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Plants Affecting the Cardiovascular System (17-Sep-2002)

A. P. Knight¹ and R. G. Walter²

¹Department of Clinical Sciences, College of Veterinary Medicine, Veterinary Teaching Hospital, Colorado State University, Fort Collins, CO, USA.

²Department of Biology, Colorado State University, Fort Collins, CO, USA.

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A large group of unrelated plants have toxic compounds that have a direct effect on the heart and blood vessels and can cause the death of animals that consume them. The most recognized of these compounds are the cardiac glycosides, of which digoxin, found in foxglove (*Digitalis* spp.), is best known. The toxic and pharmacologic properties of digoxin have been known for a long time. Because of its effects on the heart at therapeutic levels, it is routinely used to treat congestive heart failure in humans and animals [1]. Other cardiotoxic compounds found in plants such as yew (*Taxus* spp.), rhododendrons (*Rhododendron* spp.), laurels (*Kalmia* spp.), and the avocado (*Persea* spp.) are responsible for poisoning in animals and humans.

Cardiac Glycosides

Cardiac glycosides are found in 11 plant families including the Apocynaceae, Asclepiadaceae, Celastraceae, Brassicaceae, Lilaceae, Moraceae, Fabaceae, Ranunculaceae, Scrophulariaceae, Sterculiaceae, and Tiliaceae [2]. The glycosides that have specific effects on the heart are present in at least 34 genera of these plant families [2,3]. There are 2 basic groups of glycosides in plants, cardenolides and bufadienolides, both of which have direct effects upon cardiac function. The best known cardiac glycosides (cardenolides) are digoxin and digitoxin, derived from the parent glycoside digitalis. The therapeutic value of digitalis in treating heart failure was first recognized in 1775 [4] and it is now produced commercially for therapeutic purposes from foxglove (*Digitalis lannata*) [5]. Immunologic methods have enabled detection of glycoside concentrations in a variety of other plant genera [6]. Other cultivated plants that contain cardenolides include: *Euonymus* spp., Star of Bethlehem (*Ornithogalum* spp.), lily of the valley (*Convallaria majalis*), and oleander (*Nerium* spp.). Bufadienolides being found in hellebores (*Helleborus* spp.), lily of the valley (*Convallaria majalis*), and squill (*Drimia* or *Urginea* spp.).

Relatively few of the plants containing cardiac glycosides are a significant cause of animal poisoning in North America [7]. Milkweeds (*Asclepias* spp.) are the most frequent cause of cardiac glycoside poisoning in livestock in North America [8-10]. Oleander is becoming of increasing significance as a source of poisoning because of the frequency with which it is planted as an attractive, drought-resistant, ornamental flowering shrub. Foxglove (*D. purpurea*), oleander (*Nerium oleander*), and lily of the valley (*Convallaria majalis*) are widely grown as ornamental plants in North America and have escaped to become established in the wild. Dogbane or Indian hemp (*Apocynum cannabinum*) is an indigenous plant containing cardiac glycosides but is rarely a problem to livestock. In Australia and southern Africa various Kalanchoe (*Bryophyllum* spp.) have killed cattle as a result of their cardiac glycoside content [11-15]. These succulent perennial plants are now sold in the United States as house plants or garden ornamentals and can be grown in the warmer, mild climates of the southern States where they have the potential of becoming a threat to animals that eat them [12,13]. Another exotic plant containing cardiac

glycosides with the potential of being introduced to North America is pheasant's eye (*Adonis microcarpa*) [16]. Only plants that pose a risk to livestock in North America will be discussed.

Other common cultivated plants that contain cardiac glycosides include: Hellebores (*Helleborus* spp.), squill (*Urginea* spp.), *Euonymus* spp., Star of Bethlehem (*Ornithogalum* spp.), lily of the valley (*Convallaria majalis*), oleander (*Nerium* spp.), blue eyed grass (*Sisyrinchium* spp.), hyacinth (*Hyacinthium* spp.) and periwinkle (*Vinca* major).

Toxicity of Cardiac Glycosides

Cardiac glycosides are found in all plant parts, especially the leaves. Generally, only very small quantities of the plants must be ingested to produce poisoning. Drought and freezing temperatures may cause livestock to consume more of the toxic plants [17]. In cattle and horses, as little as 0.005 percent body weight of green oleander leaves is reportedly lethal [7]. Oleander leaves administered experimentally via nasogastric tube at 40 to 80 mg/kg body weight consistently caused gastrointestinal and cardiac toxicosis [18]. Although reduced, toxicity is retained in the dried plants.

