

Selenium Poisoning in Livestock

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ABSTRACT

Selenium in certain soils may be taken up by plants in amounts sufficient to make forage toxic to animals. Seleniferous forage can be found in semiarid areas on soils typically derived from Cretaceous geologic material in the Western United States and Canada. Intoxication of livestock by seleniferous plants has been classified as acute or chronic. Acute poisoning results from consuming plants containing high selenium concentrations. Chronic selenium poisoning has been described in two forms: alkali disease and blind staggers. Alkali disease results from prolonged ingestion of plants containing 5–40 ppm selenium in inorganic or organic forms. Alkali disease causes loss of hair, lameness, weight loss and probably reduces reproductive efficiency. Blind staggers is said to result from the consumption of selenium indicator plants. These plants, in contrast to the nonaccumulators, contain selenium in water soluble, nonprotein forms. Blind staggers causes animals to wander, walk in circles, and to have difficulty in swallowing; in addition, it may cause blindness. Information is presented that questions the attribution of blind staggers to selenium toxicosis.

INTRODUCTION

Selenium has long been recognized as being toxic to animals. Marco Polo had probably seen selenium poisoning during his travels through western China, circa 1295 (Lantham, 1958). He described a condition where his horses' hooves dropped off after grazing certain plants of the region. Polo's travels took him through an area of northwest China which recently has been identified as containing high to excessive soil selenium levels (Jian-An and others, 1987). The area is bounded by 36° and 45° North latitude and 75° and 107° East longitude.

In 1560, Father Pedro Simon of Columbia described a condition in livestock and humans which today is recognized as selenium poisoning (Simon, 1953). A marked loss of hair and sloughing of the hooves of cavalry horses were described by United States Army physician Dr. T.C. Madison (1860). He was stationed at Fort Randall on the Missouri River near the South Dakota and Nebraska State line. Settlers in this and adjacent areas in the northern Great Plains observed similar symptoms in their livestock. These

people called the condition 'alkali disease' because of its association with alkaline seeps. It is possible that many of these seeps were used as watering sources for the animals.

The seriousness of alkali disease stimulated initiation of research by the late 1920's to determine the cause of the problem. In the early 1930's, H.G. Knight of the United States Department of Agriculture Bureau of Chemistry and Soil suggested that selenium was the cause of alkali disease (Anderson and others, 1961). Later, high concentrations of selenium were found in some small grains grown in the area. Proof was soon obtained that selenium was the cause of alkali disease. It was also shown that certain plants accumulated selenium in relatively large concentrations. Selenium toxicosis occurred when animals consumed these plants.

During the following 15 years, intensive research was conducted by the Agricultural Experiment Stations in Wyoming and South Dakota and by the United States Department of Agriculture and U.S. Geological Survey. They described the factors governing its occurrence in soil, accumulation in plants, and toxic effects on animals. Excellent reviews of this early research are given by Anderson and others, (1961), Rosenfeld and Beath (1964), Zingaro and Cooper (1974), National Research Council (1976), Moxon and Rhian (1943), and Moxon (1937).

Selenium in Soil

The Earth's surface contains an average of 0.1 ppm selenium. Some soils contain less and some considerably more. Soil selenium concentration varies over short distances. Some soils contain selenium in amounts and forms available to plants, causing them to be toxic to animals. Selenate is the most soluble inorganic form, while selenite is less soluble. Each is readily absorbed by plants. Selenium is also present as elemental selenium or as iron selenide in certain unweathered rocks, but these forms are highly insoluble and are not absorbed by plants.

Soils containing high levels of available selenium are usually alkaline and usually receive less than 50 cm annual precipitation. The soluble selenium may be found in lower portions of the profile, where it is only available to deeply rooted perennial forbs and shrubs. Reviews of selenium in

soils have been prepared by Anderson and others (1961), National Research Council (1976), Sharma and Singh (1983), and Jacobs (1989).

Selenium in Plants

Selenium is readily absorbed by all members of the plant kingdom (Anderson and others, 1961). However, in this report we are primarily concerned with plants that accumulate selenium at levels toxic to animals. Plants, even of the same species, may differ in their selenium concentration because of differences in available soil selenium, interfering anions at the soil-root interface, root absorption, translocation from roots to tops, and dilution in the plant tops.

