Plants Affecting the Skin and Liver  
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Plants that affect the skin and liver are considered together in this chapter because some plant toxins cause liver disease that results in secondary skin disease. One of the most noticeable signs indicative of an animal with liver disease is the appearance of dermatitis and hair loss affecting the white-skinned areas. Some plants contain compounds or pigments that once absorbed from the digestive system induce a direct effect on nonpigmented skin when it is exposed to light, without any effect on the liver. Other plants contain toxic alkaloids that cause irreversible liver disease resulting in a secondary photosensitization. Yet other plants have compounds that cause a skin reaction when they come in contact with skin, or they have spines or thorns that can cause mechanical injury to the skin.

Photosensitization

Photosensitization, resembling but distinct from sunburn, is a severe dermatitis of animals resulting from a complex reaction induced by plant pigments exposed to ultraviolet (UV) wave length sunlight in the skin of animals that have eaten certain plants [1-3]. This reaction is most severe in nonpigmented skin where these reactive compounds are most directly exposed to light in the UV spectrum [2]. The precise mechanism of this reaction is unknown, but it is thought to be a light-enhanced oxidation reaction [2]. The amino acids (histidine, tyrosine, tryptophan) are particularly susceptible to oxidation and once oxidized evoke an intense inflammatory response in the blood vessels and surrounding cells that results in tissue necrosis. In addition to plant pigments, fungal toxins, chemicals, and occasionally congenital diseases affecting porphyrin metabolism in the liver may induce photosensitization [2]. Quite frequently horses and cattle develop photosensitization while on pasture with no determinable cause.

Photosensitization may be conveniently classified into two basic types - primary and secondary. Primary photosensitization is associated with photodynamic compounds in certain plants, which once absorbed from the digestive tract, react in the nonpigmented with UV light to cause a severe dermatitis. Also in this category are the congenital photosensitivity diseases associated with defective pigment (porphyrins) metabolism in the liver of animals. Secondary or hepatogenous photosensitization, as the name implies, results when an animal's liver is sufficiently diseased to be unable to remove plant...
by-products that can react with UV light to cause photosensitization. Phylloerythrin, a bacterial breakdown product of chlorophyll, is the photosensitizing compound [2]. Normally phylloerythrin is removed by the liver and is excreted in the bile, but if the liver is severely diseased, it accumulates in the blood to cause photosensitization if a white skinned animal is exposed to UV light. Hepatogenous photosensitization can be further subdivided into that attributable to liver disease as opposed to that caused by biliary system disease that causes a backup of bile. Secondary photosensitization is much more common in livestock than primary photosensitization, and because of the severity of the underlying liver disease, it always carries a poor prognosis.

Occasionally photoreactive pigments (porphyrins), produced in animals as a result of normal hemoglobin breakdown, accumulate and cause photosensitization [4]. Congenital porphyria is an inherited defect in various breeds of cattle as a result of a specific enzyme deficiency that normally regulates metabolism of porphyrins [5]. Southdown sheep may also develop photosensitization due to a congenital defect in the liver's ability to excrete the photoreactive compound phylloerythrin [6]. As it accumulates in the skin, phylloerythrin causes photosensitivity when the animal is exposed to sunlight. Chemicals such as phenothiazine sulfoxide, a derivative of the anthelmintic phenothiazine, may also produce photosensitivity in ruminants if they are exposed to sunlight after treatment with phenothiazine [2].

**Primary Photosensitization**
Primary photosensitization develops when animals eat plants containing polyphenolic pigments. These compounds are at highest concentration in the green plant and are readily absorbed from the gastrointestinal tract to circulate in the blood. In nonpigmented skin these compounds react with UV light to produce radiant energy that oxidizes essential amino acids in the skin's cells. The cells die in the photosensitization process, and the affected skin eventually sloughs off. Two plants associated historically with primary photosensitization are buckwheat (Fagopyrum esculentum), [1] and St. John's wort (Hypericum perforatum) [8-10]. Both plants contain polyphenolic pigments capable of causing primary photosensitization. Several plant species including bishop's weed (Ammi majus), spring parsley (Cymopterus watsonii), and Dutchman's breeches (Thamnosma texana) contain photodynamic furanocoumarin compounds that have been associated with photosensitivity through ingestion and direct contact with the skin [11-13] (Table 4 - 1). In southeast Texas, a seasonal photosensitivity of cattle is associated with the consumption of the dead leaves of Cooperia pedunculata, a lily of the Amaryllis family [14,15]. Photosensitivty has also been reported in Europe as a result of exposure to giant hogweed (Heracleum mantegazzianum). Cow parsnip (Heracleum spp.), which occurs in North America, has the potential to cause photosensitivity.

The detection of primary photoreactive compounds in plants can be accomplished using a screening test that is based on the sensitivity of the fungus *Candida albicans* to irradiation [16]. The simple procedure involves exposing suspect plant material on agar plates seeded with *C. albicans* to UV light. Photoreactive plants will inhibit the growth of the *C. albicans*.

<table>
<thead>
<tr>
<th>Table 4 - 1. Primary Photosensitizing Plants</th>
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<tbody>
<tr>
<td><strong>Botanical Name</strong></td>
</tr>
<tr>
<td>Ammi majus</td>
</tr>
<tr>
<td>Cooperia pedunculata</td>
</tr>
<tr>
<td>Cymopterus watsonii</td>
</tr>
<tr>
<td>Fagopyrum esculentum</td>
</tr>
<tr>
<td>Heracleum mantegazzianum</td>
</tr>
<tr>
<td>Hypericum perforatum</td>
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<tr>
<td>Thamnosma texana</td>
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**Secondary Photosensitization**
Secondary or hepatogenous photosensitization in animals occurs more commonly than primary photosensitization. Liver disease, the underlying cause of secondary photosensitivity, results from ingestion of plants containing compounds toxic to the liver. A variety of compounds toxic to the liver are found in plants, the most important of which are the pyrrolizidine
alkaloids (PAs). Once 80 percent or more of the liver is destroyed by these alkaloids, it is unable to eliminate phylloerythrin, a bacterial breakdown product of chlorophyll. Phylloerythrin then accumulates in the blood, and as it circulates through the skin and is exposed to UV light, it fluoresces and causes oxidative injury to the blood vessels and tissues of the skin [2,7]. The resulting intense inflammatory response is most severe in the nonpigmented skin. In severe cases of PA poisoning, acute liver failure and death may result before signs of photosensitization have time to develop.

Secondary photosensitization is also caused by a variety of plant toxins other than pyrrolizidine alkaloids (see Table 4 - 3). In the western range lands of North America, photosensitization in sheep (bighhead) has for many years been attributed to the grazing of horsebrush (Tetradymia glabrata and T. canescens) [17,18]. It has been shown, however, that sheep are much more susceptible to horsebrush photosensitivity if they concurrently browse on black sage (Artemisia nigra) or big sage (A. tridentata) or both [17,18]. Horsebrush and black and big sage frequently grow in the same locations in western rangelands, and when eaten together have a synergistic effect in causing photosensitivity. These plants including Lantana camara, Agave lecheguilla, Tribulus terrestris, and Panicum grass species cause secondary photosensitization through inflammation and obstruction of the biliary system.

Pyrrrolizidine Alkaloid Poisoning

Pyrrolidine alkaloids are found in many species of the three major plant families Compositae, Fabaceae, and Boraginaceae [19] (Table 4 - 2). The important plant genera that cause liver disease and secondary photosensitization in North America are Senecio, Crotolaria, Cynoglossum, and Amsinckia. The most notorious PA-containing plants are those belonging to the genus Senecio, long known to cause liver disease in man and animals that eat them. Most PA poisoning of livestock in the western United States is attributable to three species of Senecio: tansy ragwort (S. jacobaea), threadleaf or wooly groundsel (S. douglasii var. longilobus), [20,21] and Riddell's groundsel (S. riddellii) [22,23]. Throughout the world, Senecio species are also the most common cause of PA poisoning [24-28]. Severe economic losses to the livestock industry due to Senecio spp. poisoning have been estimated to exceed $1.2 million annually in the northwestern Pacific states alone [29].

