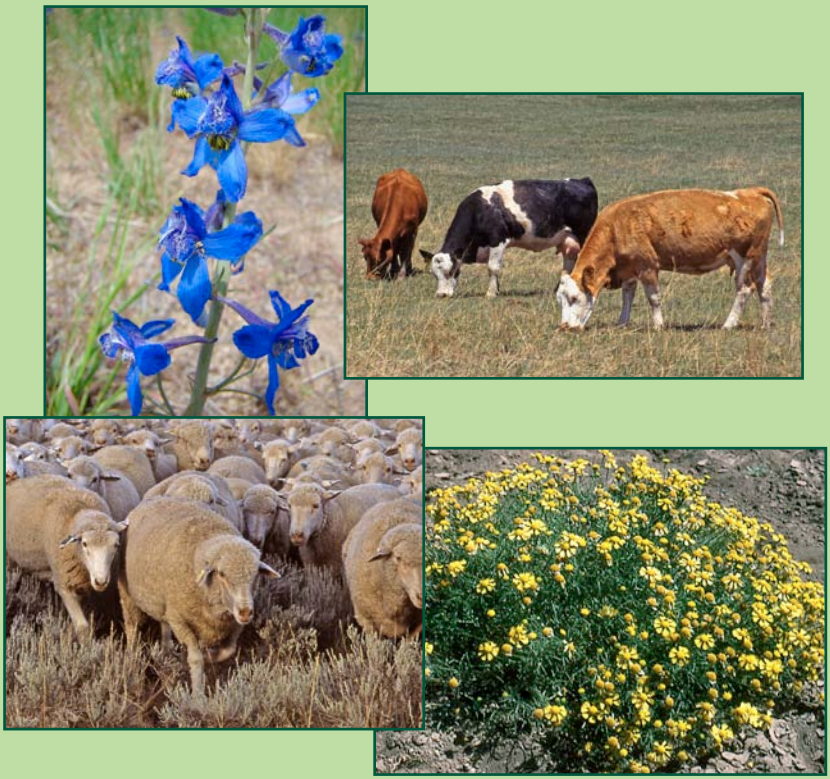
United States
Department of
Agriculture

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Bulletin Number 415

Revised April 2011

Plants Poisonous to Livestock in the Western States



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Prepared by K.E. Panter, M.H. Ralphs, J.A. Pfister, D.R. Gardner, B.L. Stegelmeier, S.T. Lee, K.D. Welch, B.T. Green, T.Z. Davis, and D. Cook, U.S. Department of Agriculture, Agricultural Research Service, Poisonous Plant Research Laboratory, Logan, Utah.

Abstract

Panter, K.E., M.H. Ralphs, J.A. Pfister, D.R. Gardner, B.L. Stegelmeier, S.T. Lee, K.D. Welch, B.T. Green, T.Z. Davis, and D. Cook. 2011. Plants Poisonous to Livestock in the Western States. U.S. Department of Agriculture, Agriculture Bulletin No. 415.

Poisonous plants are a major cause of economic loss to the livestock industry. Each year these plants adversely affect 3 to 5 percent of the cattle, sheep, and horses that graze western ranges. These losses result from death of livestock, abortions, photosensitization, decreased production, emaciation, and birth defects. In addition to these losses are those of increased management costs associated with such things as fencing, altered grazing programs, and loss of forage. This bulletin describes more than 30 of the principal poisonous plants growing on western ranges and the signs of poisoning in livestock. Suggestions are included for the prevention of livestock poisoning by plants.

Keywords: abortion, arrowgrass, birth defects, bitterweed, bracken fern, chokecherry, copperweed, death camas, emaciation, false hellebore, greasewood, groundsel, halogeton, hemp dogbane, horsebrush, kochia, larkspur, locoweed, lupine, milkvetch, milkweed, nightshade, nitrate, oak, photosensitization, pingue, poison hemlock, poisonous plants, ponderosa pine, rayless goldenrod, rubberweed, Russian knapweed, selenium, snakeweed, sneezeweed, spring parsley, St. Johnswort, sweet clover, tansy ragwort, veratrum, waterhemlock, yellow star thistle, yew

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Acknowledgments

The authors dedicate this latest revision of Plants Poisonous to Livestock in the Western States to Lynn F. James, Research Leader of the ARS Poisonous Plant Research Laboratory (PPRL) from 1972 to 2007. Under his guidance, PPRL gained a national and international reputation in the field of poisonous plants. He was the guiding force behind the original publication of this bulletin in 1980, and in 2005 he began the process of updating and revising the work. Dr. James retired from ARS in 2008 after over 50 years of service. He is a collaborator with PPRL and continues to share his insight and experience with the current staff.

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Poisonous Plants and Livestock

Poisonous plants are a major cause of economic loss to the livestock industry. Each year these plants adversely affect 3 to 5 percent of the cattle, sheep, goats, and horses that graze western ranges. All too often the losses to individual livestock operations are large enough to threaten the viability of that ranch.

Livestock losses can be heavy if animals—

- graze ranges infested with poisonous plants when plants are most toxic.
- are driven, trailed through, or unloaded from trucks onto range or pasture areas infested with poisonous plants. Animals are less selective in their grazing at these times of stress.
- are not watered regularly.
- are allowed to become hungry. Such animals are more likely to eat lethal quantities of poisonous plants.
- are grazed on rangelands early in spring when there is no other green vegetation except poisonous plants.
- are stressed, such as when they are trucked, penned, or handled (branding, vaccination, etc.).
- are not limited on how much and how fast they consume the plants.

There are no known treatments for animals poisoned by most poisonous plants. Where a treatment is available, affected animals are usually in remote places and cannot be reached until it is too late to provide treatment. Furthermore, the stress of handling poisoned animals may increase the probability of death. If the animals recover enough to be handled, treatment should consist primarily of symptomatic treatment except where a specific treatment is known.

Prevention of loss from poisonous plants in general is a problem of range and livestock management. Under normal conditions, some poisonous plants form an important part of livestock diets without negative effects on the animals. Poisoning occurs only when these animals are enticed by hunger or other stress conditions to eat too much too fast.

Many of the death losses of livestock from grazing poisonous plants are due to management error. This includes failure to examine a pasture or range prior to use because the growth of some of these plants varies from year to year. Failure to know and understand plants in an area that are toxic to livestock can lead to catastrophic losses.

Hundreds of plants are poisonous to livestock. Many are toxic at all

times, whereas others are toxic only under certain conditions. Livestock producers are urged to become familiar with the plants on their ranges and pastures that are potentially dangerous to their livestock. Help in identifying these plants can usually be obtained from the local county agricultural agent or by mailing a sample to the USDA-ARS Poisonous Plant Research Laboratory (PPRL) for identification. (See PPRL contact information at the end of this section.) Help with poisonous plant problems can also be obtained from local veterinarians.

Proper diagnosis of livestock poisoning is essential. However, under range conditions, diagnosis can be difficult.

Symptoms listed for each plant are those most likely to be observed. Not all symptoms will be seen in all toxicities and signs of poisoning may vary greatly, depending on dosage and the time taken to consume the dose. Also, individual animals respond differently to specific poisons.

To protect your animals from poisoning, do the following:

- Learn to identify the poisonous plants that grow on your range.
- Learn the conditions under which these plants can be dangerous to your livestock.
- Develop a grazing plan to improve your range and prevent poisoning. These plans should take into account the poisonous plants on the range, allowing animals to graze them at the most appropriate time or to avoid them.
- Do not allow animals that have been under stress or that are overly hungry to graze in areas infested with poisonous plants.
- Provide adequate water for your livestock.
- Be especially careful when grazing newly acquired livestock on your range.
- Provide adequate salt and other supplements as needed, but do not put them in an area where poisonous plants are growing.
- Control poisonous plants where feasible.
- If your animals get sick, consult your local veterinarian to ensure proper diagnosis and treatment. If a poisonous plant is involved, identification of the plant by an experienced botanist or poisonous plant expert is essential for any corrective action.
- Many poisonous plants can function as useful forage. Poisoning occurs when conditions develop that allow, or cause, the animal to eat too much too fast.
- Plants such as poison hemlock, water hemlock, arrowgrass,

kochia, hounds tongue, and lupine may be growing on land from which hay is harvested. This is especially true on native pastures. Care must be taken when feeding this hay.

For additional information on poisonous plants, contact—

USDA-ARS Poisonous Plant
Research Laboratory
1150 East 1400 North
Logan, Utah 84341
phone: 435-752-2941
http://www.ars.usda.gov/npa/
logan/pprl

Economic Impact of Poisonous Plants on Livestock

Direct losses (effects on animals) include the following:

- Deaths of livestock
- Abortions
- Birth defects
- Weight loss (due to illness or decreased feed intake)
- Lengthened calving interval
- Decreased fertility
- Decreased immune response
- Decreased function (due to damage to organs such as the nervous system, lungs, liver, etc.)
- Loss of breeding stock (due to deaths, functional inefficiency, etc.)

Indirect losses (management costs) include the following:

- Building and maintaining fences
- Increased feed requirements
- Increased medical treatments
- Altered grazing programs
- Decreased forage availability
- Decreased land values
- Opportunity costs
- Lost time to management
- Stress to management

Precautions for Using Herbicides

Herbicide rates in this publication refer to the acid equivalent (ae) or active ingredient (ai) of the active compound in the herbicide, except for metsulfuron (Escort), which is a dry flowable formulation (60 percent ai) and is expressed as ounces of product per acre.

Caution: *Pesticides used improperly can harm humans, domestic animals, beneficial insects, desirable plants, and fish or other wildlife. Follow the directions and heed all precautions on labels. Store pesticides in original containers under lock and key, out of the reach of children and animals, and away from food and feed.*

Note: Some States have restrictions on the use of certain pesticides. Check your state and local regulations. Also, because registrations of pesticides are under constant review by the U.S. Environmental Protection Agency, consult your local county agriculture agent or State extension specialist to be sure the intended use is still registered.

Always read the label carefully before using any herbicide. Remember that some chemicals, such as picloram, may have restricted use. Before using any herbicide, check first with your county agricultural agent or weed specialist. They can suggest the most suitable chemical for a specific problem and recommend an optimum time and rate of application.

Use only pesticides that bear the EPA registration number and carry appropriate directions.

Herbicides are safe when stored, handled, mixed, and used in accordance with label instructions and sound agricultural practices. Most herbicides are low in toxicity; however, some are poisonous to humans, domestic animals, fish, and wildlife. Most herbicides are toxic to broadleaf crops.

Label all herbicide containers clearly and appropriately.

Keep herbicides away from children, livestock, and pets. Store containers in a secured place where they are not available to children or anyone not authorized to use them. Store herbicides in closed, well-labeled containers in a dry place where they cannot contaminate food, feed, or water.

When handling herbicides, wear clean, dry clothing. Launder clothing after each spraying operation before wearing again. Wear protective clothing and equipment if specified on the label.

Apply pesticides so that they do not endanger humans, livestock, crops, beneficial insects, fish, and wildlife. Do not apply pesticides where there is danger of drift when honey bees or other pollinating insects are visiting plants or in ways that may contaminate water or leave illegal residues. Avoid spraying in windy conditions.

Do not inhale herbicides and avoid contact with spray mist and drift. Avoid repeated or prolonged contact of herbicides with your skin. Keep herbicides out of your eyes, nose, and mouth. Consult the label for treatment recommendations.

If your hands become contaminated with a pesticide, do not eat or drink until you have washed.

In case a pesticide is swallowed or gets in the eyes, follow the first aid treatment given on the label and get prompt medical attention. If a pesticide is spilled on your skin or clothing, remove contaminated clothing immediately and wash skin thoroughly with soap and water.

To protect fish, wildlife, and livestock, do not clean spraying equipment or dump excess spray material in or near lakes, streams, or ponds.

Empty herbicide containers may be hazardous. Dispose of them in accordance with label instructions or recommendations of your State extension weed science specialist. Do not burn herbicide containers.

Poisonous Plants of the Western States

Arrowgrass

Distribution of Arrowgrass



Signs and Lesions of Arrowgrass Poisoning

- Distress
- Dyspnea; rapid breathing and gasping
- Cyanosis (blue coloring of the lining of the mouth)
- Increased salivation
- Muscular twitching, staggering, and convulsions
- Bloat may occur; rumen contents may smell like almonds
- Convulsions
- Coma
- Death (animals consuming large amounts of the plant may die in 1 to 60 minutes and show only convulsions and death as signs of poisoning)
- Blood and tissues a bright cherry red (hyperoxygenation)
- Tracheal and pulmonary congestion
- Cattle and sheep may be severely affected if they eat large amounts of arrowgrass leaves or stalks in a short time. The leaves contain substances that produce the poison hydrogen cyanide on ingestion.

Arrowgrass *(Triglochin maritima* and *T. palustris)* that is growing with adequate moisture does not cause poisoning. When growth is stressed or stunted from lack of moisture or frost, plants quickly become toxic. Regrowth foliage following harvest is also toxic. The toxin in arrowgrass is the same as that in chokecherry.

Where and When Arrowgrass Grows

Species of arrowgrass are widely distributed in marshy areas and wet meadows throughout the United States. Arrowgrass grows on wet, alkaline soils and may be found growing over large areas or small patches near springs. Arrowgrass starts growing in early spring. It is often found growing in native meadows that are cut for hay. Arrowgrass cut for hay may be toxic.

How Arrowgrass Affects Livestock

The amount of arrowgrass required to poison sheep or cattle depends on the amount of poison in the plants and the rate at which the plants are eaten. There is enough hydrogen cyanide, also known as

hydrocyanic acid or prussic acid, found in ¼ to 3 lb of stunted arrowgrass to kill a 600-lb animal. The toxic dose must be eaten at one time to cause death because the poison is not cumulative. Death results from respiratory failure.

How to Reduce Losses

Keep animals off areas where the growth of arrowgrass has been retarded by drought or frost or regrowth following harvest.

The action of hydrocyanic acid is so rapid that it is usually too late to treat an affected animal after the signs of poisoning are recognized.

Some poisoned animals may be saved by immediate treatment with an intraperitoneal injection of a mixture of 20 ml of a 10-percent solution of sodium thiosulfate and 10 ml of a 10-percent solution of sodium nitrite. For animals in advanced stages of poisoning, give an intravenous injection of the sodium thiosulfate and sodium nitrite solution. Consult your local veterinarian regarding treatment.

Arrowgrass can be controlled by metsulfuron (0.5 oz product/acre) when applied during seed stalk elongation.



Arrowgrass is a perennial that has fleshy, dark-green, half-rounded, grasslike leaves that grow from the base of the plant. Although clumps of leaves are only 6 to 18 inches tall, slender flower stalks may reach a height of 5 feet. Small, green flowers appear close together along the upper part of the stalk early in the season. Later, the flowers develop into golden-brown fruits.

Bitterweed

Distribution of Bitterweed



Signs and Lesions of Bitterweed Poisoning

- Loss of appetite and weight
- Depression
- Weakness and irregular gait
- Vomiting and coughing (lips and muzzle are usually stained green with vomitus)
- Wasting and eventual death
- Head may be elevated
- Bloat
- Congestion of liver, lungs, and abomasum
- Inflammation and hemorrhage of abomasum and duodenum and sometimes the rest of the gastrointestinal (GI) tract; arched back is indicative of abdominal pain
- Affected animals may lag behind the herd
- Lambs may move with a stiff gait

Bitterweed *(Hymenoxys odorata)* poisoning is a major problem of sheep in the Southwest; cattle are occasionally poisoned. Bitterweed is a member of the sunflower family and is closely related to Colorado rubberweed (pingue) both in appearance and in its effects



Bitterweed is a yellow-flowered annual weed varying in height from a few inches to 2 feet. It is erect and branches from the base. It is a bitter-tasting plant found mainly in the semiarid rangelands of the Southwest.

on sheep. Pingue is a perennial, whereas bitterweed is an annual. Bitterweed has an extremely bitter taste and is unpalatable to livestock; thus it is generally a problem only when there is a shortage of other green forage. Toxicity increases with plant maturity. The toxin is a sesquiterpene lactone.

Where and When Bitterweed Grows

Though bitterweed occurs from southern Kansas to Mexico and from central Texas to California, it is a problem of magnitude principally in Texas. The critical time for livestock is generally in winter and early spring when animals may graze the plant because of a shortage of other green forage. Maximum plant growth occurs in spring and early summer, but seedlings may be found at any time of the year, depending on moisture and temperature conditions.

How Bitterweed Affects Livestock

As with many other poisonous plants, the tolerance of animals to bitterweed depends on many factors. Acute poisonings may result in death if large amounts are eaten rapidly. Under normal conditions, this will not occur. Chronic poisoning is common, with sheep developing a wasting disease. Under natural grazing conditions, signs of poisoning occur 2 to 4 weeks after the sheep start grazing the plant. The animal will recover if removed from access to the plant at the first signs of poisoning. Generally speaking, about 1 percent of the animal's body weight of consumed plant can be lethal.

Bitterweed is a powerful irritant to the digestive tract; thus, poisoned animals may lose their appetite, the rumen may stop functioning, and animals may show signs of pain. They lose weight rapidly and may lag behind the flock. Animals may not die until some time after removal from the plant. Post-mortem examinations of animals show lung, heart, and kidney changes and irritation of the GI tract.

