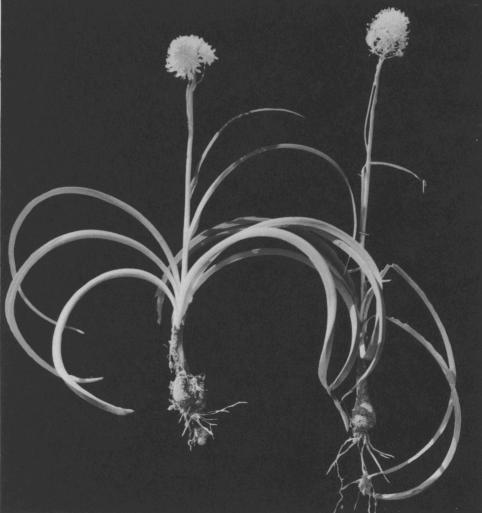
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TEXAS PLANTS POISONOUS TO LIVESTOCK



TEXAS A&M UNIVERSITY TEXAS AGRICULTURAL EXPERIMENT STATION TEXAS AGRICULTURAL EXTENSION SERVICE College Station, Texas

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TEXAS PLANTS POISONOUS TO LIVESTOCK **THE PROBLEM**—A poisonous plant is one which causes chemical or physiological disturbances when consumed by livestock. The effects may vary from mild sickness to death. Plants that interfere with growth in weight or milk production are included.

Loss from poisonous plants is one of the major economic problems in livestock production. A compilation of numerous reports indicates that the annual loss from poisonous plants in Texas is between 50 and 100 million dollars.

Approximately 80 species and varieties of poisonous plants growing in pastures and on range areas of Texas are listed alphabetically by scientific names in this publication. An additional list includes plants growing in Texas and reported to be poisonous in other areas. Some species described seldom cause trouble but are included since they have been proved toxic and may, under conditions, bring about livestock losses.

Poisoning of livestock is more commonly the result of management, range conditions, or kinds of animals rather than the presence of the plants concerned. Poor range condition from overgrazing or other conditions resulting in a lack of palatable forage is commonly the cause of poisoning. The real danger is whether or not the toxic species is grazed. Many species are seldom eaten, but some are relished by certain animals and may be taken in preference to other forage. In some instances animals will select flowers or fruits or new growth; in other situations grazing is less discriminate. Many poisonous plants are green at a time of the year when other plants are dormant. Small amounts of plant material can be lethal shortly after consumption in some cases. In others, the toxic substances are accumulative and the species must be grazed over a period of time before signs of poisoning appear.

Frequent cases of poisoning occur when hungry animals are turned into new pastures or are given access to poisonous plants near pens, watering places or along trails. Most poisonous plants are eaten because the animal is hungry and the poisonous plant is readily available.

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Poisoned animals should be treated as soon as the condition is recognized. Treatment of animals that have eaten poisonous plants is based on four principles: destruction of poisonous substance within the alimentary tract, prevention of absorption into the blood stream, promotion of excretion and evacuation, and symptomatic treatment. Poisoned animals should be removed from accessible poisonous plants and provided with feed, water and shelter. Some animals need hand feeding and watering. Specific antidotes are available for some poisons but others require symptomatic treatments such as laxatives, stimulants and good nursing.

Prevention of poisoning should be practiced when possible. Since certain plants are more poisonous to one type of animal than to another, a change in kind of livestock may be in order. Reduction of weed population and improvement of desirable forage are good management practices. Many poisonous plants can be controlled with herbicides. This method of control is practical for use on hazardous sites, and in some instances, on large-scale areas. The primary use of herbicides in weed control is to reduce the weed population to a minimum. In addition, mechanical control, modification of pasture use, correction of watering and salting, establishment of control fences and application of forage-improving management practices are most important procedures to follow to avert losses from poisonous plants.

The determination, management and control of toxic species depends upon many individuals. An operator having livestock losses normally works with the veterinarian, the county agricultural agent, or range technician in the locality. If catastrophic or continuous losses occur, the problem may move into the realm of the Agricultural Experiment Station. The research veterinarian, the range research specialist, biochemists or others may become involved in the problem. Before the operator can efficiently cope with poisonous plant problems on the range or pastures, he must be able to recognize the important species and be aware of the way in which they cause the trouble.

A field problem on poisonous plants may be brought into the research center by Extension Service or Experiment Station personnel, ranchman or practicing veterinarian. The problem, when of major importance, is directed into multi-discipline channels of research in the several departments of the Texas Agricultural Experiment Station. The program may have numerous avenues of approach. One or more of the following may be involved:

- 1. Field work to locate and determine the possible toxic species.
- 2. Feed the plants concerned to determine their toxicity and the signs and lesions produced in the affected animals.

- 3. Find laboratory animals that are susceptible to poisoning by the plant.
- 4. Determine the toxic constituent of the plant.
- 5. Determine the specific action of the poisonous principle on the animal.
- 6. Research to find specific antidotes that will neutralize the poison before it causes illness or that may be used as treatments after signs of poisoning occur.
- 7. Research to find methods of control or eradication of the more important species of poisonous plants.
- 8. Develop range management practices that will prevent or reduce losses from poisoning by plants.

Poisonous Plant Research in Texas

The problem of poisoning by plants is as old as the livestock industry. It has been recognized in Texas as long as owners and veterinarians have attempted to make differential diagnoses. Spanish missionaries described shin oak poisoning in the Southwest as early as the sixteenth century. Staff members of the Department of Veterinary Research of the Texas Agricultural Experiment Station and the USDA made many early investigations. Field Station 14, located south of Sonora and now known as the Ranch Experiment Station, was established in 1916 primarily to investigate swellhead in sheep and goats, a condition proved to be caused by sacahuista. Numerous other poisonous plants have been investigated and determined by workers on this station.

The Loco Weed Research Laboratory was established in Alpine in 1929. This work was moved to Marfa in 1945 and the name changed to Animal Disease Laboratory. In addition to the loco weeds, numerous other weeds have been investigated and poisonous species determined by workers in this station for the Trans-Pecos area. The Range Experiment Station located near Barnhart and administered by the Ranch Experiment Station in cooperation with the University of Texas was established in 1938. Emphasis on this station during the past 20 years has been aimed at determining methods for reducing losses from bitterweed. In 1947, research on poisonous plants was formalized in the Department of Range and Forestry at College Station. A bulletin entitled "Texas Range Plants Poisonous to Livestock" was published in 1955 and brought together the available information on the Texas poisonous plant problem. This publication is an expansion and updating of the 1955 work. Research on the chemistry of the toxic constituent of plants was initiated in the Department of Biochemistry and Nutrition in 1954.

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Toxic Plant Constituents

A poisonous plant may contain a single compound, several similar compounds or compounds of different chemical structure, and the poisonous principle may be organic or inorganic in nature.

The different types of toxic compounds associated with poisonous plants are alcohols, alkaloids, glycosides, phytotoxins and salts of organic and inorganic acids.

GLYCOSIDE. A glycoside is a complex compound that contains a non-sugar group attached to carbon-1 of a sugar molecule. Glucose is the sugar most frequently found in glycosides. Examples of toxic glycosides are:

1. Cyanogenetic, which releases prussic acid upon hydrolysis of the glycoside.

2. Cardiac; which yields a compound similar in structure to the sterols upon hydrolysis.

3. Saponin, which releases a steroid or terpenoid type compound upon hydrolysis.

ALKALOID. An alkaloid is a complex molecule containing nitrogen, whose free base is alkaline in reaction and capable of neutralyzing acids to form salts.

ORGANIC ACIDS. The great majority of organic acids contain a carboxyl group. Other organic acids contain the phenol group. The toxic organic acids associated with plants are fluoroacetic, oxalic and tannic acids.

PHYTOTOXINS. A vegetable protein that resembles bacterial toxins in that the injection of the



Figure 1. Guajillo, Acacia berlandieri.



Figure 2. Guajillo, Acacia berlandieri.

protein into animals causes the production of an antitoxin.

Texas Plants Poisonous to Livestock

Acacia berlandieri-Guajillo

DESCRIPTION. Guajillo is a shrub in the legume family (*Leguminosae*) with few or no prickles, twicepinnate leaves and flowers in globose heads, Figure 1. The flattened fruit pods are four to six times as long as wide and have somewhat thickened margins.

DISTRIBUTION. Guajillo grows in great density in Texas, especially in the southern part of the Edwards Plateau and in the central and northern parts of the South Texas Plains. It extends from Terrell County on the north to Webb County on the south and eastward into Bee County. Scattered plants and small patches of guajillo are found in areas adjacent to the South Texas Plains and the Edwards Plateau and in the Trans-Pecos, especially in the Chisos Mountains, Figure 2. It extends southward into Mexico.

ANIMALS POISONED. Sheep and goats are poisoned by guajillo.

CLINICAL SIGNS. Animals poisoned by guajillo develop a locomotor incoordination of the legs referred to as "limberleg" or "guajillo wobbles." The "rubbery" action of the rear limbs defies accurate description, except by means of motion pictures. At first, this sign is observed only when the animals are forced to move; after several days or weeks the incoordination is complete, with the animal recumbent and unable to rise. Affected animals retain their appetite for long periods after recumbency. Animals develop poisoning after an exclusive diet of guajillo



Figure 3. Texas Buckeye, Aesculus glabra var. arguta.

for 9 months or longer. Losses are negligible during years of favorable rainfall but may reach 50 percent in periods of extended drouth (Price *et al.* 1953).

LESIONS. No significant gross pathology has been observed.

POISONOUS PRINCIPLE. The leaves of guajillo contain three sympathomimetic amines. Camp and Lyman (1956) identified one of the compounds as N-methyl-beta-phenylethylamine, and Camp *et al.* (1963) isolated and identified two additional amines, tyramine and N-methyl tyramine, from this plant.

The oral administration of N-methyl-beta-phenylethylamine hydrochloride to sheep (375 milligrams per kilogram of animal weight) produces increased respiration, nervousness, tremors and ataxia of the hindquarters. The oral lethal dose of the amine hydrochloride in sheep is approximately 450 milligrams per kilogram of body weight.

MANAGEMENT AND TREATMENT. Guajillo is a valuable browse plant but should not be grazed to the exclusion of other range forage. When poisoning does occur, supplemental feeding and reduced stocking rates will lower the percentage of poisoning. If possible, the flock should be moved to a range free of guajillo when signs of illness first appear. Few new cases will develop after removal from guajillo range, and many early cases will show marked improvement and eventual recovery if place on an adequate ration. Guajillo may be killed with herbicides.

Aesculus glabra var. arguta-Texas Buckeye and

Aesculus pavia-Red Buckeye

DESCRIPTION. Buckeyes are trees or shrubs with opposite palmately compound leaves with 5 serrate leaflets. The flowers are polygamous in large erect panicles with a tubular, 5-lobed calyx; 4 or 5-clawed and unequal petals; 6 to 8 stamens and a 3-celled ovary. The fruit is a leathery capsule with one to three large glossy brown seed. Buckeyes are members of the horse-chestnut family (Hippocastinaceae), Figure 3. The Mexican buckeye, Ungnadia speciosa, is a member of the soapberry family (Sapindaceae) and, as the name implies, is not a true buckeye. Apparently, it is not poisonous.

DISTRIBUTION. Two species of *Aesculus* are found in Texas. These occur from East Texas to the eastern part of the Edwards Plateau with some record from the canyons in the Rolling Plains, Figure 4. Buckeye usually is found on river bottoms and along the banks of streams. Shrub forms are most common as undergrowth in canyons and on valley floors.

ANIMALS POISONED. Cattle, horses, sheep and swine have been poisoned by buckeye. Also, children have been poisoned by eating the nut-like seed.

CLINICAL SIGNS. Animals poisoned by buckeye have an uneasy, staggering gait, weakness and trembling. Mucous membranes are congested, and the pupils are dilated. The animals become depressed and go into a stupor and coma before death. Some animals will vomit, and colic may be seen in horses (Tehon *et al.* 1946).

POISONOUS PRINCIPLE. At least seven species of *Aesculus* have been reported as poisonous when eaten by animals. Several glycosides have been isolated from species of buckeye (Muenscher 1951), two of which are aesculin and fraxin. At least one species contains a narcotic alkaloid.

MANAGEMENT AND TREATMENT. In the early spring, animals should not be allowed to graze in pastures where there are buckeye seedlings and sprouts, until grass or other forage is abundant. If



Figure 4. Buckeye, Aesculus spp.

cases are found soon enough, a veterinarian should be called to administer purgatives and stimulants.

Control of buckeye can be obtained by basal applications of 32 pounds of 2,4,5-T L.V. ester in 100 gallons of diesel oil. The best kills were obtained experimentally on trees with stems under 6 inches in diameter which were notched around the bole near the ground. The kill on larger trees was erratic but effective (Sperry and Pond 1957). Seedlings and plants less than 2 feet in height can be controlled by foliage spraying. Derscheid and Ferrell (1955) reported control of red buckeye with foliage sprays of 2,4-D and 2,4,5-T emulsions in water at the rate of 4 pounds per 100 gallons of water.

Agave lecheguilla-Lechuguilla

DESCRIPTION. Each lechuguilla plant consists of a crown bearing 20 to 30 thick, fleshy leaves, 1 to 1½ inches wide and from 12 to 24 inches long. The leaves are ascending with recurving marginal prickles and terminate in a sharp spine. The flowering scapes are 6 to 12 feet long and bear a terminal panicle with short branches, Figure 5. Numerous black flattened seed are produced in a leathery, 3-celled capsule. Each plant requires 10 to 15 years to reach maturity, then flowers, sets fruit and dies. Reproduction is by both seed and offsets around the base of the parent plant. The formation of offsets is stimulated by browsing of the flowering stalks. The century plant

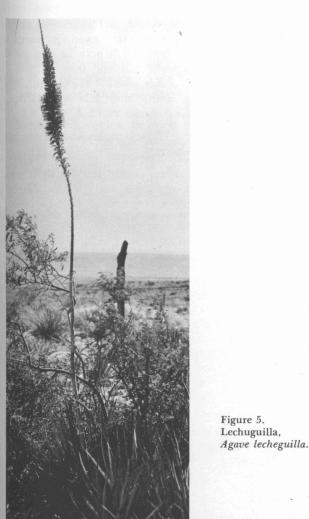


Figure 6. Lechuguilla, Agave lecheguilla.

and maguey also are species of *Agave*, which belongs to the amaryllis family (Amaryllidaceae).

DISTRIBUTION. Lechuguilla occurs in Texas westward from the southern and western portions of the Edwards Plateau across the Trans-Pecos to the Franklin Mountains at El Paso. The heaviest infestations are in the low rainfall areas paralleling the Rio Grande and on the dry hills of the Trans-Pecos, Figure 6. This plant extends westward into New Mexico and south into Mexico. Lechuguilla occurs characteristically on low limestone hills, dry valleys and bordering cayons west of the Pecos river.

ANIMALS POISONED. Sheep and goats are poisoned most frequently by lechuguilla. Although cattle are poisoned less frequently, lechuguilla poisoning may be a series problem in this species during drouth or when there is a shortage of good feed.

CLINICAL SIGNS. Poisoned animals become listless and make no effort to keep up with the flock or herd. There is a progressive decrease in consumption of water and food, with eventually a complete loss of appetite. The animals become progressively weaker and emaciated, and a short period of coma usually precedes death. Jaundice is marked. The skin and visible mucous membranes are golden yellow to orange. A yellow excretion adheres to the internal canthus and the nostrils are occluded with a yellow, tenacious discharge. The urine is occasionally port wine in color. Photosensitization with edematous swelling of face, ears and intermandibular space is seen frequently. Animals experimentally fed as little as 1 percent of their body weight of lechuguilla leaf material have developed signs of poisoning and died.

LESIONS. There is a pronounced icterus. The liver is light brownish yellow, and the capsule is usually

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thickened. The gallbladder is usually distended with normal appearing bile, but occasionally the bile has a doughy consistence. Compression along the edge of the cut surface of the liver results in the expression of numerous plugs of thickened bile from the severed bile ducts. The kidneys are swollen and greenish black. There are numerous pinpoint grey specks under the capsule of the kidney.

In some cases there is a decrease in the number of erythrocytes. There is a leucocytosis with polymorphonuclear leucocytes comprising as high as 90 percent of the leucocytes. The nonprotein nitrogen content of the blood is greatly increased, and albuminuria is evident (Mathews 1937).

POISONOUS PRINCIPLE. Lechuguilla poisoning is the combined action of two principles, photodynamic agent and nephrotoxic and hepatotoxic agent. The chemical constitution of the photodynamic agent has not been established. Mathews (1938) identified the hepatotoxic agent as a saponin. Wall *et al.* (1954) have isolated the steroidal sapogenin smilagenin from this plant.

MANAGEMENT AND TREATMENT. Lechuguilla poisoning usually occurs from grazing the leaves during the winter and spring but may appear at any time during the year, Figure 7.

When poisoning occurs, animals should be removed from pastures where lechuguilla grows, or the number should be reduced to where there is an adequate amount of other forage. Supplemental feeding is beneficial in preventing losses. When animals first exhibit signs of illness they should be placed in the shade and fed nutritious feed. Most of the animals that develop severe jaundice will die.



Figure 7. Grazed lechuguilla.

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Aloysia lycioides–Whitebrush, Beebrush

Figure 8. Whitebrush,

Aloysia lycioides.

DESCRIPTION. Whitebrush is a shrubby plant in the verbena family (Verbenaceae). It has entire or sparsely denticulate, oblong to obovate dull green leaves. The small white or bluish flowers are in open, leafy panicles of elongated spikes or spikelike racemes, Figure 8.

DISTRIBUTION. Whitebrush is frequent to abundant in Central, West and South Texas and extends into New Mexico and Mexico. It may be present as undergrowth in open woodland and may be prominent in overflow and flood areas. Whitebrush frequently increases in stand and density following mechanical and chemical brush control.

ANIMAL POISONED. Horses, mules and burros are affected by whitebrush.

CLINICAL SIGNS. When grazed heavily by horses, whitebrush produced weakness and death. Horses probably have to graze it from 30 to 40 days before symptoms develop (Mathews 1942a).

POISONOUS PRINCIPLE. A water-soluble toxin is present in whitebrush.

MANAGEMENT AND TREATMENT. Whitebrush is a valuable honey plant and has some value for browse. The most effective control of whitebrush has been by foliage spraying with 1¼ pounds MCPA with 1 gallon of diesel oil and enough water to make 8 gallons of solution per acre. Spraying should be done after plants are fully leafed and in full bloom (McCully 1959).

Amaranthus spp.-Amaranth, Carelessweed, Pigweed

DESCRIPTION. Carelessweeds are annual weedy herbs and belong to the amaranth family (Amaranthaceae). They are so commonly known that little description is necessary, Figure 9. Twenty-three species are recorded for Texas. The growth form of the various species ranges from prostrate to branching upright. Most species, especially in early growth, are palatable to domesticated animals and are relished by swine; hence, the name "pigweed" often is applied.

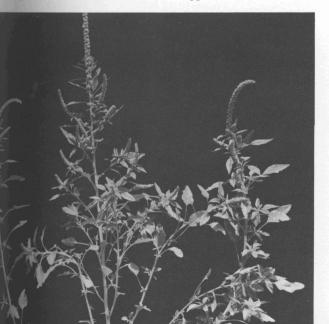
DISTRIBUTION. Amaranths are present to abundant in almost every disturbed pasture area. They are common in barnyards having rich, moist soil.

POISONOUS PRINCIPLE. It is assumed that potassium nitrate is the toxic compound, although carelessweed poisoning does not always show the classical signs (brown blood) of nitrate poisoning. Analyses of plants at time of losses were as follows: green plant in a rapid stage of growth, 0.066 percent potassium nitrate; plants that had stopped growing as a result of inadequate moisture, 0.828 percent potassium nitrate; and plants that were wilting because of inadequate moisture, 2.874 percent potassium nitrate (Dollahite and Camp 1956). For further information on nitrate poisoning, see *nitrates* under *Special Problems*.

MANAGEMENT AND TREATMENT. Frequent deaths have occurred in animals grazing amaranths and eating amaranth hay. If there are pregnant animals in the herd, there may be abortions within 2 weeks following the initial deaths. Under certain conditions, these losses reach alarming numbers. Losses almost always occur when animals are eating moisturedeficient plants.

If conditions permit, amaranths can be killed by spraying with 2,4-D or other herbicides. Rea and Wiese (1957) reported $\frac{1}{2}$ pound, acid equivalent, of silvex in 50 gallons of water per acre killed amaranths 10 inches high or less in corn up to 30 inches tall. Hand pulling when the soil is moist or mowing or chopping localized areas are good control practices.

Figure 9. Carelessweed, Amaranthus spp.



