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Advances in Equine Nutrition

Volume II

Edited by

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THE MANY PHASES OF SELENIUM

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Selenium is one of the most interesting of all nutrients. It was first known for its toxicity and was then discovered to be an essential nutrient. The power of its antioxidant activity has been widely acclaimed. The importance of selenium in immune response and as an anticarcinogen has recently received a great amount of attention. In fact, selenium has been claimed to have a protective role in at least 50 diseases of humans (Reilly, 1996). However, Reilly pointed out the evidence is not yet convincing for many of the claims. Casey (1988) rightfully stressed that the claims of many selenophiles might be exaggerated. Selenium is not a panacea. However, there is evidence that selenium can have enormous health benefits for animals and humans.

Selenium as a Toxin

Selenium was first identified as a toxin in the 1930s. Alkali disease of horses and cattle was shown to be caused by selenium. Signs in the horse included hair loss from mane and tail, sloughing of hooves, joint erosion and lameness. Blind staggers characterized by ataxia, blindness, head pressing and respiratory failure, was also thought to be caused by selenium. The mechanism of selenium toxicity is still not clear but the blocking of the function of SH groups involved in an oxidative metabolism within the cells is a likely prospect (Reilly, 1996). According to NRC (1980), the minimum toxic level for horses may vary from 5 to 40 ppm. Such a wide range perhaps relates to differences in availability among sources and other interfering factors in the environment. For example, methylselenocysteine which is found in toxic plants such as *Astragalus bisulcatus* is much more dangerous than sodium salts of selenium (Reilly, 1996).

The identification of selenium as a toxin and the knowledge of the location of high selenium soils has greatly decreased the incidence of selenium toxicity in horses and cattle. For example, Raisbeck et al. (1993) found that prior to the 1940s, reports attributed thousands of animal deaths to selenium each year in Wyoming. However, no substantiated cases were found in reports from 1947 to 1987. Raisbeck et al. contacted veterinarians in Wyoming directly in 1988 and found 4 cases of selenosis (alkali disease) in horses caused by native range and grass hay containing high levels of selenium during the previous three years. They also suggested that blind staggers in ruminants originally attributed to selenium toxicosis was in fact polioencephalomalacia. Selenosis (alkali disease) was reported in horses in western Iowa by Witte et al. (1993). The source was alfalfa hay (19-58 ppm Se) and it was hypothesized that the alfalfa extracted selenium from deeper subsoils after drought-stimulated extensive tap root development. Selenosis has also been reported occasionally because of over-zealous use of selenium supplements and water with high selenium content.

Selenium Deficiency

The essential nature of selenium was demonstrated in the 1950s. Klaus Schwarz in Germany produced liver necrosis in rats fed a brewer's yeast-based diet. Schwarz moved to the United States and continued his experiments. However, he could only produce liver necrosis when brewer's yeast from the United States was replaced with torula yeast. Selenium was identified as the factor in U.S. brewer's yeast that prevented liver necrosis (Schwarz and Foltz, 1957). Selenium was soon shown to be important in the prevention of white muscle disease in cattle, sheep, swine, and horses, and of exudative diathesis in poultry and therefore has had a major impact on animal production.

Tying-up in horses was associated with selenium deficiency in the past, but selenium does not seem to a major factor in cases of tying-up today, probably because of the widespread use of commercial feeds containing selenium. Foreman et al. (1986) reported generalized steatitis in a foal with low blood selenium values but normal vitamin E levels. Reduced reproductive performance and sudden death in horses have also been attributed to selenium deficiency (Reilly, 1996).

Other diseases such as pancreatic atrophy in poultry are caused by selenium deficiency. More recently selenium deficiency has been shown to have an involvement in Keshan disease, a juvenile cardiomyopathy, and Kaschin-Beck disease, characterized by chondrodystrophy found in China in regions where foods contain low levels of selenium.

Selenium and the Immune Response

Selenium is necessary for the development of the acquired immune system. Reilly (1996) pointed out, however, that not all classes of antibodies are affected to the same extent by selenium deficiency and that differences in animal species, age, sex and antigens affect the degree to which antibody production responds to selenium supplementation. Ponies fed 0.22 ppm selenium had a greater immune response than those fed 0.02 ppm (Knight and Tyznik, 1990). The requirement for selenium for optimal immune response in the horse is not known. However, studies in other species show that selenium supplementation at a supranutritional level may not be needed to improve immune response (Reilly, 1996).

