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7 Selenium Poisoning in Livestock: A Review and Progress

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ABSTRACT

Selenium in certain soils may be taken up by plants in amounts to render them toxic. Seleniferous forage can be found in most of the western states. Intoxication of livestock by seleniferous plants has been classified as acute and chronic. Acute poisoning results from consumption of plants having high levels of Se; chronic Se poisoning has been described in two forms— alkali disease and blind staggers. Alkali disease is said to result from the consumption of seleniferous grains and grasses, and is manifest by loss of hair, lameness, and loss of weight. Blind staggers is said to result from the consumption of Se indicator plants and is manifest by wandering, circling, loss of ability to swallow, and blindness. Some research casts doubt on the above classification of Se poisoning. Research using pigs (*Sus scrofa domesticus*) indicates that the source of Se does not alter the type of lesion or signs of poisoning observed. There are data available that suggest that blind staggers is not related to Se poisoning.

Selenium (Se)-containing plants have long been recognized for their toxic effects on animals. Marco Polo, traveling in China in 1295, was probably describing signs of Se poisoning when he wrote that the hooves of his livestock

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became swollen and dropped off when they grazed plants growing in certain areas (Rosenfeld & Beath, 1964). Loss of hair and nails in humans presumably suffering from chronic Se ingestion was described in Columbia by Father Simon Pedro in 1560 (NAS-NRC, 1983). Selenium poisoning was probably first described in the USA in 1856 by Dr. T.C. Madison, a physician with the U.S. Cavalry stationed near the Missouri River in the Nebraska Territory. Dr. Madison described hair, mane, and tail hair loss and sloughing of hooves in cavalry horses (*Equus caballus*) grazing in areas later shown to be high in Se (Rosenfeld & Beath, 1964). Reports of similar intoxications over the next 75 yr led to investigation of Se by the South Dakota and Wyoming Agricultural Experiment Stations and the U.S. Department of Agriculture. These reports demonstrated that the maladies were due to the consumption of Se-containing plants by foraging livestock (Anderson et al., 1961; Beath, 1982; Moxon, 1937).

Understanding of the biological function of Se was changed dramatically by Schwarz and Foltz (1957), who demonstrated that minute amounts of Se in the diet were protective against necrotic liver degeneration in laboratory rats (*Rattus* sp.) raised on diets using torula yeast as the protein source. This led to the concept of Se-responsive diseases in a variety of animal species, including domestic livestock, and ultimately to the recognition that Se was, in fact, an essential micronutrient. Assessment of Se's toxicity implications should be made with this other beneficial function in mind.

SELENIUM IN SOILS

In the early 1930s, research showed that some U.S. soils contained Se in concentrations and chemical forms that may be taken up by plants in amounts to render them toxic. Selenium occurs in the soil in several forms having a direct bearing on its availability to plants. Small amounts of organic-bound Se from decaying plants may be available to growing plants; however, the most important form of Se available to plants is the more soluble sodium selenate (NAS-NRC, 1976). In some kinds of unweathered rock, Se may occur as elemental Se or as iron selenite, both of which are relatively unavailable to plants.

The distribution of Se in the soil profile is closely related to its availability to plants. In arid regions, soluble Se is leached into the deeper soil profiles where deep-rooted shrubs and plants with long tap roots may absorb it (NAS-NRC, 1976). These plants act as pumps, bringing Se from the deeper soil profiles to the top 0.6 to 0.9 m (2–3 feet) of surface soil (Moxon & Rhian, 1943). Here it is available to shallower-rooted plants such as grasses and small grains.

Soils containing high levels of Se are usually alkaline and occur in rainfall regions having <50.8 cm (<20 inches) annual precipitation. These alkaline soils usually have levels ranging from <0.1 to 100 mg Se/kg soil and average about 4 to 5 mg/kg (NAS-NRC, 1983). Although Se may be high in acid soils, it is not bioavailable.

SELENIUM IN PLANTS

All plants are capable of taking up some Se if growing in soils where Se is available (NAS-NRC, 1976). Plants may accumulate Se in amounts of < 1 up to several thousand mg Se/kg plant tissue. Plants of different genera, and even different species within the same genus, growing on the same site may accumulate different amounts of Se.

The Se-accumulating plants have been divided into three general groups according to their ability to accumulate Se (NAS-NRC, 1976). These groupings are somewhat arbitrary, because some plants may appear in more than one group.

