PennState Extension

Llamas and Alpacas

Selenium Nutrition in Camelids

Selenium (Se) is one of a number of essential microminerals required by llamas and alpacas as well as all other animals and people to help maintain normal body functions.

Selenium is termed a micromineral, in contrast to a macromineral like calcium, because it is required in very small quantities; milligrams (mg) per day. However, Se has a notorious history that places it in a unique situation relative to feeding recommendations and regulations. The objective of this column is to provide an overview of Se nutrition relative to biologic functions and associated disease conditions. A companion column will address appropriate supplementation practices, evaluating mineral products, and monitoring Se status in an effort to keep llamas and alpacas healthy.

Biological Roles

Selenium was not determined to be an essential mineral until the late 1950's. 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 when Se was determined to be a functional component in the cellular antioxidant, glutathione peroxidase (GSH- Px). A number of essential and nonessential 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 (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. More recently it has been discovered that one of these selenoproteins plays an important role in thyroid function. 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.

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. White muscle disease (nutritional myodegeneration) is the best recognized clinical Se deficiency disease. Any age animal can be affected, though younger animals most commonly experience clinical disease. As the disease name implies, severe Se deficiency results in pathologic degeneration of skeletal muscle fibers with secondary fibrosis (Figure 1). 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 (Figure 2).

Figure 1. Microscopic photograph of skeletal muscle fibers undergoing degenerative changes associated with clinical selenium deficiency. Most fibers are affected and showing swelling and loss of striations typical of normal fibers (long arrow). Some muscle fibers are being replaced with fibrous tissue (short arrow).

Figure 2. Heart from a calf with white muscle disease. Notice the pale areas (arrows) of the heart wall muscle (Photograph courtesy of Dr. John King, Cornell University [http://w3.vet.cornell.edu/nst/images/catalog/1705.jpg])

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 occur equally in llamas and alpacas.

Selenium also influences immune cell function and marginal deficiencies will result in an increased susceptibility to disease. Most studies have shown a critical role for Se in the nonspecific immune response. Cells that engulf bacteria (phagocytes) are unable to kill the ingested bacteria when the animal is Se deficient. Other studies have suggested that 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 affect 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 uterine infections (metritis) around the time of breeding. Premature, weak, or poor doing babies have been attributed to Se deficiency. Although there are many potential causes of “ill-thrift” babies, 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 can result in an acute Se toxicity that occurs over a period of hours to days. 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 Dakota’s and Nebraska during the 1860’s 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 (emaciated) and show various degrees of lameness.

Of greater concern to llamas and alpacas is acute Se toxicity. With the greater propensity for Se deficiency in many regions where llamas and alpacas are raised, more owners are concerned with supplementing Se. One mode of Se supplementation is to inject a commercial Se product. One must 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 lbs (Bo- Se®) to 1 ml per 200 lbs (Mu-Se®) of body weight. With injectable Se one wants to be very careful in the amount given as over dosage 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 0.5 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.

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 lbs body weight
Bo-Se®	1 mg/ml	25 µg/lb body weight	1 ml per 40 lbs body weight
E-Se®	2.5 mg/ml	25 µg/lb body weight	1 ml per 100 lbs body weight
Mu-Se®, Velenium®	5 mg/ml	25 µg/lb body weight	1 ml per 200 lbs body weight

[1]L-, Bo-, E- and Mu-Se products from Shering-Plough, Velenium product from Fort Dodge. All are prescription products only available from veterinarians.
[2]Not currently being manufactured.

A number of veterinary diagnostic laboratories are finding very high concentrations of Se in llama and alpaca liver samples, suggesting an excessive level of supplementation. Some laboratories 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 as previously discussed, addition of Se to animal diets is regulated as a food additive by the Food and Drug Administration. 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, this means 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 with dietary Se supplementation was suggesting that the allowable level of 0.1 ppm was not sufficient to prevent deficiency disease problems. After considering the available scientific information, in 1987 the FDA 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. 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 10 kg (22 lbs) of a 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 1A). 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 your mineral product to determine Se content and expected intake rate. Compare expected intake to your animal's mineral intake to determine if the Se content is appropriate to meet daily need (use Table 1A for comparisons).

