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ROLE OF COPPER IN SKELETAL DEVELOPMENT OF HORSES

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INTRODUCTION

The trace mineral copper is required in small amounts by all animals. Copper is required for a number of enzymes in the body that are involved in important processes such as energy metabolism, antioxidant system, structural stability of collagen and elastin, pigmentation, and iron metabolism. Copper deficiency signs vary among different animal species but include bone disorders, cardiac failure, depigmentation of hair, and anemia.

Lysyl oxidase is a copper dependent enzyme that plays an important role in the structural stability of collagen and elastin. After the collagen and elastin proteins are synthesized, lysyl oxidase is required for reactions with certain amino acids (lysine and hydroxylysine) that results in cross linking of amino acids in the protein molecule. This cross linking is necessary for the normal stability and strength found in collagen in bone and cartilage, and the elasticity that elastin provides in blood vessels. In copper deficiency the normal cross linking process in collagen and elastin is impaired, and this can result in skeletal abnormalities and cardiovascular disorders. Low copper status has been associated with osteochondrosis and other skeletal problems in horses (Bridges and Harris, 1988; Knight et al., 1990; Hurtig et al., 1993). This newsletter will review research related to the relationship between dietary copper and bone and cartilage disorders in horses.

OSTEOCHONDROSIS

Osteochondrosis is an important developmental orthopedic disease in young horses (Merck Veterinary Manual, 2005). This condition results from a failure of the bone underlying the cartilage inside of the joint to form properly. The adnormalities in joint cartilage result in cracking of cartilage when the foal or young horse takes weight on its joints during exercise. Osteochondrosis can develop in any joint in the skeleton. Rapid growth rate, overfeeding of energy and protein, mineral deficiencies or imbalances, genetics, and trauma to cartilage joints are factors that have been linked with the development of osteochondrosis.

In foals less than six months of age, osteochondrosis symptoms include joint swelling and stiffness that can cause the foal to have difficulty keeping up with other horses in the pasture (Merck Veterinary Manual, 2005). Foals with mild cases of osteochondrosis generally recover naturally with time. In foals with osteochondrosis exercise should be restricted for some weeks. It may also be helpful to reduce feed intake to slow the growth rate. Inflammation associated with severe cases of osteochondrosis can result in chronic and permanent joint damage. Signs of osteochondrosis in yearlings or older horses include joint stiffness and varying degrees of lameness.

Copper and Osteochondrosis

Researchers at Texas A&M University first reported that low serum copper concentrations in foals were associated with arthritis characteristic of osteochondritis (Bridges et al., 1984). Osteochondritis refers to inflammation of bone with its cartilage. Bridges and Harris (1988) induced copper deficiency in young foals by feeding them a milk-replacer low in copper (1.7 mg Cu/kg DM) for 4 to 7 months. Serum copper decreased to less than 0.1 mg/litter in foals given the low copper diet by 13 to 16 weeks. Control foals fed adequate copper (14 mg Cu/kg DM) had serum copper concentrations ranging from 0.80 - 1.60 mg/liter. Lameness developed in copper-deficient foals 2 to 3 weeks after serum copper declined below 0.1 mg/liter. Copper-deficient foals developed a stilted gait and walked on the front edges of their hooves. They also observed cartilage lesions in copper-deficient foals that were similar to those described for osteochondrosis in horses.

Research at Ohio State University also suggested a relationship between dietary copper and cartilage lesions in foals (Knight et al., 1990). In this study pregnant mares were fed diets containing 13 (control; no supplemental copper) or 32 mg Cu/kg DM. Foals born to control mares were offered a pelleted concentrate containing 15 mg Cu/kg while foals from copper-supplemented mares received the same concentrate supplemented with 40 mg Cu/kg. Five foals in each treatment were evaluated for cartilage lesions at 90 and 180 days of age. Cartilage lesions appeared to be lower in foals receiving supplemental copper, especially at 180 days of age.

Hurtig et al. (1993) fed 3-month old foals diets containing 8 (no supplemental copper) or 25 mg Cu/kg DM for 6 months. Foals were weaned from their dams prior to initiation of the study. After 2 months of the study foals receiving supplemental copper had much higher (259 vs. 25 mg Cu/kg DM) liver copper concentrations than control foals. Liver is the major site of copper storage in the body. At the end of the 6 month study, 29 joint lesions were observed in control foals compared with only 3 in copper-supplemented foals. They concluded that inadequate dietary copper is one factor that can contribute to occurrence of ostochondrosis.

Liver copper concentrations are high in foals at birth and decline rapidly in early life. In New Zealand mean liver copper levels in Thoroughbred foals declined from 351 at birth to 21 mg Cu/kg DM at 160 days of age (Gee et al., 2000). Copper supplementation of pregnant mares, grazing pasture containing 4.4 to 8.6 mg Cu/kg DM, increased liver copper concentrations in their young foals (4 to 10 days of age) from 254 to 424 mg/kg DM (Pearce et al., 1998a). In this study copper was supplemented to mares for 13 to 25 weeks prior to foaling. Copper supplementation of mares decreased radiographic indices of physitis (swelling around the growth plate of long bones) in the distal third metatarsal bone and prevalence of joint cartilage lesions in foals at 150 days of age (Pearce et al., 1998b). Providing foals born to control mares with supplemental copper beginning at 21 days of age did not affect bone and cartilage measurements in this study (Pearce et al., 1998b). This suggests that proving adequate copper during fetal development is more important than copper supplementation after birth in preventing signs of osteochondrosis.

Even in horses grazing the same pasture large differences in liver copper levels in mares and foals have been observed. In a study conducted at The Netherlands, mares and their foals grazed pastures containing 9.5 to 12.8 mg Cu/kg DM, and were not supplemented with copper (van Weeren et al., 2003). Liver copper concentrations of foals at 2 to 4 days of age ranged from 27 to 739 mg/kg DM, with a mean value of 351 mg Cu/kg DM. In mares liver copper levels at 2 to 4 days after foaling averaged 19 mg/kg DM with a range of 5 to 97 mg Cu/kg DM. In this study joint lesions in foals decreased in number and severity from 5 to 11 months of age. However, lesions in foals

with low liver copper at birth decreased less in number and severity than those in foals with high liver copper concentrations at birth.

Meeting Copper Requirements of Horses

The most recent Nutrient Requirements of Horses (NRC, 2007) gives recommended copper requirements for various classes of horses. For horses with a mature body weight of 500 kg, the copper requirement was estimated at 10 mg/kg DM with the exception of pregnant mares. Based on research suggesting a relationship between fetal liver copper stores and osteochondrosis in foals after birth, the copper requirement of pregnant mares was estimated at 12.5 mg/kg DM for the last 3 months of gestation. Recommended daily intakes of copper were 100 mg/day for adult horses (500 kg mature weight) with light exercise, 112 to 125 mg/day for adult horses at moderate to heavy exercise, and 125 mg/day for horses lactating or in late gestation. For growing horses the copper requirement was estimated at 0.25 mg Cu/ kg of body weight (NRC, 2007). Little is known regarding factors that may affect copper requirements in horses. Supplementing with high levels of zinc has been shown to decrease copper status of horses (Bridges and Moffitt, 1990). Molybdenum is a trace mineral that greatly affect copper requirements of copper. In contrast to finding in cattle, molybdenum does not appear to have a major affect on copper requirements of horses.

The copper content of feeds varies greatly with most feedstuffs analyzing less than 10 mg Cu/kg DM. Copper can be supplemented to horses in trace mineral salt, a complete mineral supplement containing calcium and phosphorus in addition to salt and trace minerals, or in concentrate supplements. A number of forms of copper including copper sulfate, basic copper chloride, and organic copper sources (copper chelates, copper amino acid complexes, and copper propionate) can be added to horse supplements to ensure that copper requirements are met. Mineral supplements containing copper in the form of copper oxide should be avoided. Studies have indicated that copper from copper oxide is very poorly absorbed by animals.

Summary

Copper is required by all animals for a number of important biological functions. One important function of copper relates to its role in normal bone health. Osteochondrosis is a developmental orthopedic disease in young horses. Several studies have associated low copper status with osteochondrosis in young horses. Providing adequate copper to mares during late pregancy appears to be particularly important in preventing osteochondrosis in their foals. Copper can be supplemented to horses in trace mineral salt, a complete mineral, or in a concentrate supplement to ensure that copper requirements are met.

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