1. Primary Se-Accumulator, or Indicator, Plants. These are believed to require Se for growth and may accumulate large amounts (often several thousand mg/kg), although amounts of < 100 mg Se/kg are common. These plants are good indicators of seleniferous soils. Selenium-accumulator species may be found in the genera *Astragalus, Machaeranthera, Happlopappus,* and *Stanleya.* There are about 26 species of Se-accumulating *Astragalus* (Rosenfeld & Beath, 1964), which can be recognized by their offensive odor and are, for the most part, not palatable to livestock.

2. Secondary Se absorbers. These plants do not require Se for growth and rarely concentrate more than a few hundred mg Se/kg tissue, although lesser amounts are common. These secondary absorbers may be found in species of *Atriplex, Aster, Gutierrezia, Catelleja, Grindelia, Machaeranthera,* and *Mentzelia.*

3. Grasses, Small Grains, and Other Shallow-Rooted Plants. These plants usually accumulate toxic amounts of Se ranging from 5 to 12 mg/kg but seldom more than 50 mg/kg. The Se accumulation is dependent on factors, still poorly defined, other than genera, species, and availability of soil Se. Some plants such as white clover (*Trifolium repens* L.), buffalograss [*Buchloe dactyloides* (Nutt.) Englem.], and gramma grass (*Bouteloua* spp.) take up very little Se, even when growing on seleniferous soil. Other plants, such as those belonging to the *Crucifera* family (certain mustards, cabbage, broccoli, and cauliflower), may concentrate relatively high levels of Se if grown on high-Se soils.

The toxicity of Se-containing plants cannot be evaluated solely on total Se content. Several Se-accumulating plants such as the seleniferous *Astragalus* and *Happlopappus* may contain additional toxins, which complicate evaluation of Se toxicity. Plants synthesize a variety of Se-containing compounds, and the toxicity of these various compounds has not been studied.

SELENIUM INTOXICATION

All livestock and humans are susceptible to Se poisoning. However, poisoning is most likely to occur in animals grazing seleniferous forage such

as grasses, forbs, and shrubs. Poisoning may occur in poultry (*Gallus gallus domesticus*) and swine (*Sus scrofa domesticus*) as a result of including seleniferous grain or other feeds in their diet (NAS-NRC, 1976).

Selenium intoxication in animals has been classified by Rosenfeld and Beath (1964) as follows.

Acute Intoxication

Acute Se poisoning usually results from the ingestion of excessive amounts of primary, or indicator, plants containing high amounts of Se. Because plants containing high levels of Se are relatively unpalatable, acute poisoning is uncommon. Poisoning is characterized by abnormal posture and movement, watery diarrhea, labored respiration, abdominal pain, prostration, and death (NAS-NRC, 1976). The signs observed are related to the acuteness of the intoxication and the rate of Se consumption. The time interval to death is also related to the rate of Se consumption and can vary from a matter of hours to days.

Chronic Intoxication

Chronic Se intoxication is divided into two syndromes, alkali disease and blind staggers. One of the distinctions that has been made between alkali disease and blind staggers is in the kind of seleniferous forage consumed. Alkali disease in cattle (*Bos taurus*), horses, hogs, and poultry is said to be associated with the consumption of seleniferous forages, such as grasses and crops, over a period of time in which the Se is bound to protein, making the Se relatively insoluble in water. In comparison, blind staggers is said to occur in cattle, and possibly sheep, and is said to be associated with the consumption of Se indicator plants over a period of time wherein the Se is in a water-soluble form (Rosenfeld & Beath, 1964).

Alkali Disease

The condition described by Dr. T.C. Madison in 1856 was later known as alkali disease and is now known to be a type of chronic Se poisoning, but still referred to as alkali disease. The term *alkali disease* was coined by the early settlers of the semiarid Great Plains of the USA (Moxon, 1937). The early pioneers associated the disease with alkali (high salt) waters from alkali or saline seeps and with alkali spots in the soil. It was not until the early 1930s that the condition was shown to be caused by the grazing of seleniferous forages (Moxon, 1937).

Alkali disease according to Rosenfeld and Beath (1964) results from the ingestion by livestock of plants such as grasses and small grains containing 5 to 40 mg Se/kg over an extended period of time. Alkali disease has been produced by feeding soluble sodium selenate (Hartley et al., 1984; Moxon, 1937). Alkali disease is characterized by dullness, lack of vitality, emaciation, rough coat, and loss of hair, especially the long hair, hoof changes, and lameness. Cattle, horses, and swine will all develop alkali disease when

fed seleniferous feeds. Sheep do not respond in the same manner. They show neither loss of body cover nor do they develop hoof lesions (Rosenfeld & Beath, 1964).