Animals consuming plants containing cardiac glycosides develop primarily heart and digestive disturbances before death [19]. The glycosides act directly on the gastrointestinal tract causing hemorrhagic enteritis, abdominal pain, and diarrhea [7,20-22]. Cardiac glycosides are cardenolides (a steroid nucleus with an attached lactone group) that inhibit the cellular membrane sodium-potassium pump (Na⁺K⁺ adenosine triphosphatase [ATPase] enzyme system) with resulting depletion of intracellular potassium and an increase in serum potassium [3,23]. This results in a progressive decrease in electrical conductivity through the heart causing irregular heart activity and eventually completely blocking cardiac activity. In low doses, the glycosides have a beneficial therapeutic effect on the heart by increasing the force of contraction, slowing the heart rate, and increasing cardiac output. Toxic doses of the glycosides cause a variety of severe dysrhythmias and conduction disturbances through the myocardium that results in decreased cardiac output and death. Cattle on rations containing ionophore feed additives such as monensin have increased susceptibility to the cardiac glycosides.

Clinical Signs of Poisoning

Cattle especially, and less often horses consuming cardiac glycoside-containing plants are often found dead because of the profound cardiac effects of the toxins. A variety of cardiac arrhythmias and heart block, including ventricular tachycardia and first- and second-degree heart block, may be encountered with cardiac glycoside poisoning [18].

Abdominal pain (colic) and diarrhea are also signs commonly seen in animals poisoned with cardiac glycosides. If observed early in the course of poisoning, animals will exhibit rapid breathing, cold extremities, and a rapid, weak, and irregular pulse. The duration of symptoms rarely exceeds 24 hours before death occurs. Convulsions before death are not common.

In acute poisoning from cardiac glycosides as characterized by oleander poisoning, the postmortem findings include hemorrhages, congestion, edema, and cell degeneration of the organs of the thoracic and abdominal cavities. In less acute but fatal poisoning, multifocal myocardial degeneration and necrosis is often present [24].

Treatment

No specific treatment is available for counteracting the effects of the cardiac glycosides. Gastric lavage or vomiting should be induced in dogs and cats as soon as possible. Cattle and horses should be given adsorbents such as activated charcoal (2 - 5 g/kg body weight) orally to prevent further toxin absorption [13]. In ruminants known to have eaten oleander, a rumenotomy to remove all traces of the plant from the rumen may be lifesaving. The cardiac irregularities may be treated using antiarrhythmic drugs such as potassium chloride, procainamide, lidocaine, dipotassium EDTA, or atropine sulfate [1,3,13]. The use of fructose-1,6-diphosphate (FDP) has effectively reduced serum potassium levels and irregularities of the heart and will improve cardiac function in dogs experimentally poisoned with oleander [25]. The mechanism of action of FDP is not known, but it apparently restores cell membrane Na⁺ and K⁺ ATPase function [25]. Because hyperkalemia is a common feature of oleander poisoning, the use of potassium in intravenous fluids should be avoided and serum potassium levels should be monitored closely.

Intravenous fluids containing calcium should not be given because calcium augments the effects of the cardiac glycosides. Poisoned animals should be kept as quiet as possible to avoid further stress on the heart. The use of digoxin-specific antibodies to treat digoxin toxicity, although possible in humans, has not found application in animal poisoning as yet [26].

Diagnosis

A diagnosis of cardiac glycoside poisoning may be made if an animal is found dead and evidence indicates that the animal

had access to plants known to contain cardiac glycosides. Similarly, detection of cardiac dysrhythmias, heart block, and ventricular escape rhythms is suggestive of cardiac glycoside or grayanotoxin poisoning. Detection of cardiac glycosides in the serum, urine, tissues, and stomach contents is possible using high-performance liquid chromatography [27,28]. Oleander glycosides will cross-react with digoxin radioimmunoassays [29].

Postmortem findings in oleander poisoning may include focal pale areas and hemorrhages in the myocardium. Multifocal areas of necrosis and hemorrhage may be seen microscopically [18]. The clinical cardiac abnormalities, sudden deaths and the lesions present in the heart typical of cardiac glycoside toxicity also closely mimic poisoning due to monensin, a feed additive in cattle rations.

Dogbane, Indian Hemp

Apocynum cannabinum - Apocynaceae (Dogbane family)

Spreading Dogbane

A. androsaemifolium

Habitat

Dogbane is a common plant of open spaces, especially along streams, irrigation ditches, and roadsides throughout North America. Spreading dogbane is more common at higher altitudes.



