for consumption at any dosage.

PREVENTION: Pasture management is essential to prevent poisonings by cocklebur. Mature, seed-bearing plants should be removed from the pasture to prevent seeding and germination. This is particularly important if the pasture contains any wet areas that are subject to seasonal drying. If removal of the plants is impractical, fence off areas where seedlings are likely to germinate and/or provide for supplemental feed to decrease interest in the seedlings. For prepared feeds, exert extra caution when harvesting for hay, silage, or grains and do not allow any cocklebur to be included. Buy prepared feeds only from reputable dealers.

[[SECTION: Plants of Fields, Roadsides, and Open Areas]]

33. REDROOT PIGWEED

Amaranthus retroflexus

(pigweed family)

TOXICITY RATING: High. The plant is quite common and very toxic.

ANIMALS AFFECTED: Cattle and swine are the animals most likely to be affected; goats and sheep can also be poisoned.

DANGEROUS PARTS OF PLANT: Leaves, stems, roots.

CLASS OF SIGNS: Breathing problems, trembling, weakness, abortions, coma, death.

PLANT DESCRIPTION: Redroot pigweed (fig. 33) is a large (to 5 feet tall), coarse, annual with red stems and simple, egg-shaped, wavy-margined, alternate leaves. The green, inconspicuous flowers are borne in short, compact clusters along with green spines. Seeds are small, shiny, and black. Fields, barnyards, and waste areas are the favorite habitats of this weed.

SIGNS: Pigweed contains a nephrotoxin that causes kidney failure, and also contains soluble oxalates and is capable of accumulating nitrates. Therefore, toxicity can be due to any combination of these toxicoses.

Animals need to consume pigweed in fairly significant quantities over several days before signs appear. Typically, onset of signs is 3 to 7 days from the onset of ingestion. Animals will usually avoid pigweed if there are better forages available. Common incidences of poisonings have occurred when swine have been raised in confinement and are then turned out into a pigweed-infested pasture in the late summer to early fall. Under these circumstances, the swine consume large amounts of the plant quickly, with 5-90% of the animals becoming affected, with 75% or greater mortality among the affected animals. Modern management practices have largely eliminated this type of poisoning, but it can still occur. In cattle, pigweed toxicosis resembles oak toxicosis.

In affected animals, early signs include weakness, trembling and incoordination. This progresses to an inability to stand and paralysis, yet the animals may still be alert and able to eat. Near the end of the clinical course, the affected animals may go into a coma, and have edema under the skin of the abdomen and the legs, have a bloated abdomen, and die. The course of the disease is approximately 48 hours and is primarily consistent with kidney failure. Cases where animals consume smaller amounts of plants over long time periods have not been well studied, but this is also believed to cause toxicology problems.

Treatment with herbicides may render pigweed even more palatable, therefore make sure all treated plants are dead prior to introducing animals.

FIRST AID: If pigweed is being rapidly consumed, limit further access and ingestion of the plants. A veterinarian will be able to provide supportive care for the different toxicants contained in pigweed, but the animals may still succumb to the nitrates, soluble oxalates or the kidney toxin.

SAFETY IN PREPARED FEEDS: Pigweed is not safe in hay or other prepared feeds.

PREVENTION: To prevent pigweed poisoning, do not allow animals to have access to affected pastures, especially if the animals are hungry. Spray or mow plants down, making sure they are dead before animals are on pasture. Provide for supplemental feed if pasture quality is poor, since well-fed animals are less likely to consume pigweed.

34. MUSTARD FAMILY

Brassica (wild mustard)

Thlaspi (pennycress)

Lepidium (peppergrass), etc.

TOXICITY RATING: Low to moderate.

ANIMALS AFFECTED: Cattle, horses, sheep, poultry.

DANGEROUS PARTS OF PLANT: All parts, especially seeds.

CLASS OF SIGNS: Oral and gastrointestinal irritation, photosensitivity, breathing difficulty.

PLANT DESCRIPTION: Mustard family members have a pungent, sulfurous odor or taste. They may be annual, perennial, or biennial, with a basal cluster of leaves and alternate leaves on the stem that are usually smaller and shorter-stalked than the basal leaves. Flowers of most mustard species are yellow, but some are white, blue, or purple, and all have four petals in a cross-like arrangement (fig. 34A). The seedpod (silique) is dry and may be broad and flat (fig. 34B) or skinny and much longer than it is wide (fig. 34C). The mustard family includes weeds such as

yellow rocket, black mustard (fig. 34), tansy mustard, peppergrass, and pennycress. These are found in fields, pastures, lawns, roadsides, waste areas, and sometimes in woods. Cultivated mustards, which may be harmful if eaten in large quantities, include cabbage, rape, broccoli, turnip, rutabaga, horseradish, and radish.

SIGNS: Mustard plants are capable of causing several types of problems. The most common is oral and gastrointestinal irritation primarily the result of the isoallyl thiocyanates and irritant oils. The plants are not palatable and tend to be avoided unless there is little else to eat or if the seeds have been incorporated into processed feeds. Clinical signs could include oral irritation, head shaking, salivating, colic, abdominal pain, vomiting (in those species capable of vomiting), and possibly diarrhea. Swine and younger animals (lambs and calves) appear to be more susceptible to the irritant effects of mustard seeds in processed feeds. In order to cause toxicity, fairly large amounts need to be consumed over a period of time.

Mustard plants can cause other problems, although these are not reported as often. Photosensitization has been reported in cattle after rape (*B. napus*) was consumed. There are also reports of abortions, goiter and blindness due to mustard consumption, but these are not major problems. Mustard plants may accumulate nitrates under certain fertilization conditions, and thus cause nitrate toxicosis.

FIRST AID: There is no antidote for mustard poisoning. Supportive care and providing adequate quality forage is necessary. For more serious signs, or if animals do not recover when mustard feed is withdrawn, consult a veterinarian.

SAFETY IN PREPARED FEEDS: Mustard plants retain their toxic components upon drying. The seeds in particular contain a high concentration of toxins, and may be incorporated into grain mixes or hay. Care must be taken to insure that these plants and seeds do not contaminate feeds, since they are toxic in any quantity. If small amounts have been incorporated into hay, animals are likely to voluntarily avoid the plants. However, if the hay is highly contaminated and there is no other forage available, the animals will eat the mustard out of necessity and suffer toxic signs as a result. Therefore, avoid feeding mustard, but if this is not entirely possible, provide for additional and more nutritious forage.

PREVENTION: Do not feed hay or other feeds that contain mustard plants or seeds. Do not let animals onto pastures that are overgrown with mustard plants without providing adequate edible forage. Use caution when feeding animals (especially swine) crop harvest leftovers from plants in the mustard family.

35. HEMP, MARIJUANA, HASHISH, HASH

Cannabis sativa

(nettle family)

TOXICITY RATING: Low. Animals tend to avoid this plant, and toxic encounters are rarely fatal.

ANIMALS AFFECTED: All animals, pets as well as horses and livestock, may be affected.

DANGEROUS PARTS OF PLANT: Leaves, stems, flowers, seeds.

CLASS OF SIGNS: Behavioral changes, trembling, incoordination, gastrointestinal signs, sometime breathing difficulty.

PLANT DESCRIPTION: This coarse, roughly hairy, herbaceous annual, at least 3 to 6 feet tall, has opposite leaves on the lower part of the plant, alternate leaves above (fig. 35). The leaves are made up of from 3 to 7 coarsely-saw-toothed, rough-to-the-touch, long, narrow leaflets borne in a finger-like arrangement. Male and female flowers are found on separate plants, the male flowers on branch tips and the female along the length of the branches. Hemp may be found in weedy pastures, fence rows, brushy stream banks, and illicit plantings.

