ABSORPTION OF EXCESS SELENIUM AND SULFUR BY PLANTS AND ANIMALS[1]

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Abstract: High concentrations of selenium (Se) and sulfur (S) often occur in overburden soils and underlying shales associated with western coal mining areas. Knowing the role of Se and S in the soil-plant-animal system is important for proper management of mine spoil reclamation. I will discuss recent findings about Se and S forms in soil, their absorption and accumulation by plants, and their subsequent toxicity to grazing animals. Selenium absorbed by the accumulating plants is metabolized to non-protein forms, while that absorbed by the non-accumulating plants occurs predominantly as selenomethionine which is readily absorbed by animals. In animals, both acute and chronic forms of selenosis are known. Death occurs when a large dose of highly-available Se is ingested. One chronic form "alkali disease", produces symptoms of inappetence, hair loss, hardening and extension of nails and hooves, reduced weight gains, and poor reproductive performance. Rosenfeld and Beath identified "blind staggers" as another form of selenosis, but this disorder; more appropriately called polioencephalomalacia (PEM), occurs only in ruminants. However, the attribution of "blind staggers" (PEM) to excess Se is being questioned. Recent experimental evidence has shown that PEM is likely caused by excess sulfur. Cases of this disorder have been documented in the U.S. and Canada when ruminants have high sulfate intake from herbage and drinking water. Decisions regarding western mine reclamation should consider the potential for not only causing excess Se, but also excess sulfate in water, soil, and plants.

Additional Key Words: "alkali disease", "blind staggers", polioencephalomalacia, selenosis, sulfatosis, water quality

Introduction

Selenium and S have similar chemistries and are common constituents in Cretaceous geologic materials. Similar inorganic and organic forms of each element may be found in water, soil, and plants. Selenium is not required by plants (Mayland et al. 1989), but it is required for animals at dietary levels of 0.1 to 0.3 mg Se/kg (Kincaid 1995; Maas and Koller 1985; Oldfield 1989). However, concentrations in the diet greater than 3 to 15 mg Se/kg may be toxic (Combs and Combs 1986; Koller and Exon 1986). On the other hand, S is required for both plant (2 to 3 g S/kg) and animal nutrition (1 to 2 g S/kg). I will present information about the action of individual elements and their interactions that may affect both plant and animal health.

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Selenium Toxicity

In the thirteenth century, Marco Polo described a necrotic hoof disease in his horses while traveling in western China (Latham 1968). He associated the problem with the ingestion of certain plants that were generally avoided by local animals. In 1560, in Colombia, South America, Father Pedro Simon described hair and hoof loss, tender bone joints, reproductive disorders, and deaths in domestic animals (Mori 1979). The natives associated the problem with ingestion of foodstuffs grown on certain soils. The problem was documented again in the mid nineteenth century by U.S. Army Surgeon, T.W. Madison, who described necrotic and sloughed hooves and deaths of calvary horses grazing near Fort Randall, SD (Rosenfeld and Beath 1964). Anecdotal evidence suggests similar problems for the horses and mules used by Reno's troops in support of General Custer's expedition against the Indians on the Little Big Horn River (Wilcox 1944). Since then, ranchers have associated the toxicosis with saline seeps and alkaline outcrops common to much of the northern Great Plains; thus the name "alkali disease". By 1931, researchers identified "alkali disease" as chronic Se toxicosis (selenosis) characterized by hair and hoof loss and poor growth and reproduction (Rosenfeld and Beath 1964). Chronic selenosis still occurs in ruminants and monogastrics inhabiting seleniferous areas around the world and occurs in all types of animals (Rosenfeld and Beath 1964). It is more frequently encountered in grazing animals and is known to occur throughout the northern Great Plains and elsewhere where plants are rooted in Cretaceous materials.

