Minerals, Exercise and Bone: Practical Ways to Improve Soundness

Brian D. Nielsen, Ph.D., PAS, Dpl. ACAN
Equine Exercise Physiologist, Department of Animal Science
Michigan State University, East Lansing, MI 48824-1225

While constituting only a minor part of the equine diet, minerals play a critical role in the health of horses. Minerals are involved in a number of functions in the body, including formation of structural components, enzymatic cofactors, and energy transfer. Some minerals are integral parts of vitamins, hormones, and amino acids. The horse obtains most of the necessary minerals from pasture, roughage, and grain. The mineral content of feeds and the availability of minerals vary with soil mineral concentrations, plant species, stage of maturity, and conditions of harvesting. The resulting variations should be considered in assessing an animal's mineral status and formulating appropriate diets. While the amount of individual minerals in the diet is important, the amounts of all minerals should be taken into consideration as minerals often interact with each other influencing their absorption from the diet and excretion from the body. Excesses or deficiencies of one mineral can alter their requirements of another.

Developing a structurally sound horse is usually the first concern most people have when discussing minerals in the diets of their horses. In particular, people want to make sure that young horses that will be used for athletic purposes are provided with sufficient amounts to facilitate proper bone development. Much work has been done on Ca balance in mature, idle horses, as well as growing horses (Hintz et al., 1986). However, there appears to be little work conducted on 18- to 24-month old horses entering training to determine changes in mineral balance. It has been presumed that any increase in Ca requirements due to exercise would be met by increased Ca intake associated with increased dry matter intake (NRC, 1989). However, this has not been documented in young horses entering training. A study with 53 yearling Quarter Horses placed into race training demonstrated a substantial decrease in optical density of the third metacarpal during the first two months of training (Nielsen et al., 1997). This was followed by an increase that continued through the duration of the study. In a subsequent project, 10 untrained Quarter Horse geldings were put into race training and were fed a diet balanced to meet 1989 NRC recommendations to further investigate the influence of early training on bone metabolism (Nielsen et al., 1998a). A similar decrease in the mineral content of the third metacarpal was observed during the first two months of the project. The study also indicated a potential deficiency in dietary Ca in the young horses as they entered training. A follow-up study was conducted to investigate feeding different concentrations of Ca and P to two different groups of two-year-olds placed into training for the first time (Nielsen et al., 1998b). The NRC (1989) recommends 0.32% Ca in the total diet for long yearlings in training, and 0.31% Ca for two-year-old horses in training on an as-fed basis. However, after completing this study (Nielsen et al., 1998b), it appears that the recommendation is too low when formulating diets for young horses entering intense training. It is probable that the ideal concentration of Ca is at least 0.4%. Schryver et al. (1978) found no additional benefit from feeding 0.60% Ca compared to 0.40% Ca to yearling Standardbreds in training. Though no additional benefit was seen by feeding the higher concentration of Ca (Schryver et al., 1978), the exercise being performed by horses in that study may not have been loading the skeletal system sufficiently to initiate a strong remodeling response (Nunamaker et al., 1991). There was definitely a benefit in feeding 0.38% Ca compared to 0.31% Ca as demonstrated by an increase in mineral content of the third metacarpal and an increased Ca retention in horses on the high mineral diet (Nielsen et al., 1998b). However, this experiment used NRC guidelines for Ca concentration in the diet, rather than the total intake of Ca in grams per day. When total Ca intake (g/d) is considered, the control diet provided only 80 to 85% of the requirement and the high mineral diet provided about 100%. When the adequacy of the NRC recommendation is considered from this perspective, it is arguable as to whether the data can be used to support a contention that the current recommendation is too low. Hence, one needs to decide whether it is more appropriate to base mineral requirements on a given quantity per day or whether it is more appropriate to feed as a percent of total intake. Recently, a study by Buchholz-Bryant et al. (2001) found similar increased Ca retention in young (two- and three- year olds), mature (seven to eleven years of age) and aged (15 to 21 years of age) horses entering training when fed 275% of the NRC's recommendation of Ca compared to only 133%. This suggests that the NRC may be too low for all ages of horses when initially entering training after a period of disuse to allow for alterations in bone metabolism.

Interestingly, a study was conducted examining factors associated with shin soreness in human athletes (Myburgh et al., 1988). The study contained 25 athletes that developed shin soreness and 25 control athletes who matched the injured athletes in age, sex and sport. Only three athletes who developed shin soreness consumed the recommended dietary allowance (RDA) of Ca, whereas 15 control athletes met the requirement. Additionally, only two of the control athletes consumed under half the RDA of Ca compared to ten of the injured athletes. As a result, it was reported that low Ca intake was significantly ($P < .005$) related to shin soreness. This seems to indicate that inadequate Ca intake plays a role in skeletal injuries.

These studies emphasize that an increased need for dietary Ca occurs when there is a demand for bone formation. In periods of intense exercise when the skeleton is being loaded and bone is being formed, Ca demand is increased. Since the skeletal system adapts more readily to exercise while in the juvenile state, it is while a horse is still growing that the addition of exercise creates a higher demand for Ca. Interestingly, a ten year study in humans showed that Ca intake over that period was not associated with bone gain or bone strength (Lloyd et al., 2004). Instead, only exercise during adolescence was significantly associated with increased bone mineral density and bone bending strength. This underscores the importance of exercise in young horses to maximize bone strength. However, if sufficient exercise is provided, extra dietary Ca will likely be utilized to improve bone strength.

The recommended concentration of P in the diet as suggested by the NRC appears to be adequate. No differences between treatments or days were seen in P retention in the study by Nielsen et al. (1998b). Hence, there appeared to be no benefit in feeding additional P above what the NRC recommends, other than to maintain a constant Ca:P ratio. Based on the Ca and P concentrations investigated in that study, it appears that the equine is quite capable of regulating P retention when initially entering training. The same conclusions were drawn by Buchholz-Bryant et al. (2001).

It is difficult to draw definite conclusions from the Mg balance results in the study by Nielsen et al. (1998b), as Mg concentrations were greater than what the NRC recommends.

Additionally, once the feed was analyzed, it was discovered that the Mg concentration was not uniform throughout the project. Furthermore, there were no major differences in the amount of Mg fed to the control horses and the high mineral treatment group. However, additional supplementation of Mg may be greater than NRC (1989) recommendations. There was an increase in Mg retention in the latter part of the study in the high mineral group. This was the result of decreased urinary Mg, despite increased Mg intake. Since this is when the biggest increase in mineral content of the third metacarpal occurred in the high treatment group, there exists a potential benefit of feeding additional Mg during periods of high bone formation. This, however, is speculative at best. Additionally, the fact that the horses were in a negative Mg balance when fed Mg concentrations averaging 0.15% of the diet indicates that the currently recommended concentrations of Mg are inadequate.

Besides a potential increase in dietary mineral need for changes in bone metabolism, the additional sweating that accompanies exercise can cause an increased need for some minerals. Schryver et al. (1978) found that the total excretion of Ca and P in the sweat of mature polo horses ranged from 80 to 145 mg of Ca and 11 to 17 mg of P in a 20-min exercise period. Hoyt et al. (1995) estimated sweat loss of Ca to be 8.5 mg per Mcal DE consumed above maintenance for work and sweat loss of P to be 10.7 mg per Mcal DE consumed above maintenance for work. Thus, it does not appear that sweating greatly increases the need for Ca and P. In contrast, an increased need for Na, Cl and K to replace that lost in sweat has been reported (Hoyt et al., 1995; Meyer, 1987). For horses that are exercising hard and producing a lot of sweat, electrolytes should be provided.

Copper is another mineral that plays an important role in proper bone and cartilage development of young horses. A low Cu concentration in the diet appears to have links to osteochondrosis. A negative correlation between Cu concentration and perceived affliction of metabolic bone disease has been reported by Knight et al. (1985). Knight et al. (1990) also reported a reduction in prevalence and severity of osteochondrosis and other developmental cartilage lesions in foals fed elevated amounts of Cu. Gunson et al. (1982) reported several cases of foals with severe generalized osteochondrosis. The foals were suspected of having chronic zinc/cadmium toxicosis as lesions of their joint cartilage were similar to those seen experimentally in animals fed diets high in zinc. This may be partially due to the high levels of zinc interfering with normal copper absorption as similar cartilaginous lesions were also seen in foals fed a copper deficient diet (Bridges and Harris, 1988.)

In addition to osteochondrosis, low Cu concentrations have been linked to epiphysitis in the fetlock joint of weanlings (Asai et al., 1993). In a study conducted by Hurtig et al. (1990), foals fed a diet containing 7 ppm copper had greater incidence and severity of angular limb deformities, mild flexural deformities, epiphysitis, intermittent lameness and osteochondrosis lesions than weanlings fed 30 ppm of copper. Hence, it appears that the NRC (1989) recommendation of 10 ppm is minimal and that by increasing the concentration, a reduction may be seen in the incidence of developmental orthopedic disorders.

While there is no recognized requirement in horses for the element Si, the essentiality of it seems clear (Carlisle, 1974). This ultra-trace mineral is the second most abundant element in the earth's crust and hence, can be found in the diet of every horse. While most of the consumed Si is biologically unavailable and passes through unabsorbed, normally a small portion is utilized in the body or excreted without side effects (Hays and Swenson, 1984). Because of its widespread presence, Si deficiencies have never been reported except in experimental conditions (Van Soest et al., 1983). Experimentally-created deficiencies have revealed that Si-deficient diets result in improper bone, cartilage and collagen formation (Carlisle, 1972; 1976). While such deficiencies are not likely to occur, it has been shown that supplementation can have beneficial results. Nielsen et al. (1993) found that Quarter Horses in race training fed a bioavailable source of Si ran further and completed more strides before experiencing bone-related injuries when compared to non-supplemented horses. A positive correlation (R^2 = .54) between plasma Si concentrations and distance traveled before the horses experienced such an injury was also reported. Lang et al. (2001) also showed that mares fed a bioavailable source of Si demonstrated a trend for increased osteocalcin (marker of bone formation) concentrations over non-supplemented mares after parturition. Though more work needs to be done in this area, it clearly demonstrates that research into mineral nutrition may reap huge rewards when it comes to preventing skeletal injuries to performance horses.

Besides mineral nutrition, proper housing and management of horses can play a critical role in the prevention of lameness. Extensive research has been done into how bone responds to exercise and the lack thereof. While it certainly is possible to injure horses by putting too much force on their bones, it is also possible to make the skeleton less resistant to injury by not putting enough force on it. Often times people do not load the skeleton of young horses sufficiently while they are growing and the result is a weakened skeleton. One of the major problems we see is people stalling young horses with no access to free exercise or with no forced exercise that includes speed. The first evidence of this was reported when Nielsen et al. (1997) saw a dramatic decrease in the mineral content of the canon bone in yearlings brought in from pasture and placed in stalls in order to facilitate training (Figure 1). As the mineral content in the canon bone decreased, injury rates increased. When first reported, it was not certain what caused the decline. However, studies with Arabian and Quarter Horse weanlings, yearlings and two-year-olds at Michigan State University were able to reveal the cause. These studies verified that the decrease in bone mineral content of the third metacarpal was associated with stalling of young horses (Hoekstra et al., 1999; Bell et al., 2001; Nielsen et al., 2002). Even when horses (two-year-olds) were put into training, there was still no increase in bone strength because the animals were being conditioned in the slow fashion (walk, trot and canter) most people typically use (Hoekstra et al., 1999). Likewise, the same appears to be true in older horses that are not given sufficient exercise (Porr et al., 1998). As a result, it currently is recommended that horses, especially young ones, are given turn-out time to encourage normal bone development. Fortunately, for those individuals who desire to stable their horses in stalls, it is not critical to have horses turned out all day long. Research has shown satisfactory results with only 12 hours of turnout per day (Bell et al., 2001) though, conceivably, much less time is needed. Likely, only enough time to allow the horse to do a little bit of running is needed.

Besides benefits to the skeleton, turnout on pasture or in a drylot with other horses has positive effects on behavior (Heleski et al., 2002) and can even make training easier (Rivera et al., 2002). However, if not done properly, some of the major benefits to the skeleton can be missed. Using young calves as a model for young horses, it was determined that animals kept in a group setting that did not encourage the occasional sprint was ineffective at stimulating bone development (Hiney et al., 2004). In contrast, calves that were stalled, but sprinted just once per day for about 50 yards, had dramatic improvements in bone development. Therefore, developing housing-systems that encourage the occasional sprint by horses should decrease the incidence of lameness.

Further evidence of this comes from a study in the Netherlands (Bruin, 1993). In Europe, horses (warmbloods) typically do not enter training until they are at least three years old. In this study, weanlings were forced to perform short sprints over concrete covered by a few centimeters of sand. By the time they entered real training, those horses that were forced exercised while young had lower injury rates and lower incidences of OCD than did the horses that were not trained. Apparently the bones of these young horses had modeled to adapt to the high loads that were placed upon their legs while young. However, one should remember that it was rather short distances these young horses were sprinted. If it had been longer distances, serious injury to the horses could have resulted.

It is quite clear housing that prevents a horse from sprinting is detrimental to bone formation. Fortunately, it has been shown that stalled horses with limited bone development that are returned to pasture eventually return to "normal" if placed back on pasture, at least if this occurs while the animal is still young and growing (Nielsen et al., 2000). The degree to which this can occur in the older horse remains to be determined. Ideally, with proper nutrition and exercise from an early age, optimal bone strength will be achieved and there will be no need to try and regain lost mineral or to repair poorly-formed bone. With quality bone being developed from birth, the ability to withstand the rigors of training without injury will be greatly increased.

Literature Cited

Asai, Y., Y. Mizuno, O. Yamamoto and H. Fujikawa. 1993. Requirements of copper and zinc for foals in connection with the incidence of epiphysitis. Anim. Sci. Tech. (Jpn.) 64(12):1193-1200.

Bell, R.A., B.D. Nielsen, K. Waite, D. Rosenstein and M. Orth. 2001. Daily access to pasture turnout prevents loss of mineral in the third metacarpus of Arabian weanlings. J. Anim. Sci. 79:1142-1150.

Bridges, C.H., and E.D. Harris. 1988. Experimentally induced cartilaginous fractures (osteochondritis dissecans) in foals fed low-copper diets. J. Am. Vet. Med. Assoc. 193:215.

Bruin, G. 1993. The effect of exercise on the incidence of osteochondrosis in young horses. J. Equine Vet. Sci. 13(3):134.

Buchholz-Bryant, M.A., L.A. Baker, J.L. Pipkin, B.J. Mansell, J.C. Haliburton and R.C. Bachman. 2001. The effect of calcium and phosphorus supplementation, inactivity, and subsequent aerobic training on the mineral balance in young, mature, and aged horses. J. Equine Vet. Sci. 21(2):71-77.

Carlisle, E.M. 1972. Silicon: An essential element for the chick. Science. 178:619.

Carlisle, E.M. 1974. Silicon as an essential element. Fed. Proc.33:1758.

Carlisle, E.M. 1976. In vivo requirement for silicon in articular cartilage and connective tissue formation in the chick. J. Nutr. 106:478-484.

Gunson, D.E., D.F. Kowalczyk, C.R. Shoop, and C.F. Ramberg, Jr. 1982. Environmental zinc and cadmium pollution associated with generalized osteochondrosis, osteoporosis, and nephrocalcinosis in horses. J. Am. Vet. Med. Assoc. 180:295.

Hays, V.W., and M.J. Swenson. 1984. Minerals. In J.M. Swenson (Ed.) Dukes' Physiology of Domestic Animals (10th Ed.). pp 449-485. Comstock Publishing Associates, Ithaca, NY.

Heleski, C.R., A.C. Shelle, B.D. Nielsen and A.J. Zanella. 2002. Influence of housing on weanling horse behavior and subsequent welfare. Applied Anim. Behavior Sci. 78:291-302.

Hiney, K.M., B.D. Nielsen, M.W. Orth, D.S. Rosenstein, and B.P. Marks. 2004. Short duration, high intensity exercise alters bone density and shape. Accepted J. Anim. Sci. Dec. 2003.

Hintz, H.F., H.F. Schryver, and J.E. Lowe. 1986. Calcium for pregnant mares and growing horses. Equine Prac. 8(9):5.

Hoekstra, K.E., B.D. Nielsen, M.W. Orth, D.S. Rosenstein, H.C. Schott and J.E. Shelle. 1999. Comparison of bone mineral content and bone metabolism in stall- versus pasture-reared horses. Equine Vet. J. Suppl. 30:601-604.

Hoyt, J.K., G.D. Potter, L.W. Greene and J.G. Anderson. 1995. Mineral balance in resting and exercised miniature horses. J. Equine Vet. Sci. 15(7):310-314.

Hurtig, M.B., S.L. Green, H. Dobson and J. Burton. 1990. Defective bone and cartilage in foals fed a low-copper diet. Proc. Amer. Assoc. Equine Pract. p. 637-643.

Knight, D.A., A.A. Gabel, S.M. Reed, L.R. Bramlage, W.J. Tyznik and R.M. Embertson. 1985. Correlation of dietary mineral to incidence and severity of metabolic bone disease in Ohio and Kentucky. Proc. 31st Amer. Assoc. Equine Pract. p. 445.

Knight, D.A., S.E. Weisbrode, L.M. Schmall, S.M. Reed, A.A. Gabel, L.R. Bramlage and W.J. Tyznik. 1990. The effects of copper supplementation on the prevalence of cartilage lesions in foals. Equine Vet. J. 22(6):426-432.

Lang, K.J., B.D. Nielsen, K.L. Waite, G.M. Hill and M.W. Orth. 2001. Supplemental silicon increases plasma and milk silicon concentrations in horses. J. Anim. Sci. 79:2627-2633.

Lloyd, T., M.A. Petit, H.-M. Lin and T.J. Beck. 2004. Lifestyle factors and the development of bone mass and bone strength in young women. J. Pediatr. 144:776-782.

Meyer, H. 1987. Nutrition of the Equine Athlete. In: J.R. Gillespie and NE Robinson. eds. Equine Ex. Phys. 2. Davis, CA: ICEEP Publications:644-673.

Myburgh, K. H., N. Grobler and T. D. Noakes. 1988. Factors associated with shin soreness in athletes. Phys. Sports Med. 16(4):129.

National Research Council. 1989. Nutrient Requirements of Horses (5th Ed.). National Academy Press, Washington, DC.

Nielsen, B.D., C.I. O'Connor, D.S. Rosenstein, H.C. Schott and H.M. Clayton. 2002. Influence of trotting and supplemental weight on metacarpal bone development. Equine Vet. J. Suppl 34:236-240.

Nielsen, B.D., G.D. Potter, L.W. Greene, E.L. Morris, M. Murray-Gerzik, W.B. Smith and M.T. Martin. 1998a. Characterization of changes related to mineral balance and bone metabolism in the young racing Quarter Horse. J. Equine Vet. Sci. 18(3):190-200.

Nielsen, B.D., G.D. Potter, L.W. Greene, E.L. Morris, M. Murray-Gerzik, W.B. Smith and M.T. Martin. 1998b. Response of young horses in training to varying concentrations of dietary calcium and phosphorus. J. Equine Vet. Sci. 18(6):397-404.

Nielsen, B.D., G.D. Potter, E.L. Morris, T.W. Odom, D.M. Senor, J.A. Reynolds, W.B. Smith, M.T. Martin and E.H. Bird. 1993. Training distance to failure in young racing Quarter Horses fed sodium zeolite A. J. Equine Vet. Sci. 13(10):562.

Nielsen, B.D., G.D. Potter, E.L. Morris, T.W. Odom, D.M. Senor, J.A. Reynolds, W.B. Smith and M.T. Martin. 1997. Changes in the third metacarpal bone and frequency of bone injuries in young Quarter Horses during race training -- observations and theoretical considerations. J. Equine Vet. Sci. 17(10):541-549.

Nielsen, B.D., K.L. Waite, R.A. Bell and D.S. Rosenstein. 2000. Long-term pasture housing promotes bone mineral deposition in the third metacarpus of previously stalled weanlings. The Elite Show Jumper. Conf. Equine Sports Med. Sci. 2000. pg. 157-159.

Nunamaker, D. M., D. M. Butterweck and J. Black. 1991. In vitro comparison of Thoroughbred and Standardbred racehorses with regard to local fatigue failure of the third metacarpal bone. Am. J. Vet. Res. 52:97.

Porr, C.A., D.S. Kronfeld, L.A. Lawrence, R.S. Pleasant and P.A. Harris. 1998. Deconditioning reduces mineral content of the third metacarpal bone in horses. J. Anim. Sci. 76:1875-1879.

Rivera, E., S. Benjamin, B.D. Nielsen, J.E. Shelle and A.J. Zanella. 2002. Behavioral and physiological responses of horses to initial training: the comparison between pasture versus stalled horses. Applied Anim. Behav. Sci. 78:235-252

Schryver, H.F., H.F. Hintz and J.E. Lowe. 1978. Calcium metabolism, body composition, and sweat losses of exercised horses. Amer. J. Vet. Res. 39(2):245.

Van Soest, P.J., M.S. Allen and M.I. McBurney. 1983. Silicon, chromium, the rare earth elements and the remainder of the periodic table. Nat. Feed Ingred. Assoc. Nutr. Inst. Program, April 4-7, Chicago, IL.

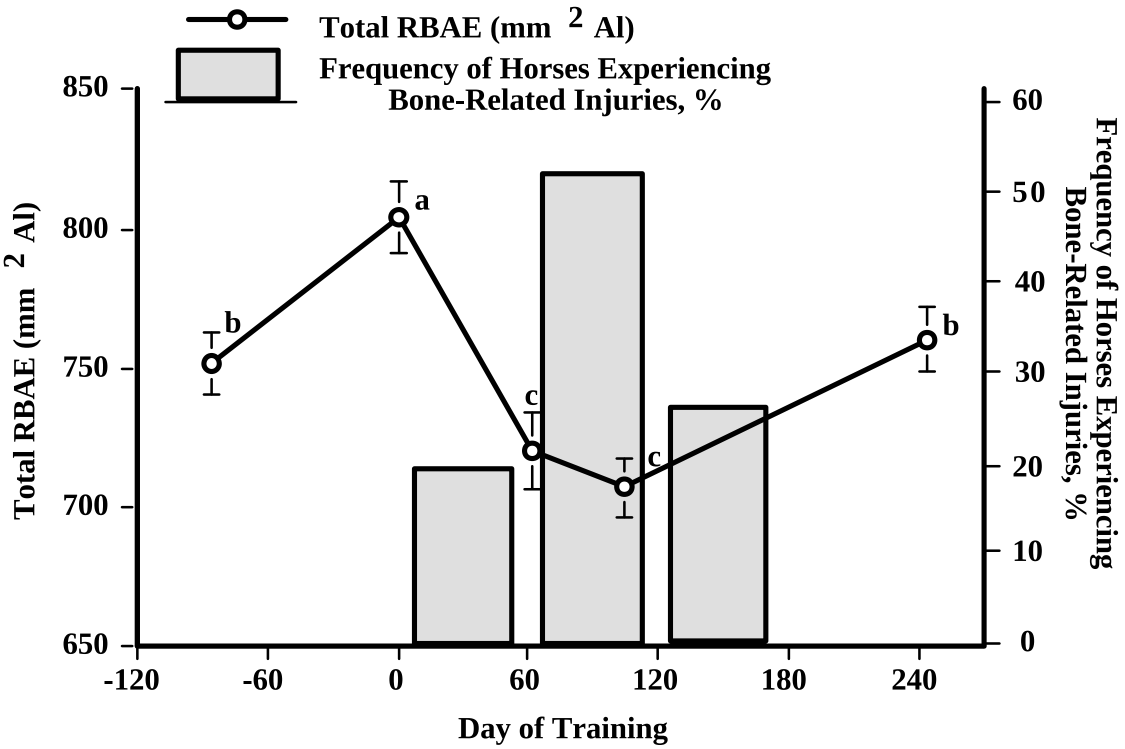

Figure 1. Total RBAEs of all horses and periods during which bone-related injuries occurred.

^{abc} Days lacking a common superscript differ ($P<0.05$)