How to Reduce Losses

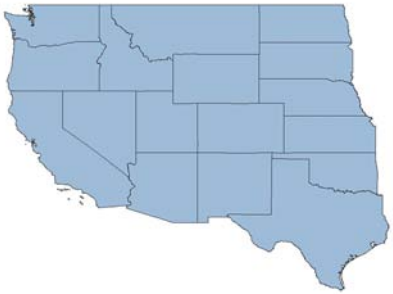
There is no treatment for bitterweed poisoning. Recent research has shown that sheep fed an activated charcoal/protein supplement were less affected by bitterweed than unsupplemented sheep. Animals not severely poisoned may be taken from infested pastures, and they may recover within a few days if given good feed and water. Ranchers with bitterweed-infested pastures should be especially aware of what their sheep are eating, particularly in winter and spring months, and should observe the flock frequently for early signs of poisoning.

Bitterweed poisoning has been prevented by moderate stocking in a four-pasture deferred rotation grazing system or light continuous stocking with sheep, cattle, and goats.

Seasonal control can be obtained from 2,4-D (1 lb ae/acre), picloram (0.5 lb ai/acre), clopyralid (0.25 lb ai/acre), or metsulfuron (1.25 oz product/acre) applied from autumn to early spring before flowering when the plant is actively growing.

Bracken Fern (Western Bracken)

Distribution of Bracken Fern



Signs and Lesions of Bracken Fern Poisoning

In cattle and sheep:

- High fever
- Loss of appetite
- Depression
- Difficulty in breathing
- Excessive salivation
- Nasal and rectal bleeding; bloody urine and feces
- Anemia, leukopenia, thrombocytopenia, and hemorrhagic syndrome
- Hemorrhages on mucous membranes
- Aplastic bone marrow
- Bladder tumors in cattle

In horses:

- Loss of weight and condition; emaciation
- Progressive incoordination
- Marked depression
- Crouching stance, back arched with legs apart
- Twitching muscles
- General body weakness
- Weak, fast pulse
- Inability to stand
- Convulsions or spasms
- Pericardial and epicardial hemorrhage

Bracken fern *(Pteridium aquilinum* var. *pubescens)* is poisonous to cattle, sheep, and horses; sheep, however, are more resistant. Bracken contains a thiaminase inhibitor that leads to the development of thiamine deficiency in horses that can be remedied by giving thiamine. Research has indicated that bracken fern is also carcinogenic. Milk from cows that graze bracken fern may be hazardous to humans.



Bracken fern has broad, triangular leaves, or fronds. The plant reaches a height of 2 to 4 feet. It grows directly from stout, black, horizontal root stalks.

Livestock losses have been high in the Pacific Coast States, as well as in the Eastern and Midwestern States and some areas of the Intermountain West.

All portions of the fern—both green and in harvested hay—are poisonous to livestock. The toxin is ptaquiloside, a nor-sesquiterpene glucoside.

Where and When Bracken Fern Grows

Bracken fern is widely distributed in many places around the world. Bracken fern grows on burned-over areas, in woodlands and other shaded places, and on hillsides, open pastures, and ranges in sandy or gravelly soils.

The plant starts growth in the early spring and usually remains green until the leaves are killed by frost.

How Bracken Fern Affects Livestock

Bracken fern produces different signs of poisoning in cattle and sheep than it does in horses. Cattle poisoning often occurs during late summer when other feed is scarce, or when animals are fed hay containing bracken fern. The disease occurs after cows have consumed large amounts of the plant and is manifested in an acute, usually fatal, form. The disease is more chronic in horses.

The poison in bracken fern has a cumulative effect. Livestock are affected only after they have eaten considerable amounts of bracken fern for 2 to 4 weeks.

Cattle are affected by a nor-sesquiterpene glycoside called ptaquiloside, which causes bleeding and damage to the bone marrow. The disease has a delayed onset: Cattle may graze the plant for several weeks and then get sick and die. Poisoned animals seldom recover. If consumed over time, ptaquiloside can also cause cancer in the urinary bladder and GI tract. These tumors often bleed, causing red urine (enzootic hematuria or redwater disease).

Bracken fern poisoning in horses can occur when they are fed hay containing about 20 percent bracken fern over a period of 30 days. Signs of poisoning include weight loss, incoordination, and lethargy. Horses may stand with their legs apart as though bracing themselves and may assume a crouching position with an arched back. Muscle tremors develop and the animal is unable to stand despite violent attempts to do so. Death will occur in several days to a week.

How to Reduce Losses

Animals seldom eat bracken fern if sufficient forage is available, so grazing should be delayed until adequate forage is available. Young

shoots are the most toxic and are relatively palatable in early growth stages.

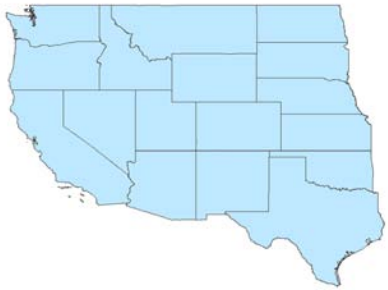
To eliminate livestock losses, do not overgraze pastures and ranges. Make sure sufficient forage is available at all times to animals in infested areas. If necessary, supplement forage near the end of the grazing period. Do not feed hay contaminated with bracken fern.

Poisoning can be treated with thiamine hydrochloride, saline cathartics, and possibly activated charcoal. Few cattle have recovered after signs of acute poisoning appear; however, horses in early stages of poisoning may be saved by intravenous injections of thiamine hydrochloride. The thiamine hydrochloride treatment should be given under the direction of your local veterinarian.

Bracken fern can be controlled. In areas where cultivation is practical, the plants can be destroyed by cultivating the soil for 2 to 3 years.

Chokecherry

Distribution of Chokecherry



Signs and Lesions of Chokecherry Poisoning

- Death (animals consuming large amounts of this plant may die in 1 to 60 minutes and show only convulsions and death as signs of poisoning)
- Distress, rapid breathing, and gasping
- Cyanosis (blue coloring of lining of the mouth and mucosal membranes)
- Salivation
- Excitement may occur
- Muscular twitching, staggering, and convulsions
- Bloat may occur; rumen contents may have odor of almonds
- Coma
- Blood and tissue are bright cherry red
- Tracheal and pulmonary congestion

Western chokecherry *(Prunus virginiana* var. *demissa)* and black chokecherry *(P. virginiana* var. *melanocarpa)* cause livestock poisoning when drought and overgrazing strip the pastures and ranges of grass and other forage. Animals become poisoned if they eat considerable quantities of the leaves in a short time. Wilting, frost, or other damage to the plant enhances toxicity.

Both sheep and cattle may be poisoned by chokecherry. Although most losses occur when feed is scarce, losses are not uncommon when sheep graze chokecherry in the spring. Cattle sometimes are poisoned by eating leaves on branches trimmed from cultivated chokecherry trees.

The toxic substance in chokecherry, hydrogen cyanide (hydrocyanic acid), is found principally in the leaves. Leaves become less toxic as the growing season advances.

Chokecherry and arrowgrass cause the same type of poisoning because they both contain hydrocyanic acid. Other plants with cyanogenic potential include serviceberry, Sudan grass, Johnson grass, white clover, birdsfoot trefoil, mountain mahogany, and reed canary grass.

Where and When Chokecherry Grows

Chokecherry grows in damp and fertile soil. It is found in thickets on hillsides and canyon slopes. It appears as a shrub or small tree among willows, poplars, and alders that grow along mountain streams.

Chokecherry begins growing early in spring. Its growth is slow at high elevations.



Chokecherry trees (center) may reach a height of 20 feet. They are often found growing with other trees and bushes. The berries are not considered to be toxic. Chokecherry may grow as a shrub to about 6-8 feet in height. It is found in thickets, along hillsides, and on canyon slopes.

How Chokecherry Affects Livestock

Although the hydrocyanic acid content of chokecherry leaves varies, ingestion of about 0.25 percent of an animal's weight in leaves can be fatal. Wilted leaves, as well as fresh leaves, are poisonous. Poisoning occurs when an animal consumes a relatively large amount over a short period of time. For chokecherry to be fatal, an animal must eat a toxic dose in a relatively short period—30 minutes to an hour. Hydrocyanic acid inhibits cellular respiration in all body organs and causes respiratory failure. Signs of poisoning come on rapidly, and death may follow within a few minutes.

How to Reduce Losses

Keep hungry or thirsty animals off areas where chokecherry is abundant.

The action of hydrocyanic acid is so rapid that it is usually too late to treat an affected animal after the signs are recognized.



Chokecherry is a perennial that bears masses of white flowers in long clusters in the spring. Small ripe cherries range in color from purple to black. Leaves are dark green and glossy.

Some poisoned animals may be saved by immediate treatment. For both cattle and sheep, use 20 cc of a 10-percent solution of sodium thiosulfate mixed with 10 cc of a 10-percent solution of sodium nitrite given intraperitoneally or intravenously. Do not let animals become overexcited while they are being treated. For advice about treatment, consult your local veterinarian.

Control of chokecherry is not practical on a large scale.

Colorado Rubberweed (Pingue)

Distribution of Colorado Rubberweed



Signs and Lesions of Colorado Rubberweed Poisoning

- Salivation
- Nausea and vomiting (area about mouth may be stained green from vomitus)
- Loss of appetite, rumen stasis, abdominal pain
- Weakness and depression
- Irregular gait and trembling; gait will become stiff
- Emaciation and death may occur if animals are allowed to graze this plant too long; death may not occur for some time after poisoning
- Congested liver, lungs, and GI tract
- Liver pale and fatty
- Enlarged spleen
- Hyperemia and hemorrhage of upper GI tract

Colorado rubberweed (pingue; *Hymenoxys richardsoni* var. *floribunda*) may cause serious losses principally in sheep herds in Colorado, Utah, and northern and western New Mexico. Poisoning is common in sheep, but occurs occasionally in cattle. The plant is poisonous to livestock throughout the growing season. Losses are heaviest when hungry animals are trailed through Colorado rubberweed or are placed on overgrazed ranges with heavy infestations of Colorado rubberweed.

All aboveground parts of the plant contain the poisonous substance, a sesquiterpene lactone. Animals grazing the plant may have a gradual buildup of the toxin to a level that is harmful or lethal, or they may eat large amounts of Colorado rubberweed and be poisoned immediately.

Where and When Colorado Rubberweed Grows

Colorado rubberweed is a perennial that grows in arid sites at elevations of 6,000 to 8,000 feet. It is found mostly on mountains and foothills and grows from early spring until the first frost. It readily increases and may replace more palatable forage plants when ranges have been overgrazed.

How Colorado Rubberweed Affects Livestock

Most poisoning occurs in spring or fall. The plant seems to act primarily as a depressant of the digestive tract. In most cases, the first sign of poisoning to be observed is a green froth around the mouth and nose. A 100-lb sheep may die if it eats ¼ to ½ lb of Colorado rubberweed daily for 1 to 2 weeks, or it may die from one large feeding.

How to Reduce Losses

Animals seldom eat toxic amounts of Colorado rubberweed if desirable forage is available.

Heavy losses during trailing may be prevented by avoiding heavily infested areas. Keep hungry animals away from Colorado rubberweed ranges at all times. If sheep losses become excessive, it may be advisable to change from sheep to cattle on certain ranges.

When grazing sheep on rubberweed-infested ranges, they should be watched closely for signs of poisoning. The rubberweed should be observed for signs of being grazed. At the first sign of poisoning (stiffness or a green stain around the mouth) or grazing, the sheep should be moved to a rubberweed-free part of the range. Lambs poisoned on rubberweed perform poorly and it may not be economical to keep them.



Colorado rubberweed may reach a height of about 1 foot. The stalk is thick and woody. Stem bases are covered with a woolly growth. Colorado rubberweed belongs to the sunflower family.



There is no known treatment for Colorado rubberweed poisoning.

Colorado rubberweed causes a wasting disease similar to that seen from bitterweed and sneezeweed poisoning. Sheep are more likely to eat it than cattle, and are probably more sensitive to it.

If Colorado rubberweed invades a range, examine your range management program carefully. On a range that is overgrazed, the desirable forage plants are gradually crowded out and Colorado rubberweed begins to spread.

To control Colorado rubberweed, spray plants in the budding stages or during active growth with 2,4-D ester at 2 lb ae/acre.

Copperweed

Distribution of Copperweed



Signs and Lesions of Copperweed Poisoning

- Loss of appetite
- Dullness
- Weakness
- Coma and death in 1 to several days after ingestion of a lethal dose
- Small hemorrhages on serosal surfaces
- Mild gastroenteritis

Cattle and sheep may be poisoned from grazing copperweed *(Oxytenia acerosa).* They usually graze this plant only when other feed is scarce. Cattle are more likely to eat the plant in the fall when they are being trailed from summer range. Sheep are occasionally poisoned in fall and winter by eating dry leaves that have fallen to the ground. Most losses occur in cattle.

Copperweed contains a toxin that is dangerous at all times. The toxin is not known but is probably a sesquiterpene lactone. Toxicity reaches a peak when the plant matures. Leaves and stems are equally toxic.

Where and When Copperweed Grows

Copperweed grows along streambeds or gullies where moisture is plentiful and the soils are usually alkaline.

Copperweed starts growing early in spring. Flowers appear during July and August.



Copperweed is a perennial that may reach a height of 4 feet. A large number of stems grow from the crown; each has many narrow leaves. Small, flowering heads turn orange to yellow when mature.

How Copperweed Affects Livestock

Cattle are more susceptible to copperweed poisoning than sheep. Three lb of copperweed or an amount equal to 0.5 percent of the animal's weight may cause death.

Poisoned cattle die in 24 to 28 hours. Poisoned sheep may linger 1 to 3 weeks before dying.

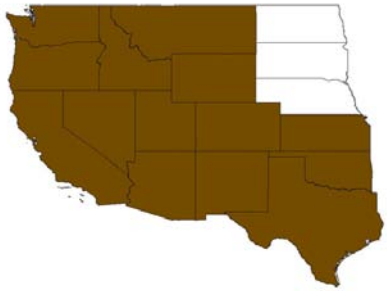
How to Reduce Losses

Animals seldom eat a toxic amount of copperweed if other forage is available. To reduce losses, provide adequate forage at all times. Supplemental feeding is beneficial before animals are driven through heavily infested copperweed ranges.

There is no known treatment for copperweed poisoning.

Death Camas

Distribution of Death Camas



Signs and Lesions of Death Camas Poisoning

- Salivation and bloody frothing
- Nausea and vomiting
- Muscular weakness and staggering
- Pulse fast and weak
- Prostration, labored breathing, gasping
- Coma
- Death due to heart failure
- Death within a few hours to a few days
- Congestion of lungs and kidneys
- Minimal necrosis of skeletal and cardiac muscle

Death camas (*Zigadenus* spp.) is the common name of several species of plants that are poisonous to livestock. The more toxic of these species are grassy death camas *(Z. gramineus),* meadow death camas *(Z. venenosus),* foothill death camas *(Z. paniculatus),* and Nuttall's death camas *(Z. nuttallii).* They are found principally in the western range States.

Death camas is one of the first plants to begin growth in early spring. Without sufficient other forage, death camas may be heavily grazed and will cause severe losses. Spring snow storms may cover all forage except death



Foothill death camas growing on a grassy hillside, a typical habitat where its density often reaches levels that are hazardous to grazing animals.





Death camas is a perennial that produces grasslike leaves in groups of three from a deeply buried bulb. The plant may be 4 to 16 inches tall. Yellowish-white flowers grow in clusters atop the stalk. Left, meadow death camas; right, foothill death camas.

camas, which may protrude through the snow and is available to the livestock. Sheep are most likely to be affected by feeding on death camas. Occasionally, cattle and horses are poisoned.

Death camas contains toxic steroidal alkaloids that occur throughout the plant; plants are dangerous at all times.

The bulb may be mistaken for those of the edible camas or quamash (*Cammassia* spp.) and can cause severe illness in humans. If bulbs are eaten, take the affected person to the emergency room of the nearest hospital immediately.

Where and When Death Camas Grows

Some species of death camas thrive on sandy soils; others grow on drier, rocky foothills. The more toxic species are seldom found above elevations of 8,000 feet. Death camas grows early in spring, matures, and enters dormancy during early summer when soil moisture declines.

The leaves appear very early in the spring. In the foothills, death camas generally flowers in April and May. At higher elevations, the plant may flower in late June and July.

How Death Camas Affects Livestock

Death camas causes marked disturbance in respiration and heart action. A 100-lb sheep may die if it eats ½ to 2 lb of green foliage. The amount of foliage that will cause an animal's death depends on the species of plant eaten and the rate of consumption. Severely poisoned animals usually die; those less seriously affected may recover.

How to Reduce Losses

To avoid poisoning, delay turnout until adequate good forage is available. Do not introduce hungry sheep into heavy stands of death camas. Avoid feeding, bedding, or trailing sheep through heavy stands of death camas.

There is no known treatment for death camas poisoning.

Research results show that early in the season, when plants have three to six leaves, death camas can be controlled by spraying with 2,4-D at the rate of 1½ to 3 lb ae/acre. After the flowering stalks appear, spraying is not effective.