Habitat of Dogbane, Indian Hemp - Spreading Dogbane. *Apocynum cannabinum* - Apocynaceae (Dogbane family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Perennial, erect herbs grow from spreading root stalks. The stems are red, smooth, tough, branched, and 4 to 6 feet (2 to 3 meters) in height. The plant contains a white milky sap. The leaves are opposite, oblong in shape, hairless, and entire margined. The small green-white flowers are produced in clusters at the ends of the branches. The fruits are characteristically 3 to 8 inches (10 to 20 cm) long, narrow pods hanging in pairs (Fig. 2-1A) The seeds are long and narrow, each having a tuft of white hairs similar to milkweed seeds.

Spreading dogbane is a smaller plant up to 3 feet (1 meter) in height, with larger pink flowers (Fig. 2-1B).



Figure 2-1A. Dogbane showing distinctive long double seed pods (*Apocynum cannabinum*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-1B. Spreading dogbane (*A. androsaemifolium*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Dogbanes contain a resin, apocynin, and two known glycosides, cymaritin, and apocynein. Livestock are rarely poisoned by dogbane, and eat these plants only when they lack other forages.

Lily of the Valley

Convallaria majalis - Liliaceae (Lily family)

Although this plant is not indigenous to North America, it is commonly planted as ground cover in shady gardens. It is a hardy plant and when abandoned can escape to establish large stands. The plant is potentially toxic to animals if they are carelessly allowed to graze it or are fed garden clippings.

Description

Lily of the valley is a perennial plant arising from a deep underground rhizome. The plant forms dense spreading colonies. The leaves are hairless, glossy, green, parallel-veined, and sheath the flower stem. The white, fragrant, drooping, bell-shaped flowers are on a raceme (Fig. 2-2A). The fruits ripen into conspicuous red berries (Fig. 2-2B).



Figure 2-2A. Lily of the valley (*Convallaria majalis*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-2B. Fruits of Lily of the valley. - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin The cardiac glycosides (cardenolides) convallerin and convallamarin amongst at least 15 others, are found throughout the plant and have similar cardiac effects to digitalis glycosides. The seeds have the highest concentration of cardenolides, but the flesh of the fruit is minimally toxic. The skin of the fruit and the flowers also contain saponins that cause abdominal pain and diarrhea. Poisoning has been reported in dogs [30].

Foxglove

Digitalis purpurea - Scrophulariaceae (Figwort family)

Habitat

A native plant of Europe, the foxglove was introduced to North America where it has escaped cultivation and now is quite widespread in the northwest. It prefers rich soils growing along roadsides, fences, and disused areas.



Habitat of Foxglove. *Digitalis purpurea* - Scrophulariaceae (Figwort family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Foxglove is a perennial herb growing 3 to 6 feet (1 to 2 meters) tall with alternate toothed, hairy, basal leaves. The characteristic purple or white tubular pendant flowers with conspicuous spots on the inside bottom surface of the tube (Fig. 2-3A and Fig. 2-3B).



Figure 2-3A. Foxglove plant in bloom (*Digitalis purpurea*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-3B. Foxglove shows the characteristic purple spots in the flowers (*D. purpurea*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Several cardiac glycosides, the most important of which are digoxin, digitoxin, and digitonin, are found in all parts of the plant, and especially in the seeds. Livestock are infrequently poisoned, but will eat the plant occasionally either fresh or in hay. All spears of digitalis and their hybrids should be considered toxic until proven otherwise.

Oleander

Nerium oleander - Apocynaceae (Dogbane family)

Habitat

Introduced from the Mediterranean area, oleander is an evergreen showy flowering shrub, growing commonly from California to Florida. It is drought tolerant and is extensively used in landscaping along highways. Oleander is also grown as a potted house plant in northern climates.



Habitat of Oleander. *Nerium oleander* - Apocynaceae (Dogbane family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Oleander is a perennial, evergreen shrub or small tree up to 25 feet (10 meters) tall with whorled, simple, narrow, sharply pointed, leathery leaves 3 to 10 inches (6 to 20 cm) long. Two primary parallel veins run perpendicular to the mid rib of the leaf. The showy white, pink, or red flowers with five or more petals are produced in the spring and summer (Fig. 2-4A and Fig. 2-4B). Fruit pods contain many seeds, each with a tuft of brown hairs (Fig. 2-4C).