<table>
<thead>
<tr>
<th>Scientific Name</th>
<th>Common Name</th>
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<tbody>
<tr>
<td>Amsinckia spp.</td>
<td>Fiddle neck, tarweed</td>
</tr>
<tr>
<td>Crotolaria spp.</td>
<td>Rattle box</td>
</tr>
<tr>
<td>Cynoglossum officinale</td>
<td>Hound' tongue</td>
</tr>
<tr>
<td>Echium vulgare</td>
<td>Blue weed, viper's bugloss</td>
</tr>
<tr>
<td>Heliotropium spp.</td>
<td>Giant hog weed</td>
</tr>
<tr>
<td>Hypericum perforatum</td>
<td>Heliotrope</td>
</tr>
<tr>
<td>Senecio spp.</td>
<td>Groundsels, Senecio</td>
</tr>
<tr>
<td>Symphytum officinale</td>
<td>Comfrey</td>
</tr>
</tbody>
</table>

Other important plant species contain PAs including Crotolaria spectabilis, a plant introduced to the southeastern United States that is known for the effects of the PA, monocrotaline, found in the seeds. In man, monocrotaline induces severe veno-occlusive disease and pulmonary hypertension by causing hypertrophy of the smooth muscle of the pulmonary arteries [30]. In horses, cattle, pigs, and monkeys, Crotolaria causes severe liver and lung disease [31-36]. Fiddleneck (Amsinckia intermedia), and hound's tongue (Cynoglossum officinale), members of the Boraginaceae, have significant quantities of PA capable of causing secondary photosensitization in horses and cattle [37-41]. In other parts of the world, species of heliotrope (Heliotropium spp.), salvation Jane (Echium plantagineum), and Trichodesma species are important causes of PA poisoning [42-47]. Blue weed or viper's bugloss (Echium vulgare) is a common introduced weed of northeastern North America that contains PA and has the potential for causing livestock poisoning.

Human poisoning from PA occurs when people regularly drink teas and remedies containing groundsel (Senecio spp.) [48].
Gordolobo yerba, a harmless herbal tea, is sometimes mistakenly made from *Senecio longilobus* that contains significant levels of PA. Consequently people and especially children fed the tea over time develop venoocclusive liver disease that can be fatal [49,50]. Teas made from comfrey (*Symphytum officinale*) are also potentially toxic because they contain at least three PAs that have been demonstrated to cause hepatogenic carcinoma in rats [51,52].

A wide range of animal species including wild and domesticated ruminants, horses, and pigs are susceptible to PA poisoning [27,37,38,53]. Pyrrolizidine alkaloids are readily absorbed from the digestive tract and are bioactivated to toxic pyrroles and possibly other reactive metabolites by the liver's mono-oxygenase system [54-57]. It is the active pyrroles that affect the endoplasmic reticulum of the liver cells inhibiting mitosis and the replication of hepatocytes [54,55]. Animals experiencing poor nutrition, pregnancy, and other metabolic stress are more susceptible to PA poisoning [58]. At high doses, PAs cause hepatocellular necrosis, while at lower doses necrosis is less severe allowing time for the characteristic pathologic changes of megalocytosis, bile duct hyperplasia, and fibrosis to occur [56,60,61]. Similar changes may also be seen in the kidneys [55]. In low doses PAs cause endothelial changes in the capillaries of the lungs with resulting pulmonary hypertension and right heart failure [30,61,62]. In the case of monocrotaline found in *Crotolaria* species, the highly toxic metabolite dihydromonocrotaline not only affects the liver, but also the lung and possibly other tissues [63]. Horses develop an acute fatal fibrosing alveolitis after eating feed contaminated with crotolaria seeds [64].

The PAs have been reported to be carcinogens, teratogens, and abortifacients [65]. Feeding toxic quantities of *S. jacobaea* to pregnant cows through the 15 to 30th days of gestation causes no detectable changes in the fetus, which may indicate PAs are not teratogenic in early gestation [21]. The PAs are secreted in the milk of cows and in very low quantities can cause mild liver changes in calves and kids consuming the milk [66-70]. The effects of PAs on people consuming milk containing PAs have not been established. Bees feeding on tansy ragwort (*S. jacobaea*), and Paterson's curse (*Echium plantagineum*) produce honey containing PAs that is potentially hazardous to those consuming it [46].

Variation in the PA content of plants, the quantity of plant eaten, and individual animal species susceptibility affect the severity of poisoning seen in animals. The PA content of plants varies considerably, generally increasing with maturation of the plant and reaching a maximum just before the flower buds open [71]. Flowers tend to contain the greatest amount of the alkaloid, and seeds of *Crotolaria* and *Amsinckia* concentrate high levels of PA [64,66]. *Senecio redellii*, when near maturity, has been reported to contain exceptionally high levels of PA (10 - 18 percent dry weight) [72]. The PA content of plants incorporated in hay remain stable for months, but appear to be largely degraded in properly prepared silage [73]. The stability of PAs in plants cured in hay can be a significant cause of poisoning in mid winter when plant toxicity may not be suspected.

Pigs are the most susceptible to PA poisoning, followed by poultry, cattle, horses, goats, and sheep [59,74]. Sheep can eat approximately 20 times the amount of *Senecio* it would take to poison a cow on an equivalent weight basis. This ability of sheep to tolerate greater amounts of PA is attributed to the presence of specialized rumen bacteria that can detoxify the alkaloids before they are absorbed [76]. It appears to be the smallest bacteria and possibly the protozoa in the rumen that are the primary detoxifiers of PAs [76,77]. Cattle and horses are highly susceptible to tansy ragwort (*S. jacobaea*), a lethal dose being 4 to 8 percent (0.05 to 0.2 kg/kg body weight) of their body of green plant [41,78]. Others have produced fatal poisoning in cattle by feeding as little as 0.2 lb dry weight of *S. jacobaea* per day for 4 weeks [57,71,74,80]. Sheep and goats, however, are quite tolerant of PA poisoning, requiring 200 to 300 percent of their body weight in green tansy ragwort to develop fatal poisoning [10,78,80]. Goats will abort, however, if they consume 1 percent of their body weight per day of dry *S. jacobaea* [81]. The chronic lethal dose of *S. jacobaea* in goats ranges from 1.2 to 4.04 kg/kg body weight. The toxicity of hound's tongue (*C. officinale*) varies considerably, ranging from 15 to 360 mg/kg body weight per day of dried plant [38,41]. The PA content of hound's tongue averages 0.8 percent in the early blossom stage [41]. This variation is possibly due to the stage of growth of the hound's tongue, and differences in the age of animals, with young animals being more susceptible to PA toxicity.

Animals will not readily eat plants containing PA unless they are forced to do so through lack of normal forage. Dryed plants with only minimal reduction in the alkaloid content are, however, quite palatable, making them a particular risk when present in hay. The effects of the PA are cumulative so that symptoms of liver disease and photosensitization may not appear for many months after animals have eaten toxic quantities of PA. This makes identification of the suspected toxic plants difficult, because the plants will often not be present in the pasture or hay when liver disease and photosensitization become evident in the animal.
**Biliary Oclusive Photosensitization**

Photosensitivity in livestock has been attributed to ingestion of various plant species other than those containing primary photosensitizing compounds (see Table 4 - 1) or those causing liver disease as a result of pyrrolizidine alkaloids (see Table 4 - 3). Some of these photosensitizing plants contain saponins that cause inflammation and obstruction of the biliary system. When bile cannot be excreted normally by the liver, photosensitizing compounds will accumulate in the animal's bloodstream and will result in photosensitization. A prime example of this is facial eczema, a disease of sheep principally in New Zealand and Australia. It is caused by the mycotoxin sporodesmin produced by the fungus *Pithomyces chartarum* growing on wet rye grass pastures. Sheep and cattle eating the affected rye grass excrete the sporodesmin in the bile, which causes a cholangitis and biliary occlusion. This in turn results in the accumulation of phylloerythrin and subsequently photosensitization [81]. Sheep eating puncture vine (*Tribulus terrestris*) develop photosensitivity secondarily to biliary obstruction that was initially thought to be caused by a mycotoxin, but it is now shown to be the result of steroidal saponins in the plant [82-84]. A similar biliary occlusive photosensitivity occurs in sheep that graze on Kleingrass (*Panicum coloratum*) during hot humid weather [85,86]. Agave poisoning (*Agave lecheguilla*), bear grass (*Nolina texana*) in North America, and alveld, a photosensitivity of sheep in Great Britain and Western Europe due to bog asphodel (*Narthecium ossifragum*) are induced by plant saponins that form crystals in the bile ducts and occlude the biliary system [88-91]. Lantana (*Lantana camara*) also causes photosensitization through cholestasis, effectively preventing the elimination of phylloerythrin through the bile, and thereby causing photosensitization [90-93].

**Alsike Clover Poisoning**

Alsike clover (*Trifolium hybridum*) (Fig. 4-1) is a perennial legume that is commonly grown for livestock consumption in northern regions of North America. Two disease syndromes encountered in horses have been associated with the grazing of alsike clover.

![Figure 4-1. Alsike clover (*Trifolium hybridum*). - To view this image in full size go to the IVIS website at www.ivis.org . -](image)

The first is an irreversible liver disease often accompanied by neurologic disturbances and referred to as alsike clover poisoning [94-97]. The second syndrome is one of photosensitivity without apparent concurrent liver disease and is referred to as trifoliosis (dew poisoning) [94,96,98,99]. The specific toxins responsible for either of these syndromes have not been determined. A fungal toxin may be involved because alsike clover poisoning is often a problem in years when there is high rainfall and humidity [100].

