Poisonous Plants
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PLANTS that poison livestock can grow anyplace, but they are a serious problem mainly in the western grazing areas where overgrazing has impaired or destroyed the good but less sturdy forage plants.

The poisonous plants usually grow among forage plants and so are available to grazing animals. Only a few are agreeable to the taste, however, and animals avoid the toxic plants unless there is a shortage of feed and better vegetation.

Overgrazing is a major factor in livestock losses from poisonous plants. Overgrazing has increased in some sections because moisture deficiencies have reduced the growth of forage. Hungry animals feed on whatever is available.

Sometimes harmful plants are harvested with the hay and their seeds become mixed with grain. Then animals can hardly separate the good feed from the bad. Livestock owners have considerable control over conditions conducive to poisoning by plants.

Some plants contain acute poisons and produce visible symptoms or death soon after they are eaten. Others may be eaten for some time before any noticeable effects are apparent. Most poisonous plants may be eaten for a considerable time with little or no ill effects.

It is not easy to control poisonous plants in pastures and on the range. Plowing and cultivation usually eradicate them, but in some areas, such as the western grazing lands, these methods are not feasible because of expense, the time they take, and the amount of benefit. Sometimes the plants are pulled up, grubbed out, or killed with herbicides, but that is slow and expensive even under favorable conditions.

Many poisonous plants are distributed so widely over grazing areas of limited value in the Western States that the cost of eradication would be much greater than the benefits. A more practical procedure is to remove the toxic plants from limited areas, including trails and watering places. The method of eradication used would depend on the character and growth habits of the species, the other plants with which the toxic plants grow, and the type of soil.

When eradication is impractical, a system of range and pasture management can be worked out to permit the use of the forage crops without excessive livestock losses. That is largely a matter of wise control of grazing.

Poisonous plants often are the first to start growth in the spring and may harm livestock if too early grazing is practiced. If pastures and ranges are stocked to full capacity in normal years and the number of livestock is not reduced in drought years, the usual forage can be supplemented with other roughage or feed in order to avoid injury to the existing vegetation and losses of animals from the poisonous plants.

Another aspect of control is that one species of animals may avoid certain plants, or one species may not be injured by plants that poison another species. Losses then may be avoided by permitting only the animals least affected to graze them.

Some examples: Horses seldom eat cyanogenetic plants—plants that can produce hydrocyanic acid—in toxic amounts, but cattle and sheep frequently do eat such plants and are poisoned by them. Pastures that contain cyanogenetic plants should be used by horses in preference to cattle and sheep. Horses are more frequently
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poisoned by ragworts or groundsels, which are species of Senecio, than are cattle or sheep, and sheep are less susceptible to their poisoning than are cattle. Pastures containing ragworts should be used by sheep in preference to cattle or horses. Cattle often are poisoned on larkspur. Sheep, however, can consume large amounts of larkspur without being poisoned but with apparent benefit. Only under abnormal conditions are sheep ever poisoned by larkspur on the range. Horses never eat enough of the larkspur to produce any ill effects. Pastures containing larkspur should be used by sheep and horses but not by cattle.

Another aspect: Some plants, such as the sorghums, become toxic under certain conditions. Sorghum in the mature stage does not contain any appreciable amount of potential hydrocyanic acid, but the young plants, or suckers (young branches from the roots), of mature sorghum may contain very much potential hydrocyanic acid. Arrowgrass grown on water-covered or very wet soil is far less poisonous than arrowgrass that continues to grow on soil that has dried after the water has receded. Oil meal made from immature flaxseed is more apt to be poisonous than oil meal made from thoroughly ripe flaxseed.

TREATMENT OF ANIMALS poisoned by plants usually is unsatisfactory and useless, because most of the damage may have been done by the time the poisoning is discovered.

Certain feeds or medicines that have some preventive value are described later in connection with plants that contain selenium. Good care and a symptomatic treatment (treatment that will reduce symptoms) will save some poisoned animals. The outcome in each case depends largely on the amount of toxic material that has been eaten and assimilated. Treatment usually is directed toward eliminating any of the toxic substance that still remains in the digestive tract. In chronic poisoning, recovery may follow a change of feed, especially if green feed is available; good care, with plenty of water and the right kind of feed, will do much to hasten recovery. (Later we suggest treatments for three types of poisoning.)

It is well to know something of the chemistry of the poisonous elements, especially those in the compounds that are physiologically active. Knowing whether the substance is an alkaloid, a glucoside, or something else makes it possible to determine by laboratory examination whether a sample of the plant is potentially deadly and to detect the poison in the tissues of animals so that a diagnosis can be made in obscure or doubtful cases.

ALKALOIDS are substances that are like the alkalies. An alkaloid turns red litmus paper blue, reacts with an acid to form a salt, and has some other properties that solutions of the alkali metals have, such as soda or potash.

The alkaloids are organic substances that contain nitrogen. Some of the alkaloids are quite stable and may be detected by a chemist in the plant tissues or the tissues of poisoned animals.