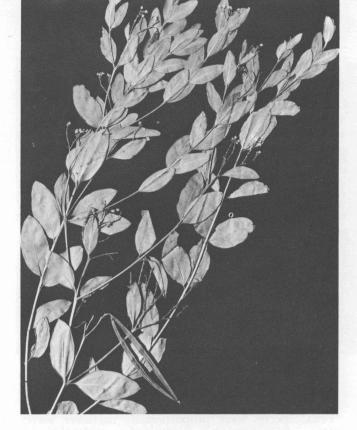


Figure 10. Dogbane, Apocynum cannabinum.

Apocynum cannabinum-Dogbane, Indian Hemp

DESCRIPTION. Dogbanes are perennial, herbaceous, erect, leafy herbs with pink or whitish flowers, Figure 10. They belong to the dogbane family (Apocynaceae).

DISTRIBUTION. Dogbane is found along streams and in moist situations over most of Texas. Another species, *A. androsaemifolium*, grows on range areas of West Texas and is considered toxic.

ANIMALS POISONED. Dogbane is poisonous to horses, cattle and sheep.

CLINICAL SIGNS. Both green and dried dogbane are poisonous. From 15 to 30 grams will cause death in horses and cattle (Muenscher 1951).

MANAGEMENT AND TREATMENT. Dogbane is not considered a serious problem on the range since domesticated animals usually avoid these plants. The bitter, milky, rubber-containing juice presumably renders dogbane unpalatable, and plants are grazed only when other forage is not available.

Asclepias latifolia-Broad-leafed Milkweed

DESCRIPTION. Broad-leafed milkweed is a perennial plant with stout simple stems and four or more pairs of large thick leaves, which are not more than 1.5 times as long as wide. The flowers are greenish and give rise to two to four smooth pods about $1\frac{1}{2}$ inches long, Figure 11. Of the 30 species of Asclepias recorded for Texas, broad-leafed milkweed is noteworthy for its robust nature and leaf size, while other



Figure 11. Broad-leafed milkweed, Asclepias latifolia.

Texas species have proportionally narrower leaves. The milkweeds belong to the family Asclepiadaceae.

DISTRIBUTION. Broad-leafed milkweed is frequent to abundant over much of the range country of the Trans-Pecos, the Plains area and the central and western portion of the Edwards Plateau, Figure 12. It is found from Nebraska to Utah, south to Texas and west to Arizona. Broad-leafed milkweed is a common constituent of grasslands and is frequent along trails and roadsides. As with many weeds of low palatability, this species increases on heavily grazed pastures.

ANIMALS POISONED. A quantity of broad-leafed milkweed fed in as small an amount as the equivalent of 0.5 percent of the body weight of the animal has poisoned cattle (Mathews 1932), and less than 0.15 percent body weight of plant material has poisoned sheep and goats (Tunnicliff and Cory 1930). The young plant is more toxic than the mature plant. Cattle graze frost-killed plants readily, but no signs of poisoning have been observed.

SIGNS AND LESIONS. Poisoned animals are restless, show signs of abdominal pain, excessive salivation and labored respiration. The liver may be swollen. The liver, spleen, kidneys and sometimes the lungs are congested. There is an enteritis which may be hemorrhagic. Poisoned animals apparently die quickly without struggling (Tunnicliff and Cory 1930).

MANAGEMENT AND TREATMENT. Since most cases of poisoning usually occur on pastures in which broadleafed milkweed is abundant, management practices to improve the range condition are the best measures of prevention. The removal of plants along trails and in holding traps may prevent many losses, especially



Figure 12. Broad-leafed milkweed, Asclepias latifolia.

when hungry animals are being trailed. When largescale poisoning occurs, animals should be moved to clean pastures or penned and fed until the range vegetation has improved.

Asclepias subverticillata-Horsetail Milkweed

DESCRIPTION. Horsetail milkweed is an erectstemmed plant growing to a height of 5 feet from horizontal rootstocks. The narrow leaves are predominantly in whorls of three, or opposite with margins rolled backwards. The umbellate flowers are greenish-white and give rise to seed pods 1 to 3 inches long, Figure 13. The seed have tufts of long silky hairs.

This species is similar to A. verticillata but differs from it by having more extensive rootstock and a

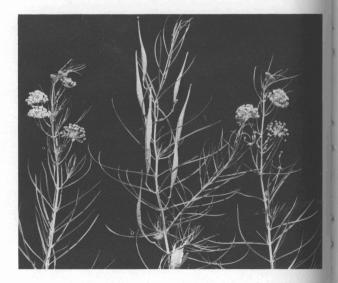


Figure 13. Horsetail milkweed, Asclepias subverticillata.

tendency to produce dwarfed, small-leaved axillary branches.

DISTRIBUTION. This plant is most abundant in the western part of Texas, but collections are also recorded from the South Texas Plains and the Gulf Coast Prairie, Figure 14. It is frequent in Northern Mexico and ranges into Arizona, Colorado and Utah. Horsetail milkweed may be abundant in open pastures, along arroyos, draws, bar ditches and roadsides.

ANIMALS POISONED. Sheep, cattle, horses, chickens and turkeys have been poisoned by horsetail milkweed. Two to 3 ounces may kill a sheep.

CLINICAL SIGNS. Animals poisoned by horsetail milkweed have a rapid and weak pulse, difficult breathing and respiratory paralysis. They show signs of loss of muscular control, trembling, staggering, falling and violent convulsion. Elevated temperature, salivation, bloating and dilation of the pupils may occur.

LESIONS. Congestion is found in the walls of the fourth stomach, duodenum, jejunum, ileum and cecum. The lungs and kidneys also are congested in some cases. The urinary bladder usually is empty.

POISONOUS PRINCIPLE. Several toxic compounds, some glycosidal in nature, have been reported for *A. galioides*, which, according to earlier determinations, is the same as this species. Cold alcohol extracts containing glycosides have caused narcosis in experimental animals without congestion of the nervous system.

MANAGEMENT AND TREATMENT. Milkweeds are distasteful to animals and are not commonly grazed





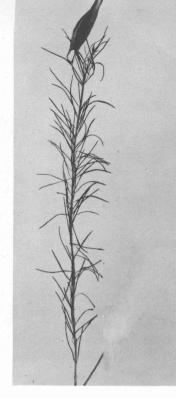


Figure 15. Whorled milk-weed, Asclepias verticillata

unless hungry animals are confined to milkweedinfested areas. Grubbing is not considered a practical control measure because of the difficulty of removing all the underground rootstocks which give rise to new growth. Where poisoning is a problem, small localized areas may be fenced off or the animals moved to pastures free of milkweed. Range management practices to maintain good forage are the best protection from poisoning. Although most of the animals reaching the convulsive stage of milkweed poisoning may die, some of them will recover. They should be moved to shade, kept quiet and given plenty of good feed and water. There is no specific treatment but sedatives and laxatives may be helpful (Tehon *et al.* 1946).

Asclepias verticillata–Whorled Milkweed

DESCRIPTION. Whorled milkweed is a slender, smooth plant which grows to a height of 3 feet from a perennial rootstock. The narrow leaves have rolled margins and are arranged in whorls of two to six at each node. The umbellate flowers are greenish-white. The seed pods are 2 to 3 inches long and produce numerous flattened, reddish-brown seed which bear long silky hairs, Figure 15.

DISTRIBUTION. Whorled milkweed is frequent throughout East Texas with occasional records from the Edwards Plateau and the Trans-Pecos, Figure 16. It ranges from Maine to Florida, west to Texas and north to Colorado and North Dakota. This weed normally grows in grasslands and marginal woodland areas and may persist in dry habitats. It may flourish along ditches, roadsides and in cultivated areas.

ANIMALS POISONED. Sheep, cattle, horses, chickens and turkeys may be poisoned by whorled milkweed.



Figure 16. Whorled milkweed, Asclepias verticillata.

CLINICAL SIGNS, LESIONS, MANAGEMENT AND TREAT-MENT. The signs and lesions of this poisoning are similar to those of horsetail milkweed, and the same prevention and treatment methods are recommended.

Astragalus emoryanus-Peavine, Red-stemmed Peavine, Emory Loco

DESCRIPTION. Peavine is an annual legume with a slender taproot and slender, decumbent stems. The stems usually branch at the base and bear odd-pinnate leaves with acute-tipped leaflets. The glabrous seed pods are 2-celled and contain about a dozen seed. A. emoryanus is closely related to and often grows in association with other species of Astragalus such as loco and garboncillo. One species called Nuttall peavine, A. nuttallianus, frequently grows with A. emoryanus, and certain amounts have inadvertently been included in the feeding experiments. This species probably is also poisonous under the same conditions. A. emoryanus, Figure 17, was separated taxonomically from A. nuttallianus by Rydberg in 1927. The obvious character of separation is: A. nuttallianus has truncate or emarginate leaflets; those of A. emoryanus are acute.

DISTRIBUTION. Peavine has been recorded for every area in Texas except the Piney Woods of East Texas and the southern portion of the South Texas Plains. It is most abundant and most troublesome in the Trans-Pecos area, Figure 18. It is distributed from Texas to California and Mexico. Nuttall peavine is also scattered over Texas with greatest abundance on the Edwards Plateau and Blackland Prairie. Peavine grows in short-grass areas and on open ground, usually assuming a prostrate habit. A single plant may cover only a few square inches or it may be 2 or 3 feet in diameter. When growing in moist, ANIMALS POISONED. Red-stemmed peavine grown on limestone soils is toxic to cattle, sheep and goats (Mathews 1940b). Peavine also has been reported to be poisonous when growing on red sandy soil in the Llano River area (Sperry *et al.* 1952). Peavine growing on soils of igneous origin is not poisonous and is considered good forage in such sections of the state. However, cattle may die from bloat after eating peavine growing on any soil. The apparent mineral relationship to the toxicity has been studied by giving calcium gluconate, calcium chloride and monosodium phosphate before experimental feeding. Sheep given the mineral compounds were poisoned more readily than when peavine was fed alone (Sperry *et al.* 1952).

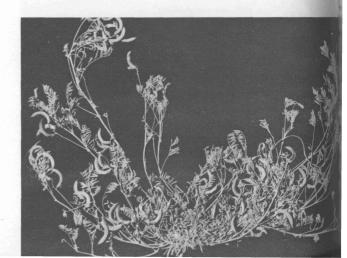
CLINICAL SIGNS. Under range conditions, the first evidence of toxic effects on cattle is momentary collapse of the leg muscles when the animal attempts a sudden movement. This is overcome after a short hesitation, and the animal then moves normally. As the toxicity progresses, the incoordination of the hind legs becomes more noticeable when the animal is forced to move. Loss of weight and paralysis are evident in later stages. A slow recovery usually is made when affected animals are given feed, water and shade (Mathews 1940b).

In addition to the incoordination of muscles of the rear legs, sheep develop a rasping noise due to labored respiration, which may be aggravated by exertion and heat. Progressive loss of weight and sporadic deaths are observed. Some may develop a cough that will recur following exertion and may persist for the life of the animal. Goats experimentally fed peavine developed similar muscular incoordination. Clinical signs were produced by Mathews (1940b) by feeding 0.73 to 2.0 percent of the body weight of peavine, usually in a 2-day period.

LESIONS. Gastroenteritis is usually present in acute poisoning, but there are no consistent lesions in chronic poisoning.

MANAGEMENT AND TREATMENT. Since peavine normally is short lived, the usual management practice is to remove animals from pastures for the dura-

Figure 17. Peavine, Astragalus emoryanus.



tion of peavine growth. Peavine is not a problem every year, and pastures free from infestation may be held in reserve for use during problem years. Light stocking of infested pastures will limit peavine poisoning most years. Since peavine often grows earlier than nontoxic plants, the prevention of peavine poisoning during early spring is often a problem which requires daily attention. Livestock should be removed from peavine pastures when the first signs of poisoning occur and placed in shaded pens with feed and water. Bitter tonics may be administered to stimulate the appetite. Most of the animals will recover if treatment is initiated early in the course of the disease.

Peavine is susceptible to herbicides and best kills, 90 percent or better, have been obtained by spraying with 4 pounds of the esters of 2,4-D and 2,4,5-T in 25 gallons of water per acre. Since germination may continue throughout the growing season, more than one treatment may be required.

Astragalus mollissimus-Loco-weeds

Three that were considered species of Astragalus have been reduced to variety status of A. mollissimus (Gould 1962). Although these three locos are similar in toxicity and management, the descriptions and distributions differ and are presented for each variety concerned. Other information concerned applies to all three varieties.

Astragalus mollissimus var. coryi (A. argillophilus)–Yellow-flowered Loco

DESCRIPTION. Yellow-flowered loco is a perennial, many-branched legume with a woody root and with



Figure 19. Yellow-flowered loco, Astragalus mollissimus var. coryi (A. argillophilus).

tips of fruiting branches ascending or erect. The entire plant is covered with matted woolly or silky hairs. The leaves have long petioles and usually 11 or 12 pairs of leaflets. The flowers are yellowish white, rarely yellow or purplish, Figure 19.

DISTRIBUTION. Yellow-flowered loco apparently is restricted in its general range in Texas to about eight counties with south Reagan County being the center of distribution, Figure 20. Yellow-flowered loco usually is found in clay soils in grasslands and along draws and in depressions or lakebeds on the divides.

Astragalus mollissimus var. earlei (A. earlei)–Earle Loco

DESCRIPTION. Earle loco is a perennial legume with a woody taproot and numerous decumbent stems. The leaves, composed of an odd number of leaflets, are $\frac{1}{2}$ inch long and about $\frac{1}{8}$ inch thick, Figure 21.



Figure 20. Yellow-flowered loco, Astragalus mollissimus var. coryi (A. argillophilus).



Figure 18. Peavine, Astragalus emoryanus.

DISTRIBUTION. Earle loco is found chiefly in seven counties of the Big Bend area of Texas, Figure 22, and extends westward into New Mexico and south into Mexico. It is most abundant in draws and flats, and in low rainfall years is restricted to these areas. In good rainfall years it is found over much of the range area on both igneous rock and limestone soils. Earle loco grows in grassland, regardless of condition, and is commonly associated with buffalograss, the gramagrasses, cane and silver bluestems and the lesser grasses such as species of *Tridens* and *Muhlenbergia*.

Astragalus mollissimus var. mollissimus Woolly Loco, Purple Loco

DESCRIPTION. Woolly loco is a stout, decumbent, many-branched perennial legume. The leaves have 19 to 29 ovate-oblong leaflets which are densely pubescent. The thick, woody root gives rise to stem branches which tend to lie close to the ground, Figure 23.

DISTRIBUTION. Woolly loco is found primarily in the Rolling and High Plains, although a few collections have been recorded for the Big Bend area, Figure 24. It is found as far north as South Dakota and east to central Nebraska and Kansas and in western Oklahoma. Its western limits approach central Colorado and New Mexico. It usually grows in localized patches, commonly in flooded draws, but is frequently associated with buffalo and blue grama grassland.

The following applies to the preceding three locos:

ANIMALS POISONED. Horses are particularly susceptible but cattle, sheep and goats may develop loco poisoning.

CLINICAL SIGNS. Cases of poisoning usually occur from eating locoweeds in the early growth stages. These stages are governed by the distribution of rain-

Figure 21. Earle loco, Astragalus mollissimus var. earlei.





Figure 22. Earle loco, Astragalus mollissimus var. earlei.

fall. Abundant growth may occur throughout the fall, winter and spring. The acute form of the disease (locoism) develops from eating large amounts of loco, and a chronic form of the disease may occur if small quantities are eaten over an extended period of time. Signs of locoism have been produced in cattle after they have consumed about 90 percent of their body weight of the plant. However, it usually takes from 200 to 350 percent of their body weight of the plant, eaten over a period of several months, to produce death in cattle, sheep and goats. About 30 percent of body weight consumption will produce signs in a horse and about 75 percent may be fatal (Mathews 1932). Animals poisoned from loco are extremely nervous. The most common signs are slow, staggering gait, rough coat, staring look, emaciation and muscle incoordination. Abortions with maceration of the fetuses will occur in affected animals. The signs of locoism may not be apparent when the animal remains quiet, but when subjected to strenuous muscular exertion, signs of locoism appear, and complete recovery rarely occurs.

LESIONS. The walls of the pregnant uterus of the animals affected with locoism usually are slightly edematous, and the foetal membranes are very edematous. In extreme cases the foetal membranes are between 2 and 3 inches thick and of a gelatinous consistency. A marked edema of the mucosa of the stomach and frequent ulcerations around the pylorus may be present. Microscopic sections of the brain and other tissues show generalized edema (Mathews 1932).

POISONOUS PRINCIPLE. Fraps and Carlyle (1936) described the isolation of a highly toxic concentrate, called "locoine" from *A. earlei*. Fraps and Wender



Figure 23. Woolly loco, Astragalus mollissimus var. mollissimus (A. mollissimus).

(1944) reported locoine and closely related compounds present in A. mollissimus, A. wootoni and A. earlei. These workers indicated that some of the compounds may have resulted from isomerism in the isolation procedure.

MANAGEMENT AND TREATMENT. When other forage is available most animals will not eat locoweeds. If they do eat locoweeds, animals frequently acquire the habit, eventually with fatal results. Animals raised where locoweeds are common are less likely to eat them than imported livestock. Susceptibility among animals of the same species is variable. When palatable range forage is scarce, the use of good supplemental feed tends to reduce the amount of loco consumed. Locoed animals should be removed from infested pastures and placed on good feed. Bitter tonics may be given to stimulate the appetite.

Loco can be killed by spraying with the L.V. ester of 2,4-D in water at 1 pound per acre. Aerial

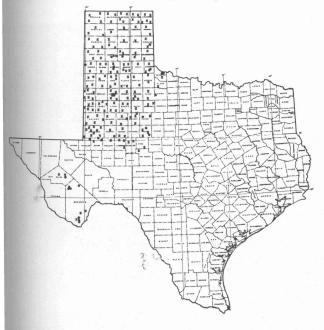


Figure 24. Woolly loco, Astragalus mollissimus var. mollissimus (A. mollissimus).



Figure 25. Garboncillo, Astragalus wootoni.

applications have been highly satisfactory when 4 gallons are applied per acre. The OC 20 boomjet spray on ground equipment applied in 15 gallons of mixture per acre has given satisfactory control. Smallscale and spot treatments applied with hand sprayers at 4 pounds of 2,4-D acid equivalent in 100 gallons of water have obtained good kills. In general, plants sprayed from October through April are killed while later spraying may obtain topkill only, with regrowth occurring in late summer following rains. When a spraying program is inaugurated, some management precautions should be followed, as sprayed plants are as palatable and as toxic as unsprayed plants. The removal of animals from a treated pasture until the treated loco has dried up prevents possible poisoning and gives range vegetation a rest and growing period. This in itself should pay a good dividend in additional forage.

Plant grubbing to 2 or 3 inches below the surface is a common practice. This procedure may need repeating, as experiments show that a large percentage of the grubbed plants regrow during the season, especially with favorable rainfall.

Astragalus wootoni-Garbancillo, Rattle-weed Loco

DESCRIPTION. Garbancillo is a much-branched, annual legume with erect, hairy stems that vary from about 3 to 12 inches long, depending on habitat. The leaves have 9 to 19 linear-oblong leaflets, hairy beneath and smooth above. The pink or purplish-towhite flowers are in axillary racemes. The plant is conspicuous in fruiting because of the large, 1-celled, inflated pods, Figure 25.

DISTRIBUTION. In Texas, garbancillo is restricted to the Trans-Pecos area, Figure 26. It is known as a common weed in the low rainfall areas of southern New Mexico, eastern Arizona and northern Mexico. Garbancillo is most abundant in valley sites which accumulate runoff water from the surrounding hills and is common in bar ditches, along trails and around earthen tanks. It often occurs on rocky slopes and hills but usually as reduced plants. It is frequently associated with dense growth of buffalo, curly mesquite and grama grasses.

ANIMALS POISONED. Garbancillo is less palatable than most loco, but cattle, horses, sheep and goats are poisoned by this plant.

SIGNS, LESIONS, POISONOUS PRINCIPLE AND TREAT-MENT. See A. mollissimus, locoweeds.



Figure 26. Garbancillo, Astragalus wootoni.

MANAGEMENT. Since garbancillo is an annual, grubbing and removal of plant material around tanks, along roadways and in other localized hazard sites often is effective. If the plant is widely scattered or abundant over large areas and is being grazed to the extent of poisoning, the removal of animals from the pasture may be necessary. The plant dies out in early summer after fruiting.

Spot and localized area control is feasible if animals cannot be moved from problem areas. The spraying of young, vigorous plants with 4 pounds of 50-50 mixture of the L. V. esters of 2,4,5-T and 2,4-D in 100 gallons of water has given satisfactory control.