Although evidence is insufficient to conclude that alsike clover (*T. hybridum*) is the primary cause of poisoning, a close association exists between the liver disease and photosensitivity syndrome seen in horses and the grazing of the plant [94]. Horses with alsike clover poisoning typically develop signs of liver failure including weight loss, jaundice, depression. Other neurologic abnormalities may develop. Significant elevations of specific liver enzymes are usually present and are indicative of severe liver disease [96,97]. The abnormal neurologic findings in affected horses are possibly due to hepatic encephalopathy caused by the failing liver. This can be substantiated by the elevation of blood ammonia levels that typically occur in advanced liver disease [96,97]. The appearance of photosensitivity can be attributed to the accumulation of phylloerythrin in the horse's circulation as a result of liver failure [95-97]. The primary lesion found in horses with alsike clover poisoning is a grossly enlarged, fibrotic liver, with marked bile duct proliferation and perilobular fibrosis [95,101].
<table>
<thead>
<tr>
<th>Scientific Name</th>
<th>Common Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agave lecheguilla</td>
<td>Agave</td>
</tr>
<tr>
<td>Avena sativa</td>
<td>Oats</td>
</tr>
<tr>
<td>Brachiaria decumbens</td>
<td>Signal grass</td>
</tr>
<tr>
<td>Bassia hysopifolia</td>
<td>Bassia</td>
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<tr>
<td>Brassica spp.</td>
<td>Rape, kale</td>
</tr>
<tr>
<td>Cenchrus spp.</td>
<td>Sandbur</td>
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<tr>
<td>Cynodon dactylon</td>
<td>Bermuda grass</td>
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<tr>
<td>Descurainia pinnata</td>
<td>Tansy mustard</td>
</tr>
<tr>
<td>Daucus carota</td>
<td>Wild carrot</td>
</tr>
<tr>
<td>Euphorbia maculata</td>
<td>Milk purslane</td>
</tr>
<tr>
<td>Hordeum spp.</td>
<td>Barley</td>
</tr>
<tr>
<td>Kalstroemia</td>
<td>Caltrops</td>
</tr>
<tr>
<td>Kochia scoparia</td>
<td>Kochia, Mexican fire weed</td>
</tr>
<tr>
<td>Lantana camara</td>
<td>Lantana</td>
</tr>
<tr>
<td>Lolium perenne</td>
<td>Perennial rye grass</td>
</tr>
<tr>
<td>Medicago sativa</td>
<td>Alfalfa</td>
</tr>
<tr>
<td>Microcystis spp.</td>
<td>Blue-green algae, water bloom</td>
</tr>
<tr>
<td>Narthecium ossifragum</td>
<td>Bog asphodel</td>
</tr>
<tr>
<td>Nolina texana</td>
<td>Sacahuiste</td>
</tr>
<tr>
<td>Panicum coloratum</td>
<td>Klein grass</td>
</tr>
<tr>
<td>Panicum spp.</td>
<td>Panic grasses</td>
</tr>
<tr>
<td>Pastinaca spp.</td>
<td>Parsnip</td>
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<tr>
<td>Psoralea spp.</td>
<td>Scurf pea</td>
</tr>
<tr>
<td>Polygonum spp.</td>
<td>Knottweed</td>
</tr>
<tr>
<td>Ranunculus bulbosus</td>
<td>Buttercup</td>
</tr>
<tr>
<td>Sorghum vulgare sudanensis</td>
<td>Sudan grass</td>
</tr>
<tr>
<td>Tetradyenia spp.</td>
<td>Horsebrush</td>
</tr>
<tr>
<td>Thamnosma texana</td>
<td>Dutchman's breeches</td>
</tr>
<tr>
<td>Tribulus terrestris</td>
<td>Puncture vine, caltrop</td>
</tr>
<tr>
<td>Trifolium spp.</td>
<td>Clovers</td>
</tr>
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</table>
**Mycotoxin Photosensitivity**
Photosensitization can develop in animals that eat moldy grains containing hepatotoxic mycotoxins (aflatoxins) produced by fungi belonging to the genera of *Aspergillus* and *Penicillium* [102]. Water-damaged alfalfa hay and moldy straw may also be implicated in photosensitivity of cattle probably as a result of mycotoxins that induce hepatitis and cholangitis [103-106]. Feeding moldy alfalfa hay and silage to cattle has been associated with a hepatogenous photosensitivity thought to be due to liver toxins produced by a variety of fungi cultured from the hay [107]. The unidentified toxins, however, appear to specifically affect cattle and not sheep, goats, horses, or mice fed the same hay, suggesting the toxin may be a rumen or liver metabolite unique to cattle [108].

**Blue-Green Algae Poisoning**
Blue-green algae or cyanobacteria may develop in large numbers in stagnant ponds and dams producing a "bloom" or green-blue discoloration of the water. Heavy growth of cyanobacteria usually occurs in late summer when water temperatures are high and the mineral or organic matter present in the water is also elevated. The primary cyanobacteria reported to cause poisoning in North America include members of the genera *Microcystis*, *Anabaena*, and *Aphanizomenon* [109-111]. As these organisms die they release potent toxins into the water that can cause severe poisoning in animals that drink the water [109-115]. The toxins have either primary neurotoxic effects with acute deaths, or they act on the liver to cause liver failure and photosensitization [110,111].

**Clinical Signs of Photosensitization**
Photophobia, excessive tearing, and swelling, redness, and increased sensitivity of nonpigmented skin initially characterize photosensitization in animals [116]. Frequently the skin around the lips, eyes, and coronary band of the hooves is most severely affected because it is heavily vascularized and poorly protected by hair or wool. White skin on the face, back, and legs of animals is particularly prone to photosensitization. White breeds of sheep often only develop lesions on the ears and face because of the protective fleece covering unless they have been recently sheered. Cows with nonpigmented udders may develop photosensitization of the teats that may be especially severe in wintertime when light is reflected off the snow. Affected skin rapidly becomes reddened, painful, and raised above areas of adjacent pigmented skin. Serum often oozes through the affected skin to form crusts in the hair. After 2 to 3 weeks, the necrotic skin becomes dry and parchment-like, and the hair and white skin slough leaving ulcerated areas that may develop secondary bacterial infections (Fig. 4-2A).

![Figure 4-2A](https://www.ivis.org) - Generalized photosensitization in a cow.

Lameness is common in horses, which have photosensitization involving the skin over joints and the coronet (Fig. 4-2B).

![Figure 4-2B](https://www.ivis.org) - Secondary photosensitization involving the white skinned areas of a horse's legs.

In cases of secondary photosensitization from PA poisoning, weight loss, abnormal neurologic signs, and typical photosensitization signs may be the first signs of disease noted. Other signs of liver disease including jaundice, abdominal distention due to ascites, diarrhea, tenesmus, and rectal prolapse may be evident [23,69,117-119]. Neurologic signs develop when the severity of the liver disease precludes the liver's ability to remove toxins from the animal's system. The accumulating toxins act on the brain interfering with normal function and causing its degeneration (hepatic encephalopathy). Horses and cattle with hepatic encephalopathy show abnormal behavior that may include yawning, drowsiness, aimless wandering, head pressing, incessant licking of objects, and terminal coma [26,116,117]. Hemolysis and hemoglobinuria from copper poisoning can occur in sheep and occasionally horses, associated with severe PA induced liver disease that results in
the release of copper stored in the liver [121]. Sheep, however, fed tansy ragwort until they developed severe PA poisoning did not develop copper poisoning, suggesting other mechanisms may be active in sheep [122]. In one instance horses died from an acute hemolytic crisis associated with the ingestion of tansy ragwort and the chewing of wooden fencing impregnated with a copper preservative [123]. Gastric impaction in ponies has been reported as an unusual finding in PA poisoning [124]. The prognosis for animals with secondary photosensitization is always poor because the liver disease is generally irreversible and affected animals eventually die from liver failure.

Diagnosis

Dermatitis limited to areas of nonpigmented skin is indicative of photosensitization. Identification of photosensitizing plants in the animal’s environment and food substantiates the diagnosis. Removal of the suspected plants from the diet with subsequent recovery of the animal suggests a primary photosensitization. Recovery is not likely to occur in cases of secondary photosensitization especially if due to PA toxicity because the underlying liver disease is usually irreversible, and signs of photosensitivity return as soon as the animal is exposed to sunlight.

