Association, Department of Agronomy, Colorado State University, Fort Collins, Colorado 80523; or the Utah Crop Improvement Association, Utah State Agricultural Experiment Station, Utah State University, Logan, Utah 84322 or the Crop Improvement Association for your state. Commercial seed growers will need 275 female and 55 male plants for a one-acre 'Rincon' orchard. Planting stock varies from 6 to 12 inches in height and cost $1.50 each plus shipping charges. Plants should begin producing seed in about two years. Cultural information is available from the Forest Service Shrub Sciences Laboratory or the U.C.E.P.C.

References

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Selenium Poisoning in Livestock

Lynn F. James and James L. Shupe

Selenium has long been recognized for its toxic effect on farm animals. Historically, Marco Polo, in 1295, may have been describing chronic selenium poisoning when in his account of his travels in western China he wrote that a poisonous plant growing there, if eaten by their beasts of burden, caused the hooves of the animals to drop off.

The first account of selenium poisoning in the U.S. was given by Dr. T.C. Madison in 1856 when he described a condition that afflected cavalry horses situated near the Missouri River in the Nebraska territory. Although the cause of the problem was not known at that time to be selenium poisoning, research done years later gave that indication. Various reports were made during the next 75 years of situations that were later recognized as due to selenium poisoning. In the early 1930s, cooperative research between the U.S. Dept. of Agriculture and the South Dakota and Wyoming Experiment Stations demonstrated that selenium in the forage in certain areas was responsible for such conditions.

After considerable research in the 1930s, little additional research was done on selenium until the late 1950s, when this element was shown to be an essential nutrient. Nothing more will be said of selenium as an essential nutrient except to point out that an essential nutrient may cause a toxic effect by its absence. Hence, the justification for the term deficiency disease.

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Selenium in Soils

The earth's crust is estimated to contain an average of 0.1 ppm selenium. Some soil may contain less and some considerably more, to a level that plants growing on them may be toxic.

In the United States, soils high on selenium are usually derived from sedimentary rocks having a high selenium content.

Selenium occurs in the soil in various forms. In unweathered rock and some soils in arid regions it may occur in its elemental form or as iron selenide. In both of these forms, selenium has a low availability to plants. Small amounts of organic selenium, derived from decaying plants, may occasionally be available to growing plants. However, the most soluble and most important for insofar as plant availability is concerned, is inorganic selenate.

The distribution of the available forms of selenium in the soil profile has an important effect on plant uptake. In arid regions, the soluble (and thus the more available) forms may leach into the deeper soil profile, where deeper-rooted plants such as shrubs and plants with long tap roots may absorb it. These plants may act somewhat as a selenium

pump, bringing the element to the soil surface. Grasses and some of the shallower rooted plants may obtain selenium from the 2nd or 3rd foot of the soil profile.

In the United States, areas of significant seleniferous soils occur in South Dakota, Wyoming, Utah, Colorado, New Mexico and Arizona.

Selenium in Plants

Plants vary significantly in their ability to accumulate selenium from the soil. Even different species of plants growing in the same area contain non-uniform amounts of selenium. The seleniferous plants have been divided into three groups according to their ability to accumulate selenium:

(1) Primary selenium indicator or accumulator plants require selenium for growth and typically contain large amounts of selenium (1000 to 7000 ppm, although amounts as low as 100 plus are common). This group of plants includes the selenium-accumulating *Astragalus* such as *A. bisulcatus, A. praelongus,* and *A. pattersonii; Stanleya* spp.

Astragalus bisulcatus, *one of the selenium accumulating plants.*

and others. These plants often have an offensive odor. Plants in this group are usually nonpalatable because of their high selenium contents.