A second disorder occurring in the northern Great Plains is "blind staggers", more correctly referred to as polioencephalomalacia (PEM). It results in varying degrees of neurological dysfunction including vision impairment. This neurological dysfunction occurs only among ruminants and has been attributed to excess Se in the forage (Rosenfeld and Beath 1964). They noted that seleniferous grains and grasses produced "alkali disease" (chronic selenosis) but that seleniferous indicator plants produced "blind staggers" (PEM). More recent research (James et al. 1994) has shown that both seleniferous grasses and Se-indicator plants produce chronic selenosis. However, the PEM disorder is now believed to be the result of ingesting excess S rather than Se (Beke and Hironaka 1991; Gooneratne et al. 1989; Gould et al. 1991; Hamlen et al. 1993; James et al. 1994; Olkowski et al. 1991; Raisbeck, 1982; Sager et al. 1990).

The initial confusion that Se caused PEM may have arisen because "blind staggers" occurs in areas where Se poisoning also occurs. It is possible that Beath associated "blind staggers" with the seleniferous areas and simply concluded that this was another symptom of excess Se. Limited experimental evidence was collected by Beath and coworkers in an attempt to reproduce the PEM. I knew Orville Beath and am familiar with the Laramie Plains region. Some of the locally derived ground water contains high concentrations of Glauber's salts ($Na_2SO_4 \bullet 10H_2O$) and Epsom salt ($MgSO_4 \bullet 7H_2O$). It is quite likely that Beath's experimental animals unknowingly received big doses of S in the drinking water and in the seleniferous forage. Thus, the misdiagnosis. Nevertheless, it is still commonly assumed that both "alkali disease" and "blind staggers" are forms of selenosis. This problem will be discussed at greater length later in this paper.

Acute Se toxicosis has been observed in grazing animals (Rosenfeld and Beath 1964) and in hatchlings of waterfowl nesting near reservoirs or evaporation ponds containing seleniferous sediments and aquatic vegetation (Ohlendorf 1989). High Se levels might also be a problem for fish and other aquatic life in ponds receiving fly ash washdown from coal-combustion or refuse incineration plants. Seleniferous conditions might also occur in seepage waters flowing from seleniferous geological materials (Mayland et al. 1989). The immediate and long term risk of Se toxicosis may vary from site to site. Studies conducted by Dheher and Finkelman (1992) showed that Se solubility is reduced with time in some surface coal mines. Continued monitoring of these areas appears warranted.

New reports of selenosis or potential selenosis still appear. Recent reports have documented chronic selenosis in horses in western Iowa (Witted et al. 1993) where the Cretaceous subsoils under the pastures contained

high concentrations of soluble Se. High Se levels have also been measured in Brazil nuts (*Bertholletia excelsa*, Secor and Lisk 1989). I recently measured 92 and 419 μg Se/g respectively, in the pericarp and fruit of a Brazil nut tree (#6411) growing in Reserve 1501 about 41 km north of Manus, Brazil. To find excess Se uptake by plants growing in Amazonia confirms the reports of Father Simon, but seems contrary to our present knowledge of Se chemistry. This illustrates the need for further research on Se chemistry and its absorption by plants and animals, including humans.

Humans have also been victims of selenosis. Common signs of poisoning are loss of hair and nails, lesions of the skin, and nervous system disorders. Yang et al. (1983) reported an outbreak of selenosis caused by drought-induced failure of the China rice crop. Water stress, and increased concentrations of Se in the soil solution undoubtedly increased Se concentration in the available rice, corn, and other vegetables. Concentration of Se in the soil water under drought may be similar to the experience of Erdman and coworkers (1991) who found that plants growing on water-stressed seleniferous soils contained 20 to 30 times greater Se concentrations than when grown on the same soils a year later when water was readily available. I am not aware of any acute selenosis in the human population in the U.S. In this country, the high quality of human nutrition may be attributed to the varied diets and foods produced on different soils. However, there are reports of chronic selenium toxocosis occurring in copper-refinery workers (Holness et al. 1989).

Grazing animals are generally restricted to the quality of herbage growing on soils in the area grazed. Both chronic and acute selenosis may thus occur. I estimate that animal-production losses to selenosis in U.S. are valued at $10 million annually. The only treatment is to change the source of feed. Some selenized animals can reduce their Se body burden via urinary excretion, even when intake continues at relatively high rates (Stowe et al. 1992; Mayland, personal observation). Another means of detoxification is attributed to anaerobic bacterial reduction of selenate and selenite to inorganic red Se^{o}, thus reducing the bioavailability (Rasmussen and James 1993, 1994).