Reduced reproductive performance is the most significant effect of alkali disease in livestock (Olson, 1978). The effect on reproduction may be quite marked, yet the animal involved may not show typical signs of alkali disease. The forages responsible for the condition probably contain between 5 and 10 mg Se/kg plant tissue.

Between the period of 1856 and 1940, numerous research and field investigations were conducted on the so-called alkali disease condition. Several good reviews discuss the soil-plant-animal interrelationships to Se poisoning (Anderson et al., 1961; NRC, 1976; Rosenfeld & Beath, 1964; Zingaro & Cooper, 1974).

Blind Staggers

Blind staggers, according to Rosenfeld and Beath (1964), results from livestock grazing a moderate amount of indicator plants over extended time periods. Selenium compounds occurring in these plants are said to be readily extractable with water (Rosenfeld & Beath, 1964). Blind staggers has been described in cattle and sheep (*Ovis aries*) but not in horses, hogs, or poultry.

Blind staggers is characterized by three stages. During Stage 1 the animal frequently wanders in circles, disregards objects in its path, becomes anorexic, and shows evidence of impaired vision. In Stage 2, Stage 1 signs intensify and the front legs become increasingly weak. During Stage 3, the tongue becomes partially or totally paralyzed, there is inability to swallow, varying degress of blindness, labored respiration, abdominal pain, grinding of the teeth, salivation, emaciation, and death (Rosenfeld & Beath, 1964). The three stages of intoxication are not clearly defined in sheep, because sheep exhibit few clinical signs and may die suddenly when intoxicated (Moxon & Olson, 1974).

Changes described in blind staggers include necrosis of functional liver cells and cirrhosis, nephritis, and impaction of the digestive tract (Rosenfeld & Beath, 1957). Treatment recommended for blind staggers consists of drenching with large amounts of water and injection of strychnine sulfate (Beath, 1982; Olson, 1978). However, there is little justification for these treatments, except that they might help relieve the impaction of the GI tract.

Hartley et al. (1984) fed pigs various sources of Se [sodium selenate, sodium selenite, two-grooved milkvetch (*Astragalus bisulcatus*), stinking milkvetch (*A. praelongus*), and seleniferous wheat (*Triticum aestivum* L.), and concluded that the Se content of the experimental diets was primarily responsible for the poisoning that resulted in the pigs. This is contrary to Rosenfeld 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 may contain some inorganic selenium compound and some in the organic form" (as quoted by Maag & Glenn, 1967). Feeding pigs different sources of Se failed to produce any differences in Se toxicoses (Hartley et al., 1984).

Blind staggers apparently was produced experimentally by administering water extract of indicator plants to animals but not by the administration of pure Se compounds (NAS-NRC, 1976; Rosenfeld & Beath, 1964). The presence of toxins, in addition to the Se in indicator plants fed as part of the diet, had been suggested as being involved in the development of the intoxication (NAS-NRC, 1976). For example, two-grooved milkvetch contains swainsonine, which is the toxic constituent of the locoweeds (James et al., 1983), and broom snakeweed (*Gutierrezia* spp.) contain organic plant toxins (Kingsbury, 1964). Because these and other indicator plants contain various organic toxins, this matter needs further study (NAS-NRC, 1976).

Various disease conditions go under the name of blind staggers. Alkali disease has been experimentally reproduced using both inorganic Se compounds and seleniferous plants and seems to be the principal manifestation of Se poisoning. Selenium-induced blind staggers has not been reproduced by using inorganic Se compounds and is questionable when using seleniferous plants. Therefore, it seems to be a condition of questionable etiology in which Se may or may not be involved. The results of recent research and evidence presented add strength to the notion that Se is not involved in the blind staggers conditions (James et al., 1983; NAS-NRC, 1976). Other conditions resembling blind staggers include forage poisoning in Colorado (Jensen et al., 1956), tansy mustard [*Descurainia pinnata* (Walt.) Britt.] poisoning in the southwest (Hershey, 1945; Kingsbury, 1964), and kochia [*Kochia scoparia* (L.) Schrad.] poisoning in Colorado (Dickie & Berryman, 1979; Dickie & James, 1983). High-sulfate waters might also be added to the list of possible causes of the so-called blind staggers syndrome (Raisbeck, 1982; Sadler et al., 1983).

Blind staggers has been reported in Wyoming but not in other western states having problems of Se poisoning in livestock (Moxon & Rhian, 1943). Jensen et al. (1956), in discussing polioencephalomalacia in cattle and sheep, point out that a condition in Colorado known as forage poisoning was clinically identical to blind staggers, but they were unable to associate Se with the forage poisoning condition. The comparison made was as follows:

> 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 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 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, the appelation polioencephalomalacia is appropriate until the etiological and pathological factors are clearly established.