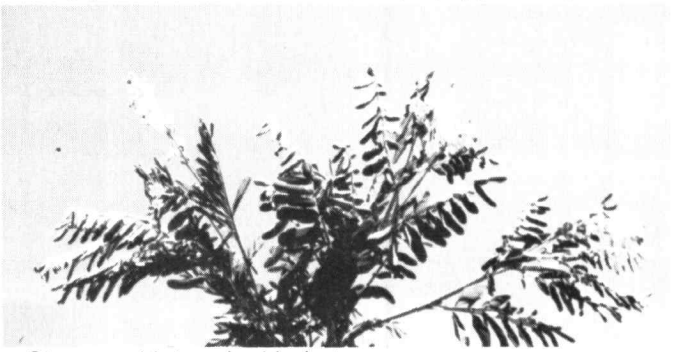
(2) Secondary selenium-accumulating plants do not require the element for growth and usually contain 50-100 ppm Se, although they may accumulate more. Included in this group are plants such as *Atriplex, Aster,* and *Gutierrezea.*

(3) Other plants, when grown on seleniferous soils, take up lesser quantities of selenium, usually not more than 50 ppm and usually less than 20 ppm Se. Included in this group are cultivated crops, grains, and native grasses.

Acute selenium poisoning in livestock is usually associated with the plants in group 1, while chronic poisonings are associated with groups 2 and 3. Many plants in groups 2 and 3 are good forage plants under normal conditions.

The part of the plant eaten is also important in determining selenium content. Seeds usually contain more selenium than leaves, which contain more than stems. Some plants contain volatile selenium compounds that are lost on drying, especially at high temperatures.

Plants are known to synthesize several selenium-containing compounds. The toxicity of many of these has not been studied. Therefore, the toxicity of the plant cannot be evaluated based on its total selenium content. In addition, selenium-accumulating plants such as the primary *Astragalus* accumulating plants or the secondary *Haplopappus* may contain



Close-up of Astragulus bisulcatus.

additional organic toxins that may complicate toxicity evaluations.

Selenium intoxications

All animals and humans are known to be susceptible to selenium poisoning. Poisoning is most common in grazing animals such as cattle, sheep, and horses, which may forage on seleniferous grasses, forbs or shrubs. Poisoning that might occur in poultry or swine would result from their being given seleniferous grain or some other seleniferous feed.

Selenium intoxication has been outlined (by Rosenfeld and Beath "Selenium" 1964, pg 145-163, Academic Press) as follows:

1. Acute intoxication
2. Chronic intoxication
   A. Alkali disease
   B. Blind staggers

Some reviews have quoted Rosenfeld and Beath as describing acute toxicity as blind staggers and chronic toxicity as alkali disease. This is incorrect according to Rosenfeld and Beath (see citation above).

Acute Selenium Intoxication

Acute selenosis in the field results from the ingestion of a lethal amount of primary-indicator or other plants containing high levels of selenium (several hundred ppm Se).

Because plants containing high levels of selenium are rather unpalatable, acute poisoning is uncommon. Poisoning is usually associated with hungry animals eating highly seleniferous plants.

Acute poisoning in cattle is characterized by abnormal posture and movement, watery diarrhea, elevated temperature, labored respiration, indication of abdominal pain, prostration, and death. Cattle at the Poisonous Plant Research Laboratory fed a high dose of seleniferous plants over several days developed quite suddenly a high-pitched bellow, excitement (running, bumping into fences, etc.), increased respiration, prostration, and death. Acute poisoning in sheep is characterized by increased respiration and sudden death.

Pigs previously given a selenium-deficient diet are more susceptible to acute intoxication than are normal pigs.

Animal responses to toxic amounts of selenium are highly variable as it involves the time dose response relationship. Thus, if death occurs rapidly, the signs may vary from those seen if death occurs some time after eating a lethal amount of selenium.

The principal gross and microscopic lesions are in the lungs. They include edema, hyperemia, and congestion. Gastroentritis may also be seen.

Chonic selenium poisoning "Alkali Disease"

Chronic selenium poisoning of the alkali disease type (as described by Rosenfield and Beath-see citation above) results from the ingestion of toxic amounts of selenium (5-40 ppm) in plants as grass, or in grain, in which the selenium is bound in the plant protein and is relatively insoluble in water. It has been shown, however, that this condition can be caused by feeding the inorganic salts of the element. The seleniferous feed must be consumed over a period of weeks or months.

In cattle and horses the principal signs of poisoning include the loss of hair, emaciation, hoof malformations, and lameness. Perhaps the most severe economic effect resulting from the consumption of seleniferous forage is reduced reproduction.

At the Poisonous Plant Research Laboratory (PPRL) we've observed that it is difficult to entice sheep and cattle to eat a diet containing more than 20 ppm Se over any length of time.