SIGNS: Nearly all animals will voluntarily avoid marijuana. Toxic cases are usually encountered when the plant is mixed in prepared feeds or bedding materials, or if the animal is forced into consuming or inhaling smoke from the plant. Signs can include nervousness and disorientation which can progress to depression, trembling, vomiting (in those species that can vomit), and sometimes diarrhea and breathing difficulties. Fatalities are rare. Animals more likely to be poisoned are pets (especially dogs and birds), and perhaps swine, but all species are susceptible.

The primary toxic component is tetrahydrocannabinol, a resin. In addition, alkaloids and glycosides are present in marijuana. Toxicity varies greatly in the plants. The top leaves and flower buds of the female plants contain the highest toxin concentration, with the rest of the plant as well as male plants being toxic to a lesser degree.

FIRST AID: In nearly all cases, effects will wear off within a few hours with little or no permanent damage done. In cases of massive (or malicious) poisoning, contact a veterinarian as soon as possible. Emergency evacuation of the gastrointestinal tract may be necessary with followup supportive care. In cases where an animal (most often a dog) has consumed marijuana in baked goods (typically brownies), chocolate toxicosis may be additive to marijuana toxicosis. A veterinarian needs to be contacted in both cases of toxicoses.

SAFETY IN PREPARED FEEDS: Marijuana remains toxic when dried, therefore prepared feeds will still be toxic.

PREVENTION: Do not allow animals to have contact with the plant or any "preparations" where it might be contained and do not allow malicious intoxication.

36. JIMSONWEED, THORNAPPLE

Datura stramonium

(nightshade family)

TOXICITY RATING: High. The plant and seeds are extremely toxic, this plant is abused as a hallucinogen in humans, and deaths in humans and animals have been reported.

ANIMALS AFFECTED: All animals (including pets and poultry) may be affected.

DANGEROUS PARTS OF PLANT: All parts, especially seeds.

CLASS OF SIGNS: Dilated pupils, agitation, trembling, delirium, may appear to be experiencing hallucinations, convulsions (which may be violent), coma, and possible death. Abortions and birth defects have also been reported.

PLANT DESCRIPTION: This stout, coarse annual (fig. 36) grows to 5 feet tall with stronglyscented, coarsely toothed, green or purplish alternate leaves. The large trumpet-shaped flowers are white or purplish and are formed singly at the forks in the stems. The fruits are hard, spiny capsules (fig. 36) which split open along four lines at maturity to release numerous tiny black seeds. Jimsonweed commonly grows in cultivated fields, waste areas, barnyards, abandoned pastures, roadsides, and feedlots. Other *Datura* species (angel's-trumpets) are grown as ornamentals.

SIGNS: Animals will avoid eating Jimsonweed whenever possible. Even when forages are scarce, animals are reluctant to consume this plant. For animals, the danger lies primarily in the consumption of seeds that contaminate prepared feeds (hay, silage, grains, processed feeds). The plants may become palatable after the application of herbicides, thus greatly increasing the risk of toxicosis.

Once the plant is consumed, signs become apparent within a few minutes up to several hours. The alkaloids in Jimsonweed act on the central nervous system as well as the autonomic nervous system that controls bodily functions. Animals may seek water to drink, have dilated pupils, become agitated, may exhibit increased heart rate, tremble, become delirious, may appear to be experiencing hallucinations, have convulsions (which may be violent), become comatose, and possibly die. Consumption of Jimsonweed during gestation may result in abortions or birth defects.

Jimsonweed contains many toxic components, in particular the alkaloids, including atropine, hyoscyamine, and hyoscine (scopolamine). As much as 0.7% of the fresh weight of the leaves may be the toxic alkaloids, which is a very large quantity. The seeds are the greatest risk, with alkaloid concentrations believed to be greater than the leaves and stems, and even the nectar is toxic.

FIRST AID: Prevent further exposure to the plant or contaminated feed. Exercise caution when working with affected animals to avoid human injury. Contact a veterinarian if signs are severe, since there are medications that can counteract the effects of the toxin. Also, if consumption was recent, contact a veterinarian quickly, since it may be possible to evacuate a large amount of the plant from the digestive tract before the toxicosis becomes severe. For less severely affected animals (a veterinarian will be able to assist in determining this), the clinical signs will resolve within a day or two, so keep animals quiet and undisturbed.

SAFETY IN FEEDS: Jimsonweed remains toxic when dry, therefore feeds are not safe for consumption. The small black seeds are very toxic and may contaminate prepared or processed feeds. Toxic signs will occur even when good forage is fed, and there is no reported "safe" quantity of Jimsonweed that can be fed.

PREVENTION: Do not allow animals to graze on Jimsonweed. Provide adequate forage whenever animals are in contact with the growing plants. If the plants are treated with herbicides, make sure they are completely dead prior to introducing animals to the pasture. Use caution when harvesting feeds, especially near the edges of fields where Jimsonweed is likely to grow. In this way, incorporation of the plant or the seeds into the feeds will be prevented. When buying processed feeds, only work with reputable dealers.

37. SPURGES, EUPHORBIA

Euphorbia spp. (spurge family) EXAMPLES:

Euphorbia cyparissias, cypress spurge, fields and gardens (fig. 37A)

Euphorbia esula, leafy spurge, noxious weed

Chamaesyce maculata (Euphorbia maculata, old name), prostrate spurge, weed (fig. 37B

E. marginata, snow on the mountain, garden plant

E. splendens, crown of thorns, houseplant

E. lactea, candelabra cactus, houseplant

E. tirucalli, tinsel tree, milk bush, houseplant

E. pulcherrima, poinsettia, houseplant

TOXICITY RATING: Moderate. Spurges are highly unpalatable, and are rarely consumed in quantities sufficient to cause serious toxicity, but are very irritating upon contact.

ANIMALS AFFECTED: Any animal consuming spurge or coming in contact with the sap may be affected.

DANGEROUS PARTS OF PLANT: All parts.

CLASS OF SIGNS: Gastrointestinal irritation, dermal and ocular irritation, poor doer, weakness.

PLANT DESCRIPTION: These spindly annuals or herbaceous, sometimes succulent or even cactus-like perennials with milky, acrid sap have simple, alternate or opposite, entire or toothed leaves. The tiny flowers are clustered in small, cup-like structures themselves resembling white-petaled flowers in some species. The fruit, three-lobed and three-seeded, is borne on a stalk extending from the cup-like flower structure. Spurges grow in old fields, open woods, roadsides, waste areas, and around homes as cultivated or escaped plantings. Some are houseplants.

SIGNS: Spurges contain sap that is highly irritating upon contact, especially to the eyes and mouth, and upon prolonged exposure to skin (legs and head primarily). Irritation, redness, pain and swelling will result, and salivation and head-shaking if the oral mucosa is affected. Blistering and open sores are possible with spurge sap, and some plants have historically been used as a chemical brand for cattle. If the plants are swallowed, stomach and intestinal irritation can occur, with vomiting (in those species that can vomit), abdominal pain, and diarrhea.

FIRST AID: Prevent further contact and ingestion of spurge. If a large quantity was consumed, if an eye is affected, or if the animal cannot eat, contact a veterinarian immediately. For minor irritation, provide supportive care, since the signs are usually self-limiting within about 12 to 24 hours.

SAFETY IN PREPARED FEEDS: Spurges remain toxic when dry, therefore feeds are not safe for consumption. If small amounts have been incorporated into hay (where the plants are still recognizable), animals may voluntarily avoid consuming spurge if there is enough good feed available. Caution must be used in more highly processed feeds where consumption is unavoidable, such as silage, chop, and pellets.

PREVENTION: Animals should not be pastured where spurges grow. Mow or spray to eliminate them, since skin irritation can occur just by the animal's contact with the plant. Do not feed spurge-contaminated feeds, especially the more highly processed ones where consumption cannot be prevented. Small amounts of spurge in hay may be safe for most animals since they typically will avoid eating it, although all efforts should be made to provide clean feed.