It is important for the farmer or student to know that certain groups or families of plants, such as the legume, lily, buttercup, potato, and some other families, are more likely to contain alkaloids than some other groups.

Some of the groups do not so metabolize their nitrogen as to yield alkaloids. The large and important mint family seldom contains any alkaloidal plants. The aster, or composite, family is another nonalkaloidal group, although there is an outstanding exception in the ragworts, or groundsel, of that family. The grasses are not characteristically alkaloid bearing, although darnel yields loliline, which is a true base.

A well-known alkaloid is strychnine, which is obtained from poisonnut (Strychnos species), a member of the logania family, whose members are mostly tropical plants. Morphine is an alkaloid that can be separated from...
the drug opium (an extract from certain kinds of poppy). Other well-known alkaloids are atropine, nicotine, and solanine, from the potato family; aconitine and several alkaloids of the larkspur group, from the buttercup family; zygodine and colchicine, from the lily family; and physostigmine, lupinine, and other lupine alkaloids, from the legume, or pea, family. Names of alkaloids usually end in “-inc.”

Alkaloids occur in many stock-poisoning plants. Among them are the larkspurs, lupines, deathcamas, ground-sels, Dutchmans-breeches and other plants of the Dutchmans-breeches family, poison-hemlock, wildtobaccos, crotolarias, and African-rue.

Larkspurs seem to attract cattle because of the pleasant acidity of their leaves, which is refreshing in hot weather. They are one of the few poisonous plants that are palatable. The larkspur alkaloids are complex.

*Delphinium barbeyi*, one of the tall larkspurs, and *D. menziesi*, a low species, are the main poisonous species, although most of the species of *Delphinium* are dangerous, especially when the plants are small or when they are available in quantities. *D. occidentale*, a tall and comparatively nontoxic larkspur, resembles *D. barbeyi* and often is mistaken for it. *D. occidentale* contains about 1 percent of an alkaloid.

A treatment recommended for cattle poisoned by larkspur during drives and roundups is a subcutaneous injection of a mixture of 1 grain of physostigmine salicylate (also called eserine), 2 grains of pilocarpine hydrochloride, and one-half grain of strychnine sulfate, thoroughly dissolved in sterile water. This formula applies to an animal weighing 500 to 600 pounds. For a larger steer or cow of 1,000 pounds or more, the dose should be twice that amount. The medicine relieves constipation and stimulates respiration. The materials can be obtained from a druggist.

*Lupines*, also known as bluebonnets and by other local names, belong to the legume family. Some species are harmless, at least at some stages of growth, and are excellent feed for grazing animals. Others are dangerous at certain times. Some are toxic at any stage of growth.

The alkaloids in lupines have the peculiarity that slight alterations in the molecular structure may convert the toxic alkaloid into a comparatively nonpoisonous substance, and vice versa. The alterations may take place in the plants and may account for the variations in the toxicity of the growing plants.

Reports from Germany in the 1860’s attributed great losses to the yellow lupine (*Lupinus luteus*) and other lupines. Actually, however, the losses were due to molds. In this country losses from lupines have been due to alkaloidal poisoning.

Deathcamas (species of *Zigadenus*) are another group of alkaloid-containing plants. They are grasslike and not conspicuous until they bloom. They are also known as the poison scgo, mystery-grass, lobelia, soap plant, alkaligrass, waterlily, squirrel food, wild onion, and hog’s potato.

Species of *Senecios*, or groundsels, have caused a number of losses in livestock in Texas and other States.

Among the other alkaloidal plants there are many in the fumitory, or Dutchmans-breeches, family (*Fumariaceae*). Two examples are fitweed (*Corydalis caseana*) and Dutchmans-breeches (*Dicentra cucullaria*, sometimes called *Bikukulla cucullaria*). In the carrot family is poisonhemlock or spotted hemlock (*Conium maculatum*). One member of the potato, or nightshade, family is wildtobacco (*Nicotiana attenuata*). A member of the legume family is crotalaria or rattlesnake (*Crotalaria sagittalis*). A member of the caltrop family is *Peganum harmala*.

Glucosides contain various sugars that are combined in the molecule and can be split out by acids and then detected by chemical reagents. The
breaking apart of a glucoside molecule is a complicated technical process.

The definition of a glucoside in a medical dictionary may be helpful: "Chemically a glucoside may be defined as a carbohydrate compound formed by a union of a sugar with a non-sugar accompanied by the elimination of water." A chemist refers to the non-sugar as an aglycone. It can be seen there is some comparison between the structure of a glucoside and the structure of a salt, but the organic components of a glucoside are more confusing than what we consider the simple inorganic components of a salt.

Flax contains the glucoside lina marin, which, when hydrolized (water added), yields acetone (a ketone), glucose (a sugar), and hydrocyanic acid (the poisonous principle).

In a similar way cherry seeds, or pits, contain the glucoside amygdalin, which, when hydrolized, yields benzaldehyde (an aldehyde), glucose, and hydrocyanic acid.