Hershey (1945) points out that tansy mustard causes a disease condition similar to the so-called Se-induced blind staggers and is often referred to as paralyzed tongue or blind staggers. Kingsbury (1964) summarized the 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 [7.6–11.4 L] 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.

It is of interest that this treatment is the same as that suggested for Se-induced blind staggers. Hershey (1945) suggests that factors other than Se may be involved in causing blind staggers.

Kochia poisoning in cattle has been shown to produce a condition characterized by progressive central nervous system dysfunction, blindness, gastrointestinal disorders, and jaundice (Dickie & Berryman, 1979; Dickie & James, 1983). Postmortem findings included brain edema, fatty, cirrhotic, enlarged livers, gastrointestinal inflammation, and rumen impaction, which is similar to Se-induced blind staggers. In addition, polioencephalomalacia has been associated with *Kochia* poisoning, which is a characteristic of forage poisoning. High sulfate in drinking water is thought to aggravate *Kochia* poisoning (Dickie & James, 1983).

A survey of cattle with sulfate in their diets indicated these diets may be associated with polioencephalomalacia or a blind staggers-like condition (Raisbeck, 1982). Additionally, feeding trials with high magnesium sulfate diets have produced polioencephalomalacia (Sadler et al., 1983). Recent field observations in Montana described a condition in cattle similar to that described as Se-induced blind staggers; however, the intoxication was associated with the consumption of water from saline seeps—water high in magnesium sulfates (James & Mayland, 1986, unpublished data). Two interesting observations are that alkali disease was early associated with alkaline or saline seeps and that much of Beath's work was done in areas of gumbo or highly alkaline soils (Moxon, 1937).

In summary, the question has been posed by several investigators as to whether the blind staggers syndrome as described by Rosenfeld and Beath (1964) is indeed associated with consumption by livestock of Se indicator plants (James & Shupe, 1984). This problem has been discussed previously in relation to polioencephalomalacia of cattle and sheep. In Colorado the condition is called forage poisoning. In Wyoming it is called blind staggers. In South Dakota, where some of the principal seleniferous areas of the USA are located, blind staggers has not been reported (Moxon & Rhian, 1943).

Alkali disease is thought to result from the consumption of seleniferous plants containing water-insoluble Se whereas blind staggers has been associated with the consumption of Se indicator plants containing water-soluble Se. Alkali disease has been described in cattle, horses, sheep, pigs, and poultry; blind staggers has been described only in cattle with some brief mention of sheep.

Treatment of Se-induced blind staggers consists of drenching with water, apparently to overcome rumen impaction. A similar treatment is used for the look-alike tansy mustard poisoning. Kochia weed also produces a condition similar to blind staggers, which may be potentiated by high sulfate in the drinking water. A condition similar to blind staggers has been associated with the feeding of diets high in magnesium sulfate. A blind staggers–like syndrome has been associated with the consumption of water from saline seeps high in magnesium sulfate. Moreover, alkali disease and blind staggers have been associated with highly saline seeps and similar areas from the beginning.

By all indications, polioencephalomalacia in cattle is often related to thiamine deficiency (Dickie et al., 1979; Galitzer & Oehme, 1978; Peirson & Jensen, 1975). It is possible that thiamine depletion is the reason blind staggers, as described by Rosenfeld and Beath (1964), is observed in cattle and sheep. Sulfates may enhance thiamine destruction.

This line of reasoning suggests that additional research is needed to clarify the etiology of Se poisoning in livestock, especially as it pertains to the aspects regarding blind staggers.

SUMMARY

Selenium-containing plants have long been recognized for their toxic effects on animals. Selenium poisoning was probably first described in the USA in 1856 in U.S. cavalry horses grazing near the Missouri River in the Nebraska Territory. Reports of similar intoxication over the next 75 yr led to the investigation of Se by the agricultural experiment stations of South Dakota and Wyoming and the U.S. Department of Agriculture. This research led to the identification of Se as the toxic element in many of these reported poisonings. It also demonstrated that Se was the toxin found in some plants growing on certain soil types of the western USA.

Selenium poisoning was classified according to the toxic effects it had on livestock. Acute poisoning results from consuming excessive amounts of plants containing high amounts of Se over a short period of time. Chronic intoxication was divided into two syndromes—alkali disease and blind staggers. Alkali disease was associated with the consumption over an extended period of time of plants containing small amounts of Se whereas blind staggers was associated with grazing plants containing larger amounts of Se. Evidence supports the concept of alkali disease, but is somewhat nebulous for blind staggers.

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