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Ecological Aspects of Selenosis on Rangelands

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INTRODUCTION

Plants containing high concentrations of selenium (Se) 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 became swollen and dropped off when they grazed plants growing in certain areas (Rosenfeld and Beath 1964). Loss of hair and nails in humans, presumably suffering from chronic Se ingestion was described in Colombia by Father Simon Pedro in 1560 [National Research Council (NRC) 1976, 1983]. Guang-Qi (1987) has also described and illustrated chronic selenium toxicosis in some Chinese people.

Selenium poisoning was probably first described in the United States in 1856 by Dr. T. C. Madison, a physician with the U.S. Calvary stationed near the Missouri River in the Nebraska Territory. Dr. Madison described loss of mane and tail hair and sloughing of hooves in calvary horses grazing in areas later shown to be high in Se (Rosenfeld and Beath 1964). Reports of similar intoxications over the next 75 years 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 these maladies were due to the consumption of seleniferous plants by foraging livestock (Moxon 1937; Anderson et al. 1961; Beath 1982). Major areas of selenosis in the United States are identified in Figure 21.1.

Low levels of Se may also be a problem. Selenium was shown to be an essential nutrient in 1957 (NRC 1983). Since then, many areas of the United States and the world have been identified where plants or feeds do not contain

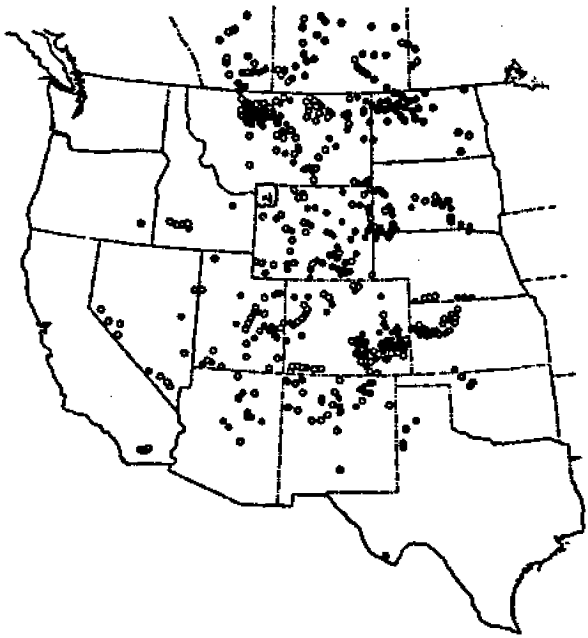

FIGURE 21.1
Distribution of seleniferous vegetation in the western United States and Canada (adapted from Rosenfeld and Beath 1964). Each open dot represents the place of collection of a plant specimen containing 50 to 500 mg Se/kg; each solid dot, specimens containing more than 500 mg Se/kg.

sufficient Se to meet animal requirements. When this occurs, animal performance suffers, e.g., reduced weight gain and poor reproduction. Young growing animals may develop potentially fatal white muscle disease, while poultry show depressed growth and exudative diathesis. In these situations, Se intake may be supplemented by fertilizing pastures or crops, supplementing feed, or providing an injectable form of Se directly to the animal. Readers interested in the nutritional requirements are directed to NRC (1983), Gissel-Nielsen et al. (1984), and Combs and Combs (1986). This chapter describes the ecological aspects of excess Se on rangelands.

SELENIUM IN SOIL

The concentration of total Se in most soils lies within the range of 0.1 to 2 mg Se/kg. However, high concentrations of total Se (1,200 mg Se/kg), of which 38 mg/kg occurred as water-soluble selenate, have been reported in seleniferous areas of the world (Lakin and Byers 1948, Swaine 1955). Soils developed from the Cretaceous shales of South Dakota, Montana, Wyoming, Nebraska, Kansas, Utah, Colorado, and New Mexico tend to have high Se values ranging from 2 to 10 mg Se/kg.

The total Se taken up by plants is closely correlated to the amount of ammonium-DTPA extractable soil Se (Soltanpour and Workman 1980). This soluble portion may not relate to the total soil Se (Lakin 1972). For example: the Se content of some Hawaiian surface soils varies from 1 to 20 mg/kg, but this Se is unavailable for vegetation because of its complexation with the iron and aluminum minerals (Anderson et al. 1961; Rosenfeld and Beath 1964).