Plants have been divided into three groups, based upon their selenium-accumulating ability (Rosenfeld and Beath, 1964; National Research Council, 1976).

1. Primary selenium-accumulator or indicator plants may contain up to several thousand parts per million selenium. This group includes species of the genera *Astragalus*, *Machaeranthera* (section *Xylorrhiza*), *Haplopappus* (section *Oonopsis*), and *Stanleya*. Selenium accumulated by these plants is largely water soluble and in the form of low molecular weight organic compounds and selenate (National Research Council, 1983). The selenium is not incorporated into protein.
2. Secondary selenium absorbers may contain up to several hundred parts per million selenium, but lesser amounts are much more common. This group includes species of the genera *Aster*, *Atriplex*, *Castilleja*, *Comandra*, *Gyria*, *Grindelia*, *Gutierrezia*, *Machaeranthera*, and *Mentzelia*. As in the first group, selenium appears primarily as water soluble selenate with smaller amounts of low molecular organic forms.
3. The nonaccumulator group includes grasses, 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 5–12 ppm. The majority of selenium intoxications probably result from the grazing of plants in this category.

These groupings are somewhat arbitrary because some plants appear in more than one group. There is, however, greater tendency for plants in group 1 or 2 to accumulate a much higher level of selenium than those in group 2 or 3.

The toxicity of selenium-accumulating plants cannot be evaluated solely on the basis of the total selenium concentration. Various selenium compounds are likely to exhibit different intensities of toxicosis to animals. Also, plants, such as *Astragalus* and *Haplopappus*, contain other nonseleniferous constituents that may be toxic to animals.

SELENIUM IN LIVESTOCK

Selenium Deficiency

Selenium has long been recognized as a mineral toxic to animals, but only recently has it been shown to be a nutritional requirement. Selenium was reported essential for animal health in 1957, nearly three decades after its implication with alkali disease and animal death (Schwartz and Foltz, 1957). Selenium deficiency (Combs and Combs, 1986) has been implicated in retained placenta in cattle, persistent diarrhea in young calves, and white muscle disease in young calves and lambs. Deficiency can cause exudative diathesis, nutritional muscular dystrophy, and nutritional pancreatic dystrophy in poultry. Hepatoses dietetica, mulberry heart disease, nutritional muscular dystrophy, and infertility may occur in ewes not receiving enough selenium.

Selenium concentrations required in the diet range from 0.05 to 0.10 ppm, depending on availability to the animal. Dietary sulfur has been shown to compete with selenium absorption, both by the plant and the animal. The interaction of selenium and vitamin E in animal metabolism is discussed by Combs and Combs (1986).

Selenium Intoxication

Toxicosis occurs when an animal's diet contains more than 3–8 ppm selenium (National Research Council, 1980). The actual concentration may vary between and within animal species. Some individuals may be conditioned to tolerate higher levels of selenium than others (Lynn James and others, unpubl. data, 1989). The concentration in forage plants leading to animal toxicosis is thus only 30–160 times that required for adequate nutrition. This window is not as narrow as that for copper toxicosis/essentiality which is less than 10 for sheep (National Research Council, 1980). Much work has been done to determine the geographical distribution (mapping) of selenium deficiency or excesses (R. W. Welch, E. E. Cary, W. H. Alloway, and J. Kubota, unpubl. data).

All animals, including man, are susceptible to selenium poisoning (National Research Council, 1980; Combs and Combs, 1986). However, poisoning occurs most frequently in animals grazing seleniferous forages. Although much emphasis has been placed on plants that accumulate selenium in thousands of parts per million, we believe that most selenium poisonings result from grazing plants containing 5–20 ppm selenium. Consumption of seleniferous grains can cause poisoning in poultry and swine. According to Rosenfeld and Beath (1964), selenium is one of the few elements absorbed by plants in sufficient concentrations to create a toxicity hazard to foraging animals.

Rosenfeld and Beath (1964) provided a widely accepted classification of selenium intoxications in livestock. The classification is as follows:

1. Acute intoxication
2. Chronic intoxication
 - a. Alkali disease
 - b. Blind staggers.

Acute Selenium Poisoning

Acute selenium poisoning often results from short-term foraging of highly seleniferous plants such as the indicator plants (Rosenfeld and Beath, 1964). This occurs principally when animals are very hungry and ingest seleniferous plants of low palatability but with high levels of selenium. Poisoning in cattle, horses, and swine is characterized by an abnormal posture, unsteady gait, diarrhea, abdominal pain, increased pulse and respiratory rate, prostration, and death. Sheep do not show signs of selenium poisoning as distinctly as do other species of domestic animals. Often all that will be seen in sheep is depression and sudden death.