Esculin is a phenolic glucoside from the horsechestnut. Dhurrin is a cyanogenetic glucoside from sorghum. Githagin is a glucoside called a saponin from corncockle. The characteristic ending of names of glucosides is "-in."

A chemist often can readily detect a glucoside or its aglycone, either or both of which may be poisonous. The plants that owe their poisonous properties to such substances are many and diverse. Some form soapy solutions with water and therefore have been called saponins. Others are called cyanogenetic glucosides because they develop hydrocyanic acid, or prussic acid, in certain circumstances. Others are outside these two categories.

The cyanogenetic glucosides have been responsible for large losses of livestock in many sections of the United States. Many plants have cyanogenetic characteristics, but usually only a few are dangerous to livestock.

The more important of the cyanogenetic plants include wild chokecherry (Prunus virginiana, P. virginiana var. melanocarpa, and P. virginiana var. demissa); sorghum (Sorghum vulgare); Sudangrass (Sorghum vulgare var. sudanense); Johnsonsgrass (Sorghum halepense); flax (Linum usitatissimum); and arrowgrass (Triglochin maritima and T. palustris).

Wild chokecherry and other wildcherries are always a potential danger in spring and early summer for animals that are unusually hungry, but like most poisonous plants they may be eaten in small amounts without causing injury. The leaves of the wildcherries contain prunasin, a glucoside. In the seeds this is associated with another cyanogenetic glucoside, amygdalin, which also occurs in bitter almonds and in peach kernels.

The sorghums—Sorghum vulgare, Johnsonsgrass, and Sudangrass—have a cyanogenetic glucoside known as dhurrin. Sorghums grown under ordinary conditions are considered good feed, but when the normal growth has been interrupted by drought, frost, trampling, or other causes, hydrocyanic acid may develop to a point where the plants become toxic.

The amount of hydrocyanic acid that the sorghums can develop varies considerably according to conditions and varieties. Young and second-growth plants can develop larger amounts than older plants. The amount of hydrocyanic acid diminishes more or less regularly as the plant matures. When the seed heads are well formed, the plants generally cannot cause fatal poisoning. A possibility always exists that young suckers or branches or second-growth plants grow in the field as some plants are maturing; poisoning may result if livestock are allowed access to them. The stubble that sprouts after harvest is high in hydrocyanic acid.

Arrowgrass growing where there is plenty of water is only slightly toxic, but under drought conditions it may become toxic.

When cyanogenetic plants are made into hay, most of the hydrocyanic acid supposedly volatilizes or evaporates. One has to be careful, however, for occasionally the hay may retain enough
acid to make it dangerous, especially for hungry animals that may overeat.

Wild flax and wild lima beans contain a cyanogenetic glucoside known as linamarin or phaseolunatin. Wild lima beans, imported into France as feed for cavalry horses during the First World War, caused several outbreaks of poisoning. Cultivated lima beans usually are not dangerous in the United States, because they contain little or no hydrocyanic acid. One or two cases of supposed hydrocyanic acid poisoning in cattle as a result of feeding ensilage made from lima beans have been reported.

A number of factors influence the absorption of hydrocyanic acid from plants in the digestive tract, but the rate at which such plants are eaten and the amount of food already in the stomach are probably the most important.

Hydrocyanic acid acts so rapidly that there is little chance for remedial treatment. If the victim can be reached in time, however, a combination of sodium thiosulfate and sodium nitrite injected into the lining of the abdomen or into the veins is effective against doses of hydrocyanic acid up to three minimum lethal doses. (It is considered that the minimum lethal dose of hydrocyanic acid is 1 milligram per pound of animal—a 100-pound animal may be killed with 100 mg. of HCN.)

Poisoning by cyanogenetic plants may cause rapid respiration, depression, stupor, convulsions, cyanosis of the mucous membranes (which causes them to turn blue), and paralysis. Death may follow in a few minutes or several hours.

When one animal in a group pasturing on plants that might be dangerous shows symptoms of poisoning, the others should be removed promptly from the pasture.

The saponin-containing plants include some that are highly dangerous.

The seeds of corncockle (Agrostemma githago), a troublesome weed, contain a mixture of saponins. The seeds sometimes get into wheat and make it dangerous to feed to poultry and livestock.

Rubberweeds, both the bitter rubberweed, or bitter actinea (Hymenoxys odorata), an annual in the Southwest, and Colorado rubberweed, or pingue (H. richardsoni), a perennial of the Rocky Mountain area, contain saponins. Both rubberweeds are poisonous to stock. Sheep especially have been poisoned by them.

The nightshade group, particularly the genus Solanum, may have a saponin (solanine) that contains basic nitrogen and so acts also as an alkaloid. Solanine occurs in sprouted white potatoes (which belong to the nightshade family) and has at times caused poisoning of persons who have eaten the tubers. The toxic effects of bullnettle (Solanum carolinense) and bittersweet (S. dulcamara)—and, somewhat doubtfully, of black nightshade (S. nigrum)—are attributed to solanine.

