The importance of selenium in equine nutrition has changed tremendously over the years. Scientists first considered selenium a toxin in the 1930s. In particular, research indicated “alkali disease” in horses was caused by selenium overload. Further research in the 1950s, however, proved selenium prevented white muscle disease in horses and other livestock, thus refuting the perception of selenium as solely a toxin and establishing it as an essential nutrient in horses when offered in limited quantities.

Because of the potential for toxicity, selenium has been labeled an environmental hazard. Government agencies strictly regulate the amount of selenium included in horse feed. For this reason, the selenium requirements established for horses may be as much a political compromise as an accurate assessment of the physiological requirements for the mineral. Whether manufacturers are permitted to add selenium to horse feed in quantities necessary for optimal performance is questionable, especially in areas where selenium deficient forage is fed to horses.

**Why is selenium important?**

Selenium has a number of roles in the body, but perhaps the most important is that of an antioxidant. Oxidation is the metabolic process by which fats, carbohydrates and proteins are converted to carbon dioxide, water and energy (burned to produce the energy needed for body functions). However, the oxidative process does not always recognize which substrates are meant for energy production and which are meant to be structural. Oxidation of the body’s structural and functional components (cell membranes, enzymes, and other intracellular substances) is harmful. In the oxidation process free radicals and superoxides (unstable compounds) are produced. These free radicals are powerful oxidizing agents, which, if they are not destroyed, damage cell structures, notably proteins and lipids (fats). Unsaturated fatty acids, which are the major component of all cell membranes, are particularly susceptible. Their oxidation is quite damaging to cell function. Free radicals attack lipids and form lipid peroxides. An antioxidant will either stop the peroxides from forming or deal with the peroxides once they are formed, which is a role of selenium.

As a part of the cellular antioxidant defense system, selenium is a component of the enzyme glutathione peroxidase, which is widely distributed throughout the body. This enzyme converts reduced glutathione to oxidized glutathione and destroys peroxides by converting them to harmless alcohols. This conversion of peroxides prevents them from reacting with lipid membranes and causing loss of membrane integrity.

Selenium is also found in other select proteins in the body. Interestingly, one of the selenoproteins (iodothyronine deiodinase) plays an important role in thyroid hormone activation. Although not yet investigated in horses, in rats there may be a link between selenium deficiency and thyroid function. Speculation exists as to whether some of the clinical manifestations of hypothyroidism may in fact be secondary to selenium deficiency.

Another selenoprotein is a muscle protein in which deficiency is known to begin muscular degeneration. This is a link between selenium and muscle integrity separate from selenium’s antioxidant properties. Selenium and vitamin E supplementation has been used to prevent muscle disorders (tying-up) in some horses. While selenium deficiency may have been the cause of some cases of tying-up years ago, the common addition of selenium to commercial grain mixes has decreased the percentage of selenium-induced tying-up drastically. Even so, an evaluation of selenium status in a horse prone to tying-up would be advisable.

Selenium is necessary for the development of the acquired immune system. Not all classes of antibodies are affected to the same extent by selenium deficiency and differences in animal age, sex and antigens affect the degree to which antibody production responds to selenium supplementation. The requirement for selenium for optimal immune response in the horse is not known, but it appears to be in line with the recommended daily amount.

Selenium has been suggested to play a role in the prevention of certain forms of cancer. Exciting research is currently being done which will enhance knowledge of selenium’s exact anticarcinogenic effect. The mechanism is not currently known, but researchers are looking at two distinct
scenarios: supplying adequate selenium to those with selenium deficient diets to decrease the incidence of cancer and using elevated intakes of modified selenium for protection against tumorigenesis.

