Kentucky Equine Research®
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Advances in Equine Nutrition

Volume IV

Edited by

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MANAGING GROWTH TO PRODUCE A SOUND, ATHLETIC HORSE

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Introduction

A horse's maximal mature body size is genetically predetermined, but growth rate can be influenced by a number of factors including environment, nutrition, and management. Optimal growth rate results in a desirable body size at a specific age with the least amount of developmental problems. Managing growth in horses becomes a balance between producing a desirable individual for a particular purpose without creating skeletal problems that will reduce a horse's subsequent athletic ability. Growing a foal too slowly results in the risk of it being too small at a particular age or never obtaining maximal mature body size. Growing a foal too quickly results in the risk of developmental orthopedic disease (DOD) manifestations such as physitis, angular limb deformities, and osteochondritis dissecans (OCD).

There is no single growth rate that is desirable for all types of horses. Therefore, horses should be managed differently for varying growth rates. Horses will generally reach physical maturity at around four to five years of age. Compared to Thoroughbreds, many breeds such as warmbloods are not expected to compete until later in life, so there is little incentive for rapid growth. Instead, a slow, steady growth rate that will allow the horse to reach maximal mature body size with the fewest problems is desirable.

Thoroughbred racehorses are a different story since they are expected to be competitive athletes at two years of age. Therefore, mature body size is not the most important end point that Thoroughbred breeders wish to achieve. In fact, there can be several important developmental milestones that must be reached even before a young Thoroughbred enters its first race.

Most Thoroughbreds are sold either as weanlings or yearlings at commercial auctions throughout the year. The size of the foal at auction can greatly impact its selling price, so there is strong incentive to market large weanlings and yearlings. At the Keeneland September sales, yearlings that were heavier and taller, but not fatter (measured by body condition score), sold for higher prices (Pagan et al., 2005). Just as important as size, however, is the foal's skeletal soundness at the time of the sale. A delicate balancing act exists between accelerated growth and skeletal soundness.

A horse undergoes rapid development in weight, height, and bone mineral content (BMC) within its first year of life. Within 30 minutes of birth, a foal can stand, and

within hours can run at speeds no human athlete will ever achieve. However, despite this early development, a newborn foal has only 17% of its mature BMC and 10% of its mature body weight (Lawrence, 2003). Thoroughbreds will reach 84% of their mature height at 6 months of age; by 12 months they will have reached 94% of mature height; and by 22 months they will have almost finished growing in height (Lawrence, 2003). Mature body weight is reached at a slightly slower rate. During the first six months of life a foal will gain 46% of its mature weight; at 12 months it will have reached 65% of its mature weight; and by 22 months it should be at 90% of its adult body weight (Lawrence, 2003).

Like height and body weight, BMC has been reported to be closely related to age. However, the rate at which maximum BMC is achieved is much slower than height and body weight. At six months of age, horses have attained 68.5% of the mineral content of an adult horse, and by one year of age they have reached 76% of maximal BMC. However, horses do not attain maximum BMC until six years of age (Lawrence, 1994, 2003).

Numerous studies have investigated the relationship between BMC and age in young growing horses. Glade et al. (1986) reported that sound velocities measured through the mid cannon bone and the apparent ultimate breaking strengths of the metacarpal and metatarsal bones of young Thoroughbreds changed with age in a curvilinear fashion, although the relationships appeared linear until 300 days of age. This is similar to findings of El Shofra et al. (1979), Jeffcott and McCartney (1985), and Lawrence and Ott (1985).

In a study conducted by Hoffman et al. (1999) there were close correlations between BMC and age ($R^2 = 0.81$), BMC and body weight ($R^2 = 0.76$), and BMC and height ($R^2 = 0.76$) from birth up to 450 days of age. In this study BMC increased with age and season in three waves with two plateaus, one at two to three months of age and the other in the winter season between November and January at eight to ten months of age. It was concluded that these seasonal changes were due to changes in diet and exercise activity. The first plateau at two to three months of age was associated with a change in diet from the mares' milk to a pasture-based diet. The winter plateau was associated with limited winter activity due to packed snow and ice.

In a more recent study conducted by Reichman et al. (2004), where BMC was measured by means of dual photon absorptiometry in Quarter Horse foals during their first year of life, correlations with age ($R^2 = 0.83$) and height ($R^2 = 0.91$) were reported similar to previously published studies. In this study a winter seasonal influence similar to the Hoffman study was not recorded; however, this study was conducted in Brazil where milder climatic conditions exist. A weaning effect was recorded in foals weaned at 17 weeks but not in foals weaned at the ages of 19 and 24 weeks.