Cattle at the Poisonous Plant Research Laboratory fed a high dose of seleniferous *Astragalus* plants over several days quite suddenly started bellowing with a high-pitched voice as if distressed, became excited, increased their respiratory rate, became prostrate and died (Lynn James, unpubl. data, 1982–86). Swine fed a selenium deficient diet are later more susceptible to acute intoxication than normal pigs.

Gross pathological changes include petechial hemorrhages in the endocardium, acute congestion and diffuse hemorrhages in the lungs, enteritis and passive congestion in the liver. Acute congestion of the endocardium occurs. The lungs evidence congestion and hemorrhaging in the alveoli. The mucosa of the stomach and intestine manifest edema, hemorrhage, and necrosis. The signs of poisoning in sheep are primarily increased respiration and sudden death. The gross and microscopic lesions reflect the marked congestion and edema of the lungs.

Chronic Intoxication

Chronic selenium poisoning has been divided into two syndromes—alkali disease and blind staggers. This separation is based on the type of forage eaten (Rosenfeld and Beath, 1964). Alkali disease, which has been described in cattle, swine, and horses, is associated with the consumption of seleniferous forages (Group 2 and 3 plants) like hay, grain, grasses, and palatable forbs for days, weeks, or even months. In these forages, the selenium is largely incorporated into the protein and is not water soluble. Blind

staggers has been described in cattle, and possibly sheep, and is associated with the consumption of selenium indicator plants over days or weeks. Selenium in these plants is largely water soluble.

Alkali Disease

The condition, described as alkali disease by T.C. Madison in 1857, is now recognized as a type of chronic selenium poisoning. This disorder, according to Rosenfeld and Beath (1964), results from the consumption of seleniferous grasses, grains, and forbs that contain 5–40 ppm selenium. The forage is usually consumed over a period of weeks or more. Alkali disease has been produced by feeding inorganic sodium selenate and sodium selenite (Moxon, 1937; Hartley and others, 1985). Alkali disease is characterized by dullness, lack of vitality, emaciation, rough hair coat, loss of hair (especially the long hair of mane or tail), and lameness. Cattle, horses, and swine all develop alkali disease when fed seleniferous feeds. Sheep do not respond in the same manner, they neither lose wool nor develop hoof lesions.

Reduced reproductive performance could be the most significant effect of chronic selenium poisoning (Olson, 1978; Ort and Latshaw, 1978; Wahistom and Olson, 1959). It apparently occurs at levels of dietary selenium lower than that required to produce typical signs of alkali disease. Adverse effects on animal reproduction may occur at forage selenium levels less than 2 ppm (National Research Council, 1976). Additional verification is needed on the effects of selenium on reproduction. Alkali disease may be the principle chronic manifestation of selenium poisoning.

Blind Staggers

According to Rosenfeld and Beath (1964), blind staggers results from grazing moderate amounts of indicator plants over a period of days or weeks. Selenium in these plants is readily extractable in water. Blind staggers has been observed in cattle and sheep but not in horses, swine, or poultry.

In cattle, blind staggers appears in three stages. In the first or early stage the animal may demonstrate impaired vision, disregards objects in its path, and stumbles over them or walks into them. The body temperature and respiration are normal, but the animal has little interest in eating or drinking. The clinical signs intensify as the animal enters phase 2. The front legs weaken, failing to support the animal. The animal becomes anorectic.

During phase 3, the tongue and mechanism for swallowing become partially or totally paralyzed. The animal is nearly blind. Respiration becomes labored and rapid. Abdominal pain is apparent. The body temperature drops below normal. The cornea becomes cloudy, and death may come suddenly. The animal dies of respiratory failure.

Often, a loss of weight occurs during the process so the animal appears emaciated. Cattle may show no outward sign of poisoning but suddenly develop severe clinical signs and die within a few days. Beath reported that this has been known to occur in cattle shipped to feed lots for fattening (Beath, 1982). Recovery only occurs during phases 1 and 2.