The optimal test for the diagnosis of PA hepatotoxicity is a liver biopsy to confirm the presence of megalocytosis, fibrosis, and biliary hyperplasia, the classic signs of PA poisoning [125]. In early cases of poisoning, intranuclear vacuoles, biliary hyperplasia, and fibrosis are more prevalent, megalocytosis being more evident in chronic cases [126-128]. Laboratory tests to determine the presence of liver disease should be performed to confirm a diagnosis of secondary photosensitization. There are, however, no commonly available specific liver tests to confirm PA poisoning, and a profile of tests is necessary to help define the severity and duration of liver disease [128,129]. Elevation of serum enzymes glutamate dehydrogenase and sorbitol dehydrogenase (horses, cattle) are indicative of active liver necrosis due to PA poisoning [130]. Serum glutamate dehydrogenase is the first enzyme to increase and also the first to return to normal following PA poisoning [126]. Serum γ-glutamyltransferase (GGT) has both sensitivity and specificity to serve as a useful screening test for PA-induced liver disease because it remains elevated especially in the presence of bile duct disease [130,131]. Alkaline phosphatase is less specific but its elevation in conjunction with GGT adds to its usefulness as part of a screening profile [131]. Other enzymes and liver function tests are often elevated with PA toxicosis but are nonspecific and vary considerably depending on the stage of liver disease [132]. A decline in the ratio of the sum of serum branched-chain amino acids (leucine, isoleucine, valine) to the sum of phenylalanine and tyrosine is indicative of the severity of liver disease and can be used in the prognosis and treatment of PA poisoning in horses [133,134]. Measurement of serum bile acid levels in horses has been also shown to have predictive value because horses with serum bile acid levels of 50 µmol/L or greater are unlikely to survive [127]. Detection of metabolites of toxic pyrroles formed in the liver have been shown to alkylate tissue-bound thiol groups forming pyrrole thioethers [135]. These compounds can be extracted and identified by various chromatography methods after the PAs are undetectable, thus providing a means of positively identifying the residual metabolites of pyrrolizidine alkaloids even in formalin-fixed tissues [27,135-137].

Treatment

Animals showing signs of photosensitization should be provided with shelter from the sun and preferably kept stalled out of the sunlight. Further access to the photosensitizing plant should be prevented. Gentle daily cleaning of the affected skin with a mild antiseptic solution will aid in the healing process. Antibiotics may be indicated in animals with severe secondary bacterial dermatitis.

A variety of different ways have been proposed for treating and preventing PA toxicosis since the toxicity of the alkaloids was first recognized. Few if any have been proven to be effective in cattle and horses [138]. Antioxidants have shown some promise in research studies because they increase liver thiol levels [138]. Administration of cysteine, B vitamins, and synthetic antioxidants may have some protective effect against PA toxicosis, although they do not prevent liver damage due to the alkaloids [133]. However, feeding butylated hydroxyanisole, cysteine, and vitamin B had no detectable beneficial effect on PA poisoning in ponies [139]. Mineral and vitamin supplements have similarly failed to prevent tansy ragwort poisoning in cattle [140].

Symptomatic treatment should include a balanced high-energy, low-protein diet to avoid overloading the liver with nitrogenous compounds, and intravenous polyionic fluids with glucose in severely affected animals. In horses, the dietary supplementation of branched-chain amino acids leucine, isoleucine, and valine may result in clinical improvement by ensuring an adequate level of these essential amino acids [133,134].

Complete recovery from primary photosensitization usually occurs provided the animal does not return to eating the photosensitizing plant. However, secondary photosensitization due to PA poisoning has a poor prognosis because of the irreversible underlying liver disease. Symptoms of photosensitization usually do not appear until at least 80 percent of the liver is affected by the disease process. Affected animals, therefore, usually die from the effects of liver failure or are
euthanized to prevent needless suffering. Plant-induced biliary occlusive photosensitivity cases have a better prognosis and can recover if the toxicity has not persisted for a long time.

Control of Photosensitizing Plants
Eradication of plants containing PAs is not practical on a large scale. Selective cultivation and removal of the plants works well in a limited area. Herbicides are effective when applied to the immature stages of Senecio and hound’s tongue. Regardless of the control method implemented it is important to do so before heads are produced. Biologic control methods offer possibilities for control in large geographic areas. A beetle (Chrysolina guadrigemina) has been successfully used in the past to control St. John's wort. In limited areas of northern California, the larvae of the Cinnabar moth (Tyria jacobaeae) in conjunction with a root-feeding flea beetle (Longitarsus jacobaeae) have been used successfully to control S. jacobaea [141]. It is also feasible to use sheep to graze Senecio spp. because they are relatively resistant to pyrrolizidine poisoning. In due course new biological methods may be developed to aid in the control of these poisonous plants.

Bishop's Weed, Greater Ammi
_Ammi majus_ - Apiaceae (Parsley family)

Habitat
Introduced from Asia, bishop’s weed has become established in the wet, brackish meadows of the coastal region of the southern United States. It is also commonly grown as an ornamental annual.

Description
Bishop’s weed is an annual, 1 to 2 feet (0.30 to 0.60 meters) tall with ascending branches and leaves that are finely dissected into filiform segments. The inflorescence is umbellate with small white flowers (Fig. 4-3). The involucral bracts are pinnately parted, the involucels are of several linear bracts.

Figure 4-3. Bishop’s weed (_Ammi majus_). - To view this image in full size go to the IVIS website at www.ivis.org.

Principal Toxin
The Principal Toxins are the furocoumarins. All parts of the plants, but especially the seeds, may be phototoxic to cattle, sheep, fowl, and humans as a result of ingestion or skin contact with the plant and subsequent exposure to sunlight [142-145].

Buckwheat
_Fagopyrum esculentum_ - Polygonaceae (Buckwheat family)

Habitat
Cultivated as a grain or cover crop, buckwheat has since escaped and become a plant of disturbed soils and roadsides. It is commonly used as a cover crop for soil enrichment.
Habitat of Buckwheat. *Fagopyrum esculentum* - Polygonaceae (Buckwheat family). - To view this image in full size go to the IVIS website at www.ivis.org.

**Description**

Buckwheat is a glabrous, herbaceous annual plant with an erect stem. The leaves are alternate and have hastate or cordate blades. The stipules are united as a sheath (ochrea) around the stem at the nodes. The greenish white flowers occur at terminal or axial panicles (Fig. 4-4). The stamens number eight, styles three-parted, and the achenes three-angled and brown in color.

![Figure 4-4. Buckwheat (*Fagopyrum esculentum*). - To view this image in full size go to the IVIS website at www.ivis.org.]

**Principal Toxin**

Fagopyrin, a plant pigment present in the green and to a lesser extent the dry plant, is capable of producing photosensitization in domestic livestock and people [146]. Buckwheat flour is not toxic.

St. John’s Wort, Klamath Weed  

Hypericum perforatum - Hypericaceae (St John’s wort family)

**Habitat**

St. John’s wort is an introduced weed of dry soils throughout most of North America, especially in the northwestern states. There are numerous cultivated hybrids in existence.

![Habitat of St. John’s Wort, Klamath Weed. *Hypericum perforatum* - Hypericaceae (St John’s wort family). - To view this image in full size go to the IVIS website at www.ivis.org.]

**Description**

St. John’s wort is an erect perennial herb up to 3 feet (1 meter) tall with woody lower stems. The branches are opposite and sterile. Usually both stems and branches are two-edged or winged. The leaves are opposite, sessile, and linear-oblong, 1 inch long and spotted with tiny dots that are translucent when held against the light. The inflorescence is a cyme with numerous flowers, two-thirds to an inch in diameter having five bright yellow petals, five green sepals, many stamens in three to five clusters and an ovary with three widely spreading styles (Fig.4-5). The petals have finger-like margins and may have black glandular dots on the margins.

![Figure 4-5. St. John’s wort (*Hypericum perforatum*). - To view this image in full size go to the IVIS website at www.ivis.org.]

Principal Toxin
Hypericin, a photoreactive pigment, is readily absorbed from the digestive tract and remains chemically intact through the digestion process. Hypericin has no effect on the liver and causes primary photosensitization after ingestion [147]. Hypericin is present in the glandular dots on the leaves suggesting that all Hypericum spp. with similar glands are potentially toxic. Hypericin is stable to drying and therefore hay containing St John’s wort may cause poisoning. The young plants are as toxic as the mature plant and more palatable to livestock although the content of hypericin varies with growing conditions of the plant [148]. Hypericin has found recent popular use in people as an herbal stimulant and will induce photosensitivity in some individuals, especially if overdosed. Hypericin has also shown some antiviral properties and has been investigated for its effects on human immunodeficiency virus [149,150].

Lechuguilla
*Agave lecheguilla* - Agavaceae (Agave family)

Habitat
Lechuguilla plants are found in low limestone hills, dry valleys, and bordering canyons especially in the southwestern United States and Mexico.

Description
The plant is a stemless perennial with leaves 10 to 30 in number, fleshy, bayonet-like, erect and attached to a short, broad crown at ground level. Each leaf is 1 to 1.5 inches (2.5 to 4 cm) wide at the base and tapering to a very sharp point (Fig.4-6). They range from 12 to 20 inches (30 to 51 cm) long with recurring marginal teeth. The plant flowers once after 10 to 15 years of vegetative growth, producing a large terminal panicle on a thick stalk ranging from 6 to 12 feet (2 to 3.5 meters) tall. The flowers are tubular with three sepals, three petals, six stamens, and a three-carpellate ovary that matures into a leathery capsule producing many flat black seeds. After flowering, the plant dies. Reproduction may continue vegetatively by offshoots from the parent plant.