Selenium Deficiency

The nutritional value of Se was first recognized in 1957 when it was found to have a complementary role to vitamin E in preventing dietary hepatic necrosis and exudative diathesis in rats and chicks (Combs and Combs 1986). Later, Se was shown to be nutritionally important for all animals. Selenium is an essential constituent of the biologically important enzyme glutathione peroxidase, which along with vitamin E, decreases the oxidative stress in the body. Recently, additional metabolic roles have been identified for Se (Arthur and Beckett 1994).

Combs and Combs (1986) review Se deficiencies affecting fish, laboratory animals, poultry, livestock, and humans. Clinical signs include reduced appetite, growth, production, and reproductive fertility, a general unthriftiness, and muscular weakness. Specific disorders include exudative diathesis and increased embryonic mortality in birds. Nutritional muscular dystrophy is found in birds, fish, and animals. Retained placenta is reported in Se-deficient cows, while mulberry heart disease is noted in pigs. White muscle disease is the common term applied to sheep, cattle, horses, and other herbivores having inadequate Se in their diets. Selenium deficiencies cost U.S. cattle and sheep producers an estimated $545 million and swine and poultry producers $82 million annually (Ullrey 1980).

Severe nutritional Se deficiency is associated with endemic juvenile cardiomyopathy (i.e., Keshan disease) in young children from a discrete area in China. Se may also be involved in the etiology of chondrodystrophic disease (i.e., Keshan-Beck disease) in young Chinese children. Changing cropping practices and importing food containing higher levels of Se have significantly reduced the incidence of these Se deficiencies in these people (Tan and Huang 1991).

Several syndromes in cattle and sheep have been classified as Se-responsive conditions based on current information (Mayland 1994). Some of these syndromes are complex because they involve interactions with other nutrients. Scientists have just begun to learn about the involvement of Se with the immune system (Nicholson et al., 1993). Blood levels of over 100 µg Se/L in cattle (Nicholson et al., 1993) and 180 to 230 µg Se/L in swi365ne (Wuryastuti et al., 1993) are needed to maintain optimum immunocompetence. Measures of whole-blood Se and SeGSH-px in the hemoglobin are useful in interpreting the Se nutritional status in cattle and sheep. Similar criteria are used in determining Se status of human nutrition (Combs and Combs, 1986)

Producers and veterinarians have several methods for treating Se-deficient animals (Maas and Koller 1985). The most commonly used therapies in the U.S. are *i*) injectable Se products, *ii*) salt-mix formulations with supplemental Se, or *iii*) total-ration formulations with supplemental Se. In 1993, The U.S. Food and Drug Administration limited supplementation levels to only 0.1 mg/kg Se in the diet. On 30 September 1994, action was taken suspending that ruling until 31 Dec. 1995. Meanwhile, 0.3 mg/kg is the maximal amount of Se allowed in complete feed for all poultry and animals.

Soils, plants, animals, and humans in New Zealand and Finland are deficient to marginally deficient in available Se. These countries have resorted to Se fertilization of crop-producing areas to increase Se concentration in pasture, cereal, and other food crops (Mayland et al. 1989). Increasing soil-Se levels has effectively increased the general level of Se in feedstuffs for both animals and humans (Mayland et al. 1989). Sulfur deficiencies in the U.S. Pacific Northwest have necessitated S fertilization. The added S has reduced the bioavailability of soil Se and increased the incidence of Se deficiencies in calves and lambs (Mayland, personal observation).

Selenium in Soils

Depending on the redox potential of the soil, Se occurs in many different forms. Concentrations in most soils lie within the range of 0.01 to 2 mg Se/kg. However, some seleniferous soils may contain as much as 38 mg Se/kg as water-soluble selenate. Other soils like those in Hawaii and Ireland also contain high levels of total Se, but it is relatively unavailable to most plants (Mayland et al. 1989). Inorganic Se forms like SeO_4, SeO_3, and Se^o have a wide range of solubility in water and subsequent bioavailability to plants and animals. Selenium concentrations in plants are related approximately to broad areas described by geology and soils.