Resinoids, a large group of somewhat indefinite substances, occur in some plants. One of the most common is andromedotoxin, a poison, in ericaceous plants. A complex substance, it is considered the active principle in some of the rhododendrons and azaleas and possibly the laurels. We have no precise knowledge of the peculiar structure of the molecule involved in the toxic action. Waterhemlock (Cicuta vagans), an extremely poisonous plant, contains a poisonous resinoid.

Milkweeds (Asclepias species) contain resinoids. Little is known about the chemistry of the milkweeds, but some investigators have concluded that a resinous substance extracted from milkweeds produced typical cases of range poisoning when fed to animals. They did, however, find other substances of a slightly toxic nature.

Among the species that have been studied are Asclepias subverticillata, the whorled milkweed of the Southwest; A. eriocarpa, the broadleaf (wooly-pod) milkweed of California; A. fascicularis, the whorled milkweed of the Western States; and A. pumila and A. verticillata.
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var. geyeri, the whorled milkweeds of the Central and Eastern States. The toxicity of the different species varies greatly and ranges approximately in the order in which we list them.

Losses from the whorled milkweed (*A. subverticillata*) of the Southwest can be severe. Once, near Hotchkiss, Colo., 400 sheep died within 24 hours after feeding on the plant. In 1917 a loss of 736 sheep out of a band of 1,000 near Cortez, Colo., was traced to the same species. Next to waterhemlock, this whorled milkweed has been considered the most toxic plant in the United States, but another species of milkweed, *A. labriformis*, is much worse. The toxicity of *A. labriformis* is highest during the early part of the growing season, when about 1 ounce of its green leaves can kill a sheep weighing 100 pounds. The lethal amount for cattle is even less in proportion to weight.

The plant loses some of its toxicity at maturity, but the dry leaves retain enough of the toxic substance to cause heavy losses of livestock in winter and early spring.

Apparently *A. labriformis* grows only in eastern Utah, mostly in sandy places and along watercourses. This species, like other poisonous milkweeds, is not palatable. Animals eat it only when they have no other feed. On overgrazed ranges and trails it is a hazard at all times.

We know of no remedy for milkweed poisoning. If losses are to be avoided, hungry animals must be kept away from places in which poisonous milkweeds grow or the plants should be eradicated.

The symptoms of milkweed poisoning are uneasiness; some lack of muscular coordination; rapid, shallow, noisy respiration due to edema (swellings filled with fluid) of the lungs; spasms; and considerable struggling. Death may result from respiratory failure. The kidneys usually are affected more than the other organs.

Oxalic acid occurs in sorrels, docks, halogeton (*Haloegeton glomeratus*), greasewood (*Sarcobatus vermiculatus*), and other plants. The oxalic content of the dried leaves and fine stems of greasewood may exceed 10 percent in late summer and early fall. Halogeton may contain 18 to 20 percent.

Cattle and sheep are susceptible to poisoning by greasewood and halogeton. Most of the losses have occurred in bands of hungry sheep that have been grazed in places where these plants predominate and desirable forage is scarce. Animals may eat small amounts of the plants daily over a long period of time without apparent injury. A sheep that eats them slowly all day with other forage may safely consume twice the amount that would be toxic if eaten in an hour without other forage. The animals can eliminate the oxalates fairly rapidly and thus prevent the accumulations of toxic levels within the body. With good management of ranges and livestock, especially during trailing and following shipping, losses from poisoning by greasewood and halogeton can be kept down.

Books giving antidotes to poisons recommend either chalk (which is mostly carbonate of lime or calcium carbonate), or limewater as an antidote for oxalic acid poisoning. The lime forms an insoluble compound (salt of lime) called oxalate of lime (calcium oxalate). When formed in the intestines, the compound would be eliminated as an insoluble compound with the feces instead of being absorbed into the blood stream as might be the case if it were either oxalic acid or oxalates (salts) of other elements prevalent in most foods and vegetation, such as sodium (forms oxalate of soda called sodium oxalate) and potassium (forms oxalate of potassium called potassium oxalate). Both are water-soluble salts of oxalic acid.

In one experiment, however, with a sheep poisoned on halogeton, we gave the animal some chemically pure calcium carbonate as an antidote. It did not prove so effective as in another experiment, when the antidote we gave was the leaves of alfalfa hay. Possibly
the lime in the alfalfa hay was more soluble and consequently more reactive with the oxalic acid (to form an insoluble salt) than was the lime in the calcium carbonate.

Tremetol produces a disease known as trembles in livestock and milksickness in people. Tremetol, an oily alcohol, can be isolated from white snakeroot (Eupatorium rugosum), which grows in the Eastern States, and from rayless goldenrod or jimmyweed (Apolopappus heterophyllus), which grows in the Southwestern States.

Cases of trembles or milksickness have been reported in Virginia, North Carolina, South Carolina, Georgia, Maryland, Missouri, and Michigan, where white snakeroot grows, and in Texas, New Mexico, and Arizona, where rayless goldenrod grows.

Milksickness has been caused by milk and by butter made from milk of cows that had fed on tremetol-containing plants. The isolation of tremetol and the production of trembles by feeding poisoned butter to sheep completed the chain of evidence linking the plants to milksickness in human beings.