The chemical forms of Se present in soils and sediments are closely related to the oxidation-reduction potential and pH of the soil (van Dorst and Peterson 1984). Inorganic Se exists in the selenate or selenite form in aerated, alkaline soils that predominate on western rangelands. In poorly aerated acidic soils, inorganic Se exists as selenide or elemental Se. The bioavailability of inorganic Se is selenate > selenite >> elemental Se = selenide.

SELENIUM IN WATER

Selenium generally occurs as a minor constituent in drinking water in a concentration range of 0.1 to 100 µg/liter (NRC 1983). Samples rarely exceed the 10 µg Se/liter upper limit established by the 1977 Safe Drinking Water Act of the Environmental Protection Agency (EPA). Rivers draining some of the seleniferous regions may contain as much as 10 µg total Se/liter (R.A. Smith, personal communication). Water from wells drilled into the Cretaceous Colorado formations of central Montana may contain as much as 1,000 µg Se/liter (Donovan et al. 1981). Waters that are high in Se are also likely to be high in total salts (Donovan et al. 1981).

Rumble (1985) reported 2.2, 10.6 and 1.1 µg Se/liter of water in coal and bentonite mine impoundments, and livestock ponds, respectively, in the Northern High Plains. Mayland (unpublished research) found 0.4 to 0.7 µg Se/liter in most

of the sampled livestock waters in the seleniferous areas of Montana and Wyoming; however, several reservoir samples contained as high as 270 µg Se/liter. That high value is many times greater than the EPA (1977) allowable value of 10 µg Se/liter in drinking water, but it is only three times the 100 µg/liter level of Se in blood considered adequate for animal requirements.

SELENIUM IN PLANTS

Accumulators vs. Nonaccumulators

Rosenfeld and Beath (1964) and Shrift (1973) divided plants into three groups on the basis of their ability to accumulate Se when grown on high-Se soils. These groupings are somewhat arbitrary as some of the plants listed may appear in more than one of the groups.

Group I plants include the so-called Se accumulator or indicator plants. These plants grow well on soil containing high levels of available Se, and their presence is generally indicative of seleniferous soils. Plants in this group include 26 species of Astragalus (Rosenfeld and Beath 1964) plus many species in the genera of Machaeranthera, Haplopappus, and Stanleya. These plants absorb relatively high concentrations of Se that may be in the hundreds and occasionally even thousands of milligrams per kilogram dry weight. Lesser amounts of Se are often found in these plants (Figure 21.2).

Plants in Group II include species of the genera Aster, Atriplex, Castilleja, Grindelia, Gutierrezia, Machaeranthera, Mentzelia, and some species of Astragalus. These plants rarely accumulate more than 50 to 100 mg Se/kg.

Plants in Group III include grains, grasses, and many forbs that may accumulate toxic amounts of Se from 5 to 12 mg/kg, but seldom more than 50 mg Se/kg, even when grown on seleniferous soil (Figure 21.2). These plants are perhaps more important than the Se accumulating plants in overall livestock management, because they are more palatable and more likely to be grazed.

Some plants that grow on seleniferous soils accumulate only low levels of Se. Examples of these plants include white clover (Trifolium repens), buffalo grass (Buchloe dactyloides), and grama grasses (Bouteloua spp.). High sulfur (S) containing plants like the Brassica species (mustard, cabbage, broccoli, and cauliflower) and other Cruciferae are relatively strong concentrators of Se.

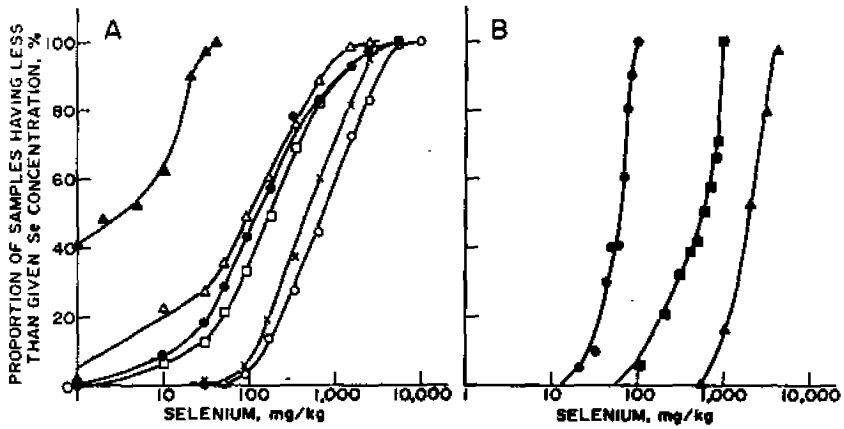

FIGURE 21.2
Proportion of samples having less than given Se concentration (%).