Figure 2-4A. Oleander (*Nerium oleander*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-4B. Oleander (*N. oleander*). White variety shows the characteristic appendages at base of the petals. - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-4C. Oleander pod and seeds. - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Oleandrin and neriine are two potent cardiac glycosides (cardenolides) found in all parts of the plant. Red flowered varieties of oleander appear to be more toxic. Oleander remains toxic when dry and is very poisonous to humans, many animals, and birds [7,17,18,20,22,31]. A single leaf can be lethal to a child eating it, although mortality is generally low in humans [32]. The lethal dose of the green oleander leaves for cattle and horses is 0.005 percent of the animal's body weight [7]. The minimum lethal dose of oleander for cattle is 50 mg/kg body weight [24]. Horses given 40 mg/kg body weight of green oleander leaves via nasogastric tube consistently developed severe gastrointestinal and cardiac signs of poisoning [18]. Livestock are usually poisoned when they are allowed to graze in places where oleander is abundant or when pruning are carelessly thrown into animal pens.

Yellow Oleander, Be-still Tree, Tiger Apple, Lucky Nut

(*Cascabela thevetioides*, *Thevetia thevetioides*) - Apocynaceae (Dogbane family)

Habitat

Native to tropical America, this plant is widely cultivated in the southern United States and Hawaii, and most tropical areas of the world.



Habitat of Yellow Oleander, Be-still Tree, Tiger Apple, Lucky Nut. (*Cascabela thevetioides*, *Thevetia thevetioides*) - Apocynaceae (Dogbane family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Yellow oleander is a perennial branched shrub or tree growing to 30 feet (12 meters) tall with dark green, glossy, alternate linear leaves up to 6 inches (15 cm) long and 0.5 inch (1 to 2 cm) wide, with milky sap. The yellow showy, tubular flowers are produced in clusters at the ends of branches (Fig. 2-5A). Fruits are fleshy drupes turning yellow to black when ripe.

Thevetia peruviana is a similar, but smaller species that is a large shrub or small rounded tree growing to 15 feet (7 meters) in height. The leaves are light green and glossy, often rolling under at the edges. The funnel-shaped flowers are smaller than *T. thevetioides* and orange-yellow to salmon-pink in color depending on the variety (Fig. 2-5B). The spherical fruits have rounded corners and are green, turning black when ripe. Each fruit contains two black seeds.



Figure 2-5A. Yellow oleander (*Thevetia thevetioides*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-5B. Yellow oleander (*T. peruviana*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Thevetin A and B and thevetoxin are potent cardiac glycosides found in all parts of the plant and are concentrated in the fruits [33,34].

Milkweeds

Asclepias spp. - Asclepiadaceae (Milkweed family)

Habitat

Milkweeds are widely distributed throughout the world and much of North America. They cause poisoning in sheep, goats, cattle, horses, and domestic fowl [8,35-38]. Greatest losses have occurred in sheep on western rangeland, but all animals are susceptible to poisoning especially when other forages are scarce or milkweeds are incorporated in hay [8,39]. Milkweeds grow in open areas along roadsides, waterways, and disturbed areas, preferring sandy soils of the plains and foothills. Overgrazing will enhance the encroachment of milkweeds.

Description

Milkweeds are erect perennial herbs that have either 6 to 12 cm broad veined leaves or narrow linear leaves less than 0.5 inches (1 cm) wide, arranged either alternately or in whorls (Fig. 2-6A and Fig. 2-6B). Most species (except *A. tuberosa*) contain a milky sap or latex (Fig. 2-6C). The flowers are produced in terminal or axillary umbels consisting of two, five-parted whorls of petals, the inner one being modified into a characteristic hornlike projection (Fig. 2-6D) The color of the flowers varies among species from greenish white to red. The characteristic follicle or pod contains many seeds each with a tuft of silky white hairs that aids in its wind-borne dispersal.



Figure 2-6A. Showy milkweed (*Asclepias speciosa*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-6B. Whorled milkweed flowers and narrow leaves (*A. subverticillata*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-6C. Butterfly weed (*A. tuberosum*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-6D. Woolypod milkweed (*A. eriocarpa*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Although milkweeds characteristically have a milky sap that is readily seen when the stem, leaves, or pods are ruptured, they should be differentiated from other plant genera that also have milky sap. These include the spurges (*Euphorbia* spp.), dogbane (*Apocynum* spp.), and wild lettuce (*Lactuca* spp.). The presence, therefore, of a milky sap or latex is not limited to milkweeds.