Baileya multiradiata-Desert Baileya

DESCRIPTION. Desert baileya is a low-growing composite densely covered with short hairs with a woolly appearance. It has numerous, alternate, toothed leaves on numerous basal branches. The prominent yellow-flowered heads on elongated stems are present from spring until late fall, Figure 27.

DISTRIBUTION. This species occurs frequently in the seven most-western counties of Texas, especially in the low rainfall areas of the Big Bend, Figure 28. From this area it extends to southern Utah, Nevada, Southern California and south into Mexico. Desert baileya often is abundant on sandy and gravelly soils in the semidesert grassland and shrub areas. It often is conspicuous along roadsides and over extensive range areas.

ANIMALS POISONED. Sheep, goats and rabbits are susceptible experimentally to bailey poisoning. Sheep are the only animals reported to be poisoned on the range. Rabbits are more susceptible than sheep, having died within 2 days following feeding on green plant material. All parts of the green or dried plant are poisonous. The flowers and seed heads are more poisonous than the leaves. The flowers and seed heads are palatable to sheep and will be eaten even though an ample amount of green grass is available (Dollahite 1960).

CLINICAL SIGNS. Sheep and goats show loss of appetite, emaciation and regurgitation of green material with accumulations around the mouth. They become depressed, sluggish, weak and develop pneumonia with audible rales. Some of the animals tremble and develop opisthotonos; they are unable to right themselves or to remain upright when helped. They develop incoordination, especially of the rear legs, a rapid, thumping or pounding heart action and excrete red urine.

LESIONS. Lesions observed in sheep and goats include petechial and ecchymotic hemorrhages on the endocardium and epicardium, and pneumonia with large areas of hepatization. Many have ascites of amber to red. The livers are congested, friable with subcapsular petechiae and usually contain unclotted blood. The kidneys are dark and congested with some subcapsular hemorrhages. The spleen is softer and thicker than normal. Severe hemorrhagic gastritis with ulcers in the abomasum and severe enteritis with some hemorrhage in the duodenum and caecum is present. The brain usually is congested and often edematous.

MANAGEMENT AND TREATMENT. Since the flowers and seed heads are palatable to sheep, these animals cannot be maintained economically on range that is heavily infested with baileya. Losses in Presidio County were between \$50,000 and \$100,000 annually in 1958 and 1959. When sheep first show signs of baileya poisoning, they should be moved to range







Figure 28. Desert baileya, Baileya multiradiata.

where the plant does not grow. Baileya is susceptible to 2,4-D if sprayed following periods of rainfall while the plants are in a vigorous stage of growth. Spraying done during dry periods is ineffective.

Cassia occidentalis-Coffeesenna, Stypicweed

DESCRIPTION. An annual legume naturalized from the Tropics. The compound leaves have 4 to 6 pairs of ovate-lanceolate, acute-tipped leaflets. The longlinear, glabrous pods are 4 to 6 inches long, Figure 29.

DISTRIBUTION. Coffeesenna usually grows in waste places in East and South Texas. It is found from Virginia to Florida and west to Texas. CLINICAL SIGNS. After animals have been fed 1 percent of their body weight of coffeesenna for about 7 days they refuse to eat, become weak and may develop diarrhea. They may have red urine. Animals usually die within 24 hours after signs of acute illness develop (Dollahite and Henson 1963).

LESIONS. The only gross lesion which occurs in rabbits, sheep and cattle is generalized pulmonary edema. In addition, cattle often have portions of one or more skeletal muscles which are pale and in some instances almost ivory-colored. Microscopically, there is a degeneration of skeletal muscles and varying degrees of toxic hepatitis and nephritis.

ANIMALS POISONED. Rabbits, sheep and cattle have been poisoned with beans and leaves of this plant.

MANAGEMENT AND TREATMENT. Cattle should not be allowed access to coffeesenna unless there is ample forage available.

Centaurium beyrichii-Mountain Pink, Centaury Centaurium calycosum-Buckley Centaurium, Centaury

DESCRIPTION. Species of *Centaurium* are annual or biennial herbaceous plants in the gentian family (Gentianaceae). The leaves are simple, entire, commonly opposite and sessile. The pink flowers are produced in simple or compound cymes from March to November, Figure 30.

DISTRIBUTION. The mountain pink grows on dry soil in Central and West Texas. It is found eastward to Arkansas. Buckley centaurium normally grows in moist habitats in Central and West Texas. It is found from Missouri to New Mexico.



Figure 29. Left: Coffeesenna, Cassis occidentalis.

Figure 30. Right: Mountain Pink, Centaurium beyrichii. ANIMALS POISONED. In 1959 mountain pink was suspected of causing cattle and goat losses north of Monterrey, Mexico. Feeding of mountain pink produced illness in 5 goats, death in 4 of the 5, and produced illness in 1 of the 2 sheep. The sheep were fed a smaller quantity than the goats. Buckley centaurium produced signs and lesions similar to those produced by mountain pink when fed to one goat. This plant was suspected of being the cause of death in bighorn sheep on the Black Gap Game Reserve in Brewster County (Dollahite and Allen 1962).

CLINICAL SIGNS. Lack of appetite, abdominal pain and diarrhea are signs of centaury poisoning.

LESIONS. Poisoning by centaury produces lesions of congestion in the liver and kidneys, severe gastroenteritis with hemorrhage and ulcers in the rumen and the abomasum.

Cephalanthus occidentalis-Buttonbush

DESCRIPTION. Buttonbush is a shrub growing to about 8 feet with 4-angled branches. The leaves are opposite or in threes. The flowers are in peduncled, terminal or axillary globose heads, Figure 31.

DISTRIBUTION. This shrub is found around springs, streams and in swamps. It is cosmopolitan in distribution throughout Texas and most of temperate North America.

POISONOUS PRINCIPLE. Although buttonbush contains two glycocides, cephalin and cephalanthin, which produce an emetic action, paralysis, spasms and hemolysis (Cary *et al.* 1924), it is not considered palatable and livestock losses are negligible.

Cicuta maculata-Spotted Waterhemlock

DESCRIPTION. Spotted waterhemlock is a perennial herb in the parsley family (Umbelliferae). The two to three palmately compound leaves are alternate with clasping petioles and serrate margins, Figure 32.

Figure 31. Cephalanthus occidentalis, Buttonbush.



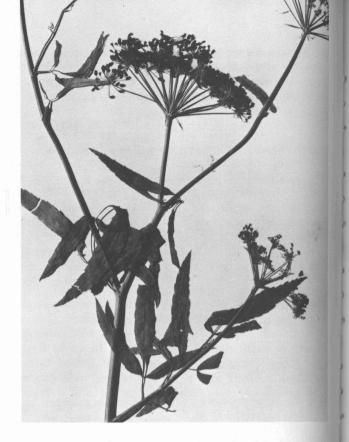


Figure 32. Cicuta maculata, Spotted waterhemlock.

The major veins of the leaves extend to the notches between the teeth. The stems are purple striped or mottled and hollow except for partitions at the nodes. The root stocks are short and thickened and have air cavities separated by plate-like cross partitions.

DISTRIBUTION. Spotted waterhemlock has been recorded in the Post Oak Savannahs, the Blackland Prairies, the Edwards Plateau and the Rolling Plains. It is distributed throughout much of the eastern United States. Waterhemlock, *C. douglassii*, is found throughout the western United States. Hemlocks are found in wet meadows and pastures, along streams and around permanent springs.

POISONOUS PRINCIPLE. Cicutoxin is the poisonous principle of these plants. It is abundant in the roots but is also found in the young plant. Mature stems and leaves are not considered toxic. The toxin is poisonous to all warmblooded animals, including humans. The greatest danger comes in the spring when young plants may be grazed. Except for localized areas, waterhemlock poisoning is not a livestock problem in Texas.

Colubrina texensis-Hogplum

DESCRIPTION. Hogplum is a shrub of the buck thorn family (Rhamnaceae). The leaves are alternate, petioled, entire or denticulate. The shrub branches freely and some of the branches form slender spines, Figure 33. The small, brownish fruit is a hard, three-celled, drupelike capsule. DISTRIBUTION. Hogplum is most commonly found in the Cross Timbers, Edwards Plateau and the South Texas Plains areas. It grows on rocky or gravelly slopes and along washes and arroyos from Colorado to South Texas.

POISONOUS PRINCIPLE. Hogplum causes a condition in sheep similar to lechuguilla poisoning. The seed apparently contain a hepatic toxin since they will produce a marked icterus and death (Boughton 1936).

Conium maculatum-Poison Hemlock, Poison Parsley

DESCRIPTION. Poison hemlock is a large, branching, herbaceous biennial of the parsley family (Umbelliferae). The pinnately dissected leaves are sessile on the upper part of the plant and petioled with dilated sheaths on the lower. The stems are often streaked or spotted with purple, Figure 34.

DISTRIBUTION. Poison hemlock is widespread in disturbed and waste places. It has been recorded in Texas from the Gulf Prairies westward to the Trans-Pecos. It has been naturalized in the United States from Eurasia.

POISONOUS PRINCIPLE. The principal toxic substance in poison hemlock is coniine, an alkaloid, which is a heart depressant (Massey and Hatch 1943). All parts of the plant, including the seed, are poisonous to domestic livestock.

MANAGEMENT. Poison hemlock is of greatest danger to livestock in the spring, but has low palata-

Figure 33. Right: Hogplum, Colubrina texensis.Figure 34. Center: Poison hemlock, Conium maculatum.Figure 35. Left: Conyza, Conyza coulteri.

bility and is not eaten to any extent if good forage is available. Plant grubbing before seed maturity is practical if they are not too abundant. This plant is susceptible to 2,4-D and control by spraying of localized areas has been effective.

Conyza coulteri-Conyza, Coulter Conyza

DESCRIPTION. Conyza is a herbaceous annual of the composite family. The small many-flowered heads are in leafy panicles. The plant commonly branches, is publicated or hirsute and has simple, toothed to coarsely pinnatifid leaves, Figure 35.

DISTRIBUTION. Conyza is frequent on range areas of the Trans-Pecos and may become abundant on heavily used and trampled areas and after flooding or during better rainfall years. It extends into Colorado, California and Mexico.

ANIMALS POISONED. Sheep, goats and cattle have been poisoned experimentally by feeding conyza. Apparently young plants are more toxic than mature specimens. Serious losses of cattle in the Trans-Pecos area have been attributed to this plant. It appears to cause more losses during times of drouth and when it grows around watering areas (Boughton 1941; Dollahite 1955, 1956, 1958, 1959, 1963).

Corydalis aurea-Golden Corydalis

DESCRIPTION. Golden corydalis is an early flowering annual, biennial or short-lived perennial in the fumitory family (Fumariaceae). The diffusely branched, smooth, leafy-stemmed plants have yellow flowers with a spurred petal in spikelike racemes, Figure 36.



DISTRIBUTION. Golden corydalis grows in disturbed areas, along stream banks, in open woods and commonly in sandy soil throughout Texas. It is found from Alaska to California and Mexico and eastward across the United States.

POISONOUS PRINCIPLE. Corydalis contains alkaloids. It is more poisonous to sheep than to cattle (Kearney and Peebles 1951). Corydalis poisoning has not been recognized as a problem in Texas.

Datura spp.-Jimsonweeds, Thornapples

DESCRIPTION AND DISTRIBUTION. There are three species of Datura recorded for Texas. They are coarse, annual, ill-scented, herbaceous weeds. The large, showy, erect flowers are white or purplish and solitary in the leaf axils. The leaves are alternate, simple, glabrous and toothed. The fruits are conspicuous, prickly, four-valved, many-seeded capsules. Jimsonweed, Datura stramonium, is naturalized in the United States from South America and the Eastern hemisphere and is found throughout Texas. Indian apple, D. wrightii (D. metaloides), is found on the South Texas Plains, the Edwards Plateau and the Trans-Pecos. It is recorded from Colorado to Texas. westward to California and south into Mexico. The oakleaf thornapple, D. quercifolia, is purple flowered and normally a smaller plant than the above. It is found on the High Plains and in the Trans-Pecos area and extends into southern Arizona and Mexico.

The jimsonweeds normally grow in rich soil on disturbed sites, waste places, old fields and in open areas.

ANIMALS POISONED. All parts of the plants are poisonous to horses, cattle, sheep and pigs.



Figure 36. Corydalis aurea, Golden corydalis.

Figure 37. Plains larkspur, Delphinium virescens.



CLINICAL SIGNS. The toxic substances in the *Daturas* produce subnormal temperature, restlessness, muscular twitching, incoordination, paralysis, delerium, respiratory paralysis and death (Hubert and Oehme 1961).

POISONOUS PRINCIPLE. The jimsonweeds contain various alkaloids, including atropine (daturine), scopolamine and hyscyamine.

Delphinium virescens-Plains Larkspur, Delphinium

DESCRIPTION. Native Texas larkspurs are perennial herbs with erect, branching stems and alternate, lobed or divided leaves. The irregular flowers have a spurred sepal. The delphiniums belong to the crowfoot family (Ranunculaceae). Although several western larkspurs are poisonous, the only Texas species considered toxic is the plains larkspur. This species has white or slightly bluish flowers, Figure 37. The Carolina larkspur (D. carolinianum) has blue flowers and is not known to be poisonous.

DISTRIBUTION. The plains larkspur is distributed uniformly over Texas but is of greatest abundance on prairie sites.

POISONOUS PRINCIPLE. The poisonous larkspurs contain several toxic alkaloids but the plains larkspur, although reported to be poisonous to cattle (Durrell *et al.* 1952), is not considered hazardous in Texas.

Drymaria arenarioides-Alfombrilla

DESCRIPTION. Alfombrilla, Drymaria arenarioides, is a prostrate, short-lived perennial in the pink family (Caryophyllaceae). The stems branch profusely to form a somewhat-tufted plant with a tap root. The linear-lanceolate to narrowly elliptic leaves are more or less facicled. The petals of the white flowers are two-cleft or parted, Figure 38.

DISTRIBUTION. Alfombrilla grows in northern Sonora and Chihuahua, Mexico, with greatest abundance in the State of Chihuahua. It extends southward into Central Mexico (Sperry and Walker 1957). Soil analyses of native habitats of alfombrilla showed that it normally grows on soil of an acid nature. Only 1 plant out of 75 extensive samples has been found on soils on the transition between acid and alkaline. Although this plant has been collected in *Mexico, about 85* miles southwest of El Paso, 20 miles south of the Arizona border, and about 7 miles south of Antelope Wells, New Mexico, no known collections have been made in the United States.

ANIMALS POISONED. Alfombrilla is poisonous to cattle, sheep and goats. Severe losses have been reported in cattle in the State of Chihuahua, Mexico. Sheep fed as little as 0.1 percent of their body weight of the plant died within 21 hours. All of the animals died when fed as much as 0.5 percent of their body weight (Dollahite 1959).

CLINICAL SIGNS. Signs develop rapidly and usually there is only a short time from the appearance of the first signs of poisoning until death. This period may be as short as 2 hours. Animals have a normal or subnormal temperature, dropping as low as 94° F. before death. They tremble, have muscular spasms, salivate and develop labored breathing. Poisoned animals are reluctant to move and "get down" and struggle before death. Occasionally there is straining and bloating.

LESIONS. The lungs usually are congested with small petechial hemorrhages on the surface. There are extensive hemorrhages on the inside and outside of the heart that sometimes extend into the muscle. The spleen is thick, congested and soft, and blood drips or runs from the cut surface. The liver is congested and friable, and usually there are hemorrhages under the capsule. An edematous infiltration into the walls of the gall bladder may extend to the bile ducts. There usually are hemorrhages into the wall of the gall bladder and the bile may vary from dark green to red, and in some cases contains intact blood cells. The kidneys may be congested and some-

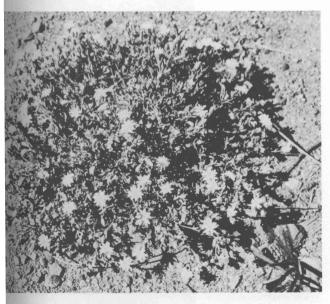


Figure 38. Alfombrilla, Drymaria arenarioides.



Figure 39. Thickleaf drymary, Drymaria pachyphylla.

times are surrounded by as much as 6 inches of semigelatinous, amber fluid. The abdominal cavity sometimes contains a large amount of amber fluid. There is a mild gastritis. There may be mild to severe enteritis.

POISONOUS PRINCIPLE. By means of paper chromatography, alkaloids have been isolated from this plant.

MANAGEMENT AND TREATMENT. Since the time lapse between the appearance of the first signs of illness and death is so short, and by the time these signs appear irrepairable damage has been done to the body tissues, treatment is of little or no help. Animals should be kept away from ranges when alfombrilla is growing luxuriantly. If animals must be driven through areas where this plant grows, they should be given ample feed and water before they are exposed to alfombrilla.

Drymaria pachyphylla-Thickleaf Drymary, Inkweed

DESCRIPTION. Thickleaf drymary is a glabrous, short-lived annual which grows close to the ground in a somewhat circular pattern up to 8 or 10 inches in diameter. The leaves are blunt-pointed and are usually about as wide as long. Small flowers and small-seeded fruits are produced in the axils of the leaves, Figure 39. Thickleaf drymary is a member of the pink family (Carophyllaceae) and is related to and sometimes confused with the chickweeds, species of Stellaria and Cerastium.

DISTRIBUTION. Drymary is frequent to abundant in the Trans-Pecos area with a few records east of the Pecos River, Figure 40. It extends across New Mexico, southeastern Arizona and south into Mexico. Thickleaf drymary grows on sites with sparse vegetation, most commonly on heavy clay soil and in low areas subject to flooding. Soil analyses from sites



Figure 40. Thickleaf drymary, Drymaria pachyphylla.

inhabited by thickleaf drymary in Texas showed that it grows on soils of an alkaline nature.

ANIMALS POISONED. Cattle, sheep and goats are poisoned by both dry and green drymary plants. Most poisoning occurs on over-grazed ranges and plants are most commonly grazed in the early part of the day while they are turgid. Feeding experiments have shown that 0.6 percent of the body weight of the plant will kill a sheep, 0.4 percent a cow, and 0.9 percent a goat (Mathews 1940b).

CLINICAL SIGNS. Animals poisoned by drymary on the range usually die before the signs are recognized. This is in accord with the results of feeding experiments in which death occurred a short period after the first signs appeared. Diarrhea and loss of appetite are early signs but animals remain on their feet and stand with an arched back and tucked-up abdomen.

LESIONS. The heart muscle is hemorrhagic, the wall of the gall bladder is edematous and thickened, and the liver and spleen show marked congestion and dark color. Hemorrhages and congestion also may appear in other organs (Mathews 1940b).

MANAGEMENT AND TREATMENT. Most cases of poisoning occur on pastures with little forage during dry seasons when light showers are sufficient to bring on a weed crop. For information on management, see Drymaria arenarioides.

Eupatorium rugosum-White Snakeroot, Richweed

DESCRIPTION. White snakeroot is an erect, branching, herbaceous perennial composite of from 1 to 4 feet. The slender, round stems may develop a purplish tinge, especially when growing in the open. Small clusters of white flower heads are produced at the ends of the numerous branches. The leaves are opposite, have three distinct veins and have coarsely toothed margins, Figure 41.

There are approximately 30 species and varieties of *Eupatorium* in Texas, some similar to *E. rugosum* but, as far as known, not toxic. Hershey (1949) reported another species, *E. wrightii*, as poisonous in the Rocky Mountain area.

DISTRIBUTION. White snakeroot occurs in East Texas and as far west as the Chisos Mountains but is most abundant and troublesome in the Hill Country. Luxuriant growth is found in north Uvalde and Medina Counties, over most of Real and Bandera Counties and in portions of Kerr County, Figure 42. The general distribution of white snakeroot is from eastern North America westward to Minnesota and Texas (Couch 1933). White snakeroot is most frequent in wooded areas but may persist in open clearings. It frequents most hardwood areas in East Texas and is associated with juniper and oak in the Hill Country. This plant may become abundant following timber clearing, especially in juniper areas. In the Hill Country, white snakeroot is not confined to ravines and valleys but extends up the slopes and occasionally over hilltops.

ANIMALS POISONED. White snakeroot is poisonous to goats, cattle, sheep, horses and swine. Humans may be affected by drinking milk from cows grazing on this plant. Goats are most commonly poisoned in Texas.

CLINICAL SIGNS. Signs of white snakeroot poisoning are trembling of muscles, especially after exercise, depression, weakness, inactivity, stiff movements with frequent stumbling and falling, labored respiration,



Figure 41. White snakeroot, Eupatorium rugosum.



Figure 42. White snakeroot, Eupatorium rugosum.

constipation, blood in feces and an odor of acetone in the breath (Couch 1933). Humans acutely affected by the disease usually show delirium and coma preceding death (Graham and Michael 1935).

