



Selenium Nutrition in Camelids – Part 1

Robert J. Van Saun, DVM, MS, PhD
*Department of Veterinary and Biomedical Sciences
Penn State University*

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 (T₄) 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).

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 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 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.

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. The subject of requirements and oral supplementation will be the focus of the next column.

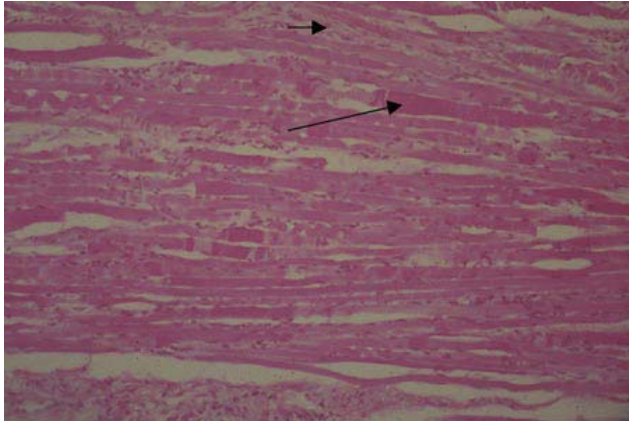


Figure 1. Microscopic photograph of skeletal muscle fibers undergoing degenerative changes associated with clinical selenium deficiency. Most fibers are affected and showing swelling and loss of striations typical of normal fibers (long arrow). Some muscle fibers are being replaced with fibrous tissue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

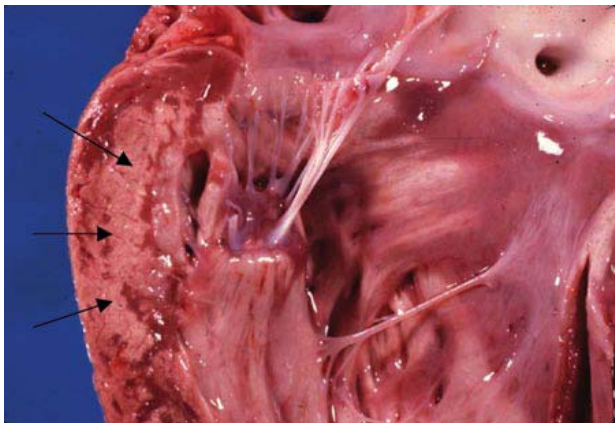


Figure 2. Heart from a calf with white muscle disease. Notice the pale areas (arrows) of the heart wall muscle (Photograph courtesy of Dr. John King, Cornell University <http://w3.vet.cornell.edu/nst/images/catalog/1705.jpg>)

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

Selenium Product¹	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

¹L-, Bo-, E- and Mu-Se products from Shering-Plough, Velenium product from Fort Dodge. All are prescription products only available from veterinarians.

²Not currently being manufactured.

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).

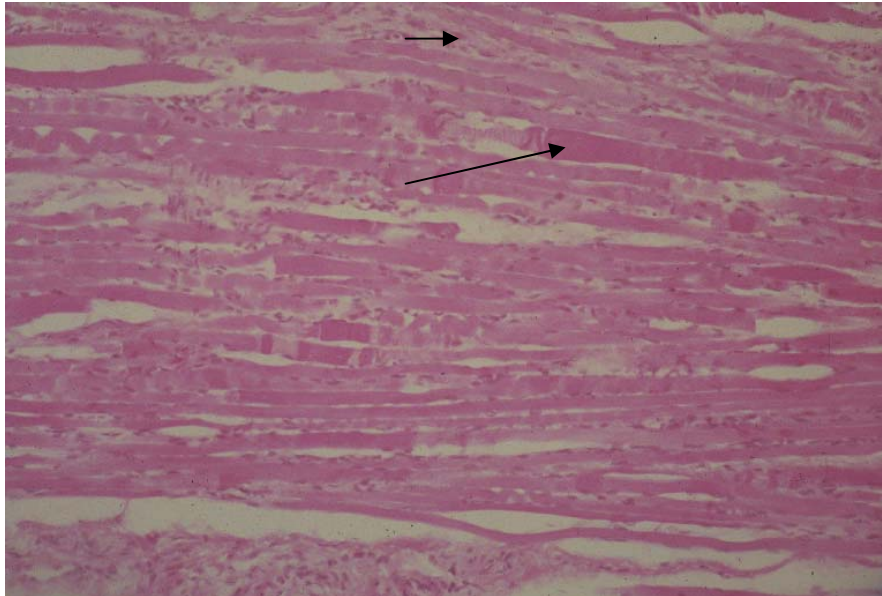


Figure 2. Heart from a calf with white muscle disease. Notice the pale areas (arrows) of the heart wall muscle (Photograph courtesy of Dr. John King, Cornell University <http://w3.vet.cornell.edu/nst/images/catalog/1705.jpg>).