NITRATE POISONING in animals has been reported to be caused by animals eating oat hay, Russian-thistle (Salsola pestifer), tarweed (Amsinckia intermedia), redroot pigweed (Amaranthus retroflexus), puncturevine (Tribulus terrestris), beet tops (Beta vulgaris), white ragweed (Fraseria discolor), variegated thistle (Silybum marianum), and many others.

Selenium is a nonmetallic element. Chemically it resembles sulfur, however, the physiological action of selenium is different from that of sulfur.

Selenium-bearing plants sometimes cause considerable damage. Some plants known as indicator plants, which seem to require selenium in the soil for their growth, such as princes-plume (Stanleya species), woody aster (Xylorrhiza parryi), and some species of Astragalus, contain considerable selenium and poison the animals that eat them in large quantities. Some crop plants and others tolerate selenium in the soil and may absorb selenium and metabolize it in their tissues in quantities that would make them poisonous to livestock if eaten in sufficient quantity.

Experimental work with laboratory animals indicates the resistance of animals to selenium poisoning is increased by a high-protein diet.

Research workers in Wyoming learned that increasing the protein and vitamin A intake of sheep improves the quality of the diet to the extent that the poisonous effects of selenium are lessened. If the selenium intake was reduced or stopped and a high protein diet was continued, the animals recovered from selenium poisoning within a short time and the selenium was completely eliminated in about 30 days. If the protein content of the diet was low, however, the recovery was slow or the animals died. It was found also that iodine increases the susceptibility of animals to selenium poisoning. The recommendation was made that in places where selenium occurs, salt that contains no iodine should be used.

Arsenic compounds, especially compounds with sodium, tend to protect animals against the toxic action of selenium. In one experiment, a group of steers that were given 25 parts per million of arsenic in the salt made better gains and sold for higher prices than others.

Selenium poisoning is mentioned in another chapter (p. 117).

Some compounds of molybdenum (a heavy metal that belongs to the chromium group), are soluble and may be absorbed by plants so as to make them harmful to stock that eat them. We discuss these compounds under the term “molybdenum.”

A discussion of molybdenum poisoning appears in a previous chapter (p. 115).

English stockmen have known for a long time that certain pastures, which they called “teart,” were harmful to
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stock. The teartness was especially prevalent in Somerset, and they became known as the "teart pastures of Somerset." The outstanding symptom of cattle grazing teart pastures was scouring, or diarrhea. Affected animals also might show emaciation, anemia, stiffening or enlargement of the joints, dry skin, and loss of coat color or hair. Black tended to become gray, red became tan, and white became a light tan. This discoloration was first noticed around the eyes and the ears. Young, growing cattle were more susceptible to the teartness of pastures than old cattle.

Several possibilities were mentioned to explain the disease, but the real cause of the trouble was shown in 1938 to be an excess of molybdenum in the vegetation. Studies were started later in Europe, Australia, the islands of the Pacific, and the United States.

The first recorded molybdenosis (a condition of stock effected by excess molybdenum in their feed) in the United States was from an area of the San Joaquin Valley of California by J. W. Britton and H. Goss less than 10 years after the discovery in England.

According to the early reports on molybdenosis, any pasture whose vegetation contained 20 p. p. m. (parts per million) of molybdenum or more was a dangerous one. Recent studies show that a certain amount of molybdenum in the vegetation or in the soil is not the only factor to be considered as the cause of the disease or the toxicity of the pastures. The acidity of the soil, the species of plants, the stage of growth of the plants, the part of the plant eaten, the amount of molybdenum in the soil, the condition of the animals and the character of any supplementary food they eat all contribute to the poisoning of livestock.

Generally speaking, dry hay from a high-molybdenum area is less likely to cause scouring than green, succulent pastures in the same area. Some alfalfa hay occasionally has caused severe symptoms of molybdenum, but hay usually loses its toxicity with storage. Legumes in general absorb more molybdenum than nonlegumes. The greatest concentration is found in most instances in the blades of the leaves and in the actively growing parts of the plant. That fact may account for the beneficial effect of feeding roughages to reduce excessive scouring to which cattle are subject when they graze succulent leguminous pastures.

Molybdenum is widely distributed in the soils of many places in the United States. Most of the reports of trouble that we have had have come from San Joaquin Valley, although the molybdenum in the troublesome soils there is not always high. It may range from 1.5 to 10 p. p. m., and most of it is soluble. The high solubility of those soils is due mainly to their alkaline reaction, for water soluble molybdenum has been greatly reduced by acidification. In this respect molybdenum behaves as selenium does, and the opposite of manganese, boron, zinc, and copper. Soils that are overlimed become alkaline and may produce toxic vegetation even in humid regions if the soil contains enough molybdenum.