False Hellebore (Veratrum)

Distribution of False Hellebore



Signs of False Hellebore Poisoning

- Excessive salivation with frothing
- General body weakness; animal may be unable to stand
- Irregular gait
- Vomiting
- Fast, irregular heartbeat
- Slow, shallow breathing
- Coma
- Convulsions
- Birth defects

False hellebore *(Veratrum californicum),* or veratrum (sometimes called wild corn and cow cabbage), is a range plant that causes severe poisoning in sheep. It also affects cattle and goats. If ewes, nanny goats, and cows eat it during early pregnancy, false hellebore causes a cyclopian-type (single eye) facial deformity in the offspring.

Sheep and goats readily eat the whole plant. Cattle may eat it if other forage is scarce.

False hellebore is poisonous from the time it starts to grow until after it is killed by freezing, but toxicity decreases as plants mature. Roots are 5 to 10 times as poisonous as leaves or stems. The toxins in false hellebore are cyclopamine and other steroidal alkaloids.

Where and When False Hellebore Grows

False hellebore grows on moist, open meadows and hillsides at elevations of 6,000 to 11,000 feet. It emerges as soon as snow melts in the spring. Flowers appear in July and August, and the plant produces seeds in late August and September.

How False Hellebore Affects Livestock

False hellebore poisoning may occur 2 to 3 hours after an animal eats the plant. Sheep may show slight or marked signs of poisoning after eating 6 to 12 ounces of green stems or leaves.

If pregnant ewes eat false hellebore on the 14th day after breeding, the young may have congenital deformities of the head. These offspring, commonly called monkey-faced lambs, may have a protruding



Leaves of false hellebore may measure 9 to 12 inches long and 3 to 6 inches broad. Cream-colored flowers grow in clusters at the top of a single unbranched stalk in a way that resembles corn. Seed pods turn black as they ripen.

lower jaw, underdeveloped upper jaw, proboscis-like nose, cyclopia, hydrocephaly, and a variety of deformities of the eyes as well as related deformities. There is a high incidence of embryonic loss if the plant is ingested between the 15th and 19th day of gestation. When ewes eat the plant later in gestation (28th to 33th days), they give birth to lambs having tracheal stenosis or shortened limbs.

Ewes carrying deformed fetuses fail to lamb at the end of the normal gestation period. As the ewe approaches term, the udder will start to fill with milk and the external genitalia will swell. At term, all indications of parturition will disappear and the fetus continues to grow to an abnormal size and will eventually kill the ewe unless the lamb is removed by caesarian. Gestation has extended as long as 240 days with the lamb weighing over 30 lb when removed from the ewe (normal birth weight is 8 to 9 lb).

How to Reduce Losses

Losses of newborn animals from deformities can be avoided by keeping sheep, goats, and cattle away from false hellebore during early gestation. Ideally, a flock of sheep should not be grazed on veratrum for 60 days after the rams are turned in with the ewes.

Research results show that false hellebore may be controlled by applying amine salts of 2,4-D at



Typical cyclopic lamb, caused when the pregnant ewe grazed false hellebore on the 14th day of gestation.

the rate of 2 lb ae/acre after the last leaves have expanded and before bud stage. A second treatment may be required the following year. Picloram pellets at 1 lb ae/acre may also provide long-term control.

Greasewood

Distribution of Greasewood



Signs and Lesions of Greasewood Poisoning

- Depression
- Dullness, weakness, reluctance to move
- Breathing rapid and shallow
- Drooling
- Recumbency
- Coma
- Death between 2 hours and several days
- Uremia, hypocalcemia, death due to interference with energy metabolism
- Renal tubular necrosis with crystals
- Crystalluria

Greasewood *(Sarcobatus vermiculatus)* is a range shrub that livestock can safely eat in moderate amounts with other forage. It is quite palatable to livestock. Death occurs when livestock eat large amounts in a short period of time. Poisoning occurs in sheep and cattle.

Losses may occur when sheep or cattle eat large quantities of leaves that have fallen to the ground in fall and winter or new growth in spring.

The toxic substances, soluble sodium and potassium oxalates (also found in halogeton), accumulate in the leaves of the plant. The amount of toxin varies considerably among the different areas where it grows. Greasewood increases in toxicity as the growing season advances.

Where and When Greasewood Grows

This shrub is adapted to the heavy saline soils of arid and semiarid regions. It is confined to alkaline soils and may form the dominant vegetation on alkaline flats.

Greasewood starts growing in early spring. The leaves remain succulent until fall, when they dry and drop off. Buds remain on plants most of the year.

How Greasewood Affects Livestock

Signs of poisoning may develop 4 to 6 hours after an animal eats a toxic amount of greasewood. Greasewood poisoning in livestock is similar to that caused by halogeton.

A sheep may die if it eats as little as 2 lb of green leaves and fine stems in a short period. Cattle may die after eating 3 to 3 ½ lb of greasewood in a short period. Most poisonings occur when hungry animals are turned into a patch of greasewood.

How to Reduce Losses

Rumen bacteria can metabolize the toxin to a nontoxic form. Therefore, introduce livestock into heavy stands of greasewood slowly so as to allow time for them to adapt to the toxic substance in the plant (2 to 3 days). Livestock can also be conditioned to this toxin by grazing them on shadscale, which contains nontoxic levels of oxalate. Make sure animals are not hungry when first allowed to graze greasewood. Supply adequate water and a good variety of forage.

There is no known treatment for greasewood poisoning.

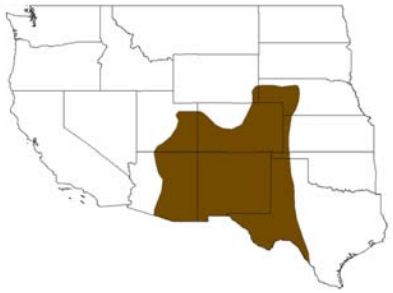
Research shows that greasewood can be effectively controlled with 2,4-D (2 lb ae/acre). Two or more successive years of treatment may be necessary for effective control because of its resprouting ability.



Greasewood is an erect, spiny, woody perennial shrub that grows 2 to 5 feet tall. Flowers are small and light green to whitish.

Groundsel (Threadleaf and Riddell) and Houndstongue

Distribution of Groundsels



Signs and Lesions of Groundsel and Houndstongue Poisoning

- Signs may not appear until 6 months or more after the plant is eaten
- Lethargy; loss of interest in food and abdominal pain
- Crustiness around eyes and nose; eyes may also be red and watery, especially in bright sunlight
- There may be diarrhea or constipation
- Weakness as manifested by wobbling and dragging rear feet
- Animals may wander aimlessly and appear blind, and may become belligerent
- Cattle may develop an unpleasant odor with a sweetish quality
- Ascites
- Death may occur within a few days after symptoms appear
- Icterus and hyperbilirubinemia
- Liver cirrhosis, fibrosis, and bile duct proliferation
- Photosensitization

Threadleaf groundsel *(Senecio longilobus),* often called woolly groundsel, and Riddell groundsel *(S. riddellii)* contain pyrrolizidine alkaloids similar to those in tansy ragwort and thus may poison cattle, horses, and sheep. These groundsels occur in greatest abundance in the Southwest, notably in Texas, New Mexico, and Arizona.

Riddell groundsel may also poison livestock in the Midwest. Both plants have similar leaves that are thickish, elongated, and narrow. Threadleaf groundsel leaves are gray, hence the name "woolly groundsel," whereas Riddell groundsel leaves are bright green. Riddell groundsel has stems that are somewhat less woody than those of threadleaf groundsel, which also tends to be more bush-like.

Houndstongue *(Cynoglossum officinale)* is a biannual forb that infests pastures, ranges, and hayfields throughout the Western United States. It is sometimes harvested in hay where it can cause poisoning. Houndstongue also contains pyrrolizidine alkaloids (see

tansy ragwort). There are a number of other plants that contain toxic amounts of pyrrolizidine alkaloids such as *Amsinckia* and *Crotalaria.* In some areas of the world, these plants grow mixed in with small grains and subsequently end up in the food of people, with toxic consequences including carcinogenicity. There is some evidence that pyrrolizidine alkaloids can pass through the placental membranes and have an effect on the fetus.



Threadleaf groundsel, often called woolly groundsel because of its gray color, has many stems. Each stem may have several branches.

Toxicity increases up until time of flowering; then it begins to decrease. Younger plants tend to be more palatable to livestock. Sheep and goats are more resistant to poisoning than cattle and horses. Younger animals are more susceptible than older animals.

Where and When Groundsels and Houndstongue Grow

Threadleaf groundsel typically grows in dry, gravelly, or hardpan soils and may be found on plains and foothill areas; Riddell groundsel is generally found in slightly moister sites, such as drainages and in sandy places. Houndstongue can be found growing in croplands and in the better soils.

Both groundsels flower in late summer, although in the Southwest threadleaf groundsel may grow and flower any time temperature and moisture conditions are right. Riddell groundsel usually dies back to the crown after flowering and follows a more definite growth cycle.

How Groundsels and Houndstongue Affect Livestock

These groundsels and houndstongue contain pyrrolizidine alkaloids, which primarily affect the liver. Cells of the liver are slowly killed or prevented from reproducing and are gradually replaced by scar tissue. Whether an animal survives depends on the number of liver cells it loses, and the duration of survival depends upon the rate at which it loses liver cells.





Houndstongue is a biennial. The first year it produces a rosette with large rough leaves resembling a dog's tongue. The second year it produces a flowering stalk with reddish-purple flowers that produce four prickly nutlets.

Animals may survive for 6 months or longer after they have ingested a lethal amount of plant and may show no outward symptoms during this period. They may suddenly become lethargic, fill with fluid in the abdominal cavity, and die within 2 to 4 days. They may also show central nervous system effects such as impaired vision, belligerency, and an incessant stumbling walk.

The onset of symptoms may be dependent on other stresses imposed on the liver, such as cold weather, poor feed, shipping, or parturition, which it cannot handle in its weakened condition; thus, the true cause of death is often not ascertained.

Cattle eating 5 percent or more of their total daily diet of mature plant for periods exceeding 15 consecutive days can be expected to die within 1 to 6 months. A lesser amount of young plant may cause the same symptoms. Intoxications are often subchronic or chronic with clinical signs developing only after long periods of time. Such animals perform poorly throughout their lives and are culled because they are inefficient producers. Young animals are more easily poisoned than mature animals.

How to Reduce Losses

When symptoms of poisoning appear, it is too late to save the animal; thus, the best prevention is avoidance of the plant.

Animals may graze pastures containing these plants without ill effects if an ample amount of good forage is present; thus, pastures or ranges should be maintained for good production of grasses and high quality forage.



Riddell groundsel is a bright green plant which has several stems that originate from a woody base. Stems, if they branch, only branch terminally.

Groundsels may be controlled with 2,4-D (1 lb ae/acre), picloram (1 lb ae/acre), or a combination of 2,4-D and picloram (0.75 + 0.25 lb ae/acre) when the plants are growing rapidly, but plants are generally spread over large areas and herbicide treatment may not be economically feasible.

Houndstongue can be controlled with 2,4-D (2 lb ae/acre) or picloram and 2,4-D (0.25 to 0.5 lb ae/acre). Herbicide should be applied in spring prior to bloom.

Halogeton

Distribution of Halogeton



Signs and Lesions of Halogeton Poisoning

- Depression, weakness, and reluctance to move
- Respiration rapid and shallow
- Drooling
- Recumbency
- Coma
- Death between 2 hours and several days; death due to interference with energy metabolism
- Hypocalcemia, uremia
- Renal tubular necrosis and crystals, hemorrhage, and hyperemia
- Rumen wall may be hemorrhagic and edematous

Halogeton *(Halogeton glomeratus),* an alien invasive annual plant of the Western States, frequently causes poisoning in sheep and cattle. Most losses occur when hungry animals are allowed to graze in heavy stands of halogeton.

The toxic substance in halogeton is sodium oxalate. Halogeton becomes more toxic as the growing season advances, reaching a peak of toxicity at maturity. Cattle and sheep readily graze dried halogeton; thus, losses occur during fall, winter, and early spring. Losses generally occur when these livestock become hungry—during trailing, after trucking, or overgrazing.

Where and When Halogeton Grows

Halogeton often grows along railroad beds, roads, sheep trails, and in places where the soil has been disturbed. Dense stands are found on burned-over areas, overgrazed ranges, dry lakebeds, and abandoned dry farms. It thrives in the saline soils of colder arid and semiarid regions of the Great Basin, especially where native plant cover is thin. However, halogeton lacks the capacity to compete with vigorous perennial plants and the more aggressive annuals.

Halogeton is a prolific seed producer. New plants germinate from February to mid-August and produce a seed crop before the growing season ends. Seeds are spread by wind, water, animals, and vehicles and equipment such as road graders. Seed may remain alive in soil 20 years or longer.



Halogeton grows 3 to 18 inches tall; its height depends on the moisture available during the growing season. Each plant generally has five main stems that come directly from the base of the plant.



Halogeton has a characteristic small hair—about one-twelfth inch long—on the end of each leaf.



Halogeton forms a solid line along the roadside and extends into the sagebrush.

How Halogeton Affects Livestock

Toxicity is due primarily to sodium oxalate, which can accumulate to high levels in the plant. Oxalates precipitate calcium from the blood causing hypocalcemia; oxalate crystals damage the kidneys and rumen walls, and enzymes are inhibited in the citric acid cycle of energy metabolism, causing rapid death.

Sheep can tolerate large amounts of halogeton if they eat it slowly or with other forage. The tolerance of sheep doubles if they are preconditioned by grazing light stands or other plants containing low levels of oxalates.

About 12 ounces of halogeton will kill a sheep that has been without feed for a day or longer; 18 ounces are required to kill a sheep that has been feeding on other forage. First signs of halogeton poisoning occur 2 to 6 hours after an animal eats a fatal amount; death usually occurs in 9 to 11 hours.

How to Reduce Losses

Livestock losses may be reduced by maintaining range in good condition and by proper management of animals. Supplemental feeding helps prevent halogeton poisoning when animals trail through or graze infested areas.

Do not introduce livestock into areas heavily infested with halogeton unless it can be done slowly to allow time for adaptation to the toxin. This can be accomplished by grazing plants such as shadscale or light stands of halogeton.

Livestock should not be allowed to become hungry or thirsty while grazing in areas infested with halogeton as they graze indiscriminately. Death in livestock occurs when an animal eats a large amount of halogeton in a short period of time. Animals being trucked in should not be unloaded in halogeton-infested areas. These animals may benefit from supplemental feeding before grazing in the halogeton-infested areas. There is no known treatment for halogeton poisoning.

Halogeton does not compete well with other vegetation; therefore the best way to control halogeton is proper range management. Because each plant produces vast numbers of seeds, some of which may survive for 20 years or more in soil, it is not practical to eradicate any population that has been in existence for 2 years or more. Plants can be held in control by proper use of herbicides, and very small infestations can be eradicated if treated early. Research results indicate that few herbicides are effective for halogeton control. The low volatile ester of 2,4-D (2 lb ae/acre) is effective when applied in late May or early June. Applications of 2 lb ai/acre of tebuthiuron as late as August will kill the halogeton and prevent reinvasion for 3 to 5 years. However, tebuthiuron is a sterilant and also kills the broadleaf forbs and shrubs, resulting in further (and potentially stronger) invasion by halogeton (from seed in the soil) or other pioneer invaders, such as Russian thistle and rabbitbrush.

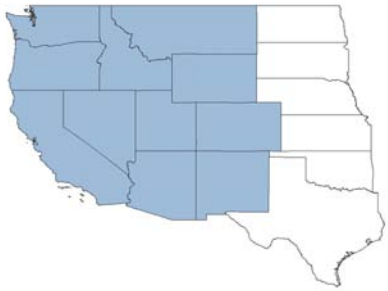
Caution: Use the herbicide only to treat small infestations of halogeton. Repeated treatments are necessary for control.



Some of the 1,200 sheep that died from halogeton poisoning in a case from west central Utah.

Hemp Dogbane

Distribution of Hemp Dogbane



Signs and Lesions of Hemp Dogbane Poisoning

- Rapid pulse
- Dilation of pupils
- Vomiting
- Blue coloration of mucous membranes
- Progressive body weakness
- Convulsions may occur
- Coma
- Death
- Mild myocardial degeneration

Hemp dogbane *(Apocynum cannabinum),* a poisonous plant found throughout the United States, may cause occasional livestock losses in the western range States. The plant gets its name from the fact that American Indians used fiber from the bark for making rope. It also is called dogbane, Indian physic, American hemp, and rheumatism weed.

Normally, animals avoid hemp dogbane because of its bitter, sticky, milk-white juice. Sheep are more frequently affected than other animals; they will eat large quantities of hemp dogbane leaves and tops if other forb-type plants are not available. Hungry animals may eat hemp dogbane when they are turned onto harvested fields or onto new range in the fall or when other forage is scarce. Poisoning may also occur when sheep, cattle, and horses are trailed from summer to winter ranges.

Leaves are poisonous at all times, even when they are dry. The chief toxic substance in hemp dogbane is thought to be cymarin, a glycoside once used as a heart stimulant for humans.

Where and When Hemp Dogbane Grows

Hemp dogbane grows on plains and foothills at elevations up to 7,000 feet. It commonly is found in gravelly or sandy fields, in meadows, and along creekbeds, irrigation ditches, and fence lines in cultivated pastures. The plant begins growing in late spring or early summer.

How Hemp Dogbane Affects Livestock

Death from hemp dogbane poisoning may occur 6 to 12 hours after



Hemp dogbane is a perennial that grows 2 to 5 feet tall. Leaves turn yellow in the fall.