A. ▲—▲ data for western wheatgrass (Pascopyrum smithii) and Sandberg bluegrass (Poa secunda) sampled from seleniferous areas of Montana and Wyoming by Mayland (unpublished data); △—△ Stanleya spp.; ●—● Xylorrhiza section of Machaeranthera; □—□ Astragalus bisulcatus; X—X Astragalus pectinatus; and ○—○ Conopsis section of Haplopappus: latter data adapted from Rosenfeld and Beath (1964).

B. ●—● data for vegetative wheat (Triticum aestivum), ■—■ Astragalus bisulcatus, and ▲—▲ Astragalus pectinatus reported in plants from North Dakota by Lakin and Byers (1948).

Absorption of Se and S by plants may be correlated because of their similar chemistries (Shrift 1973).

Plant Response to Selenium Species

Gissel-Nielsen (1973) reported that more selenate than selenite was taken up by plants. There was a rapid decline in the bioavailability of the selenate form, presumably because it was converted to less available forms. Adding sulfate to the soil greatly decreased the uptake of selenate

but had a lesser effect on selenite uptake. The ratio of Se in roots to Se in tops shows that Se was more readily translocated from the roots when taken up as selenate than as selenite.

Hamilton and Beath (1963) measured Se uptake by rangeland forbs and grasses from selenite, selenate, and organic (ground Astragalus bisulcatus plus A. preussii) Se sources. Selenate was generally absorbed more efficiently than selenite, and organic Se was absorbed least efficiently. However, plants identified in Groups I and II (see above) readily absorbed large amounts of organic Se provided as finely ground mixtures of A. bisulcatus and A. preussii added to the soil.

Plant Response to High Bioavailable Selenium

Selenium is known to be required for animal health but is not yet considered an essential element for plant growth. Several studies suggest that Se is required at least for the accumulator plant species. Trelease and Trelease (1938, 1939) reported a pronounced stimulating effect in the growth of Astragalus racemosus and A. pattersonii (grown in nutrient culture) when up to 9,000 µg Se/liter, as selenite, was provided. Both species are identified as Se accumulator plants. However, A. crassicarpus (identified as A. succulentus by Rosenfeld and Beath 1964), which is a nonaccumulator, was poisoned by Se at rates as low as 300 µg/liter.

These findings led Trelease and Trelease (1938, 1939) and others (Rosenfeld and Beath 1964, Shrift 1969) to suggest that Se might be an essential element for the Se indicator plants. This apparent Se requirement by the accumulator species may be confounded by a Se-phosphorus interaction, whereby the increased growth in the presence of Se could have been due to a depression of phosphorus toxicity by selenite (Broyer et al. 1972).

If either A. bisulcatus (accumulator) or A. crotolariae (nonaccumulator) has a requirement for Se, the critical level probably would be less than 80 µg Se/kg dry plant tops (Broyer et al. 1972). This value is of the same order reported for alfalfa and/or subterranean clover (Trifolium subterraneum) (Broyer et al. 1966) and much less than the value suggested by Trelease and Trelease (1939).

Selenium Compounds in Plants

Shrift (1973) has summarized the findings on many chemical compounds of Se isolated from plants. Much of the Se in nonaccumulating species is found in the form of protein-bound selenomethionine. In contrast, the Se in accumulator plants is mostly water soluble and is not associated with protein. Formation of Se-methylselenocysteine has been found to occur only in accumulators and has been suggested as a chemotaxonomic basis for distinguishing accumulators from nonaccumulators. Another distinction between these plant groups is the existence of a large amount of selenocystathionine in accumulator species and only trace amounts in nonaccumulator plants (Shrift 1969, 1973).

There is little incorporation of the selenoamino acids into the proteins of the accumulator plants tested (Peterson and Butler 1962). However, extensive protein incorporation of the selenoamino acids occurred in ryegrass (*Lolium perenne*), wheat (*Triticum aestivum*), and clover. Lewis (1976) suggested that Se accumulator species have evolved a detoxification mechanism, whereby Se is excluded from protein incorporation, while nonaccumulator species do not have this mechanism. Selenium incorporation into proteins could result in alteration of the protein structure, inactivation of the protein, and eventual poisoning of the plant (Peterson and Butler 1962).