Principal Toxin

Asclepias species contain various toxic cardenolides (cardiac glycosides) [40-42]. Acute death from milkweed poisoning results from the cardiotoxic effects of the cardenolides that act like ouabain, a digitalis glycoside [43]. The cardenolides act by inhibiting Na^+K^+ -ATPase, thereby affecting myocardial conduction and contractility [42,43]. Milkweeds are most toxic during rapid growth and retain their toxicity even when dried in hay. Toxicity varies with the species and growing conditions [44]. However, all milkweeds should be considered potentially poisonous, especially the narrow-leafed species (see Fig. 2-6B and Fig. 2-6E). The highest concentration of cardenolides occurs in the latex, with the lowest concentrations in the roots [44].



Figure 2-6E. One of the smallest toxic milkweeds (*A. pumilla*). - To view this image in full size go to the IVIS website at www.ivis.org . -

The relative toxicity of the more common milkweeds is shown in Table 2 - 1. Fatal poisoning of an adult horse (450 kg) may occur with the ingestion of as little as 1.0 kg of green milkweed plant material [36]. As little as 0.1 to 0.2 percent body weight of plant on a dry matter basis of *A. labrifloris* and *A. subverticillata*, respectively, induced fatal poisoning in sheep [37]. In addition to the cardiotoxic effects of the cardenolides common to most milkweeds, other glycosides and resinoids identified in milkweeds have direct effects on the respiratory, digestive, and nervous systems causing dyspnea, colic and diarrhea, muscle tremors, seizures, and head pressing [36,37].

The presence of cardenolides in milkweeds, as many as 20 in *A. eriocarpa*, is apparently a defense mechanism for the plant to discourage most animals and insects from feeding on the plant [44,45]. Some insects, however, including the larvae of the monarch butterfly (*Danaus plexippus*) have the ability to feed on milkweeds and store the cardenolides in their own tissues as a protective mechanism [40,44,45]. The adult monarch butterfly retains the cardenolides as a defense mechanism. Birds that feed on the monarch butterfly that has fed on toxic milkweeds will experience the intense emetic effects of the cardenolides, and by association, avoid eating the insect in the future.

Clinical Signs

Signs of poisoning usually begin within 8 to 10 hours of the milkweed plants being eaten; the severity of symptoms depends on the quantity of plant consumed. In acute milkweed poisoning the animal may be found dead without any prior symptoms.

Poisoned sheep show a labored and slow respiratory rate, pain and inability to stand, muscular tremors, staggering gait, a weak, rapid pulse, bloating, colic and dilated pupils prior to death [8,19,35,37]. A variety of cardiac dysrhythmias may be detected using electro- cardiography. Once recumbent, the poisoned animals exhibit periods of tetany and chewing movements [35,37]. Postmortem signs in animals poisoned by milkweeds consist of nonspecific congestion of the lungs,

stomach, and intestines, with hemorrhages present on the surfaces of the lungs, kidneys, and heart.

Table 2 - 1. Common Toxic Milkweeds [36,38]		
Common Name	Scientific Name	Toxicity *
Labriform milkweed	<i>Asclepias labriformis</i>	0.05
Western whorled milkweed	<i>A. subverticillata</i>	0.2
Eastern whorled milkweed	<i>A. verticillata</i>	0.2
Woolypod milkweed	<i>A. eriocarpa</i>	0.25
Spider antelopehorn milkweed	<i>A. asperula</i>	1 - 2
Plains or dwarf milkweed	<i>A. pumila</i>	1 - 2
Swamp milkweed	<i>A. incarnata</i>	1 - 2
Mexican whorled milkweed	<i>A. mexicana</i>	2.0
Showy milkweed	<i>A. speciosa</i>	2 - 5
Broad leaf milkweed	<i>A. latifolia</i>	1.0
Narrow-leaved milkweed	<i>A. stenophylla</i>	-
Butterfly Weed	<i>A. tuberosa</i>	1.5
Antelope horn milkweed	<i>A. viridis</i>	1
* Amount of green plant as a percent of the animal's body weight that is lethal.		

Treatment and Prevention

No specific treatment is available for milkweed poisoning. Animals that have not consumed a lethal dose of the plants recover over several days. Affected animals should be moved from the source of the plants and given fresh water, good quality hay, and shade. Sedatives, laxatives, and supportive intravenous fluid therapy with addition of potassium chloride, if carefully monitored, may help to control the severity of signs. Intravenous fluids containing calcium are contraindicated.