The relationship of vitamin E and selenium

No discussion on selenium would be complete without mentioning vitamin E. The functions of the two are intricately linked; in fact, deficiencies of one can be compensated for if there is adequate supply of the other. Vitamin E present in the cell membrane will decrease the formation of lipid peroxides. Selenium in the intracellular fluid will remove the lipid peroxides that do form. Inadequate amounts of either vitamin E or selenium result in increased oxidation-induced damage and therefore similar effects of deficiency. The administration of either tends to treat or alleviate these effects. In addition, the amount of either needed by the animal depends on the amount of the other available. If there is inadequate vitamin E, more peroxides are formed and, therefore, more selenium is needed. Conversely, if there is inadequate selenium, fewer peroxides can be removed and, therefore, more vitamin E is needed to prevent peroxide formation. Optimum amounts of both, however, are necessary to minimize oxidation-induced tissue damage.

Deficiency and toxicity

Many of the areas of major concentrations of horses in the U.S. have selenium deficient soils. Generally, if the soil is deficient, the forage grown on it will be selenium deficient. Plants growing in acidic soils do not efficiently take up selenium from the soil, so areas that are acidic with low levels of selenium in the soil will produce forage even more deficient. The majority of the horse population in the U.S. does not receive adequate amounts of selenium from forage. The responsibility of supplying adequate selenium in the diet of the horse rests upon feed manufacturers and horse owners. Care should be taken to find out whether the forage is adequate in the area of origin before beginning selenium supplementation.

Gross outward signs of selenium deficiency are not normally seen in free-living animals whose diets are adequate in vitamin E. Vitamin E is abundant in green grass and high quality alfalfa hay, so this may account for the reason why many horses on pasture in selenium deficient areas do not show outward signs of deficiency.

Subclinical signs of selenium deficiency may be easily overlooked. Because the major role of selenium is in the oxidative defense system, deficiency would first compromise cellular integrity. This damage is difficult to quantify but may eventually surface as work intolerance, poor hair coat, and early onset of problems normally associated with aging. Hypothyroidism may also be a subtle sign of selenium deficiency, although the connection has not been proven in horses. Deficiency effects on the muscle can cause cardiomyopathy, myositis (muscle inflammation), and white muscle disease (weak, pale muscles). Gross deficiency signs are growth retardation, cataract formation, retained placentas and eventual reproductive failure.

The requirement for selenium and the toxic level are not widely separated. The minimal requirement according to the National Research Council is 0.1 mg/kg of diet (1 mg per day for the average horse) depending on the age and use of the animal, while toxic levels start at 2 mg/kg of diet for the horse. A few zones in the U.S. have soils very high in selenium, and crops or forages grown in these soils can be toxic. Furthermore, there are selenium accumulator plants, such as milk vetch or woody aster, that superconcentrate selenium from the soil. Selenium accumulator plants can have 5 to 50 mg/kg selenium, so the potential for toxicity is obvious. Horses will generally avoid these plants because they have an unpleasant garlic-sulfur odor, except when forage availability is limited. Selenium appears to be more available for uptake by plants in alkaline soils. Horses grazing forage in zones of excessive soil selenium or selenium accumulator plants develop “alkali disease” which is characterized by hair loss from mane and tail, sloughing of hooves, joint erosion and lameness. Excessive selenium can damage the liver, heart and skeletal muscle, cause degeneration of bones and joints and eventually lead to death.

The presence of certain minerals (sulfur, copper, silver, mercury and arsenic) can decrease the toxicity of selenium because they can either interfere with absorption or increase excretion of selenium. Horses can graze forage in areas with high levels of selenium in the soil when they concomitantly ingest these minerals. This is not an ideal situation, but it is an explanation as to why some horses survive well on high selenium soils.

Availability and absorption of selenium

Selenium available to the horse is either organic (found in forages and grains) or inorganic (salts used to supplement). Not all forms of selenium are equally available to the horse. Absorption of selenium, in general, appears to be fairly high (>50%). The limited research that has been performed indicates that organic selenium is slightly better absorbed than inorganic. Selenium enriched yeast (organic) was found to have higher digestibility and greater retention than selenium salts (inorganic). Levels of selenium in plant foods and grains will vary with soil content. Grains can be important sources of selenium because many are grown in selenium adequate zones of the coun-
try. Garlic can also be high in selenium. Brans in general, whether wheat or rice, are good sources of selenium. The most common source of selenium in horse feeds, sodium selenite, is absorbed efficiently.

