A review of studies which investigate nutritional causes of Developmental Orthopaedic Disease in growing foals

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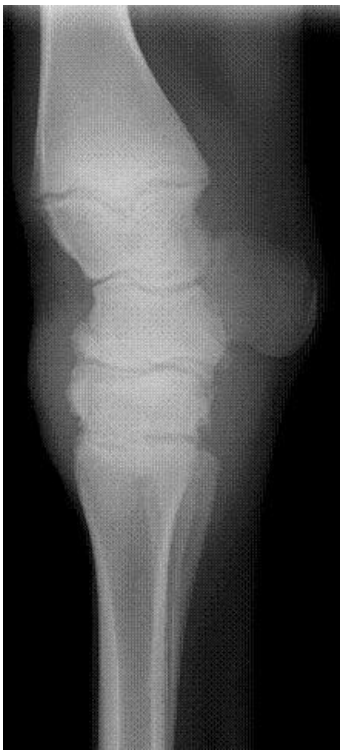
Excess energy and rate of growth as a cause of Developmental Orthopaedic Disease



It is sometimes stated that Developmental Orthopaedic Diseases (DODs) are due entirely to excessive energy intake and not to other nutrient imbalances. After feeding weanlings a high energy diet (129% of NRC recommendations), Savage et al., (1993) did indeed find a very high incidence of DOD at necropsy (11/12) when compared to the control (1/12) or at a high protein diet (1/6). However in a more recent study, no statistically significant feeding level effect could be established on limb DOD in foals with fast and moderate growth rates induced by two levels of balanced nutrient intake (at 130% and 150% of recommendations) from birth to one year of age (Donabédian et al., 2006).

It is generally believed that DOD is more common in large, rapidly growing horses, possibly as a result of excessive weightbearing and other biomechanical stresses on bones and joints whereas other studies have found no difference in the size or average daily gain of horses with and without DOD. In a study by Pagan and Jackson (1996), foals that developed Osteochondritis Dissecans (OCD) as yearlings tended to be large foals at birth and grew rapidly, however it was undetermined if the possible effect of size and growth rate on bone development were the result of nutrition or other factors such as genetics. In another study by Petersen et al. (2001a,b), there were no differences observed in bone mineral quality in horses fed for rapid gain versus foals fed for slow gain. They noted however that three of six foals fed for rapid gains developed clinical signs of physitis and joint pain, but these signs were no longer evident within 60 days.

These studies suggest that feed imbalance and high energy intake are stronger DOD inducing factors than a high but balanced feeding level. Current feeding practices are designed to control growth via energy intake, however growth rate is not only a factor of nutrition, but also of genetic capacity. A review of the occurrence of DOD in relation to energy and nutrient intake will assist in our understanding of energy and mineral requirements of these foals with a genetic disposition to a fast growth rate.

Type of dietary carbohydrates and their influence on the occurrence of osteochondritis dissecans

Feeding diets high in soluble carbohydrates to growing horses has been implicated in the development of orthopaedic diseases and it has been suggested that glucose intolerance caused by insulin resistance may be associated with OCD in young horses.

Pagan et al. (2001), studied the possible implication of hyperglycaemia and hyperinsulinemia with OCD in a field study where young horses were fed concentrate feeds with different glycaemic indexes. The results described a strong positive correlation across all farms between the incidence of OCD and serum glucose and insulin levels 120 minutes after feeding. However, within a farm, there were no significant differences in the glycaemic response between horses that had lesions and those that did not. The authors suggested that diet-induced hyperglycaemia or hyperinsulinemia predisposes every weanling to OCD, but other factors such as biomechanical stress or trauma are needed to produce a clinically relevant lesion and that it would be prudent to feed foals concentrates that produce low glycaemic responses. The authors also suggested that further research is required to determine if a glycaemic response test using a more

standardized oral glucose challenge (i.e. dextrose) can be used to identify younger individuals predisposed to OCD.

In a further study, Ott et al. (2005) investigated the influence of starch intake on growth and skeletal development of weanling horses. Body weight and length gains were highest in those weanlings fed high-starch rations but bone osteochondrotic lesions were not found to be related to diet. The authors concluded that at least under the experimental conditions, weanlings need some readily available glucose to support normal growth and that skeletal development of weanling horses was not affected by consumption of high-starch concentrates. In an investigation of dietary energy source on serum concentration of insulin-like growth factor-1, growth hormone, insulin, glucose, and fat metabolites in weanling horses, Ropp et al. (2003) found that insulin-like growth factor-1 did not differ between fat supplemented and conventional carbohydrate-supplemented foals. The authors suggested that substitution of fat for soluble carbohydrate may not necessarily alleviate orthopaedic diseases associated with rapid growth in susceptible animals.

Growth and bone development in foals was examined as affected by seasonal changes in pasture and dietary supplementation with concentrates rich in sugar and starch or in fat and fibre (Hoffman, 1999). In weanlings and yearlings fed the fat and fibre supplement, their bone mineral content was lower at certain points, possibly due to a reduction in calcium availability through cation exchange and waterholding capacity of fibre or the formation of calcium soaps with fat in the small intestine, and also a reduction in exercise (as these foals tended to be less active on a daily basis).

Some feed companies are now marketing “Low Glycaemic Index” feeds to the stud farm market. The inconsistencies in research data to date suggest that the effects of these “novel” feeding strategies on OCD and growth require further investigation.