The toxicity of molybdenum to animals seems to be due at least partly to an imbalance of nutrients or minerals in the animals' system. Research in Australia revealed that the symptoms of animals with excess molybdenum resemble the symptoms of a disease known as peat scours, which affects animals pastured on peat soil and occurs in animals that have a deficiency of copper. Symptoms of copper deficiency and molybdenosis are relieved by giving animals soluble compounds of copper.

Investigators in England considered that teartness or molybdenosis was a disease only of cattle. Early investigators in the United States recognized that sheep also may be affected. It is now known that, although it is primarily a disease of ruminants and that field cases of reported molybdenum poisoning seem to be confined to cattle and sheep, horses, swine, pou-
try, and laboratory animals may be poisoned if they get too much molybdenum. Nonruminant animals are relatively immune to poisoning, however.

Locoweeds, copperweed, paperflower, horsebrush, bracken fern, and eupatorium are examples of plants that contain unknown and miscellaneous poisons.

The locoweeds (species of *Astragalus* and *Oxytropis*) were so important that at one time the term “locoed” was used synonymously with “poisoned,” especially in the Western States.

The locoweeds have been analyzed extensively in the past 80 years. White loco (*Oxytropis lamberti*) was studied by James F. Couch, who found that the active principle belongs to none of the recognized groups of poisonous compounds but appears to be a new type.

Some members of the genus *Astragalus* are nontoxic. Others produce poisoning but not the true locoism. Examples of the first group include the red locoweed (*A. drummondii*) and *A. tenellus*. In the second group is *A. tetrapterus*. Nothing is known about their chemistry.

Another group of the genus is poisonous not because of any constituent normally produced by the plant itself but because of selenium, which the plants may take up from the soil in the form of a compound and accumulate in dangerous quantities.

Copperweed (*Oxytenia acerosa*) grows over much of the Colorado Basin along streams and seepage areas where the soil is strongly alkaline. Copperweed only recently was proved to be poisonous. It affects cattle and sheep. The plant increases in toxicity as it reaches maturity. Poisoning usually occurs in the fall when the cattle are being driven down from the summer ranges over trails that are overgrazed and contain little or no desirable forage. Enough copperweed, which ordinarily is quite unpalatable, may be eaten then to cause poisoning. Less than one-half pound of green leaves to 100 pounds of animal weight is lethal for cattle.

Most animals that show severe symptoms die. Losses may be prevented by providing suitable feed along the trails and on the ranges so that animals are not compelled to eat the toxic plants.

In copperweed poisoning, the liver seems to be affected first and the kidneys later. The symptoms usually are marked depression, weakness, and coma. Death follows without much struggling.

Occasionally an animal shows considerable nervousness and excitement. Frequently animals are found dead in the morning without having shown symptoms the previous evening.

Greenstem paperflower (*Psilostrophe sparsiflora*) has become a problem of sheepmen in northern Arizona. It has been spreading over a wider area, so that it is becoming increasingly difficult to utilize the range. Two other species of *Psilostrophe* in Texas have caused losses of sheep.

The paperflower grows in semiarid regions that are used largely as intermediate and winter ranges. Overgrazing may have been responsible for its spread. Very likely cattle do not eat enough of it on the range to be poisoned because the paperflower is not palatable. In places where the forage consists largely of plants low in palatability, it is necessary to move the sheep to uninfested areas as soon as loss starts in order to prevent further losses. Some deaths occur even then after the animals are moved.

Because *P. sparsiflora* is abundant over a rather wide region, eradication, even if it could be accomplished, would not be economically practicable, and the control of sheep losses in the region must depend on improvement in range conditions. That is, the range area should be widened so there would be larger grazing area for each sheep.

In paperflower poisoning, the kidneys appear to be affected primarily. Poisoning is usually the result of feeding for several days on the plant. Consequently the symptoms may develop
rather slowly. The principal symptoms are loss of appetite, depression, and weakness. Death follows a week or more of partial coma.

Horsebrush is the name of two related plants. One is called littleleaf horsebrush, spring rabbitbrush, or coal oil brush (\textit{Tetradymia glabrata}) and has been known for many years as a cause of heavy losses of sheep. It is common in dry areas of western Utah, Nevada, eastern California, southern Oregon, and southwestern Idaho.

Spineless horsebrush (\textit{T. canescens}) is poisonous to sheep, but rather large amounts are necessary to produce poisoning. It grows to a limited extent in the same areas as those occupied by littleleaf horsebrush. It is found in greater abundance at the higher elevations and extends through central Utah to southwestern Wyoming, southwestern Montana, eastern and southern Idaho, eastern Oregon, eastern Washington, eastern California, and northern New Mexico.

The most toxic stage of both plants is during their active growth period—April and May for littleleaf horsebrush and May and June and early July for spineless horsebrush.

In sheep the horsebrushes cause a disease known as bighead or swellhead. Bighead is essentially a range disease and has been prevalent in many areas of the intermountain region since the early days of the sheep industry. It probably has caused a greater financial loss to the sheep owners in the affected areas than any other disease.