38. COMMON ST. JOHNSWORT, KLAMATH WEED

Hypericum perforatum

(St. Johnswort family)

TOXICITY RATING: Low to moderate.

ANIMALS AFFECTED: Cattle, sheep, goats, horses, and swine.

DANGEROUS PARTS OF PLANT: All parts.

CLASS OF SIGNS: Sunburn, skin slough, eye irritation.

PLANT DESCRIPTION: This perennial herb (fig. 38) grows 1 to 11/2 feet 1/2 to 1 inch long and flat-topped clusters of golden yellow flowers 3/4 to 1 inch broad which bloom from midsummer to late fall. The five petals often have distinctive black dots around their edges (fig. 38A) and the leaves may have similar dots (fig. 38B). St. Johnswort commonly grows in droughty, poor, or over-grazed meadows, pastures, fields, and waste areas, usually on dry, gravelly, or sandy soils in full sunshine. Other species of *Hypericum* occur in Indiana, some as garden plants. Although the evidence of their toxicity is not as clear, it may be prudent to avoid them, too.

SIGNS: The toxin in St. Johnswort is called hypericin. When animals ingest the plant, the hypericin is absorbed from the intestinal tract and goes into the circulation. Hypericin is photodynamic, able to convert sunlight into energy (primarily heat), causing cellular damage and sunburn (which can be severe). Cattle and sheep are the most sensitive to this toxin, but swine and horses may also be affected.

St. Johnswort is not palatable and is eaten only when better food is unavailable. Animals must consume the plants for 4 to 5 days or more before clinical signs are noted. The affected skin first becomes swollen and tender, then reddened. This occurs primarily on the lightly pigmented areas (pink or white skin), and on the areas of the body that receive more sunlight (head, neck, back). The skin can be burned to the point where large areas of skin peel off. This is extremely painful, and predisposes the animal to infection. Affected animals are reluctant to have the areas examined, and may act abnormally and not want to eat due to the discomfort. Occasionally the eyes will be affected, causing redness and inflammation of the eyelids and the eye itself. These animals may not be able to see.

There are many plants that can cause sunburn either by contact or ingestion. In addition to St. Johnswort, some types of clover, vetches and buckwheat (*Fagopyrum*) have caused sunburn and skin scald in animals.

FIRST AID: The first steps are to prevent further consumption of the plant and to get the animals into the shade or a barn. Emergency evacuation of the gastrointestinal tract is not required since the toxin takes several days to build up in the body and cause signs. If the sunburn is mild, conservative treatment and supportive care is all that is required. Animals will resent handling, and horses will not be able to be ridden for at least 1 to 2 weeks. Keep animals in the shade, consider turning them out at night only. For more severely affected animals, including animals whose eyes are affected, or where the skin is blistered or sloughing, a veterinarian needs to be contacted, and antibiotics and anti-inflammatory medications provided.

SAFETY IN PREPARED FEEDS: The toxin in St. Johnswort remains active even when the plants are dry, therefore hay or processed feeds will still be toxic and should not be fed. However,

if the hay needs to be fed under extreme circumstances, keep all animals out of direct sunlight for up to one week after the contaminated feed is no longer being used.

PREVENTION: Animals will voluntarily avoid St. Johnswort if more nutritious and palatable forage is made available. If the pasture contains large stands of this plant, it may be best to mow, spray, or re-seed to improve the pasture quality and eliminate potential problems. If the plants are to be sprayed, keep animals off the pasture until the plants are completely dead, since herbicide treatment often increases plant palatability.

39. STAR-OF-BETHLEHEM, SNOWDROP, NAP-AT-NOON

Ornithogalum umbellatum

(lily family)

TOXICITY RATING: Moderate to low. While very toxic, exposure is not commonly reported.

ANIMALS AFFECTED: Cattle, sheep, horses, and potentially any grazing animal.

DANGEROUS PARTS OF PLANT: All parts, especially bulbs.

CLASS OF SIGNS: Stomach and intestinal irritation, abdominal pain, irregular heart rate, death (rarely).

PLANT DESCRIPTION: This perennial (fig. 39), a close relative of wild garlic (but without the smell), reproduces mostly by clumps of bulbs. The central flower stem grows 4 to 12 inches long. The leaves are about as long as the stem and have a light green midrib. Star-shaped flowers, six white petals with green stripes on the back, appear in spring. Usually the tops die back after flowering and before the fruit, a capsule, can be produced. Originally introduced to Indiana as a garden plant, star-of-Bethlehem has now gone wild along roadsides, in fields, and in woods, especially in the southern and western parts of the state.

SIGNS: Star-of-Bethlehem contains cardiac glycosides in all parts of the plant, with the bulbs containing a higher percentage of the toxin. This is not a commonly reported toxicosis, but it can be severe if encountered and if enough of the bulbs have been consumed. The bulbs may become more readily accessible after plowing, frost heaving or other such activity, thus increasing the risk of toxicosis. The toxic component (and therefore the toxic signs) are very similar to foxglove (*Digitalis*). The first signs are stomach and intestinal irritation, which is followed by abnormalities in the heart's rate and rhythm, and this can progress to fatal cardiac arrythmias.

FIRST AID: If animals are observed eating Star-of-Bethlehem, contact a veterinarian immediately, since evacuation of the gastrointestinal tract may be attempted to remove the toxin. Beyond this, therapy is symptomatic and supportive, often necessitating a veterinarian's care.

SAFETY IN PREPARED FEEDS: The toxin remains after the plant has dried. Since the toxin is powerful, there is no level that can be considered safe when feeding processed feeds containing Star-of-Bethlehem.

PREVENTION: Do not let animals graze Star-of-Bethlehem, and avoid incorporating this plant into hay and other feeds. It would be advisable to eliminate the plant from pastures, especially if they grow in any significant numbers.

40. COMMON POKEWEED, POKEBERRY, POKEROOT, INKBERRY, POKE

Phytolacca americana

(pokeweed family)

TOXICITY RATING: Low.

ANIMALS AFFECTED: All animals may potentially be affected.

DANGEROUS PARTS OF PLANT: All parts, especially roots and seeds.

CLASS OF SIGNS: Gastrointestinal irritation (colic, diarrhea which may be bloody). Rarely: anemia, possibly death. Birth defects and tumors may also be possible.

PLANT DESCRIPTION: Pokeweed (fig. 40) is a tall (to 10 feet), smooth-stemmed, perennial herb with a large, fleshy taproot (fig. 40A). Stems are succulent, purplish, and bear alternate, lance-shaped, shiny leaves with smooth, curled margins. The small, white to greenish flowers hang in long, drooping, grape-like clusters. Each flattened, spherical, green berry turns dark-purple or ink-black and usually contains 10 seeds. Pokeweed commonly grows on recently cleared land, in open woods, barnyards, pastures, fence rows, and roadsides.

SIGNS: Animals do not voluntarily eat this plant unless there is no other forage available. If the animals are forced to eat pokeweed (especially if it has been incorporated into processed feeds), the primary signs relate to the irritant effects of the saponin toxins, in particular phytolaccigenin. Salivation, abdominal pain, diarrhea (which may become bloody) can be noted. Horses and ruminants do not exhibit vomiting, which is seen in humans, dogs, cats, and pigs. Signs usually resolve within a day or two. Only if large doses are consumed will the animal display more serious signs: anemia, alterations in the heart rate and in respiration, and in very rare cases, death.

Noted in the human literature but not well published in the veterinary literature is the mutagenic and teratogenic properties of pokeweed, that is the ability to induce mutations (and possibly cancer) and birth defects. For humans, even handling the plant is considered dangerous, so it would seem wise to not only prevent human contact with the plant, but animal contact as well. Despite this, the plant is eaten as a spring vegetable in the southern U.S. after cooking it first in several changes of water. Consumption of the plant is not advised.