Leaves of hemp dogbane grow on opposite sides along reddish stems that contain a milky sap. The smooth-edged leaves are hairy on the lower surface. Flowers are greenish white. Two long pods, which develop from each flower, contain numerous seeds with tufts of silky white hairs at their ends.

animals eat the plant. A lethal dose for sheep is about ½ to 1 ounce per 100 lb of body weight. A lethal dose for cattle and horses is about ½ to ¾ ounce per 100 lb of body weight. A toxic dose depends largely on rate of consumption. Poisoned animals are usually found dead; post-mortem pathology shows heart lesions.

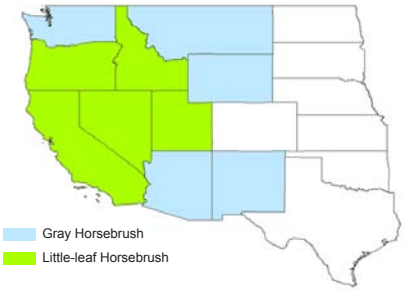
How to Reduce Losses

There is no known treatment for hemp dogbane poisoning.

Research results show that hemp dogbane may be controlled by repeated treatment of 2,4-D at 1 to 4 lb ae/acre.

Horsebrush

Distribution of Horsebrush



Signs and Lesions of Horsebrush Poisoning

- Loss of appetite
- Depression
- Lagging behind the herd
- Weakness, recumbency; death may occur in severe cases, or photosensitization may develop
- Itching and uneasiness may develop with the head held in a "looking up" position
- Swelling of the ears, lips, and face
- Animal seeks shade
- Peeling of skin from face and ears
- Abortion may occur
- Liver is swollen, engorged, and often shows severe fatty changes
- Kidneys show low grade nephritis
- Petechial hemorrhages can be found in subcutis and serosal surfaces

Two species of horsebrush that grow in the Great Basin region of the West are poisonous to sheep. Littleleaf horsebrush *(Tetradymia glabrata)* is commonly called coal-oil brush or spiny rabbitbrush. Spineless horsebrush *(T. canescens* var. *inermis)* is also called gray horsebrush.

Sheep that feed on horsebrush just following or in conjunction with black sage *(Artemesia nova)* and then are exposed to bright sunlight may develop a characteristic swelling of the head (photosensitization) called bighead. Sheep grazing horsebrush may die without developing bighead; the liver is extremely friable. However, with care, many of these sheep survive.

Most losses from horsebrush occur during stormy periods when sheep change their grazing habits as they are trailed through heavily infested areas. Hungry sheep may also eat toxic amounts of horsebrush after they are watered. The plant is especially dangerous during the bud stage, perhaps because it is more palatable.

All plant parts of both species are poisonous, but sheep eat only buds, leaves, and fine stems.



Spineless horsebrush is also called gray horsebrush. Photo courtesy U.S. National Park Service.

Where and When Horsebrush Grows

Littleleaf horsebrush is most abundant on benchlands, well-drained slopes, and low elevations on sheep winter ranges. It is one of the earliest desert range plants to start growing in spring; it may be green by late March and in full flower by the end of June. Leaves dry and drop off in early July, and the plant is dormant until the next spring.

Spineless horsebrush is most abundant in sagebrush areas and foothill regions. It starts growing later than littleleaf horsebrush and flowers in June or July. The plant usually remains green until fall.

How Horsebrush Affects Livestock

Horsebrush causes severe liver damage when large amounts are eaten, and sheep may die within a day or two. Survivors, if pregnant, often abort and may require weeks to recover. Sheep eating lesser amounts of the plant may become sensitive to sunlight and develop various degrees of photosensitivity. Severe photosensitization results in bighead wherein the sheep's head is swollen. Sheep vary considerably in their susceptibility to horsebrush, but often ½ to ¾ lb of littleleaf horsebrush will cause bighead, and larger amounts may result in death. Nearly twice that amount of spineless horsebrush must be eaten to cause similar symptoms.



Severe photosensitization (bighead) on sheep that grazed horsebrush. As little as one-half pound of littleleaf horsebrush can cause bighead.

Bighead develops only if the sheep has recently eaten black sagebrush. This plant enhances the action of horsebrush on sheep.

How to Reduce Losses

Bighead is principally a trail disease. To prevent losses, select trail routes devoid of horsebrush and do not let animals graze in infested areas. Sheep are more apt to graze horsebrush during stormy periods.

Move affected sheep to shade as soon as signs of bighead appear. Give them water and supplemental hay. If possible, these sheep should be left undisturbed for a few days.

An effective economical method for the control of horsebrush has not been developed. Chemical and mechanical control in the area where littleleaf horsebrush grows is economically and physically impractical and ecologically unsound. Littleleaf horsebrush grows in areas where it would be difficult and impractical to replace it with better forage. It is most practical to avoid horsebrush populations where possible.

Kochia

Signs and Lesions of Kochia Poisoning

- Depression
- Dehydration
- Weight loss
- Muscular weakness
- Photosensitization
- Ocular discharge
- Crusty muzzle
- Blindness
- Chronic nephrosis
- Degenerative hepatopathy
- Elevated GOT (glutamic-oxaloacetic transaminase), GGT (gamma-glutamyl transpeptidase), and serum bilirubin
- Brain edema
- Fatty, cirrhotic, enlarged livers
- Gastrointestinal inflammation
- Icterus
- Ataxia
- Incoordination
- Polioencephalomalacia may occur

Kochia *(Kochia scoparia),* or summer cypress, is an annual weed introduced into the United States from Eurasia. This plant can now be found growing throughout the United States as one of our serious weed pests. Kochia has been promoted in some areas of the country as a forage plant. During drought years, it has been used both as forage and often as pasturage. However, kochia can cause a variety of problems in cattle and sheep. Although not common, it has been associated with oxalate and nitrate poisoning. On occasion, it has been associated with photosensitization, polioencephalomalacia, toxic nephrosis, and hepatitis. Diets having much kochia will result in decreased growth rates and death can occur. The principal manifestation of kochia poisoning is photosensitization. Poisoning due to kochia apparently depends on the environmental conditions under which the plant is growing.

Where and When Kochia Grows

Kochia weeds can be found growing on abandoned farm land, cultivated land, ditch banks, road sides, waste lands, and other disturbed areas. It thrives in a great variety of environments and soil types. Kochia is very drought tolerant. Kochia weed should not be confused with prostrate kochia *(Kochia prostrata),* which is considered excellent winter forage.

How Kochia Affects Animals

Cattle and sheep may become intoxicated after grazing kochia. Cattle are more susceptible to intoxication from kochia than are sheep. Cattle grazing pastures with a high percentage of kochia lose weight or gain it slowly. Cattle grazing near pure stands of kochia may show depression, dehydration, weight loss, photosensitiza-



Kochia is a bushy plant with stems either green or reddish. It is often confused with tumbleweed *(Salsola kali)* since it also breaks off and blows in the wind when dry.

tion, and discharge from the eyes and nose. They may show signs of polioencephalomalacia. Death frequently occurs. The toxin may be any or all of the following compounds: oxalate, nitrate, sulfate, saponin, and alkaloid.

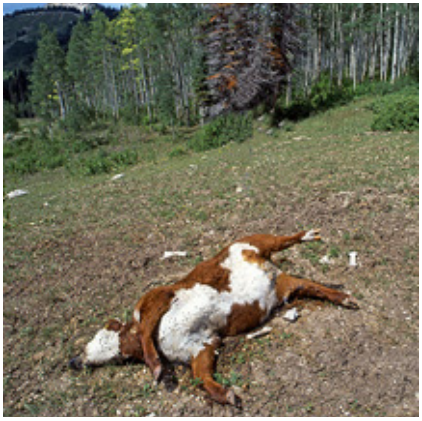
How to Reduce Losses

Ensure that alternative feed is available. During periods of drought, livestock producers frequently ask if it is safe to feed kochia to their cattle. In general, the advice given is to dilute the kochia with other hay if at all possible and watch the cattle closely for signs of poisoning. Grazing cattle on this plant leads to unpredictable results.

Larkspur

Distribution of Larkspur





Signs and Lesions of Larkspur Poisoning

- Nervousness
- Weakness and staggering gait; animal may fall suddenly
- Salivation
- Muscular twitching
- Nausea and vomiting may occur
- Bloating may occur
- Rapid, irregular pulse
- Animal may die suddenly; excitement intensifies all signs of poisoning
- Lack of gross lesions
- Pulmonary congestion

Larkspur (*Delphinium* spp.) causes heavy cattle losses in western range States. Larkspur is highly palatable to cattle, and losses can be expected when cattle are allowed to graze larkspur-infested ranges, especially where the plant is abundant or grows in large, dense patches. Cattle consume tall larkspur most often after plants begin flowering; consumption increases into the pod stage. Summer storms may lead to increased consumption of tall larkspur.

Losses rarely occur in sheep or horses, but if subjected to sudden physical activity after ingesting large amounts of larkspur, these animals may show clinical effects.

Plants are most toxic during early growth, but toxicity gradually declines over the growing season. The toxic substances are alkaloids, and over 40 of these compounds have been evaluated. The relative toxicity and concentration of individual alkaloids varies among species. The toxic alkaloids cause death by muscular paralysis, leading to respiratory failure or bloat.

All parts of the plant are poisonous, but new growth and the seed contain the highest concentrations of toxic substances. Normally, seeds do not pose any special prob-



Tall larkspur is a perennial found on hillsides and in meadows above 7,000 feet. It ranges in height from 2 to 6 feet. A hollow stem distinguishes larkspur from poisonous monkshood, which has a similar blue or purple flower without a spur.

lem if cattle have grazed the area prior to seed maturity; however, under a rest-rotation system, where cattle enter an ungrazed unit after seeds have started to mature, losses can be significant.

Where and When Larkspur Grows

Based on height at maturity and geographic location, larkspurs have arbitrarily been divided into three groups: the tall larkspurs *(D. barbeyi*, *D. occidentale*, and *D. glaucum),* the low larkspurs *(D. nelsonii*, *D. andersonii*, and *D. nutallianum),* and the plains larkspur *(D. geyeri).*

Tall larkspurs tend to grow at higher elevations on deep soils where a plentiful supply of moisture is available. They grow in mountain meadows on sites where deep snowdrifts persist well into the growing season, under aspens on north-facing slopes, along streams, or around seeps and springs. Tall larkspur begins growing as soon as snow melts, but at the upper limits of their distribution this may not occur until July.

Low larkspurs tend to grow at lower elevations where they mature and become dormant before the soil moisture is depleted. They begin growing in early spring, often before other forage begins growth. Low larkspurs grow best when springs are cold and wet. Cattle will graze low larkspur at all stages of growth, but most often graze it after flowering.

Plains larkspur is found primarily on the high plains of Colorado and Wyoming. It begins growth in spring before other plants.

How Larkspur Affects Animals

Plains larkspur may be eaten by cattle at any time during summer, but early green growth and pods may be most appealing to cattle. Both low and plains larkspurs may



Low larkspur has spurred blue flowers that grow on the top third of a single, unbranched stem. It is found on grassy hillsides and in sagebrush areas, where it may reach a height of 2 feet. Leaves alternate and are divided into deep, narrow lobes. The stem is hollow.

be the only green herbage available to cattle in early spring.

The larkspurs contain a number of alkaloids of varying toxicity. The most toxic of these are the MSAL (methyl succidimino acetyl lycoctonine) types, which include methyllycaconitine.

How to Reduce Losses

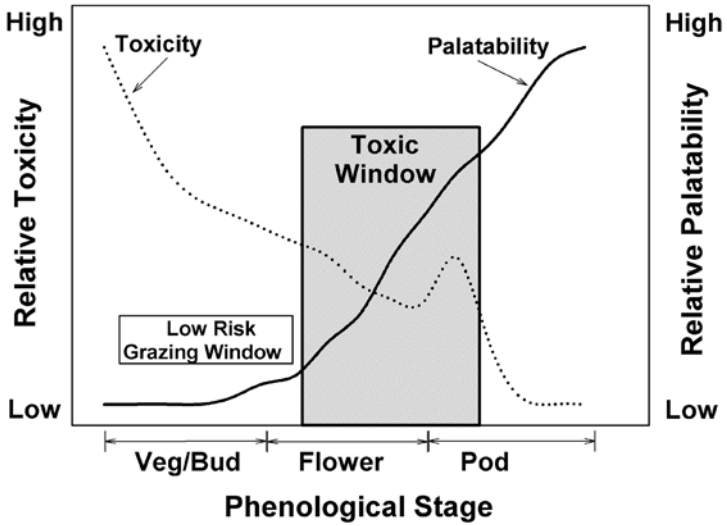
Placing an affected animal on its brisket or chest with its head uphill may reduce bloating. Treatment for bloat (intubation or rumen puncture with a trocar) may save some animals. Avoid unduly exciting affected animals.

Toxicity of tall larkspurs declines as it matures through the growing season. Research has identified a toxic window of high risk during the flower and early pod stages when it becomes palatable and toxin levels are moderate.

Since cattle do not generally consume tall larkspurs before flowering, grazing early before plants flower may be an option. Cattle should be moved off of the larkspur areas during the flower stage but can graze larkspur in the late pod stage when toxicity declines. Using sheep to graze or trample tall larkspur patches ahead of cattle grazing may reduce cattle losses.

Low larkspur losses may be prevented by deferring grazing until plants lose their flowers and pods, as they rapidly senesce after producing pods.

The cholinergic drug neostigmine (0.02 mg/kg i.m.) has been successfully used under pen conditions to reverse clinical larkspur intoxication. This reversal lasts about 2 hours, and repeated injec-



Toxic window theory of high risk for larkspur poisoning. When toxins are high, palatability is low; thus there is low risk of poisoning. Risk is highest when palatability is high and toxins are at moderate concentrations.

tions of neostigmine are sometimes required. Under field conditions, neostigmine temporarily abates clinical signs and animals quickly (about 15 minutes) become ambulatory. Depending on the larkspur dose, the intoxication can resurface. Nonetheless, there are risks associated with the use of neostigmine. The use of neostigmine-based treatments may actually aggravate losses in the absence of further treatment because suddenly mobile animals may later develop increased muscular fatigue and dyspnea and may die.

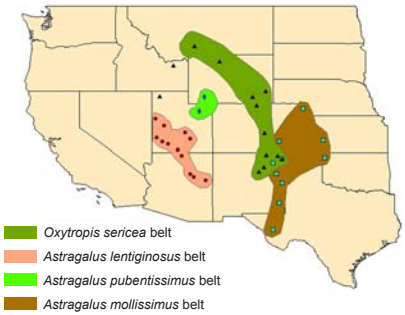
Research results show that low larkspurs can usually be controlled by applying 2,4-D at the rate of 4 lb ae/acre when the vegetative development approaches its maximum but before the first flowers open.

Tall larkspur can be controlled with picloram (1 to 2 lb ae/acre) up through the flowering stage. Metsulfuron (1 to 2 oz of product/acre) is effective when applied in the early vegetative stage of growth. Plains larkspur can be controlled with picloram (0.25 to 0.5 lb ae/acre) in the bud stage.

Do not graze cattle on larkspur ranges treated with herbicide until larkspur is senescent in the fall. Herbicide treatment may increase palatability to cattle, but toxicity remains high.

Locoweed

Distribution of Locoweed



Signs and Lesions of Locoweed Poisoning

- Depression
- Dull, dry hair coat
- Eyes dull and staring
- Irregular gait or some loss of muscular control
- Weakness
- Some animals show extreme nervousness
- Loss of sense of direction
- Withdrawal from other animals
- Some animals develop inability to eat or drink
- Abortions are common; hydrops may occur in some cattle
- Skeletal malformations may occur
- Animal may become violent if stressed
- Reduced libido
- Cessation of spermatogenesis and oogenesis
- Congestive heart failure when grazed at high elevations
- Vacuolation of neurons, renal tubular epithelium, hepatocytes, etc.
- Recumbency and death may follow prolonged consumption of locoweed

Horses, cattle, sheep, and goats are poisoned by eating locoweed. Wildlife such as elk and antelope also have been poisoned; locoweed is suspected in poisoning of deer and bighorn sheep as well. The locoweeds belong to certain species of the *Astragalus* and *Oxytropis* genera. Other species of *Astragalus* accumulate selenium or 3-nitro-propanol, which cause different types of intoxication.

The plant gets its name from the Spanish word “loco” (crazy), which describes the abnormal behavior of poisoned animals. “Locoed” animals do not recover completely. Horses affected by locoweed have no value as saddle or draft animals. However, the females may be used as brood mares.



Woolly loco is found from southwestern South Dakota south to Texas and New Mexico.

Generally, beef cattle poisoned on locoweed do not make economic gains, although they may appear to recover. Calf, lamb, and foal losses from abortion may be high. Skeletal birth defects are common; many lambs are lightweight at birth.

The more common species include white locoweed *(Oxytropis sericea),* woolly loco *(Astragalus mollissimus* var. *mollissimus),* spotted loco *(A. lentiginosus),* bigbend loco *(A. mollissimus* var. *earlei),* and garboncillo *(A. wootonii).* Loco is poisonous at all stages of growth. Plants are dangerous throughout the year—even when they have matured, dried, and turned black. All plant parts are toxic. The toxin is the indolizidine alkaloid swainsonine, which is produced by an endophyte (fungus growing within the plant). Absence of the endophyte may result in toxicity being greatly reduced or absent in some plant populations.