LESIONS. Necropsy reveals extensive degeneration of kidneys and liver (Wolf *et al.* 1918).

POISONOUS PRINCIPLE. Couch (1927) gives the principle toxin of white snakeroot as an unsaturated alcohol, tremetol. This alcohol is found primarily in green tissue and decreases as the plant dries. The poison is cumulative in effect, and is transmitted in the milk.

MANAGEMENT AND TREATMENT. Animals should not be allowed to graze in pastures where white snakeroot grows unless there is adequate palatable feed. The milk should not be used for animal or human consumption.

Poisoned animals should be removed from infested pastures as soon as possible. The young should be given milk from cows that have not had access to white snakeroot. The use of purgatives, stimulants and laxative feeds improve chances of recovery Drugs should be given by stomach tube or injection as some animals have throat paralysis (Tehon *et al.* 1946).

Localized areas may be isolated by fencing, or plants may be pulled and burned. White snakeroot is susceptible to herbicidal sprays. The best control has been obtained with amine formulations.

Euphorbia spp.-Spurges

DESCRIPTION. The spurges are a large group of annual and perennial herbs and shrubs with milky, acrid juice. The leaves are simple and alternate, whorled or opposite on the stem. The monoecious flowers are in cup-shaped heads surrounded by four or five-lobed involucres. A single pistilate flower is surrounded by staminate or sterile flowers and from it develops a three-carpeled, three-seeded fruit. The spurges vary in habit from mat form growing close to the ground to upright leafy or almost leafless plants. Figure 43 of snow-on-the-mountain, *Euphorbia marginata*, is an example of an upright leafy form.

DISTRIBUTION. Some species of the approximately 65 species and varieties listed for Texas are present in every section of the state. At least eight of these are known or suspected of being poisonous. Most species are weeds and are fairly abundant on overgrazed pastures and disturbed areas.

POISONOUS PRINCIPLE. The acrid juice of a number of species of this genus is reported to be toxic. A resinous substance, euphorbin, which is toxic, or at least an irritant, has been extracted. Species other than those listed by various workers undoubtedly are potentially poisonous. One of the common properties of seed of the spurges and other parts of spurge plants is the strong purgative effect. Approximately 3 kilograms of both *E. prostrata* and *E. marginata* fed to cattle produced severe scours and emaciation (Hoffman 1954). The animals concerned recovered from the effects in several months. The milky juice of many of the spurges is often irritating to the skin of animals and may cause loss of hair.

MANAGEMENT AND TREATMENT. Intestinal astringents should be administered to poisoned animals to relieve diarrhea.

Since most of the spurges can be grazed to a limited degree without noticeable reaction, light in-

Figure 43. Snow-on-the-mountain, Euphorbia marginata.



festations in pastures should not be a problem. When a heavy infestation is present, poisoning may be expected. As most pasture species of *Euphorbia* are annual weeds, a good practice is to mow, reseed with good forage plants and stock lighter to improve desirable forage. The bitter juice of the spurges apparently makes most species unpalatable and plants are grazed only accidentally when better forage is available. Snow-on-the-mountain can be controlled with 2,4-D or 2,4,5-T.

Flourensia cernua-Blackbrush, Tarbush

DESCRIPTION. Blackbrush is a much-branched, leafy shrub of the composite family. The leaves are alternate, entire, obovate or oblong. The rayless flowers are solitary in the leaf axils, forming a leafy inflorescence. The fruit is a one-seeded achene. Blackbrush plants may not exceed 12 inches in height in dry sites, but attain a height and spread of 5 to 6 feet in more favorable situations, Figure 44.

DISTRIBUTION. Blackbrush is frequent to abundant in the Trans-Pecos area and in counties immediately east of the Pecos River. It extends across New Mexico into Arizona and southward into Mexico. This shrub grows on dry hills, plains and mesas, often on limestone areas.

ANIMALS POISONED. Sheep, goats, cattle and rabbits are poisoned by blackbrush. Mathews (1944) demonstrated the toxicity of the ripe blackbrush fruit by experimental feeding to sheep and goats. A marked variation in the susceptibility of individuals was observed as well as a narrow margin between slightly toxic and lethal amounts. As little as 1 percent of the animal's body weight of dry fruit eaten in one day will kill some animals.

Figure 44. Blackbrush, Flourensia cernua.





Figure 45. Bitter Sneezeweed, Helenium amarum.

Although Mathews only reported severe losses in sheep and goats when they were hungry, sporadic losses occur regularly in sheep, goats and cattle on ranges where blackbrush grows, when good forage is scarce. The blossoms, buds and immature green fruit are as toxic as the ripe fruit (Dollahite and Allen 1957 and 1959).

CLINICAL SIGNS. The signs of blackbrush poisoning are loss of appetite, a listless attitude, muscular twitching, abdominal pain, groaning, grinding of teeth and poor equilibrium.

LESIONS. There is usually severe gastroenteritis with some hemorrhage in the abomasum and first part of small intestines. There is marked congestion of the liver and kidneys with albuminous degeneration in some cases.

MANAGEMENT AND TREATMENT. Hungry animals should be kept off ranges where blackbrush grows unless there is adequate forage or adequate supplementary feed provided.

Large range areas infested with blackbrush have been cut with roller brush cutters and regrowth has provided some cattle forage during winter months.

Helenium amarum (Helenium tenuifolium)– Bitter Sneezeweed

DESCRIPTION. Bitter sneezeweed, also called east ern bitterweed and fineleaf sneezeweed, is an erect, branching annual with narrow alternate leaves. The many-flowered, composite heads have yellow, cleft, ray flowers. The glabrous, much-branched plant may reduce the lower leaves and branches during dry summers and put on a profusion of terminal growth before late summer flowering, Figure 45. *H. nudi florum*, purplehead sneezeweed, recorded for East Texas, also is reported as toxic to livestock (Tehon *et al.* 1946). Orange sneezeweed, *H. hoopesii*, is an other poisonous sneezeweed which infests much of the Western United States.

DISTRIBUTION. Bitter sneezeweed occurs from Central and East Texas across the Southeastern United States. It grows in old fields, overgrazed pastures, is a frequent roadside weed and may be abundant on waste and disturbed areas.

ANIMALS POISONED. Sheep, cattle, goats, horses and rabbits have been poisoned by eating the different species of sneezeweed. Sneezeweed poisoning is more a problem in sheep than in other species. Sneezeweeds have a bitter, sharp taste, but some animals graze them in quantity when more palatable vegetation is scarce. In addition to their poisonous properties, the sneezeweeds give a bitter taste to milk.

CLINICAL SIGNS. Animals affected with sneezeweed poisoning become weak and stagger, develop diarrhea, vomiting, salivation and bloating. They groan, grind their teeth and retract their lips. Respiration is forced and usually fast. The pulse is irregular and rapid and they have a nasal discharge.

LESIONS. Sneezeweed poisoning produces gastroenteritis with an edema in the walls of the stomachs. Endocardial hemorrhage, pulmonary edema, fluid in the pleural cavity and ascites are usually produced (Hardy and Boughton 1940; Dollahite 1963).

MANAGEMENT AND TREATMENT. Mineral oil administered in large doses in the early stages of sneezeweed poisoning may help prevent losses. Pasture management to promote ample forage or to keep cattle out of pastures infested with sneezeweed should be practiced. Mowing before seed maturity greatly reduces the amount of the plant.

Bitter sneezeweed is susceptible to 2,4-D and good control has been attained by spraying with the amine formulations at the rates of $\frac{1}{2}$ to 1 pound per acre in water when the weed is small (Harris 1956, 1957). Restrictive regulations and the proximity of susceptible crops, such as cotton, must be adhered to if a spraying program is initiated. Older weeds in the bloom stage also may be killed if the rate of applica-

Figure 46. Smallhead Sneezeweed, Helenium microcephalum.

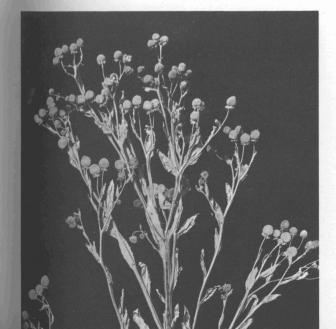




Figure 47. Bitterweed, Hymenoxys odorata.

tion is increased to 2 pounds per acre (Harris 1956, 1957). Tests have shown that good control also is obtained with the ester formulations and 2,4-D applied at the rate of $\frac{1}{2}$ pound per acre (Dunbar 1956).

Helenium microcephalum-Smallhead Sneezeweed

DESCRIPTION. Smallhead sneezeweed is an erect, branching herb with alternate, lanceolate or oblong leaves decurrent on an angled stem. The disk flowers of the composite heads assume a somewhat spherical appearance and are equal to or more obvious than the yellow ray flowers, Figure 46. The plant usually grows as an annual but may be biennial. The Rio Grande sneezeweed, *H. ooclinum* is quite similar to the smallhead but has larger flower heads.

DISTRIBUTION. Smallhead sneezeweed is usually found in moist soil around tanks, ponds and bar ditches in South Texas and adjacent Mexico. The Rio Grande sneezeweed is found in the area of the Rio Grande in southwest Texas.

ANIMALS POISONED. Smallhead sneezeweed is very poisonous. The plant, when in flower, is particularly poisonous and consumption of as little as 0.25 percent of their body weight of the plant will produce death in sheep and cattle (Hardy and Boughton 1940; Dollahite 1963).

CLINICAL SIGNS AND LESIONS. See bitter sneezeweed.

MANAGEMENT AND TREATMENT. In general, management and treatment are similar to that for bitter sneezeweed. Since smallhead sneezeweed usually is in localized areas, hand pulling and burning is easily accomplished. This plant is susceptible to herbicides, and when possible, may be killed by spraying with 2,4-D.

Hymenoxys odorata-Bitterweed

DESCRIPTION. Bitterweed is a member of the composite family. It is a much-branched annual plant that varies in height from a few inches to about 2 feet according to environmental conditions. Each



Figure 48. Bitterweed, Hymenoxys odorata.

of the ascending stem branches terminates in a yellow-flowered head. The flower heads are made up of many small flowers, and under normal growth conditions each head produces from 50 to 75 seeds, Figure 47. Seedlings or older green plants may be found at almost any time of the year, but most growth is from early spring to early summer. If climatic conditions are favorable, growth may start as early as December. The plant has a bitter taste that apparently is strongest in mature plants and plants growing on dryer sites. Crushed or bruised leaves have an aromatic odor. Pingue, *Hymenoxys richardsonii*, another poisonous plant of the same genus, causes heavy loss of sheep on grazing areas of Colorado, Arizona and New Mexico.

DISTRIBUTION. Bitterweed is found over most of Texas west of the 99th meridian. The heaviest infestations and the most severe losses in Texas occur in about 12 counties in the eastern portion of the Edwards Plateau and the adjacent Trans-Pecos region, Figure 48. Bitterweed ranges from Central Texas to California and from Kansas south into Mexico.

ANIMALS POISONED. Bitterweed produces poisoning in sheep and occasionally in cattle. Poisoning occurs in winter and early spring, or at other times when there is inadequate green range forage. The average poisonous dose is 1.3 percent of the animal's body weight. This plant is more poisonous during times of drouth.

CLINICAL SIGNS. The signs of acute poisoning are loss of appetite, cessation of rumination, depression, indications of abdominal pain, bloating and green regurgitated material about the mouth and nose. Loss of weight is the most common sign of chronic bitterweed poisoning.

26

LESIONS. The most constant lesion observed is congestion of the lungs. There usually are hemorrhages on the epicardium and occasionally on the endocardium and costal pleura. The submaxillary and retropharyngeal lymph nodes are frequently congested or hemorrhagic. The fourth stomach is congested and usually contains hemorrhages; these lesions usually continue into the duodenum and may be found scattered throughout the intestinal tract. The spleen may be tumefied, the kidneys may be congested, and the liver may be congested and friable (Hardy et al. 1931).

MANAGEMENT AND TREATMENT. When bitterweed poisoning occurs, animals should be moved to clean pastures or supplied feed. There is no medical cure for severely poisoned animals. When taken in small quantities, the weed must be grazed for several days before the animal becomes noticeably ill. The animal will recover within a few days if it is removed from the bitterweed range.

Reduced stocking rates, change in the type of livestock, and supplementary feed will help prevent losses.

Temporary relief and a reduction of bitterweed are obtained by hand pulling and destroying the weeds or by spraying with herbicides. Complete or near complete control of bitterweed has been obtained over a period of several years when plants growing under optimum soil moisture conditions and with related vigorous growth were sprayed with the L.V. ester formulations of 2,4-D in water at the rate of 1 pound per acre by either air or ground equipment. The importance of proper soil-moisture plant-vigor relationships has been confirmed by experiments. The best results were obtained when soil moisture was near or above the determined wilting point for the soil type concerned (Sperry and Sultemeier 1965).

Figure 49. Rayless goldenrod, Isocoma wrightii.





Figure 50. Rayless goldenrod, Isocoma wrightii.

Isocoma wrightii (Aplopappus heterophyllus)-Rayless Goldenrod, Jimmyweed, Alkaliweed

DESCRIPTION. Rayless goldenrod is a member of the composite family but is not a true goldenrod. It is a bushy half-shrub with numerous upright branches which arise from a perennial, woody, root crown. The branches, which range from 2 to 4 feet high, give rise to numerous yellow flower heads and then die back to the ground each year, Figure 49. Growth on Texas range areas start in early spring, and flowering begins in late summer. Reproduction is by seed following fall, winter and early spring rains.

DISTRIBUTION. Rayless goldenrod is abundant in the Pecos River and Rio Grande valleys, and is frequent around water sites and irrigated areas of the Trans-Pecos, the South Texas Plains and the High and Rolling Plains area, Figure 50. Rayless goldenrod often frequents drainage areas, springs and irrigation canals. Large acreages along the upper Pecos have been infested through irrigation, Figure 51.

ANIMALS POISONED. Cattle, horses and sheep may be poisoned by consuming the weed, and their young may be poisoned by their milk. Humans may develop milk sickness or trembles by consuming milk from cows grazing on rayless goldenrod.

CLINICAL SIGNS. The most noticeable sign of rayless goldenrod poisoning is trembling, especially in the muscles about the nose, legs and shoulders. The trembling may be preceded by a period of depression and inactivity. The animal stands in a "humped-up" position and moves with a stiff gait. This stiffness and weakness is most pronounced in the forelegs. The inactivity gradually increases and culminates in extended weakness. In later stages, the animal lies down most of the time and eventually may be unable to rise. Most cases of poisoning result in constipation, vomiting, quickened and labored respiration and almost continuous dribbling of urine.

The abnormal respiration in sick animals a short time before death is especially characteristic. Affected animals breathe with a prolonged inspiration being followed by a pause and then a short and somewhat forcible expiration. In still later stages, the animal may breathe in a series of gasps. Horses sweat profusely in early stages of poisoning.

LESIONS. Necropsy findings are not especially marked but are uniform. There usually is congestion in the fourth stomach and intestines. The liver is generally pale, the bile thick, dark and viscid and the gall bladder distended. The brain and spinal cord are congested in some cases (Marsh 1926).

POISONOUS PRINCIPLE. Tremetol, an alcohol, is present in both dry and green plant material, and is thought to be the principal toxin. From 1 to 1.5 percent of the animal's body weight of the plant fed over a period of 2 to 3 weeks has been found toxic. The poison of rayless goldenrod is cumulative, and since there may be some elimination, larger amounts would be required to be poisonous if taken over a longer period of time. Sometimes calves, colts and lambs become ill from the milk and die before their mothers show signs of illness. An average daily feeding of $1\frac{1}{2}$ pounds of the green plant per 100 pounds of animal, continued for a week, usually produces toxic symptoms or death (Couch 1929, 1930).

MANAGEMENT AND TREATMENT. The treatment of poisoned animals is the same as for White snakeroot, *Eupatorium rugosum*. Eradication of rayless goldenrod is imperative if areas are to be grazed by livestock. Infested areas are commonly fenced off, and large areas are idle due to heavy infestation. Plants have been grubbed in some localized areas. Late summer, before flowering, is apparently the best time for grubbing.

Individual plants may be killed economically by basal spraying with 2 to 4 pounds of L. V. 2,4-D mixed in 100 gallons of diesel oil. Control by foliage spraying has been erratic in control studies to date.

Figure 51. Rayless goldenrod, Isocoma wrightii.



Jatropha dioica (J. spathulata)–Leatherstem Jatropha cathartica–Berlandier Nettlespurge

DESCRIPTION. The species of Jatropha differ from other native members of Euphorbiaceae by having petals and united filaments in the flower. They do not have stinging hairs as do some related genera. Leatherstem is an erect, perennial, shrubby plant with simple or somewhat lobed leaves. The stems are quite flexible, thus the name leatherstem, Figure 52. Nettlespurge has long-petoiled, palmately 7-parted leaves. The stems are from a large starchy rootstock, Figure 53.

DISTRIBUTION. Leatherstem grows on gravelly bluffs, hillsides, and ravine slopes of the South Texas Plains, the Edwards Plateau and Trans-Pecos areas. Nettlespurge grows in the southern portion of the South Texas Plains.

CLINICAL SIGNS AND ANIMALS POISONED. Berlandier nettlespurge has been shown to be poisonous to sheep, goats and rabbits. Rabbits died in convusions within 4 hours after eating the equivalent of 2 percent of their body weight of plant material (Dollahite 1963). A goat that was fed 3.7 percent of its body weight of leatherstem leaves developed progressive anemia and died (Boughton and Hardy 1939).

Kallstroemia hirsutissima–Hairy Caltrop Kallstroemia parviflora–Warty Caltrop

DESCRIPTION. The caltrops are much-branched annuals with long prostrate stems from a central root, Figure 54. The growth habit and general appearance is similar to the puncturevine or goathead, *Tribulus terrestris*. Both are members of the caltrop family (Zygophyllaceae). The fruit of the caltrops is beaked and breaks up into 8 to 12 one-seeded nutlets at maturity. The stems and leaves of hairy caltrop are conspicuously hairy and the beak of the fruit is not more than 3 millimeters long. The beaks of the fruits of warty caltrop are commonly longer than the nutlets

Figure 52. Leatherstem, Jatropha dioica.



Figure 53. Berlandier nettlespurge, Jatropha cathartica.



(4-6 mm. long), which are tuberculate (warty) on the back. The nutlets of the hairy caltrop have ridge-like tubercules.

DISTRIBUTION. Both species of caltrop are widely distributed over Texas; hairy caltrop is more common in the western portions and warty caltrop more common in the eastern sectors. They may be found in old fields, heavily grazed pastures and disturbed areas.

ANIMALS POISONED. The caltrops are poisonous to cattle, sheep, goats and rabbits.

CLINICAL SIGNS. This plant was first shown to be the cause of cattle losses in the Trans-Pecos area by Mathews (1944a). The first signs of hairy caltrop poisoning, according to Mathews, is a weakness in the hind legs with a knuckling of the fetlock joint, followed by posterior paralysis. Frequently convulsions occur before death.

Sporadic losses due to eating warty caltrop have been observed in sheep. Signs of illness are similar to those in cattle, except that sheep were observed to be walking on their front knees before convulsions developed. Lesions of congestion with hemorrhages were observed in the lungs, heart, kidneys, stomach and intestines (Dollahite 1955).

MANAGEMENT AND TREATMENT. In recent years extensive losses in cattle have been reported as a result of eating hairy caltrop. These losses have occurred in widely scattered areas of Texas but have been most severe in the Trans-Pecos.

Animals should not be grazed where a majority of the forage is caltrop. Animals that have not reached the convulsive stage will usually recover if they are placed in the shade with ample feed and water. Excessive handling or driving of these animals is contraindicated.

Since hairy caltrop is an annual weed, general practices to eliminate the weed population and provide better forage is the best precaution. Turning hungry animals into heavily infested fields should be done only with close observation and caution.

Karwinskia humboldtiana-Coyotillo

DESCRIPTION. Coyotillo is a spineless shrub of the buckthorn family (Rhamnaceae) with mostly opposite-veined leaves. The small greenish flowers and brownish black fruits are in the axils of the leaves. The fruit is an ovoid-shaped drupe. The simple veins end in the untoothed margins of the leaves and, being quite distinct, are an aid in recognizing the plant in the field, Figures 55 and 56.

DISTRIBUTION. Coyotillo occurs in the southern portions of the Edwards Plateau and Trans-Pecos and in the South Texas Plains, Figure 57. Marsh (1929) gives the general distribution as Southwest Texas and Mexico. This shrub grows along arroyos, river canyons and on gravelly hills and ridges.



Figure 55. Coyotillo, Karwinskia humboldtiana.