Protein and its effect on skeletal disease

While likely to be rare on the larger commercial stud farms in Australia, it has been suggested that inadequate protein intake may result in the inhibition of bone remodelling, which may contribute to DOD. Foals reared on mature grass hay or pasture forage, and a cereal grain mix containing minimal protein may be at risk of a protein deficiency which may be exacerbated under drought conditions.

Excess protein however has been frequently implicated as a cause of DOD, potentially due to excessive calcium loss (Barzel and Massey, 1998) although direct evidence linking excess protein to occurrence of DOD is lacking. Excess protein consumption is more likely to be a concern rather than insufficient protein as equine rations are generally formulated on the basis of crude protein rather than exact amino acid requirements. *Ad libitum* grazing of pastures also makes for the control of protein intake difficult.

Minerals and their importance in OCD

Developmental orthopaedic diseases frequently attributed to excessive energy or protein intake may instead be caused by an amount of phosphorus, calcium, zinc or copper in the diet inadequate to support the rate of growth permitted by the amount of energy and protein consumed. Deficiencies and imbalances of calcium and phosphorus have been widely recognized as causes of DOD. Less attention has been paid to excesses of calcium and phosphorus. High levels of calcium can lead to hormonal changes (increased calcitonin) which may inhibit the replacement of cartilage by bone, resulting in OCD. This may be of particular concern due to the increased use of Lucerne hay which is high in calcium due to current drought conditions and warrants further investigation.

Trace minerals

Investigations on trace minerals have suggested that copper and zinc supplementation could decrease the incidence of DOD. In a study investigating the effects of copper supplementation on the prevalence of cartilage lesions in foals, mares were fed rations containing 13 ppm copper (control group) or 32 ppm (supplemented group). Foals were fed a pellet containing 15 or 55 ppm copper and were destroyed at 90 or 180 days. In foals killed at 90 days, there were over twice as many lesions of osteochondrosis and more than four times as many articular lesions of osteophyte formation in the control group versus the supplemented group. In foals killed at 180 days, there were seven times more articular lesions of osteophyte formation or thinning, nearly twice as many lesions of osteochondrosis in the physis and over five times as many involving the A-E complex in control foals compared to supplemented foals (Knight et al., 1990). In another study, five of nine foals fed from 4 to 10 months of age a diet containing 7 ppm copper developed OCD and subchondral bone cysts (SBC), whereas no DOD occurred in any of nine foals fed 30 ppm dietary copper (Hurtig et al., 1990). The liver copper content was six times lower in the low copper-fed foals, but there was no difference in their plasma copper, calcium, phosphorus or zinc concentration or their plasma alkaline phosphatase activity. In another report, seven of eight nursing foals with osteochondrosis had plasma copper concentrations below normal (Bridges et al., 1984). A more recent study however showed that there was no relationship between foal or mare liver copper concentration and osteochondrosis status at either 5 or 11 months. However, osteochondrotic lesions in foals with low-level copper status at birth decreased significantly less in number and severity than those in foals with high-level copper status at birth. The authors of this paper concluded that copper is not likely to be an important factor in the aetiopathogenesis of osteochondrosis, but there may be a significant effect of high copper status on the natural process of repair of early lesions (van Weeren et al., 2003). Radiographically visible OCD lesions present at weaning resolved in foals fed a diet containing 30 ppm copper, whereas in those fed a 7 ppm copper-containing diet, the lesions worsened and mild angular and flexure deformities, intermittent lameness, and joint effusions occurred (Hurtig, 1993). In addition, their growth plates and metaphyseal bone contained microfractures, and they had wider zones of provisional calcification and higher rates of metaphyseal bone accretion as compared to the weanlings fed the high copper diets.

Dietary zinc deficiency has also been postulated as a cause of DOD (Knight et al., 1985). In an unpublished report, it was stated that three-month old foals fed a diet containing 152 ppm zinc had less cartilage defects at 6 months of age than those fed 42 ppm (Lewis, 1995). This diet also contained 35 ppm copper. In another study, foals with osteodystrophy administered 200mg Zn/day (equivalent to increasing their dietary zinc from 25 to 65 ppm) increased their rate of recovery as compared to those not given additional zinc (Spais et al., 1976).

Excess zinc, iron and cadmium have been implicated in decreasing copper absorption or utilization resulting in a secondary copper deficiency (young horses grazing pastures contaminated with zinc, iron and lead from industrial smelters near Newcastle, NSW, were found to have enlarged joints, flexural deformities, lameness, and multiple OCD lesions (Eamens et al., 1984)). This may be of particular importance to stud farms in the Hunter Valley and warrants further investigation.

Despite these studies, debates continue over the level of trace minerals required to prevent DOD. The NRC (1989) recommended that all horses receive 40ppm zinc and 10 ppm copper in the diet and the recent 2007 publication does not appear to have increased these recommendations significantly. The importance of copper particularly and its significance in DOD requires further examination.

Pasture effects

Anecdotal evidence suggests that the incidence of DOD is reduced in times of drought, and increases in good seasons. Lush, rapidly growing pastures generally have a higher content of soluble carbohydrates and protein than the dry, mature pastures found in the drier times of the year. The higher incidence of DOD noted in good seasons may, therefore, be a result of increased intake of soluble carbohydrates, as this factor has been experimentally shown to cause DOD (Savage et al., 1993).

Lush pasture diets also may affect calcium balance and absorption. Dairy cows grazing lush pastures have a much higher incidence of milk fever (low blood calcium) than those grazing mature pastures or hay (Harris, 1981). This is thought to be a result of changes in calcium absorption and excretion (Hyde, 1994). There has been no published research on the effect of lush pasture on calcium balance in the horse.

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