Principal Toxin
Hepatotoxic saponins in lechuguilla appear to be the primary cause of poisoning in sheep and goats and less frequently cattle [151]. Photosensitivity results from the accumulation of phylloerythrin as a result of bile duct obstruction similar to that which occurs in *Tribulis terrestris* photosensitivity [82,83]. Animals seldom eat the plant except in times of drought. As little as 1 percent of the animal's body weight of the leaves has caused poisoning and death. Liver disease with icterus, weight loss, and secondary photosensitization are common features of lecheguilla poisoning.

Fiddleneck, Tarweed
*Amsinckia intermedia* - Boraginaceae (Forget-me-not family)

Habitat
Fiddleneck is a weed of dry cultivated soils, waste ground, and wheat fields. It continues to spread eastward from western
Description
Fiddleneck is an erect, sparsely branching, 2 to 3 feet (1 meter) tall annual weed, covered with numerous white hairs. Leaves are hairy, lanceolate, and alternate (Fig.4-7a). The perfect five-parted small orange to yellow flowers are born terminally on a characteristic fiddleneck-shaped raceme, the flowers all inserted on one side of the axis (Fig.4-7b). Mature fruits separate into two to four black-ridged nutlets.

Principal Toxin
Pyrrolizidine alkaloids are the principle toxins responsible for poisoning in horses, cattle, and pigs that eat the plants, especially the seeds [31]. The symptoms and lesions of Amsinckia poisoning in all species of animals consist of liver necrosis and fibrosis characteristic of PA poisoning. Amsinckia spp. may also accumulate potentially toxic levels of nitrate as rapidly growing annual weeds.

Rattlebox
*Crotalaria* spp. - Fabaceae (Legume family)

Habitat
Some species of *Crotalaria* were introduced as a soil-building cover crop for the sandy soils in the southeastern United States and have since become established in disturbed soils along fences and roadsides in Florida and Georgia. An indigenous species, *C. sagittalis* is common along river bottomland.

Description
*Crotalaria* are erect, herbaceous, variably hairy plants, and may be annual or perennial. The leaves are simple, alternate, lanceolate to obovate, with a finely haired under surface (Fig.4-8A). The flowers are yellow, with the leguminous calyx longer than the corolla (Fig.4-8B). The fruit is a leguminous pod, inflated, hairless, becoming black with maturity, and contains 10 to 20 glossy black, heart-shaped seeds, which often detach and rattle with the pod. Several species of *Crotalaria*
have been associated with livestock poisoning including *C. sagittalis*, *C. spectabilis*, and *C. retusa*.

**Figure 4-8A. Crotalaria spp.** - To view this image in full size go to the IVIS website at www.ivis.org.

**Figure 4-8B.** Rattlebox flower and seed pods (*Crotalaria spectabilis*). - To view this image in full size go to the IVIS website at www.ivis.org.

**Principal Toxin**

The principal toxins in *Crotalaria* spp. are the pyrrolizidine alkaloids (PA), the most notable of which is monocrotalamine. The alkaloid is present in greatest quantity in the seeds, with lesser amounts in the leaves and stems. All livestock, including domestic fowl are susceptible to poisoning. Although acute deaths will occur from eating large quantities of the crotalaria seeds or plant, more typically animals will develop signs of liver disease and photosensitization from a few days up to 6 months later. Monocrotaline also causes severe pulmonary changes, and horses have been reported to die after developing an acute fibrosing alveolitis from eating a feed containing 40 percent crotalaria seeds [63,64].

---

**Senecio**

Groundsel, tansy ragwort, butterweed

*Senecio* spp. - Asteraceae (Sunflower family)

**Habitat**

More than 1200 species of *Senecio* are distributed throughout the world, with about 70 species occurring in North America. Approximately 25 species have been proven poisonous to animals, but there is high probability that other species of *Senecio* are toxic. *Senecio* spp. have a wide overlapping geographic range, but many are, however, selective in their habitat, some preferring high altitude, subalpine, moist conditions, whereas others prefer dry, rocky, or sandy soils at lower elevations. The more important toxic species of *Senecio* in North America are listed in Table 4-4.

**Habitat of Senecio. Senecio spp.** - Asteraceae (Sunflower family). - To view this image in full size go to the IVIS website at www.ivis.org.

<table>
<thead>
<tr>
<th>Scientific Name</th>
<th>Common Name</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Senecio jacobaea</em></td>
<td>Tansy ragwort, stinking willie</td>
</tr>
<tr>
<td><em>S. intergerritum</em></td>
<td>Lamb’s tongue groundsel</td>
</tr>
<tr>
<td><em>S. douglasii</em></td>
<td>Woody or threadleaf groundsel</td>
</tr>
<tr>
<td><em>S. riddelli</em></td>
<td>Ridell’s ragwort</td>
</tr>
<tr>
<td><em>S. plattensis</em></td>
<td>Prairie ragwort</td>
</tr>
</tbody>
</table>
**Description**

Identification of individual *Senecio* spp. is difficult without being an experienced taxonomist. However, recognition of a plant as a member of the genus *Senecio* can be based on the presence of a single layer of touching, but not overlapping, greenish bracts surrounding the flower (Fig.4-9A). *Senecio* spp. have alternate leaves, generally lanceolate to ovate, dentate and often irregularly and deeply pinnately divided (Fig.4-9B). The composite flower heads are flattened terminal clusters with showy yellow ray flowers (Fig.4-9C). Seed is produced in both disc and ray florets, each seed with a tuft of white hairs that aid in wind dissemination (Fig.4-9D).

### Table 4 - 4. Specie Associated with Livestock Poisoning

<table>
<thead>
<tr>
<th>Scientific Name</th>
<th>Common Name</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>S. spartioides</em></td>
<td>Broom groundsel</td>
</tr>
<tr>
<td><em>S. glabellus</em></td>
<td>Butterweed</td>
</tr>
<tr>
<td><em>S. vulgaris</em></td>
<td>Common groundsel</td>
</tr>
</tbody>
</table>

**Principal Toxin**

Pyrrolizidine alkaloids (PA) are the principal toxins in senecios [19,59,63]. The quantity of alkaloid in *Senecio* spp. varies with the species and stage of growth, the young pre-flowering plant being most toxic. Acute liver necrosis and death in 1 to 2 days has been associated with feeding cattle 4 to 8 percent of an animal's body weight in green plant over a few days [78]. Chronic poisoning, which is the more natural form of poisoning encountered in cattle and horses, is usually associated with ingestion of smaller quantities of *Senecio* over a period of 3 weeks or more. Cattle fed 1.5 g of *S. jacobaea* kg of body weight daily for a minimum of 20 days had 100 percent mortality. This equates to a 20-day cumulative dose of 2 percent of an animal's body weight of dry plants. The cumulative effects of PAs on the liver may take 6 or more months to reach the point...
where 80 percent or more of the liver is affected and clinical signs of liver disease become evident.

Hound’s Tongue
*Cynoglossum officinale* - Boraginaceae (Forget-me-not family)

**Habitat**
Imported in wheat seed from Russia, hound’s tongue has become widespread in most regions of North America and especially in the Rocky Mountain region where it is considered a noxious weed. It prefers waste areas, roadsides, meadows, and hay fields.

![Habitat of Hound’s Tongue. *Cynoglossum officinale* - Boraginaceae (Forget-me-not family). - To view this image in full size go to the IVIS website at www.ivis.org.](image)

**Description**
Hound’s tongue is a biennial, forming a rosette the first year, with basal leaves up to 18 inches (45.5 cm) long, densely pilose, oblong or oblong-lanceolate in shape (Fig.4-10A). The second year the plant produces stems that are 2 to 4 feet (0.5 to 1 meter) tall, erect, stout, softly pilose, and flowering. The upper leaves are lanceolate and sessile. The flowers are regular, reddish purple, in scorpioid racemes (Fig.4-10B). The fruit is pyramidal, separating into four nutlets at maturity, which are covered with hooked barbs (“velcro”) facilitating their adherence to clothing and animal hair.

![Figure 4-10A. Hound’s tongue first-year rosette (*Cynoglossum officinale*). - To view this image in full size go to the IVIS website at www.ivis.org.](image)

![Figure 4-10B. Hound’s tongue flowers and fruits (C. officinale). - To view this image in full size go to the IVIS website at www.ivis.org.](image)

**Principal Toxin**
Hound’s tongue contains significant quantities of pyrroliidine alkaloids (PA) comparable to the levels found in the most toxic *Senecio* spp. The plant is rarely eaten in the green state, but livestock find it palatable when it is dried in hay. As little as 15 mg of dried plant/kg body weight fed to horses over a 2-week period induced fatal liver disease [137]. This equates to about 6 percent of a horse's daily intake of food if the hound’s tongue is dried in the early blossom stage when the PA content averages 0.08 percent [38].

