Selenium nutrition and therapy in small ruminants

Robert J. Van Saun, *DVM, MS, PhD, Diplomate ACT and ACVN*
Department of Veterinary and Biomedical Sciences, Pennsylvania State University, University Park, PA 16802

Abstract

The objective of this presentation is to address selenium metabolism, disease relationships, and nutritional assessment in small ruminants. Selenium has long been recognized for its toxicity potential, which has driven the regulations restricting dietary supplementation. Selenium deficiency diseases are prevalent in small ruminants raised in geographic regions where soil selenium is marginal to deficient. Toxicity disease most often results from inappropriate parenteral supplementation. Sheep, goats, and camelids respond similarly to selenium deficiency or toxicity diseases, though diagnostic criteria assessing selenium status differ.

Résumé

Cette communication traitera du métabolisme du sélénium chez les petits ruminants, des problèmes de santé qui y sont associés et de son évaluation nutritionnelle. On connaît depuis longtemps les problèmes de toxicité que peut entraîner le sélénium, d'où les restrictions réglementaires qui le concernent dans la préparation des suppléments alimentaires. Les maladies dues aux carences en sélénium sont fréquentes chez les petits ruminants élevés dans les régions où le sol est de marginal à pauvre en cet élément. Quant à la toxicité en sélénium, elle résulte la plupart du temps d'une supplémentation parentérale inadéquate. Les ovins, les caprins et les camélidés réagissent de façon semblable aux maladies reliées à la carence ou à la toxicité en sélénium, bien que les critères d'évaluation diagnostique qui le concernent diffèrent.

Introduction

Selenium (Se) is one of a number of essential microminerals required by small ruminants, as well as all other animals, to help maintain normal body functions. Nearly all of the United States and Canada, with the exception of the central plains regions, are considered to have soil conditions producing forages with marginal to deficient Se content. This places nearly all grazing animals in a situation where supplemental Se needs to be provided to complement the forage component of the diet to meet total daily Se requirements. However, Se has a notorious history that places it in a unique situation relative to feeding recommendations and regulations. The objective of this presentation is to provide an overview of Se nutrition relative to biologic functions and associated disease conditions. Secondly, this review will address appropriate supplementation practices, evaluating mineral products, and monitoring Se status in an effort to minimize disease concerns in small ruminants.

Biological Roles

Selenium was not determined to be an essential mineral until the late 1950s.[12] Immediately following this discovery, Se supplementation was observed to prevent a disease process termed "stiff lamb disease", or what has become known as white muscle disease or nutritional myodegeneration. It was not until 1973 that Se was determined to be a functional component in the cellular antioxidant, glutathione peroxidase (GSH-Px).[11] A number of essential and non-essential nutrients have biologic roles as antioxidants that collectively protect cell function and structure. There is tremendous interest in dietary antioxidants in human and animal nutrition, as they have been linked to preventing or protecting against heart disease, aging, cancer, and many other diseases.

All cells undergoing normal metabolism generate potentially toxic end products termed reactive oxygen species (ROS; peroxide radicals) that have strong oxidizing capabilities. Oxidizing metabolic byproducts, when left unchecked, can result in damage to cellular components including chromosomes, proteins, and cell membranes, ultimately destroying the cell. Selenium is only one of a number of biologic antioxidant agents the body has at its disposal to inhibit damage from internal or external (e.g., pollution, UV radiation, smoke) oxidizing agents. Another well known biologic antioxidant is vitamin E, which works in concert with Se to collectively protect cell membranes (vitamin E) and cell contents (Se) from oxidative damage. The interrelated antioxidant function of vitamin E and Se accounts for why they are often found together in nutritional supplements. Additional dietary vitamin E or Se can replace the other in situations where one is marginally deficient.

Other biologic functions of Se are not fully understood, though a limited number of selenoproteins have been identified.[13] More recently it has been discovered that one of these selenoproteins plays an important role in thyroid function.[3] Secretions from the thyroid gland are important regulators of cellular activity and overall body metabolic rate. This deiodinase selenoprotein

converts thyroxine (T_4) to the biologically active form triiodothyronine (T_3), which mediates rate of cellular metabolism. In the presence of Se deficiency, T_4 can accumulate while T_3 levels will diminish, thus inducing a state of hypothyroidism. Thioreductase is another selenium-dependent compound that regulates intracellular oxidation-reduction state. Selenoproteins S, W, and P have also been identified, through specific functions are not completely elucidated.