FIRST AID: For gastrointestinal irritation, provide better feed and symptomatic care, and signs should abate in about 24 hours. Discard all feeds containing pokeweed, since the plant is never safe for consumption. For severely affected animals, or if it is known that a large amount was consumed, consult a veterinarian promptly for emergency care.

SAFETY IN PREPARED FEEDS: Reports are not clear, but consider pokeweed as unsafe in hay and other feeds.

PREVENTION: Pokeweed should be removed from pastures and barnyards. Exercise caution when doing so, since the plant is toxic to humans as well. Good pasture management, with mowing and weed removal, will suffice in keeping pokeweed under control.

41. BOUNCING BET, SOAPWORT

Saponaria officinalis

(carnation family)

TOXICITY RATING: Low.

ANIMALS AFFECTED: All animals have the potential to be affected.

DANGEROUS PARTS OF PLANT: All parts, especially seeds and roots.

CLASS OF SIGNS: Mouth, stomach, and intestinal irritation.

PLANT DESCRIPTION: A very common and familiar weed of summer and autumn throughout the United States, bouncing bet (fig. 41) is found in colonies along roadsides and railroad tracks, in meadows, and waste areas. It is a knee-high, spreading, perennial weed with jointed stems. The leaves are opposite, simple, toothless, and slightly hairy. Phlox-like, flat-topped flower clusters consist of white or pinkish-white to red blossoms that have five petals, each with a slight notch at the tip.

SIGNS: Bouncing bet contains saponins, substances that when mixed with water produce a soaplike foam. These saponins produce gastrointestinal irritation upon ingestion. Animals will typically avoid eating this plant, however they may ingest it if extremely hungry and no better feed is available, or if parts of the plant (especially the seeds or the roots) are incorporated into prepared feeds. The plant needs to be consumed for several days before toxic signs are noted, which can include: mild depression, vomiting (in those species that can vomit), abdominal pain, diarrhea (which may become bloody). Overall, this toxicosis is not encountered frequently.

The seeds of corn cockle (*Agrostemma githago*) contains saponins and cause toxicity similar to that of bouncing bet. In the case of corn cockle, the poisonings occurred when seeds contaminated prepared feeds. Swine are particularly susceptible to this toxicity.

FIRST AID: If the animals are eating the plant in the pasture, either remove the animals or eliminate the plants. Provide better forage, since most animals will not eat this plant unless there is little else to eat. If the feed is contaminated, it may need to be discarded, since the continual presence of the saponins will perpetuate the gastrointestinal irritation. Call a veterinarian if animals are severely affected and do not recover within 12 to 24 hours, or if the animals were observed eating a large amount of the plants.

SAFETY IN PREPARED FEEDS: Feed and hay contamination with bouncing bet (especially the seeds) is the primary route of poisoning. The toxicity remains with drying and upon storage, and the feed is not safe for consumption.

PREVENTION: Maintain pastures in good condition, keeping weeds mowed. This alone will discourage most broadleaf plants from growing, including bouncing bet. Exercise caution when preparing hay or other feeds, so that no seeds or other plant part becomes incorporated into the mixture. When buying feeds, only do business with reputable suppliers.

42. BLACK NIGHTSHADE

CAROLINA HORSENETTLE, BULL NETTLE

Solanum carolinense

Solanum nigrum

BITTER NIGHTSHADE, CLIMBING BITTERSWEET

Solanum dulcamara

(nightshade family)

TOXICITY RATING: Moderate. While the plant itself is very toxic, it is also unpalatable, and rarely does an animal consume enough to cause a serious or potentially lethal poisoning. Toxic risk is higher if the plant is included in processed feeds.

ANIMALS AFFECTED: All animals, including pets, may be affected.

DANGEROUS PARTS OF PLANT: All parts are potentially toxic, the berries are often higher in toxicity.

CLASS OF SIGNS: The nightshade plants cause problems with the gastrointestinal tract and can also affect the central nervous system. Signs can include abdominal pain, vomiting, diarrhea, incoordination, weakness, depression, apparent hallucinations, convulsions, and possible death.

PLANT DESCRIPTION: Black nightshade is a low-branching annual (fig. 42A), 1 to 2 feet tall with triangular stems that bear oval, thin-textured, alternate leaves with wavy margins. The tiny white flowers, borne in drooping clusters on lateral stalks between the leaves, resemble tomato flowers. The berry fruit is green when immature, purplish-black when ripe. Bitter nightshade resembles black nightshade except that the stems are climbing, the lower leaves are lobed at the base, the flowers are purple, and the ripe fruit is red. Horsenettles (fig. 42B) are similar but have coarser, prickly stems, larger white to purplish flowers in loose clusters, and yellow fruits that look much like small tomatoes. All three species commonly grow in open woods, old fields, waste areas, pastures, along roadsides, and around farm buildings.

SIGNS: Clinical signs of poisoning in the nightshade family tend to reflect gastrointestinal irritation and/or effects on the central nervous system. The plant is not palatable and is eaten only when animals have no other forage available. The plant may be a contaminant in hay, where it will still cause toxicity. Pets may eat the green, red, or black berries and be poisoned. The major toxin is solanine, an alkaloidal glycoside, and along with other glycosides and atropine have numerous and powerful effects on the body.

Gastrointestinal signs can include: vomiting (in those species that can vomit), poor appetite, abdominal pain, and diarrhea which may become bloody. Central nervous system signs can include depression, difficulty breathing, incoordination, weakness, collapse, convulsions, and possible death. In one report, one to ten pounds of plant material was potentially lethal for a horse.

A chronic toxicity has also been reported, where the animal eats small amounts of the plants each day. These animals tend to present with general unthriftiness, depression, and diarrhea or constipation.

FIRST AID: If a large amount of nightshade plant was consumed, contact a veterinarian immediately, since emergency measures may need to be undertaken. In most cases the animals will avoid eating this plant, so clinical cases are rare. Curious or bored pets are particularly at risk, and a veterinarian should be contacted if these animals are seen eating a nightshade plant. Treatment is largely symptomatic until the clinical signs wear off (which can take a day or two, sometimes longer). Death is rare in animals, but has occurred in people who have abused these plants.

SAFETY IN PREPARED FEEDS: The nightshade plants lose some toxicity with drying, but the toxin is not eliminated. Therefore, feeds containing nightshade are not considered to be safe. If there are just a few plants in hay bales, the animals may voluntarily avoid the nightshade if provided with enough nutritious and safe hay. In the case of hay then, careful observation to see that the nightshades are being left may make it allowable to feed the rest of the affected bales. If the feed is more highly processed (silage, chop, pellets), the feed is not safe at all since the animals will not be able to avoid the nightshade plants.

PREVENTION: Nearly all grazing animals will avoid eating plants in the nightshade family unless they are extremely hungry and there is little else to eat. The exception to this is if nightshade plants are incorporated into prepared feeds and the animals eat them unknowingly, therefore only feed quality feeds and only purchase from reputable dealers. Pets may be attracted to and eat the berries, so always keep pets away from nightshade plants, especially if the pet is confined, bored or unattended.

NOTE: Other Solanum species contain the same poisonous principle. These include buffalobur (*Solanum cornutum*), the ornamental Jerusalem cherry (*Solanum pseudocapsicum*), and the common white potato (*Solanum tuberosum*). Sprouts and sunburned (green) or spoiled potato tubers should not be mixed in feed because they also contain solanine. Vines of tomatoes (*Lycopersicon esculentum*, nightshade family) contain similar glycoalkaloids. Toxicity is also related to that seen with Jimsonweed.

43. JOHNSONGRASS

Sorghum halepense

(grass family)

TOXICITY RATING: Moderate to high.

ANIMALS AFFECTED: All types, especially ruminants.

DANGEROUS PARTS OF PLANTS: Leaves and stems, especially young plants.