ANIMALS POISONED. The seeds and leaves of coyotillo are poisonous to cattle, sheep, goats, guinea pigs, horses, swine and chickens. Poisoning may result from a single feeding but usually several days or even weeks elapse after the initial feeding before symptoms appear. While as little as 0.2 percent of the body weight of a sheep of ground coyotillo fruit may cause death, as much as 20 to 25 pounds of leaves may be required to obtain this toxicity (Marsh *et al.* 1928).

CLINICAL SIGNS. Coyotillo produces signs of unthriftiness, depression, weakness, trembling, incoordination and respiratory distress. This condition is commonly known as "limberleg."

LESIONS. Animals that die of coyotillo toxicity have a severe pulmonary edema. Microscopically,



Figure 54. Hairy caltrop, Kallstroemia hirsutissima.



Figure 56. Coyotillo, Karwinskia humboldtiana.



Figure 57. Coyotillo, Kawinskia humboldtiana.

there is usually degeneration of some of the skeletal and the cardiac muscles. There is a mild toxic nephritis and hepatitis (Henson 1963).

MANAGEMENT AND TREATMENT. Heaviest losses occur when animals that have never been on coyotilloinfested range are first exposed to this plant. Animals unfamiliar with coyotillo should not be-turned into infested areas when the plants are in fruit. Supplemental feed and proper stocking rates will help prevent poisoning. Poisoned animals should be placed in pens and fed. Purgatives and stimulants may help. In severe cases, death usually occurs, but mildly poisoned animals often recover.

Experimental control studies have shown that coyotillo is difficult to kill with herbicides (Sperry *et al.* 1962). The best kills were obtained with fenuron pellets applied at the base of the plant before summer rains. Selective herbicides obtain reasonably good kill if sprayed on the lower portions of the stems when the plants are in lush growing condition following spring and early summer rains.

Kochia scoparia–Summercypress

DESCRIPTION. Summercypress is an annual herb of the goose-foot family (Chenopodiaceae). The many-branch stems give the plant a bushy appearance when mature. The leaves are petiolate, lanceolate, thin and flat. Some plants have small leaves $1/_2$ to 1 inch in length while others have leaves to about 3 inches in length. Some plants have both types of leaves, Figure 58. The fruit has five wedgeshaped, horizontal wings.

DISTRIBUTION. Summercypress is a native of Eurasia. It is cultivated as a garden plant and frequently escapes. It often grows abundantly on ANIMALS POISONED, CLINICAL SIGNS, POISONOUS PRINCIPLE. Cattle grazing summercypress have developed ataxia, incoordination, muscular spasms and recumbency. Many of these cattle die. The leaves of summercypress contain as much as 10.24 percent of oxalic acid on a dry weight, basis (Camp 1963). Cattle that had signs of poisoning after eating summercypress recovered following intravenous injections of calcium gluconate (Hardy 1963). Death losses were prevented in sheep grazing Halogeton, a plant containing large quantities of oxalic acid, by feeding pellets made of alfalfa and 5 percent dicalcium phosphate (Binns 1961).

Lantana camara-Largeleaf Lantana

DESCRIPTION. Lantana is an irregularly shaped shrub with spreading or ascending branches. The opposite, petioled leaves are ovate to oblong. The many-flowered heads are on long peducles usually from the axils of the leaves, Figure 59. The flowers vary from yellow to red-orange. The drupaceous fruits are fleshy or juicy and nearly black when mature. Two varieties of the largeleaf lantana and three additional species are known in Texas. The common lantana, *L. horrida*, is the most widely distributed form in the State.

DISTRIBUTION. Largeleaf lantana usually grows in sandy soil as an escape, but thrives under various conditions when cultivated. It grows from Texas to Florida and south into Mexico.

CLINICAL SIGNS. Symptoms of lantana poisoning include sluggishness, partial paralysis and bloody

Figure 58. Summercypress, Kochia scoparia.





Figure 59. Largeleaf lantana, Lantana camara.

diarrhea (West and Emmel 1952). Acute poisoning may cause death within 3 to 4 days.

POISONOUS PRINCIPLE. In addition to gastrointestinal irritants, this plant contains a substance that will cause photosensitization (see under special problems). The amount necessary for poisoning may be so small ($\frac{3}{4}$ to 1 pound of dried-frosted leaves for a 400 pound animal) that evidence of browsing may be overlooked (Sanders 1946). Lantana poisoning also is a problem in Southern Africa, Australia and India.

MANAGEMENT AND TREATMENT. For treatment and care of poisoned animals, see photosensitization. Although lantana can be killed by basal spray applications of 8 pounds of 2,4,5-T L.V. ester, acid equivalent, in 100 gallons of diesel oil, grubbing and destruction of plants is most practical.

Lobelia berlandieri-Berlandier Lobelia

DESCRIPTION. Berlandier lobelia is an erect, branching, herbaceous member of the bellflower family (Campanulaceae). The small, blue, bilabiate flowers are in loose racemes, Figure 60.

DISTRIBUTION. Varieties of berlandier lobelia are recorded in moist habitats in South, Central and West Texas.

POISONOUS PRINCIPLE, ANIMALS POISONED AND CLIN-ICAL SIGNS. Some species of *Lobelia* have been known to be poisonous for a long time, but losses from this plant have not been reported in Texas. In February, March and April 1959, approximately 1,500 cattle and 500 goats were reported to have died from suspected plant poisoning in a mountain area 30 to 50 miles north of Monterrey, Mexico. The signs of sickness in these cattle, similar to those produced by lobeline poisoning, were profuse salivation, dilation of the pupils, atrophy of leg muscles and extreme narcosis. The cattle had been eating berlandier lobelia. The air-dried lobelia was found to contain .079 percent of the alkaloid lobeline, and plant material fed to one sheep produced signs similar to those seen in most of the cattle. Some of the poisoned cattle were in a comatosed condition for 3 weeks, but continued to eat when food was placed in their mouth and drink when their mouth was placed in water. About half of the poisoned animals recovered when carefully nursed (Dollahite and Allen 1962).

Melia azedarach-Chinaberry

DESCRIPTION. Chinaberry is a tree with rounded crown and large, twice-pinnate leaves. The purplish flowers are produced in panicles. The fruit is a a smooth, yellow drupe, $\frac{1}{2}$ to $\frac{3}{4}$ inch in diameter. The drooping clusters of fruits mature in the fall and may persist on the tree into the winter.

DISTRIBUTION. Chinaberry was introduced into the United States as an ornamental from Asia and has naturalized as an escape from Texas to Florida and north to Oklahoma, Arkansas and the Carolinas.

ANIMALS POISONED AND CLINICAL SIGNS. The fruit is most toxic, but the flowers, leaves and bark also contain poison. Hogs are more frequently poisoned than other animals, and develop signs of illness 3 to 4 hours after eating the green or dried berries. They become stiff, incoordinated, lose their appetite, are constipated with blood stained feces and develop general weakness. Death usually occurs within 24 hours or the animal recovers. The lethal dose for swine is about 3 grams of berries per pound of body weight. The berries are less toxic for chickens, ducks and goats (West and Emmel 1952).

Melilotus alba–White Sweetclover Melilotus officinalis–Yellow Sweetclover

DESCRIPTION. Sweetclover is a well-known, valuable forage and soil improvement legume.

POISONOUS PRINCIPLE. Dicumarol is the toxic agent in sweetclover.

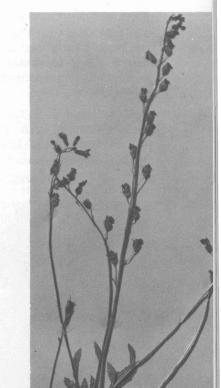


Figure 60. Berlandier Lobelia, Lobelia berlandieri.

ANIMALS POISONED. Sweetclover hay or silage may cause extensive internal hemorrhages in cattle and sheep. Sheep have been observed to become ill and have died after grazing green sweetclover.

CLINICAL SIGNS AND TREATMENT. Poisoned animals should be kept quiet and given blood transfusions. Intravenous or intraperitoneal injections of hemostatic solutions should be given to speed blood coagulation (Milks 1949). Administration of vitamin K also will speed blood coagulation. Small or alternate feeding of alfalfa hay will help prevent losses.

Nerium oleander-Oleander

DESCRIPTION. Oleander is a shrub which may grow from 15 to 20 feet in height. The variously colored, odorless flowers are produced in terminal cymes during the summer. The dark green, entiremargined, linear-to-elliptic leaves are opposite or in whorls of three or four.

DISTRIBUTION. Oleander is a common cultivated plant in Southern United States and is a native of Asia.

ANIMALS POISONED, CLINICAL SIGNS, POISONOUS PRIN-CIPLE AND TREATMENT. Animals may be poisoned when oleander garden cuttings are thrown into a dry lot or when animals have access to the shrub. People have been poisoned by using the twigs as meat skewers. All parts of the plant contain highly toxic cardiac glycosides which are more concentrated in the seeds. Oleander produces abdominal pain, vomiting, diarrhea, stimulation of the heart and constriction of the blood vessels. Poisoned animals tremble, develop a progressive paralysis, become comatose and die. Animals poisoned by oleander should be kept warm and quiet, and be given atropine and emetics or gastric lavage (Hubert and Ochme 1961).

Nicotiana glauca-Tree Tobacco

DESCRIPTION. Tree tobacco is a shrub or small tree with mostly entire glabrous and glaucus leaves. The terminal paniculate or racemelike inflorescence is diurnal and is composed of tubular-funnelform yellow flowers.

DISTRIBUTION. Tree tobacco is frequent along the Rio Grande, in the Big Bend and is often planted as an ornamental in Southwest Texas. Tree tobacco

Figure 61. Sacahuista, Nolina texana.

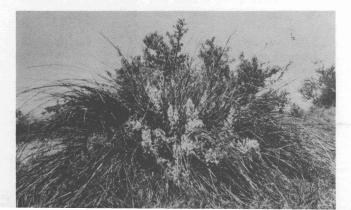




Figure 62. Sacahuista, Nolina texana.

is naturalized from South America and is found from Texas to central California.

POISONOUS PRINCIPLE. Tree tobacco contains nicotine and is poisonous to most animals. It also contains an alkaloid, anabasine, reported to be more efficacious than nicotine in killing certain species of aphid (Kearney and Peebles 1951).

ANIMALS POISONED. Cattle and horses are poisoned frequently by tree tobacco. Sheep are poisoned occasionally.

CLINICAL SIGNS. After the plant is eaten, signs of poisoning appear rapidly. Animals have a weak pulse, staring eyes, unsteadiness, stumbling and trembling of the entire body. Salivation and frequent urination are characteristic, and breathing becomes difficult before death. (Sampson and Malstrom 1942).

Nolina texana-Sacahuista

DESCRIPTION. Sacahuista is a perennial of the lily family (Liliaceae). The plants have a thick, woody caudex which gives rise to numerous clustered, long, narrow, fibrous leaves. The several flower stems bear numerous small, white flowers in somewhat elongated clusters. The fruit is a dry, three-parted capsule. The flower stalks usually are not apparent until the plant is in full bloom, Figure 61.

DISTRIBUTION. Sacahuista occurs in Texas from Bell and Travis Counties to the western boundary of the state. It is abundant on the Edwards Plateau and the Trans-Pecos area, but is less frequent on the Rolling and High Plains, Figure 62. It extends into southeast Arizona and northern Mexico. Sacahuista usually grows in open grassland on rolling hills and slopes. ANIMALS POISONED. The flower buds, flowers and fruits are toxic to cattle, sheep and goats.

CLINICAL SIGNS. Sacahuista produces signs of generalized jaundice, loss of appetite and progressive debilitation. Affected animals may have a yellow nasal discharge. Ingestion of sacahuista flowers or fruit and some green plants may produce photosensitization, as evidenced by a swollen face and ears. A purplish band may appear around the top of the hoof above the coronary band. Dermatitis with itching may occur in the early stages of photosensitization.

LESIONS. Lesions of sacahuista poisoning are generalized jaundice, yellow-brown liver and greenish brown to greenish black swollen kidneys.

MANAGEMENT AND TREATMENT. Animals should be removed from the sacahuista-infested pastures during the time that the plant is in bloom and early fruit. When animals first exhibit signs of intoxication they should be placed in the shade and fed nutritious feed. Most of the animals that develop severe jaundice will die.

Most operators do not like to eradicate or control sacahuista unless the stand is dense. When range grasses are dry, the green leaves of sacahuista are browsed and, although the plant is very fibrous, some nutrition is obtained.

Notholaena sinuata var. cochisensis-Jimmy Fern

DESCRIPTION. Jimmy fern is an evergreen, erect fern with simple, pinnate leaves. The numerous leaflets are scaly beneath and smooth above. The leaves



Figure 63. Jimmy fern, Notholaena sinuata var. cochisensis.



Figure 64. Jimmy fern, Notholaena sinuata var. cochisensis.

are from a short, chaffy, woody stem, Figure 63. Growing in dry habitats, this fern is of the "resurrection" type, in that the leaflets roll and become quite dry when moisture is lacking. They unroll and appear green and fresh following rain.

DISTRIBUTION. In Texas, this fern is found on the southwest part of the Edwards Plateau and the Trans-Pecos area, and there are a few records from canyons in the Rolling and High Plains, Figure 64. It extends into New Mexico and Arizona and south into Mexico. Jimmy fern grows on rocky slopes and crevices and is often closely associated with some of the grasses characteristic of dry habitats.

ANIMALS POISONED. Jimmy fern poisoning occurs in sheep, goats and cattle, in this order of severity. The trembling reaction called "jimmies" develops about 48 hours after animals are fed as much as 0.5 percent of their body weight of the fern and are exercised. It takes from 10 to 60 minutes of walking for animals to develop signs.

CLINICAL SIGNS. After exercise, animals affected with jimmy fern poisoning have an arched back, a peculiar stilted movement of the hind legs and usually increased respiration. The animals stop and tremble violently from head to foot, and by this time both respiration and heart action are increased. If exercise is continued, the spasms of trembling will recur. Sheep and goats usually die. Cattle tremble but usually live. Non-fatal cases require 5 to 19 days to recover (Mathews 1942).

MANAGEMENT. Most fatalities occur in winter; thus sufficient forage or supplemental feed should be supplied to animals on infested pastures during this period. Under severe conditions, animals should be moved from pastures in which jimmy fern is abundant during the winter months before losses occur. Under range conditions, it is important to leave sheep strictly alone during the danger period, since excitement or exercise will aggravate their condition. Ample watering places should be provided in pastures where jimmy fern grows so that animals can water conveniently. Since jimmy fern usually grows in rough, rocky areas, control of the plant would be difficult.

Oxytropis lambertii-Lambert Loco, Crazyweed, Point Loco

DESCRIPTION. Lambert loco is a perennial legume with basal pinnate leaves from the crown of the deep taproot. The leaves have 11 or more linear leaflets which often are covered with appressed silky hairs. The flowers are violet to bluish purple, Figure 65. Flowers ranging from white or yellowish white have been reported for the species in some mountain states. The genus Oxytropis may be distinguished from Astragalus (i.e. earle loco, woolly loco, etc.) by a peculiar point or appendage on the keel of the flower, and by the absence of stems or branches above the crown of the plant other than the scapelike flower stalks.

DISTRIBUTION. Lambert loco occurs on open grassland of North-Central Texas and on the Rolling and High Plains. It extends westward to Eastern Arizona and northward across the Rocky Mountain States into Canada. This loco usually grows on welldrained sandy or gravelly soil and frequently on rocky knolls.



Figure 65. Lambert Loco, Oxytropis lambertii.



Figure 66. Blue Panicum, Panicum antidotale.

ANIMALS POISONED. Horses, cattle and sheep are susceptible to Lambert loco poisoning.

TOXIC PRINCIPLE, SIGNS, LESIONS, MANAGEMENT AND TREATMENT. See *Astragalus mollissimus*, locoweeds.

Panicum antidotale-Blue Panicum

DESCRIPTION. Blue panicum is native to Australia, Afghanistan and India. It was introduced in the United States in 1912 from India and was brought from Australia in 1920 and 1935 by the USDA. Blue panicum is a perennial, warm-season bunchgrass that grows in large, dense tufts. It is bluish green and grows over 4 feet tall. The short, bulbous rhizomes enable the plant to withstand short drouth. The stems branch freely at the nodes, are woody and coarse when mature, Figure 66.

DISTRIBUTION. Blue panicum is best adapted to the South Texas Plains, Rolling Plains, High Plains, Edwards Plateau and Trans-Pecos areas west of the 30-inch annual rainfall line. It is adapted to a wide range of soils from fine sandy loams to clays generally having a high lime content.

ANIMALS POISONED. Losses of 30 to 50 percent of flocks of sheep on irrigated blue panicum following heavy application of nitrogen fertilizer have been observed. Fewer losses occur in sheep grazing unfertilized blue panicum. Severe losses have been observed in cattle grazing irrigated and fertilized blue panicum. CLINICAL SIGNS. Animals poisoned by blue panicum suddenly become ill, breathe rapidly and may die within a few minutes of the first signs of illness.

LESIONS. The lesions of blue panicum poisoning are primarily pulmonary emphysema and edema. In sheep dying on irrigated pastures, the thoracic cavity may be half or entirely filled with a straw colored fluid containing a fibrinous exudate. The blood is dark, almost black, indicating anoxia. Cattle grazing on dry blue panicum may have pulmonary emphysema and edema without the fluid in the thoracic cavity.

MANAGEMENT AND TREATMENT. Cattle and sheep should not be allowed to graze blue panicum for at least 40 days after fertilization and irrigation, and should be closely observed at all times. All animals should be removed from blue panicum as soon as signs of illness are observed. Animals that are acutely ill will probably die, but quiet nursing may save the less seriously affected animals.

Blue panicum is a valuable forage crop when properly managed for grazing or hay. A rotation grazing program in small blocks is necessary to obtain good utilization during peak production periods. The coarse, unpalatable stems that are not grazed should be mowed to force nutritious basal growth that can be grazed easily during the next rotation period. Proper amounts of fertilizer are necessary to maintain the stand for high production of forage.

An established stand of blue panicum can be eliminated by cultivation.

Peganum harmala-African Rue

DESCRIPTION. African rue is a much-branched perennial herb of the caltrop family (Zygophyllaceae). The alternate, dissected, succulent leaves are glabrous and divided into narrow segments. The flowers have five white petals and the fruit is a two, three or fourcelled, many-seeded capsule, Figure 67.

DISTRIBUTION. Records show that African rue has been collected in Texas in six or seven counties of the Trans-Pecos area and Edwards Plateau, Figure 68. It also is recorded for New Mexico and Arizona.

Figure 67. African rue, Peganum harmala.





Figure 68. African rue, Peganum harmala.

African rue, native to North Africa and Asiatic deserts, was introduced near Deming, New Mexico, (Cory 1949) and apparently has spread into Texas and Arizona from this introduction. The heaviest infestation of the plant in Texas appears to be on the Pecos Air Field near Pecos and it has spread along highways leading from the city.

ANIMALS POISONED. African rue is poisonous to cattle, sheep, guinea pigs and probably horses.

CLINICAL SIGNS. The signs of chronic poisoning include loss of appetite, listlessness, weakness of hind legs and knuckling of the fetlock joints. In acute poisoning, stiffness, trembling, incoordination and frequent urination occurs. The temperature is subnormal. There is excessive salivation and the lower jaw and muzzle may be wet. Acute poisoning usually is caused by eating the seeds.

LESIONS. There is severe gastroenteritis and a congestion of the kidneys. Hemorrhages are present under the capsule of the liver. The lungs are congested and usually there are hemorrhages on the heart.

POISONOUS PRINCIPLE. African rue has been proved poisonous by experimental feeding (Black and Parker 1936, Moran *et al.* 1940; Mathews 1941). The seed are most toxic, and the leaves and stems less so. The leaves of young plants are more toxic than leaves of the mature plant. This plant contains the alkaloids vasicine, harmaline, harmine and harmalol (Manske and Holmes 1952).

MANAGEMENT AND TREATMENT. Although African rue is not palatable, hungry animals should not be allowed access to this plant, particularly when the fruit is ripe. Cattle that previously have consumed a large amount of mesquite beans will develop a depraved appetite and then will graze African rue. Poisoned animals should be removed from the weed and given water and good feed. Chronic cases will recover if properly nursed.

Hazard areas may be cleaned by pulling or grubbing the plants. African rue is not easily controlled with herbicides but experimental tests have shown that this method of control is possible.

Phyllanthus abnormis-Abnormal Leafflower

DESCRIPTION. Abnormal leafflower is an annual or short-lived perennial herbaceous plant with upright stems and spreading or recurved branches, Figure 69. It is a member of the spurge family (Euphorbiaceae).

DISTRIBUTION. Collections of abnormal leafflowers in Texas are from about 17 counties of the Rolling and High Plains, the Trans-Pecos area and South and South-central Texas. This plant usually grows in sand or on sandy soils.