**Note** - Viper’s bugloss or blue weed (*Echium vulgare*) is an introduced European plant that contains PAs. It is an invasive weed in some areas and is often cultivated as an ornamental flower. In the same family as hound’s tongue (*Boraginaceae*), it is a very hairy plant with showy blue, pink, or white, tubular flowers, produced in one-sided clusters that uncoil as the flowers bloom sequentially (Fig.4-10C). The leaves are hairy, 2 to 6 inches (5 to 15 cm) in length and lanceolate. Numerous rough nutlets are produced that promote its proliferation.

![Habitat of Viper’s bugloss, blue weed (*Echium vulgare*). - To view this image in full size go to the IVIS website at www.ivis.org.](image)
**Lantana**

*Lantana camara* - Verbenaceae (vervain family)

**Habitat**
Lantana is an ornamental shrub in the southern warmer areas of North America, often escaping along stream bottoms in the southwest to become an invasive noxious weed.

**Description**
The plant is a shrub with square stems and a few scattered spines. The leaves are simple, opposite or whorled, and oval-shaped. The margins are serrate. The flowers are born in flat-topped clusters and are small, tubular, and white, yellow, orange, red, or purple (Fig.4-11). The fruits are produced in clusters and turn black when ripe.

**Principal Toxin**
At least 15 of the 29 described taxa of *L. camara* are known to be toxic to livestock [91,92]. Lantana poisoning is a disease of ruminants characterized by an intrahepatic cholestasis induced by the triterpene acids lantadene A and B. Horses are apparently not affected. Acute poisoning frequently results in death in 7 to 10 days. In more chronic poisoning, sheep in particular stop eating and develop a metabolic acidosis with resulting increases in serum potassium. Death probably results from a combination of liver disease, anorexia, and accumulation of triterpene acids. Photosensitization may be evident in white skinned areas of affected animals.

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**Sacahuista, Beargrass**

*Nolina texana* - Agavaceae (Agave family)

**Habitat**
Sacahuista grows on dry hillsides at elevations from 3000 to 6500 feet (914 to 1,981 meters) in milder climates of the southwestern states.
**Description**
Sacahuista is an evergreen perennial grasslike plant with a thick woody, mostly haired basal stem growing 2 to 3 feet (0.5 to 1 meter) tall (Fig.4-12). Many linear leaves, 0.5 inch (1 cm) wide, 5 feet (1.5 meters) long, with finely toothed margins arise from the basal stem. Small white to green flowers are born on stalks in elongated clusters. Fruits are three-parted capsules containing many seeds. *Nolina microcarpa* is very similar in appearance and distribution.

**Principal Toxin**
An undefined hepatotoxin saponin concentrated in the flower buds, flowers, and fruits appears to be responsible for a crystalloid material that obstructs the biliary system, causing phylloerythrin accumulation and photosensitivity [152]. The leaves are not toxic and are eaten by livestock at times when other forages are unavailable. All ruminants are susceptible to poisoning, but sheep and goats are most commonly affected [152]. About 1.1 percent of an animal's body weight in flowers or fruits will induce poisoning. In severe cases animals die from liver failure.

**Note** - Beargrass is the common name given to *Xerophyllum tenax* that grows in British Columbia, Montana, and from Wyoming to northern California, and is not related to *Nolina* species. It is not known to be toxic.

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**Horsebrush**
*Tetradymia* spp. - Asteraceae (Sunflower family)
*T. glabrata* - Coal oil brush, little horsebrush, spring rabbit brush
*T. canescens* - Spineless or gray horsebrush

**Habitat**
Horsebrush is generally confined to southwest North America and is found growing in sagebrush (*Artemisia* spp.) habitats. *Tetradymia glabrata* is found in semidesert foothills, whereas *T. canescens* grows at higher elevations in the Rocky Mountains.

**Description**
Horsebrush is a low shrub with stiffly, much-branched stems that are usually covered with white, wooly hairs, especially when young (Fig.4-13A). The leaves are alternate, narrow, and entire. The inflorescence is a head that is solitary from the upper axils or clustered at the tips of the branches (Fig.4-13B).
There are four bracts present. The florets are yellow with a large number of straw-colored bristles or pappus. *Tetradymia spinosa* is a shrub between 1.5 to 4 feet (0.45 to 1 meter) tall and varies from *T. canescens* by the fact that the flowers number five to nine to a head and the bracts five to six. The primary leaves are converted to spines.

**Principal Toxin**
Tetradymol, a furanoeremophilane, is the main toxic compound that causes liver toxicity. Sheep are primarily affected by horsebrush, but must eat black sagebrush *(Artemisia nova)* before or at the same time they eat horsebrush if poisoning is to occur [153,154]. The sagebrush preconditions the sheep to the toxic effects of the horsebrush indicating strong synergistic action of the two plants on the liver. Sheep vary in their susceptibility to horsebrush but 0.5 to 1.0 lb of little leaf horsebrush (*T. glabrata*), which is about twice as toxic as *T. canescens*, will produce photosensitization (big head). Consumption of larger amounts of the plant causes death in a few days. Abortions have also been attributed horsebrush when the plant is grazed by pregnant ewes.

---

**Puncture Vine, Caltrop, Goat’s head**
*Tribulus terrestris* - Zygophyllaceae (Caltrop family)

**Habitat**
Puncture vine is found in the dry soils of wastelands, roadsides, and deserts in most eastern, southern, and western states, and other regions of the world.

**Description**
Puncture vine is a prostrate, annual weed with recumbent stems that are pubescent and branching occasionally up to 3 feet (1 meter) in length. Leaves are opposite and pinnately compound with leaflets occurring in four to seven pairs, oblong, elliptical, and about 0.5 inch (1 cm) long. Flowers are solitary, occurring in the axils of the leaves with a corolla composed of five yellow petals (Fig.4-14). The fruit is a small hard capsule that breaks apart into five spiny sections, each having two prominent, sharp, woody spines, that resemble a goat head in appearance.

Puncture vine is similar in appearance to hairy caltrop (*Kallstroemia hirsutissima*), differing in the very hairy leaves and stems of the latter.

**Principal Toxin**
A number of steroidal sapogenins have been found in *Tribulus terrestris* that form an insoluble crystalloid substance in the bile ducts [155]. The development of photosensitivity appears to be secondary to obstruction of the biliary system with this calcium rich crystalloid substance that forms microliths (caleuli or stones). These microliths in the bile ducts increase the retention of phylloerythrin and the development of secondary photosensitivity [155]. A mycotoxin present in the plant has
also been suspected of being associated with hepatogenic photosensitivity in livestock [82]. An entirely different syndrome of sheep characterized by hindleg weakness and paresis has been associated with the chronic consumption of *T. terrestris* in Australia [156]. Caltrops (*Kalstroemia* spp.) have produced a similar neurologic syndrome in sheep, goats, and cattle in Texas [157]. The plant toxins causing the neurologic signs are the \(\beta\)-carboline indoleamines harmane and norharmane [158].

The spiny burrs are mechanically injurious to animals and people.

### Panic grasses

*Panicum spp.*  
**Kleingrass** (*Panicum coloratum*)  
Blue panicum (*P. antidotale*) - Poaceae (Grass family)

**Habitat**  
Panic grasses are mostly confined to southwest North America in dry woodlands, plains, and valleys.

**Description**  
Panic grass is a warm-season bunch grass growing to 5 feet (1.5 meters) tall with blue-green leaf blades to 24 inches (70 cm) long. There are often soft straight hairs on the upper side of the leaf next to the culm. The inflorescence is an open, widespreading panicle 6 to 18 inches (15 to 45 cm) long.

**Principal Toxin**  
Sheep and occasionally cattle grazing on panicum pastures in some years under hot humid conditions can develop photosensitization. Saponins in the grass are assumed to be the cause of the formation of crystalloid substances in the biliary system that subsequently obstruct the bile duct and cause secondary photosensitization [159].

### Tansy Mustard

*Descurainia pinnata*  
**Flixweed**  
*Descurainia sophia* - Brassicaceae (Mustard or crucifer family)

**Habitat**  
These plants are commonly found throughout North America, being opportunistic weeds of cultivated fields, disturbed soils, waste areas, roadsides, and open dry areas.