Some have questioned whether selenium was the cause of alkali disease, and whether the form in which it was ingested was important. Ample evidence demonstrates that alkali disease can be caused by grains and grasses with high selenium contents and by inorganic selenium salts.

The South Dakota Agricultural Experiment Station uses the following guidelines for diagnosing alkali disease.

Selenium (ppm) in Blood	Hair	
Under 1.0	Under 5.0	Chronic selenosis should not be expected.
1.0-2.0	5.0-10.0	Suggests a borderline problem.
Over 2.0	Over 10.0	Selenium intake excessive and selenosis can be expected. However animals with these levels will not necessarily exhibit signs of the toxicosis.

Chronic selenium poisoning "Blind Staggers"

Chronic selenium poisoning of the blind staggers type (Rosenfeld and Beath - see above citation) is said to be caused by organic selenium compounds, with or without small amounts of selenate, which are readily extractable with water from native, selenium-indicator plants. The animals must consume moderately toxic amounts of these plants over a considerable period of time to evidence poisoning.

Native range weeds such as *Astragalus bisulcatus* and *Machaenanthera glabriuscula* contain a moderately high concentration of water-soluble selenium that is responsible for poisonings. The severity of the intoxication depends upon the indicator plant ingested. The presence of other toxic substances in these plants may produce different manifestations of the disease. *A. bisulcatus,* however, has been judged to contain little toxic material other than selenium.

The selenium-accumulating *Astragalus* plants are probably the principal group of selenium indicator plants concerned with livestock poisoning.

The blind staggers syndrome expresses itself in three stages. In the first stage the animal has some impairment of vision, and wanders in circles, disregarding objects in its path. In the second stage, the signs in stage one intensify in severity, and the animal's front legs become weak and give way. In the third stage, the tongue and swallowing mechanism become paralyzed, respiration is labored and quickened, there is obvious evidence of abdominal pain, and the cornea becomes cloudy. The third stage appears suddenly and death often follows within a few hours, apparently as the result of respiratory failure.

Treatment consists of drenching with copious amounts of water and injecting strychnine sulfate.

There is some question as to whether blind staggers is associated with selenium poisoning. The National Research Council suggests further study on the matter because blind staggers has been associated with the consumption of Se-indicator plants but has not been produced with selenium compounds. Therefore, other plant toxins could be involved.

Jensen et al. (JAVMA 129:311-321, 1956), in comparing a condition called forage poisoning in Colorado with blind staggers in Wyoming, said:

> Polioencephalomalacia, a noninfectious disease of pasture and feedlot cattle and sheep, is characterized by multiple foci of necrosis in the cerebral cortex. In Colorado, the disease is known as "forage poisoning." In Wyoming, where the disease has been studied extensively, it is known as "blind staggers" from selenium poisoning. The clinical syndromes of the disease in Colorado and Wyoming are identical. The cause of the disease in cattle and sheep of Colorado has not been studied adequately, while the neuropathology of the disease in cattle and sheep of Wyoming has not been reported.
>
> "Although it is assumed that blind staggers reported from Wyoming, and forage poisoning reported from Colorado, are a single entity. . .

Forage poisoning in Colorado was not associated with selenium poisoning.

Tansy mustard, when grazed by cattle for extensive periods of time, produces a syndrome very much like blind staggers, and it too responds to treatment with copious amounts of water.

Kochia scoparia has also been shown to produce a blind staggers like syndrome in cattle in various parts of the west. The kochia must be grazed over an extended period of time.

The selenium-accumulating *Astragalus* plants are the principal group of selenium indicator plants. Much of the research work on blind staggers was done using *A. bisulcatus,* a selenium indicator plant. Recent research at the USDA Poisonous Plant Research Laboratory suggests that, contrary to what was thought, *A. bisulcatus* does contain organic toxins in addition to selenium.

It is not uncommon to hear of conditions referred to as "Blind Staggers." If animals have problems with locomotion and staggering gait, etc., there is tendency to describe the condition as blind staggers. Because of the classification of selenium poisoning—alkali disease, blind staggers, etc.—such occurrences are often considered to be selenium related. ●

Calves fed *A. bisulcatus* for a period of time at the PPRL, circled, bellowed, became uncoordinated, and died suddenly. We have not obtained similar results to date from feeding the organic selenium compound, nor have we observed these phenomena when feeding the nonselenium-accumulating *Astragalus*. Perhaps this intoxication is associated with the form of selenium as Beath suggested, or with a nonselenium unknown organic toxic involved, or with both acting in concert.