Selenium Requirement

NRC (1989) concluded that the selenium requirement for horses was 0.1 ppm on a dry matter basis. However, I prefer to use the suggestion of 0.1 to 0.3 ppm by NRC (1980). It has been shown in studies by Stowe (1967) and Roneus and Lindholm (1983) that 0.1 ppm Se can be adequate to prevent white muscle disease and maintain glutathione peroxidase levels, but as discussed below many factors can influence selenium utilization. However, I feel that situations such as decreased selenium availability could exist under practical situations where 0.1 ppm Se may not be adequate for the health of the horse.

Selenium Availability

Factors such as form of selenium and interrelationships with other dietary ingredients can influence selenium utilization. For example, copper, sulfur, mercury and arsenic can influence selenium. The effect of the increased supplementation of commercial feeds with copper on selenium has not been studied. Stowe (1980), however, found that an oral dose of 20 or 40 mg of copper/kg of body weight prior to giving ponies 6 or 8 mg of selenium/kg of body weight prevented toxicity by selenium in ponies. The copper did not appear to prevent selenium absorption but enhanced excretion.

Arsenic could influence selenium metabolism in horses by increasing biliary excretion of selenium (Traub-Dargatz et al., 1986). Mercury and selenium form a mercury-selenium complex that decreases the activity of both minerals (Goyer, 1997).

Sulfur has been shown to decrease selenium activity in sheep (Hintz and Hogue, 1964) and presumably in horses, but I am not aware of any studies with horses which titrate the effect. Silver and cadmium are examples of other metals that have been shown to influence selenium metabolism in other species but have not been tested in the horse (Jamall and Roque, 1990).

A low vitamin E content of the diet could influence the need for dietary selenium. Sometimes the interaction could be indirect. For example, a diet containing a high content of raw, cull kidney beans can decrease the effectiveness of vitamin E in the prevention of nutritional muscular dystrophy in lambs. Cooking the beans or increasing the selenium supplementation can alleviate the problem (Hogue et al., 1962).

As mentioned earlier Kaschin-Beck disease is associated with selenium deficiency. It has also been suggested that the mold *Fusarium* might also be involved. A toxin extracted from *Fusarium tricenatum* can cause a decrease in collagen microfibroids in chicken embryo chondrocytes. The addition of selenium prevented the decrease (Reilly, 1996). It has been suggested that selenium might help prevent Kaschin-Beck disease by its effect on *Fusarium* in the food (Reilly, 1996). Tibial chondroplasia in growing chicks can be caused by *Fusarium*. Tibial chondroplasia in chicks is similar to osteochondrosis in foals. Would there be a benefit from increasing selenium intake if foals were consuming moldy feed? Were the raw, cull beans fed to sheep to produce nutritional muscular dystrophy contaminated with mold?

The form of selenium can influence activity. As mentioned earlier, methylselenocysteine is more available than sodium salts of selenium. Schwarz and Foltz (1957) demonstrated that the selenium in swine kidney powder was much more effective than other selenium compounds in the prevention of liver necrosis in rats. Selenium in milk was more effective than Na_2SEO_3 in the prevention of exudative diathesis in chicks (Mathias et al., 1965). Many other factors affecting selenium availability are discussed by Combs and Combs (1986).

Few studies have compared the effectiveness of selenium compounds in horses. Podoll et al. (1992) reported no difference between the utilization of

dietary selenate and selenite by horses. Pagan et al. (1999) measured selenium utilization in two diets containing 0.41 ppm selenium. In one diet, about 3/4 of the selenium was from sodium selenite and in the second diet it was from selenium enriched yeast (Sel-Plex, Alltech Inc.). The apparent digestibility of yeast selenium was greater than for selenite (57% vs 51%). Selenium retention was 25% greater for yeast selenium than for selenite. Exercise increased urinary excretion of selenium more in the selenite group than in the yeast fed group and plasma selenium remained higher in the selenium yeast group than in the selenite group. The authors concluded that more research is needed to quantify the selenium requirements of horses at various intensities of exercise and to determine the effect of form of selenium on antioxidant status.