Disease Conditions

A wide spectrum of disease conditions have been attributed to Se deficiency, though not all have been well documented to be true deficiency conditions.[13] Nutritional myodegeneration, previously termed "white muscle disease", is the best recognized clinical Se deficiency disease. Any age animal can be affected, though younger animals most commonly experience clinical disease. Severe Se deficiency results in pathologic degeneration of skeletal muscle fibers (i.e., Zenker's necrosis) with secondary fibrosis and calcification. These lesions change the physical appearance of muscle tissue from its normal red to a pale white color. Affected clinical animals will show signs reflective of specific muscles affected and severity of degenerative changes to muscle fibers. Typically both hind legs are symmetrically affected; however, tongue and heart muscles are commonly involved in newborn or young growing animals.

With skeletal muscle damage, affected young or older animals will show various degrees of lameness, weakness, or difficulty moving. Acute death can occur in those younger animals where the heart muscle is damaged. Newborn animals with tongue lesions will have difficulty nursing and may be diagnosed as a "dummy" animal. Severe Se deficiency has been attributed to causing abortion and stillbirth. All of these problems have been documented in most domesticated species and believed to similarly occur in all small ruminants.

Selenium also influences immune cell function, and marginal deficiencies will result in an increased susceptibility to disease. Most studies in ruminants have shown a critical role for Se in the non-specific immune response.[1,2,4] Phagocytic cells are unable to kill the ingested bacteria when the animal is Se deficient. Other studies in humans and sheep have suggested Se also influences the body's ability to mount an appropriate antibody response to an infectious agent. Subclinical Se deficiency in growing animals, through its effect on immune response, may predispose them to diarrhea and pneumonia conditions. Young animals infected with coccidia may not be able to mount a sufficient immune response to help them recover from the disease. This will result in a prolonged disease condition and a perception of disease treatment failure. Adult females with marginal Se deficiency may be more susceptible to metritis around the time of breeding. Premature, weak, or poor-doing neonates have been attributed to Se deficiency. Although there are many potential causes of "ill-thrift" neonates, selenium's role in thyroid function might explain a possible link to this disease syndrome.

Deficiency problems primarily gain our attention when discussing Se, but we should not forget that Se is more notorious for its toxicity concerns. Consumption of specific Se-accumulator plants (*Astragulus* spp) can result in an acute Se toxicity that occurs over a period of hours to days.[10] More common is a chronic Se toxicity syndrome termed alkali disease. The disease is associated with prolonged consumption of seleniferous plants. These plants, and the high Se soils on which they grow, are scattered throughout the northern Great Plains of North America. This disease was first recognized in the Dakotas and Nebraska during the 1860s, and has even been suggested to have contributed to the defeat of General George Custer at the Battle of Little Bighorn. Alkali disease is characterized by cracks and lesions of the hoof wall, abnormal hoof wall growth, brittle hair, and hair loss. Affected animals are often in poor body condition and show various degrees of lameness.

Of greater concern to small ruminants is acute Se toxicity. With the greater propensity for Se deficiency in many regions where small ruminants are raised, more owners are concerned with supplementing Se. One mode of Se supplementation is to inject a commercial Se product. One must be careful in using injectable Se products as their concentration varies, thus the dosing amount will vary (Table 1). Suggested dosage will range from 1 mL per 40 lb (18.2 kg) (Bo-Se®) to 1 mL per 200 lb (90.9 kg) (Mu-Se®) of body weight. With injectable Se one wants to be very careful in the amount given, as overdosage can result in acute toxicity. Injectable Se has high biologic availability and is readily absorbed. There is no antidote for a situation of acute Se toxicity. In such cases the animal will show signs of distressed breathing, salivation, and cardiovascular collapse. This all may occur within minutes to an hour following an injection of an excessive amount of Se. Toxic dosages have not been well defined for all species, but more than 0.23 mg/lb (0.5 mg/kg) body weight is considered toxic for sodium selenite injections. This is about 20 times the suggested dosage for these products (Table 1), but easily achieved by using an inappropriate product. Acute Se toxicity needs to be differentiated from an anaphylactic reaction, which can occur with Se injections. Anaphylactic reactions can be successfully treated with an appropriate dosage of epinephrine.