The Se metabolites in plants are analogs of S compounds. Nevertheless, Se metabolism in plants cannot be identified from known mechanisms involving S metabolism (Shrift 1973). It is now known that many isolated enzyme systems can utilize S and Se analogs interchangeably. What was traditionally thought to be the mechanism of Se toxicity, namely, a general interference with the enzymes involving S assimilation, has proved to be more complex.

SELENIUM ABSORPTION IN ANIMALS

Selenium toxicosis in aquatic and terrestrial animals has been well described in several reviews (NRC 1983; Sorensen 1986; Ohlendorf et al. 1986). Generalizations concerning the acute toxicity of Se to animals are exceedingly difficult to make because of the large array of chemical compounds, aqueous solubility, methods of administration, animal species, and variability within species (Combs and Combs 1986). However, selenite,

selenate, and several seleno-amino acid sources have the greatest bioavailability, while the reduced forms of Se have the least bioavailability. Feedstuffs vary with respect to Se bioavailability, with most plant sources being moderately to highly available, while the Se in soybean, meat, and fish is only slightly available. The various sources produce similar toxicological effects (Moxon and Rhian 1943).

The toxicity of Se-containing plants cannot be evaluated solely on total Se content. There are several Se-accumulating plants such as the seleniferous Astragalus and Happlopappus that contain additional toxins that may complicate the evaluation of Se toxicity. In general, Se as selenomethionine is more readily absorbed than when ingested as selenite, selenate, or selenocystine (NRC 1983). Nearly one-half of the Se in wheat grain is present as selenomethionine (Olson et al. 1970). The Se in wheat is more readily available to animals than is the Se in selenocystine, Astragalus, or fish meal (Combs and Combs 1986, NRC 1983). Selenium from plant forms is generally more available to animals than that from animal forms (Combs and Combs 1986).

As in plants, Se in animals interacts with other trace elements. Frank et al. (1986) found 20% lower liver-copper concentrations in cattle supplemented with 0.58 mg Se/kg diet compared with 0.18 mg Se/kg in the control diet over a seven to eight month period. This has practical applications because copper deficiency has been reported in areas where cattle and sheep are often supplemented with Se. A synergistic relationship was reported by Mayland et al. (1986) when high, but not toxic, levels of Se increased the absorption of lead and its subsequent toxicity in mature sheep.

Increasing dietary S intake has been observed to increase the clinical incidence of white muscle disease. The addition of 3.3g S/kg (as Na_2SO_4) to the diet reduced the availability of 0.17 mg Se/kg (as Na_2SeO_3) to lambs (Hintz and Hogue 1964). Halverson et al. (1962) reported that the addition of sodium sulfate to diets containing 10 mg Se/kg reduced the toxicity to rats (Rattus rattus) when Se was added as selenate but not when it was added as selenite or as seleniferous wheat. However, the efficacy of sulfate-S supplementation of larger animals ingesting seleniferous feeds has not been tested.

Selenium toxicity (i.e., that of SeO_2 or Na_2SeO_3) has been reduced in rats by feeding rather high levels of arsenic, copper, iron, silver, or some organic feedstuffs like Torula yeast and linseed oil meal (Combs and Combs

1986). The active factor in linseed oil meal appears to be a cyanogenic glycoside (Palmer et al. 1980). Arsenic appears to increase the biliary elimination of Se (Combs and Combs 1986). There are many interacting factors associated with increasing the tolerance of animals to various forms and sources of Se. In large animals, arsanilic acid supplementation has been somewhat effective in protecting pigs against Se toxicosis during reproduction and growth (Wahlstrom and Olson 1959a, 1959b). Even though some livestock grazing on seleniferous forage are supplemented with arsenic (H.F. Mayland, unpublished research), there is no experimental evidence that arsenic or any of the other factors discussed above have any beneficial effects on reducing Se toxicosis in grazing animals.

FECAL AND URINARY SELENIUM

Urine is the primary route of Se excretion by monogastric animals, regardless of whether the Se is given orally or injected. The main route of Se excretion in ruminants, though, is a function of the method of administration and the age of the animal (NRC 1983). When Se is ingested by ruminants, most of it is excreted in feces. In contrast, Se that is injected either intravenously or subcutaneously into ruminants is excreted mostly in urine. Lambs (Ovis aries) and, presumably, calves (Bos taurus) that have not developed rumen function can excrete 66 to 75% of the orally ingested Se in the urine. It is likely that exogenous fecal Se is predominantly Se which has been reduced to an unavailable form such as elemental Se (Langlands et al. 1986). Rumen organisms undoubtedly contribute to this age effect.