In sheep, the blind staggers form of chronic selenium poisoning is not easily diagnosed, and the three stages are not distinct. However, the pathological lesions are the same in sheep as in cattle (Rosenfeld and Beath, 1964).

Microscopic changes, as described in blind staggers, include necrosis and cirrhosis of the liver, nephritis, and impaction of the digestive tract (Rosenfeld and Beath, 1964). Nothing is known of neurological changes even though neurological aberrations are indicated by the signs of poisoning.

Blind staggers, while presently attributed to selenosis, may be a disease of unknown etiology in which selenium may or may not be involved.

Is Blind Staggers a Misnomer?

Some question exists as to whether blind staggers as described by Rosenfeld and Beath is a condition associated with selenium intoxication (National Research Council, 1976; James and others, 1983; Jensen and others, 1956). Alkali disease may be the principle chronic manifestation of selenium poisoning; whereas, "blind staggers" may be a disease condition of unknown etiology in which selenium may or may not be involved.

This section includes several quotations illustrating the confused descriptions of selenium-induced blind staggers. The reader should be aware that blind staggers has never been experimentally produced using selenium compounds.

Beath claimed to have induced blind staggers in cattle by feeding selenium-containing plants. These plants may have contained other toxins in addition to selenium. This work has not been verified (National Research Council, 1976). Beath described a treatment for blind staggers as follows:

TREATMENT—In the treatment of a type case of blind staggers some success can be expected by following this schedule. First of all, one should not allow a time lag to develop after the first suggestion of illness is noticed. Drenching with copious amounts of lukewarm water is an important step to take. This should be followed with foodstuffs, such as corn syrup (glucose), along with baby calf feed. When the digestive tract has been cleared, an animal should be offered some green, succulent forage. In the meantime, it is advisable to inject a small amount of strychnine to stimulate certain body functions. One of the accompanying disorders in this malady is an impaction of the fourth stomach. The use of laxatives should be considered advisedly so as not to bring on excessive irritation. Recovery is slow in most cases; but, when the animal regains its normal appetite and can find its way around, one can consider the illness arrested.

There would seem to be little justification for this treatment for simple selenium toxicosis. However, the treatment is very appropriate for cases of impaction of the digestive tract.

Tansy mustard (*Descurainia pinnata*) causes a condition in New Mexico that is very similar to so-called selenium-induced blind staggers (Hershey, 1945). The syndrome was called woody tongue, paralyzed tongue, or blind staggers. Kingsbury (1964) summarized the tansy mustard condition as follows:

POISONOUS PRINCIPLE—unknown. Despite similarity of the symptoms with those produced in one type of selenium poisoning, tansy mustard's selenium content is insufficient to produce the disease.

TOXICITY, SYMPTOMS, AND LESIONS—Continued ingestion of large quantities of this plant over a relatively long period of time is required before symptoms appear. In cattle on range the symptom first observed is partial or complete blindness. This is followed by, or accompanied with, inability to use the tongue or to swallow. The disease is popularly termed "paralyzed tongue." Because of blindness, animals may wander aimlessly until exhausted, or stand pushing against a solid object in their path for hours. Because of inability to swallow, animals may be observed standing at water unable to drink, or unsuccessfully cropping forage. Animals become thinner and weaker, and death will eventuate if treatment is not undertaken. Treatment is simple and effective. It consists of administering 2 to 3 gallons (7.6 to 11.4 liters) of water (with nourishment such as cotton seed meal in it if the animals are seriously weak) twice daily by stomach tube. This gets the digestive system functioning again and symptoms gradually disappear.

Treatment by Southwestern ranchers and veterinarians for tansy mustard poisoning is the same as that recommended by Beath (1982) for treatment of blind staggers. Hershey (1945) suggested that elements other than selenium may also be involved in the blind staggers described by Beath (1982).

Jensen and others (1956) reported a polioencephalomalacia in pasture and feedlot cattle in Colorado, which was not associated with seleniferous forage. The problem was clinically identical to blind staggers as described by Rosenfeld and Beath (1964). Jensen and others (1956) noted that:

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" and (is said) results 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 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, the appellation polioencephalomalacia is appropriate until the etiological and pathological factors are clearly established.