Organic Se forms, including selenomethionine, have been extracted from soils and represent an important source of plant-available Se (Abrams et al. 1989, 1990a, 1990b). Selenomethionine is two to four times more available to plants than selenite (Williams and Mayland 1992) and its uptake is under metabolic control (Abrams et al. 1990a, 1990b). Selenocystine is less bioavailable than selenomethionine (Williams and Mayland, 1992). In some soils, nearly 50% of the Se may be in organic forms (Abrams et al., 1990a, 1990b). Identifying these forms will be challenging, but necessary if scientists are to better understand Se cycling.

Selenium in Plants

Among plants growing on moderately low-Se soil, alfalfa accumulates more Se than many other forage plants (Mayland et al. 1989). This characteristic may be related to differences in rooting depth and to genetic traits that affect the absorption and translocation of Se to shoots. Sulfur fertilization of legumes will often reduce Se uptake and concentration in the forage (Westermann and Robbins 1974). McQuinn et al. (1991) estimated that Se concentrations could be increased 19% in tall fescue (Festuca arundinacea Shreb.) through genetic selection. This species is adapted to most of the Se-deficient pastoral areas in the United States. Genetic selection in this forage species promises to increase herbage Se levels, thus meeting Se requirements of grazing animals in marginally

deficient areas. Similar breeding opportunities may exist in other forages. Plants exhibit genetic differences in Se uptake when growing on seleniferous soil. Some plants accumulate surprisingly low levels of Se. For example, white clover (Trifolium repens L.), buffalograss (Buchloe dactyloides [Nutt.] Engelm.), and grama (Bouteloua spp.) are poor accumulators of Se. On the other hand, S-rich plants like the Brassica spp. (mustard, cabbage, broccoli, and cauliflower) and other Cruciferae are good concentrators of Se (NAS, 1983).

Rosenfeld and Beath (1964) identified three plant groups based on their ability to accumulate Se when growing on Se-rich soils. The first two groups of plants were identified by their potential to accumulate moderate or very high concentrations of Se. These are the plants that grow successfully on soil containing high levels of available Se. The presence of these plants, and the characteristic dimethylselenide odor (garlic smell), are indicative of seleniferous soils (Lewis 1976). These plants have a different metabolic pathway that shunts Se into non-protein forms (Mayland, 1994). A Se requirement has not been shown for any plants (Läuchli 1993). Plant genera that can accumulate very high concentrations of Se include many species of Astragalus, Haplopappus, Machaeranthera, and Stanleya. On a dry weight basis, these species absorb high concentrations of Se, from hundreds to occasionally even thousands of milligrams per kilogram. These plants are found in semi-arid environments throughout west-central North America and other continents. The absence of deep percolation, neutral to alkaline soil pH, and oxidative conditions have allowed much of the soil Se to remain in place. Precipitation in excess of evapotranspiration normally leaches out the soluble Se salts. An exception seems to occur in the Amazonian Plateau where several members of the Amazonian Lecythidaceae family (Brazil nut) also accumulate high concentrations of Se (Mori 1979).

Plant genera having the potential to accumulate moderately high concentrations of Se include many species of Aster and some species of Astragalus, Atriplex, Castilleja, Grindelia, Gutierrezia, Machaeranthera, and Mentzelia. They rarely concentrate more than 50 to 100 mg Se/kg. The remaining are non-accumulator plants and include grains, grasses, and many forbs that do not usually accumulate more than 50 mg Se/kg when grown on seleniferous soil.

Alfalfa (Medicago sativa L.) is commonly grown in seleniferous areas like the seleniferous Kendrick Reclamation Project area of central Wyoming. A Se survey of alfalfa conducted there during 1988 reported a range of 0.1 to 40 mg Se/kg with a median of 0.9 mg Se/kg (Erdman et al. 1991). However, the next year, alfalfa that had previously contained 17 and 25 mg Se/kg, now contained only 0.7 and 0.2 mg Se/kg, respectively. The significant reduction in Se values was attributed to percolation of soluble Se beyond the rooting zone and to dilution in the plant material resulting from increased dry matter production.