Locoweed is found on foothills and semiarid regions. It grows in tufts or clumps 4 to 24 inches tall.





Locoweed flowers resemble sweetpeas. Blossoms may be blue, purple, yellow, or white. Each stem contains numerous leaves with a leaflet on its tip. Top, white locoweed; bottom, spotted locoweed.

Where and When Locoweed Grows

Locoweed is commonly found on mountains, foothills, and plains and in semiarid desert regions. It starts growth in late fall, winter, or early spring—depending on locality, species, and moisture.

White locoweed grows from Montana, Idaho, and North Dakota south to Arizona, New Mexico, and Texas. Spotted loco grows from British Columbia, Canada, to Mexico and east to Kansas and Oklahoma. Bigbend loco is native to the region of Texas for which

it was named; it also occurs in southern New Mexico. Wooton loco (garboncillo) grows in eastern Arizona, southern New Mexico, and southwestern Texas. Seeds may remain viable in the soil 50 years or more.

Populations of all locoweed species are cyclic. White loco is relatively long-lived but its populations die out during major regional droughts and gradually increase during wet cycles. Woolly and spotted loco are short-lived perennials, germinating during autumn or spring rains. They grow for 2 to 3 years and then die from drought or insect damage. Wooton loco is a winter annual growing during wet winters.

How Locoweed Affects Livestock

Signs of poisoning appear after 2 to 3 weeks of continuous grazing on the plant. Locoweed has four principal effects on livestock: neurological damage; emaciation; reproductive alterations, such as abortion and birth defects; and congestive right heart failure when grazed at high elevations. The toxin is excreted in the milk; calves nursed by cows poisoned on locoweed have become intoxicated. The neurological signs of poisoning will disappear in time but may recur when an animal poisoned on locoweed is moved and stressed.

How to Reduce Losses

Locoweeds are relatively more palatable than other forage at certain seasons. Cattle prefer green-growing locoweed to dormant forage on short-grass prairies in early spring, but will cease grazing locoweed when it matures and warm-season grasses begin rapid growth. On mountain summer range, cattle prefer the young succulent pods of white locoweed. On desert winter range, cattle and sheep graze dry senescent spotted locoweed (which retains the toxin) in proportion to its availability. Livestock should not be allowed to graze locoweed-infested sites when they are likely to eat it. Once consumption of locoweed stops, swainsonine is rapidly cleared from the body.

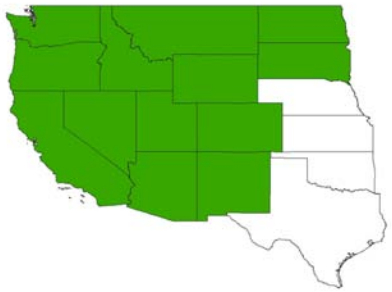
Livestock owners can reduce losses by keeping animals off locoweed-infested ranges until good forage is available.

There is no effective treatment for locoweed poisoning.

Locoweeds are particularly susceptible to clopyralid (0.12 to 0.5 lb ae/acre), picloram (0.25 to 0.5 lb ae/acre), metsulfuron (0.25 oz product/acre), and 2,4-D (2 to 3 lb ae/acre). All locoweeds have a large seed bank in the soil and will germinate and re-establish the population when environmental conditions are favorable.

Lupines

Distribution of Lupine



Signs and Lesions of Lupine Poisoning

- Nervousness
- Excessive salivation, frothing at the mouth
- Depression
- Reluctance to move about
- Lethargy, inappetence
- Difficulty in breathing
- Twitching leg muscles
- Loss of all muscular control
- Convulsions
- Coma
- Death
- Cleft palate and skeletal defects in fetus when grazed during 40th to the 100th day of gestation

Sheep in the Western States are frequently poisoned by feeding on lupine. Poisoning usually occurs when hungry animals are allowed to graze lupine. Losses may be especially heavy when hungry sheep are trailed through lupine ranges in late summer. Sheep and cattle have been poisoned by eating lupine plants that have been cut and dried for hay.

Cows may give birth to calves with cleft palate and skeletal defects if the cows ingest certain lupines during early gestation (crooked calf syndrome).



Five poisonous species are silky lupine *(Lupinus sericeus),* tailcup lupine *(L. caudatus),* velvet lupine *(L. leucophyllus),* silvery lupine *(L. argenteus),* and lunara lupine *(L. formosus).* Some lupine species are not poisonous to livestock, and not all species cause birth defects.

Piperidine and quinolizidine alkaloids (ammodendrine and anagyrine) are the compounds causing toxicosis and cleft palate as well as skeletal defects.

Poisonous species of lupine are toxic from the time they start growth in spring until they dry up in fall. Younger plants are more

toxic than older plants; however, plants in the seed stage in late summer are especially toxic because of the high alkaloid content of the seeds. Under proper conditions, some lupines make good forage.

Where and When Lupines Grow

Lupines grow on foothills and mountain ranges in sagebrush and aspen areas.

Silky lupine grows in northern areas from Washington and Oregon east to South Dakota. Tailcup lupine is found in Oregon, California, Idaho, Utah, Montana, and Wyoming. Velvet lupine is found in an intermountain region from Oregon to Wyoming. Silvery lupine occurs from North Dakota and Idaho south to Arizona and New Mexico. Lunara lupine grows in California.

How Lupines Affect Livestock

The amount of lupine that will kill an animal varies with species and stage of plant growth. It is not safe to let sheep graze species such as *L. argenteus* under any condition.

Cattle may be poisoned by eating 1 to 12 lb of lupine without other forage. Smaller amounts are poisonous if cattle eat lupine daily for 3 to 7 days. Hay containing lupines has caused poisoning and death of cattle. Crooked legs and other congenital deformities occur in newborn calves if cows graze certain species of lupine *(L. sericeus*, *L. caudatus*, and *L. laxiflorus)* between the 40th and 100th days of gestation.



Closeup shows typical circular pattern of lupine leaves.



Lupine is found on open and wooded hillsides. Poisonous species grow 1 to 3 feet tall. Leaves are composed of several leaflets, which radiate from a central point. Although blue is the most common color, flowers may also be white, pink, yellow, or blue and white.





Cleft palate of calf whose dam grazed teratogenic lupine during the early part of the susceptible gestational period.

How to Reduce Losses

Poisoning can be reduced by keeping hungry animals away from lupines in the early growth stage, in late summer when the plant is in the highly toxic seed stage, and from dense plant stands at all times. Supplemental feeding is beneficial, especially when animals are trailed through lupine ranges. If animals are poisoned on lupines, do not try to move them until they show signs of recovery.

If cows in the susceptible gestational period (40th to 100th days of gestation) are kept from lupine when it is most teratogenic (very

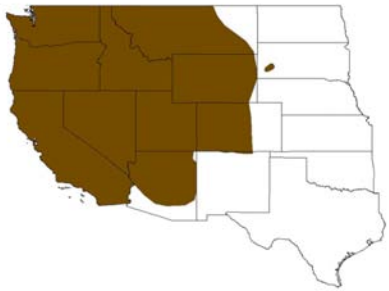
early growth or mature seed stage), most deformities can be prevented. The congenital deformity hazard is minimal at other gestation periods and after seeds have shattered from pods. The malformations can be avoided by adjusting the breeding season and the grazing of lupine-infested range to avoid the critical periods of gestation.

There is no known treatment for lupine poisoning.

Lupine can be controlled with 2,4-D (2 lb ae/acre), 2,4-D + dicamba (1 + 0.5 lb ae/acre), or triclopyr (0.5 to 1.5 lb ae/acre). Spray actively growing plants after they are 5 inches high but before they bloom. Reinvasion is rapid and re-treatment may be necessary every 4 to 5 years.

Milkvetches

Distribution of Milkvetches



Signs and Lesions of Milkvetch Poisoning

Acute Poisoning:

- Respiratory distress
- Muscular weakness primarily in pelvic limbs; prostration
- Death usually occurs in 3 to 4 hours
- Lobular alveolar emphysema; collapsed lungs and constricted bronchioles with interlobular edema
- Forced movement may cause these animals to collapse and die

Chronic Poisoning:

- Nervousness
- Labored, rapid respiration
- As intoxication progresses, respiration develops a wheezing or roaring sound
- Knuckling of fetlocks
- Goose stepping, knocking of hocks and/or feet when walking
- Drooping of pelvic limbs and loss of control of hind limbs, which may be dragged when animal moves
- Indications of temporary blindness
- Drooling; rough hair coat; constipation or diarrhea may occur
- When forced to move rapidly, animal may collapse and die
- All signs of poisoning increase with forced movement
- Lactating cows are more commonly affected than non-lactating
- Animals with advanced poisoning seldom recover but waste and die after several months
- Sheep show more respiratory and less neuromuscular involvement
- Horses can be intoxicated; cannot get them to back up
- Focal hemorrhages in brain
- Wallerian degeneration in spinal cord in pelvic region
- Alveolar emphysema, interlobular edema
- Death

Some milkvetches contain nitro compounds that are poisonous to cattle, sheep, and horses. These include plants such as Wasatch milkvetch *(Astragalus miser* var. *oblongifolius),* Columbia milkvetch (var. *serotinus*), Yellowstone milkvetch (var. *hylophilus*), red stemmed pea vine *(A. emorya-*

nus), and other species and varieties of *Astragalus.* Cattle and sheep of all ages are highly susceptible to the poisoning. Even when other forage is available, cattle readily eat some species of milkvetch.

Plants are poisonous from the time they emerge but decrease in toxicity as they mature and dry up.

The poisonous substance in Wasatch, Yellowstone, and Columbia milkvetches is the ß-D-glucoside of 3-nitro-l-propanol, or miserotoxin. Other milkvetches contain glucosides of 3-nitropropionic acid. Poisonous nitro compounds are found in varying quantities in 263 species and varieties of North American *Astragalus.*

Where and When Milkvetches Grow

Milkvetches grow throughout much of North America. Poisonous species grow on the meadows, deserts, and forests in the Rocky Mountain States. Milkvetches emerge from late April to June, depending on elevation and snowmelt. Leaves and stems become dry and are less dangerous after seed dispersal in July or August. Red-stemmed pea vine *(Astragalus emoryanus)* occurs in western Texas and New Mexico.

How Milkvetches Affect Livestock

The poison in milkvetches acts quickly. Two lb of green milkvetch may cause acute poisoning or death in a 1,000-lb cow. Some deaths occur within 1 hour, so fast that cattle show no signs; more often, animals die within 3 or 4 hours after eating the plant. Lactating cows are more readily affected than dry cows. Do not stress cows that are grazing or have recently grazed milkvetch.

Acute poisoning is characterized by a general muscular weakness so that affected animals fall after the slightest excitement. The heart beats very rapidly before the animal dies from heart failure.

Chronic intoxication may occur in cattle and sheep from grazing any of the toxic varieties of *Astragalus miser, A. emoryanus,* and *A. Canadensis*—or other nitro-containing *Astragalus* species over a period of several days or weeks. Intoxication in cattle is characterized by goose stepping (cracker heels), varying degrees of posterior paralysis, and respiratory problems characterized by loud breathing sounds (roaring disease). This condition occurs in most of the Western States and Western Canada.



Flowers of Wasatch milkvetch resemble sweetpeas. They vary in color from creamy white to shades of violet.



Cow poisoned on milkvetch.

How to Reduce Losses

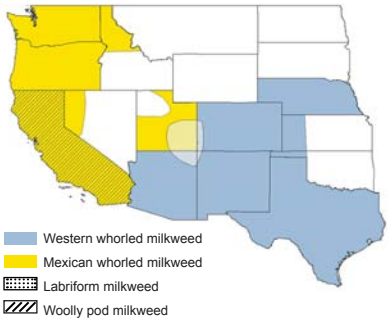
To reduce losses, prevent animals from grazing these plants for extended periods. Feeding protein supplements has been shown to reduce intoxication. Avoid stressing cattle that have grazed these milkvetches. Cattle 3 years or older are less susceptible than younger ones.

There is no known treatment for milkvetch poisoning.

Milkvetches can be controlled with 2,4-D (2 lb ae/acre), picloram, or clopyralid (0.25 lb ae/acre). Treat plants before they reach full bloom.

Milkweed

Distribution of Milkweed



Signs and Lesions of Milkweed Poisoning

- Depression, weakness, and staggered gait
- Difficulty in breathing with expiratory "grunting" sounds
- Dilation of pupils
- Rapid, weak pulse
- Loss of muscular control
- Elevated temperature
- Violent spasms
- Bloating
- Respiratory paralysis
- Congestion of visceral organs
- Renal tubular degradation and necrosis
- Gastroenteritis

Several species of milkweed are poisonous to livestock. Labriform milkweed *(Asclepias labriformis)* is the most toxic. Other species, in order of toxicity, include western whorled milkweed *(A. subverticillata),* woollypod milkweed *(A. eriocarpa),* and Mexican whorled



Milkweed is a perennial that often bears blossoms and fruit at the same time. The plant may be 1 to 3 feet tall. Greenish-white flowers are borne in umbrella-like clusters. Leaves may be narrow or broad.

milkweed *(A. fascicularis).* The whorled, narrow-leaf milkweeds are most toxic while the common broad-leaf milkweed is relatively nontoxic.

Milkweed poisoning occurs in chickens, turkeys, rabbits, horses, cattle, sheep, and goats, with avian species being less susceptible than mammals. Most livestock losses are a result of hungry animals being concentrated around heavily infested milkweed areas (such as corrals, roadways, and bedding grounds). However, poisoning may also occur when animals are fed hay contaminated with milk-



Illustration of broadleaf (left) and narrow-leaf milkweed (right). Drawing courtesy of Holly Broome-Hyer, graphic artist, Logan, Utah.

weed. The narrow-leaf, whorled milkweed species appear to be more palatable than the broadleaf species. Thus, poisoning incidents from contaminated hay are more commonly associated with the whorled milkweeds. Similarly, field poisoning cases are more common from the whorled milkweeds. Milkweed may cause losses at any time, but it is most dangerous during the active growing season. Livestock may be more apt to graze these plants after a frost or when other available forage is limited.

Leaves and other aboveground parts of the plant are the typical cause of poisonings, but roots may also contain toxic constituents. The nonwhorled milkweeds contain a variety of glycosidic substances called cardenolides that are highly toxic. In contrast, the whorled milkweeds generally contain very low cardenolide concentrations. The whorled milkweed species are thought to contain as yet uncharacterized neurotoxin(s).

Where and When Milkweed Grows

Milkweed is often found in sandy soils of plains and foothills. It grows on ranges and abandoned farms, along roadsides, in pastures, in ditches, and in waste places or overgrazed areas. This plant gets its name from the milky, sticky juice that oozes out quickly when any plant part is broken.

How Milkweed Affects Livestock

The toxicity of milkweed species is quite variable, ranging from relatively nontoxic to highly toxic. An average-size sheep that eats 1 to 3 oz of green leaves of one of the more toxic species is likely to die of poisoning within a few hours to 2 to 4 days. On a body weight basis, for the milkweeds that have been identified as being toxic, ingestion of as little as 0.05 percent to greater than 2 percent of an animal's body weight can result in poisoning.

How to Reduce Losses

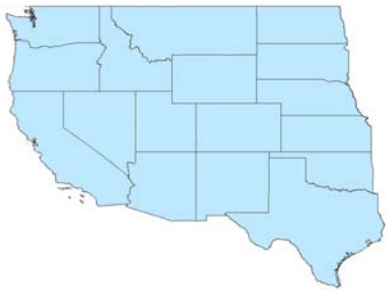
Animals usually do not eat milkweed unless good forage is scarce or when plants have been frosted.

Livestock owners can reduce losses by keeping animals out of milkweed-infested areas. Supplemental feeding usually is beneficial during trailing. Hay contaminated with milkweed can be poisonous to sheep and cattle and should not be fed to them. When possible, rid the areas of milkweed and then manage to prevent reinvasion.

Milkweeds can be controlled with 2,4-D plus picloram (1 + 0.5 lb ae/acre) or glyphosate as a spot spray.

Nightshades (*Solanum* spp.)

Distribution of Nightshades



Signs and Lesions of Nightshade Poisoning

- Labored breathing and expiratory grunt
- Salivation and nasal discharge
- Body temperature may be slightly elevated
- Yellow discoloration of the skin may occur in chronic poisoning
- Apathy, drowsiness, progressive weakness, paralysis, and trembling
- Increased heart rate
- Fat may be yellowed and gelatinous
- Gall bladder may be distended
- Gastrointestinal irritation including inflammation, hemorrhage, and ulceration

There are several species of nightshades that are toxic to horses, cattle, swine, sheep, and poultry.

The genus includes annual and perennial herbs and shrubs that can be found throughout the United States.

The principal species that serve as examples of the genus are black nightshade *(Solanum nigrum),* silverleaf nightshade *(S. eleagnifolium),* and buffalo burr *(S. rostratum).* Black nightshade is an introduced herbaceous annual weed that can be found growing mostly on disturbed soils and waste areas in the Eastern United States and into the Midwest. Silverleaf nightshade is a perennial with long creeping rootstocks. Buffalo burr is an annual native to the Great Plains and introduced to the West Coast.

The toxins include a combination of a number of sugars and at least six different steroidal amines combined to form a variety of glycoalkaloids. One example is the toxin solanine. Potatoes are included with this group because the vines and tubers that have been exposed to light can be toxic to livestock. Drying does not destroy the toxin.