Beath (1982, p. 20–22) in an apparent response to Jensen's report (Jensen and others, 1956) wrote:

The action of the poison may be delayed, so that several weeks or several months may elapse before the attack occurs. Cattle may show no outward sign of poisoning until severe symptoms develop suddenly, with death following within a few days. This has been known to occur after cattle have been shipped to a feedlot and are being fattened for the market. Diagnosis would obviously be impossible by one not familiar with this type of delayed poisoning.

QUESTIONABLE INTERPRETATIONS—Some investigators think of blind staggers as a form of arrowgrass poisoning. Others merely refer to it as a kind of forage poisoning***Perhaps such an experiment should be undertaken to either prove or disprove the role of arrowgrass as a forage capable of inducing a chronic form of injury to cattle. The term forage poisoning is so all inclusive that any abnormal behavior, of an animal might come under this classification.

This response did not account for Jensen's observations nor was any justification given for assuming that the delayed action response was selenium related.

Kochia (Kochia scoparia) weed poisoning in cattle has been shown to produce a condition characterized by progressive central nervous system dysfunction, blindness, gastrointestinal disorders, and jaundice (Dickie and Berryman, 1979; Dickie and James, 1983). Post-mortem findings included edema of the brain, fatty, cirrhotic, and enlarged livers, gastrointestinal inflammation, and rumen impaction. In addition, polioencephalomalacia has also been associated with kochia poisoning. High sulfate drinking water is thought to aggravate kochia poisoning (Dickie and James, 1983).

High sulfate water is also a potential cause of polioencephalomalacia or blind staggers syndrome (Raisbeck, 1982). The work of Sadler and others (1983) supports that hypothesis. Blind staggers has been repeatedly noted in cattle drinking water from wells or saline seeps in Montana. These saline waters may contain high concentrations of magnesium sulfate (L.F. James, and H.F. Mayland, unpubl. data, 1987). The hypothesis is that ingestion, by ruminants, of large amounts of sulfur, either in the forage or water, results in sulfur reduction by specific rumen flora (Coleman, 1960) to hydrogen sulfide. The H_2S escapes to the oral cavity. The toxic gas is then absorbed by the respiratory system, causing symptoms similar, in many respects, to those of blind staggers (National Research Council, 1980).

Blind staggers was produced experimentally by feeding indicator plants to animals but not by administering pure selenium compounds (Rosenfeld and Beath, 1964).

The presence of toxins, in addition to the selenium in the indicator plants, may have been involved in the intoxication (National Research Council, 1976). For example, two-grooved milkvetch (*A. bisulcatus*) contains swainsonine, which is the toxin in locoweeds (James and others, 1983). Broom snakeweed (*Gutierrezia* spp.) contains various organic toxins (Kingsbury, 1964). Rosenfeld and Beath (1964) erroneously indicated that selenium was the only toxin present in *A. bisulcatus*.

Hartley and others (1985) fed swine various sources of selenium including sodium selenite, sodium selenate, *A. bisulcatus*, *A. praelongus*, and seleniferous wheat (*Triticum aestivum*) to test the hypothesis that accumulator plants produced one type of intoxication (blind staggers) and nonaccumulators produced another (alkali disease). They concluded that selenium in experimental diets was responsible for the poisoning that occurred in swine. Feeding swine different sources of either organic or inorganic selenium produced similar selenium toxicoses. This is contrary to Rosenfeld and Beath (1964) who said "It is well known that alkali disease or hoof injury occurs when selenium is in the organic form." "Blind staggers occurs in connection with indicator plants which contain some inorganic selenium compounds and some in the organic form" (Maag and Glenn, 1967).

Maag and others (1960) fed sodium selenite to steers. The animals did not develop signs of alkali disease, blind staggers, or forage poisoning, although 2 of 8 steers fed the selenite developed microscopic lesions of polioencephalomalacia.

CONCLUSIONS

Alkali disease, a chronic selenium poisoning, has been produced by feeding inorganic selenium compounds or seleniferous plants. Alkali disease seems to be the principal manifestation of selenium intoxication.

Blind staggers describes various disease conditions. It has not been reproduced by feeding selenium compounds, and it is highly questionable if it has been produced using selenium-accumulating plants. Conditions resembling blind staggers include forage poisoning in Colorado, tansy mustard poisoning in the Southwest, and kochia poisoning. High sulfate in water and feed should also be added to the list of potential causes of so-called blind staggers.