A number of veterinary diagnostic laboratories are finding very high concentrations (>10 µg/g dry weight) of Se in llama and alpaca liver samples, suggesting an excessive level of supplementation. Some laboratories

Table 1. Comparison of selenium content and suggested dosage for injectable selenium (sodium selenite) products.

Selenium product[1]	Selenium concentration	Recommended selenium dosage	Dosage amount
L- Se®[2]	0.25 mg/mL	25 µg/lb body weight	1 mL per 10 lb body weight
Bo-Se®	1 mg/mL	25 µg/lb body weight	1 mL per 40 lb body weight
E-Se®	2.5 mg/mL	25 µg/lb body weight	1 mL per 100 lb body weight
Mu-Se®, Velenium®	5 mg/mL	25 µg/lb body weight	1 mL per 200 lb body weight

[1]L-, Bo-, E- and Mu-Se products from Shering-Plough, Velenium product from Fort Dodge.
[2]Not currently manufactured.

have identified Se toxicosis as a potential contributor to the death of the animal in a number of these cases. Whether the high liver Se concentration is due to injection or oral Se supplementation has not been clearly defined. Our understanding of Se metabolism in llamas and alpacas is very limited, especially related to injectable Se distribution, and requires further research. In some of these "toxicity" cases, no injectable Se supplementation was documented, suggesting excessive oral supplementation.

Feeding Regulations

Due to toxicity concerns, addition of Se to animal diets is regulated as a food additive by the Food and Drug Administration (FDA) even though it is recognized as an essential nutrient. It was not until 1978, some 20 years after the discovery of Se essentiality, that animal feed manufacturers were permitted to include supplemental Se in animal feed products. Being classified as a food additive dictates that Se cannot be used as a medicine, and veterinarians are not able to write prescriptions for higher level of use. Addition of Se was initially allowed for poultry and pigs at an incorporation rate of 0.1 parts per million (ppm) of the total diet. Over the next couple of years, this allowance for Se inclusion was expanded to include cattle, sheep, and goats.

Field experience in the 1980s with dietary Se supplementation was suggesting the allowable 0.1 ppm level was not sufficient to prevent deficiency disease problems. After considering available scientific information, the FDA in 1987 increased the allowable supplemental dietary Se incorporation rate to 0.3 ppm of the total diet. Use of organic Se (selenomethionine) from Se-fortified yeast cultures was approved in 2000 for chickens, and then other species over the next couple of years. Llamas and alpacas are not mentioned in any of these FDA regulations. By the letter of the law, this would mean supplemental Se cannot be legally added to their diets. However, this is not a situation the FDA would pursue as long as the current regulations for similar species are being followed.

Current FDA regulations allow supplemental Se to be added to the total diet at a level of 0.3 ppm. This is in addition to what is naturally in the diet. However, Se cannot be easily added to forage, thus making it difficult to supplement Se for animals consuming primarily forage-based diets. To address this issue, the FDA permits Se to be incorporated into mineral mixes for animals. Again, the Se concentration of a mineral mix is regulated to conform to expected total dietary intake of Se from the product. Mineral products formulated for sheep and goats are allowed to have a maximum of 90 ppm Se, while products for cattle can contain a maximum of 120 ppm Se. These Se concentrations are based on estimated mineral intake and the total amount of Se to be consumed to meet the 0.3 ppm of supplemental Se in the total diet.