Nightshade species are not very palatable to livestock. However, these plants often grow as weeds in hay and silage crops and small grains where they can be harvested with the crop and then fed to livestock.

Where and When Nightshades Grow

Black nightshade (both the native and introduced varieties) is an annual 6 inches to 3 feet tall. Leaves are simple, ovate to lanceolate, entire to sinuate-dentate. Flowers are white; berries are black when ripe. It grows peripherally in moist areas of fields and pastures of disturbed loamy or gravelly soils throughout the United States.

Silverleaf nightshade is a perennial that grows 1 to 3 feet tall with white, hairy leaves and stems. Leaves are simple, thick, lanceolate to linear, entire to sinuate. Stems and ribs usually have short stiff spines. Flowers are violet or blue; berries are yellow or orange. Silverleaf nightshade grows in fields, pastures, and roadsides from Missouri to Texas and California.

Buffalo burr is an annual spiny weed 1 to 2 feet tall. Leaves are irregularly round-lobed or once or twice pinnately deeply lobed; veins are spiny. Flowers are yellow, and the berries are enclosed. Native to the Great Plains and introduced to the West Coast, buffalo burr grows in old fields, overgrazed pastures, and roadsides.

How Nightshades Affect Livestock

Nightshades are generally unpalatable and are not grazed by livestock except under the stress of overgrazing or in contaminated hay and grain. Poisoning by this group of plants does not always end in death. In acute poisoning, the nervous symptoms develop rapidly. Death or recovery occurs within a few hours to 1 or 2 days. Death apparently is related to the paralysis. Chronic poisoning is accompanied by emaciation, rough hair coat, anorexia, constipation, and ascites.

How to Reduce Losses

Losses can be kept at a minimum by good pasture management and weed control. Harvested forage such as hay, grain, or silage can be contaminated with nightshades. Contaminated forage can be fed if it is diluted (mixed) with nightshade-free forage: an on/off feeding strategy should be used. Animals being fed this diluted forage should be kept under close surveillance and immediately removed from the contaminated feed if signs of poisoning appear.

Nitrate-accumulating Plants

Signs and Lesions of Poisoning by Nitrate-accumulating Plants

Acute poisoning:

- Blue coloration of membranes of mouth, eyes, and other mucous membranes (cyanosis)
- Shortness of breath
- Staggering gait
- Death
- Chocolate-brown blood
- Muddy, cyanotic mucus membranes
- Congestion of rumen and abomasum

Subacute poisoning:

- Watering eyes
- Unthrifty appearance
- Reduced milk flow
- Reduced weight gain
- Abortion and infertility

Many plants, both crops (oat hay, sorghum, corn, sudangrass, Johnsongrass, and beets) and weeds (carelessweed, kochia, pigweed, Russian thistle, and nightshade), can accumulate nitrate. Plants containing more than 1.5 percent nitrates (as KNO_3) dry weight may be lethal to livestock. Sublethal effects may occur in livestock from eating feed containing between 0.5 percent and 1.5 percent nitrate. Nitrate poisoning can also occur in animals that consume nitrate fertilizers, machine oil, and some natural well and pond waters.

Plants differ in their ability to accumulate nitrate. Often the type of soil present and the form of nitrogen therein influences the amount of nitrate that accumulator plants may contain. Drought conditions or cloudy weather may enhance nitrate accumulation. Treatment of nitrate-accumulating plants with 2,4-D may also cause plants to accumulate excessive amounts of nitrate. Nitrate accumulates primarily in the vegetative tissue of plants while the seed remains safe.

Where and When Nitrate Poisoning Occurs

Nitrate poisoning occurs throughout the United States. Livestock may be poisoned after eating either harvested or nonharvested plants, feed materials high in nitrate, nitrogen fertilizer, etc.

How Nitrate Poisoning Affects Livestock

Nitrates are converted to nitrite in ruminants. Nitrite causes the production of methemoglobin, a form of hemoglobin that cannot carry oxygen. Thus, the effects of nitrate poisoning result largely from oxygen starvation or suffocation.

The amount of plant material required to poison an animal depends on the amount of nitrate in the

plant and on the rate at which the plant is eaten. Many factors affect toxicity, but in general about 0.05 percent of an animal's weight of nitrate is near a minimum lethal dose. Abortions in cattle have been associated with the consumption of high-nitrate forage or with maternal poisoning resulting in fetal hypoxia.

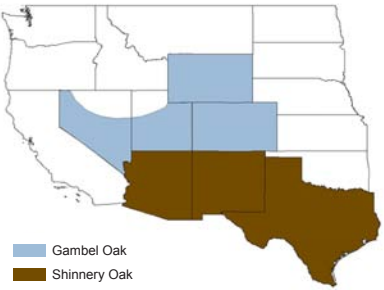
How to Reduce Losses

Crops that accumulate nitrate and grow under conditions favoring nitrate accumulation should be checked for nitrate content. High-nitrate forage should be diluted with low-nitrate forage to decrease nitrate intake; or if there are indications of nitrate toxicity, the feeding of nitrate-accumulating forage should be discontinued. Adequate levels of readily fermentable carbohydrates (such as corn) can provide some protection against nitrate intoxication. Ruminants can adapt to relatively high dietary nitrate levels if the high-nitrate feed is increased gradually in the diet. Otherwise, mix a low-nitrate feed with the high-nitrate feed.

If the condition is identified soon after onset of clinical signs, animals poisoned on nitrate can be successfully treated by giving methylene blue intravenously at 2 to 4 mg/kg body weight.

Oak

Distribution of Oak



Signs and Lesions of Oak Poisoning

- Lack of appetite
- Gaunt, tucked-up appearance
- Constipation, frequently followed by profuse diarrhea
- Weakness
- Rough, dry hair coat; dry muzzle
- Tendency to remain near water, excessive thirst
- Reluctance to follow the herd
- Emaciation
- Mucus in feces
- Frequent urination; dark-colored urine
- Swelling under the jaw
- Subnormal temperature
- Collapse
- Death
- Gastritis: abomasum and small intestine inflamed
- Swollen kidneys
- Nephritis with necrosis and renal tubular casts
- Ascites and subcutaneous edema
- Uremia; elevated BUN (blood urea nitrogen)

Cattle may be poisoned by browsing on oak in fairly large amounts over a period of 2 to 3 weeks. Shinnery oak *(Quercus havardii)* and Gambel oak *(Q. gambelii)* are the species responsible for most losses. However, many other species can cause poisoning when a large amount of the oak plant material is ingested. Shinnery oak, or shinnery, is common in the Southwest. Gambel oak, also called scrub oak, is found throughout the central part of the Western States.

Poisoning occurs primarily when animals graze buds and early growth leaves, sprouts in cut-over areas, mature foliage, and acorns. Oak is most dangerous in the budding and leafing stages and when acorns are available. As leaves mature, oak decreases in toxicity.

Cattle may get as much as 50 percent of their diet from oak browse without showing signs of poisoning. More than 50 percent of oak browse in the diet will cause sickness, and more than 75 percent will cause death. Poisoning is most likely during times of drought.

The toxic substance in oak is probably a complex of tannins called gallotannins.

Where and When Oak Grows

Shinnery oak is a low spreading shrub that grows in sandy areas. It is found primarily in western Oklahoma, western Texas, and eastern New Mexico.

Gambel oak is a shrub or small tree. It grows in dense thickets on foothills and mountain slopes, up to an elevation of 9,000 feet.

Oak usually starts growing in early spring before other range plants.

How Oak Affects Livestock

Death from oak poisoning may occur from a few days to 2 weeks after the cattle first show signs. Chronic poisoning results in poor-doing animals that never fully recover once oak consumption ceases. Milk production will decrease in lactating cows, and they will become emaciated. Goats are less susceptible than cattle.

How to Reduce Losses

Livestock losses from oak poisoning can be reduced by conservative grazing and proper seasonal use of shinnery and Gambel oak ranges. Oak leaves decrease in toxicity as they mature.

In shinnery areas of the Southwest, livestock owners should reserve pastures with the least amount of oak for early spring grazing. The use of supplemental feed containing 10 percent calcium hydroxide will reduce losses.



Scrub oak may be a shrub 3 to 4 feet high or a tree up to 20 feet high.

In Gambel oak areas, animals should be kept off oak ranges until other forage becomes available and the oak leaves mature.

At first signs of poisoning, cattle should be moved to an oak-free area. In some cases, the use of a mild laxative, such as mineral oil, may be beneficial.

Oak can be controlled with tebuthiuron at 0.5 to 1 lb ai/acre applied to the soil in winter.

Caution: *tebuthiuron is a sterilant and will kill all vegetation.*

Poison Hemlock

Distribution of Poison Hemlock



Signs and Lesions of Poison Hemlock Poisoning

- Nervous trembling
- Neuromuscular stimulation followed by depression and paralysis
- Ataxia, especially lower and hind limbs
- Salivation
- Lack of coordination
- Dilation of the pupils
- Rapid, weak pulse
- Respiratory paralysis
- Coma
- Death
- Convulsions have been reported
- Occasionally bloody feces and gastrointestinal irritation
- Skeletal birth defects and cleft palate in calves and piglets if cows or sows eat poison hemlock during susceptible stage of gestation: 40th to 100th days for cows, 30th to 60th days for sows

Poison hemlock *(Conium maculatum)* can be found growing throughout the United States. Sheep, cattle, swine, horses, and other domestic animals are poisoned by eating a small amount. It is also extremely poisonous to humans.



Poison hemlock has white flowers that grow in small erect clusters. Each flower develops into a green, deeply ridged fruit that contains several seeds. After maturity, the fruit turns grayish brown.



Poison hemlock grows 4 to 10 feet tall. Leaves are delicate, like parsley, and it has a white taproot.

Poison hemlock is sometimes confused with western waterhemlock—a more deadly plant—because the names are similar. (See Waterhemlock chapter in this volume.) Poison hemlock has a number of common names, including deadly hemlock, poison parsley, spotted hemlock, European hemlock, and California or Nebraska fern.

Roots of poison hemlock may be mistaken for wild parsnips and eaten by people. The stem of poison hemlock has purple spots on it.

All parts of poison hemlock—leaves, stem, fruit, and root—are poisonous. Leaves are especially poisonous in spring up to the time the plant flowers. Fresh leaves are unpalatable, so livestock seldom eat hemlock when other feed is available. The toxic compounds are coniine, γ–coniceine, and related piperidine alkaloids.

Where and When Poison Hemlock Grows

Because of its attractive flowers, poison hemlock was brought to the United States from Europe as a garden plant but has escaped cultivation and can be found growing in many pastures and in some areas on rangeland. Poison hemlock is found at roadsides, along fences and ditch banks, on edges of cultivated fields, along creekbeds and irrigation ditches, and in waste areas and may invade fields or pastures.

Poison hemlock is a biennial and belongs to the carrot family. It starts growing in early spring but does not flower until its second year. In favorable locations it may be a perennial. Poison hemlock harvested with hay can be toxic to livestock and produce birth defects.

How Poison Hemlock Affects Livestock

Poison hemlock ingestion is often fatal. Sheep may be poisoned by eating as little as 4 to 8 ounces of green leaves. Cattle that eat 10 to 16 ounces may be affected. Signs



The hollow stem is marked with purple spots, a distinguishing feature.

usually appear within an hour after an animal eats the plant. Animals die from respiratory paralysis in 2 to 3 hours. Convulsions, which are common in waterhemlock poisoning, seldom occur with poison hemlock.

Skeletal deformities or cleft palate may be induced in offspring of cows, sheep, goats, and pigs if poison hemlock is ingested by the mother during susceptible stage of gestation: 40th to 100th days in cows and 30th to 60th days in sheep, goats, and pigs. Palate and skeletal deformities in calves are indistinguishable from the lupine-induced crooked calf disease.

How to Reduce Losses

Avoid stressing poisoned animals that are not recumbent. For recumbent animals, support respiration and treat with activated charcoal and a saline cathartic. Gastric lavage may be beneficial, with atropine therapy to control parasympathetic signs.

Animals that recover seldom show lingering effects.

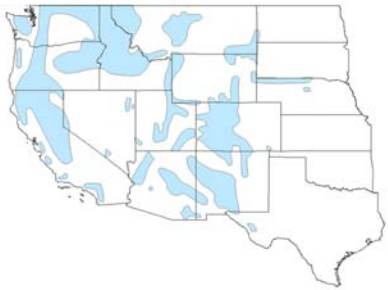
Research results show that poison hemlock may be controlled by treating plants before they begin to bud with 2,4-D plus dicamba (2.5 lb + 1 lb ae/acre). Repeat applications may be needed.

Hemlock Poisoning in Humans

People may be poisoned by eating any part of a hemlock plant. Often poisoning occurs when the victim has confused hemlock root with wild parsnips, hemlock leaves with parsley, or hemlock seed with anise.

Ponderosa Pine Needles

Distribution of Ponderosa Pine



Signs and Lesions of Ponderosa Pine Needle Poisoning

- Abortion characterized by weak parturition contractions, excessive uterine hemorrhage, and incomplete dilation of the cervix
- Calves are weak but may survive if abortion is near term and extra care is provided
- Persistently retained placenta
- There may be indications of the impending abortion
- Following the abortion, cows may develop endometritis and septicemia accompanied by a marked increase in body temperature; cow may die if treatment is not immediately provided

The needles of ponderosa pine *(Pinus ponderosa)* cause abortion when grazed by cattle. The abortions occur primarily from late fall to early spring, during the last trimester of pregnancy. Pine needles are generally grazed by cattle during cold winter weather when forage is lacking because of previous grazing or snow cover. However, cows may incidentally ingest dry fallen pine needles when grazing under tree cover. They may also eat needles from slash piles or windfall branches. The abortifacient compounds are diterpene acids including isocupressic acid and its metabolites.



Where and When Ponderosa Pine Grows

Ponderosa pine grows in all of the States west of the Great Plains and in Western Canada. Lodgepole pine, some cedars, junipers, and Monterey cypress can also cause abortions when consumed by cows.

How Ponderosa Pine Needles Affect Livestock

Abortions may begin within 48 hours after pine needles are ingested. Some cows abort as much as 2 weeks after having ingested the needles. Only a small amount

of needles is required to cause an abortion. Incidence can vary from only a few to 100 percent of the cows involved. There are no indications of overt toxicity to cattle eating the needles. If calves are far enough developed when the cow eats pine needles, they will be small and weak but may survive with proper care. Surviving calves may not nurse or may not be allowed to suckle by the dam; the dam may not have sufficient milk to feed the calf.

A persistently retained placenta is a constant finding regardless of the gestational stage of the abortion. Cows should be observed closely the first few days following an abortion. In case of complications from the retained placenta, consult a veterinarian. Some of these cows will die. The mortality of cows aborting from eating pine needles can be significantly higher than normal.

How to Prevent Poisoning

Prevent access to pine trees during the last trimester of pregnancy.

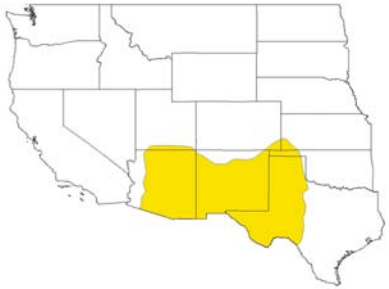




The ponderosa pine is a hardy tree extensively harvested for lumber. Both the dry and green needles and bark from the tree can cause abortion in cows.

Rayless Goldenrod

Distribution of Rayless Goldenrod



Signs and Lesions of Rayless Goldenrod Poisoning

- Lassitude and depression
- Standing in a humped-up position; stiff gait when walking
- Trembling of muscles of nose and legs, especially after exercise; as the condition worsens, the whole body may shake
- Weakness
- Constipation and dribbling of urine
- Recumbency
- Acetone odor on the breath
- Coma
- Terminal respiration consists of prolonged inspiration, a short pause, and forced expiration
- Death
- Myocardial necrosis and fibrosis of cardiac and skeletal muscles in horses and goats
- Degenerative changes of the liver

Rayless goldenrod *(Isocoma wrightii* or *pluriflora),* rosea, or jimmy weed, is an erect, bushy, unbranched perennial shrub, growing 2 to 4 feet tall. The leaves are alternate, linear, and sometimes sticky, and the flowers are yellow. It is toxic to horses, cattle, sheep, and goats. The toxin, referred to as tremetol (a mixture of compounds including tremetone, dehydrotremetone, 3-hydroxytremetone and 3-oxyangeloyl-tremetone), is excreted in the milk of lactating animals, so the offspring of the animal or humans drinking the milk may become poisoned by the consumption of contaminated milk. The toxin is cumulative and is present in both green and dry plant material.

Where and When Rayless Goldenrod Grows

This shrub grows on the dry rangelands from southern Colorado into Texas, New Mexico, and Arizona. It grows especially well in river valleys and along drainage areas. Poisoning is most common in late fall and winter.

How Rayless Goldenrod Affects Livestock

Daily consumption of 1 to 1.5 percent of an animal's weight of the green plant for 1 to 3 weeks will produce signs of poisoning in horses, cattle, and sheep. Death will result if the affected animals are not removed from access to the plant. The condition it produces in cattle is known as trembles.

How to Reduce Losses

Poisoning of livestock by rayless goldenrod can best be prevented by not overgrazing livestock on ranges infested with this plant and by feeding hay following snowstorms that cover other forage. There is no treatment for poisoning by this plant but livestock may recover if they are not stressed and are given fresh feed and water; long-term effects are unknown.