**Description**  
Flixweed is an erect, terminally branched, annual, herbaceous plant growing 1 to 2 feet (0.5 meters) in height. The leaves are alternate, two to three times pinnately compound, each segment narrow or linear, the basal leaves being largest. The plant is pubescent or nearly hairless, with hairs being branched. The small greenish yellow flowers are produced on a raceme (Fig.4-15A). The seed pod (silique) is cylindrical, 0.5 to 1.25 inches (1 to 3 cm) in length, the silique septum having two to three longitudinal nerves. Tansy mustard is a very similar annual that is differentiated from flixweed on the basis of its shorter, fatter, siliques (less than 0.75 inches (2 cm) in length), that contain two rows of seeds in each seed pod (Fig.4-15B). Both species spread readily by seed.
Principal Toxin
No specific toxin has been identified in either tansy mustard or flixweed that has been associated with the clinical signs of liver disease and photosensitization reported in cattle grazing the plants [160,161].

Clinical Signs
Two apparently different syndromes in cattle have been attributed to tansy mustard. In cattle, a neurologic syndrome of partial or complete blindness, accompanied by the inability to use the tongue or swallow, aimless wandering, head pressing, and eventual death from dehydration and starvation has been sporadically reported [1]. In some areas, and in some years, the principal toxicity associated with tansy mustard is one of liver disease and photosensitization without neurologic signs [160,161]. Attempts at reproducing the disease by feeding tansy mustard have not been successful [161].

Kochia, Summer cypress, Mexican Fireweed, Belvedere, Morenita
Kochia scoparia - Chenopodiaceae (Goosefoot family)

Habitat
Originally from Asia, and introduced from Europe, kochia has become an established weed of North and South America because of its wind-distributed seeds and adaptation to dry conditions. It is commonly found in disturbed soils, croplands, pastures, and dry rangeland. In many areas of southwestern North America, kochia weed is favored as a useful forage for livestock and is often cut and baled as a winter feed.

Description
Kochia is an annual, herbaceous, rapidly growing plant with branching stem and simple, entire, hairy leaves (see Fig.1-17A and Fig.1-17B). The leaves are alternate and vary in size from 0.5 to 3.0 inches (1 to 7.5 cm) in length on the mature plant. Stems are either green or red-tinged, the stems becoming particularly red in the fall. The size of the plant varies considerably, attaining heights of 6 feet (2 meters) where the moisture and organic content of the soil are high. The flowers are green and produced in dense spikes in the upper leaf axils (see Fig.1-17C). The seeds are small, brown, and slightly ribbed. Kochia weed is often erroneously called tumbleweed because it breaks off at ground level in the winter and gets blown about by the wind. True tumbleweed is another common weed called Russian thistle (Salsola kali).

Principal Toxin
Kochia has a mixed reputation as a nutritious forage and under some circumstances as a poisonous plant to cattle and sheep. A specific toxin has not been identified in kochia that will account for the various syndromes of toxicity encountered in cattle and sheep whose diet consists predominantly of kochia. Nitrates, oxalates, sulfates saponins, and alkaloids have been identified in the plant depending on the stage of growth, growing conditions, and geographic region [162-166]. Kochia is used as a forage and is made into hay for livestock in many arid regions of North America. If grazed or cut before it reaches maturity, kochia does not appear to be toxic, although if high in nitrates it is hazardous to ruminants [162]. Its nutritive value is similar to that of alfalfa [165]. However, livestock grazing kochia when it is mature or stressed by drought are prone to poisoning at least in some regions [162,163]. Poisoning is more likely to occur if the animal’s diet consists predominantly of kochia. Experimental feeding trials in which cattle were allowed to graze pure stands of irrigated and
fertilized kochia from 2 to 15 weeks resulted in poor weight gain when compared to cattle grazing native grasses [167]. Furthermore, those cattle eating kochia alone developed degenerative liver and kidney disease. Sheep may be fed kochia up to 50 percent of their total dry matter intake without affecting weight gain or causing toxicity [162]. Other studies have shown that sheep can graze greater than 95 percent kochia with only slight elevations of liver enzymes indicative of mild liver degeneration without apparent clinical disease or effect on growth weights [166]. The variable toxicity of kochia would appear to be related to the stage of maturity of the plant, and perhaps a combination of the effects of the various toxic components of the plant. The general toxic effect appears to be that of degenerative liver disease with secondary photosensitization and liver failure.

**Clinical Signs**

Cattle, sheep and occasionally horses appear to be the most commonly affected when mature kochia is the predominant constituent of their diet [163,167-170]. The most common clinical signs observed in cattle include poor weight gains, depression, weakness, excessive tearing, incoordination, and photosensitization [167,169]. Other clinical signs associated with kochia poisoning include loss of appetite, diarrhea, icterus, and mouth ulcerations [162]. Affected animals frequently have elevated serum liver enzymes and bilirubin levels [166,167]. Some animals will be blind and walk in circles or follow fences endlessly [162,169]. These neurologic signs may be due to the high levels of sulfate that once reduced in the rumen to hydrogen sulfide result in degenerative changes in the brain leading to depression and blindness [171]. Long term consumption of mature kochia results in eventual death of the animal. However, cattle and sheep will recover from kochia poisoning provided they are given a balanced ration where kochia weed is not the only food available.

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**Common Cocklebur**  
*Xanthium strumarium* - *Asteraceae* (Sunflower family)

**Habitat and Description**

Found throughout North American, cockleburs are annual, bushy weeds 2 to 5 feet (0.5 to 1.5 meters) tall, with stout stems, often with dark spots. The leaves are large, rough, glandular, and triangular from 2 to 14 inches (5 to 35.5 cm) long and 1 to 8 inches (2.5 to 20 cm) wide. Flowers are produced in the leaf axils, the inconspicuous male flowers being clustered at the top, with the larger female flowers that form the burs toward the base (Fig.4-16B). Hooked spines cover the characteristic oval burs. Each bur contains two seeds, which can remain dormant in the soil for years. Consequently cockleburs can reappear many years after the parent plant disappeared.

Habitat of Common Cocklebur. *Xanthium strumarium* - *Asteraceae* (Sunflower family). - To view this image in full size go to the IVIS website at www.ivis.org. -

Figure 4-16A. Cocklebur (*Xanthium strumarium*). - To view this image in full size go to the IVIS website at www.ivis.org. -

**Principal Toxin**

Carboxyactractyloside, a sulfated glycoside, is the primary liver toxin present in cockleburs [172,173]. The glycoside is present in high concentration in the seeds and the two-leafed cotyledon stage (Fig.4-16B), but disappears by the four-leaf stage and is not present in the mature plant. The liver appears to be the primary target organ with pigs, ruminants, and horses being susceptible to poisoning.
Fatalities occur when 0.75 to 3.0 percent body weight of cotyledons are consumed [174]. Acute deaths, convulsions, blindness, and recumbency have been encountered in cattle eating hay contaminated with mature cockleburs [175]. The most consistent effect of the toxin on the liver is acute diffuse central-lobular and paracentral coagulative necrosis [173]. Similar renal tubular necrosis may occur. Severe hypoglycemia and marked elevations of liver enzymes (sorbitol dehydrogenase) are encountered in experimental poisoning of pigs [172,177]. The spiny burs are a source of mechanical injury to the mouth of dogs, especially long haired breeds, that pull them from their hair coat with their teeth when self grooming. If dogs swallow the burs they vomit subsequently, but it is unknown whether this is due to mechanical irritation or the toxic glycoside. Significant economic losses can also occur to wool producers when the burs become entangled in the fleece reducing its market value.

**Clinical Signs**

Poisoning usually occurs when animals eat large numbers of the two-leaved stage of the cocklebur or eat the seeds that may contaminate cottonseed or other food sources. Pigs and cattle show similar signs of poisoning including depression, reluctance to move, hunched back, ataxia, and recumbency [174-176]. Hypoglycemia may be present in severe cases. Paddling of the limbs, convulsions succeeded by coma, and death in 24 hours are common. Serosanguinous ascites, edema of the gallbladder, pericardial and pleural effusion, and hepatic congestion are the usual postmortem findings. There is no known effective treatment for cocklebur poisoning. Attempts to eliminate the toxin from the gastrointestinal tract may be made with neostigmine and mineral oil. Activated charcoal may also be effective as an adsorbent. Intravenous glucose will help counteract the severe hypoglycemia induced by the toxin. Cockle burs are best controlled by mowing, cultivation, or herbicides, provided the plants are destroyed before they produce burs. A single mature plant that goes to seed will effectively repopulate the area becoming a troublesome and aggressive weed.

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**Hairy Vetch**

_Vicia villosa_ - Fabaceae (Legume or pea family)

**Habitat**

Introduced from Europe as a legume for pasture improvement and as a cover crop, hairy or woolly vetch has become an established weed in many areas of North America, especially along roadsides, waste areas, and in croplands.

**Description**

Hairy vetch is a climbing or sprawling annual 4 to 6 feet (1 to 2 meters) in length, with hairy stems and leaves. The leaves have 10 to 20 leaflets up to 1 inch (2.5 cm) in length that are narrow and lance-shaped. Tendrils at the end of the leaves are well developed. Flowers are purple to red in color, 20 to 60 per spike, all on one side of the flower stalk (Fig.4-17A). Pea-like seed pods are about 1 inch in length containing several hard seeds.