For example, beef cattle are estimated to consume on average about 22 lb (10 kg) of forage diet per day. If one is to supplement Se at the legal level (0.3 ppm), then a beef cow could consume 3 mg Se per day (0.3 mg/kg x 10 kg/day = 3 mg/day). If this 3 mg is to be packaged into a mineral product, the Se concentration of the mineral will need to be adjusted to expected mineral intake. For beef cows, this is estimated between 0.75 and 1 oz per day. In completing the calculations to package 3 mg Se into 0.75 oz, this works out to 120 ppm Se in the salt (refer to Table 2A). Similar calculations were undertaken for sheep and goats on the basis of meeting the defined supplemental Se amount of 0.7 mg/day. Based on a lower expected mineral intake for sheep and goats, allowed maximal Se content of free choice mineral is 90 ppm Se. In allowing the use of Se-fortified mineral supplements, the expectation is the mineral will be the only source of supplemental Se in the animal's diet. To make this issue even more confusing, the allowable Se content for mineral mixes is a maximal value. Feed manufacturers can add any level of Se to their mineral products up to the maximal value for the given species of animal. Therefore, you will need to assess the feed tag information for the selected mineral product to determine Se content and expected intake rate. Compare expected intake to the animal's mineral intake to determine if

the Se content is appropriate to meet daily need (use Table 2A for comparisons).

Requirements

Reports from the National Research Council (NRC) consider the dietary requirement for Se to be between 0.045 and 0.136 mg/lb (0.1 and 0.3 mg/kg) of dietary dry matter (DM).[9] This requirement range was defined for all production animal species from pigs and horses to sheep and cattle. A new NRC report on small ruminant nutrient requirements, which includes llamas and alpacas, recommends 0.74 mg Se/day, or 0.091 mg/lb (0.2 mg/kg) of diet DM for llamas and alpacas. This NRC recommendation is a minimal value and based on extrapolations from other species and information from a single published survey of Se supplementation in llamas.[7] In this survey, supplementation around 1 mg Se/day to adult llamas was associated with adequate blood Se concentrations and ability to maintain normal Se concentrations in crias born to these females. Unfortunately there are no feeding trial reports to document this requirement, though one study reported adequate whole blood Se concentrations (179 ng/mL) for llamas (10-14 months of age) fed alfalfa hay containing 0.091 mg/lb (0.2 mg Se/kg).[5] However, this study did not document total dry matter intake or any mineral feeding to these animals. Measures of whole-blood Se and glutathione peroxidase reflect Se status up to three months prior to supplementation. Another report feeding 0.05 mg/lb (0.11 mg Se/kg) in dromedary camels observed whole-blood Se concentrations averaged 114 ng/mL, which would be considered a deficient concentration.[6]

Another fundamental challenge in defining Se requirement for camelids is the documented difference in dry matter intake compared to other species. Llamas and alpacas consume less food per unit of body weight compared to sheep, goats, and cattle. This results in a disconnection between dietary requirements and conforming to current FDA regulations. In using the current daily (0.74 mg/day) and dietary content (0.091 mg/lb or 0.2 mg/kg) recommendations, predicted total intake would be 1.7 lb or 3.7 kg (0.74 mg/day ÷ 0.2 mg/kg = 3.7 kg/day). This intake greatly exceeds any estimated intake for llamas or alpacas in the NRC report (NRC, 2007).[9] Using this estimate of daily requirement (0.74 mg/day) and estimates of daily intake for maintenance from the NRC report, calculated dietary Se content would range from 0.9 to 0.4 ppm, all values being greater than FDA allowances. These differences underscore the need for further research on mineral requirements for llamas and alpacas. Dietary Se content issues must be resolved; however, the recommended daily amounts of Se to supplement, between 0.74 and 1.0 mg/day, are reasonable guidelines to incorporate into a feeding program.

Feeding Recommendations

To this point, supplemental Se in the diet has been discussed. Inherently there is some Se content to feed ingredients. With small ruminants the primary feed ingredient is forage with supplemental concentrate (pellets, mineral, or both). Forage Se content is extremely variable across all of North America and dependent upon soil conditions. Selenium content of the soil is variable (ranging from < 0.1 to > 80 ppm) and soil acidity, rainfall amount, and other factors can greatly influence its availability to plant tissues. Acid soil conditions, heavy rainfall, and presence of inhibiting substances (iron and aluminum) will result in very low plant Se content. Essentially the eastern coast, north to south, over to the Great Lakes region and the entire western coast areas are low (< 0.1 ppm) in forage Se content. Most all Canadian provinces are low in Se. Only the central plains states up into Manitoba and Saskatchewan have moderate to high soil Se and variable to high plant Se content. Although somewhat expensive ($30-45), forage Se content can be determined at some forage testing laboratories. Unless you have forages from these Se-adequate areas, you should ignore the Se contribution from forage and add the maximal amount of supplemental Se via mineral or pellet products.