Bighead apparently has two separate stages—first, a toxic condition produced by the plants and affecting primarily the liver; second, a swelling, or edema, which affects principally the head. This edema affects only animals with white or light-colored skins and is the result of sensitiveness to light. Either white sheep or black sheep may be affected by the toxic substance in the plants, but the pigment in the skin of black animals protects them from the effects of the light rays, so that photosensitivity does not occur. The same is true of sheep kept in total darkness after eating \textit{Tetradymia}. The exact nature of the substance causing photosensitization and the manner in which it gets into the circulation near the body surface are not known definitely.

The areas where bighead occurs correspond with the distribution of the species of \textit{Tetradymia}, but although the plants are the main cause of bighead, other factors are involved in the occurrence of the disease. The kind and character of the feed on which the sheep subsist at the time they eat the plants are probably the most important factors in producing the edema or photosensitization. Horsebrushes are among the first to begin growth in the spring and usually are well leaved out by the time sheep start on the trails to the shearing corrals and summer ranges. Most of the bighead outbreaks occur at that time.

Since plants are not palatable and are seldom eaten in toxic amounts when good forage is available, sheep may graze normally in an area where \textit{Tetradymia} is abundant with a very little danger of bighead, but a hungry herd that is being trailed through the same area may develop the disease.

Stormy weather also is apt to change the feeding habits of animals to the extent that unpalatable or poisonous plants will be eaten. Early spring use of ranges before the forage plants are well started may cause sheep to eat \textit{Tetradymia} in sufficient amounts to produce bighead.

Bighead usually appears quite suddenly in a band. Many animals may become affected within a few hours, although symptoms usually appear 16 to 24 hours after the plants are eaten.

Prevention of range bighead depends on a knowledge of the distribution of the \textit{Tetradymia} plants. Avoiding \textit{Tetradymia} areas on the ranges or trails when sheep are hungry will eliminate the greatest danger.

\textbf{Bracken (Pteridium aquilinum)} may be poisonous to both horses and cattle.
and may cause rather severe losses.

Several species of ferns have been suspected of being poisonous to livestock. Because of their low palatability, bracken are seldom eaten when other forage is available. Therefore they are not considered of much importance.

The bracken fern occurs in many parts of the United States, especially in moist places and in meadows.

Most of the cases of bracken poisoning in horses have been caused by ferns that were cut and cured in meadow hay. Poisoning in cattle usually is the result of grazing on the green plants when other forage is scarce. Hay containing ferns is more dangerous in winter if it is the only feed. Poisoning from green plants usually occurs during the latter part of the grazing season until frost.

Bracken poisoning does not usually occur until after the animals have been feeding on ferns for 3 or 4 weeks, unless the feed consists very largely of these plants. When large daily amounts are consumed, the symptoms may appear earlier and be more acute than when the ration contains less of the toxic material.

The toxic substances in the ferns apparently have a cumulative effect, so that a period of time must elapse before symptoms become noticeable. The course of the disease and the severity of the symptoms largely depend on the amount of the plants consumed daily, although there is some variation in the individual susceptibility of different animals.

The symptoms of bracken poisoning in horses may be emaciation, weakness, staggering, nervousness, and constipation. The temperature may remain nearly normal, although the pulse rate is usually higher. The disease may extend over a week or more. The appetite may remain fairly good for some time after symptoms appear.

The symptoms in cattle are usually more acute and may include a high temperature, rapid loss of flesh, salivation, hemorrhage from the nostrils, small hemorrhagic spots (petechiac) in the membranes of the eyes, nostrils, and mouth, and a bloody diarrhea.

The symptoms of fern poisoning may vary, but when such a condition is suspected and it is known that the plants are being eaten, a change to other feed should be made.

The mortality rate of animals showing symptoms of bracken poisoning is usually high. Some benefit may be derived from treatment of the symptoms, such as a diet of readily available soft, nutritious food to relieve exertion, constipation, and irritation of the hemorrhagic intestine, but no specific cure is known. Prevention is the best way to control the disease.

**Limestone Disease** of southern New Mexico and southeastern Arizona, which has resulted in heavy losses of cattle over a period of years, is probably caused by Wrights eupatorium (*Eupatorium wrightii*). The nature of its toxic substance has not been determined. Unlike white snakeroot (*E. rugosum*), a related and highly toxic species of the East Central States, Wrights eupatorium does not contain tremetol.

Toxicity is confined principally to the leaves, and the margin between a nontoxic and a lethal dose is very narrow. Poisoned animals are usually found dead on the range, having died during the night while lying in a normal position without any signs of having struggled. The plant is as toxic during the summer as later, but apparently it is not eaten until fall; the losses start about the time of the first heavy frosts and continue for a month or longer. In experimental feeding, sheep were found to be as susceptible as cattle to the poisonous effects of the plant, but the reported losses have all been among cattle.

Wrights eupatorium is found mainly in limestone outcroppings on ridges and slopes of foothills and lower mountains. These areas may occupy only a small portion of a pasture and, if fenced off, the remainder of the pasture would be safe for cattle. If additional
fencing is not feasible, the cattle should be moved to other pastures shortly before the first of October.