CLASS OF SIGNS: Breathing problems, staggering, severe anxiety, convulsions, coma, death (may be very sudden).

PLANT DESCRIPTION: Johnsongrass (fig. 43), a coarse perennial grass, produces large, scaly rootstocks and grows in dense stands up to 6 feet high. Seed heads are large and loose. This plant grows commonly in the fields, fencerows, and ditch banks of the southern part of the state and is rapidly spreading northward. Once grown for dike stabilization or for hay, it is now classified as a "noxious" weed.

SIGNS: The toxic signs resulting from ingestion of Johnsongrass are due to the presence of cyanide in the leaves and stems. This toxicity is identical to that resulting from the ingestion of wilted or damaged cherry leaves. In normal, healthy Johnsongrass plants, the levels of free cyanide are low, and the plant can be consumed safely. Other members of this grass genus have been bred as grain or forage plants (milo, Sudan grass, etc.) and also have the potential to produce cyanide, although not as much as Johnsongrass. The young shoots are the most dangerous, and when wilted, trampled, herbicide treated or frost damaged, a great deal of free cyanide is liberated in the leaves. Upon ingestion, the animals quickly develop signs related to cyanide poisoning. Mature plants have much lower toxicity, and well-cured hay is relatively safe for consumption.

The most common occurrence of Johnsongrass toxicity is on pasture after the plants have been damaged somehow. Cyanide prevents the body from being able to utilize oxygen at the cellular level, so although the animals physically can breath, their tissues and cells "suffocate". After consumption, signs will manifest within a few minutes, but sometimes up to an hour may pass. The animals will try to breath more rapidly and deeply, and then become anxious and stressed. Later, trembling, incoordination, attempts to urinate and defecate and collapse is noted, which can proceed to a violent death (respiratory and/or cardiac arrest). If an affected animal is still

alive 2 or 3 hours after consumption, chances are good that it will live. Under rare circumstances, a chronic cyanide toxicity can develop, which causes weakness and partial paralysis, but this is not a common occurrence. In the southwestern U.S., a syndrome affecting the urinary bladder and nervous system in horses has been reported after consumption of sorghum grasses. This disorder is called equine sorghum cystitis ataxia, and the animal is uncoordinated, has urinary incontinence (dribbles urine uncontrollably), and foals of affected mares may be aborted or be born with birth defects. The disorder can also cause death.

Johnsongrass, like other plant species, can accumulate toxic levels of nitrates, depending on fertilization practices. Nitrates are extremely toxic and can also cause death, especially in cattle, sheep and goats. Look in the section on oats for more discussion of nitrate poisoning. The signs of nitrate poisoning may be confused with cyanide poisoning.

FIRST AID: The clinical signs of cyanide poisoning tends to come on quite rapidly, and the animals may be found dead without much warning. If the animals are exhibiting toxic signs, call a veterinarian immediately. There is an antidote, but it needs to be given intravenously and within a few minutes of the onset of signs, and it is often impossible to get help in time. Prevent the animals (especially the unaffected animals) from eating any more of the grass or feed. Do not handle or stress affected animals any more than absolutely necessary, since this will worsen the signs. Also, affected animals are extremely stressed and may be dangerous to work with, therefore exercise caution so no human injury results. Similar recommendations exist for nitrate poisoning, although death usually doesn't occur as quickly as with cyanide.

SAFETY IN PREPARED FEEDS: When johnsongrass is dry, the level of cyanide is extremely low, so properly cured hay is safe for consumption. If the grass is used for silage or green chop, the levels of cyanide may still be quite high and potentially lethal. If the grass has accumulated toxic levels of nitrates, the hay, silage and chop will continue to be toxic. This may be fed in small amounts along with an adequate supply of good forage, although it would be best to discard feeds that are high in nitrates.

PREVENTION: Johnsongrass and other sorghums may be grazed safely throughout most of the growing season. Extra care must be taken when the grass is young, especially if damaged somehow. Animals should not be allowed access to the grass if these circumstances occur. Care must also be taken when fertilizing johnsongrass (as well as many other types of grasses) so as to not accumulate toxic levels of nitrates. Do not use johnsongrass as green chop or silage.

[[SECTION: Trees and Shrubs]]

44. OHIO BUCKEYE HORSECHESTNUT

Aesculus glabra Aesculus hippocastanum

(horsechestnut family)

TOXICITY RATING: Moderate to high.

ANIMALS AFFECTED: All animals may be affected, especially grazing animals and those consuming the honey.

DANGEROUS PARTS OF PLANT: Buds, nuts, leaves, bark, seedlings, and honey.

CLASS OF SIGNS: Two effects: gastrointestinal and neurologic: excessive salivation, gastrointestinal irritation, vomiting in those species that can vomit, abdominal pain, diarrhea.

Neurologic signs can include staggering, trembling, breathing difficulty, dilated pupils, collapse and paralysis, which can proceed to coma and death.

PLANT DESCRIPTION: The thick twigs of these medium-sized trees have glistening buds in spring and bear opposite leaves composed of five leaflets in a finger-like arrangement (fig. 44). The yellowish flowers rise in large, upright, dense, candle-like clusters at branch ends during June. The prickly fruit contains 1 to 3 nutlike seeds, glossy and leathery brown with a pale scar on each that gives the tree its name. These trees commonly grow in rich, moist woods or along river banks and are often planted as ornamentals.

SIGNS: The toxins in Buckeye and Horsechestnut affect the gastrointestinal tract as well as the nervous system. The saponic glycoside aesculin in addition to suspected alkaloids cause the toxic signs. Initially, gastrointestinal signs manifest, which can include salivation, vomiting (in those species that can vomit), abdominal pain, and diarrhea. If enough was ingested, neurologic signs may develop, including trembling, staggering, and difficulty in breathing. Toxicity may then progress to collapse, paralysis, coma and death.

These trees are among the first to leaf out in the spring, and hungry animals on pasture may be tempted to eat them if no other forages are available.

FIRST AID: If animals are observed eating Ohio Buckeye or Horsechestnut, contact a veterinarian immediately; emergency measures can be used to remove plant material from the digestive tract. Once clinical signs are present, and if it has been several hours after the plants were consumed, supportive care is all that can be provided, since there is no antidote for this toxicosis.

SAFETY IN PREPARED FEEDS: Reports are unclear concerning the safety of Ohio Buckeye or Horsechestnut in processed feeds. Therefore, in the interest of safety, consider these plants as toxic and do not feed processed feeds if contaminated.

PREVENTION: If animals are to be pastured with these trees, be certain that adequate, nutritious forage is available. In this way, the animals are likely to avoid consuming toxic quantities of these trees.

45. BLACK WALNUT

Juglans nigra

(walnut family)

TOXICITY RATING: Moderately toxic, depending upon length of exposure.

ANIMALS AFFECTED: Horses, dogs, possibly other animals.

CLASS OF SIGNS: Laminitis, breathing problems, gastroenteritis.

PLANT DESCRIPTION: These familiar trees are recent additions to the list of poisonous plants. Little information is yet available about their toxicity. Black walnuts are large (60-80 foot) forest trees often

planted as ornamentals. The bark has characteristic broad, round ridges. The leaves are alternate, pinnately compound, 1 to 2 feet long, with 13 to 23 sharply toothed, tapered-pointed leaflets (fig. 45). Often there is no terminal leaflet. The fruit is a very rough nut enclosed within a clammy glandular husk, 2 to 4 inches in diameter (fig. 45A).

SIGNS: Horses are most often affected. When horses are bedded on wood shavings containing more than about 20% of black walnut shavings (which tend to be dark in color), clinical signs of laminitis (inflammation of the laminae in the hoof) can occur within 12 to 18 hours of contact, but 24 hours may elapse before signs manifest. Consumption of the shavings may cause signs of laminitis as well as mild colic. Affected horses become unwilling to move or have their feet picked up, are depressed, may exhibit limb edema and signs of laminitis. Difficulty in breathing (increased rate and depth) may be noted. Horses on pasture may show mild respiratory signs from pollen or fallen leaves. Poisoning in dogs is reported occasionally when the seed hulls are consumed, causing stomach upset and diarrhea (gastroenteritis).

The toxin causing equine laminitis has not been clearly identified. It was once thought to be juglone, but this is not believed to be the toxin. Juglone is produced by the tree which limits the growth of other plants in the vicinity. Walnuts are also lethal to earthworms.

FIRST AID: Remove black walnut shavings immediately; cooling the hooves and legs with a hose can help. Call a veterinarian if signs are severe or if shavings were consumed. Prompt removal of shavings typically results in complete recovery. Respiratory signs in horses usually do not require treatment unless severe or long-lasting. Stomach upset in dogs will resolve when hulls are no longer eaten.

SAFETY IN PREPARED FEEDS: Reports are not clear concerning the safety of black walnut leaves in processed feeds. The bark and seed hulls are toxic, and these should never be incorporated into any feeds. In the interest of safety, any part of the black walnut plant should not be allowed to contaminate feeds.

PREVENTION: Do not use shavings containing black walnut, limit access of horses to pastures with walnut trees. Purchase bedding shavings only from reputable dealers. Do not let dogs eat walnut hulls.

46. WILD BLACK CHERRY

Prunus serotina

(rose family)

TOXICITY RATING: High.

ANIMALS AFFECTED: All animals may be affected. Ruminants (cattle, sheep, goats, deer) are more at risk than monogastric animals (dogs, cats, pigs, horses) and birds.

DANGEROUS PARTS OF PLANT: Damaged leaves pose the greatest risk. All parts are potentially toxic.

CLASS OF SIGNS: Anxiety, breathing problems, staggering, convulsions, collapse, death (which may be sudden).

PLANT DESCRIPTION: This cherry may grow as a tree or shrub. Bark of young branches and twigs is scaly and reddish-brown with prominent cross-marks ("lenticels"). Leaves (fig. 46) are alternate, simple, elliptic-pointed, leathery in texture, and finely toothed on the margins. Flowers are showy, fragrant, and white, hang in drooping clusters, and produce dark-red to black cherry fruits (fig. 46A). The wild black cherry commonly grows in fence rows, roadside thickets, and rich open woods.

SIGNS: Black cherry contains cyanogenic precursors that release cyanide whenever the leaves are damaged (frost, trampling, drought, wilting, blown down from the tree during storms). Most animals can consume small amounts of healthy leaves, bark and fruit safely; however when hungry animals consume large amounts of fresh leaves or small amounts of damaged leaves (as little as 2 ounces), clinical cases of poisoning will occur, and many animals may die. This is especially true if there is no other forage for the animals to consume, or in the case of pets, when confined and/or bored, the chances for toxic levels of ingestion can occur. The conditions of cyanide poisoning have also been discussed under Johnsongrass.

Healthy cherry leaves contain prunasin, a cyanide precursor that in itself is non-toxic. When the leaves are damaged, the prunasin molecule is split and free cyanide (also called prussic acid or hydrocyanic acid) is liberated. Many plants, especially those in the rose family, have the potential to produce toxic levels of cyanide under certain conditions. Chokecherry (*Prunus virginiana*) is also toxic. There are reports of peach sprouts, leaves, and pits poisoning sows. Apricot pits and apple seeds are toxic as well. Arrowgrass (*Triglochin maritima*) contains a cyanogenic glycoside and has caused poisoning in livestock. Johnsongrass, discussed earlier, has a similar toxicity.

Cyanide prevents the body from being able to utilize oxygen at the cellular level, so although the animals physically can breath, their tissues and cells "suffocate". After consumption, signs will manifest within a few minutes, but sometimes up to an hour may pass. The animals will try to breath more rapidly and deeply, and then become anxious and stressed. Later, trembling, incoordination, attempts to urinate and defecate and collapse is noted, which can proceed to a violent death from respiratory and/or cardiac arrest within a few minutes to an hour. If an affected animal is still alive 2 or 3 hours after consumption, chances are good that it will live.

FIRST AID: The clinical signs of cyanide poisoning tends to come on quite rapidly, and the animals may be found dead without much warning. If the animals are exhibiting toxic signs, call a veterinarian immediately. There is an antidote, but it needs to be given intravenously and within a few minutes of the onset of signs, and it is often impossible to get help in time. Prevent the animals (especially the unaffected animals) from eating any more of the grass or feed. Do not handle or stress affected animals any more than absolutely necessary, since this will worsen the signs. Also, affected animals are extremely stressed and may be dangerous to work with, therefore exercise caution so no human injury results.

SAFETY IN PREPARED FEEDS: Cyanide is lost to the air with time, so processed feeds containing cherry may technically be free of the toxin. However, green chop and silage containing cherry will still retain large amounts of cyanide aside from being feeds of poor quality. Dried products would also not be of high quality if they contain cherry, but the cyanide levels will be much lower. Caution is still advised when feeding cherry-contaminated feeds.

PREVENTION: Do not allow animals to have access to damaged cherry leaves, especially if they are hungry and there is no other forage available. Do not place fallen branches or tree trimmings where animals can graze them. Exercise caution with animals on pasture after storms, during droughts or after a frost since these conditions will increase the chances of toxic levels of ingestion. For pets, do not house or confine animals in the vicinity of cherry, since boredom will increase the likelihood that the plant will be eaten. For most species of cherry, the fruit is safe for consumption. It is the leaves and bark which pose the greatest risk.

47. RED OAK

Quercus rubra

(beech family)

TOXICITY RATING: Moderate high.

ANIMALS AFFECTED: All animals may potentially be affected, but the primary risk is to cattle.

DANGEROUS PARTS OF PLANT: Buds (fall), young shoots (early spring), sprouts, acorns.

CLASS OF SIGNS: Poor doer, poor appetite, weight loss, diarrhea or constipation, increased drinking, increased urination, edema, death is possible.

PLANT DESCRIPTION: Oaks are trees with leaves that turn brown but hang on through the winter. In the southwestern U.S., Gambel's oak, shinnery oak, and post oak frequently cause poisonings. In our part of the country, red oak has produced problems. Red oak is a large tree of well-drained woodlands, parks, and home plantings that bears broad-bladed leaves with deep lobes ending in bristle-tips (fig. 47). The

fruit is the familiar nut borne in a scaly cup and called an acorn (fig. 47A).

SIGNS: This discussion refers primarily to cattle, the species most often affected by oak toxicosis. It also seems that cattle less than 2 years of age succumb to oak toxicosis more than do older animals, however older animals are still at risk. Other species at risk include sheep and possibly deer. Goats and swine are more resistant to poisoning, and horses are rarely affected (likely due to a unwillingness to consume oak). Pets rarely consume sufficient quantities to do harm. Many species of oak have been implicated in the poisoning of livestock, with red and black oak exhibiting greater toxicity than white oak.

While short-term acute poisoning by oak has been reported, the most commonly encountered oak poisoning is of a chronic nature. Oak is most dangerous early in the spring when the leaves and buds are the highest in toxicity and when there is little else to eat. The fall is another at risk period, when acorns and leaves fall and better forage dies back. Therefore, management plays a key role in preventing oak toxicosis.

The toxins in oak are called gallotoxins and are converted in the body to tannic acid, gallic acid and pyrogallol, all of which are very toxic to the kidney. It is the resulting kidney failure that causes the clinical signs. Typically, a significant amount of oak needs to be consumed over a period of time before clinical signs appear. Signs can develop over 2 to 14 days, or signs may be present with the animals becoming progressively worse over many weeks. The number of animals affected in the herd can vary greatly, but of those showing clinical signs, up to 80% may die. Signs of oak poisoning can include depression, lack of appetite, a gaunt and emaciated appearance, poor or rough hair coat, dependent edema (fluid buildup under the skin under the neck, abdomen or on the legs), digestive disturbances (both diarrhea and constipation have been reported, with mucus covered or tarry stools), increased drinking, passage of copious amounts of urine which may contain blood, and death.

FIRST AID: The most important step is to get the animals back on to plentiful and nutritious feed, and to limit stress, shipping and handling during the recovery period. Also, make sure that plenty of fresh water is always available, since affected cattle cannot maintain their own water balance very well while recovering. Since this is a long-term chronic toxicity, there is little in the way of an antidote to relieve signs. Severely or more chronically affected animals may not recover, but the less affected animals may, and may return to previous rates of gain and milk

production. A veterinarian will be able to assist in management, and will be able to assist in emergency measures if large amounts of oak were recently consumed. Beyond this, treatment is supportive and symptomatic.

SAFETY IN PREPARED FEEDS: The toxin in oak remains when dry, so no feeds are safe that contain oak.

PREVENTION: Oak toxicosis is easily preventable with proper livestock and pasture management. Animals tend to eat oak only out of necessity, therefore by providing adequate and nutritious feed in the spring when the oak leaves bud out and again in the fall when leaves and acorns drop, the incidence of toxicosis should be minimized or eliminated.

48. BLACK LOCUST

Robinia pseudo-acacia

(pea family)

TOXICITY RATING: High to moderate.

ANIMALS AFFECTED: Horses are particularly at risk, but all animals ingesting the plant may be poisoned.

DANGEROUS PARTS OF PLANT: Leaves, especially wilted leaves, young shoots, pods, seeds, inner bark.

CLASS OF SIGNS: Depression, poor appetite, weakness, paralysis, abdominal pain, diarrhea (which may be bloody) and abnormalities in the heart rate and/or rhythm. Death is possible.

PLANT DESCRIPTION: These moderate-sized trees with rough bark often bear two short spines at the base of each leafstalk (easiest to see on young leaves). Leaves are alternate and pinnately compound with oval, entire leaflets (fig. 48). The fragrant flowers are creamy white, sweet-pea-like, and arranged in long drooping clusters. The fruit is a flat brown pod which contains kidney-shaped beans (fig. 48A). Black locusts are common in well-drained woods, thickets, and waste areas, especially in the southeastern part of the state. They are often planted along highways and fencerows as ornamentals and for erosion control.

SIGNS: This discussion will center on the effects in horses, the species most likely to be poisoned by black locust. Horses may ingest the bark or leaves when hungry and no other forage is available, or if they are confined or bored in the vicinity of the tree.

There are several toxic components in black locust including the toxic protein robin, the glycoside robitin, and the alkaloid robinine. The toxins affect the gastrointestinal tract as well as the nervous system. Clinical signs can manifest as soon as one hour after consumption and can include depression, poor appetite, generalized weakness to paralysis, abdominal pain, diarrhea (which may be bloody) and abnormalities in the heart rate and/or rhythm. With sufficient amounts ingested, death may occur within a few days, although black locust is not always lethal. Some animals recover despite showing clinical signs, an indication of the dose-dependent nature of the toxin.

Honey locust (*Gleditsia triacanthos*, pea family) has been implicated in causing similar toxic signs, but the information on this is not clear. Prickly ash (*Zanthoxylum americanum*, citrus family) superficially resembles black locust in vegetative aspect and has been blamed for loss of sheep.

FIRST AID: If horses are observed eating black locust, contact a veterinarian immediately, since emergency measures to rid the gastrointestinal tract of toxin may be implemented. Beyond this, therapy is aimed at preventing further exposure and keeping other animals away from the trees, and treating clinical signs symptomatically. Recovery may take days to weeks. Be extra cautious around affected horses to prevent human injury, and these horses should not be ridden until all clinical signs have resolved.

SAFETY IN PREPARED FEEDS: Reports are not clear on this matter, but given the potentially toxic nature of black locust, it should never be allowed to contaminate feeds, especially those destined for horses.

PREVENTION: Do not confine horses in an area where black locust grows. If this is unavoidable, provide enough palatable feed so that the horses leave the trees alone. Some horses are wood and bark chewers, however, and for these horses is may be necessary to fence off the trees or utilize a different pasture to prevent toxicosis. Paints and sprays to prevent wood chewing may be tried, but long-term success with these treatments may be difficult.

49. ENGLISH YEW JAPANESE YEW

Taxus baccata Taxus cuspidata

(yew family)

TOXICITY RATING: Extremely toxic, death is likely.

ANIMALS AFFECTED: All animals (livestock, pets and birds).

PLANT DESCRIPTION: Several species of yew are planted as ornamental shrubs or hedges. They are woody perennials with flat 1/2-1 inch long evergreen leaves (fig. 49) lighter green on the underside and broader than pine needles. The "berry" (technically called an aril) is grapesized, juicy, and bright scarlet, with a hole in the end which makes it look cup-like.

CLASS OF SIGNS: Sudden death is the typical sign. Occasionally: breathing problems, trembling, weakness, heart problems, stomach upset.

SIGNS: "Found dead" is the typical presenting sign. Very rarely will animals show signs up to 2 days later: trembling, slow heart rate, difficulty breathing, gastroenteritis (stomach upset and diarrhea). The plant is exceptionally toxic, with one mouthful able to kill a horse or cow within 5 minutes. Toxicity is compounded by the apparent palatability of yew. Many animals are poisoned accidently when yew trimmings are thrown into the pasture or when yew is planted as an ornamental within browsing reach. Infrequent reports of dogs chewing the leaves resulted in gastroenteritis, seizures, and aggressive behavior.

The toxin is taxine, a mixture of alkaloids, that slow down cardiac conduction. As little as 0.1 to 0.5% of the fresh plant per body weight is lethal. Death is due to cardiac and/or respiratory collapse.

FIRST AID: First aid is usually impractical, since the animals die so quickly. Prevent other animals from being exposed and use caution around animals showing clinical signs to prevent human injury. If animals are still alive, contact a veterinarian. Cardiac drug therapy may be attempted, but success is unlikely.

SAFETY IN PREPARED FEEDS: Yew is toxic even when dry, therefore hay with yew in it is never safe in any amount.

PREVENTION: Never allow yew plants or trimmings within reach of horses, cattle, sheep, goats, pigs, birds, or any other animal likely to eat plants. Dogs and cats rarely chew on this plant, so it is not necessary to remove it from ornamental gardens. Toxicities in dogs occurred when puppies were confined to a pen with yew and chewed the plant out of boredom. The fleshy red "berry" is not considered toxic, but consumption is not advised.

[[PRINT edition included an index of scientific names and an index of common names. There also was an index of animals affected, and a pullout table of poisoning symptoms.]]

[[PLANT SPECIES included in the online version but not in the print edition appear below.]]

50. Red Maple

Acer rubrum

(maple family)

TOXICITY RATING: High, death is common.

ANIMALS AFFECTED: Horses only.

DANGEROUS PARTS OF PLANT: Leaves, especially when fallen, damaged, or wilted.

CLASS OF SYMPTOMS: Breathing difficulties, jaundice, dark brown urine, death.

PLANT DESCRIPTION: Red maple is a tree of medium size, occurring naturally or planted as an ornamental. Young bark is a smooth gray color, older bark is dark and broken. Leaves are 3 to 5 lobed, with shallow notches between lobes. Underside of leaves are white. Leaves are green during the growing season and turn red in the fall. Buds, twigs, flowers, and petioles are red. The sap is not milky.

