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MICROMINERAL REQUIREMENTS IN HORSES

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Introduction

Minerals required in minute amounts by horses are specified as microminerals. These nutrients play an important role in a wide range of biochemical systems which affect virtually every metabolic function in the horse. With the exception of selenium, little attention has been given to microminerals in horse nutrition until shortly before the publication of the current NRC Nutrient Requirements of Horses in 1989. Since then, a great deal of research has been directed towards microminerals, particularly as they affect skeletal development in growing horses. Still, many questions remain unanswered about specific requirements for microminerals in many classes of horses. This paper will briefly review the principal functions of copper, chromium, iodine, iron, manganese, selenium and zinc and will highlight recent research that has been conducted with each micromineral in horses. It will also attempt to combine these data with practical experience to provide recommendations for inclusion of these minerals in horse rations.

Expressing Micromineral Requirements

The 1989 NRC expresses micromineral requirements as a concentration (ppm or mg/kg) of the dry matter intake of a total ration. While this is an easy way to remember requirements, it is fundamentally flawed because it presumes a single dry matter intake for a particular class of horse when in reality dry matter intake can range considerably depending on the overall energy density of the ration that is consumed. Since most of the micromineral requirements are not actually related to the energy density of the ration, a better way to describe micromineral requirements would be on a daily intake basis (mg/day) or on a body weight basis (mg/kg BW/day). This paper will attempt to describe requirements both in terms of concentration of diet dry matter (ppm) and on the basis of daily intake.

Copper

Copper (Cu) is essential for proper functioning of enzymes involved in the synthesis and maintenance of elastic tissue, mobilization of iron stores, preservation of the integrity of mitochondria, and detoxification of superoxide. Copper has received a great deal of attention since the last publication of the NRC because of its purported role in the pathogenesis of developmental orthopedic disease (DOD).

The 1989 NRC estimated that all classes of horses require 10 mg Cu/kg of dry diet. Pagan (1998a) and Hudson et al. (2000) both estimated that 500-600 kg

mature, idle horses require about 95-100 mg Cu/day (10.5-11 ppm at 9 kg/d dry matter intake), which is very close to the NRC. Other studies, however, suggest that the requirements of copper for young growing horses and broodmares are considerably higher, especially in certain breeds.

New Zealand researchers (Pearce et al., 1998a) studied the effect of copper supplementation on the incidence of DOD in Thoroughbred foals. Pregnant Thoroughbred mares (n=24) were divided into either copper supplemented or control groups. Live foals born to each group of mares were also divided into copper supplemented or control groups. The four treatment groups were: 1) mares supplemented with copper, but their foals were not supplemented; 2) both mares and foals were supplemented with copper; 3) mares were not supplemented, but their foals received supplementation; 4) neither mares nor foals received supplementation.

Supplemented mares received 0.5 mg Cu/kg body weight (BW) daily (~30 ppm at 9 kg/d dry matter intake) while copper supplemented foals received 0.2 mg Cu/kg BW from 21-49 days of age and 0.5 mg Cu/kg BW (~20 ppm at 5-6 kg/d dry matter intake) from 50 days to 150 days. Mares were supplemented for the final 13 to 25 weeks of gestation. At 150 days of age, the foals were sacrificed and an exhaustive postmortem examination was performed which included investigation of all limb and cervical spine articulations and examination of the physes from the proximal humerus, proximal and distal radius and tibia and distal femur, third metacarpus and third metatarsus. The number of articular and physeal cartilage lesions was noted for each treatment group along with a physitis score that was determined from radiographs of the distal metatarsus.

Copper supplementation of mares was associated with a significant reduction in the physitis scores ($p<0.01$), assessed radiographically, of the foals at 150 days of age. Foals from mares that received no supplementation had a mean physitis score of 6, while foals out of supplemented mares had a mean score of 3.7. A lower score indicates less physitis. When only foals were supplemented with copper, no significant effect on physitis scores was noted. There was a significantly lower ($p<0.05$) incidence of articular cartilage lesions in foals from mares supplemented with copper. However, no significant effects on articular and physeal cartilage lesions occurred in foals supplemented with copper.

Two North American dose-response studies examining the effect of increased dietary copper intakes on bone and cartilage abnormalities (Knight et al., 1990; Hurtig et al., 1993) found that the incidence of DOD decreased by increasing the copper content of the diet above NRC recommendations. In Knight's study, both mares and their foals received copper supplementation, so it is difficult to determine whether the effect resulted from supplementation of the mare or the foal. New Zealand research (Pearce et al., 1998a,b) would suggest that supplementation of the pregnant mare is more important than supplementation of the foal. Oral copper supplementation of mares in late gestation altered the copper balance in these horses and resulted in an increase in the foal's liver copper stores at birth. Increased liver copper stores of the neonate may be important for ensuring healthy development of the skeleton during the period of maximum postnatal growth.

The studies cited above certainly provide proof that copper supplementation of mares and their foals can play an important role in skeletal development. Copper is not, however, the only factor involved in the pathogenesis of DOD, and it is questionable whether the lesions produced by copper deficiency are the same as those most often seen in the field. Pagan and Jackson (1996) documented the incidence of DOD on a commercial Thoroughbred farm over a four-year period. Two hundred and seventy-one foals were monitored. DOD was diagnosed in 10% of the foals even though the horses were fed a ration that provided pregnant mares >250 mg Cu per day (~30 ppm at 9 kg/d dry matter intake) and foals began receiving copper supplementation at 90 days of age.

Copper deficiencies may either be primary in origin because of a lack of copper intake or induced (secondary) due to interactions with other substances in the ration. Zinc (Zn) and molybdenum (Mo) have often been implicated as minerals that can interfere with copper absorption in horses, but several studies have suggested that neither Zn (Coger et al., 1987; Young et al., 1987; Bridges and Moffitt, 1990; Pagan, 1998b) nor Mo (Cymbaluk et al., 1981; Strickland et al., 1987) affects copper utilization when fed at levels found in practical diets. Pagan (1998b) found significant negative correlations between true copper digestibility and the concentration of both crude protein and calcium in thirty different diets. These interactions may be particularly relevant where horses are fed predominantly legume forage.

Chromium

Chromium (Cr) is a component of glucose tolerance factor (GTF). GTF is thought to potentiate the action of insulin in Cr-deficient tissue (Mertz, 1992). Insulin has anabolic characteristics as it promotes glucose uptake by the cell, stimulates amino acid synthesis and inhibits tissue lipase. Chromium excretion is greater in athletic than in sedentary humans and the chromium requirement is increased by physical activity (Anderson et al., 1991). Chromium supplementation has increased lean body mass in humans and pigs and has resulted in a partitioning effect on nutrients which favors tissue anabolism and muscle protein accretion. In calves chromium excretion is greater during stress and chromium supplementation has resulted in a stimulation of the immune system and less mortality and morbidity in shipped feedlot cattle. The 1989 NRC does not include a recommendation for chromium supplementation in horses.

Pagan et al. (1995) reported that supplementing performance horses with 5 mg/day of chromium (0.5 ppm at 10 kg/d dry matter intake) in the form of chromium yeast had a beneficial effect on the response of horses to exercise stress. Horses were subjected to a standardized exercise test on a high speed treadmill and blood and heart rate were monitored. Horses receiving chromium cleared blood glucose following a meal more quickly than control horses and showed lower peak insulin values and lower cortisol levels. Chromium supplemented horses also had higher triglyceride values during exercise indicating perhaps more efficient fat mobilization. There was no difference in the heart rate in response to exercise between the two groups but peak lactic acid concen-

trations in the chromium supplemented group of horses were significantly lower than for the controls.

Iodine

Iodine (I) is an essential nutrient for reproduction and normal physiological function in the horse. Thyroxine (T_4) contains iodine and this hormone, along with triiodothyronine (T_3), has powerful effects on the overall health of the horse. These hormones influence nearly every process in the body, from heat regulation and feed utilization to proper bone growth and maturation. Based on data from other species, the 1989 NRC estimated that the iodine requirement of horses was 0.1-0.6 mg/kg of diet.

Nearly 75% of the iodine in an animal's body is in the thyroid gland. Iodine deficiency may result in goiter as the thyroid becomes enlarged in an attempt to produce adequate levels of thyroxine. In the horse, goiters often occur in the foal at birth. Foal goiter may result from a deficiency in iodine in the mare's ration during pregnancy or it may be caused by a goitrogenic substance. Symptoms of iodine deficiency may be a stillborn foal or a very weak foal that cannot stand and nurse. The foal may also have a rough haircoat, contracted tendons, angular limb deformities or other abnormal bone development. A Russian study (Kruzkova, 1968) indicated that mares which had shown anovulatory cycles responded to iodine supplementation.

While iodine deficiency is the primary cause of goiter in foals, excessive levels of iodine may also cause this condition. The maximal tolerable dietary concentration of iodine has been estimated to be 5 mg/kg (ppm) of dry matter (NRC, 1980), equivalent to 50 mg of iodine/day for a horse consuming 10 kg of dry matter daily. The horses most sensitive to high iodine levels are foals from mares who are supplemented with high levels of iodine. Iodine is concentrated across the placenta and in milk so that the fetus and nursing foal receive much higher concentrations than are present in the mare's ration. Therefore, goiters may be present in newborn foals while sparing the mother. A dietary intake of 83 mg I/day is the lowest level reported to have caused goiter in a horse more mature than a suckling foal (Drew et al., 1975).

Baker and Lindsey (1968) reported that goitrous foals were born on three farms which were feeding mares high levels of iodine. The incidence of goiter was proportional to the level of iodine fed and equaled 3% on one farm feeding 48-55 mg I/day, 10% on a farm feeding 36-69 mg I/day and 50% on another farm feeding 288-432 mg I/day. A neighboring farm which did not have any goitrous foals fed iodine at a rate of 6.3-7 mg I/day. In a separate paper, Sipple (1969) reviewed a case in which 11% of the foals born on a farm had goiters. Analysis of the diet revealed that the mares received between 160-400 mg I/day. Coincidentally, the author discovered that the manager of this farm was the brother of the manager of one of the farms in Baker's study in Florida. Apparently, the Florida horseman had prescribed the same iodine supplement for his brother's horses 1,000 miles away.

Drew et al. (1975) reported that on one stud farm in England four foals were born with greatly enlarged thyroids and leg weaknesses. One mare also had an enlarged thyroid. Feed analysis showed that the mares had received 83 mg I/day from a proprietary feed during pregnancy. The year before the introduction of this proprietary feed, the mares received a vitamin/mineral supplement which supplied about 12 mg I/day and there was no problem with goiter on the farm.

It appears from these reports that around 50 mg of dietary iodine is required in the daily rations of mares to produce any incidence of goiters in their foals. One other study (Driscoll et al., 1978) reported goitrous foals from mares receiving 35 mg I/day. There is some question, however, about what levels of iodine the mares in this study actually received. The authors reported that the mares were given 12 ounces per day of a supplement which was reported to contain 58 ppm iodine. The guaranteed analysis on the product's label stated that it contained 340 ppm iodine and independent analyses of the same product revealed that it contained at least 580 ppm iodine, a level 10-fold higher than reported in the paper. Using the manufacturer's guarantee, the mares would have received a total of 131 mg I/day and according to the independent analyses, a total of 212 mg I/day. These levels are within the ranges reported to produce goitrous foals in other studies.

Toxic dietary iodine concentrations may result from adding excessive supplemental iodine, such as from ethylenediamindihydroiodide (EDDI), to concentrates or from using feedstuffs high in iodine. A common feedstuff that may contain excess iodine is kelp (*Laminariales*), a specific family of seaweeds that may contain as much as 1,850 ppm iodine (NRC, 1989). Unfortunately, people have a tendency to classify all seaweeds as kelp just as the layman might consider every breed of horse a Thoroughbred. There are numerous other specific seaweeds, including *Fucaceae, Palmariaceae, Gigartinaceae, Bangiaceae,* and *Ulvaceae* that contain considerably less iodine than kelp.

Iron

Iron (Fe) is the trace mineral most often associated with exercise, even though its true relevance is questionable. A recent survey conducted at a California racetrack indicated that many trainers used some type of iron supplement (Carlson, 1994). This concern with iron stems from the well-known function of iron as part of the heme molecule. The first symptom associated with iron deficiency is anemia. The anemia associated with iron deficiency is hypochromic, microcytic anemia. There are few instances, however, when practical diets would result in iron deficiency anemia. In the previously mentioned study of Carlson, horses which received supplemental iron had iron levels which were in the normal range for adult horses. Very few of the unsupplemented horses examined had any evidence of anemia and those with resting hematocrits below 34% (defined as anemia) showed no evidence of impaired iron status. This scenario is frequently the case and it is rare that a horse with a lowered hematocrit responds to supplemental iron. Lawrence et al. (1987) failed to show an increase in hemoglobin, hematocrit or serum iron when ponies were supplemented with high

levels of iron. More times than not, low hematocrits are an indicator of infection, low-grade systemic disease or even marginal B vitamin status.

Clinically significant anemia in the athletic horse is rare. Exceptions to this are severely parasitized horses, horses with gastric ulceration that leads to blood loss and perhaps horses which suffer from severe EIPH (exercise-induced pulmonary hemorrhage). Meyer (1987) suggested that the iron requirement of the 500 kg horse is 500, 600 and 1200 mg/day for light, moderate and heavy exercise, respectively.

High levels of iron supplementation may affect the availability of other minerals in the diet. Lawrence et al. (1987) reported that high levels of dietary iron supplementation (500 and 1000 mg/kg) depressed both serum and liver zinc. Pagan (1998b), however, failed to show any correlation between iron content and mineral digestion in diets with a wide range of iron content (127-753 ppm). Most of the iron in this study was not supplemented and was probably in the form of iron oxide.

Manganese

Manganese (Mn) is essential for carbohydrate and lipid metabolism and for synthesis of the chondroitin sulfate necessary for cartilage formation. The 1989 NRC based its recommendation of 40 mg Mn/kg of diet on research from other species. Pagan (1998a) estimated that the maintenance requirement for manganese in idle 500-600 kg horses equaled about 385 mg/d, or 43 ppm of the diet, when the horses consumed 9 kg of dry matter per day. Recently, Hudson et al. (2000) measured manganese retention when mature idle horses were fed four different levels of copper, zinc and manganese supplementation (0, 50, 100 or 200% of NRC) added to the same basal ration. The maintenance requirement for manganese calculated from linear regression of intake against retention equaled 540 mg per day or 60 ppm of diet (Figure 1). When data from these two studies were combined, true manganese digestibility was estimated to be 37%, endogenous losses equaled 151 mg/d (0.27 mg/kg BW), and the maintenance requirement equaled 409 mg/d or 45 ppm of diet.

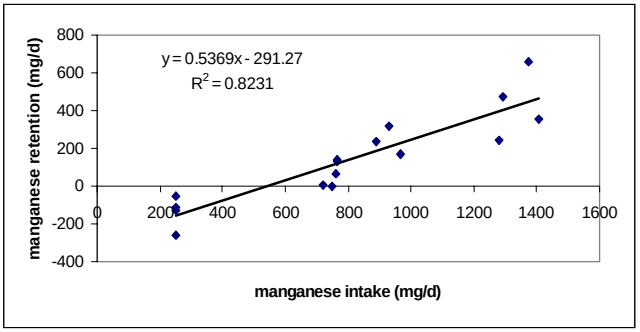

Figure 1. Relationship between manganese intake and retention.

Selenium

Selenium (Se) plays an important role in the maintenance of membrane integrity, growth, reproduction and immune response. A deficiency of Se in foals may produce white muscle disease, a myopathy which results in weakness, impaired locomotion, difficulty in suckling and swallowing, respiratory distress, and impaired cardiac function (Dill and Rebhun, 1985).

Strenuous exercise is known to induce oxidative stress, leading to the generation of free radicals. An increased generation of free radicals may induce lipid peroxidation and tissue damage in both the respiratory system and working muscle. This is particularly true if the animal has a deficient or impaired antioxidant status. Many antioxidants, including glutathione peroxidase (GSH-Px), are selenoproteins, making selenium an extremely important mineral for performance horses.

Although the Food and Drug Administration (FDA) has approved maximal selenium supplementation at 0.3 mg/kg of dry matter in complete feeds for cattle, sheep, and swine (FDA, 1987), selenium supplementation of equine feeds is restricted only by nutritional recommendations and industry practices (NRC, 1989). The selenium requirement for mature idle horses was estimated by the NRC to be 0.1 mg/kg of diet. This requirement is based on studies that evaluated the relationship between selenium intake and blood selenium in mature idle horses (NRC, 1989). Shelle et al. (1985) investigated the effect of supplemental selenium on plasma selenium and on glutathione peroxidase in Arabian and crossbred horses subjected to a conditioning program. They reported that conditioning increased erythrocyte glutathione peroxidase activity and suggested that horses at high work intensities may have higher requirements for selenium than the 0.1 ppm requirement suggested by the NRC. Stowe (1998) has suggested that the appropriate concentration of selenium in the total diet of a horse is 0.3 ppm. This would mean that if a concentrate mix was 50% of the diet and the forage component of the diet supplied 0.1 ppm Se, the grain mix would need to supply roughly 0.5 ppm.

Recent research from the University of Kentucky (Janicki et al., 2000) demonstrated that foals from mares receiving 3 mg Se/d had higher concentrations of IgG at 2 wk ($P<0.05$) and at 4 and 8 wk ($P<0.1$) compared to foals from mares receiving 1 mg Se/d.

Selenium in forages and seed grains is normally present as organic selenium in the form of selenocystine, selenocysteine, and selenomethionine. Sodium selenite and sodium selenate are common inorganic sources of supplemental selenium for horses, and evidence in horses (Podoll et al., 1992) indicates there is no difference between them in potency as measured by blood selenium status. Measurement in laboratory animals, however, shows that organic plant sources of Se are more potent than inorganic (Frape, 1998). Pagan et al. (1999) measured selenium utilization by exercised Thoroughbreds 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. 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. 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.

The maximal tolerable level of selenium in horses is estimated to be 2 mg/kg of diet (NRC, 1980). Acute selenium toxicity (blind staggers) is characterized by apparent blindness, head pressing, perspiration, abdominal pain, colic, diarrhea, increased heart and respiration rates and lethargy (Rosenfeld and Beath, 1964). Chronic selenium toxicity (alkali disease) is characterized by alopecia, especially of the mane and tail, as well as cracking of the hooves around the coronary band (Rosenfeld and Beath, 1964; Traub-Dargatz and Hamar, 1986).

Zinc

Zinc (Zn) is present in the body as a component of many metalloenzymes. The biochemical role of zinc relates largely to the functions of these enzymes. The 1989 NRC suggests that all classes of horses require 40 mg zinc/kg of dry matter. Pagan (1998a) estimated that idle, mature 500-600 kg horses require 257 mg/d, or 28.5 ppm zinc at 9 kg/d dry matter intake. Hudson et al. (2000) estimated that horses of this size require 230 mg Zn/d. Combining data from these two studies (Figure 2) yields a Zn requirement of 248 mg/d, or 27.5 mg Zn/kg diet. This requirement assumes a true zinc digestibility of 21% and endogenous losses of 52 mg/d (0.10 mg/kg BW). Pagan (1998b) evaluated interactions between zinc digestibility and a number of nutrients in 30 different diets. The only nutrient that was significantly correlated to zinc digestibility was magnesium. None of the trace minerals, including iron, affected zinc digestibility.

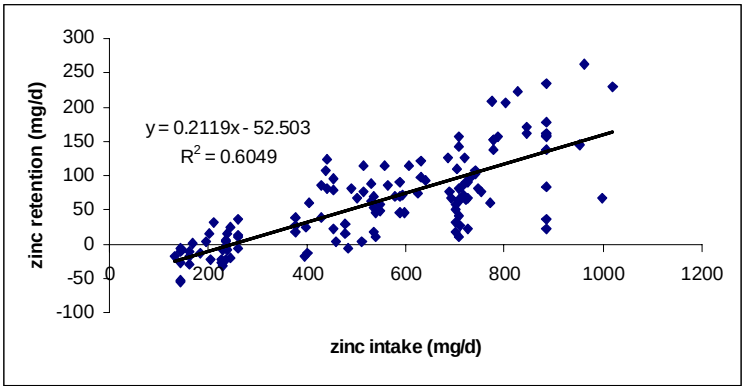

Figure 2. Relationship between zinc intake and retention.

The 1989 NRC noted two studies in which 40 mg Zn/kg diet resulted in acceptable growth rates and no deficiency symptoms in young horses. Ott and Asquith (1987), however, suggested that while yearlings grow satisfactorily with this level of supplementation, optimum bone mineralization may require intakes above NRC recommendations. The current NRC recommendations for zinc appear adequate for mature horses, but they may be low for growing horses.

Recommendations

Table 1 contains a comparison of the current NRC recommendations and those currently used by Kentucky Equine Research (KER). Both sets of recommendations are given as concentrations in the total ration for comparison purposes because this is how the NRC requirements are commonly expressed. The KER recommendations are listed as ranges to account for variability in dry matter intake within each class of horse. KER recommendations are generally more liberal than those of the NRC for several reasons. First, the NRC requirements are considered "minimum amounts needed to sustain normal health, production, and performance of horses." The KER recommendations are generally higher than minimal to account for potential metabolic differences among horses and to protect against possible interactions with other substances in the ration that might result in depressed digestibility or utilization. Second, there is current research which shows that some of the NRC recommendations are too low, particularly for pregnant mares and growing horses. Finally, the NRC provides no recommendation for chromium for any class of horse. Research in other species and with exercised horses suggests that chromium may be an essential mineral for all classes of horses. More research is certainly warranted in this area.

	Maintenance		Pregnant Mares		Lactating Mares		Growing Horses		Working Horses	
	NRC	KER	NRC	KER	NRC	KER	NRC	KER	NRC	KER
Iron	40	40	50	40-50	50	40-50	50	40-50	40	40-50
Manganese	40	40-50	40	40-60	40	40-50	40	60-80	40	40-60
Copper	10	10-15	10	15-25	10	10-15	10	20-30	10	10-15
Zinc	40	40-50	40	50-60	40	40-50	40	60-80	40	40-60
Chromium	None	0.1-0.3	None	0.2-0.4	None	0.2-0.4	None	0.3-0.4	None	0.4-0.5
Selenium	0.1	0.1-0.3	0.1	0.2-0.3	0.1	0.2-0.3	0.1	0.2-0.3	0.1	0.2-0.3
Iodine	0.1-0.6	0.1-0.2	0.1-0.6	0.15-0.3	0.1-0.6	0.15-0.25	0.1-0.6	0.15-0.25	0.1-0.6	0.15-0.25

Table 1. A comparison of 1989 NRC micromineral recommendations and those currently used by Kentucky Equine Research (KER). Recommendations are expressed as a concentration (mg/kg) of dry matter in the total ration.

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