Because milkweeds generally tend to grow singly or in relatively small stands, they can be controlled by digging out individual plants or selectively spraying the plants with a herbicide such as 2,4-dichlorophenoxyacetic acid (2,4-D). Care should be taken during the hay-making process to avoid incorporating the narrow-leaved milkweeds in the hay.

Yew

Taxus species - Taxaceae (Yew family)

Habitat

Several species of yew grow naturally or as ornamentals in North America, generally preferring more humid, moist environments. Western yew (*T. brevifoliat*) and American yew (*T. canadensis*) are two indigenous species [46]. English yew (*T. baccata*) and Japanese yew (*T. cuspidata*) are commonly cultivated species in North America.

Description

Yews are evergreen shrubs or small trees with glossy, rigid, dark green, linear leaves 1.5 to 2 inches (4 to 5 cm) long with pointed ends, closely spaced on the branches. Inconspicuous axillary male and female flowers are produced on separate plants, forming showy red to yellow fruits (aril) containing a single seed (Fig. 2-7a).



Figure 2-7A. Japanese yew leaves and fruits (*Taxus japonicus*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Podocarpus (*Podocarpus macrophyllus*), a nonpoisonous tree found commonly in milder climates of the southern portion of North America, is frequently but incorrectly called yew. It has dark green lance-shaped leaves 3 to 4 inches (7 to 10 cm) long, and the fruits are greenish purple that help differentiate it from yew (Fig. 2-7B).



Figure 2-7B. Yew (left) and *Podocarpus* (right) leaves. - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

The toxicity of yews to humans and animals has been known for many years [46-48]. Yews contain a group of 10 or more toxic alkaloids, the most toxic of which are taxine A and B, collectively referred to as taxine. Taxine inhibits normal sodium and calcium exchange across the myocardial cells, depressing cardiac depolarization and causing arrhythmias [51,52]. Taxol is a different diterpenoid alkaloid found in *T. brevifolia* and other yews that has anticancer activity [51,52]. Yews also contain nitriles, ephedrine, and irritant oils [46,48]. All parts of the plant, green or dried, except the fleshy part of the aril surrounding the seed are toxic [47,48]. Livestock are frequently poisoned when fed prunings from cultivated yews. The highest concentration of the alkaloids is generally found in the leaves in winter time. All domestic animals including birds are susceptible to the cardiotoxic effects of the alkaloid. Adult cattle and horses have been fatally poisoned with as little as 8 to 16 oz of yew leaves or 0.1 to 0.5 percent of their body weight. Drying of the leaves does not appreciably decrease their toxicity [47]. As little as 200 g of dried leaves fed to a 550 kg steer proved fatal [49]. Animals generally will not eat yew if they are fed a balanced diet [50]. Interestingly, deer appear to be able to eat yew without problem.

Although not proven, the yew alkaloids are not apparently excreted through the milk of cows fatally poisoned [49]. However, it is wise to not use the milk from lactating animals that survive yew poisoning for at least 48 hours and to withhold animals from slaughter for 35 days [53].

Clinical Signs

Sudden onset of muscle trembling, incoordination, nervousness, difficulty in breathing, slow heart rate, vomiting, diarrhea, convulsions, and death are characteristic of yew poisoning in animals [46,50-62]. Sudden death may be the only observed sign in many cases [56]. Deaths may however occur several days after the yew was eaten.

No postmortem lesions are diagnostic of yew poisoning. Diagnosis must be based on eliminating other causes of sudden death, evidence of access to yew, and the presence of yew leaves in the rumen and stomach contents of the animal. Finely chewed leaves may have to be examined microscopically to positively identify them as yew. Identification of taxine from chewed plant material and rumen contents using mass spectrometry affords a more precise means of confirming yew poisoning [63,64].

There is no specific treatment or antidote for acute yew poisoning. If an animal is observed eating yew, immediate veterinary attention is indicated. Activated charcoal (2 g/kg body weight) and magnesium sulfate (2 g/kg body weight) as a cathartic should be given via stomach tube to decontaminate the rumen [53]. A rumenotomy to remove the yew leaves from the rumen of cattle in early confirmed cases of yew consumption may be lifesaving. Atropine sulfate is reportedly effective in counteracting the slow heartbeat and heart failure but should be used with caution [49]. When possible, intravenous fluid therapy and other supportive measures should be instituted to support the cardiovascular system [53].