Nearly all of the Se excreted in the feces of ruminants is in an insoluble form, and very little is available for uptake by plants. Peterson and Spedding (1963) showed that during a 75-day period, less than 0.3% of the Se taken up by three pasture species originated from the Se contained in sheep manure.

Trimethylselenonium ion ($TMSe^+$) is the major urinary Se metabolite (NRC 1983). When added to nutrient solutions, $TMSe^+$ was absorbed and translocated to leaves and stems but not to the grain of wheat (Olson et al. 1976a). Large differences were observed in Se uptake by barley, wheat, and alfalfa when $TMSe^+$ was applied in a soil-pot study in the greenhouse. Olson et al. (1976a) noted that very little of the Se from $TMSe^+$ was absorbed by plants. In addition, some

of the $TMSe^+$ was lost to the atmosphere through volatilization from the plant. It is likely that the $TMSe^+$ excreted in animal urine contributes little biologically active Se to plants because it is not metabolized.

A portion of the $TMSe^+$ added to soil was biologically volatilized, and this loss was increased by liming of the soil at rates to increase the pH from 5.45 initially to values approaching pH 7.05 (Olson et al. 1976b). In addition, 30 to 50% of the $TMSe^+$ added to several different soils was sorbed to the soil particles during a 21-day period. The biologically inactive $TMSe^+$ in urine plus the stimulation of plant growth by the added nitrogen and sulfate in urine may explain the lowered Se content in grass growing on urine patches (Joblin and Pritchard 1983).

VOLATILE SELENIUM

Volatile Se compounds are naturally released into the atmosphere as a result of biological activity in aquatic (Chau et al. 1976) and terrestrial ecosystems (Abu-Erreish et al. 1968; Shrift 1973; Doran and Alexander 1977). The pathways for Se volatilization in higher plants have been reviewed by Lewis (1976). The volatile compounds include dimethyl selenide (DMSe), which produces the garlicky odor associated with Se accumulator plants. Also included are dimethyl diselenide, dimethylselenone, methane selenol, and hydrogen selenide (Zieve and Peterson 1981, 1984c). Dimethyl selenide is volatilized from soils by microbial activity (Zieve and Peterson 1984b) and sorbed on the surfaces of soil colloids or lost to the atmosphere (Zieve and Peterson 1985). Dimethyl selenide is also absorbed by plants through the leaves (Zieve and Peterson 1984a), but this source accounts for only a small portion of the Se in most plants (Zieve and Peterson 1986).

SELENIUM INTOXICATION

All livestock and humans are known to be susceptible to Se poisoning. However, poisoning is most likely to occur in animals grazing seleniferous forage. Poisoning may occur in poultry and swine as a result of including seleniferous grain in their diet. The Food and Nutrition Board of the National Academy of Sciences (1980) has suggested 5 mg Se/kg diet as the critical level between toxic and nontoxic feeds.

Selenium intoxication in grazing animals has been classified by Rosenfeld and Beath (1964) as follows: (1) acute intoxication; and (2) chronic intoxication, the latter including two syndromes identified as alkali disease and blind staggers.

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. The signs observed are related to the acuteness of the intoxication.

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, horses, hogs, and poultry is said to be associated with the consumption of seleniferous forages such as grasses and crops in which the Se is bound to protein and is relatively insoluble in water. Blind staggers is said to occur in cattle and possibly sheep and is thought to be associated with the consumption of Se indicator plants wherein the Se is in a water soluble form (Rosenfeld and Beath 1964).

Alkali Disease. The condition described by Dr. T.C. Madison in 1856 (Rosenfeld and Beath 1964) was later known as alkali disease and is now known to be chronic Se poisoning. This form of Se poisoning is still referred to as alkali disease. The term alkali disease was coined by the early settlers of the semi-arid Great Plains of the United States (Moxon 1937). The early pioneers associated the disease with waters from alkali or saline seeps and with alkali spots in the soil. In fact, water from some of the saline seeps in the northern Great Plains may contain an average of 300 µg Se/liter (Miller et al. 1981).