ANIMALS POISONED. Mathews (1945) demonstrated the toxicity of this species to cattle, sheep and goats. Sheep and goats were more resistant to the toxicity than cattle.

MANAGEMENT. As with most spurges, abnormal leafflower is relatively unpalatable. Supplemental feed or a change of pastures should alleviate further poisoning.

Phytolacca americana-Pokeweed

DESCRIPTION. Pokeweed or pokeberry is a tall, usually branched, juicy, perennial herb which dies back to the ground each year. The large leaves are



Figure 69. Abnormal leafflower, *Phyllanthus abnormis*.



Figure 70. Pokeweed, Phytolacca americana.

ovate-lanceolate and usually acute tipped. The flowers are in terminal racemes but develop opposite to the leaves as the stem continues to grow. The fruit is flattened-globose and dark purple or nearly black at maturity, Figure 70.

DISTRIBUTION. Pokeweed is found in fields, along fencerows, in rich low ground and may be around barn lots and in waste places. It is recorded from Maine and Ontario to Florida, Texas and Arizona.

POISONOUS PRINCIPLE. Pokeweed contains an alkaloid and a substance called phytolascotoxin (Hardin 1961).

ANIMALS POISONED. Pokeweed is poisonous to cattle and sheep, and occasionally is poisonous to horses, goats and swine. The entire green plant is toxic, but the roots are more toxic (Hubert and Oehme 1961). Leaves are sometimes eaten as greens, but in the cooking process, water should be changed twice (Hardin 1961).

CLINICAL SIGNS. This plant is a gastrointestinal irritant. It causes abdominal pain, vomiting and purging. There may be convulsions and death usually is due to respiratory failure (Hubert and Oehme 1961).

Prosopis glandulosa-Mesquite

DESCRIPTION. Mesquite is a small tree or shrub, somewhat thorny and often branching near the ground. The alternate leaves are deciduous, bipinnately-compound and dark green. The fruits are loosely-clustered legumes (beans) up to 8 or 10 inches in length, Figure 71. Three varieties are recognized within the state.

DISTRIBUTION. Mesquite grows throughout the Southwestern United States from Texas to Kansas, in Utah and California, south through Mexico and Central America to Colombia and Venezuela (Little 1953). ANIMALS POISONED. Primarily cattle are affected by mesquite beans, but goats also may be affected. Poisoning is not a serious problem in goats since they are seldom found on a range where only mesquite grows. An acute form of mesquite poisoning occurs where the cattle become acutely ill with an acetonemia-like condition and die within a few days. During prolonged drouths an entire herd may become affected. Usually the toxicity is chronic in nature and over a period of time cattle develop "jaw and tongue trouble."

CLINICAL SIGNS. Signs of acute poisoning include loss of appetite, rapid loss of weight, nervousness, a wild expression and bulging eyes. These animals usually die within 2 to 4 days of the first signs of illness. The chronic form of the disease, or jaw and tongue trouble, develops more gradually, usually after the cattle have been eating beans for two months or more. They gradually become emaciated and may lose 50 percent of their weight. These cattle develop atony of the rumen, salivate profusely and chew continuously, sometimes without anything in their mouth, and will hold their head to one side as if chewing were painful. They have difficulty eating anything except finely-ground feed, and when regurgitating feed from the rumen have difficulty holding it in their mouth. About 25 percent of the affected animals have a partial paralysis of the tongue; it will protrude from their mouth for 1 to 4 inches. They can lick themselves and usually keep their hair damp in this way. At least 10 percent of these cattle will have swelling under their jaws or tongue, and some will have enlarged salivary glands. They are extremely nervous and with some muscular tremor about the lips and face. They are anemic, and in hot weather large areas of their body will be denuded of hair. They cannot digest cellulose and pieces of ground hay will be found unchanged in the fecess (Dollahite and Anthony 1957).

LESIONS. The carcass is emaciated and the brain usually is congested. Hemorrhages may be seen on the heart of some animals. The kidneys are small and very firm. The liver is small, firm, has a tough capsule and sometimes is slate-colored. The rumen is filled with mesquite beans, dry grass and usually some soil. There may be hemorrhages into the papillae. A gastroenteritis is present in about 50 percent of the animals. The masseter and lingual muscles are flabby, light gray and may contain petechial hemorrhages. The salivary glands are often enlarged.

MANAGEMENT AND TREATMENT. Most of the acute poisoning occurs in pastures where large quantities of mesquite beans accumulate and where pack rats store them. Cattle should not be allowed to consume

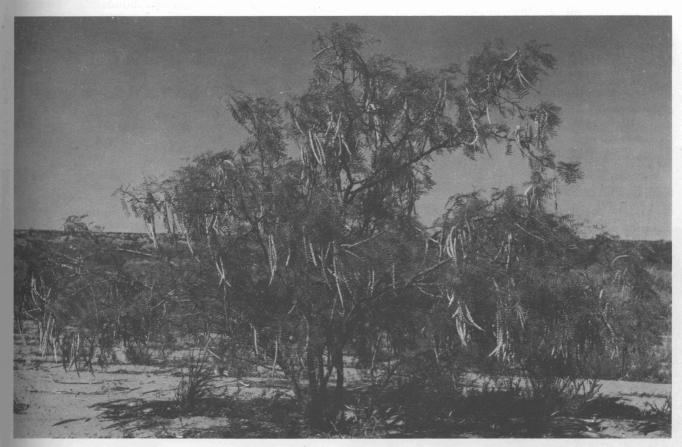


Figure 71. Mesquite, Prosopis glandulosa.



Figure 72. Species of *Prunus* in fence rows and localized in pastures are potential hazards to grazing animals.

large quantities of beans for more than 60-day periods. A combination stocking of cattle and sheep will reduce cattle losses, since sheep are apparently not affected by mesquite beans. Animals may be treated if the course of the disease has not progressed too far. Three out of four animals recovered when treated in the following manner:

1. Injected subcutaneously with Lentin at the rate of 1 milliliter per 100 pounds body weight and repeat 3 days later.

2. Administered 1 to 2 quarts of rumen material from a cow that was eating alfalfa hay and administered again 7 days later.

3. Place on a daily ration of 1 pound of cottonseed meal, 1 pound of molasses, and all of the ground grain sorghum (hay and grain) that they would eat.

Extensive areas infested with mesquite have been treated by both chemical and mechanical methods. Good range management must be followed with any method or combination of methods if control and forage production is to be achieved (Hoffman and Ragsdale 1964; Fisher *et al.* 1959).

Figure 73. Paperflower, Psilostrophe tagetinae.



Prunus spp.-Wild Plum, Wild Cherry, Chokecherry

DESCRIPTION. Plants of the genus *Prunus* are shrubs or small trees with alternate or fascicled, simple leaves. The inflorescences are elongated, somewhat flat-top clusters or solitary in the axils of the leaves. The fruit is a fleshy, one-seeded drupe. There are numerous species in this genus with a wide distribution. Twenty-eight species and varieties, some escapes from cultivation, have been recorded for Texas. The genus *Prunus* usually is included in the rose family (Rosaceae).

DISTRIBUTION. Several species of *Prunus* are present in every section of Texas. Some grow in open areas, others constitute undergrowth in wooded sites. Some of the shrubby species form motts in fields and pastures or along fence rows, Figure 72. These are usually considered hazard sites for livestock grazing in the area.

POISONOUS PRINCIPLE, SIGNS AND TREATMENT. Several species of *Prunus* are cyanogenic and develop hydrocyanic (prussic) acid under certain conditions. Influences such as bruising, wilting, withering or drying of leaves appear to contribute to the glucoside enzyme reaction. Wilted leaves, due to cutting the plant, appear to be most dangerous. For further information, see "hydrocyanic acid" under special problems.

MANAGEMENT. Mechanical and chemical eradication are suggested if followed by removal of root sprouts which may develop abundantly. From the practical viewpoint, the motts, which are the biggest problem, can be fenced off and in turn will serve as excellent sources of wildlife cover and food.

Psilostrophe gnaphalodes-Cudweed Paperflower Psilostrophe tagetinae-Woolly Paperflower

DESCRIPTION. The paperflowers are perennial composites with erect, woolly, tufted stems branching from the base. The leaves are alternate, entire or lobed.

DISTRIBUTION. Psilostrophe tagetinae and P. gnaphalodes are common in West and Southwest Texas. In general, habit and growth of these two species are similar, Figure 73. Woolly paperflower grows primarily in the Trans-Pecos area and extends into the Rolling and High Plains. From Texas it extends south into Mexico, west into Arizona and northwest into Colorado. Cudweed paperflower is frequent in the Trans-Pecos area and South Texas Plains.

ANIMALS POISONED. The two species of *Psilostrophe* are quite palatable to sheep. Limited feeding tests indicated that *P. tagetinae* is slightly more toxic than *P. gnaphalodes* (Mathews 1934). Dry and young plants are more toxic than mature blooming plants, although losses of sheep have been recorded when the plants are in full bloom. Both species are

suspected to be toxic to cattle. Field cases appear to confirm this, but experimental feeding of cattle has not ascertained its actual poisoning relation. Extensive sheep losses in the Trans-Pecos area have been caused by the grazing of paperflower.

CLINICAL SIGNS. Signs of paperflower poisoning are stumbling, sluggishness, coughing and vomiting. The vomitus is generally a greenish liquid (Mathews 1934).

LESIONS. Lesions are not significant, but an occasional animal will have inflammation of the kidneys.

MANAGEMENT AND TREATMENT. Sheep have to eat the plant for approximately two weeks before they show signs. They will recover if removed from the plant when signs first appear; thus pasture rotation is effective. Poisoned sheep, removed from the range and placed on feed, usually recover.

Pteridium aquilinum var. pseudocaudatum-Bracken Fern

DESCRIPTION AND DISTRIBUTION. Bracken ferns are perennial plants with subterranean rhizomes and much-divided leaves, Figure 74. They are found primarily in the eastern part of the state in wooded and timber areas, often in abundance. The species and its varieties, however, have a widespread distribution in woodlands over much of the United States and also are found in Europe.

POISONOUS PRINCIPLE. The poisonous nature of bracken fern and possibly other ferns and horsetails, which are related to the ferns, is due to the action of the enzyme thiaminase. Thiaminase is not a poison but destroys an essential constituent (thiamine) of the animal's food (Pohl 1955).

ANIMALS POISONED AND MANAGEMENT. Bracken fern is poisonous to cattle and horses. Most cases of poisoning in the United States have been reported



Figure 74. Bracken fern, Pteridium aquilinum var. pseudocaudatum. for the variety *pubescens*. This variety, known as western bracken fern, is common in northwest United States. Extensive losses have been reported in the Allegheny Mountains in the eastern part of the United States. Although frequent on timber-range areas of East Texas, no records of poisoning are known definitely for the Texas variety. Livestock owners should know that it is potentially poisonous. Should poisoning occur, animals should be moved to bracken-free pastures or placed on feed.

Quercus spp.-Oaks, Shin Oak, Shinnery

DESCRIPTION. The oaks are shrubs or trees with alternate, entire, toothed or lobed, persistent or deciduous leaves. The staminate flowers are in catkins; the pistillate flowers are solitary to clustered. The fruit is a one-seeded nut (acorn), partly enclosed in a saucer-shaped involucre. About 40 species of oaks with numerous varieties and hybrids have been recognized in Texas (Muller 1951).

DISTRIBUTION. Species of oak are present throughout Texas and range in habitat from dry to wet situations, and from sandy to limestone and clay soils.

ANIMALS POISONED. Cattle, sheep, goats, rabbits and guinea pigs have been poisoned by oak. The buds, small leaves, flowers and stems are palatable and poisonous. As the leaves mature they become less palatable, and animals usually stop eating them by the time the leaves are three-fourths grown. After leaves become older, animals again will eat them, but seldom in the quantity that they eat the young growth. Acorns, when eaten in quantity, produce signs and lesions similar to oak bud or leaf poisoning (Dollahite 1961).

CLINICAL SIGNS. The signs of oak poisoning include emaciation, edema, constipation or diarrhea and mucus and/or blood in the feces. The animals appear drawn, have a rough hair coat and a dry nose. They are depressed and show evidence of discomfort.

LESIONS. Gastroenteritis with degeneration of the epithelial lining and edema of the intestinal walls may occur. The kidneys are inflamed and often contain petechial hemorrhages. There may be subcutaneous edema and fluid in the abdominal cavity.

POISONOUS PRINCIPLE. Pigeon *et al.* (1962) report the isolation of a gallotannin from shin oak which produces pathological lesions in rabbits similar to lesions observed in livestock poisoned by this plant. The tannin of shin oak is the toxic principle or a contributory factor. The tannin content of the leaves (dry weight) from *Q. havardii*, Figure 75, for the months of April, May, August and October was 15.13, 8.68, 7.67, and 4.19 percent, respectively.

MANAGEMENT AND TREATMENT. Due to the severity of the losses from shin oak poisoning, many cattle are penned and fed for 3 to 4 weeks when the oak first buds in the spring. Even so, some of them later



Figure 75. Shin oak, Quercus havardii.

will eat sufficient oak to be poisoned. Many feed supplements have been used on the range, and may reduce losses, but on ranges where sand shin oak (Quercus havardii) grows, feed supplement alone fails to prevent losses.

Calcium hydroxide (hydrated lime) is an efficient antidote to prevent poisoning by tannic acid and oak tannins in rabbits (Dollahite and Camp 1962). Supplemental feed containing 9 percent hydrated lime have reduced losses in cattle in experimental feeding trials (Dollahite *et al.* 1963). Feeds containing 10 percent hydrated lime are being tested to reduce or prevent oak poisoning in cattle on the range.

A satisfactory grazing management practice is complete control of the oak on an acreage estimated to carry the entire ranch stock until the oak leaves are fully developed. The area is then deferred until the next oak bud season.

Sand shin oak may be effectively controlled with low-volatile esters of 2,4,5-T or silvex at 1/2 pound per acre in an emulsion of 1 gallon of diesel oil and water to give 4 gallons of solution per acre applied aerially. Applications should be made for 3 consecutive years to obtain maximum control. Shin oak on limestone soil does not respond readily to aerial spray applications of herbicides, but two consecutive treatments of 1 pound of 2,4,5-T in an emulsion of 1 gallon diesel oil and water to make 4 gallons of solution per acre have produced effective control. Shin oak in tree form may be controlled by trunk base applications of 16 pounds of 2,4,5-T of low-volatile ester in 100 gallons of diesel oil (Robison 1963).

Post and blackjack oaks may be controlled by basal bark application of 16 pounds of 2,4,5-T esters in 100 gallons of diesel oil and/or by aerial spray application of 2 pounds per acre of 2,4,5-T esters or silvex in an emulsion of 1 gallon of diesel oil and water to make 4 gallons of solution per acre. Repeating the application with 11/2 pounds of 2,4,5-T or silvex the following year obtains maximum control (Darrow and McCully 1959).

Ricinus communis-Castorbean

DESCRIPTION. Castorbeans are tall glabrous herbs cultivated as a source of oil for industrial and medicinal uses. They are planted as ornamentals, become established as escapes in some areas and commonly reseed in gardens and fields if left to mature fruit. Castorbeans were introduced from the Old World Tropics.

POISONOUS PRINCIPLE, ANIMALS POISONED AND SIGNS. The castorbean fruit contains ricin which is toxic to humans and all livestock. The other parts of the plant contain ricin in lesser amount. Poisoned animals develop nausea and violent purging. There is often blood in the feces. Muscular tremors, general weakness and emaciation develop in cases of prolonged illness (West and Emmel 1952). Children are reported to be especially susceptible to castorbean poisoning and Kingsbury (1963) states that one or two castorbeans may be lethal to adults.

Senecio longilobus-Threadleaf Groundsel, Woolly Groundsel

DESCRIPTION. Threadleaf groundsel is a manystemmed, perennial composite. It is evergreen on Texas ranges. The leaves usually are pinnately divided into three to seven segments and may be hairy or nearly smooth. The stems are herbaceous except at the base and also have variable hairiness. Yellow flowers are produced throughout mild winters and the blooms are abundant following summer rains, Figure 76.

DISTRIBUTION. Threadleaf groundsel is infrequent to abundant in grassland areas over most of the western half of Texas, Figure 77. It ranges south into Mexico, north into Nebraska and Wyoming and

Figure 76. Threadleaf groundsel, Senecio longilobus.





Figure 77. Threadleaf groundsel, Senecio longilobus.

west into Arizona. Threadleaf groundsel is a native forb or half-shrub, usually growing in association with grama and buffalograss. It extends into short-grass desert scrub areas and increases with disturbance and overgrazing.

ANIMALS POISONED. Senecio is poisonous to cattle, horses, sheep and goats. On Texas ranges, cattle are most commonly poisoned. It is eaten throughout the year, especially when the range forage is dry and during ice and snow storms, but stock losses are heaviest in the late spring and the summer months.

CLINICAL SIGNS. There is usually a time lapse between eating the plant and the appearance of the first signs of intoxication. The advanced stage of the disease is characterized by continuous walking, the sudden appearance of nervous disturbances and frequent voiding of small amounts of liquid, bile-stained feces. The voiding of feces is usually accompanied by prolonged painful straining. In the advanced stage, the poisoned animals are likely to attack any moving object that may be near. Death soon follows the appearance of the advanced stage of the disease (Mathews 1933).

LESIONS. Lesions of *Senecio* poisoning are jaundice, ascites which impregnate the peritoneal covering of the intestines, hardening of the liver and distention of the gall bladder, frequently to an enormous size.

POISONOUS PRINCIPLE. The toxic principle of threadleaf groundsel is probably the alkaloid longilobine (Manske 1931, 1950). The leaves are more toxic than the stems and the young leaves are more toxic than older growth.

MANAGEMENT AND TREATMENT. Animals eating groundsel should be removed from the herd or, if practical, the herd should be moved to a pasture free of groundsel. Supplemental feeding, if pastures are in poor condition, is a good management practice. Mathews (1934) demonstrated that sheep and goats are less susceptible to groundsel poisoning than are cattle. Even though sheep and goats may be poisoned experimentally by large quantities of groundsel, this occurs infrequently on the range. Sheep, when grazed with cattle, usually control the groundsel plants. This practice significantly reduces the total loss from groundsel poisoning.

Threadleaf groundsel is highly susceptible to 2,4-D and good kill has been obtained by spraying with the low-volatile formulation at 1 pound per acre. Spraying from April through June is best with either ground or aerial equipment.

Senecio spartioides, (S. riddellii)–Riddell Groundsel, Broom Groundsel

DESCRIPTION. Riddell groundsel is a herbaceous perennial. Several stems grow from a woody base. These produce lateral branches, at least terminally. The leaves are pinnatifid, green and without hairs. Yellow flowers are produced in somewhat flattish inflorescences during late summer and early fall, Figure 78. This species differs from threadleaf groundsel by being bright green, having less diffuse branching and dying back to the ground after frost.

DISTRIBUTION. Riddell groundsel is frequent to abundant in the western half of Texas, with records from several counties along the Gulf Coast south of the Guadalupe River, Figure 79. This species occurs in grasslands, foothills and sandhills, and extends from West Texas and New Mexico north into Colorado, Wyoming and Nebraska. It is often abundant in disturbed and overgrazed areas, gravelly washes, flooded areas and along roads and trails.

POISONOUS PRINCIPLE, ANIMALS POISONED, SIGNS, LESIONS, MANAGEMENT AND TREATMENT. Except for chemical control, refer to *Senecio longilobus*. Satisfactory control of Riddell groundsel has been obtained

Figure 78. Riddell groundsel, Senecio spartioides.





Figure 79. Riddell groundsel, Senecio spartioides.

by spraying with the L.V. ester of 2,4-D at the rate of 1 pound per acre during the summer and fall after effective rainfall.

Sesbania drummondii–Drummond Sesbane, Poison Bean

DESCRIPTION. Drummond sesbane is a perennial shrub of the legume family. The pinnately compound leaves are alternate and have 12-60 oblong, pointed leaflets, Figure 80. The scarlet to bright yellow flowers are in racemes. The indehiscent fruit (legume) is four-winged with cross partitions between the seeds.

[•]DISTRIBUTION. Drummond sesbane occurs from Florida to Texas and south into Mexico. It grows

Figure 80. Drummond sesbane, Sesbania drummondii.



in the Gulf Coast area and extends into the state along sluggish waterways and around wet sites. The plants are often found growing in shallow water.

Figure 81. Bagpod sesbane, Sesbania vesicaria.

ANIMALS POISONED. The seed of sesbane are poisonous to sheep, goats and cattle. Marsh (1929) states that about an ounce of seed will kill a sheep and less than two ounces will kill larger animals.

CLINICAL SIGNS. The primary signs of cases of light poisoning by sesbane are depression, diarrhea and rapid pulse. In fatal cases, weakness and labored breathing precede death. Signs ordinarily appear about a day after eating sesbane (Marsh 1920).