Closeup of rayless goldenrod showing the leaves and flowers.



Rayless goldenrod in its natural environment. Photo courtesy of Dr. Samantha R. Uhrig, Desert Willow Veterinary Services, LLC, Carlsbad, NM.

Rayless goldenrod can be controlled by picloram or dicamba (1 to 2 lb ae/acre) in late summer following adequate rainfall.

White Snakeroot

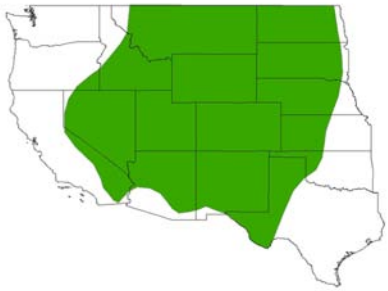
White snakeroot *(Eupatorium rugosum)* contains the same toxin as rayless goldenrod (tremetol compounds), and it is toxic to cattle, horses, sheep, and goats. It affects all animals, including humans who consume milk from affected animals ("milk sickness"). It damages nearly all muscle types including the heart muscle, disrupting cardiac function. Death may result, especially when an animal is stressed. White snakeroot grows in the Midwest and south into eastern Texas and is most commonly found in moist, shady, wooded areas.



White snakeroot.

Selenium-accumulating Plants

Distribution of Selenium-accumulating Plants



Signs and Lesions of Selenium Poisoning

Acute Poisoning:

- Abnormal posture
- Unsteady gait
- Diarrhea
- Abdominal pain
- Increased pulse and respiration rate
- Prostration
- Death
- Petechial hemorrhage, endocardia, acute congestion and hemorrhage of the lungs; enteritis and passive congestion of the liver

Sheep do not show the typical signs of poisoning observed in cattle, horses, and pigs. Depression, increased respiration, and sudden death are seen in sheep.

Chronic Poisoning:

- Dullness
- Rough hair coat
- Emaciation
- Lack of vitality
- Lameness
- Hooves may become overgrown
- Loss of long hair

Selenium is accumulated by a number of plants in sufficient amounts to be chronically toxic, and in some cases acutely toxic, if consumed by livestock. Plants have been divided into three groups, based on their selenium-accumulating ability.

Plants containing more than 5 ppm (parts per million) selenium are potentially toxic.



Two-grooved milkvetch *(Astragalus bisulcatus)* is a selenium-accumulating plant.

Primary selenium accumulator or indicator plants may accumulate up to several thousand parts per million selenium. This group includes species of milkvetch *(Astragalus),* woody aster *(Machaeranthera* section *Xylorrhiza),* goldenweed *(Haplopappus* section

Oonopsis), and princes plume *(Stanleya pinnata).* Selenium accumulated by these plants is largely water soluble and in the form of low-molecular-weight organic compounds and selenate. The selenium is not incorporated into protein.

Secondary selenium accumulator plants may contain up to several hundred parts per million selenium, but lesser amounts are much more common. This group includes species of aster *(Aster),* milkvetch *(Astragalus),* saltbush *(Atriplex),* Indian paintbrush *(Castilleja),* toadflax *(Comandra),* gumweed *(Grindelia),* snakeweed *(Gutierrezia),* woody aster *(Machaeranthera),* and stickleaf *(Mentzelia).* In rare instances, these plants will accumulate up to several thousand parts per million. As in the first group, selenium appears primarily as water-soluble selenate with smaller amounts of low-molecular organic forms.



Aster sp., a secondary selenium accumulator

The nonaccumulator group includes grasses such as western wheat grass, small grains, and alfalfa. Selenium absorbed by these plants is metabolized into plant proteins. These plants rarely accumulate more than 50 ppm selenium and usually only 5 to 12 ppm. On rare occasions, however, several hundred parts per million may be taken up.

Where Selenium-Accumulating Plants Grow

The major seleniferous areas of the West can be found in North and South Dakota, Montana, Wyoming, Colorado, and Utah. Selenium poisoning occurs in the areas that have soils high in selenium.

How Selenium Affects Livestock

Selenium intoxication can occur as acute (rapid death) or as chronic (hair loss, hoof lesions, etc.) poisoning. Selenium is an essential nutrient. The problem in dealing with selenium is that the difference between the amount required for good nutrition and the amount that is toxic is very small.

The nonaccumulator plants are the most problematic of the selenium accumulators because they are commonly grazed by livestock. Cattle and sheep grazing on these plants may have problems with overgrown hooves and reproduction. Animals (both males and

females) may show few obvious signs of selenium poisoning but may suffer reproductive effects.

How to Reduce Losses

The only practical method of reducing losses in livestock to selenium poisoning is to prevent animals from eating excessive amounts of selenium-containing plants. Soil and forage surveys or maps of the selenium content over a ranch or a range can be of great assistance in developing a grazing or forage-production program. The amount of selenium in soil and forage can vary markedly over an area.



Hooves of pigs fed selenium-accumulating plants. Note the cracks on the tops of the hooves.

The chemical form of selenium in the soil can dramatically affect plant uptake. Identifying high-selenium and low-selenium areas, and when possible the form of selenium present, can help in deciding where to graze animals or if selenium supplementation is needed. Breeding horses or cows should not be pastured in a high-selenium area. Affected animals



Princes plume *(Stanleya pinnata),* a primary selenium accumulator.

that are removed from seleniferous forage may recover without apparent permanent effects; however, recovery depends on the severity of intoxication.

Snakeweed (Broom and Threadleaf)

Distribution of Snakeweed



Signs and Lesions of Snakeweed Poisoning

- Listlessness
- Periodic mucopurulent nasal discharge with crusting and sloughing of nasal mucosa
- Anorexia and weight loss
- Rough hair coat
- Diarrhea followed by constipation
- Hematuria may occur
- Gastroenteritis
- Hydropic degeneration and necrosis of the liver; mild icterus
- Toxic nephrosis
- Uterine edema and possible hydrops of fetal membranes
- Pregnant cows may have periodic vulvar swelling and earlier-than-normal udder development; they may abort and retained placenta is present.

Broom snakeweed *(Gutierrezia sarothrae)* and threadleaf snakeweed *(G. microcephala)* are perennial weeds common on rangelands from Canada to Mexico. Cattle, sheep, and goats have been poisoned in large numbers from eating snakeweed; however, most losses occur in cattle. Snakeweed should be considered for its toxic proper-



ties and for its abortifacient effects. Death may occur, but the principal effect of poisoning is abortion. Toxin levels increase through the growing season. Cattle will not eat snakeweed unless other feed is scarce. Abortions often follow a snowstorm because snow covers the grasses leaving only the snakeweed for the animals to graze.



Snakeweed is a densely branched perennial shrub that grows to 2 feet tall on dry range areas from Texas to California north to Colorado and Idaho and south into Mexico.

Where and When Snakeweed Grows

Broom snakeweed and threadleaf snakeweed are very similar in appearance. They are distinguished by the number of ray flowers per head: three or more ray flowers for broom snakeweed and only one or two for threadleaf snakeweed. Snakeweed grows on the drier range areas. Snakeweed is said to be more toxic when growing on sandy soils than on limestone soils (referred to as hard soils). In the Southwest, it resumes growth following flowering in the fall and remains green over the winter. Farther north it begins growth in the spring and flowers in the late summer and early fall. Its populations are cyclic, increasing in wet years and dying back during drought.

How Snakeweed Affects Livestock

The amount of snakeweed needed to cause death or abortion in cattle varies greatly. Cows near term may abort or give birth to small, weak calves that may survive if properly cared for. Cows that abort have a persistently retained placenta. Cows aborting may develop endometritis and septicemia accompanied by an increase in body temperature. These cows are at risk unless properly treated by a veterinarian. Factors affecting dosage include site and stage of growth and rate of consumption. The toxins are thought to be diterpene acids.

How to Reduce Losses

Snakeweed increases with disturbance (overgrazing, fire, drought). Maintaining ranges in good condi-

tion can prevent dense concentrations of snakeweed. Snakeweed is not palatable. Adequate forage will prevent consumption of snakeweed. Cows in poor body condition probably eat more snakeweed than cows in better condition because the former graze less selectively.

Poisoned cows should be removed from snakeweed areas and given proper care until they recover.

In the Southwest, snakeweeds can be reliably controlled by spraying in the fall using picloram (0.25 to 0.5 lb ae/acre) or metsulfuron (0.5 g ai/acre). Further north in the Plains and Intermountain regions, these same herbicides and rates must be applied in early summer while snakeweeds are in the vegetative stage and growing rapidly.

Sneezeweed

Distribution of Sneezeweed



Signs and Lesions of Sneezeweed Poisoning

- Decreased appetite
- Lips and muzzle become stained green from vomiting (spewing sickness)
- Lambs become stiff and wasted
- Depression
- Weakness with irregular pulse
- Frothing at the mouth
- Coughing
- Emaciation and wasting
- Ascites
- Gastrointestinal hyperemia
- Renal tubular necrosis
- Liver degeneration

Animals that feed on sneezeweed may become affected with "spewing sickness." The disease gets its name from its most characteristic sign—chronic vomiting or spewing.

In the Western States, western sneezeweed *(Hymenoxys hoopesii;* formerly *Helenium)* is common to many mountain ranges. Sheep are frequently poisoned by eating sneezeweed; cattle are rarely poisoned. Animals eat sneezeweed during summer and fall, when other forage is scarce or has become less palatable. Lambs poisoned on sneezeweed become unthrifty and are difficult to market. Poisoned ewes are often emaciated (wasting) and may not die until some time after removal from sneezeweed.

All plant parts are poisonous. The toxin is a sesquiterpene lactone. Sneezeweed, bitterweed, and rubberweed have the same type of toxin.

Where and When Sneezeweed Grows

Sneezeweed grows at elevations of 5,000 to 12,000 feet on moist slopes and well-drained meadows from western Montana and eastern Oregon southward to California and New Mexico. It starts growing in early spring and matures in late summer and early fall. Sneezeweed increases with overuse of the range. Heavy stands of sneezeweed will inhibit growth of other plants.

How Sneezeweed Affects Livestock

An animal may die if it eats small quantities of sneezeweed over a long period. Sheep may be poisoned by eating about 2 lb of green

sneezeweed leaves daily for 10 days.

Some animals die within a few days after the first signs appear. Others that develop a chronic form of poisoning may live for 2 to 3 weeks or more. Complete recovery from the poisoning is possible if animals are taken off the plants as soon as the first signs are observed.

Lambs usually show signs of stiffness before other symptoms appear. Lambs left to graze sneezeweed after signs first appear become unthrifty (waste) and are unprofitable to keep. Ewes left on sneezeweed after signs appear will waste and die.

How to Reduce Losses

Proper range management and improved range conditions will reduce the risk of sneezeweed poisoning. Alternate grazing (on and off) of sneezeweed areas at the proper times allows time for recovery. As soon as signs of poisoning appear, remove all animals from sneezeweed-infested range for at least 14 days.

Practice open herding. Allow animals free movement. Avoid grazing sheep continuously on sneezeweed-infested rangeland.

Sneezeweed can be controlled with 2,4-D at 4 lb ae/acre.



Sneezeweed has one or several stems. Leaves are alternate, lance shaped, and smooth edged. Orange flowers with darker orange centers grow in clusters. Sneezeweed is a perennial of the sunflower family and is closely related to bitterweed and Colorado rubberweed.

Spring Parsley

Distribution of Spring Parsley



Signs of Spring Parsley Poisoning

- Sunburn of exposed areas
- Reddening and blistering of exposed areas of the body (nose, udder, external genitals)
- Scabs form after blistering
- Ewes and cows may refuse to allow young to nurse

Spring parsley *(Cymopterus watsonii)* causes a severe sunburn—or photosensitivity—in sheep and cattle. The plant also is known as cymopterus or wild carrot.

Animals do not die from eating spring parsley, but losses occur when affected ewes or cows with blistered, sore udders refuse to let their offspring nurse. Lamb losses are often high; calf losses usually are low.

Spring parsley poisoning differs from bighead poisoning, a type of



Spring parsley is a perennial that grows 4 to 6 inches tall. It gets its name from the finely divided leaves that resemble parsley. Small white or cream-colored flowers are borne in umbrellalike clusters about one inch across. The plant has a long taproot. Spring parsley is a member of the carrot family.

photosensitivity caused by horsebrush, but is similar to St. Johnswort poisoning. The poisonous substances in spring parsley are two furocoumarins—xanthotoxin and bergapten.

Plants are poisonous from early spring until they mature and dry in early summer.

Where and When Spring Parsley Grows

Spring parsley grows on well-drained soils, on rolling foothills, and with sagebrush, piñon pines, and junipers. It occurs at elevations of 4,000 to 8,000 feet. This is one of the first plants to begin growing in early spring. It flowers from late April to June and disappears by early summer.

How Spring Parsley Affects Livestock

Sheep and cattle are especially susceptible to spring parsley poisoning during March, April, and May.

Sheep are affected if they are exposed to direct sunlight after eating as little as ¼ lb of the green plant. The photosensitization varies from slight to severe; blisters form on areas of the sheep's body not covered by wool. In advanced poisoning, all of the white areas of the body may be affected.

Losses are primarily confined to lambs. On ranges where spring parsley is one of the first spring plants to emerge, a ewe may eat it soon after lambing. The udder and teats become so painful that the ewe will not allow the lamb to nurse. Newborn lambs may die of starvation or dehydration. The growth of surviving lambs is usually stunted from lack of milk.

Cattle are affected if they are exposed to direct sunlight after eating about 1 lb of the green plant. White areas on the body become "sunburned." Cows refuse to let calves nurse. In severe poisoning, all of the white areas of the body may blister, and animals may lose weight rapidly.

Sheep and cattle gradually recover after they stop eating spring parsley.

How to Reduce Losses

To reduce losses, keep sheep and cattle off ranges infested with spring parsley in early spring until adequate forage is available.

There is no known treatment for animals that are poisoned by spring parsley, but they will recover if kept in the shade and if skin lesions are treated.

Research results show that spring parsley can be controlled by

spraying plants in the bud to early bloom stages of growth with an amine salt of 2,4-D applied at the rate of 2 lb ae/acre.

St. Johnswort

Distribution of St. Johnswort



Signs and Lesions of St. Johnswort Poisoning

- Restlessness
- Scratching head with hind legs and rubbing head against solid objects
- Crouching
- Rapid pulse, increased temperature
- Redness and swelling of white-skinned areas (sunburn)
- Swollen eyelids, clouded eyes; possibly blindness
- Peeling or sloughing of affected skin
- Convulsions
- Necrotic dermatitis

St. Johnswort *(Hypericum perforatum),* or Klamath weed, is a range plant that, when grazed by livestock, results in photosensitization. Animals that eat St. Johnswort and then are exposed to direct sunlight develop severe photosensitization in white areas of the body.

Young cattle and sheep are most often affected, but almost all white-skinned cattle, sheep, and horses react to eating the plant. Recently sheared sheep are especially susceptible. Although St. Johnswort seldom kills, it can cause severe economic losses.

St. Johnswort is toxic at all stages of growth. Young tender shoots may attract animals in spring. Normally, cattle and sheep will not eat mature St. Johnswort if they have other forage. Hay containing dry St. Johnswort can cause poisoning.



St. Johnswort is a perennial that grows along roadsides and in meadows, pastures, rangelands, and waste places. In the Pacific Coast States, it may reach a height of 5 feet; in other areas, it grows 1 to 3 feet tall.

Where and When St. Johnswort Grows

St. Johnswort is an aggressive weed. It grows in old meadows, on pastures, along roadsides, on rangeland, and in waste areas. It usually is found growing on dry, gravelly or sandy soils. It may grow in dense patches or mixed among other plants. It is considered a noxious weed in many States.

How St. Johnswort Affects Livestock

When an animal eats St. Johnswort, the poisonous compound in the plant, hypericin, sensitizes the skin to sunlight. Pigments in the skin shield colored skin from the sun's rays so that only white or unpigmented areas are affected. These areas itch and become red, swollen, and sore, and the skin may peel or come off in large sheets (photosensitization).

White-skinned cattle are more susceptible to St. Johnswort poisoning than white-skinned sheep. Cattle are poisoned by St. Johnswort if they eat an amount equal to approximately 1 percent of their body weight and are then exposed to direct sunshine for 2 to 5 days. In experimental feedings, sheep fed 5 percent of their body weight showed symptoms.

Signs of poisoning usually appear 2 to 21 days after animals begin to graze St. Johnswort.



St. Johnswort is a smooth-branched, erect plant. Leaves are covered with clear, small dots that contain the toxic substances. Five-petaled flowers grow in clusters; they are orange-yellow with occasional black dots along the edges.



Photosensitization due to eating St. Johnswort.

How to Reduce Losses

At the first signs of poisoning, move affected animals out of direct sunlight to shady or dark quarters. Treat affected skin areas as directed by your veterinarian. Give animals free access to fresh water and feed.

Research results show that St. Johnswort may be controlled by applying 2,4-D at 2 to 3 lb ae/acre. Biological control with the Klamath beetle is recommended for extensive infestations. For information about using the Klamath beetle, see your county agricultural agent.

Sweet Clover

Distribution of Sweet Clover

Sweet clover grows throughout the Western United States.