Feed and Food Sources of Selenium

The selenium content of grains and forage is determined largely by the selenium content of the soil on which the crop is grown. For example, alfalfa has been found to contain values of .03 to .69 ppm selenium and corn may contain .03 to 1.0 ppm. As mentioned earlier, brewer's yeast in Europe contained low levels of selenium whereas brewer's yeast in the United States contained enough to prevent liver necrosis in rats. Wheat products in the United States are likely to contain higher levels of selenium because much of the wheat is raised in areas of the country that contain selenium. Linseed products also are likely to be raised in higher selenium soils and therefore may contain significant amounts of selenium.

Values for various feedstuffs which can supply a significant amount of selenium are shown in Table 1.

Table 1. Selenium contents of feedstuffs.

Feed	Selenium (ppm)
Brewer's dried grains	0.7-1.0
Dried brewer's yeast	1.0-1.2
Linseed meal	1.0-1.5
Wheat bran	0.6-1.0

Selenized salt is often recommended as a source of selenium. It could be a very useful source for animals grazing forages growing on low selenium soils. Such products containing 30 to 90 ppm selenium have been marketed. The voluntary salt intake of horses will vary depending on environmental temperature, work and on anything that can influence sweat production. One study reported a range of voluntary intakes of 19 to 143 g of salt per day with an average of 53 g (Schryver et al., 1987). If the salt contained 90 ppm selenium, a horse would need to eat 11 g of salt per day to obtain about 1 mg of selenium. If the horse

consumed 53 g of selenized salt, it would obtain 4.8 mg. If the horse ate 143 g of selenized salt containing 90 ppm per day, it would obtain 12.9 mg of selenium per day.

The maximum dietary tolerable level of selenium suggested by NRC (1989) is 2 ppm. A 500 kg horse eating 10 kg of feed should be able to tolerate at least 20 mg of selenium daily. Thus, even at the intake of 143 g of salt per day, the tolerable level of selenium would not be exceeded. A horse with a pyschogenic intake of salt could exceed the tolerable level. For non-range conditions, I prefer that the concentrate be the source of selenium rather than free choice salt because the intake of selenium can be more closely controlled.

The selenium content of human food is shown in Table 2. The high selenium contrast of Brazil nuts has not been explained.

Table 2. Selenium in humans foods[a].

Food	Selenium (micrograms)
Brazil nuts (solid unshelled) ½ ounce (4 medium nuts)*	436
Tuna, light 3 ½ ounces	80+
Flounder, 3 ½ ounces	58+
Pork, sirloin, 3 ½ ounces	52
Clams, canned, 3 ½ ounces	49+
Turkey, dark meat, 3 ½ ounces	41
Turkey, white meat, 3 ½ ounces	31
Pasta, cooked, 1 cup	30

[a]Adapted from Environmental Nutrition 22(5):6, 1999.
*Brazil nuts sold in the shell have significantly more selenium than Brazil nuts sold shelled.
+Only 20% is thought to be usable by the body.

As mentioned in the opening paragraph, many health benefits have been claimed for selenium. Reilly (1996) concluded there is strong evidence for the connection of selenium and prevention of heart disease, certain forms of cancer and problems of the immune system but less convincing for the other claims. However, he also pointed out that there is interest in selenium for the prevention of cystic fibrosis, intrahepatic cholestasis of pregnancy and age-related macular degeneration.

One of the most convincing studies showing a relationship between cancer and selenium was conducted by Clark et al. (1996). They reported significant decreases in lung, prostate and colorectal cancer in subjects given 200 µg of selenium per day in a high selenium brewer's yeast tablet supplied by Nutrition 21, LaJolla, CA.

Combs (1998) concluded that the antioxidant protection provided by selenium is directly related to the prevention of overt selenium deficiency and that it corresponds to the expression of SeCys enzymes. He also concluded that other responses, including those involved in anti-carcinogenesis, are related to higher (supranutritional) levels of selenium and those responses may relate to the production of one or more specific intermediary metabolites of selenium. Will higher intakes of selenium by horses have health and performance benefits not previously recognized? Not even "The Shadow" knows, but that possibility exists and should be explored.

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