It was not until the early 1930s that alkali disease was shown to be caused by grazing 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 mg Se/kg over an extended period of time. According to these investigators, the Se producing this disease condition is bound to plant protein and is therefore relatively insoluble in water. However, alkali disease has been produced by feeding soluble sodium selenate (Moxon 1937, Hartley et al. 1984). Alkali disease is characterized by dullness, lack of vitality, emaciation, rough coat, 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 and Beath 1964).

According to Olson (1978), reduced reproductive performance is the most significant effect of alkali disease in livestock. The effect on reproduction may be quite marked, yet the animal involved may not show typical signs of alkali disease. Reproduction has been adversely affected in cattle (Dinkel et al. 1963), swine (Wahlstrom and Olson 1959a, 1959b), and mice and rats (Schroeder and Mitchener 1971, Halverson 1974). The forages responsible for the reproduction losses in cattle probably contained between 5 and 10 mg Se/kg.

As in many other situations, sheep respond differently to Se than cattle. Glenn et al. (1964) and Maag and Glenn (1967) reported that feeding as much as 0.55 mg Se/kg body weight as selenate did not lower the conception rate nor noticeably affect the developing fetuses of two-year-old ewes. This rate (0.55 mg/kg) corresponds to a dietary intake of 15 to 20 mg Se/kg forage. The Se was administered with a linseed oil meal carrier which has since been shown to contain several compounds that reduce the toxicosis of Se (Palmer et al. 1980). Thus, compounds in the linseed oil may have reduced any toxicological effect of the selenate on reproduction in sheep.

Ranch managers have learned through experience that some areas or pastures are "hotter" or more seleniferous than others and that grazing time on these areas should be limited to a few weeks to minimize the effects of the high Se levels in the forage. Many of these ranges are now utilized by stocker cattle rather than cow-calf units (H.F. Mayland, unpublished research). This shift away from cows may be a response to reduced reproductive performance

and/or other economic factors (Wahlstrom and Olson 1959a, 1959b). More individual attention is often given to horses than to cattle, and managers are very careful which pastures these animals are allowed to graze. Even so, the effective life of these animals may be shortened because of chronic selenosis.

Blind Staggers. This disorder, according to Rosenfeld and Beath (1964), results from livestock grazing moderate amounts of indicator plants over extended time periods. Blind staggers has been described in cattle and sheep but not in horses, hogs, or poultry. Many of the selenium compounds occurring in these plants are readily extractable with water.

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 degrees of blindness, labored respiration, abdominal pain, grinding of the teeth, salivation, emaciation, and death (Rosenfeld and Beath 1964). The three stages of intoxication are not clearly defined in sheep, as sheep exhibit few clinical signs and may die suddenly when intoxicated (Moxon and Olson 1974).

Blind staggers has been reported in Wyoming but not in other western states having problems of Se poisoning in livestock (Moxon and 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 from Se 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 appellation polioencephalomalacia is appropriate until the etiological and pathological factors are clearly established." (p. 321)

CONCLUSIONS

Large areas of the semiarid western United States are underlain with seleniferous geological materials of Cretaceous origin. Soil, water, and plants associated with these materials are also likely to be seleniferous. Plants growing on these areas may vary significantly in their ability to accumulate Se. Those plants accumulating high (hundreds of mg Se/kg) or moderate (50 to approximately 100 mg Se/kg) amounts are termed primary and secondary accumulators, respectively. Most of the Se in these plants occurs in water-soluble forms like selenate and several seleno-amino acids. Ingestion of these plants at a rate providing about 10 to 15 mg Se/kg body weight can be lethal. Fortunately, these plants are generally not very palatable. The third group is composed of the grasses, small grains, and many of the palatable forbs. These plants may contain 5 to 10 mg Se/kg, much of which is incorporated into protein.

Contrary to earlier conclusions that Se was an essential element for plant growth, it has been shown that the presence of Se in the soil solution ameliorates excess phosphorus uptake and subsequent phosphorous poisoning in the accumulator plants.

Animal intoxication varies between and within animal species and Se sources. Both chronic and acute forms are recognized. Chronic forms are identified as alkali disease and blind staggers. Alkali disease (characterized by loss of hair, inappetence, and elongated hooves) occurs when animals, over long periods of time, graze Group III forages containing organic Se in moderate amounts. Consumption of plants in Groups I and II may produce blind staggers in cattle and sheep but not horses and swine. This neurological disorder has been attributed to the high levels of inorganic Se present in these plants. However, some of these plants also contain other toxic principles that may produce blind staggers. Research is needed to determine the cause of this neurological disorder.

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