Yews should never be planted as hedges around animal enclosures, and prunings from yews should not be given to animals.

Avocado

Persea americana - Lauraceae (Laurel family)

Habitat

Avocados are grown in southern areas of North America and throughout tropical areas of the world for their edible fruits. As early as 1942, avocado poisoning was reported in California, [65] and since then a variety of species including cattle, horses, goats, rabbits, canaries, budgerigars, cockatiels, ostriches, and fish have been poisoned by eating the leaves and fruits of the avocado tree [66-72]. The leaves, bark, seeds, and skin of the fruit are toxic. The leaves remain toxic when dried. Both the Guatemalan and the Mexican varieties of avocado are toxic to animals and birds, with most poisoning being associated with the consumption of the Guatemalan and Nabal varieties of avocado [73-75] (Fig. 2-8). There is some variability in toxicity between different varieties of avocado, the Mexican variety being considered the least toxic [74]. However, hybrid varieties of avocados are toxic to birds [74,75].



Figure 2-8. Avocado fruits (*Persea americana*). Guatemalan varieties have a dark rough skin. - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

The toxin present in avocados, as yet unidentified, has a direct toxic effect on the myocardium and tissues of the lactating mammary gland [72-75].

Clinical Signs

Horses develop edematous swelling of the lips, mouth, eyelids, head, and neck, which can cause upper respiratory distress [70]. Colic is seen in some horses. The serum enzymes creatine phosphokinase (CPK) and aspartate aminotransferase (AST) are generally elevated, indicating muscle necrosis and in particular myocarditis [70]. Edematous swelling of the head and neck area is suggestive of heart failure. Brisket and neck edema, and acute pulmonary edema as a result of cardiomyopathy and heart failure, have also been reported in rabbits and goats that have died from avocado poisoning [72]. The cardiomyopathy is manifested as an acute noninfectious degeneration and necrosis of myocardial tissue, which, if severe, will result in acute death of the animal [72-75].

Ostriches eating the leaves and immature fruits of the Guatemalan variety of avocado have developed fatal cardiomyopathy and congestive heart failure with 96 hours of eating the plants [69]. In other birds, avocado poisoning is characterized by respiratory distress and sudden death. Deaths usually occur 1 to 2 days after consumption of the avocado.

Cattle, horses, goats, and rabbits develop noninfectious mastitis after eating avocado leaves [72,74]. Goats fed as little as 31 g/kg body weight of avocado leaves showed dramatic reduction in milk production and developed hard swollen udders 24 hours after they had eaten the leaves. The milk was of a cheesy consistency and contained clots. The milk somatic cell counts also became markedly elevated. If no further avocado leaves were fed, the udder edema regressed and milk production returned, but not to the levels before feeding the avocado leaves [72]. The serum liver enzyme AST was increased in intoxicated goats [91]. Generalized necrosis of the mammary gland epithelium with no significant cellular inflammatory response and sloughing of necrotic cells are characteristic microscopic findings [72,75].

At postmortem examination, generalized congestion of the lungs, hydropericardium, and subcutaneous edema in the pectoral area are typical [68,69]. Microscopic examination of the tissues reveals scattered nonsuppurative inflammation in the liver, heart, and kidneys and eosinophilic material in the cytoplasm of many Kupffer cells [72-75]. Similar noninflammatory necrosis of the myocardium and lactating mammary gland were observed in goats and mice fed avocado leaves [73-75].

Horses and other livestock should not be allowed into avocado orchards and the trees should not be planted adjacent to livestock enclosures. The fruits and seeds should not be fed to pet birds.

Death Camas

Zigadenus spp. - Liliaceae (Lily family)

Habitat

Approximately 15 species of death camas occur in North America, their habitat ranging from moist mountain valleys to dry sandy hills and plains. Death camas appears in early spring, often growing among wild onion from which it can be readily differentiated on the basis of the smell.

Description

Death camas is a herbaceous, hairless perennial with grasslike, linear, V-shaped, parallel-veined leaves arising basally from an onion-like bulb 6 to 8 inches below the soil surface. The leaves are not hollow like those of onions, nor do they smell like an onion. The bulb is covered with a black membranous outer layer. The inflorescence, a terminal raceme or panicle, has small, perfect, greenish white to yellow flowers (Fig. 2-9A). A showy species is mountain death camas (*Z. elegans*) (Fig. 2-9B). The six perianth segments are separated, each with a gland at its base (Fig. 2-9C).