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Common vetch (_Vicia sativa_) that can be mistaken for hairy vetch, is similar but is not hairy and the flowers are not arranged on one side of the flower stalk (Fig.4-17B).
Principal Toxin
The specific toxin(s) in hairy vetch responsible for the symptoms encountered in hairy vetch poisoning has not been determined. The generalized granulomatous disease that is characteristic of hairy vetch poisoning in cattle and horses is suggestive of a hypersensitivity (type IV) reaction induced by a foreign substance that activates the immune system response [178-184]. Vetch lectins have been proposed as the instigators of the immune mediated response that results in the granulomatous inflammatory response seen in many different tissues of the affected animal [185]. Not all animals are susceptible to vetch hypersensitivity, the disease being more prevalent in cattle over 3 years of age [178,183]. Although reported in many breeds of cattle, hairy vetch poisoning appears to be more common in the Angus and Holstein breeds [181,183]. Hairy vetch poisoning occurs most often when it forms a major part of the diet of cattle and horses, and when the plant is nearing maturity. The plant is less likely to cause a problem in hay or when ensiled [183]. However, the mechanism by which hairy vetch induces poisoning remains unclear as the plant is frequently consumed by cattle without apparent problem.

Clinical Signs
Hairy vetch poisoning sporadically affects adult cattle and occasionally horses and is characterized by signs of pruritic dermatitis, weight loss, conjunctivitis, and diarrhea [179,181,183]. The dermatitis is not confined to white skinned areas. Initially the hair coat is rough and stands erect where lesions develop. Papules then develop that exude serum and result in superficial crusts. Continual rubbing and scratching of the lesions causes hair loss and thickening of the skin [183]. Abortions and red urine have also been associated with hairy vetch poisoning [183]. Lymphocytosis and hyperproteinemia are a feature of hairy vetch poisoning. A more acute form of hairy vetch poisoning characterized by subcutaneous swellings, ulcers of the oral mucous membranes, purulent nasal discharge, and coughing with significant mortality may also occur. Ingestion of large amounts of hairy vetch seed may induce neurologic signs and death in cattle and horses [186]. At post mortem examination, granulomatous lesions can be found in multiple organs including the skin, liver, kidneys, heart, spleen, lymph nodes, and digestive system. Histologically, the lesions consist of monocytes, eosinophils, multinucleated giant cells and lymphocytes [180,181,183].

Leucaena, lead tree
*Leucaena leucocephala* - Mimosaceae (Legume family)

Habitat
Leucaena is a tropical legume found in many parts of the world that is well established in Hawaii and has been introduced into Florida, Texas, and coastal areas in between. As a legume it has potential as a protein source for livestock in tropical areas of the world.

Description
Rapidly growing shrub or tree attaining heights of 30 feet (10 meters), with bipinnately compound leaves, 6 to 10 inches (15 to 25 cm) long, with 4 to 8 pairs of pinnae, each with 10 to 15 pairs of lanceolate leaflets. Numerous white to pink tightly clustered flowers are produced in spherical heads on long pedicels from the leaf axils. Fruits are many seeded legume-like pods (Fig.4-18).
**Principal Toxin**

Leucaena contains 25 to 35 percent crude protein in its leaves that has potential as a livestock protein source especially in those parts of the world deficient in available sources of protein rich forages for animals. However, leucaena contains the amino acid mimosine that is poisonous to animals that are not adapted to eating the plant. The precise mode of action of mimosine is unclear, but it and its metabolites are presumed to act as amino acid antagonists and/or will complex with metals such as zinc, copper, and magnesium thereby inhibiting a variety of enzymes important to protein metabolism [187-189]. Mimosine may also act as goitrogen, causing goiter and signs of thyroid deficiency [187]. In some parts of the world cattle and goats thrive on eating leucaena, while in other areas animals lose weight, lose their hair and grow poorly when eating the plant. The ability of some animals to thrive on leucaena and others not is related to the ability of the animal’s rumen microflora to detoxify mimosine. Specifically it is the presence of the anaerobic rumen bacterium Synergistes jonesii that detoxifies mimosine and enables the animal to make use of the plant’s proteins without ill effect [190-192]. It is possible and practical to transfer this bacterium from one animal to the next through transferring rumen contents thereby populating the animal’s rumen with the bacteria and enabling it to eat leucaena without problem. The organism once established in the rumen will survive from season to season even if the animal does not have daily access leucaena [193]. Nonruminants do not have the capability of adapting to leucaena and should not be fed the plant.

**Clinical Signs**

Cattle, sheep, and goats eating leucaena exhibit signs of poor growth, weight loss, hair loss, lameness due to swollen and ulcerated coronary bands, and mouth and esophageal ulcers [193,194]. Goiter with decreased thyroxine levels, cataracts, atrophy of the gums, decreased fertility due to embryonic death, and fetal abnormalities may also be seen in animals consuming leucaena [194,195]. Once ruminants establish mimosine degrading bacteria in their rumen they are able to utilize leucaena as a good protein source. Nonruminants should not be fed leucaena.

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**Scorpion Weed**

*Phacelia crenulata* - Hydrophyllaceae (Waterleaf family)

**Habitat**

Scorpion weed is a common plant of drier climates and desert areas of southwestern North America, preferring sandy soils of roadsides and waste areas. Other species of Phacelia are found throughout the continent and are considered a desirable wild flower.

**Description**

Often forming dense stands, scorpion weed is an annual or biennial, growing to 18 inches (45 cm) in height, with pinnately lobed hairy leaves. Flowers are small, blue or white, and open sequentially on one-sided terminal coils (Fig.4-19). The name scorpion weed refers to the coiled flower head that resembles a scorpion’s tail.
**Principal Toxin**
The hairs of the plants contain phenolic compounds that are contact allergens [196]. These compounds have an allergic equivalent to the compound urushiol found in poison ivy. Some dogs and people coming in contact with the hairs, especially on the dry plant, develop an acute skin reaction.

**Clinical Signs**
Dogs especially develop swollen feet and may also have swelling and inflammation of the lips, eyelids, and mouth if they are running through dense stands of the scorpion weed. Affected animals should have the affected skin areas washed with a mild soap and water to remove the residual plant hairs. Antihistamine and anti-inflammatory medications may be necessary in severe cases.

**Poison Ivy**  
*Toxicodendron radicans* - (Synonym Rhus radican) Anacardiaceae (Cashew family)

**Habitat**
Poison ivy is found throughout North America except on the West Coast where poison oak (*Toxicodendron quercifolium*) prevails. Varieties of the plant may be found in dry sandy soils, or moist loamy soils of woodlands and forests.

**Description**
Poison ivy is a perennial, woody plant that is typically a trailing or climbing vinelike plant in eastern North America, in the drier western areas of North America it is a low growing, bushy shrub. The leaves are glossy, hairless, alternate, always three-parted, and may be toothed, or lobed (Fig. 4-20A). The climbing stems produce aerial roots that anchor the pant to the bark of trees on which it is climbing. The flowers are small, whitish, hanging clusters, and the fruits are white and berry-like with longitudinal grooves. The leaves turn a bright red in the fall.

**Poison oak** (*T. quercifolium*) is a woody, perennial shrub, with alternate, deeply lobed, oaklike leaves found in western North America (Fig. 4-20B). The flowers are small, greenish, and are produced in hanging clusters. The fruits are yellowish, hairy berries. Also found in the same region is western poison oak (*T. diversilobum*), which is similar to poison oak but is a vine that can reach 50 feet (15 meters) in length. Its fruits are white berries.

**Poison sumac** (*T. vernix*) is a woody perennial, branching shrub or small tree with gray bark and leaves to 12 inches (30 cm)
long, each with a distinctive red-purple rachis. Leaflets are smooth, elliptic and entire (Fig.4-20C). The flowers are small and yellowish white and produced in panicles. The fruits are white berries. Poison sumac is usually confined to wet areas such as pineland bogs in southern North America.

Figure 4-20C. Poison sumac (T. vernix) (Courtesy of Drs. John and Emily Smith, Baldwin, Georgia). To view this image in full size go to the IVIS website at www.ivis.org.

Principal Toxin
Poison ivy, poison oak, and poison sumac all contain a highly irritating allergenic phenolic compound urushiol (oleoresin). The most toxic being the resin 3-n-pentadecyl-catechol [197]. All parts of the plant, green or dried, contain these compounds. The oily resin is not volatile or soluble in water but is soluble in alcohol. Smoke from burning the plants can contain droplets of the toxin and will affect people who are highly allergic to the toxin. The hair coat of animals may become contaminated with the resinous toxin if they are in close contact with the plants, and therefore can transmit the toxin to people who handle the animals. The animals themselves are rarely if ever affected by the urushiol, and goats and sheep have been observed eating poison ivy without apparent problems.

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