SIGNS: The toxin has not been identified, but is believed to be an oxidant due to its effects on red blood cells. Only horses are known to be affected. The ingestion of wilted or fallen leaves causes massive destruction of red blood cells, and the blood can no longer carry sufficient oxygen. Ingestion of 1.5 pounds of leaves is toxic, and ingestion of 3 pounds is lethal. Wilted or dry leaves remain toxic for about a month. Fresh and undamaged leaves have not been implicated, but ingestion is still not advised. Clinical signs develop within one or two days and can include depression, lethargy, increased rate and depth of breathing, increased heart rate, jaundice, dark brown urine, coma, and death. Approximately 50% to 75% of affected horses die or are euthanized.

FIRST AID: The first step is to prevent further consumption by the horse (and any other horses on the same pasture). There is no specific treatment, and contacting a veterinarian is advised. The veterinarian may use methylene blue, but this is not often effective in horses, but can be tried early in the course of the disorder. Treatment is symptomatic and supportive and can include mineral oil and activated charcoal to prevent further absorption in the stomach, oxygen, fluid support, and blood transfusions.

SAFETY IN PREPARED FEEDS: Reports are lacking, but red maple should not be considered safe in hay, especially if it is freshly baled.

PREVENTION: Do not allow horses access to red maple. Most poisoning occur in the late summer and fall when leaves fall into pastures, or at any time when fallen limbs are placed in

pastures. Apparently the leaves are palatable, thus increasing the risk of toxicosis. Do not incorporate red maple leaves into hay bales.

51. Easter lily

Lilium longiflorum

Lily family

TOXICITY RATING: High.

ANIMALS AFFECTED: The only reported toxicity is in cats.

DANGEROUS PARTS OF PLANT: Leaves primarily, stems and flowers may also be toxic.

CLASS OF SIGNS: Gastrointestinal irritation (vomiting), depression, lack of appetite.

PLANT DESCRIPTION: (I need to get this to you)

SIGNS: This is a newly reported toxicosis, apparently lethal only to cats. Upon consumption of Easter lily (the exact amount is unknown), the cats begin to vomit within an hour or so. The cat then becomes depressed over the next half day, presumably as the toxin begins to affect the kidneys. Within 48 to 96 hours after consumption, the cat will tend to show signs of clinical kidney failure: increased urination, depression, stomach upset, dehydration. Death tends to occur within 5 days.

FIRST AID: If a cat is seen eating Easter lily, contact a veterinarian immediately. If emergency treatment is begun within 6 hours of consumption, the chance are good that the cat will recover. This generally consists of emptying the gastrointestinal tract of the affected cat and intravenous fluid therapy in a hospital setting. If more than 18 hours has elapsed, the cat may not survive, even with emergency care.

PREVENTION: Easter lily is a popular plant at certain times of the year, and extra caution must be used when bringing these plants into the house where cats can get at them. Make sure the plant is kept away from cats, especially ones that like to nibble on things. If nibbling plants is unavoidable, have a selection of safe plants available (grass or catnip are two possibilities).

52. Oleander

Nerium oleander

Apocynaceae (Dogbane family)

TOXICITY RATING: High. Ingestion of even small amounts can kill.

ANIMALS AFFECTED: All animals can be affected.

DANGEROUS PARTS OF PLANT: The entire plant is toxic. Most animals are poisoned by consuming leaves, fresh or dried.

CLASS OF SIGNS: Gastrointestinal irritation, cardiac abnormalities, death (may be sudden).

PLANT DESCRIPTION: This plant grows outdoors in warmer regions, and in Indiana is grown as a houseplant. Oleander grows as a shrub or sometimes to the size of a small tree. The leaves are lance-shaped, thick and leathery, and grow opposite each other. Sometimes, leaves may grow in whorls. The leaves are 8 to 10 inches long, although smaller specimens will have shorter

leaves. Flowers are showy, approximately 1 to 3 inches in diameter, and grow in large clusters at the ends of the branches, and can be white or any shade of pink or red.

SIGNS: Oleander contains the toxins oleandrin and nerioside, which very similar to the toxins in foxglove (*Digitalis*). This is a tropical plant, but is grown as an ornamental and as a houseplant in Indiana. Apparently the plant is not palatable, but will be eaten by hungry animals. It is reported that dried or wilted leaves may be slightly more palatable than fresh leaves, and the leaves are still toxic when wilted or dried. In one report with horses, it was indicated that approximately 1/4 pound of leaves (about 30 or 40 leaves) could deliver a lethal dose to an adult horse.

Clinical signs may develop rapidly, and the animal may be found dead with no prior warning. In other cases, depression coupled with gastrointestinal distress is evident: vomiting (in those species that can vomit), diarrhea (which may be bloody), and abdominal pain. Irregularities in the heart rate and rhythm will occur: the heart may speed up or slow down, and beat erratically. As the toxicosis progresses, the extremities may become cold and the mucous membranes pale. Trembling and collapse can occur, followed by coma and death within a few hours.

FIRST AID: If animals are observed eating oleander, contact a veterinarian immediately. The toxin acts quickly, and is lethal in small amounts. Emergency measures may be used to empty the gastrointestinal tract of remaining plant matter, and medications may be administered to control the effects that the toxin has on the heart. Despite emergency care, the animal may still die, but the sooner treatment is begun, the better the prognosis for survival.

SAFETY IN PREPARED FEEDS: Oleander is extremely toxic, even in small quantities, and the toxin is not eliminated by drying. Therefore, feeds containing oleander are never safe for consumption.

PREVENTION: Be able to identify oleander and exercise extreme caution when pets (and humans) are in the vicinity of these plants. The plants should never be placed where animals can have contact with them. Extra care needs to be taken in cases where leaves can fall into a pasture or in the vicinity of a confined, bored or hungry animal.

53. Rosary Pea, Precatory beans, crabs eye, jequirity bean

Abrus precatorius

legume or pea family

TOXICITY RATING: High. Even one bean can kill.

ANIMALS AFFECTED: All animals may be affected, although the primary risk is to pets.

DANGEROUS PARTS OF THE PLANT: The beans are the primary risk.

CLASS OF SIGNS: Severe gastrointestinal irritation, vomiting, diarrhea, abdominal pain, collapse, death.

PLANT DESCRIPTION: Twisting perennial vine, grows naturally in tropical climates. Rosary pea is established in certain areas of southern Florida. The leaves are alternate and compound, with 8 to 15 leaflets. The flowers are small, and can be any shade of white to red to purple. The seed pod is about 1 and 1/2 inches long, containing several seeds, bright red with a black spot.

SIGNS: The toxic signs resulting from rosary pea ingestion are very similar to those of castorbean, except rosary pea contains a more powerful toxin. It is reported that one seed if well-chewed can kill an adult human. The toxins are a protein called abrin and a glycoside called abric acid, which cause severe gastrointestinal signs: vomiting (if the species can vomit), diarrhea (which may be bloody) and abdominal pain. This progresses to weakness, shock and death within a short period of time.

The plant does not grow in Indiana, but sometimes seeds are imported to make jewelry and rosaries. If the seed is swallowed without damage to the seed coat, poisoning is unlikely, and the seed will tend to pass without incident. In cases where the seed coat is chewed or opened (as in drilling to make jewelry), toxic signs and death are likely.

FIRST AID: If rosary pea was ingested, contact a veterinarian immediately. Prevent further exposure and get other animals away from the source. Emergency measures may be used to eliminate the toxin from the stomach and intestines. Once gastrointestinal signs appear, it may be inadvisable to try to evacuate the stomach and intestines for fear of doing even greater damage, but a veterinarian will decide this. Beyond this, care is symptomatic and supportive of the digestive upset, weakness, and shock. Affected animals are likely to die even with care.

SAFETY IN FEEDS: The seed is toxic in feeds, and is never safe at any level of feeding. Discard all feeds if contaminated with rosary pea.

PREVENTION: Rosary pea should never be allowed around the home or pasture for the safety of animals and humans alike. If jewelry or rosaries are made of rosary pea, discard them immediately.