The ovary is superior or partly inferior forming a trilobed, capsuled seed pod.



Figure 2-9A. Death camas in bloom (*Zigadenus venenosus*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-9B. Showy death camas (*Z. elegans*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-9C. Showy death camas flowers (*Z. elegans*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Members of the genus *Zigadenus* contain several steroidal alkaloids, the best known being zygacine and zygadenine [76]. The alkaloids are similar to those found in false hellebore (*Veratrum californicum*) and decrease blood pressure by dilating arterioles, constricting veins, and slowing the heart rate. The plants vary considerably in their toxicity, with most toxic species seldom growing at elevations above 8000 feet. All parts of the plant are toxic, especially the bulbs, which have caused severe gastrointestinal disease, hypotension, and even death in people when mistakenly eaten as wild onion [77-79]. Sheep are most frequently poisoned by death camas, but cattle, horses, and pigs may be affected [80-85]. Deer and other wild ruminants may be more tolerant of the toxic effects of death camas [86]. Poisoning is most likely to occur in early spring when few other plants are available and the succulent shoots are especially enticing.

Clinical Signs

Sheep show signs of poisoning after eating as little as 0.5 lb of the green plants. Salivation, nausea, vomiting, muscular weakness, and staggering gait are typically seen. Convulsions, coma, and death soon occur if sheep eat 2 to 2.5 lb of green plant per 100 lb body weight [82-84]. No specific lesions are visible on autopsy.

Diagnosis of death camas poisoning is therefore usually made by eliminating other causes of sudden death, the presence of *Zigadenus* species in the animal's environment, and the detection of *Zigadenus* alkaloids in the rumen contents using thin-layer chromatography [76,85].

Treatment

In most cases of death camas poisoning, little can be done to reverse the toxicity of alkaloids. The subcutaneous injection of 2 mg atropine sulfate and 8 mg picrotoxin per 100 lb body weight is reported to be effective in treating early poisoning of sheep [86,87]. Supportive therapy with intravenous fluids is helpful in combating the hypotensive effects of the death camas. Bloated animals should be kept in a sternal position and a stomach tube passed to relieve rumen pressure.

Keeping animals out of the areas where the plant is present, especially in the early spring when other forages are not available, can prevent poisoning from death camas. Herbicides can be selectively used to control dense stands of the death camas in problematic areas.

Plants Causing Similar Clinical Signs

Similar signs and death losses have occurred in sheep after eating fly poison or stagger grass (*Amianthium muscaetoxicum*, synonym *Chrosperma muscaetoxicum*), a plant similar in appearance to death camas, and found in the eastern United States from New York to Florida, and west to Arkansas [87]. Fly poison has basal grasslike leaves and showy white flowers borne on multiple dense racemes (Fig. 2-10A and Fig. 2-10B).

It prefers moist soils, emerging in the early spring when it can induce death in cattle and sheep that graze it before other forages are available. The name fly poison was given to this plant because at one time the ground up bulb was used as a fly poison.



Figure 2-10A. Fly poison or stagger grass plant in bloom (*Amianthium muscaetoxicum*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2-10B. Fly poison or stagger grass flowers (*A. muscaetoxicum*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Star of Bethlehem (*Ornithogalum umbellatum*) was introduced from the Mediterranean area and has become established as a common pasture plant of eastern North America. This onion-like perennial herb, growing from a bulb, forms clumps of showy white star-shaped flowers (Fig. 2-11). The bulbs appear to be the most toxic and result in rapid death of animals, particularly sheep, that consume them [88]. Members of the genus *Ornithogalum* are some of the most poisonous plants found in South Africa [89].



Figure 2-11. Star of Bethlehem (*Ornithogalum umbellatum*). - To view this image in full size go to the IVIS website at www.ivis.org . -

The Atamasco or rain lily (*Zephyranthes atamasco*) found in wooded areas or pastures of southeastern North America has been associated with deaths in cattle and horses [90]. The 1 inch (2.5 cm) diameter bulb is the most poisonous part of the plant. Several bluish green leaves 6 to 10 inches (15 to 25 cm) long emerge from the bulb in the spring and are followed by showy star-shaped, white flowers (Fig. 2-12). The faded flowers turn pink. Within 24 hours of eating the bulbs, affected animals develop hemorrhagic diarrhea, staggering gait, and coma before death.



Figure 2-12. Atamasco or rain lily (*Zephyranthes atamasco*). - To view this image in full size go to the IVIS website at www.ivis.org . -

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