

Osteochondrosis

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Juvenile osteochondral conditions (JOCC) include a wide variety of joint-related lesions that occur in growing foals. Osteochondrosis (OC) or osteochondritis dissecans (OCD) is one type of JOCC that develops when there is a failure in the process of endochondral ossification. The inflammation and damage that occurs at the articular cartilage and underlying bone can result in the development of subchondral bone cysts and osteochondral fragments which often require surgical intervention. The prevalence of OCD varies depending on the age of the horse, the number of joints examined, and the lesion inclusion criteria. Prevalence of JOCC lesions has been reported to be as high as over 67% in both Dutch Warmbloods¹ and in foals from a group of farms in Normandy.² A variety of factors including genetics, breed, and growth rate to biomechanical stress through exercise are thought to influence the development of OCD. Nutrition is another important factor that influences OCD and is one area where therapy can be directed to help prevent OCD and to manage OCD if it does develop.^{3,4}

Dietary Energy

Young growing foals that consume more energy than required often experience a rapid rate of growth. This results in more body weight transmitted to bones and joints that are unable to support the weight. Periods of rapid growth that can occur if foals are fed an inconsistent volume of a high energy feed, like a creep feed, can also contribute to OCD and other JOCC like physitis. Even foals that obtain all of their nutrients from mare's milk may experience a rapid rate of growth that increases the risk of JOCC. Arguably, the most important factor to prevent JOCC is to ensure that young growing foals are fed to meet their nutrient requirements, preventing excess caloric intake that can lead to excessive weight gain at an early age.⁵⁻⁸

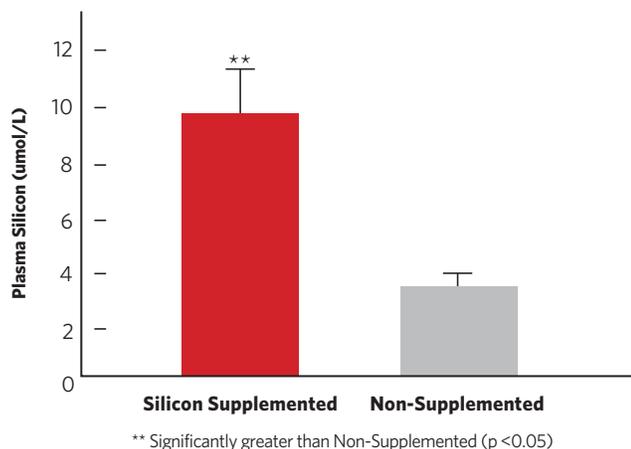
Minerals

Mineral imbalances can also predispose the growing horse to developing OCD. Adequate phosphorus is required for proper bone mineralization, and a deficiency could result in developmental disorders.

Calcium is also necessary for proper bone formation, but excess should be avoided because this can interfere with normal endochondral ossification. The calcium to phosphorus ratio in the ration of a growing horse should be between 2:1 to 3:1. Zinc is a trace mineral that is important for bone development, but excess dietary zinc has been linked to osteochondrosis in foals.⁹ Dietary copper also plays a significant role in the pathogenesis of OCD. Among its many roles, copper is required for collagen formation and stabilization. Osteochondrosis due to copper deficiency has been reported in several species.¹⁰⁻¹² Foals exposed to a copper deficient diet (either in utero or through nursing or later offered as a copper deficient feed) have been shown to be at a higher risk of osteochondral lesions compared to foals that are fed a copper-replete diet.¹³ Foals born to mares that were supplemented with oral copper sulphate at a rate of 0.5 mg Cu/kg body weight during the last three to six months of gestation had OCD lesions at 150 days of age that were significantly less severe

than foals born from unsupplemented mares.¹⁴ The authors speculated that the copper supplementation had a protective effect and enabled foals to more easily repair the OCD lesions that developed. Silicon may also protect against OCD lesions. Silicon is required for cartilage development and bone mineralization.^{15,16} Calves supplemented with silicon for nearly 6 months showed positive correlations between the increased serum silicon and cartilage collagen concentration.¹⁷ Pigs supplemented with silicon had lower overall joint lesion severity scores compared to non-supplemented controls.¹⁷ Silicon supplementation in yearling horses, at 2% of their diet for 45 days, may alter bone resorption as noted by a reduction in the marker carboxy-terminal pyridinoline crosslink telopeptide region of type I collagen.¹⁸ Because silicon supplementation was not shown to resolve pre-existing lesions in 2 year old horses,¹⁹ it may be prudent to begin silicon supplementation earlier as a preventive, during in utero development through to after the foal has been weaned. The transfer of silicon from mare to foal is feasible since silicon supplementation increases the silicon content of mare's milk.²⁰ Silicon is a naturally-occurring element and is rather ubiquitous in nature; however, not all sources of silicon are the same. Choosing a bioavailable silicon supplement is critical.

Figure 1. **Plasma silicon after 8 weeks of supplementation^A in horses**



^A Platinum Performance Oston®

Thoroughbreds supplemented with zeolite,^A a natural form of silicon, for eight weeks demonstrated significant increases in plasma silicon over non-supplemented controls (Figure 1) suggesting this product is a viable choice for silicon supplementation.

Amino Acids and Protein

Certain amino acids may afford some support against severe OCD lesions. Methionine supplementation has been associated with lower overall OC lesion scores (a factor of lesion number and severity) in pigs, a benefit attributed to methionine being either a sulfur donor or precursor to SAME.²¹ In this same study, the combination of the non-essential amino acids proline and glycine, both found in high quantities in collagen, reduced overall lesion scores when compared to non-supplemented pigs. Although controlling growth spurts is important to preventing JOCC, one should not drastically decrease protein intake while reducing dietary energy intake. Severe protein malnutrition during growth may result in improper bone development in the horse.⁶

Managing Inflammation

Although not present in all cases of JOCC, inflammation is a key factor in the diagnosis of osteochondritis dissecans. Osteochondritis dissecans exists when osteochondrosis is present with accompanying synovial inflammation.²² Omega-3 polyunsaturated fatty acids (PUFAs) are well recognized for their management of various inflammatory conditions, although likely most associate this with the long chain PUFAs, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). It is of note, however, that the shorter chain PUFA, alpha-linolenic acid, also has anti-inflammatory activities.²³ A natural source of alpha-linolenic acid is flaxseed. Although not studied regarding OC/OCD, flax supplementation may successfully control inflammation that is associated with certain forms of JOCC in the horse when synovitis is present.

Case Study A farm with a high incidence of JOCC began supplementing pregnant and lactating mares with silicon^A plus a blend of polyunsaturated fatty acids, vitamins and minerals^B. Supplementation of mares continued through weaning, at which point foals were also supplemented. Clinical results from the supplemented foals at one year of age suggest an improvement in the incidence of JOCC within the herd. Three quarter horse colts, each born to a dam with a strong history of JOCC in offspring, currently have clean radiographs.

Putting it Into Practice

- Supplement the pregnant and lactating mare with a well-balanced vitamin, mineral, amino acid and anti-inflammatory product to ensure adequate growth and development of foal in utero and while nursing.
- After foals are weaned provide a high quality, forage based ration and avoid excess energy intake by limiting the volume of grain or commercial foal feed that is offered. For the weanling, keep high-energy feed intake and free access to highly digestible legumes to a minimum to prevent growth spurts.
- Supplement the weanling and young foal with minerals, including silicon, vitamins and amino acids to meet nutrient requirements and to promote proper bone and joint development.

Literature Cited

1. van Grevenhof E, Ducro B, van Weeren P, et al. Prevalence of various radiographic manifestations of osteochondrosis and their correlations between and within joints in Dutch Warmblood horses. *Equine Vet J* 2009;41:11-16.
2. Denoix JM, Jacquet S, Lepeule J, et al. Radiographic findings of juvenile osteochondral conditions detected in 392 foals using a field radiographic protocol. *The Veterinary Journal* 2013;197:44-51.
3. Hurtig M, Pool R. Chapter 20: Pathogenesis of equine osteochondrosis In: McIlwraith C, Trotter G, eds. *Joint Disease in the Horse*. Philadelphia: W.B. Saunders Co, 1996;335-358.
4. Philipsson J. Chapter 21: Pathogenesis of osteochondrosis -- genetic implications In: McIlwraith C, Trotter G, eds. *Joint Disease in the Horse*. Philadelphia: W.B. Saunders Co, 1996;359-362.
5. Cymbaluk NF, Christison GI, Leach DH. Longitudinal growth analysis of horses following limited and ad libitum feeding. *Equine Veterinary Journal* 1990;22:198-204.
6. Lewis L. Chapter 18. Developmental Orthopedic Diseases in Horses In: Cann C, ed. *Equine Clinical Nutrition: Feeding and Care*. Media, PA: Williams & Wilkins, 1995;420-437.
7. Busch ME, Wachmann H. Osteochondrosis of the elbow joint in finishing pigs from three herds: Associations among different types of joint changes and between osteochondrosis and growth rate. *The Veterinary Journal* 2011;188:197-203.
8. Gee E, Firth E, Morel P, et al. Articular / epiphyseal osteochondrosis in Thoroughbred foals at 5 months of age: influences of growth of the foal and prenatal copper supplementation of the dam. *N Z Vet J* 2005;53:448-456.
9. Bridges C, Moffitt P. Influence of variable content of dietary zinc on copper metabolism of weanling foals. *Am J Vet Res* 1990;51:275-280.
10. Handeland K, Bernhoft A. Osteochondrosis associated with copper deficiency in a red deer herd in Norway. *Vet Rec* 2004;155:676-678.
11. Woodbury M, Feist M, Clark E, et al. Osteochondrosis and epiphyseal bone abnormalities associated with copper deficiency in bison calves. *Can Vet J* 1999;40:878-880.
12. Bridges C, Harris E. Experimentally induced cartilaginous fractures (osteochondritis dissecans) in foals fed low-copper diets. *J Am Vet Med Assoc* 1988;193:215-221.
13. Knight D, Weisbrode S, Schmall L, et al. The effects of copper supplementation on the prevalence of cartilage lesions in foals. *Equine Vet J* 1990;22:426-432.
14. Pearce S, Firth E, Grace N, et al. Effect of copper supplementation on the evidence of developmental orthopaedic disease in pasture-fed New Zealand Thoroughbreds. *Equine Vet J* 1998;30:211-218.
15. Carlisle E. Biochemical and morphological changes associated with long bone abnormalities in silicon deficiency. *J Nutr* 1980;110:1046-1056.
16. Carlisle E. The nutritional essentiality of silicon. *Nutr Rev* 1982;40:193-198.
17. Calomme M, Vanden Berghe D. Supplementation of calves with stabilized orthosilicic acid. Effect on the Si, Ca, Mg, and P concentrations in serum and the collagen concentration in skin and cartilage. *Biol Trace Elem Res* 1997;56:153-165.
18. Lang KJ, Nielsen BD, Waite KL, et al. Supplemental silicon increases plasma and milk silicon concentrations in horses. *J Anim Sci* 2001;79:2627-2633.
19. Turner K, Nielsen B, O'Connor C, et al. Silicon supplementation and osteochondritic lesions in 2-year-old Standardbreds: a preliminary study. *Equine Comp Exer Physio* 2007;4:53-58.
20. Lang K, Nielsen B, Waite K, et al. Increased plasma silicon concentrations and altered bone resorption in response to sodium zeolite A supplementation in yearling horses. *J Equine Vet Sci* 2001;21:550-555.
21. Frantz N, Andrews G, Tokach M, et al. Effect of dietary nutrients on osteochondrosis lesions and cartilage properties in pigs. *Am J Vet Res* 2008;69:617-624.
22. Olstad K, Ekman S, Carlson CS. An update on the pathogenesis of osteochondrosis. *Veterinary Pathology* 2015;52:785-802.
23. Anand R, Kaithwas G. Anti-inflammatory potential of alpha-linolenic acid mediated through selective COX inhibition: Computational and experimental data. *Inflammation* 2014;37:1297-1306.

^A Platinum Performance Osteon®

^B Platinum Performance® Equine Wellness Formula