As previously described, free-choice mineral products can range widely in Se content; from minimal (10 ppm) up to the legal maximum. What is of interest is the total amount consumed. To assess mineral adequacy relative to Se, one needs to determine the Se content of the mineral and daily animal intake. Mineral Se content can be determined from the feed tag with the product, though recent events have shown this may not be very accurate. Selenium content may be expressed as ppm (mg/kg) or as a percent (%). To convert percent to ppm, move the decimal point to the right 4 places. For example, 0.005% Se is the same as 50 ppm. The bigger challenge is determining average daily mineral intake. Mineral intake is controlled by salt content of the mineral product. Most products will have some intake guidelines on their feed tag. However, expected intake is often over-estimated.

Mineral intake will be variable over time, but typically llamas and alpacas can be expected to consume between 0.25 and 0.33 ounces per day, similar to what is expected for sheep and goats. Again, it is best to determine this for the animals. Also, do not have both white salt and a trace mineral salt available for the animals to choose. They only seek out a salt source. Armed with intake and Se content information, information in Table 2A can be used to assess Se adequacy. For sheep and goats or llamas and alpacas, the combination of mineral Se content and intake that achieves at least 0.7 or 1 mg Se intake per day, respectively, is desired. From these

data, it can be seen that only mineral that has at least 90 ppm Se will achieve near 1.0 mg Se intake, with a daily mineral intake less than 0.5 oz per day. Many commercial mineral products contain less than 90 ppm Se.

Another method of supplementing Se is through the pellet or grain supplement. Using the data shown in Table 2B, one can determine a reasonable Se concentration for their pellet or grain product. If 1.0 mg Se/day is the goal (highlighted row in Table 2B), read across the row to see how many pounds of concentrate would need to be fed to achieve this Se intake amount. The variation is due to the different concentration of Se in the pellet or concentrate product. In this table, the Se concentration of the pellet or concentrate is varied from 0.3 to 8 ppm. It is only when you have a Se concentration of 2.0 ppm or greater where you would be feeding 1 lb or less of the pellet or concentrate product to achieve the desired 1.0 mg Se/day delivery rate. These two examples demonstrated how one could provide the entire supplemental Se allotment from either mineral or pellet sources. Be careful not to provide both sources and potentially double the amount of supplemental Se.

Potential problems with Se toxicity have been highlighted; is there a concern with oral supplementation? Non-ruminant animals, such as pigs and horses, absorb Se from the diet very efficiently and hence are susceptible to toxicity problems. The maximal tolerable level for Se in the total diet of non-ruminants is 2 ppm, or about 10x the requirement.[8] Remember, this is the total diet consumed and not a single ingredient. In contrast, ruminant animals, including llamas and alpacas, are less efficient at Se absorption due to rumen alteration of the Se molecule. Though not determined directly for llamas and alpacas, maximum tolerable level for Se in ruminant diets is considered 5 ppm or even higher.[8] This means that ruminant animals are less susceptible to Se toxicity, but with excessive supplementation it can occur. There is some concern that the new organic Se sources will be more predisposing to toxicity issues, as they are concentrated in tissues to a higher concentration than inorganic Se sources.

Monitoring Se Status

Even when feeding a balanced diet that contains sufficient Se to meet current dietary recommendations, not all animals consume the diet to the desired specifications. To this end, some method of evaluating the nutritional status monitoring process to ensure neither deficiency nor toxicity potential exists should be utilized. Dietary Se content is difficult to determine and an expensive procedure. One is best served by monitoring Se content of supplemental feeds (mineral or pellets) and determining the response of the animals.

Table 2. Calculated amounts of either selenium intake from free-choice mineral supplements (A) or selenium-fortified pellet supplement (B) needed to achieve specified levels of selenium intake.