Requirements

Reports from the National Research Council (NRC), a scientific body that reviews available research to determine nutrient requirements for animals, consider the dietary requirement for Se to be between 0.1 and 0.3 mg/kg of dietary dry matter (DM). This requirement range was defined for all production animal species from pigs and horses to sheep and cattle. A new NRC report [http://books.nap.edu/catalog.php?record_id%20=11654] on small ruminant nutrient requirements, which includes llamas and alpacas, recommends 0.74 mg Se/day, or 0.2 mg/kg of diet DM for llamas and alpacas. Unfortunately there are no feeding trial reports to document this requirement. 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 (Herdt, 1995). 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.

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 (0.2 mg/kg) recommendations, predicted total intake would be 3.7 kg (0.74 mg/day ÷ 0.2 mg/kg = 3.7 kg/day). This intake greatly exceeds any estimated intake for llamas in the NRC report. 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 one's feeding program.

Feeding Recommendations

To this point I have discussed supplemental Se in the diet. Inherently there is some Se content to feed ingredients. With llamas and alpacas, 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 difficult, 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 you are interested in 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. 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. Again, it is best to determine this for your 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, you can then use Table 1A to assess Se adequacy. In reviewing this table, the italicized cells show the combination of mineral Se content and intake that achieves at least 1 mg Se intake per day. 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 of the commercial mineral products contain less than 90 ppm Se.

Table 1. 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 mg/day	**Supplement Selenium Concentration (ppm)**				
	0.3	**1.0**	**2.0**	**4.0**	**8.0**
	lbs 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

Another method of supplementing Se is through the pellet or grain supplement. Using the data shown in Table 1B, one can determine a reasonable Se concentration for their pellet or grain product. If we set 1.0 mg Se/day as the goal (italicized row in Table 1B), 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.

We have highlighted potential problems with Se toxicity, is there a concern with oral supplementation? Nonruminant 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 nonruminants is 2 ppm or about 10x the requirement. 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. This means that ruminant animals, including llamas and alpacas, are less susceptible to Se toxicity, but with excessive supplementation it can happen. There are still many questions to be answered relative to Se supplementation in llamas and alpacas.

Monitoring Se Status

One can do their best to provide a balanced diet that contains sufficient Se to meet current dietary recommendations. However, we cannot ensure that the animals consume the diet to be specifications we desire. To this end we should incorporate some method of evaluating nutritional status monitoring process to ensure neither deficiency or toxicity potential exists. Selenium content of the diet is difficult to determine and an expensive procedure. One is best served by monitoring Se content of supplemental feeds (mineral or pellets) and determine response of the animals.

Fortunately, Se is one of the nutrients that can be adequately assessed through the use of blood concentrations, though there is some debate on the preferred method. Selenium concentration can be determined in serum (without cells), whole blood (with cells), 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 2). 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 if one is to properly interpret laboratory results. These observations may partially explain some of the current issues of interpreting high (toxic) Se concentrations in llamas and alpacas that have not been known to be exposed to toxic intake or injections.

Table 2. Diagnostic criteria for evaluating serum selenium concentrations (ng/ml) in sheep, goats, and camelids (data based on Michigan State Nutrition Laboratory values).

Age Category	Sheep/Goats		Camelids	
	Deficient	Adequate	Deficient	Adequate
Neonate (1-9 days)	<25	45-80	<50	75-130
Suckling (10-29 days)	<30	50-90	<50	75-130
Weanling (30-150 days)	<40	60-90	<60	75-150
Yearling (151-500 days)	<50	70-110	<110	130-230
Adult (>501days)	<50	100-145	<110	130-230

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