Pigs fed on diet containing 25 ppm Se or more have developed severe spinal cord lesions, which ultimately led to varying degrees of paralysis.

Selected Reading

Rosenfeld, Irene and Orville A. Beath. Selenium, Biochemistry, Toxicity and Nutrition. Academic Press. New York.

Those wanting more information can obtain a complete list of references by writing to the authors.

How to Race a Dead Man and Lose

A.A. Beetle

Frederic E. Clements is our dead man—he lived from 1874 until 1945 and therefore died before the Society for Range Management was formed. One fondly hopes that F.E. Clements, had he lived, would have been a charter member.

Born at Lincoln, Nebr., September 16, 1874, Clements died at Santa Barbara, Calif., on July 26, 1945. He received his B.S. degree in 1894, M.A. in 1896, Ph.D. in 1898, and LL.D. in 1940, all from the University of Nebraska. He was Phi Beta Kappa. He taught at the University of Nebraska (1894 until 1907) and at the University of Minnesota (1907-917) and from 1917 until retirement in 1941 he was research associate (in charge of ecological research) at the Carnegie Institution of Washington. In retirement he transplanted timothy *(Phleum pratense)* from Santa Barbara, his winter home, to the Alpine Laboratory on Pike's Peak, Colorado, his summer home. And he carried alpine timothy *(Phleum alpinum)* from in *Phleum* was a factor of the environment. I visited Dr. Clements to Santa Barbara, planning to show that form in 1943 and was shown this garden.

How can F. E. Clements have outrun, and still be outrunning the range managers of today? His 176 mentions in the first 31 volumes of the *Journal of Range Management* ranks him 88th. He was a teacher of A. W. Sampson (4:362; 8:44 in the *Journal*); (here and subsequently are shown volume and page references for the *Journal of Range Management*); he was a teacher of J.E. Weaver (7:217); was one of the first ecologists to study grasslands (15:243; 17:124); and was a leader of American conservation (25:75).

Clements' productive life in the field of range management,judging from his cited work in the *Journal of Range Management*, lasted from 1905 when he published "Research Methods in Ecology" until 1949 (posthumus) when B.W. Allred and Edith S. Clements (his wife) compiled and edited his "Dynamics of Vegetation", a memorial volume. Between these two he published 18 other books or papers that have been cited in the *Journal of Range Management*.

What did Clements do to interest these authors, Allred, Anderson, Arnold, Blauer, Bleak, Blydenstein, Cable, Claveron A., Collins, Cronemiller, Dwyer, Dyksterhuis, Eckert, Edgar, England, Fichter, Frischknecht, Giunta, Hanks, Harkness, Hulett, Humphrey, Jameson, McArthur, Miles, Nettleton, Nord, Parker, Plummer, Poulton, Sampson, Schmutz, Smeins, Pond, Poulton, Talbot, Thatcher, Tisdale, Tomanek, Tueller, Turner, Van Amburg, West, Wright and others? In the first place he published with J.E. Weaver a book entitled "Plant Ecology" in 1929. This volume produced, according to Campbell (20:274), the original use of the term "forb"; it described the use of a meter line transect (26:61) and it documented the presence of mycorrhiza in grasslands (31:149). The second edition in 1938 is Clements' most cited work (21 times). What caught range men's attention? The control of rabbits and rodents (6:18); the success of seedlings (7:90); sagebrush seedlings (8:69); competition always occurs where two or more plants make demands for light, nutrients, or water in excess of the supply (11:115); the tap root system of big sagebrush (16:245); the Palouse grassland (15:275); climax vegetation as a guide for organizing man's mind concerning ecological process that occurs in natural plant communities (16:289); true prairie can prevent weed invasion (18:312); the effect of repeated burning (20:170); the sagebrush climax (20:288); shortgrass disclimax (22:87); soil depth influences vegetation heterogeneity (22:196); alpine communities are little understood (25:276); a relatively low water content, provided there is enough to insure good growth, stimulates the roots to a greater development, resulting in a greatly increased absorbing surface (28:475).

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