A. Free-choice mineral supplements					
Selenium mineral content (ppm)	**Daily salt intake (oz)**				
	0.25	**0.33**	**0.5**	**1.0**	**1.25**
	Amount of selenium (mg/day) consumed				
30	0.21	0.28	0.4	0.85	1.1
50	0.35	0.47	0.7	1.4	1.8
70	0.50	0.66	1.0	2.0	2.5
90	0.64	0.84	1.3	2.6	3.2
120	0.85	1.12	1.7	3.4	4.25
B. Selenium containing pellet or grain supplements					
Desired selenium intake	**Supplement selenium concentration (ppm)**				
	0.3	**1.0**	**2.0**	**4.0**	**8.0**
mg/day	*lb supplement needed to be consumed per day*				
0.5	3.7	1.1	0.6	0.3	0.15
0.75	5.5	1.7	0.8	0.4	0.20
1.0	7.3	2.2	1.1	0.55	0.27
1.5	11.0	3.3	1.7	0.85	0.43
2.0	14.7	4.4	2.2	1.1	0..55
2.5	18.4	5.5	2.8	1.4	0.7

Fortunately, Se is one of the nutrients that can be adequately assessed by measuring blood concentrations, though there is some debate on the preferred method. Selenium concentration can be determined in serum, whole blood, or in tissue (primarily liver) as a method of Se status assessment. Selenium status can also be assessed by determining glutathione peroxidase (GSH-Px) activity in blood. Laboratories that offer GSH-Px analysis are limited, and it is a more expensive and technically difficult procedure. Liver Se content is a good determinate of Se status, but one must obtain a liver tissue sample either by biopsy on a live animal or from a dead animal. It is well worth the cost to have liver mineral analyses completed on any animal that dies (young or old) as a routine monitor of nutritional status.

Most laboratories will perform either serum or whole-blood Se concentration analyses. Serum Se concentration reflects more acute or recent changes in Se nutrition, whereas whole-blood Se reflects more chronic or historical Se status. This is a result of GSH-Px residing primarily in the red blood cell, and each red blood cell lives for 105 or more days. However, llamas and alpacas are slightly different from other species. They have more GSH-Px outside the red blood cell, and thus have generally higher serum Se concentrations compared to other species (Table 3). The Michigan State Nutrition Laboratory reports expected whole blood Se concentrations for adult llamas to be between 150 and 220 ng/mL with deficient value below 120 ng/mL (T. Herdt, personal communication). Many laboratories do not have extensive databases to determine appropriate reference values for llamas and alpacas. Therefore, interpretation from a given laboratory might vary, given the reference values being used. Selenium concentrations in serum and whole blood will also vary by age of the animal. These are important considerations interpreting laboratory results. These observations may partially explain some of the current issues of interpreting high (toxic) Se concentrations in llamas and alpacas that have no history of toxic intake or injections.

Conclusions

Selenium is an essential trace mineral that, due to its potential for toxicity, is regulated as a food additive by the Food and Drug Administration. Published reports of Se deficiency or toxicity disease in llamas and alpacas are scarce, though clinical and anecdotal observations suggest similarity with other small ruminant species. The recent NRC report on small ruminant nutrient requirements has established a daily Se requirement of 0.74 mg/day for llamas and alpacas, while supplemental Se for sheep and goats is 0.7 mg/day. Expression of Se requirements on a dietary concentration basis is confounded in camelids as a result of their inherently lower dry matter intake. Calculated dietary content would exceed current FDA regulations for supplemental Se. Methods to assess daily Se intake from mineral or concentrate sources were described. Animal Se status can be accurately defined by measuring either serum or whole blood Se concentrations. Liver Se content can also be used when tissue samples are available. Recent cases have highlighted concerns with Se supplementation and risk of toxicosis.

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Table 3. Comparison of diagnostic criteria for evaluating serum selenium concentrations (ng/mLl) in sheep and goats, alpacas and llamas (data based on Michigan State Nutrition Laboratory values).

Age category	Sheep/goats		Alpacas		Llamas	
	Deficient	Adequate	Deficient	Adequate	Deficient	Adequate
Neonate (1-9 days)	<25	45 - 80	<40	60 - 120	<50	75 – 130
Suckling (10-29 days)	<30	50 - 90	<50	70 - 140	<50	75 - 130
Weanling (30-150 days)	<40	60 - 90	<60	80 - 150	<60	75 – 150
Yearling (151-500 days)	<50	70 - 110	<80	110 - 160	<110	130 – 230
Adult (>501days)	<50	100 - 145	<80	120 - 160	<110	130 - 230

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