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Growing more durable equine athletes

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Abstract

Discussions on growing more durable equine athletes often revolve around developmental orthopaedic disease (DOD). What degree of DOD, if any, is acceptable or inconsequential to long-term athletic performance remains a question. Much effort has been directed toward diminishing or eliminating the incidence and severity of DOD, but with limited success. We must continue to define what degree the management decisions affect the horse during different stages of its growth. It would seem logical that the most profound influence would occur during the most rapid periods of growth. Therefore, concerns have been directed toward the last third of gestation and the possible indirect effect that broodmare management has on foetal development. The early postnatal period is another period of rapid growth; thus, the effect of broodmare management on lactation may affect the growth of the foal. Management will also have a direct effect on growth and development from weaning through to maturity. Recent research in other species as well as in horse suggests that genetic predisposition is the primary determinant of DOD. Non-nutritional management also may have significant effects on articular and skeletal health and athletic durability. Much effort has been directed toward nutrition and potential impact on DOD, but with limited success. Except for overt deficiencies, or perhaps excessive imbalances, nutrition does not appear to be a primary determinant of DOD, particularly of focal lesions associated with osteochondrosis. However, nutrition may serve a secondary role in diminishing the severity and incidence of DOD in horses genetically predisposed to such conditions. Producing horses that will have long-term and productive athletic careers requires sound management that starts at conception and continues throughout the life of the horse.

Keywords: horse; growth; developmental orthopaedic disease

Introduction

Developmental orthopaedic disease (DOD) and its potential effects on future athletic performance and longevity in growing horses are often a concern. The term osteochondritis dissecans was used as early as 1887 in association with human knee injuries[1]. Osteochondrosis, osteochondritis and osteochondrosis dissecans have often been used interchangeably and with some confusion. Osteochondrosis can be considered the disease specifically associated with the bone cartilage condition, osteochondritis the inflammatory response and osteochondrosis dissecans the condition when a piece of cartilage is actually avulsed from the surface of a joint[2–4]. Further classification of osteochondrosis is proposed as osteochondrosis lateens (focal cartilage necrosis of the epiphyseal cartilage), osteochondrosis manifesta (microscopic and/or radiographic evidence of focal failure of endochondral ossification) and osteochondrosis dissecans (a loose or avulsed flap of necrotic cartilage)[5]. In 1986, the term DOD was introduced as a heading for all orthopaedic problems in growing horses[3,6]. Today, osteochondrosis, acquired limb deformities, physitis, subchondral cystic lesions, flexural deformities, cuboidal bone malformation and juvenile arthritis can all fall under the general heading DOD.

Osteochondrosis is currently thought to be the result of a failure of endochondral ossification that may affect the articular epiphyseal cartilage or the metaphyseal growth plate. Studies in other species as well as in the horse suggest that initial lesions are derived from growing cartilage and are associated with cartilage thickening and subchondral bone (SCB) defects[2,6–10]; thus, dyschondroplasia may be considered to be a more accurate description of the condition[3]. However, observations during surgery have indicated the separation of cartilage and bone without cartilage thickening or defect in SCB. It has been suggested that until the pathogenesis

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of DOD and the correlations with research models and clinical observations are better understood, the use of the term osteochondrosis would be most appropriate[3].

From a pathogenic standpoint, osteochondrosis has been associated with various factors and interactions among these factors. Incomplete angiogenesis resulting in ischaemic necrosis of SCB has been one factor initially considered[11,12]. Other evidence suggested no association with cartilage necrosis and incomplete vascularization[10]. However, studies in swine show a significant association between reduced blood supply and the pathogenesis of osteochondrosis[13]. Some of these differences in observed pathologies may be due to the site of the lesion as well as the age of the animal[14]. In addition, the influence of physical trauma that may shear vessels and thus contribute to cartilage necrosis and subsequent osteochondrosis lesions can also confound the issue regarding reduced blood supply and the pathogenesis of osteochondrosis[2,8,15–17]. More recent works in swine suggest damage to defined local temporal vessels, present only during a specific phase of growth, as the primary contributor to disruption of blood supply and subsequent focal osteochondrosis[5,18]. This type of pathogenesis has also been shown in horses[19].

Although much knowledge has been gained regarding the nutrient requirements, health and management of the young, growing horse, there are still questions concerning optimum nutrition and management for growth and the avoidance of DOD. What degree, if any, of DOD can be considered normal? If any DOD is abnormal, then what degree can be considered acceptable or recoverable, such that future athletic performance will not be compromised? Is there a perceived increase in the incidence due to a greater ability to detect DOD, or due to changes in management practices that predispose horses to the disease, or both? Our ability to recognize and detect DOD has certainly increased, which leads back to the question of how much of what we detect is normal or inconsequential regarding the development, maturity and soundness of the tissue. Whether changes in management practices have affected the incidence of DOD is difficult to ascertain, because DOD can potentially be influenced by many variants of the confounding factors of genetics, environment, nutrition and other management actions. For the sake of discussion, this article summarizes all of these variants with the term 'management' unless specifically identified otherwise. Beyond genetic predisposition, the complexity of different management interactions makes the establishment of accurate and precise recommendations to avoid DOD complex and difficult. The goal is to grow horses that will be more durable throughout their athletic career, and thus the work will continue until the development of management practices that will help to develop horses with improved articular cartilage and skeletal strength, integrity and maturity.

Genetics

Heritability for osteochondrosis has been established in the horse and can vary depending on specific joints and breeds[5,8,20,21]. As a population, feral horses have significantly less radiographic incidence of DOD than the domestic population[22]. This difference may be related to natural selection pressures as well as to management differences between feral and domestic horses. More recent research in horses is focused on specific quantitative trait locus associated with osteochondrosis[23,24]. In pigs, associations have been made with the heritability of specific anatomical conformation and the incidence and severity of DOD[18]. Selection against specific anatomical features may result in significant reductions in the incidence and severity of specific types of DOD. Simulated active genetic selection in Maremmano horses over a five-generation period indicated a 87.5% reduction in the frequency of osteochondrosis dissecans (reduction in frequency from 16 to 2%)[25]. It could be suggested that genetic selection for soundness in horses has been practiced for centuries. Selecting superior individuals for breeding is often predicated by their past performance records. In order for a given horse to construct a superior athletic performance record, the horse has to be durable enough to sustain training and high-level competition. Thus, the selection of breeding individuals with superior performance records complements to some degree the selection of a durable equine athlete. More emphasis on futurity performance and less on long-term athletic careers may diminish the association with durability and athletic performance.

As genetic research progresses, we will better understand the interactions of genetic and non-genetic factors, as well as the implications of genomics. Such knowledge may already be implemented in breeding selection and could also provide insight into the possible genetic predisposition of a particular horse, which may then allow adjustment to management programmes in order to reduce the risk of DOD.

Maternal influences on growth

A linear increase in foetal body weight occurs after 200 days of gestation, and increases in length are greatest during mid-gestation[26]. Growth in length of the cannon bones continues rapidly until 8–9 months of gestation and then slows; however, the pastern bones reach almost adult length by the time of birth.

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In general, the more distal the bone the sooner it reaches its mature size and shape. At birth, the third phalanx no longer has an active growth plate, and postnatal growth proceeds perichondral[27].

Concerns regarding the maternal effects on foal growth and development should include the prenatal period, particularly during the last two-thirds of gestation. Evidence suggest that neuroendocrine control of growth hormone secretion in the horse may occur before parturition, which is earlier than in other species and may emphasize the importance of broodmare nutrition during gestation and lactation[28]. To date, no research has been done to examine the role of broodmare nutrition on foetal growth hormone secretion. A series of studies have shown that copper supplementation in the mare is reflected in increased copper concentrations in the liver of the foal, and also resulted in reduced radiographic indices for physitis in the distal third metatarsal[29–31]. However, subsequent research showed no difference in the incidence of osteochondritic lesions in foals from mares given injectable supplementation of copper compared with mares that did not receive copper supplementation[32].

Increasing the amount of nutrients available to the broodmare during the last third of gestation and during lactation is well accepted. Such increases help to ensure proper foetal growth and development. It also provides adequate milk production and maintains broodmare body condition. Mares entering the breeding season with body condition scores of <5 may be less reproductively efficient[33,34]. However, to date, research has not established that manipulation of nutritional intricacies beyond current recommendations for the broodmare has a significant effect on skeletal soundness of the foal *in utero,* during growth or throughout adulthood.

Growth rate

The association of rapid rates of growth with osteochondrosis dissecans (OCD) has long been recognized in several species, including the horse[3,9,18,35]. Several comparative studies have been done on height and weight measurements of a horse's growth rate with its estimated mature body size. At 6 months of age, a horse has obtained approximately 83 % of mature height and 46 % of mature weight, at 12 months, 90 and 67 % and at 18 months, 95 and 80 %, respectively. Much of the growth in length from the knee and hock to the ground has occurred between 4 and 6 months of age[36,37]. Growth data show that Thoroughbreds from 1989 to 1990 grew slightly heavier and taller compared with Thoroughbreds from 1958 to 1976[38]. This may be related to genetic selection for larger, faster-growing horses and/or increased nutrient intake, better-defined nutrient requirements, improved nutritional management and improved health management. Data from 1979 showed that colts of the same age were heavier and taller than fillies, and this difference persisted to maturity[39]. However, data from Quarter Horses showed no difference in height between colts and fillies after 48–60 months of age. Interestingly, growth patterns of horses seem to be similar across breeds, geography and diet[40,41]. Studies in Thoroughbreds and Standardbreds indicate an increased incidence of OCD lesions, particularly in the shoulder, stifle and tibiotarsal joints, in horses with greater than average weight gains. Lesions in the fetlock were not associated with weight gain prior to weaning[42,43]. It has been suggested that DOD lesions occur in the first 3 months of life, which is the most intense postnatal growth phase[44]. In addition, the hind limbs of the horse tend to grow at a more accelerated rate than the front limbs, and there is a greater frequency of DOD associated with the hind limbs[45]. Also, timing may play a role in the development of DOD in that there may be a 'window of opportunity' along some point of the growth curve or at some transition period that allows for a greater susceptibility to DOD. In swine, there are contradictory data regarding growth rate and predisposition to DOD. Associations between genetic predisposition and DOD may be associated more with anatomical conformation as it relates to specific joints, rather than with the rate of growth[5].

Although genetic predisposition, as it relates to anatomical form and perhaps growth rate, is the primary determinant of DOD, nutrition may be a contributing factor in horses predisposed to such conditions. The effects of supplemental feeding programmes on young, growing horses may be most profound during the transition period when the young horse begins receiving most of its nutrients from feed sources rather than from mare's milk. This transition period is usually associated with weaning (3–5 months of age), a period of rapid growth that may compound the effects of feeding programmes on growth. Creep fed foals (at approximately 1.5 % of body weight) generally show a greater growth response than non-creep fed foals[46]. Growth in the foal has been expressed in a curvilinear fashion as it relates to age. Studies in other species have shown that daily growth is variable and may not follow a precise growth curve. Growth in children has been suggested to be a series of 0.5–1.0 cm spurts, each approximately lasting <24 h and separated by periods of stasis[47]. Growth curves for horses may represent a similar stair-step type of pattern, with a potential negative rate of gain for 2–3 days post-weaning. Where on the growth curve a horse should be at a given point in time is dependent on breed, the growth potential of the individual horse

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and the desired growth rate. It is generally accepted that moderate or lower rates of growth may have less of an association with the incidence of DOD than rapid rates of growth. Horses that may be genetically predisposed to DOD (i.e. large-framed individuals with an apparent potential for rapid growth) may benefit from diets of lower caloric density. This is sometimes confused with nutrient deprivation (starvation), which is counter productive to reducing the potential for DOD. Protein, vitamins and minerals are needed to ensure sound tissue development. Although specific amino acid supplementation has been shown to facilitate growth rate[48], protein concentrations at 126% of 1989 National Research Council recommendations have not been shown to result in an increased incidence of DOD[49,50]. Furthermore, a review of studies in humans shows a positive association between protein intake and bone mineral density, bone mineral content and reduced bone resorption markers[51]. Deficiencies of these nutrients certainly retard the growth rate, but may also affect the quality of growth. Managing the growth rate of predisposed horses is best achieved by reducing the calories while still providing recommended amounts of protein, vitamins and minerals to maintain the quality of growth.

The other aspect of the growing horse's diet that is often neglected is the forage component, which may vary from 30% or less to 70% or more of the diet. Depending on the quality of the hay or pasture, this aspect may have a greater impact on the amount and quality of nutrients consumed than the concentrate portion of the diet, and thus should be considered in the overall management of predisposed horses[52].

Exercise/mechanical stress

Proposals have been made to reduce the incidence and severity of DOD by eliminating the athletic events and strenuous training regimens to which young horses are subjected. Although such proposals may be effective, it is not a realistic solution given the current industry focus. In addition, several studies actually support the reverse contention and suggest that early training and competition are beneficial to healthy bone and cartilage development[53]. Exercise affects bone density, and those effects can be dependent on the intensity and duration of exercise[54–56]. Exercise may have a dual role in its effects on cartilage and SCB, depending on the condition of the tissue when exercise is introduced. Research suggests that non-clinical lesions present at an early age manifest into clinical signs with the increased trauma of exercise[1,44,56]. However, exercise during the growth phase appears to be of much benefit to the development of a healthy skeletal system. Early research showed young, growing horses subjected to forced exercise increased bone mineral density without an increase in DOD, and that such exercise may actually serve as a potential prophylactic to the development and aggravation of DOD[55,57,58]. The difference reported in the incidence of DOD issues in feral horses compared with domestic horses may be due, in part, to the amount of exercise (distance travelled) that feral horses experience from day 1 of age compared with most domestic horses[22].

More recent research has examined the potential benefit of exercise to the development of a healthy skeletal system in the young, growing horse. Growing horses maintained in a stall confinement system and subjected to short, intense bouts of forced exercise resulted in negative consequences to skeletal health[53]. Few statistical differences were observed in articular-calcified cartilage thickness, mineralization density and tidemark count and linear accretion rate in growing horses subjected to either low-level forced exercise or no forced exercise[14]. In 2008 and 2009, a series of articles were published that were the result of an extensive and collaborative effort to examine in detail the short- and long-term effects of exercise conditioning in young, growing horses[59–61]. In order to determine the long-term effects on skeletal health of young, growing horses subjected to forced exercise programmes, Rogers *et al.*[61] subjected young, growing horses to a controlled exercise programme and continued to assess the skeletal health through their 2–3-year-old racing careers. Phase 1 consisted of one of the two groups of horses in a pasture management system being subjected to a controlled exercise programme beginning at approximately 3 weeks of age and continuing until the initiation of race training at approximately 20 months of age. No negative effects of forced exercise were observed through this period. During phase 2, horses from phase 1 were entered into race training at approximately 20 months of age and maintained a normal race training and race event schedule to 3 years of age. No negative effects were observed in horses subjected to exercise conditioning programmes as young, growing horses prior to the initiation of race training. Horses that did receive exercise conditioning at an early age tended to respond better to initial race training and, in general, were better able to compete[61]. A detailed assessment of the biochemical composition of the articular cartilage extracellular matrix showed that the cartilage degeneration index was not different compared with control[62]. Although the exercise group had a lower glycosaminoglycans (GAG) and collagen content at one of two sites assessed, GAG distribution reflected a more mature profile. The lower collagen content may be a result of an early maturation process. Other barometers of collagen remodelling were higher in the exercise group, indicating a more advanced stage of maturity. In total, early

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exercise seems to facilitate an accelerated process of maturation in articular cartilage extracellular matrix components[62].

An observational study indicated a greater risk for DOD in foals reared in large pasture areas at a young age (< 2 months of age)[41]. In addition to the effects of exercise on articular cartilage, SCB mineral density is also influenced by early exercise. Foals subjected to exercise programmes had significantly greater SCB density than non-exercised controls. Exercised foals continued to have a greater bone density than non-exercised controls even 6 months after the cessation of the forced exercise programme[54]. These results support the observations from earlier studies regarding bone mineral density and exercise in young, growing horses[55].

In contrast to the potential benefits exercise may have on bone and articular cartilage, no significant differences were observed in tendon properties between exercised and non-exercised growing horses reared under pasture management[63,64]. Although no positive associations were observed for early exercise and tendon properties, it is important to note that early exercise did not result in any negative consequences to tendon structural properties.

Several factors may contribute to variability in specific results of studies associated with exercise in young, growing horses and its effect on skeletal health. There may be limitations of benefit based on time of implementation of an exercise programme as it relates to extracellular matrix[63]. Nutritional programmes may also influence the outcome. The most common variable that could have the most profound effect on outcome is an accurate assessment of locomotor activity not related to specific controlled exercise programmes. Use of the cumulative workload index can provide an accurate reflection of controlled exercise regimens but can provide only approximate assessment of the activity of comparative groups of horses in pasture or stalled environments[59]. Moderate variations in exercise may have significant effects on articular collagen properties and bone density. In addition, the difference may be relatively narrow between a beneficial or detrimental amount of exercise applied to young, growing horses. A system utilising a global positioning satellite device may provide more accurate and precise recommendations and control of exercise programmes in young, growing horses in order to benefit skeletal health[65,66].

Hormonal interactions

Different growth factors acting in a complicated orchestration, systemically and locally, regulate the growth of cartilage. Currently, it is thought that chondrocyte proliferation and differentiation are influenced by cell location and density *via* a feedback loop involving parathyroid hormone-related peptide, Indian hedgehog and transforming growth factor β (TGF-β)[67,68]. In addition, other systemic and local growth factors such as insulin-like growth factor-1 (IGF-1) may have effects on this regulatory process[69]. Furthermore, IGF-1 and TGF-β also may play a role in changes of cartilage protein and proteoglycan seen in osteochondritic lesions in the horse[2,15,70]. Initial research regarding nutrition suggested that diets rich in starch and sugar were a predisposing factor due to an increased insulin response to feeding, which precipitated episodic transient hypothyroidism[71]. Subsequent research suggested that increased concentrations of insulin in response to dietary-induced elevations in blood glucose may be more directly associated with DOD than the hypothesis of transient hypothyroidism. Increases in glycaemic response measurements have been associated with increased incidence of DOD[72]. Dietary glycaemic response may affect, directly and indirectly, the hormonal responses that may affect cartilage metabolism. How nutrition interacts with various growth-related hormonal theories associated with DOD is not completely understood. It has been suggested that feeding diets rich in starch and sugar may contribute to hormonal interactions that facilitate DOD issues in growing horses. However, such interpretations may be complicated because glycaemic response may be affected by rate of consumption, physical form of the diet, diet composition and activity level. Substituting fat and fibre calories for sugar calories has been shown to affect systemic concentrations of IGF-1 in foals[73,74]. Other studies that address the effect of added fat and/or reduced starch and sugar diets on systemic IGF-1 or TGF-β[75,76]or TGF-β in synovial fluid[77] have not observed any significance.

The optimal amount of calories and the form in which they are presented to reduce the potential of DOD is not yet known. To date, the relatively weak association of nutrition and DOD has suggested a diminished role of nutrition in the aetiology and pathogenesis of DOD, particularly in the consideration of bilateral focal point lesions, as one would expect nutritional insult to have a more systemic effect. However, an old management rule of thumb that may be very applicable regarding nutrition and DOD would be 'the more of the nutrient requirements of a horse that can be met with long-stem forage/roughage, the better will be their overall health'. In addition, what is not met with forage/roughage should be compensated for with a well-formulated, research-based, high-quality concentrate. When feeding with such concentrates, their nutritional balance and quality should not be compromised with the added supplements.

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Other considerations

There has been little work addressing the possible associations between vitamin status and DOD. The pathology of the various forms of DOD does not suggest a vitamin deficiency or excess issue. Mineral nutrition for the growing horse seems to be well established as it relates to object deficiencies, particularly for minerals of primary concern such as calcium, phosphorus, zinc and copper[78,79]. Additional research in this area may be warranted as we continue to learn more about the intricate relationships of vitamins and minerals and growth physiology. However, consideration should be taken that deficiencies of vitamins and minerals are associated with systemic disease conditions and not reflective of localized growth cartilage osteochondrosis. Furthermore, supplementation of minerals should not be considered a prescription for DOD.

Association of DOD with other disease states is not yet known. General stressors of weaning or other systemic stressors also may have an association with incidence and severity of DOD, but research in this area is lacking.

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