MANAGEMENT AND TREATMENT. The major problem in Drummond sesbane-infested areas is to keep animals from browsing the plant and consuming the fruit during winter. Ample supplemental feed at this time usually keep animals from consuming toxic quantities. Laxatives help poisoned animals eliminate the toxic material. The removal of plants is a good precaution. Hazard sites also may be fenced to prevent browsing. Young plants can be controlled by spraying with 1 pound of 2,4-D in 15 to 20 gallons of water per acre. Treated areas should be deferred a month or more following spraying to allow sprayed plants to dry completely.

Sesbania vesicaria-Bagpod Sesbane

DESCRIPTION. Bagpod sesbane is a smoothstemmed annual legume with compound leaves. The racemes are four to eight-flowered. The one-celled, usually two-seeded fruit has a loose-fitting outer portion and a thin white inner membrane, Figure 81.

DISTRIBUTION. Bagpod sesbane extends from the Gulf States into Texas. It is frequently abundant in old fields, overgrazed pastures and in disturbed and waste places.

ANIMALS POISONED, SIGNS, MANAGEMENT AND TREAT-MENT. See Sesbania drummondii. Areas to be grazed should be mowed if the plant is abundant.

Solanum carolinense-Horsenettle, Treadsalve

DESCRIPTION. Horsenettle is a herbaceous perennial of the nightshade family (Solanaceae). The leaves are mostly ovate, lobed or toothed with stellate hairs. The flowers are blue and the globose fruit is berry-like. Prickles are present on the stems and leaves, Figure 82.

DISTRIBUTION. Horsenettle may be abundant in overgrazed pastures, overflow areas, cultivated fields and waste places. It extends from Massachusetts to Nebraska, south to Texas and Florida and in some western states.

ANIMALS POISONED, SIGNS, LESIONS, MANAGEMENT AND TREATMENT. See silverleaf nightshade.

Solanum elaeagnifolium-Silverleaf Nightshade

DESCRIPTION. Silverleaf nightshade is a perennial with long, creeping rootstocks. The leaves are oblong to linear, undulate to deeply sinuate, silvery-white or stellate canescent. The flowers are violet and yellow or bluish. The plants usually are prickly, varying in degrees from sparse to heavily-covered, Figure 83.

DISTRIBUTION. This species is widely distributed in fields, pastures and roadsides. It is found from Missouri to Texas and California and south into Mexico.

ANIMALS POISONED. Horses, cattle, sheep, goats, swine, chickens, ducks, rabbits and men have been poisoned by eating *Solanum* spp.

CLINICAL SIGNS. The signs vary with the species of *Solanum* but usually include labored breathing, with an expiratory grunt, salivation and nasal dis-



Figure 82. Horsenettle, Solanum carolinense.

Figure 83. Silverleaf Nightshade, Solanum elaeagnifolium.



charge. The temperature may be normal to slightly above normal. In subacute cases a yellow discoloration of the skin may be observed in lightly pigmented areas. Other signs observed are weakness and incoordination, trembling of the muscles of the hind legs, anemia and accelerated heart rate. The animals may bloat. Often they are found dead with evidence of excessive salivation (Dollahite and Allen 1960).

LESIONS. The lungs are congested and emphysematous, the liver and kidneys are congested and the heart, intestinal mucosa and spleen are hemorrhagic and congested. In Hereford cattle, the fat frequently is yellow and contains a gelatinous infiltration. The gall bladder may be distended with dark green bile. The plant may be identified in the rumen of animals that die suddenly from eating green plant material. In cases of poisoning from eating the ripe fruit, the small, tomato-like seed will usually be found in large quantities between the folds of the omasum and in lesser numbers in the abomasum.

POISONOUS PRINCIPLE. The leaves and fruit of the toxic species of *Solanum* contain alkaloids, especially solanine. Of the species included, silverleaf nightshade is the most poisonous in Texas and frequently causes extensive losses in cattle. Cattle will eat the green plant when there is an inadequate supply of other green feed. They will also eat the ripe fruit, which is as toxic as the green fruit. In addition to the four species described, other plants in the nightshade family (Solanaceae) are known to be poisonous. The foliage of tomato and potato



Figure 84. Black Nightshade, Solanum nigrum.

plants and silage made from potato plants have poisoned animals. When the potato tuber is exposed to light it turns green. The green portion contains the poisonous principle and should not be eaten (Konig 1953).

MANAGEMENT AND TREATMENT. Hungry animals should not be allowed to graze areas where there is an abundance of silverleaf nightshade. Animals should not be fed from the ground in areas where there is a large quantity of the ripe fruit of silverleaf nightshade or horsenettle. Most of the cattle that have survived for 24 hours after eating silverleaf nightshade will recover if they are placed in the shade, fed, watered and kept quiet.

Solanum nigrum-Black Nightshade

DESCRIPTION. Black nightshade is an herbaceous annual with alternate, wavy-toothed or nearly entire leaves. The white flowers are in umble-like drooping clusters. The globose fruit is purple or black, Figure 84.

DISTRIBUTION. Black nightshade grows on disturbed areas of fields and pastures and in somewhat moist, open woodlands on loamy or gravelly soils. It is widespread throughout the United States and southern Canada.

POISONOUS PRINCIPLE, ANIMALS POISONED, SIGNS, LESIONS, MANAGEMENT AND TREATMENT. See silverleaf nightshade, Solanum elaeagnifolium.

Solanum rostratum-Buffalobur

DESCRIPTION. Buffalobur is a prickly annual with pinnatifid leaves. The flowers are yellow and the fruit is enclosed by a close-fitting, prickly calyx creating the "bur," Figure 85.

DISTRIBUTION. It is a weed in old fields, overgrazed pastures and along roadsides. It is found growing from North Dakota to Texas, westward and south into Mexico.

ANIMALS POISONED, SIGNS, LESIONS, MANAGEMENT AND TREATMENT. See silverleaf nightshade, Solanum



Figure 85. Buffalobur, Solanum rostratum.



Figure 86. Mescalbean, Sophora secundiflora.

elaeagnifolium. In addition to the toxic substances in this plant, the prickles cause internal irriation when grazed. Mowing in the early growth stage is a good control measure.

Sophora secundiflora-Mescalbean, Mountain Laurel, Frijolito

DESCRIPTION. Mescalbean is a shrub or small tree of the legume family, Figure 86. The oddpinnate leaves are evergreen, leather-like, dark green above and light green below. The showy bluish flowers smell sweet and the fruit is a several-seeded, woody pod, Figure 87. The seed are bright orange to scarlet-red and have a very hard seedcoat.

DISTRIBUTION. Mescalbean is most frequent on the Edwards Plateau and Trans-Pecos areas with records from Duval, Live Oak and Kleberg counties in the southern part of the state, Figure 88. This shrub extends into New Mexico on the west and Mexico on the south. It is found on hills, rock ledges and canyons and may become abundant following fire and mechanical brush control.

ANIMALS POISONED. Cattle, sheep and goats have been poisoned by eating the leaves or seeds of mescalbeans. Cattle are very susceptible to poisoning from the leaves, but goats and sheep are more tolerant. The seeds are quite poisonous if crushed, but due to their hardness they usually are swallowed without being broken. They are expelled unchanged during the process of rumination or are passed unchanged in the feces.

CLINICAL SIGNS. When animals poisoned by mescalbean are exercised, they have an increased pulse and a stiffening of the hind legs. Muscular trembling is especially noticeable over the shoulders and rump. They fall and usually become comatose. Sheep usually recover after a rest period, but cattle often die. Goats were not poisoned by experimental feeding (Boughton and Hardy 1935).

POISONOUS PRINCIPLE. Mescalbean is highly poisonous and contains the narcotic alkaloid cytisine or sophorine. Feeding tests of leaves have demonstrated



Figure 87. Fruiting branch of Mescalbean.

their toxicity to sheep, cattle and goats (Boughton and Hardy 1935). Cattle are very susceptible to the poison, while goats and sheep are less susceptible. The poison is not cumulative.

MANAGEMENT AND TREATMENT. When other forage is scarce, animals should be provided supplemental feed so that they will not consume poisonous amounts of mescalbean. Affected animals should be confined and fed a good ration until they recover.

Control experiments with herbicides obtained satisfactory control by basal application of 16 pounds of the ester formulations of 2,4,5-T mixed in 100 gallons of diesel oil when applied during the late spring and summer following effective rainfall (Sperry 1964).



Figure 88. Mescalbean, Sophora secundiflora.

Sophora sericea-Silky Sophora

DESCRIPTION. Silky sophora is a herbaceous, lowgrowing legume with alternate, pinnately-compound leaves, Figure 89. This species is common over much of West Texas and extends north to South Dakota and Wyoming, and west to Utah and Arizona.

POISONOUS PRINCIPLE AND ANIMALS POISONED. The seed, as with other species of *Sophora*, contain a toxic alkaloid. If eaten in large quantities, poisoning may result. Cattle losses in extreme West Texas have been attributed to this plant.

Sorghum halepense–Johnsongrass Sorghum vulgare–Sorghums Sorghum almum–Sorghum Almum

DESCRIPTION AND DISTRIBUTION. A large number of sorghum varieties and strains are under cultivation or growing as escapes. Sorghums generally are coarse grasses with large, terminal panicles. They are grown for grain, hay, silage and pasturage and are widely distributed.

Johnsongrass was introduced into South Carolina from Turkey about 1830. Ten years later Col. William Johnson obtained seed and planted it in the Alabama River bottom near Selma, Alabama. Since then, Johnsongrass has spread throughout the South. Johnsongrass is used primarily for hay, and grazing is secondary.

Sudangrass, Sorghum vulgare var. sudanese, was introduced into the United States from the African Sudan in 1909 and was first grown at the Chillicothe Experiment Station. The strains of Sudangrass include common, sweet, tift, piper, lahoma and green-

Figure 89. Silky sophora, Sophora sericea.



leaf. Sudangrasses are used for hay and grazing and the strains generally are classed as annuals, though some are weak perennials.

Sorghum almum was introduced into the United States from Argentina, South Africa, Australia, Nigeria and Algeria in 1949 and first grown in Georgia. It is also known as Columbusgrass. It originated in Argentina and is a hybrid between Johnsongrass and a sorghum. It is a weak perennial but should be managed as an annual.

ANIMALS POISONED. Cattle and horses are most susceptible to poisoning by sorghums.

CLINICAL SIGNS, LESIONS AND TREATMENT. See "Hydrocyanic Acid" under special problems.

POISONOUS PRINCIPLE. Under certain conditions, Johnsongrass and the sorghums produce a glycocide which yields several toxic substances during digestion. A deadly poison which may be formed is hydrocyanic (prussic) acid.

MANAGEMENT. Sorghums used for grazing should be 18 to 24 inches tall before livestock is allowed to consume them. Grazing during a younger stage apparently increases the danger of poisoning. Mature forage that is not grazed should be mowed to allow new growth. To maintain Johnsongrass in a healthy state, it is necessary to allow it to mature, seed and restore food reserve in the rhizomes. Plowing established stands at four year intervals stimulates growth. The hay sorghums should be cut during the boot stage of growth for best yield and quality. Crimping freshly mowed forage hastens drying and maintains high quality. Grazing the sorghums in small blocks in rotation allows highest yield and ease of management. Sorghums used for silage should be cut at the stage of growth that produces the largest yield.

Stillingia treculiana-Trecul Queensdelight

DESCRIPTION. Plants of queensdelight are monocious, many-stemmed herbs with a large central root. The stems may be somewhat woody toward the base. The smooth leaves usually are alternate, entire or toothed. The staminate flowers usually are terminal, pistillate flowers are at the base of the spike, Figure 90. The capsules are three-lobed. In addition to Trecul queensdelight, Texas queensdelight, *S. texana* and the more common species, *S. sylvatica*, are recorded for Texas.

DISTRIBUTION. Certain species are present in every area of Texas. S. sylvatica is most widely distributed in the state, Texas queesdelight is in the Cross Timbers, prairies and Edwards Plateau. Trecul queensdelight ranges from the Gulf Prairies to the Trans-Pecos area.

ANIMALS POISONED. Numerous sheep losses have been attributed to queensdelight in the Edwards Plateau area. Sheep fed experimentally have been killed with hydrocyanic acid poisoning, (Hardy 1954). POISONOUS PRINCIPLE. The analysis of dry plant material of *S. treculiana* showed that it contained HCN, (Hardy 1954).

MANAGEMENT. Since both lambs and mature sheep have been lost on poor-condition pastures infested with queensdelight, supplemental feeding or removal of animals is advocated.

Tribulus terrestris-Puncturevine, Goathead

DESCRIPTION. Puncturevine is an annual weed in the caltrop family (Zygophyllaceae). The prostrate stems radiate from a tap root. The opposite leaves are pinnate. The flattened fruit breaks into five nutlets, with two strong spines.

DISTRIBUTION. Puncturevine is a weed naturalized from Europe. It is widely distributed in waste places, along trails, roadsides and may become abundant in overgrazed pastures.

POISONOUS PRINCIPLE. Puncturevine contains a photodynamic agent and causes photosensitization, comonly called big head or swell head, of sheep. The preflowering, flowering and fruiting plants are toxic (Clare 1952).

CLINICAL SIGNS, LESIONS, MANAGEMENT AND TREAT-MENT. See Photosensitization. Puncturevine in fields can be controlled by cultivation. Effective control on local sites in noncultivated areas is possible by burning or spraying young plants with herbicides. Plants bearing seed, if cut off with a hoe or shovel, should be dried and burned to destroy the seed.

Xanthium spp.-Cocklebur

DESCRIPTION. Cockleburs are coarse, rough annual weeds with alternate, toothed or lobed leaves. The flowers are inconspicuous and imperfect, the pistilate flowers developing into a bur-like fruit, Figure 91. The fruit is usually two-beaked, covered with many spines and has two compartments, each containing a seed. *Xanthium* is a genus of the composite family.

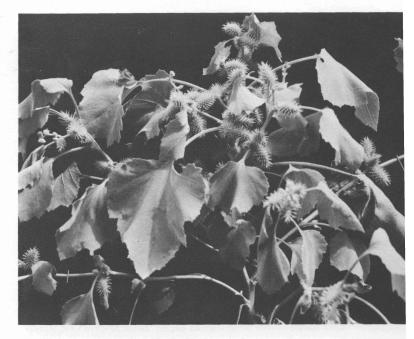


Figure 91. Cocklebur, Xanthium spp.

DISTRIBUTION. Over 40 species of Xanthium are widely distributed, six of them in Texas. One species, X. spinosum, has triple spines in the axils of the leaves. Cockleburs are most frequent in disturbed and flooded areas of fields, pastures and roadways. They may be present in bar ditches, around earthen tanks, barnyard lots, old lake beds and river bottoms. In many low areas where water flows or stands after a rain, the cocklebur plants are among the first green plants to appear.

ANIMALS POISONED. Swine, cattle, sheep, chickens and horses are poisoned by cocklebur plants. Swine are most frequently poisoned, but poisoning is quite frequent in cattle. The burs are seldom eaten. Most poisoning is caused by eating of plants in the cotyledon stage (Marsh 1929).

CLINICAL SIGNS. As little as 0.75 percent of an animal's weight of young seedlings will produce signs

Figure 90. Texas Queensdelight, Stillingia texana.



of illness in a few hours and death within 24 to 48 hours. Animals poisoned by cockleburs have low temperatures and show signs of depression and general weakness. They have a rapid and weak pulse, labored breathing, nausea, vomiting and spasmodic contraction of leg and neck muscles. Pigs may squeal and have convulsions.

LESIONS. There usually is gastroenteritis with thickening of the walls of the inflamed tissues. The liver is congested, the bile is viscid and there is a slight icterus. Ascites may be seen.

POISONOUS PRINCIPLE. Cockleburs are mechanically injurious when consumed and also contain a toxic glucoside.

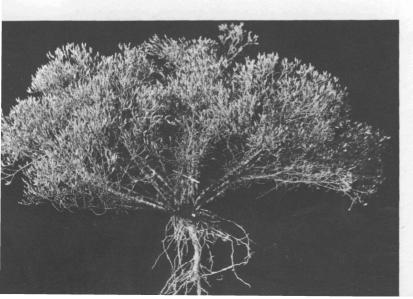
MANAGEMENT AND TREATMENT. Animals should not be allowed to graze areas where there are abundant young cocklebur plants unless there is adequate green forage. Poisoned animals should be kept warm and large doses of mineral oil or milk should be given by mouth with heart and respiratory stimulants administered. Hubert and Oehme (1961) recommended physostigmine (5-30 mg.) injected intramuscularly and repeated at $\frac{1}{2}$ to 1 hour intervals for pigs.

Cockleburs, in addition to being poisonous, are obnoxious when burs get in the wool, hair and tails of animals. Young plants are highly susceptible to herbicides and can be controlled by broadcast spraying with 2,4-D at the rate of $\frac{1}{2}$ to 1 pound per acre.

Xanthocephalum microcephalum (Gutierrezia microcephala)-Threadleaf Broomweed, Perennial Broomweed, Turpentineweed, Slinkweed

DESCRIPTION. Threadleaf broomweed is a manybranched, perennial, herbaceous composite. The numerous yellow-flowered heads are narrow turbinate (top-shaped). Each head has from one to three disk and from four to five ray flowers. The leaves are alternate and filiform, Figure 92. There are five

Figure 92. Threadleaf Broomweed, Xanthocephalum micro-cephalum.



additional perennial species classified under Xanthocephalum in the Texas flora.

DISTRIBUTION. The most abundant stands of threadleaf broomweed are in the Trans-Pecos area. It extends from Central and West Texas south into Mexico and west to Nevada.

ANIMALS POISONED. Threadleaf broomweed is poisonous to cattle, sheep, goats, swine, rabbits, guinea pigs and young chickens. The most common problem with this plant is abortion in cattle. This problem becomes much more serious when broomweed grows on sandy soil; often 60 percent of the cows abort, delivering dead or small weak calves. Abortion has been produced experimentally in cows, sheep, goats and rabbits. Swine grazing broomweed may abort, and breeding has been delayed by experimental feeding (Dollahite and Allen 1959).

CLINICAL SIGNS. Acute poisoning by broomweed produces different clinical signs from those usually seen in cows prior to abortion. They develop periodic mucopurulent nasal discharge, the skin of the muzzle becomes crusted and sloughs and buccal ulcers may be present. The cattle lose their appetite, lose weight, have a listless attitude and develop a rough coat. Occasionally they have dark brown or reddish urine. Animals urinate frequently and twist as if urination causes pain. Diarrhea, observed in early stages, changes to constipation and large amounts of mucus are found in the foul-smelling feces. Pregnant cows often have periodic vulvar swelling and an earlierthan-normal udder development. Hereford cows, which usually do not have large udders, often develop painful engorgement of the udder before aborting. The placenta usually is retained following abortion and secondary infection causes some deaths (Dollahite and Anthony 1957).

LESIONS. Although there is no clinical evidence of icterus, the subcutaneous fat and connective tissue is light yellow to golden-orange. The liver is soft and friable, the capsule peels easily and there are often pinpoint hemorrhages under the capsule. The gallbladder shows inflammation of the mucous membrane which is stained yellow by the thick yellow bile. The kidneys and spleen are congested and the pulp of the spleen is darker and softer than normal. There is a mild to severe gastroenteritis. The fetal membranes usually are edematous.

POISONOUS PRINCIPLE. Dollahite et al. (1962) reported the isolation of a saponin from Xanthocephalum spp. which will induce abortion when injected intravenously. Shaver et al. (1964) have demonstrated that the saponin will induce abortion when administered orally to rabbits. The isolated saponin was shown to stimulate contractions in isolated rat and rabbit uteri. Qualitative tests indicate that the saponin is of the triterpenoid type.

MANAGEMENT AND TREATMENT. Heaviest losses occur when broomweed leaves grow rapidly. Abor-

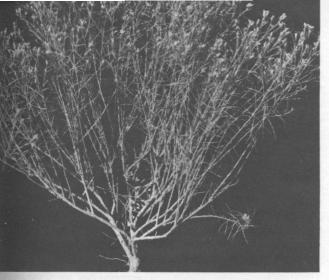


Figure 93. Perennial Broomweed, Xanthocephalum sarothrae.

tions were produced in experimental cows with smaller quantities of the plant at this stage of growth. Rapid leaf growth may occur occasionally in October and November, but usually occurs in late winter and early spring. Cattle eat broomweed readily at this stage of growth when other green feed is scarce. Cattle should be moved from sandy soils to loam soils during danger periods. In areas where poisoning occurs on other soil types, cattle should be moved to pastures that are free from broomweed. Cows poisoned by broomweed should be placed in pens and fed nutritious feed until they recover. Cows with retained placentas should be treated by a veterinarian. Calves that are born alive, but small and weak, should be placed in the shade and helped to nurse until they are strong enough to care for themselves.