Signs and Lesions of Sweet Clover Poisoning

- Signs occur 3 to 6 weeks after first exposure to spoiled plant
- Bleeding into muscles, joints, or other tissues
- Hematomas appear on neck, flanks, and hips
- Excessive bleeding during minor surgery
- Sudden death due to massive hemorrhage

Where and When Sweet Clover Grows

White sweet clover *(Melilotus alba)* and yellow sweet clover *(M. officinalis)* are biennials that can be found growing along roadsides, ditch banks, and often invading fields throughout the United States. These plants have been used extensively as a forage crop and soil builder. They are the cause of sweet clover poisoning, a severe hemorrhagic disease of cattle. Only minor losses have occurred in sheep. This bleeding condition is the result of the consumption of moldy or spoiled sweet clover hay or silage by cattle.



Yellow sweet clover.

How Sweet Clover Affects Livestock

Sweet clover contains coumarin, which is nontoxic but can be converted to the dicoumarol by molds that grow on spoiled sweet clovers. The anticoagulant dicoumarol is an antagonist to Vitamin K. Dicoumarol interferes with the synthesis in the liver of the vitamin-K-dependent clotting protein prothrombin. The likelihood of poisoning depends on the level of dicoumarol in the clover and the time over which the moldy clover is consumed by an animal.

The signs of poisoning usually occur 3 to 6 weeks after the start of feeding spoiled sweet clover. Sudden death due to massive hemorrhaging may occur. Bleeding may occur in muscles, joints, or other tissues. Hematomas usually occur in the neck, flanks, hips, and other body areas where the animal may be bumped, such as when eating in a stanchion or similar device. Excessive bleeding is associated with minor surgery.

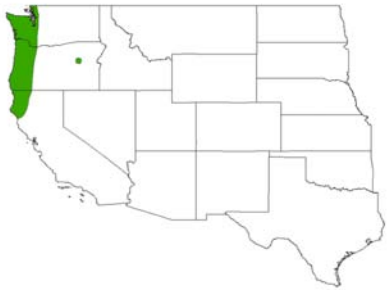
How to Reduce Losses

Treatment consists of removing the moldy clover from the diet. A blood transfusion can be given to badly poisoned animals or vitamin K can be administered.

Prevent poisoning by not feeding moldy clover to cattle, especially just prior to castration, dehorning, or any surgical procedure. Cattle grazing sweet clover also bloat and die.

Tansy Ragwort

Distribution of Tansy Ragwort



Signs and Lesions of Tansy Ragwort Poisoning

- Signs may not appear until 6 or more months after the plant is ingested
- Lethargy and loss of interest in food
- Crustiness around eyes and nose; eyes may also be red and watery, especially in bright sunlight
- There may be diarrhea or constipation
- Weakness as manifest by wobbling and dragging rear feet
- Animals may wander aimlessly and appear to be blind and may become belligerent
- Cattle may develop an unpleasant odor that has a somewhat sweetish quality
- The abdominal cavity may fill with fluid up to several gallons, and death may occur within a few days after symptoms appear
- Liver cirrhosis (necrosis and fibrosis) and bile duct proliferation
- Ascites
- Gastroenteritis



Mature tansy ragwort may grow 4 or 5 feet tall. It has sturdy stems, has a strong "weedy" odor, and produces great numbers of seed. Photo courtesy of Michael Shephard, USDA Forest Service.

Tansy ragwort *(Senecio jacobaea)* is a weedy biennial plant that infests woodlands, pastures, and hayfields of the coastal northwest United States. It is generally unpalatable to livestock and, therefore, is eaten only when other food is scarce or when it cannot be avoided as a contaminant in hay, ensilage, and lush pastures. The toxins are pyrrolizidine alkaloids that are toxic to all classes of livestock but most toxic to cattle and horses. They cause a chronic,

cirrhosis-like condition of the liver, which may lead to death of the animal several months after the tansy ragwort has been eaten. The poisoning is thus difficult to diagnose and death is often attributed to other causes. Death losses have been reported from short-term grazing of these plants.

The rapid spread of the plant has been slowed due to the development of biological control procedures.

Where and When Tansy Ragwort Grows

Tansy ragwort grows in areas with over 20 inches of rainfall annually; however, it could possibly grow in heavily irrigated locations with less rainfall. In the United States, tansy ragwort occurs principally in coastal Washington, Oregon, northern California, and in the New England States.

How Tansy Ragwort Affects Livestock

Tansy ragwort contains pyrrolizidine alkaloids, which primarily affect the liver. Cells of the liver are slowly killed or prevented from reproducing and are gradually replaced by connective tissue. Whether an animal survives depends on the number of liver cells it loses, and the length of survival depends on the rate at which it loses liver cells. Animals may survive for 6 months or longer after they have ingested a lethal amount of plant and may show no outward symptoms during this period. They may suddenly become lethargic, fill with fluid in the abdominal cavity, and die within 2 to 4 days. They may also show central nervous system effects such as impaired vision and continuous walking with a stumbling gait. The onset of symptoms may be dependent on other stresses imposed on the animal, which it cannot handle in its weakened condition. Animals eating 5 percent or more of their total daily diet of prebloom tansy ragwort for periods exceeding 20 consecutive days can be expected to die within 6 months.

How to Reduce Losses

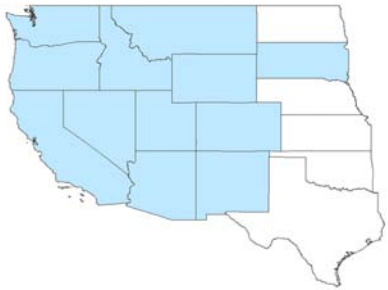
The poisoning is irreversible and the animal will die. The best way to prevent tansy ragwort poisoning is to avoid the plant. Young animals are more easily poisoned than mature animals. Pastures for horses and cattle with over 5 percent tansy ragwort should be avoided until the tansy ragwort has been controlled. Avoid hay and ensilage contaminated with tansy ragwort because animals cannot sort out the tansy ragwort.

Tansy ragwort can be controlled by cultivation. In other areas, research results show that tansy ragwort can be controlled with 2,4-D (low

volatile ester or emulsifiable acid) at 1 to 3 lb ae/acre, dicamba at 0.5 to 1 lb ae/acre, picloram at 0.25 to 0.5 lb ae/acre, or metsulfuron at 1 to 1.5 oz product/acre. Spray tansy ragwort in the rosette stage. These herbicides will injure or kill legumes. Biological control methods have been developed; contact your extension agent.

Waterhemlock

Distribution of Waterhemlock



Signs and Lesions of Waterhemlock Poisoning

- Nervousness
- Excessive salivation and frothing
- Muscle twitching
- Dilation of the pupils
- Rapid pulse
- Rapid breathing
- Tremors
- Violent convulsions, grand mal seizures
- Coma
- Death may occur as early as 15 minutes after a lethal dose is consumed
- No significant gross lesions

Waterhemlock *(Cicuta douglasii)* is the most violently toxic plant that grows in North America. Only a small amount of the toxic substance in the plant is needed to produce poisoning in livestock or in humans. The toxin, cicutoxin, acts on the central nervous system and is a violent convulsant.

Waterhemlock may be confused with poison hemlock because of their similar flowers. However, these two are different plants and cause different types of poisoning. (See Poison Hemlock chapter in this volume.)

The underground portions of the plant, especially the tuberous roots, are very toxic. People are sometimes poisoned by eating the roots, which they mistake for wild parsnip.



Waterhemlock grows in wet seepage areas such as meadows, pastures, and along the banks of streams. It reaches a height of 2 to 4 feet. The plant is a perennial in the carrot family.

In cases of waterhemlock poisoning in humans, take the affected person to the emergency room of the nearest hospital immediately.

Call poison control and seek emergency treatment immediately.

Cattle have been known to eat lethal amounts of waterhemlock in pastures having adequate forage; therefore, animals should be prevented from grazing over waterhemlock-infested areas. Animals have been poisoned by eating roots that have been brought to the surface by plowing or cleaning ditches.

The toxic substance in waterhemlock is cicutoxin, a highly poisonous unsaturated alcohol that has a strong carrot-like odor. It is found principally in the tubers but is also present in the leaves, stems, and immature seeds. Leaves and stems lose most of their toxicity as they mature.

Where and When Waterhemlock Grows

Waterhemlock is most commonly found growing in wet meadows and pastures and along stream banks. It starts growing in spring. Waterhemlock usually flowers in June or July.

How Waterhemlock Affects Livestock

Livestock usually show signs of poisoning 15 minutes to 6 hours after eating the plant. They develop violent convulsions and may die within 15 minutes to 2 hours after signs appear.



Waterhemlock has small, white flowers that grow in umbrella-like clusters. Side veins of the leaves lead to notches, not to tips at the outer margin.

How to Reduce Losses

The toxic substances act so rapidly that an affected animal can seldom be saved. Treatment consists of preventing seizures with barbiturates or tranquilizers and supporting respiration. Gastric lavage, activated charcoal, or saline cathartic may be helpful. Seek immediate medical or veterinary treatment.

To reduce losses, keep animals away from places where waterhemlock grows. Prevent waterhemlock poisoning in livestock by carefully surveying pastures and ranges at a time when the plant can be identified, and eradicate it.

The plants, which usually grow in small patches, are easy to locate.



The thick rootstalk of waterhemlock contains a number of small chambers. These hold a highly poisonous brown or straw-colored liquid that is released when the stem is broken or split. Thick, fleshy tubers and slender individual roots grow from the bottom of the rootstalk.

They can be eradicated by spraying or grubbing. Actively growing plants can be controlled with 2,4-D at 2 lb ae/acre. Repeat spray treatments until eradication is complete. The stems and leaves of waterhemlock increase in palatability immediately after being sprayed with herbicide. Therefore, keep animals away from treated plants for 3 weeks after spraying. Most losses occur early in the spring or after the plants have been sprayed with 2,4-D.

Note: If grubbing the waterhemlock, use gloves and be careful to get all of the plant, including roots. Gather and burn every part.

Yellow Star Thistle and Russian Knapweed

Distribution of Yellow Star Thistle and Russian Knapweed

Both plants grow throughout the Western United States.

Signs and Lesions of Poisoning

- Clinical signs occur after the horse has eaten large quantities of either plant for 30-60 days
- Chewing disease (dysfunction of facial, mouth, and throat muscles)
- Facial paralysis
- Depression
- Dehydration and malnutrition
- Incoordination
- Muscle tremors
- Irreversible necrosis of the brain

How and When Yellow Star Thistle and Russian Knapweed Grow

Yellow star thistle *(Centaurea solstitialis)* is an annual weed growing throughout much of the Eastern, Southern, and Western United States. Russian knapweed *(C. repens)* is an invasive perennial weed that spreads via seed and rhizomes. It is abundant throughout the intermountain west and the northern plains. They are both characterized as noxious plants.

How Yellow Star Thistle and Russian Knapweed Affect Livestock

Yellow star thistle and Russian knapweed are toxic primarily to horses. They are grazed only after other forage is gone.

It takes weeks to months of exposure to large amounts of yellow star thistle or Russian knapweed before clinical signs develop. Horses must consume 86 to 200 percent of their weight in green plant.



Yellow star thistle is an annual weed that grows up to 12 inches tall. Leaves are covered with cottony hair, and the yellow flowers are surrounded by the characteristic stiff yellow spines up to an inch long.

Horses develop chewing disease; signs of poisoning include twitching of lips, tongue flicking, involuntary chewing motion, drowsiness, weight loss, and emaciation. Eating and drinking are severely impaired. Prehension and mastication are affected, but swallowing is unaffected. The mouth is often held partly open with the tongue partially protruding.

Most poisoning cases are in younger horses.

How to Reduce Losses

There is no effective treatment for poisoning. The necrosis in the brain is irreversible.

The best prevention is to control the plant and keep pastures where horses graze in good condition, provide adequate good quality feed, and avoid overgrazing.



Russian knapweed is a perennial that grows 1 to 3 feet tall. The flowers are similar to some thistles and are lavender to white. There are no spines around the flowers.

Yew *(Taxus)*

Signs and Lesions of Yew Poisoning

- Gastric distress
- Diarrhea
- Vomiting
- Tremors
- Dyspnea
- Dilated pupils
- Respiratory difficulties
- Weakness
- Fatigue
- Collapse
- Coma
- Convulsions
- Bradycardia
- Circulatory failure
- Death (may be so rapid that signs of poisoning may not have time to develop)
- Survival is rare
- Moderate irritation of the upper digestive tract

Yews (*Taxus* spp.) are shrubs that are used extensively in landscaping around homes and other buildings. Yews have long, slender, alternate dark-glossy-green flat needles and bright-scarlet fleshy cup-shaped fruits. In some areas, the yew is considered the most toxic tree or shrub and is probably one of the most toxic of all poisonous plants.

Yew is toxic to all classes of livestock, large game animals, and humans. Poisonings usually occur when these plants are trimmed and the trimmings are fed to animals. Children are poisoned when they eat or chew on the scarlet fruit.



Closeup of leaves of Japanese yew *(Taxus cuspidata).* Japanese yew is commonly used in horticulture.

Foliage, bark, and seeds are all toxic. The foliage is readily eaten by livestock.

Where and When Yew Grows

There are about 40 species of *Taxus* that can be found growing throughout the United States. Four of these are commonly used in horticulture.

How Yew Affects Animals

Green foliage is toxic to horses and other monogastric animals at about 0.1 percent of body weight. In ruminants, about 0.5 percent of body weight of foliage is toxic. The toxin is an alkaloid, taxine. The compound is a cardiac depressant causing the heart to stop.

How to Reduce Losses

Do not allow animals access to this group of plants. Control is mostly a matter of horticulture and landscaping decisions to prevent grazing animals from access. The general public should be educated not to put clippings of those plants where animals have access.

Other Poisonous Plants



Cocklebur.

Cocklebur *(Xanthium strumarium)* is an annual plant that grows in dry lakebeds, river bottoms, and disturbed and flooded areas of fields, pastures, and roadways after water disappears. Poisoning occurs primarily in cattle, sheep, horses, and swine when the plant is eaten in the cotyledon stage (seedling). Signs of poisoning include rapid, weak pulse, labored breathing, nausea, vomiting, and spasmodic contractions of the legs and neck muscles.

Desert baileya *(Baileya multiradiata)* is a small annual that grows in dry, sandy and gravelly areas from Texas to southern California. Sheep are sometimes poisoned on this plant. Signs of poisoning include depression, anorexia, and slobbering of a green material from the mouth.

Drymary or inkweed *(Drymaria pachyphylla)* is a short-lived annual that grows close to the ground on alkaline clay soil in western Texas, southern New Mexico, and Arizona. Poisoning occurs primarily in cattle. Signs of poisoning include loss of appetite, diarrhea, restlessness, arched back, depression, coma, and death. Drymary is exceedingly distasteful to livestock.

Horsetail (*Equisetum* spp.) is an herbaceous perennial. It is rush-like in appearance with hollow, jointed stems. This group of plants is common throughout the United States. It is toxic primarily to horses and cattle.



Horsetail.

Jimsonweed or thornapple (*Datura* spp.) is a large annual herb with worldwide distribution. It is toxic to all classes of livestock as well as humans. Signs of poisoning include dilated pupils, subnormal

temperature, restlessness, gastric stasis, bloat, muscular twitching, incoordination, paralysis, rapid heart rate, and respiratory paralysis.

Tansy mustard *(Descurainia pinnata)* is an annual weed that grows on dry, sandy soils in arid areas. Intoxication occurs primarily in cattle only after large amounts of this plant have been eaten over extended periods. Signs of poisoning include partial or complete blindness, inability to use the tongue, and aimless wandering.

Hundreds of plants are poisonous to livestock. Many are toxic at all times, whereas many others are toxic only under certain conditions. Livestock producers are urged to become acquainted with the plants on their ranges and pastures that are potentially dangerous to their livestock. Help in identifying these plants can usually be obtained from the local county agricultural agent. Help with poisonous plant problems can also be obtained from local veterinarians.



Jimsonweed.



Tansy mustard.

Noxious Weeds

A noxious weed is any plant that is designated by a Federal, State, or county government as injurious to public health, agriculture, recreation, wildlife, or property and must be controlled by the legal owner of the land on which it appears. There are several poisonous plants that have also been classified as noxious weeds. The following is a partial list:

- Mustards: white top, hoary crest, perennial pepperweed. Tansy mustard has not been declared noxious as yet.
- Knapweeds: diffuse, Russian, spotted, squarrose, yellow star thistle
- Leafy spurge
- Goatsrue
- Houndstongue
- St. Johnswort
- Nightshades: sliver leaf, buffalo bur, black nightshade
- Kochia weed
- Milkweeds
- Johnson grass
- Poison hemlock

Under ordinary conditions, these plants are seldom grazed. However, poisoning can and does occur under stress conditions such as on overgrazed pastures or after a frost. Noxious weeds compete with useful forages such as grasses, some legumes, etc. for space, nutrients, and moisture.

Some noxious weeds can be found growing in crops and may be harvested with hay or small grains. Care must be taken when these crops are fed to livestock.

Most of these plants are aggressive and spread rapidly. It is important to monitor all range and pasture land for poisonous and noxious plants to prevent infestations and manage these plants. Early treatment to control these plants is the most effective. The spread of noxious weeds may signal the decline of entire ecosystems. They severely affect the biodiversity of natural areas and cause economic losses to those relying on range resources.