Kentucky Equine Research tracked body weight and skeletal growth in a group of 30 Thoroughbred foals born in 2003 and raised on a large commercial breeding farm in central Kentucky. Dorsopalmar radiographs of the third metacarpal bone (McIII) were taken on a monthly basis. An aluminum step wedge was exposed simultaneously with the McIII. This was used as a reference standard. Radiographic bone aluminum

equivalencies (RBAE) were recorded at three sites: lateral and medial sites with peak densities, and a central site of least density in the medullary cavity. The bone mineral content in grams per 2-cm cross section of bone was estimated using regression equations derived by Ott et al. (1987).

Bone morphological measurements (bone width, medullary width, lateral and medial cortical width) were determined from radiographs. Body weights were also measured monthly using in-ground digital scales.

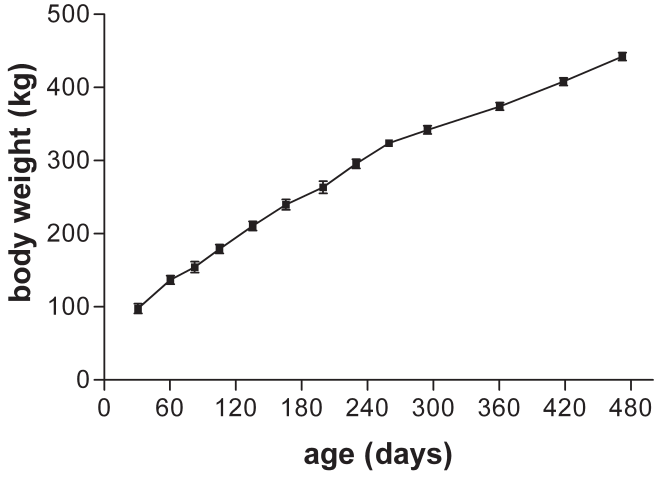

Figure 1. Body weight (kg) of Thoroughbred foals.

Table 1. Bone mineral content (BMC) and bone morphological measurements in the third metacarpal bone of Thoroughbred foals.

Month	*Age*	*Mean BMC (g/2cm)*	*Width (mm)*	*Medial Cortical Width (mm)*	*Lateral Cortical Width (mm)*	*Medullary Bone Width (mm)*
Mar-03	30.7 ± 4.6	6.7 ± 0.8	4.7 ± 0.3	4.1 ± 0.2	18.8 ± 0.6	27.5 ± 0.5
Apr-03	60.3 ± 4.6	8.3 ± 0.4	5.1 ± 0.2	4.8 ± 0.1	18.1 ± 0.3	28.0 ± 0.3
May-03	82.4 ± 6.8	10.6 ± 0.8	6.9 ± 0.3	5.8 ± 0.2	18.1 ± 0.3	30.8 ± 0.5
Jun-03	105 ± 5.9	11.0 ± 0.3	7.2 ± 0.2	5.8 ± 0.1	18.3 ± 0.3	31.3 ± 0.3
Jul-03	135.3 ± 6.1	13.6 ± 0.5	7.4 ± 0.2	7.5 ± 0.4	18.8 ± 0.4	33.8 ± 0.4
Aug-03	165.5 ± 5.9	14.0 ± 0.6	8.2 ± 0.2	6.6 ± 0.1	18.2 ± 0.3	33.0 ± 0.3
Sep-03	199.5 ± 6.3	15.8 ± 0.6	8.7 ± 0.2	7.0 ± 0.2	17.9 ± 0.3	33.6 ± 0.3
Oct-03	229.3 ± 6.5	16.2 ± 0.7	8.4 ± 0.3	6.8 ± 0.2	17.9 ± 0.5	33.2 ± 0.5
Nov-03	259.4 ± 6.3	13.7 ± 0.5	10.1 ± 0.3	7.1 ± 0.2	18.2 ± 0.3	35.3 ± 0.3
Dec-03	294.6 ± 6.2	13.9 ± 0.4	9.7 ± 0.3	7.6 ± 0.2	17.8 ± 0.3	35.1 ± 0.3
Feb-04	360.4 ± 6.6	13.6 ± 0.5	9.6 ± 0.3	7.9 ± 0.2	18.3 ± 0.4	35.7 ± 0.4
Apr-04	418.4 ± 6.3	17.2 ± 0.4	9.7 ± 0.3	7.3 ± 0.2	18.2 ± 0.4	35.2 ± 0.4
Jun-04	472.1 ± 5.9	18.3 ± 0.3	10.3 ± 0.4	7.4 ± 0.2	17.9 ± 0.3	35.6 ± 0.4

BMC increased significantly from the previous month in May 2003, July 2003, September 2003, and April 2004. November 2003 saw a dramatic reduction in values from those recorded in October (P<0.001) with values not increasing again until April 2004 (Figure 2). The reduction in BMC through the fall and winter months coincided with a reduction in body weight average daily gain (ADG), which is a typical growth pattern for Thoroughbred foals raised in central Kentucky (Pagan et al., 1996). Other factors such as pasture availability, day length, and voluntary activity may have also contributed to this reduction in BMC and warrant further investigation.