Experimental control with herbicides has shown threadleaf broomweed to be susceptible to 2,4-D. See Xanthocephalum sarothrae for more detail.

Xanthocephalum sarothrae (Gutierrezia sarothrae)-Perennial Broomweed, Broom Snakeweed, Turpentineweed, Slinkweed

DESCRIPTION. This species of Xanthocephalum is similar in growth habit to X. microcephalum. The flower heads are slightly thicker and have from three to eight ray flowers and from three to eight disk flowers, Figure 93.

DISTRIBUTION. It is found in greatest abundance on the Edwards Plateau, the High and Rolling Plains and the Cross Timbers and Prairies. Collections also have been made in the Gulf Prairies and Trans-Pecos area.

POISONOUS PRINCIPLE, ANIMALS POISONED, SIGNS, LESIONS, MANAGEMENT AND TREATMENT. Experimental feeding and control studies have involved both X. microcephalum and X. sarothrae. See discussions under X. microcephalum.

Since X. sarothrae is more widely distributed than X. microcephalum, most of the control experiments have been with the former. Sperry and Robison (1963) reported the progress of control experiments in which 2,4-D has obtained the best results to date. Spraying must be done under optimum growth and moisture conditions and it is indicated that two successive years of treatment may be necessary to get satisfactory control.

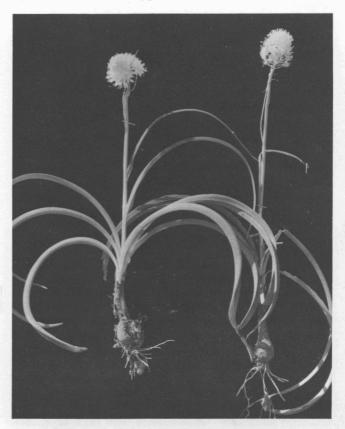
Zygadenus nuttallii-Nuttall Deathcamas

DESCRIPTION. Nuttall deathcamas, a member of the lily family (Liliaceae), is one of several poisonous species of the genus Zygadenus. It has long, somewhat curved leaves, an underground bulb and flower stalks 1 to 2 feet tall. The white flowers are borne in a dense terminal cluster, Figure 94. The fruit is a dry many-seeded capsule. Deathcamas plants are related to, and may be confused with, wild onions, especially before flowering.

DISTRIBUTION. This species is most prevalent in Texas on the eastern portion of the Edwards Plateau, in the central and northern prairies and in post oak areas. It grows northward into Kansas and eastward into Tennessee. Other species have been reported for Texas but are not frequent enough to be listed. Nuttall deathcamas grows mostly in grasslands and open woodlands.

POISONOUS PRINCIPLE. The toxicity of deathcamas is thought to be due to an alkaloid. Considerable

Figure 94. Deathcamas, Zygadenus nuttallii.



work has been done on range species, which vary in their poisonous properties (Marsh and Clawson 1929).

ANIMALS POISONED. Nuttall deathcamas is poisonous to sheep, cattle and horses. This species is reported to be more poisonous than deathcamas in the western states. All parts of the plant are poisonous, even when dry (Marsh 1929).

CLINICAL SIGNS. Signs of deathcamas poisoning are salivation, nausea, vomiting, depression, weakness, low temperature, a weak and irregular pulse and irregular, difficult breathing. Death is usually preceded by coma.

LESIONS. Lesions are minimal, but there is usually congestion of the kidneys, and there may be congestion of the lungs and other organs.

MANAGEMENT AND TREATMENT. Livestock should be kept away from heavily infested areas of deathcamas, especially in the spring. Supplementary feeding will help prevent losses. Although there is no effective treatment, many poisoned animals will recover if kept quiet and fed.

Since nuttall deathcamas are conspicuous when in flower and usually occur as scattered plants, it is practical to dig out and destroy them. The bulb may be from a few inches to a foot or more below the surface.

Special Problems

ERGOT

Claviceps cinerea, Tobosagrass Ergot Claviceps paspalum, Dallisgrass Ergot Claviceps purpurea, Ergot of Rye or

Other Cereal Grasses

ANIMALS POISONED. Most cases of ergot poisoning in Texas occur in cattle. Under favorable climatic conditions, enough of the ergot sclerotia may develop to cause poisoning. Ergot may be associated with other fungi, complicating toxic conditions. Some of these fungi cause a "honey-dew" to form on the sclerotia making them palatable to livestock.

CLINICAL SIGNS. Ergotism is divided into two different disease syndromes: acute, usually is characterized by nervous signs, and chronic, characterized by gangrene. Acute or nervous ergotism in cattle caused by dallisgrass ergot is the more common problem in Texas. Ergot growing on tobosagrass in western Texas causes a similar nervous condition (Dollahite 1963). Signs of nervous ergotism are extreme nervousness, increased heart rate, muscular trembling and frequent urination. There may be a loss of the special senses followed by ataxia, prostration, convulsions and death. Gangrenous ergotism is more often caused by Claviceps purpurea growing on some of the cereal grasses. Gangrene affects parts of the body having poorest blood supply: feet, legs, tail and ears, any or all of which may drop off.

Frequently cattle are able to walk without hooves, apparently without pain. Abortion is thought to be a sign of chronic ergotism. Although research workers usually have failed to produce abortion with ergot, abortions are frequently associated with gangrenous ergotism in the field. Gangrene and abortions have been observed in herds following outbreaks of the nervous form of abortion.

MANAGEMENT AND TREATMENT. Grass containing ergot should be mowed, or cattle should be moved. Animals with gangrene often have to be destroyed for humane reasons. Animals with nervous ergotism usually will recover if removed from the ergot-infested pasture, given good feed, water and not unduly disturbed.

HYDROCYANIC OR PRUSSIC ACID POISONING

Some plants are capable, under certain conditions, of producing hydrocyanic (prussic) acid, a highly poisonous substance. They are known as cyanogenetic plants. The acid forms when an enzyme or rumen bacteria act on a glycoside. Neither the glycoside nor the enzyme is individually poisonous, and under normal conditions they do not come in contact. Poisoning usually follows plant damage such as wilting, freezing, drouth, cutting, crushing, etc. Young growing plants usually contain more of the glycoside than mature plants. Drying of plants usually reduces the toxicity, but animals occasionally are poisoned by eating hay from cyanogenetic plants.

Of the plants that grow in Texas, the Sorghum species are the ones that most frequently produce cyanide poisoning. This includes all of the grain sorghums, Johnsongrass and Sudangrass. Other plants that produce cyanide poisoning are cherries, plums, velvetgrass (Holcus lanatus), Mountain mahogany (Cercocarpus brevifolius), flax (Linum spp.), queensdelight, elder (Sambucus spp.), American basket flower (Centaurea americana), eastern mannagrass (Glyceria septentrionalis) and sticky palefoxia (Palafoxia tripteris).

ANIMALS POISONED. All domestic animals are susceptible to hydrocyanic acid poisoning but cattle are most susceptible.

SIGNS OF ILLNESS. Hydrocyanic acid is one of the most rapid acting poisons. Signs of illness may start within 5 minutes of the time the animal starts eating the plant. Death may occur within 15 minutes, or the animal may live for several hours. Salivation and labored breathing are usually the first signs, followed by muscle tremors, incoordination, bloating, tetanic muscle contractions, convulsions and death due to respiratory failure. The heart may continue to beat for some time after breathing stops.

LESIONS. The blood is usually bright red and there is congestion of the lungs and other internal organs. There may be hemorrhages on the serous surface of the digestive tract.

MANAGEMENT AND TREATMENT. Animals should not be allowed to eat wilted cherry and plum leaves and root sprouts. Second growth sorghums should be grazed with caution. When any of the cyanogenetic plants have been subject to drouth, freezing or other damage, they should not be grazed unless tested for hydrocyanic acid.

Sodium nitrite and sodium thiosulfate are specific antidotes for hydrocyanic acid poisoning. Solutions should be given intravenously because the poison acts rapidly. Most veterinarians carry solutions ready to administer at all times. Methylene blue given intravenously is another antidote.

NITRATE POISONING

Nitrate poisoning in animals usually is caused by their consuming fertilizers, water, feed or forage plants containing poisonous nitrogen compounds such as potassium nitrate. Potassium nitrite is much more toxic than potassium nitrate. Moisture and heat may change the nitrate to nitrite, but most of this change occurs in the gastro-intestinal tract, and is thought to occur in rumen of ruminants. The condition is characterized by abortion, reduction of milk flow, signs of vitamin A deficiency and sudden death. When non-protein nitrogen is excessive in the plant, it may combine with other elements to produce poisons that add to the complexity of the nitrate poisoning. One of these compounds is nitrogen dioxide, a brown or yellowish-red gas heavier than air which may form in silos.

Nitrogen is an indispensable constituent of the protein molecule, which in turn is a vital part of the protoplasm of plants. Some plants may accumulate nitrates to a toxic level. The enzyme, nitrate reductase, is important in this building process and requires light to function. Most investigators have associated nitrate poisoning with plants that are grown in soils having a high nitrogen content, such as in areas that once were livestock pens or where the soil has been highly fertilized. Gilbert et al. (1946) showed that high nitrate in the soil will increase the amount of nitrate in growing plants. They also showed that when nitrogen alone is applied to soil the nitrogen in plants is higher than when nitrogen is applied with phosphorus. Plants usually accumulate nitrate when under stress, such as growing in the shade or on certain soil types (Gilbert et al. 1946); suffering from lack of moisture (Dollahite and Camp 1956); or suffering from physical damage, such as being trampled or blown down during a storm (Dollahite 1960).

In the Rocky Mountain States, oat is most commonly responsible for nitrate poisoning, and the illness has been known as "oat poisoning." Other field crops that have been known to cause nitrate poisoning are barley, wheat, rye, sorghum, corn, Sudan, alfalfa, millet, soybean, rape and fescue. Corn

has been reported to have as much as 25 percent nitrate on a dry weight basis (Gilbert, et al. 1946). Some grasses and many weeds accumulating nitrates are amaranths, sunflower, lambsquarter, Russian thistle, ragweed, witchgrass, nightshade, white cockle, velvetweed, smartweed, burdock, hounds-tongue, fiddleneck, Canada thistle, bull thistle, bindweed, sourdock, stinging nettle, elderberry, goldenrod, sweetpotato vines and blue-green algae. Vegetables accumulating large quantities of nitrate are beets, turnips, kale, radishes, mangels, swiss chard, lettuce, celery, squash, parsnips, cucumbers and spinach. Humans usually are not poisoned by these vegetables because they seldom eat them exclusively, but when fed to animals in quantity they often cause nitrate poisoning.

The poisonous effects of nitrate vary with different species of animals, and with the ration that the animal is eating. Because of these varying conditions, the amount of nitrate reported by different workers to produce poisoning varies widely. Under most conditions, 1.5 percent of the ration in nitrate on a dry weight basis produces acute poisoning and death, while lesser amounts will produce abortion. One-half percent of the ration over an extended period will cause lower milk production and lower weight gains. Nitrate is thought to interfere with the conversion of carotene to vitamin A (Pugh *et al.* 1962).

ANIMALS POISONED. Cattle, sheep, goats, swine, horses, dogs and birds are susceptible to nitrate poisoning. Cattle are more frequently poisoned than other animals.

In acute nitrate poisoning, CLINICAL SIGNS. animals are often found dead with no previous history of illness. Numerous abortions may occur in the herd a few days after the deaths, or there may be abortions and no deaths. Poisoned cattle have signs of nausea, vomiting, abdominal pain, bloating, fast pulse, prostration and dyspnea. The blood is usually dark brown, but may be cherry red. Sheep exhibit the same signs as cattle, and in addition have frequent urination and convulsions. Fowl stop eating, develop a watery diarrhea with green flakes, cyanosis and depression. The damage is caused by vasodilation as a result of the nitrate and the lower oxygen carrying capacity of the blood due to methemoglobin (brown blood) formation.

LESIONS. Lesions of nitrate poisoning are lung congestion, hemorrhages on the heart, brownish liver, chocolate-brown blood, irritation of the stomach and intestines and sometimes, hemorrhage in the urinary bladder. The brown color of the blood disappears 3 or 4 hours after death. Microscopically, there is degeneration of the vascular tissues of the lungs, heart, liver, kidneys, testes and brain.

MANAGEMENT AND TREATMENT. Rations high in carbohydrates will reduce and sometimes prevent

losses from nitrate poisoning. Water for human or animal consumption, if in question, should be checked for nitrates and nitrites. Weed control may prevent nitrate poisoning. If there is any reason to suspect that plants contain an excess of nitrates, they should be tested before animals are allowed to eat them.

Poisoned animals should be handled quietly and methylene blue should be administered intravenously. It is usually administered in a 1-4 percent solution which also contains 5 percent dextrose at the rate of 1 gram of methylene blue for each 250 pounds of animal weight. Due to the vasodilation effect of the nitrate, vasoconstrictor drugs such as adrenalin should be administered. In chronic poisoning, vitamin A should be given.

PHOTOSENSITIZATION

Photosensitivity is that condition in animals characterized by hyper-reaction to sunlight. Photosensitization is the syndrome which develops when an animal in a state of photosensitivity is exposed to sunlight (Clare 1952). It has been classified by Runnels *et al.* (1960) into three types:

TYPE I. In primary photosensitivity the photodynamic agent is absorbed directly from the digestive tract. It does not depend on functional or metabolic disturbances of the body organs. Plants that cause this type of photosensitivity are *Polygonum persicaria* (ladies-thumb), *Hypericum perforatum* (St. John's wort) and species of *Eriogonum* (wild-buckwheat).

TYPE II. Photosensitivity due to abnormal pigment synthesis is due to a hereditary defect in porphyrin metabolism and occurs in some breeds of cattle and in Southdown sheep.

TYPE III. Hepatogenous photosensitivity is due to the accumulation of phylloerythrin in the peripheral circulation. Hepatic or biliary injury is the primary lesion, which interferes with excretion of phylloerythrin and produces photosensitivity. Plant and chemical hepatotoxins, certain infectious diseases and mechanical obstruction of the bile ducts are capable of producing enough damage to interfere with excretion of phylloerythrin. Most photosensitivity diseases are of this type. Plants that cause hepatogenous photosensitivity are puncturevine, lechuguilla, sacahuista and species of Lippia and Lantana. The fungus Pithomyces chartarum, growing on rye grass, produces a hepatotoxin, sporidesmin, which causes facial eczema, a photosensitive disease important in New Zealand.

Several annual grasses in certain states and stages of growth are apparently responsible for hepatogenous sensitivity (Sperry *et al.* 1955).

The etiology of the photosensitivity of some syndromes is not well understood and cannot be classified in the above groups. They usually occur in animals grazing on luxuriant pastures of oat, wheat, rye, Sudangrass, Johnsongrass or clovers. Early workers placed these plants with those in Type I. Evidence of hepatic damage usually is not observed under these conditions, but there are many exceptions where there is a pronounced icterus.

Photosensitization has been reported from many areas as a result of eating common forage plants such as Bermudagrass and clover infected with mold. Alfalfa and clover hay which contain molds have also caused photosensitization. These outbreaks usually follow heavy rains and climatic conditions that are conducive to growth of molds. Certain additional plants, not considered photodynamic, frequently produce hepatic damage with marked icterus when infested with molds.

ANIMALS POISONED. Cattle, horses, sheep, goats and swine are susceptible to photosensitization.

CLINICAL SIGNS. The first signs of photosensitization usually are conjunctivitis and reddening of light colored skin. Thin-skinned areas and those having no hair or thin hair, such as the muzzle, udder and scrotum, are also affected. Occasionally dark-pigmented skin is affected but less severely. The inflammation of the skin is followed by swelling, serous exudate, blisters and usually sloughing of the skin. Bacterial and screwworm infections may follow. The animals are sensitive to sunlight and seek shade. Evidence of itching is exhibited by scratching, kicking, rubbing against objects and biting the affected parts. The skin of dark animals does not blister and slough, but usually becomes thickened and the hair crusted with exudate.

Animals may develop laminitis (founder) with swelling of the coronary band. Animals with primary photosensitization seldom die, but animals with signs of icterus often die.

MANAGEMENT AND TREATMENT. When the plant causing photosensitization is known, preventive measures may be taken (Hoffman et al. 1962). It may be completely avoided or the amount consumed may be reduced by furnishing supplementary feed. Where climatic conditions occur that may lead to photosensitization, pasture rotation and supplementary feed may help prevent trouble. When the first cases of photosensitization occur, the animals should be moved to new pasture if possible. The sick animals should be placed in the shade with feed and water. Placing in screened barns to prevent exposure to flies is advisable. Painting or spraying the affected parts with methylene blue solution or some other nontoxic dye is beneficial. Applications of grease, oils or tannic acid should be avoided.

WHEAT PASTURE POISONING (Grass Staggers, Grass Tetany, Green Oat Tetany)

ANIMALS POISONED. Cattle, and sometimes sheep are poisoned after grazing wheat, oats and some native grasses. Although lactating or pregnant cows are usually affected, dry cows, steers and calves have also been poisoned. Most cases in cows develop between the fifth month of pregnancy and the time the calf is 60 days old. The cows usually have been grazing wheat between 60 and 150 days (Sims and Crookshank 1956).

CLINICAL SIGNS. Wheat pasture poisoning signs begin with nervousness, incoordination and loss of appetite. Viciousness, staggering, falling and muscular twitching of the extremities develop. The animals grind their teeth, have an anxious expression and salivate profusely. The third eyelid protrudes or flickers, and general tetanic contractions of the muscle follows until the animal becomes prostrate. A reflex response will occur if the animal is touched or if it hears a sudden noise. Labored breathing and a pounding heart develop followed by coma within 6 to 10 hours from the appearance of the first signs. If left untreated, animals will have convulsions and die.

MANAGEMENT AND TREATMENT. Supplemental feeding with hay and various trace minerals may delay but not necessarily prevent wheat poisoning. Treatment by intravenous or intraperitoneal injections of calcium gluconate solution fortified with magnesium and phosphorus is usually successful if started before coma develops. Recovery is more rapid and more certain if the animal is removed from the pasture for a few days. Animals that recover enough to stay on their feet for 24 hours usually do not have the disease again.

Plants Growing in Texas Known to Have Caused Animal Losses in Other Parts of the Continental United States

Agrostemma githago	Corncockle	Lactuca spp.	Wild lettuce
Allium spp.	Onion	Leucaena glauca	White popinac
Amanita spp.	Mushrooms	Ligustrum lucidum	Glossy privot
Argemone mexicana	Yellow poppy	Lolium temulentum	Darnel
Arisaema triphyllum	Jack-in-the-pulpit	Lyonia ligustrina	He-huckleberry
Baptisia spp.	False indigo	Maclura pomifera	Osage-orange, bois d' arc
Berberis repens	Creeping barberry	Melanthium virginicum	Bunchflower
Batis maritima	Maritime saltwort	Onoclea sensibilis	Sensitivefern
Buxus spp.	Boxwood	Papaver somniferum	Opium poppy
Cannabis sativa	Indian hemp (marihuana)	Phalaris tuberosa	Hardinggrass
Celastrus scandens	American bittersweet	Phoradendron spp.	Mistletoe
Chenopodium spp.	Lambsquarter, goosefoot	Physalis spp.	Ground-cherry
Croton spp.	Croton, doveweed	Pinus spp.	Pine
Crotalaria spp.	Crotolaria, rattleweed	Podophyllum peltatum	Mayapple
<i>Cyanophyta</i> genera	Blue-green algae	Psoralea tenuiflora	Scurfpea
	Yellow ladyslipper	Ranuculus spp.	Buttercup
Cypripedium calceolus		Rhododendron spp.	Rhododendron
Equisetum spp.	Horsetail, scouring-rush	Rhus spp.	Poison ivy, poison oak
Festuca elatior	Tall fescue	Robinia pseudo-acacia	Black locust
Gelsemium sempervirens	Yellow jasmine	Rumex spp.	Dock
Gossypium herbaceum	Cotton	Salvia coccinea	Tropical sage
Gymnocladus dioica	Kentucky coffee tree	Sanguinaria canadensis	Bloodroot
Hedera helix	English Ivy	Saponaria officinalis	Bouncingbet
Hydrangea macrophylla	Bigleaf hydrangea	Sartwellia flaveriae	Sartwellia
Ilex spp.	Holly	Solidago spp.	Goldenrod
Iris spp.	Iris	Spigelia marylandica	Pinkfoot

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The designation of vegetational areas in Texas for plant distribution follows the outline of vegetational areas of Texas by Gould *et al.* (1960). Plant names, both scientific and common, have followed, in most instances, The Check List of Texas Plants by Gould (1962). Binns, Wayne. 1961. U.S.D.A. News release, Aug. 1.

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