Infrequent incidence of selenosis in animals has been reported on the Kendrick Project in central Wyoming. Tolerance to high Se levels varies considerably among individual animals and birds. In addition, experimental evidence suggests that some animals can accommodate high levels of dietary Se after observing some symptoms of chronic toxicosis, such as lameness and hair loss (H. F. Mayland, personal observation). The tolerance to increased Se intake may result from the activity of Se-reducing bacteria such as those found in Se tolerant heifers by Rasmussen and James (1993, 1994). The selenate and selenite forms are reduced to red selenium metal which has very low bioavailability.

Many different Se compounds have been identified in plants (Shrift 1973). Much of the Se in non-accumulating species is found as protein-bound selenomethionine. In contrast, the Se in accumulator plants is mostly water-soluble and found in nonprotein forms like Se-methylselenocysteine. Only trace amounts of the latter compound are found in non-accumulator species. Selenomethionine, selenocystine, and possibly Se-methylselenomethionine and selenonium have been detected in non-accumulators but not in the accumulators tested (Lewis 1976). The Se metabolites in plants are generally analogs of S compounds. Nevertheless, Se metabolism in non-accumulator plants cannot be identified from known mechanisms because of scientists' limited understanding

of the metabolic pathways for Se in plants (Shrift 1973). Two methylated-Se compounds dimethylselenide and dimethyldiselenide are respiratory products of microorganisms, plants, animals, and humans (Mayland, 1994). Hydrogen selenide (H_2Se) is another volatile Se compound. It is highly toxic, but under atmospheric conditions quickly decomposes into innocuous Se° and water (Läuchli, 1993).

Microorganisms can reduce selenate to elemental Se° and Se^{-2}. Many microorganisms, plants, and animals reduce selenite to selenide giving rise to volatile organic forms (Läuchli 1993). Dimethylselenide is the volatile and odiferous Se compound that is characteristic of Se accumulator plants. The compound is also detected in the breath of animals and humans respiring excess Se (Combs and Combs 1986; Lewis 1976; Mori 1979). I (Mayland, unpublished) detected the aroma of dimethylselenide within an hour of spraying sodium selenite on alfalfa foliage. Obviously, the selenite was rapidly metabolized to the dimethylselenide by the plants or by the microorganisms present on the plants or on ground.

Bioavailability of Se in Feces, Urine and Respiratory Products

Urine is the primary route of Se excretion by monogastric animals. The main route of Se excretion in ruminants, though, depends on the method of administration and the age of the animal (Mayland et al. 1989). 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, and presumably calves, which have not developed rumen function can excrete 65 to 75% of the orally ingested Se in the urine. As these animals develop functioning rumen systems, the micro-organisms transform the Se to unavailable forms such as elemental Se, which are then excreted in the feces. Nearly all of the Se excreted in the feces of ruminants is in an unavailable form, and very little is available for uptake by plants. Research reports summarized by Mayland et al. (1989) noted that, during a 75-day study, < 0.3% of the Se taken up by plants originated from the Se contained in sheep manure.

Trimethylselenonium ion ($TMSe^+$) is the primary urinary metabolite. This source is readily absorbed and translocated to leaves and stems of wheat, but not to the grain (Mayland et al. 1989). However, large differences were observed in Se uptake by barley, wheat, and alfalfa when $TMSe^+$ was applied in a soil-pot study in the greenhouse. Very little of the Se from $TMSe^+$ was absorbed by plants, and some absorbed $TMSe^+$ was even lost to the atmosphere through volatilization from the plant or perhaps from microbial respiration (Mayland et al. 1989). Therefore, $TMSe^+$ excreted in animal urine contributes little biologically-active Se to plants. Dimethylselenide is the principal respiratory product of animals ingesting excess Se. Dimethyldiselenide may also be respired and the proportion of the two compounds depends on the Se source (Combs and Combs 1986). Dimethylselenide is also respired by plants (Mayland et al. 1989) and accounts for the distinctive odor of Se-accumulator plants. These methylated forms are likely absorbed by plants. The Se enrichment of plants growing in Se-free nutrient culture could have occurred by foliar absorption of Se volatilized from adjacent plants growing in selenized nutrient culture (Williams and Mayland 1992).

Sulfur in Water

Polioencephalomalacia has occurred in areas where available drinking water contains excess sulfate (Hamlen et al. 1993). They reported 7,200 mg SO_4 in drinking water utilized prior to the onset of PEM. Blindness was verified in five of six affected animals. James et al. (1994) drenched cattle with sodium selenate or sodium sulfate or fed cattle *Astragalus praelongus* or selenized alfalfa hay. Four of five animals receiving the sulfate treatment became blind while none of the other animals were affected. Gould et al. (1991) reported the odor of hydrogen sulfide in eructated rumen gas associated with the onset of PEM. The role of increased sulfate intake and its interactions with other elements like copper and with thiamine metabolism have not been clearly identified.

Sulfur in Soils

Organic S constitutes more than 90% of the total S present in most surface soils (Germida et al. 1992). Sulfate adsorption is influenced by many factors. In calcareous soils, the usually abundant S is coprecipitated/cocrystallized with calcium. Mayland and Robbins (1994) attempted to maximize plant uptake of sulfate only to find that the gypsum ion activity limited the amount of soluble S available to the plants. Nevertheless, *Kochia scoparia* and several other plants contained nearly 900 mg S/kg. Kochia is often used as forage in the areas where "blind staggers" occur and was identified by Dickie and Berryman (1979) as a likely cause of PEM in range cattle. These S levels could contribute significant amounts to the S intake by animals, adding to the potential health risk. Severson and Gough (1992) and Westermann and Robbins (1974) acknowledged that S fertilization often reduces the concentration of Se in the forage, especially if soil-Se levels result in marginal Se concentration in the plant. Severson and Gough (1992) found that the S was effective in reducing the bioavailability of Se, even when both were present in high concentrations in the soil.

Sulfur in Plants

Most of the inorganic S taken up by plants is converted to organic S and used for protein synthesis (Rennenberg 1984). Protein will contain about 80% of the organic S and organic nitrogen in plants adequately supplied with both elements. Sulfur accumulates in higher plants, when applied to the soil in amounts exceeding those optimal for growth. The excess S may be metabolized to organic non-protein forms or remain as sulfate. Hydrogen sulfide can be emitted by plants when S dioxide, sulfite, sulfate, or L-cysteine is present in excess.

Summary

Selenium is an essential element for adequate nutrition and health in animals. It serves as the metal cofactor for the biologically important enzyme, glutathione peroxidase. Se deficiency reduces growth, productivity, reproduction, and even causes death in fish, birds, animals, and humans. Plants, while not requiring Se, absorb it from the soil solution and cycle it to ingesting animals. Plants differ in their Se metabolism, with most food plants converting much of the Se into protein where the Se is readily available to animals. Animals have a dietary-Se requirement of about 0.1 mg/kg in maintenance situations. The requirement increases to 0.3 mg/kg when high levels of S or other Se antagonists are present.

Animals develop a chronic selenosis when the Se concentration of the diet increases to levels greater than 3 to 15 mg Se/kg. This is a problem in some areas of the USA and elsewhere, where plants grow on seleniferous soils and accumulate excess Se. Animals feeding on these plants may develop health problems and do very poorly. Animal sensitivity to selenosis depends on animal species and preconditioning. Some plants can accumulate Se in excess of 25 mg Se/kg when grown on highly seleniferous soils and if consumed by animals would result in acute selenosis of those animals. The actual lethal concentration of Se is dose related.

"Blind Staggers," or clinically Polioencephalomalacia (PEM), in ruminants has been attributed to the ingestion of excess Se-accumulator plants. Recent investigations in Canada and the Western U.S. strongly suggest that the ingestion of excess sulfur (S) whether in diet or drinking water, will increase the risk of PEM.

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