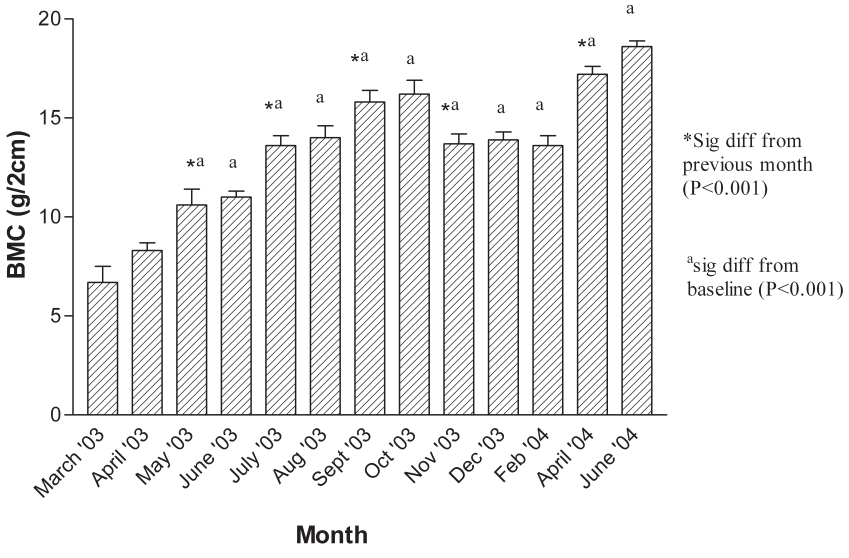

Figure 2. Bone mineral content (BMC) in the third metacarpal bone of Thoroughbred foals.

Factors Affecting DOD and Bone Strength Development

DIET

A balanced feeding regime is critical in the production of a sound equine athlete. Proper feeding begins with the pregnant broodmare. How the pregnant mare is fed will impact both the size and the skeletal soundness of the foal (Lawrence, 2006). Feeding is most important during the last trimester of pregnancy when 75% of fetal growth occurs (Huntington et al., 2003). The most critical nutrients for broodmares and foals are energy, protein, lysine, calcium, phosphorus, copper, and zinc.

ENERGY

Energy intake controls growth rate when all other nutrients are in the foal's ration in sufficient quantities. The most effective way to manage growth in foals is to adjust

concentrate and/or forage intake to provide a level of calories that fuels a desired growth rate. Therefore, knowledge of energy requirements for growth is critical.

During the first two months of life, mare's milk contains enough energy and protein to meet the needs for growth. Foals with an expected mature weight of 450-500 kg require approximately 9 kg of milk for each kg of gain at seven days of age, 13 kg at one month of age, and 15 kg at two months of age (Kohnke et al., 1999).

The 1989 NRC does not give specific feeding recommendations for the suckling foal other than to say that supplemental feed prior to weaning may be desirable in foals nursing mares that are poor milkers. This differs from the previous NRC edition (1978) which recommended that a three-month-old suckling foal gaining 2.64 lb/day should receive 6.89 Mcal DE/d as supplemental feed. In a Ralston Purina study, Quarter Horse foals averaged about 2.41 lb ADG over this same time period (Pagan, 1998a). These foals consumed an average of 3.05 Mcal DE per day as supplemental feed over this two-month period. Regression analysis of these data (weighted for mare weight) yields the equation:

$$\text{Foal ADG (lb/day)} = 2.08 + 0.109 \text{ (foal DE (Mcal DE/d))}$$

Brown-Douglas and Pagan (2006) reported on growth rates in 13,429 Thoroughbred foals from around the world. These foals averaged about 2.56 lb gain/day during the second and third months of lactation. Using the above equation, foals would need to consume 4.36 Mcal of supplemental DE/day during the second and third months of lactation to fuel this rate of growth. No supplemental feeding would result in a growth rate of 2.08 lb/day. These figures agree with Doureau et al. (1982) who stated that foals require 15 kg of milk per kg live weight gain at eight weeks of lactation. Fifteen kilograms per day is the expected lactation. Thus, milk alone during this period of lactation could be expected to support a growth rate of only 2.1-2.2 lb/day, a rate which is below that desired by today's horseman.

Typical foal concentrates contain around 1.4 Mcal DE/lb. Therefore, a foal of this age would need to eat about 3.0 lb of concentrate per day to support the type of growth rate typically seen under commercial conditions. It is not known how much hay and pasture a suckling foal will consume, but these energy sources should be kept in mind when deciding how much supplemental grain to feed. Ideally, weighing the foal regularly can aid in adjusting its grain intake.

It should also be noted that higher supplemental DE intakes may result in elevated growth rates which are undesirable since accelerated growth at this early age may contribute to developmental orthopedic disease in foals. It would therefore seem unwise to allow suckling foals free access to supplemental feed in a "creep" feeding arrangement. Instead, the foal should be fed a weighed amount of supplemental feed when the mare is fed. This can easily be accomplished by simply tying up the mare at meal time. It is a good management practice to feed both the mare and the foal at least twice daily, and three or four times per day if possible.

The energy requirement for growing horses is calculated by adding the foal's maintenance requirement to the requirement for tissue energy deposition calculated using the efficiency of conversion of dietary energy for gain (Ott, 2001). The digestible energy requirement for maintenance is DE (Mcal) = 1.4 + 0.03 BW (body weight, kg) (NRC, 1989). Combining the requirements for maintenance and growth yields the following equation: DE (Mcal) = (1.4 + 0.03 BW) + (4.81 + 1.17X – $0.023X^2$) (ADG), where X is age in months and ADG is average daily gain in kg.

Nutritional Factors as a Cause of Developmental Orthopedic Disease

MINERAL DEFICIENCIES

A deficiency of minerals, including calcium, phosphorus, copper, and zinc, may lead to developmental orthopedic disease. The ration of a growing horse should be properly fortified because most commonly fed cereal grains and forages contain insufficient quantities of several minerals. A ration of grass hay and oats would supply only about 40% and 70% of a weanling's calcium and phosphorus requirement, respectively, and less than 40% of its requirement for copper and zinc. The best method of diagnosing mineral deficiencies is through ration evaluation. Blood, hair, and hoof analyses are of limited usefulness.

Copper has received a great deal of attention in recent years due to its suggested role in the pathogenesis of developmental orthopedic disease. Lysyl oxidase is a copper-containing enzyme involved in the cross-linking of elastin and collagen in cartilage. Disruption to this process affects normal bone cartilage development. The 1989 NRC estimates that all classes of horses require 10 mg/kg dietary copper. This recommendation appears reasonable for horses at maintenance. However, research suggests that copper requirements of growing horses and broodmares may be considerably higher.

Two dose response studies examining the effects of increased dietary copper intake on bone and cartilage abnormalities (Knight et al., 1990; Hurtig et al., 1993) found that the incidence of DOD was decreased by increasing the copper content of the diet above NRC recommendations. van Weeren et al. (2003) examined the copper status of foals at birth and the incidence of radiographic signs of osteochondritis dissecans (OCD) in warmblood foals genetically prone to OCD. Foals with high liver copper levels at birth had decreased severity of stifle OCD from 5 to 11 months, whereas foals with low liver copper levels had more severe signs of OCD at 11 than 5 months.

Zinc is present as a component of many enzymes and the biochemical role of zinc relates largely to the functions of these enzymes. Zinc is required for the synthesis of alkaline phosphatase, an enzyme actively involved in bone development. Ciancaglini and coworkers (1990) reported a zinc deficiency can cause a decrease in the synthesis of this enzyme.

Much of the research on trace minerals, in particular copper, has focused more on bone abnormalities such as OCD than on bone strength. However, Ott and Asquith

(1995) examined the effect of trace mineral supplementation on bone mineral content. Yearlings supplemented with a complete trace mineral package had greater final BMC values than yearlings fed a basal diet containing no trace mineral supplementation. Horses fed the complete trace mineral package also had greater BMC gain than horses receiving only supplemental Cu and Zn.

MINERAL EXCESSES

Horses can tolerate fairly high levels of mineral intake, but excesses of calcium, phosphorus, zinc, iodine, fluoride, and certain heavy metals such as lead and cadmium may lead to developmental orthopedic disease. Mineral excesses may occur because of overfortification or environmental contamination.

Massive oversupplementation of calcium (>300% of required) may lead to a secondary mineral deficiency by interfering with the absorption of other minerals such as phosphorus, zinc, and iodine. Excessive calcium intake may be compounded by the use of legume hays as the primary forage source. Iodine and selenium oversupplementation occurs if supplements are fed at inappropriate levels. A ration evaluation is the best way to identify this type of mineral imbalance.

Environmental contamination is a more likely cause of developmental orthopedic disease because contamination may result in extremely high intakes of potentially toxic minerals. In addition, chemical analysis of hoof and hair samples may reveal valuable information in such a situation. Farms that are located near factories or smelters are the most likely candidates for this type of contamination. Horses grazing pastures contaminated with zinc, iron, and lead from industrial smelters in Australia were found to have enlarged joints, flexural deformities, lameness, and multiple OCD lesions (Eamens et al., 1984). On post mortem, tissue levels of zinc and lead were elevated, while copper was decreased. The authors concluded that a zinc-induced copper deficiency was responsible for the abnormalities. OCD from a zinc-induced copper deficiency has also been reported on farms using fence paint containing zinc or galvanized water pipes. If a farm is experiencing an unusually high incidence of developmental orthopedic disease or if the location and severity of skeletal lesions are abnormal, environmental contamination should be investigated. Blood, feed, and water analysis should be performed.

MINERAL IMBALANCES

The ratio of minerals may be as important as the actual amount of individual minerals in the ration. High levels of phosphorus in the ration will inhibit the absorption of calcium and will lead to a deficiency, even if the amount of calcium present was normally adequate. The ratio of calcium to phosphorus in the ration of young horses should never dip below 1:1 and ideally it should be 1.5:1. Too much calcium may affect phosphorus status, particularly if the level of phosphorus in the ration is marginal. Calcium to phosphorus ratios greater than 2.5:1 should be avoided if possible. Forage

diets with high calcium levels should be supplemented with phosphorus. The ratio of zinc to copper should be 3:1 to 4:1.

DIETARY ENERGY EXCESS

Excessive energy intake can lead to rapid growth and increased body fat, which may predispose young horses to developmental orthopedic disease. A Kentucky study showed that growth rate and body size may increase the incidence of certain types of developmental orthopedic disease in Thoroughbred foals (Pagan, 1998b). Yearlings that showed osteochondrosis of the hock and stifle were large at birth, grew rapidly from three to eight months of age, and were heavier than the average population as weanlings.

The source of calories for young horses may also be important, as hyperglycemia or hyperinsulinemia have been implicated in the pathogenesis of osteochondrosis (Glade et al., 1984; Ralston, 1995). Foals that experience an exaggerated and sustained increase in circulating glucose or insulin in response to a carbohydrate (grain) meal may be predisposed to development of osteochondrosis. In vitro studies with fetal and foal chondrocytes suggest that the role of insulin in growth cartilage may be to promote chondrocyte survival or to suppress differentiation, and that hyperinsulinemia may be a contributory factor to equine osteochondrosis (Henson et al., 1997).

Research from Kentucky Equine Research (Pagan et al., 2001) suggests that hyperinsulinemia may influence the incidence of OCD in Thoroughbred weanlings. In a large field trial, 218 Thoroughbred weanlings (average age 300 ± 40 days, average body weight 300 kg ± 43 kg) were studied. A glycemic response test was conducted by feeding a meal that consisted of the weanlings' normal concentrate at a level of intake equal to 1.4 g nonstructural carbohydrate (NSC) per kilogram body weight. A single blood sample was taken 120 minutes post feeding for the determination of glucose and insulin.

In this study, a high glucose and insulin response to a concentrate meal was associated with an increased incidence of OCD. Glycemic responses measured in the weanlings were highly correlated with each feed's glycemic index (GI), suggesting that the GI of a farm's feed may play a role in the pathogenesis of OCD. Glycemic index characterizes the rate of carbohydrate absorption after a meal and is defined as the area under the glucose response curve after consumption of a measured amount of carbohydrate from a test feed divided by the area under the curve after consumption of a reference meal (Jenkins et al., 1981). In rats, prolonged feeding of high GI feed resulted in basal hyperinsulinemia and an elevated insulin response to an intravenous glucose tolerance test (Pawlak et al., 2001). Hyperinsulinemia may affect chondrocyte maturation, leading to altered matrix metabolism and faulty mineralization or altered cartilage growth by influencing other hormones such as thyroxine (Jeffcott and Henson, 1998). Based on the results of this study, it would be prudent to feed foals concentrates that produce low glycemic responses. More research is needed to determine if the incidence of OCD can be reduced through this type of dietary management.

Conclusion

Feeding and managing growing foals is a balancing act between achieving a commercially desirable level of growth and prevention of developmental orthopedic disease. The keys to success are assuring that the foal's ration is correctly balanced and then regulating feed intake to obtain a safe, desirable growth rate.

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