**Tall Fescue** (*Festuca arundinaceae*) has received attention lately as a possible cattle-poisoning plant. We have not been able to learn what is its poisonous principle.

Tall fescue, a coarse, reedlike grass, is a prominent grass crop in some swampy areas of New Zealand. In areas where this fescue grew, some cattle in the fescue pastures became affected with a condition in which lameness sometimes developed within 14 days after they first ate the fescue. One or both hind limbs were involved. Heat, swelling, and pain occurred in the affected limb. Later the pain and the swelling subsided, but drying and hardening of the skin and cold in the extremity of the leg or foot set in. An indented line formed in the hide of the leg, which separated the lower part of the leg and foot, which was cold and apparently dead from the normal skin above. The warm and cold places usually were sharply separated at this point. Sometimes the extremity of the limb was shed.

Such a description is like a description of poisoning of cattle by the black, hard, hornshaped sclerotia (horny mass of fungus tissue) that form the resting stage in the growth of the ergot fungus. Because ergot sclerotia commonly replace some of the seeds of many grasses on which they grow (they often replace rye grains in the head), it was thought it was ergot of the tall fescue that was causing the trouble in New Zealand.

That assumption was readily made from the similarity of symptoms of ergot and fescue poisoning. The ergot sclerotia forms only in the seed heads of a grass which it infects. Study and observation led to the discovery that the cattle-foot condition occurred in places where there were no seed heads of fescue and that consequently the poisoning from fescue could not be as a result of the ergot.

The first published report we have on the effect of fescue is one by I. J. Cunningham, of Wallaceville, New Zealand, in the New Zealand Journal of Agriculture, November 1948, page 519. Dr. Cunningham reported in the Australian Veterinary Journal, February 1949, that the fescue grass under consideration should be classed as a toxic plant and not as a plant that acquired toxicity through infestation by ergot.

After they received the reports from New Zealand and Australia, research workers in the United States undertook to determine whether some trouble in the United States that had been attributed to the ergot actually might have been caused by fescue.

Lameness in cattle that had been eating fescue had been reported at various times by cattlemen in western Colorado. They believed it was due to ergot poisoning, foot rot, or frozen feet, but after the reports came from New Zealand, the disease was associated with fescue.

*Festuca arundinaceae* is often referred to as tall fescue, but other common names for it are alta fescue, king fescue, giant fescue, ditchbank fescue, and reed fescue. A. A. Goodman, extension veterinarian of the Colorado Agricultural and Mechanical College, in an early report of the trouble in Colorado expressed the opinion that other more palatable species of fescue grasses, which are recommended for pasture grass mixture, do not carry the toxic substance which was reported to be in the coarse tall fescue.

Dr. T. J. Stearns, a practicing veterinarian in Louisville, Ky., in an article in the Journal of the American Veterinary Medical Association, May 1933, page 388, reported a similar ailment among cattle grazing fescue 31, a smaller more palatable variety. He wrote: "It would seem that most fescue species of grasses contain the toxic substance, and under certain climatic conditions will cause trouble."

Other references to fescue foot or
fescue lameness, as the disease is often called, have come from Tennessee, Alabama, California, and Texas. Research workers in Colorado, Kentucky, and Tennessee began experiments in an effort to determine the causative agent and develop a corrective treatment.

A report from the Tennessee Agricultural Experiment Station said that cattle have recovered when removed from fescue to other pastures, sometimes slowly and after loss of considerable weight. Some of the recovered animals were returned to the fescue pastures without recurrence of toxicity symptoms or any further untoward effects.

The report stated, "Based on the lack of complaints from farmers and Extension people, many fescue pastures may not be toxic." Because of the large number of pastures of tall fescue on which cattle graze and the small number of cases reported, the percentage of toxic pastures may be small. It is likely that toxicity in certain pastures does not persist from year to year.

Most of the cases of toxicity from tall fescue have been in localities where because of ecological factors the coarse, persistent plants were nearly in pure stands.

The danger of toxicity probably can be reduced considerably by providing a variety of feed rather than depending on fescue as the only feed. Tall fescue, even though the palatability is low and the danger of poisoning from it has to be considered, serves a purpose because of its hardiness, persistence, and tolerance of swampy conditions.

If vigilance and careful observation is made for initial symptoms and the cattle are removed promptly from pastures, possibly tall fescue has a useful place under certain conditions as an economic plant for cattlemen.

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EDWARD A. MORAN, an animal physiologist in Noninfectious Diseases Section of the Animal Disease and Parasite Research Branch, has been studying stock poisoning plant problems as an employee of the Department of Agriculture since 1929. Until 1951 his field quarters were in Utah during the summer. He is a graduate of the New York State College of Agriculture at Cornell University.

WAYNE BINNS holds degrees from Iowa State College and Cornell University. He was a member of the veterinary science department of the Utah State Agricultural College from 1940 to 1942 and head of the department from 1946 to 1954. He joined the Agricultural Research Service as a veterinarian in charge of studies of livestock poisoning by plants in 1954 with headquarters in Logan, Utah.

For further reading: