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SELENIUM *in the* ENVIRONMENT

edited by

W. T. FRANKENBERGER JR.
University of California
Riverside, California

SALLY BENSON
Lawrence Berkeley Laboratory
Berkeley, California

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Selenium in Plant and Animal Nutrition

H. F. Mayland

Agricultural Research Service, U.S. Department of Agriculture
Kimberly, Idaho

INTRODUCTION

Selenium (Se), while not required by plants, is an essential trace element for adequate nutrition and health for fish, birds, animals, and humans. Generally, diets containing 0.1–0.3 mg/kg Se will provide adequate Se for these various animals. However, many soils are incapable of providing that amount to the plants growing on them. Animals consuming low-Se diets will be Se-deficient, grow poorly, or even die. Conversely, there are soils that provide an abundance of soluble Se. Some plants growing on these Se-rich soils may accumulate Se in excess of the 3–15 mg/kg concentration at which animals begin to show Se toxicity symptoms. This multifaceted characteristic of Se makes it imperative that scientists and policy makers recognize the deficiency, adequacy, and toxicity effects of Se on animal health. This chapter presents information about these aspects of Se in the plant and animal system.

HISTORICAL PERSPECTIVE OF SELENIUM NUTRITION

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 [1,2]. Ensuing reports were published of similar nutritional interactions between Se and vitamin E in birds and animals. In the late 1960s, a specific nutritional requirement for Se was established for chicks [3].

Eventually, Se was shown to be an essential constituent of the biologically important enzyme glutathione peroxidase (SeGSHpx) [4]. SeGSHpx, superoxide dismutase, and catalase convert free radicals to peroxides and then to water and oxygen; whereas, vitamin E scavenges the free radicals and neutralizes their potential damaging effects. Thus, low selenium intake with vitamin E deficiency increases oxidative stress and contributes to the development of oxidative damage.

Combs and Combs [5] reviewed the 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. Severe nutritional Se deficiency is associated with endemic juvenile cardiomyopathy (i.e., Keshan disease) in youngsters from a discrete area in China. Selenium may also be involved in the etiology of chondrodystrophic disease (i.e., Kaschin-Beck disease) in young Chinese children.

Toxicity

Marco Polo described a necrotic hoof disease in his horses during his travels in western China in the thirteenth century [6]. 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, reproduction disorders, and deaths in domestic animals [7]. These disorders were later attributed to Se toxicosis [7]. Father Simon also noted malformation of children and chickens and Indian women giving birth to monsters that were abandoned by their parents. The natives associated the problem with ingestion of foodstuffs grown on certain soils.

The problem was documented again in the mid-nineteenth century by a U.S. Army surgeon, T. W. Madison, who described similar necrotic and sloughed hooves and deaths of horses grazing near Fort Randall, South Dakota [8]. Ranchers associated the toxicosis with the saline seeps and

outcrops common to much of the northern Great Plains and named the problem "alkali disease." Alkali disease still occurs in ruminants and monogastrics inhabiting seleniferous areas.

By 1931, researchers identified alkali disease as chronic Se toxicosis (selenosis) characterized by hair and hoof loss and poor growth and reproduction. A second disorder occurring in the area, "blind staggers," results in varying degrees of vision impairment. This neurological dysfunction occurs only among ruminants [8] and was attributed to excess Se in the forage. However, this disorder may be the result of ingesting excess sulfur (S) rather than Se [9].

Selenium toxicosis has been observed in waterfowl inhabiting areas where sediments and aquatic vegetation contain excess Se levels. Ohlendorf [10] described embryocidal deformities in birds feeding on Se-enriched feedstuffs. Earlier reports of these deformities had stimulated investigation of both natural and anthropogenic factors associated with Se cycling.

SELENIUM IN WATER

Selenium naturally enters the food chain through water. It occurs as a minor constituent in water at concentrations ranging from <0.1 to 100 μg Se/L [11]. Few samples exceed the 10 μg Se/L upper limit established by the 1977 Safe Drinking Water Act of the U.S. Environmental Protection Agency [12]. Water derived from Cretaceous geological zones may contain as much as 1000 μg Se/L. Regional rivers that drain such areas may have concentrations approaching 10 μg Se/L [13]. Most of these areas are found in the central plains and western deserts of North America and the interior deserts of other continents. Another area of economic interest is the accumulation of seleniferous drainage waters in the San Joaquin Valley of central California. Selenium may also accumulate in other catchment areas where evaporation concentrates soluble salts.

Small amounts of Se may be found in aerosols that enter the atmosphere and contribute to global cycling of Se. This Se originates from volcanic eruptions and the fine particulates that are generated from fossil fuel combustion and the incineration of municipal wastes. Water used to wash down smoke stacks or precipitators also contains significant levels of Se [13]. Some organic forms are volatilized directly from biological activities. Eventually, these Se compounds are returned to earth. Globally, wet deposition of Se aerosols returns about 1.5 g Se/ha annually.

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 0.01–2 mg Se/kg. However, some seleniferous soils may contain as much as 38 mg Se/kg as water-soluble selenate. Other soils such as those in Hawaii, Ireland, and the Amazonian rain forest also contain high levels of total Se, but the Se is relatively unavailable to most plants [13]. Inorganic Se forms like SeO_4, SeO_3, and Se^0 have a wide range of solubility in water and subsequent bioavailability to plants and animals (Table 1). Selenium concentrations in plants are related approximately to broad areas described by geology and soils (Figure 1).

Organic forms, including selenomethionine, have been extracted from soils and represent an important source of plant-available Se [14,15]. Selenomethionine is two to four times as available to plants as selenite [16], and its uptake is under metabolic control [17]. Selenocystine is less bioavailable than selenomethionine [16]. In some soils, nearly 50% of the Se may be in organic forms [15]. Identifying these forms will be challenging but is necessary if scientists are to better understand Se cycling.

Table 1 Selenium Solubility in Water and Relative Uptake of Se by Plants from Various Sources Labeled with ^{75}Se in Pot Experiments Using a Loamy Sand Having 2.8% Organic Matter, 5.7 pH, and 0.12 mg Se/kg

Se source	Solubility of Se in cold water[a] (g/L)	Se added to soil (mg/kg)	Uptake relative to added Se (%)		
			Clover	Barley	Mustard
Se	i	2.5	0.005	0.02	0.07
SeO_2	i	0.5	1.0	0.9	1.2
K_2SeO_3	22.4	0.5	1.0	1.1	1.3
Na_2SeO_3	s	0.5	1.0	1.0	1.1
$BaSeO_3$	0.05	0.37	0.9	0.9	0.9
$FeSeO_3$	i	0.35	1.1	1.0	1.1
$CuSeO_3$	i	0.30	0.8	0.8	0.7
K_2SeO_4	390	0.50	24.	12.	24.
$BaSeO_4$	0.03	0.10	63.	27.	61.
$CuSeO_4$	68	0.13	53.	28.	48.

[a]i = insoluble; s = slightly soluble.
Source: Adapted from Mayland et al. [13].

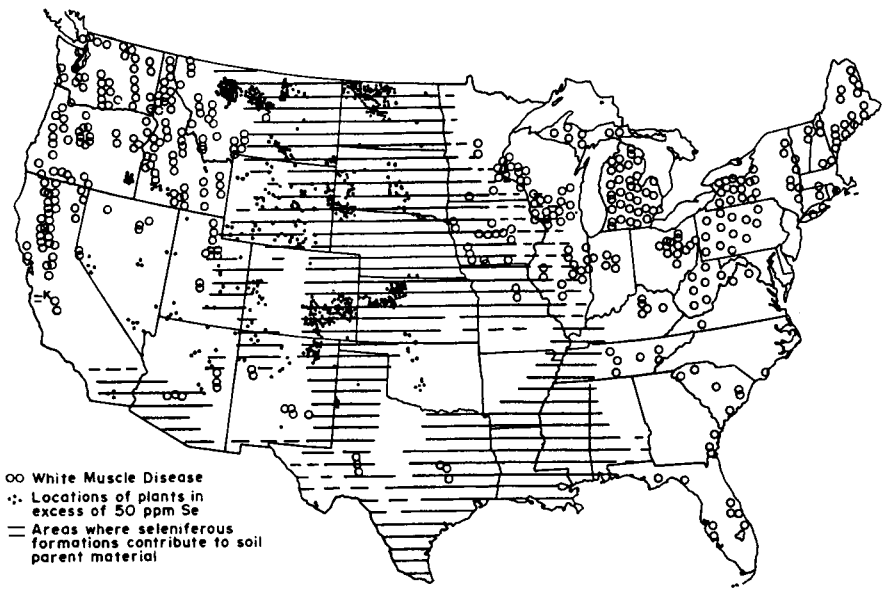

Figure 1 The geographical distribution of Se-rich soils (horizontal lines), locations where plants contain in excess of 50 μg Se/kg (solid dots), and locations of Se deficiency in animals (white muscle disease, clinically termed nutritional muscular dystrophy, open circles). (Adapted from Muth and Allaway [35].)

SELENIUM IN PLANTS

On moderately low Se soil, alfalfa accumulates more Se than many other forage plants [13]. 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 [18]. McQuinn et al. [19] estimated that Se concentrations could be increased by 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 and satisfy the Se needs of grazing animals in marginally deficient areas. Similar breeding opportunities may exist in other forages.

Plants exhibit genetic differences in Se uptake when grown 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 [11].

Rosenfeld and Beath [8] identified three plant groups according to 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 are indicative of seleniferous soils. These plants have a different metabolic pathway that shunts Se into nonprotein forms [20]. Nevertheless, a Se requirement has not been shown for any plants.

Plant genera that can accumulate very high concentrations of Se include many species of *Astragalus, Machaeranthera, Haplopappus,* 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 semiarid environments throughout west central North America (Figures 2 and 3) 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 also accumulate high concentrations of Se [7]. Investigations into Se cycling in this area of high precipitation would be interesting.

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 50–100 mg Se/kg. Nonaccumulator plants make up the third group. It includes 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 Kendrick Reclamation Project area of central Wyoming. A Se survey of alfalfa conducted there during 1988 reported a range of 0.1–40 mg Se/kg with a median of 0.9 mg Se/kg [21]. However, the next year, alfalfa that had 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.

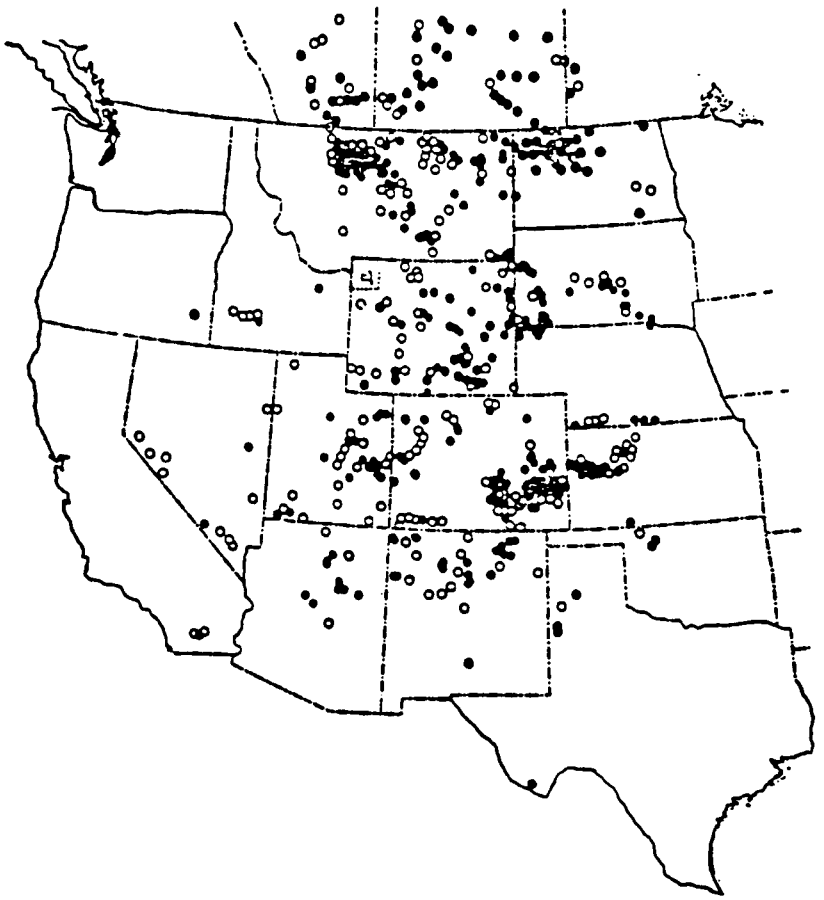

Figure 2 Distribution of seleniferous vegetation in the western United States and Canada. (Adapted from Rosenfeld and Beath [8].) Each open dot represents the place of collection of a plant specimen containing 50–500 mg Se/kg; each solid dot represents specimens containing more than 500 mg Se/kg.

Infrequent incidence of selenosis has been reported on the Kendrick Project in central Wyoming. Tolerance to high Se levels varies considerably among individual animals and birds [22]. In addition, experimental evidence suggests that some animals can accommodate high levels of dietary Se after evidencing some symptoms of chronic toxicosis, such as lameness and hair loss (H. F. Mayland, personal observation).

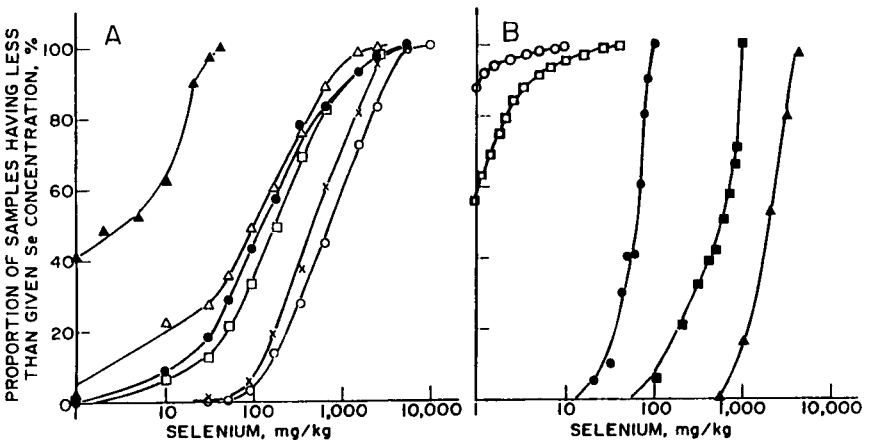

Figure 3 Proportion of samples (percent) having less than given Se concentration. (A) Data for (▲) western wheatgrass (*Pascopyrum smithii* [Rydb.] A. Love) and Sandberg bluegrass (*Poa secunda* Presl.) sampled from seleniferous areas of Montana and Wyoming; (Δ) *Stanleya* spp.; (●) *Xylorhiza* section of *Machaeranthera*; (□) *Astragalus bisulcatus*; (X) *Astragalus pectinatus*; (○) *Oonopsis* section of *Haplopappus*. (B) (●) Vegetative wheat (*Triticum aestivum* L.); (■) *Astragalus bisulcatus*; and (▲) *Astragalus pectinatus* reported in plants from North Dakota. (Data adapted for graphical presentation by Mayland et al. [13]). (○) Big sagebrush (*Artemisia tridentata* Nutt.) and (□) alfalfa (*Medicago sativa* L.) data from Kendrick Project, central Wyoming (adapted from Erdman et al. [36]).

Many different Se compounds have been identified in plants [20]. Much of the Se in nonaccumulating species is found as protein-bound selenomethionine. In contrast, the Se in accumulator plants is mostly water-soluble and found in nonprotein forms like selenium methylselenocysteine. Only trace amounts of the latter compound are found in nonaccumulator species. Selenomethionine, selenocystine, and possibly selenium-methylselenomethionine and selenonium have been detected in nonaccumulators but not in the accumulators tested [23]. The Se metabolites in plants are generally analogues of S compounds. Nevertheless, Se metabolism in nonaccumulator plants cannot be identified from known mechanisms because of scientists' limited understanding of the metabolic pathways for Se in plants [20].

Microorganisms can reduce selenate to elemental Se^0 and Se^{2-}. Many microorganisms, plants, and animals reduce selenite to selenide, giving

rise to volatile organic forms. Dimethylselenide is the volatile and odoriferous Se compound that is characteristic of Se accumulator plants. The compound is also detected in the breath of animals and humans respiring excess Se [5,7]. I have 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.

Two methylated Se compounds, dimethylselenide and dimethyldiselenide, are respiratory products of microorganisms, plants, animals, and humans [7, 10, 13, 16]. Hydrogen selenide (H_2Se) is another volatile Se compound. It is highly toxic but under atmospheric conditions quickly decomposes into innocuous Se^0 and water [5].

BIOAVAILABILITY OF SELENIUM IN FEEDSTUFFS

Selenium utilization varies greatly, depending upon the chemical form in which it is fed. Selenium compounds that are insoluble or have low digestibility pass through the digestive tract and are excreted in the feces. Apparent absorption of Se in feedstuffs, inorganic compounds, and Se amino acids is about 70%; however, absorption is highly variable among and within sources [5,24].

Not all Se absorbed is physiologically important. Some is metabolized to methylated forms that are excreted readily. For example, most of the Se in urine is trimethylselenonium. Plants and animals alike metabolize some Se to the volatile dimethylselenide and dimethyldiselenide, which are respired and lost to the atmosphere.

Selenium is normally metabolized to Se analogues of the S-containing amino acids and then to Se-containing polypeptides and proteins. Of these, the Se-dependent enzyme glutathione peroxidase (SeGSHpx) is the only physiologically critical species known [5]. As an analogue of S, Se may be incorporated into some Se proteins that are physiologically inert.

The biological utilization of dietary Se depends on the form and the integrated response of several physiological and metabolic processes of varying complexity. The net response is quantitatively called the bioavailability of the given Se source, an experimentally derived value that must be considered in the context of the measured response. Disease prevention, tissue Se levels, and SeGSHpx activities are criteria of bioavailability. Thus, Se bioavailability values are often expressed on a percentage basis, with the bioavailability of sodium selenite assigned a value of 100%.

Combs and Combs [5] list bioavailability estimates for nearly 300 inorganic and organic Se compounds based on their ability to prevent hepatic necrosis in rats and exudative diathesis in chicks. Assuming sodium selenite to be 100% bioavailable, Se in animal by-product feedstuffs (including fish meal) have low availability (9–25%), while that in various plant products have a much higher bioavailability, of about 80%.

Blood Se concentrations less than 120 ng/L are positively correlated with blood SeGSHpx in humans and other animals. To maintain this concentration, the bioavailability of Se in plant products must be comparable with or greater than that of sodium selenite. However, the Se in foods or feedstuffs of animal origin has a much lower bioavailability [5].

Measures of SeGSHpx activity provide another approach to estimating bioavailability, which generally substantiates the relative value of Se in various feedstuffs determined by the other methods. Bioavailability of Se can be affected by factors other than Se source. A reduction in feed intake often increases the utilization of ingested Se. Dietary fats, and especially unsaturated plant oils, increase the utilization of dietary Se. Suboptimal levels of the S-containing amino acid methionine have been shown to reduce the utilization of Se from selenomethionine [5]. High levels of S, however, have been shown to decrease the metabolism of Se to tissue SeGSHpx in animals and result in increased frequency of skeletal myopathies in lambs and calves. Selenium has been identified as an antagonist against the toxicity of other heavy metals [5,25]. However, it has been shown to exhibit a synergistic effect on lead poisoning in sheep [26].

Studies with common foods and animal feedstuffs have shown that despite variations between bioassay methods and species, some materials, like yeasts, wheat grain, and alfalfa, generally provide highly available sources of Se. Compared to the bioavailability of Se in selenate or selenite, the Se in plant-derived foods and feedstuffs is moderately available, whereas Se in materials of animal origin is poorly available [5].

The toxicity of high levels of Se can be reduced by feeding high levels of protein. Protection is provided by feeding casein, lactalbumin, linseed oil meal, and torula yeast [5]. The active factor in linseed-oil meal is a cyanogenic glycoside. Some heavy metals may increase the biliary elimination of Se but may also potentiate the acute toxicity of trimethylselenonium [5,26]. Supplemental methionine has been shown to protect against Se toxicity by forming readily excreted methylated metabolites like dimethylselenide and trimethylselenonium ion. Large-scale application of these remedies may not be economically practical. Changing diets is often the only realistic solution.

Studies with rats have shown that males are less sensitive than females to intoxication by selenite but more sensitive to methylated forms [5]. Males can adapt to higher levels of Se than females. Genetic strains of chickens and swine have shown heritable traits in response to Se deficiency or toxicity levels, respectively. Animals may be conditioned or acclimated to moderately toxic levels of Se (H. F. Mayland, personal observation). They may adapt to the high Se exposure by increasing their metabolic production of methylated Se compounds, which are readily excreted.

BIOAVAILABILITY OF SELENIUM 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 [11]. 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, that have not developed rumen function can excrete 65–75% of the orally ingested Se in the urine. As these animals develop functioning rumen systems, microorganisms transform the Se into 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. [13] noted that <0.3% of the Se taken up by plants originated from the Se contained in sheep manure during a 75-day study.

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 [27]. However, large differences were observed in Se uptake by barley, wheat, and alfalfa when $TMSe^+$ was applied in a pot study in the greenhouse. Very little of the Se from $TMSe^+$ was absorbed by the plants, and some absorbed $TMSe^+$ was even lost to the atmosphere through volatilization from the plant or perhaps from microbial respiration [27]. 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 is dependent upon the Se source [5]. Dimethylselenide is also respired by plants [13] 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 [16].

DIAGNOSTICS FOR ADEQUATE SELENIUM NUTRITION IN ANIMALS

Several syndromes in cattle and sheep have been classified as selenium-responsive conditions (Table 2) on the basis of current information [28]. 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 [29]. Blood levels of over 100 μg Se/L in cattle [30] and 180–230 μg Se/L in swine [31] are needed to maintain optimum immunocompetence. Measures of whole-blood Se and SeGSHpx in the hemoglobin are useful in interpreting the Se nutritional status in

Table 2 Selenium-Responsive Diseases of Cattle and Sheep

Syndrome	Major clinical features
Nutritional myodegeneration (white muscle disease)	Acute onset, stiffness, skeletal and/or cardiac muscles affected. Signs vary from acute death to chronic lameness. Pale, necrotic areas of muscle; Zenker's necrosis present histopathologically.
Retained placenta	Retained placenta.
Abortions, stillbirths	Late third-trimester abortions and stillbirths.
Neonatal weakness	Neonates born weak, with or without gross lesions of nutritional myodegeneration.
Diarrhea	Diarrhea, usually profuse, and weight loss in young and adult cattle.
"Ill thrift" syndrome	Decreased feed efficiency, decreased weight gains, and unthrifty appearance.
Immune system effects	Cell-mediated immune response suppression.
Myodegeneration in adult cattle	Weakness, myodegeneration, myocardial fibrosis, myoglobulinuria.
Infertility	Decreased conception rate, irregular estrous cycles, early embryonic death.

Source: Adapted from Maas and Kohler [28].

Table 3 Selenium Diagnostics for Cattle and Sheep

Category	Whole-blood selenium (mg/kg)	GSHpx μg/(mg heme·min)	Results of Se supplementation
Deficient	0.01–0.04	0–15	Usually beneficial
Marginal	0.05–0.06	15–25	Often beneficial
Normal	0.07–>0.10	25–500	Seldom beneficial

Source: Adapted from Maas and Kohler [28].

cattle and sheep (Table 3). Similar criteria are used in determining Se status of human nutrition [5].

MANAGEMENT OF SELENIUM-RESPONSIVE DISEASES

Producers and veterinarians have several methods for treating Se-deficient animals. The most commonly used therapies in the United States are (1) injectable Se products, (2) salt-mix formulations with supplemental Se, and (3) total-ration formulations with supplemental Se. The U.S. Food and Drug Administration regulations in the United States [32] now permit only 0.1 mg/kg as Se supplementation in the diet. Some practitioners question whether this limit is adequate to meet the nutritional needs of animals [31], especially in areas where feeds may contain high levels of S that reduce Se bioavailability. More information is needed about the bioavailability and cycling of various Se forms in the ecosystem.

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 [13]. To overcome widespread Se deficiencies, these two countries applied selenate fertilizer to crops in the early 1980s. Increasing soil Se levels has effectively increased the general level of Se in feedstuffs for both animals and humans [13,33].

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 (H. F. Mayland, personal observation).

SELENIUM IN HUMAN NUTRITION AND HEALTH

In the mid-1960s, severe nutritional Se deficiency was identified in specific areas of China [34]. The deficiency was associated with an endemic juvenile cardiomyopathy (i.e., Keshan disease). Selenium deficiency was also implicated in the etiology of a chondrodystrophic disease (i.e., Kaschin-Beck disease) of children in severely Se-deficient parts of China. Such severe deficiencies have not been noted elsewhere.

The efficacy of sodium selenite for the prevention of Keshan disease was evident as early as 1974. Selenium was supplemented to the human population in the affected areas by (1) distribution of selenite tablets, (2) enrichment of table salt with sodium selenite, and (3) foliar sprays of sodium selenite on grain crops. Recent surveys in the affected areas indicate a decline in the incidence of Keshan disease. However, selenite has failed to provide a cure for people already stricken with Keshan disease. Positive results have been obtained for the prevention and cure of Kaschin-Beck disease by supplementation with sodium selenite [34].

Other areas of China are plagued with endemic Se toxicity in humans, characterized by loss of hair and nails [34]. These symptoms are similar to the alkali disease described in the Northern Great Plains and Prairie Provinces of North America. In addition, Kerdel-Vegas [7] earlier reported cases of selenosis in Amazonian peoples. These cases occurred after the consumption of too many nuts of the *Lecythidaceae* family. The ingestion of foods containing excess Se produced symptoms of nausea, vomiting, chills, diarrhea, and breath characteristic of dimethylselenide. In several days there was a loss of body hair and some finger nails and pronounced arthralgia of joints [7]. Hair regrew almost immediately, and few deaths were reported.

SELENIUM TOXICITY IN BIRDS AND ANIMALS

Acute selenosis is characterized by intake of large doses of Se. Clinical signs of toxicosis are dependent on Se source, dose rate, route of administration, and animal species. Acute lethalities associated with Se compounds are greater when the Se is administered parenterally than when it is given orally [5]. Common inorganic Se salts such as sodium selenite, sodium selenate, selenomethionine, and selenodiglutathione are among the more toxic species. Important characteristics associated with the short-term toxicity of these Se forms include their oxidation state and aqueous solubility. The reduced and poorly soluble forms are the least toxic Se compounds.

Subacute or chronic selenosis can occur when birds, fish, and animals are gradually exposed for periods of weeks to moderately high concentrations of Se (5–25 mg/kg) in their diets. The major signs include lesions on the skin, hoof necrosis, loss of long hair, and emaciation. Other signs include anorexia, weight loss, and increases in serum transaminases and alkaline phosphatase [5]. Signs of chronic selenosis may be expected among animals with whole-blood Se concentrations above 2 mg/kg.

Chronic Se toxicosis decreases conception in animals and may cause embryocidal damage in birds and feticidal loss in animals. The method of administration and the Se source both have significant effects on the extent of the toxicosis.

It is not yet possible to rank chronic toxicities of Se compounds by direct comparison [5]. However, sodium selenate and sodium selenite appear to be quite toxic, and selenomethionine appears to have moderate toxicity. The insoluble forms of Se, like elemental Se, exhibit the least long-term toxicity.

SUMMARY

Selenium is an essential element for animal nutrition and health. It serves as the metal cofactor for the biologically important enzyme glutathione peroxidase. Selenium deficiency reduces growth, productivity, and 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 uncomplicated situations. The requirement increases to 0.3 mg/kg when high levels of S or other Se antagonists are present. In many parts of the United States and elsewhere, there is not enough Se in the feedstuffs to provide adequate nutrition for animal health requirements. In these areas Se may be injected into animals, provided as a mineral mix, or supplemented in a complete feed mix. However, the U.S. Food and Drug Administration currently limits such supplementation to 0.1 mg Se/kg in the diet. Several Se-deficient countries are successfully increasing the Se concentration of their feedstuffs by fertilizing pastures and cropland.

Animals develop a chronic selenosis when the Se concentration of the diet increases to levels of 3–15 mg Se/kg. This is a problem in some areas of the United States and elsewhere, where plants grow on seleniferous soils and accumulate excess Se. Animals feeding on these plants may

develop moderate to severe health problems. Animal sensitivity to selenosis is dependent upon animal species and preconditioning. Some plants can accumulate Se in excess of 25 mg Se/kg when grown on highly seleniferous soils. Animals consuming these plants often will die of acute stenosis. The actual Se concentration is dose-related.

Current regulations of Se supplementation and management of seleniferous areas are largely driven by the political process. Factually based decisions await more information about the Se cycling in the soil–plant–animal system and the bioavailability of various Se species. Continued progress in understanding Se cycling will require additional methodologies to determine Se speciation.

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