How much is enough?

The National Research Council (1989) defines the selenium requirement for the horse as 0.1 mg/kg of diet (mg/kg is the same as ppm). This is sufficient to prevent obvious signs of selenium deficiency in the mature, idle horse. The FDA has also chosen this same amount as the legal limit for horse feeds. For a 1000 pound horse eating 10 kilograms of forage and grain per day, 1 milligram of selenium per day would meet his requirement. Since this would be the minimal requirement, how much would be required for optimal health of the horse? How much does age, health, and performance influence that requirement? Because exercise causes increased oxygen delivery to tissues and oxidation of energy substrate resulting in the generation of peroxides, the selenium requirement for the athletic horse should be increased. The recommendation for selenium in the performance horse may reasonably range from 2.5 to 3.5 mg per day, depending on work intensity, weight and environmental factors. This amount may or may not be supplied by a commercial grain mix if the feeding rate is high enough.

Determining selenium status

To assess selenium status of a horse, concentrations can be measured in serum, plasma, whole blood or liver. Serum, plasma and whole blood are simple measures; liver is a much more invasive procedure and probably not practical for the live horse. In addition to direct selenium measurements, activity of the selenium-dependent enzyme glutathione peroxidase in whole blood also adequately reflects selenium status. The majority of selenium in whole blood is in the red blood cell as glutathione peroxidase. Therefore, there is a strong correlation between whole blood selenium concentration and glutathione peroxidase activity in most animals. Selenium concentrations may differ in serum or plasma as compared to whole blood or glutathione peroxidase activity. Serum and plasma concentrations are acutely sensitive to changes in selenium status as opposed to whole blood and glutathione peroxidase activity which are more chronic indicators of selenium status. Serum and liver selenium concentrations will increase and decrease rapidly with changing selenium intake. In contrast, whole blood glutathione peroxidase activity will increase slowly and remain elevated, up to nine months, after selenium supplementation has ceased. Age will affect selenium concentration. Normal selenium levels for younger horses are lower than those of adult horses. Further, whole blood levels are much higher than those for serum or plasma and have different normal values. For a thorough evaluation of selenium status, both serum and whole blood should be analyzed if the supplementation history is unknown.

Recommendations

Horseowners can ensure a horse has enough selenium in the diet with a few simple steps.

1) A test for blood selenium is the most effective measure to establish if a horse is getting enough selenium from its diet. Whole blood provides a clearer picture of the long term efficacy of selenium supplementation; however, serum or plasma will suffice if that is all that is available.

2) Calculate how much the horse is getting from the forage and grain. If the hay comes from a selenium deficient region of the U.S., the horse is not likely to be deriving much selenium from the forage. The amount of selenium in straight grains (oats, corn, barley) is variable. A few laboratories will analyze forage and feed for selenium content, but the assay is rather expensive.

3) Find out how much selenium is in the horse feed. Most commercial mixes have added selenium, and many manufacturers list the amount on the tag. If selenium is not listed, the manufacturer should provide this information. If the horse is getting small amounts of a commercial mix, it may not be getting enough selenium because feeds are designed to be fed at a certain feeding rate. If a horse is getting 1 pound of a mix that is designed to be fed at 5 pounds per day, the horse in only getting 1/5 of the amount of selenium necessary. In a case like this, selenium supplementation may be prudent.

4) Check to see how much selenium is in any of the supplements being fed. It may be advisable to supplement selenium and vitamin E to any horse with a history of tying-up.

5) The total amount of selenium in the diet should be between 1 and 10 mg per day, depending on the size of the horse and the amount of work the horse is doing. Be careful to stay below 20 mg per day, as toxicity may develop at this point.

Selenium is an integral part of normal body function. Although it is only needed in small amounts, selenium is important in antioxidant defense, immune function, thyroid hormone production, and has possible anticarcinogenic effects. Although vitamin E can compensate for a deficiency of selenium, for optimal health and well-being of a horse adequate amounts of both should be provided.