Pferdeheilkunde 12 (1996) 3 (Mai-Juni) 338–342

Nutrition and the development of Osteochondrosis (Dyschondroplasia)

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Summary

This paper reviews the effects of overfeeding of foals, particularly with dietary excess of digestible energy and crude protein. The other important nutritional component in the development of osteochondrosis involves mineral imbalance. This may be associated with excessive dietary phosphorus while excess calcium does not appear to induce the condition. There is evidence that a deficiency of copper predisposes to osteochondrosis as well as other forms of development orthopaedic disease. Toxicity due to zinc and cadmium can produce cartilagenous lesions similar to osteochondrosis. It is clear therefore that nutrition plays an important role in the pathogenesis of ostechondrosis, but many other factors (e.g. growth rate, genetics, biomechanics, endocrinological and growth factors) also need to be considered along with diet.

keywords: osteochondrosis, nutrition, energy, minerals

Ernährung und die Entwicklung von Osteochondrosen (Dyschondroplasien)

In der Übersicht werden die Effekte einer Überfütterung von Fohlen insbesondere mit verdaulicher Energie und Protein behandelt. Ein anderer wichtiger Komplex bei Entwicklung der Osteochondrosen ist die Imbalanz von Mineralstoffen. In diesem Zusammenhang sind Überschüsse an Phosphor von Bedeutung, während ein Überschuß an Kalzium die Erkrankung nicht zu begünstigen scheint. Bisherige Befunde zeigen, daß auch ein Cu-Mangel für Osteochondrosen und andere Arten orthopädischer Erkrankungen beim wachsenden Tier disponiert. Toxische Mengen von Zink und Kadmium verursachen ähnliche Knorpelschäden, wie sie bei der Osteochondrose beobachtet werden. Insgesamt ist eindeutig, daß die Fütterung eine wichtige Rolle bei der Entstehung von Osteochondrosen spielt, aber viele andere Faktoren (wie Wachstumsgeschwindigkeit, Vererbung, Biomechanik, Endokrinium) in Zusammenhang mit der Ernährung beachtet werden müssen.

Schlüsselwörter: Osteochondrose, Ernährung, Energie, Mineralien

Introduction

The term Developmental Orthopaedic Disease (DOD) is used to describe the numerous disturbances of cartilaginous and skeletal growth in foals and many factors have been implicated in their pathogenesis. Putative nutritional, endocrinological, genetic and biomechanical mechanisms have been focused upon, yet the pathophysiological basis for the primary lesion has not yet been elucidated. One of the most important developmental problems is that referred to as osteochondrosis or dyschondroplasia. The initiating lesion of dyschondroplasia has been defined as a failure of endochondral ossification, with major effects recognized in the zone of hypertrophy of the articular/epiphyseal cartilage complex, affecting the differentiation and maturation of chondrocytes. There is considerable evidence that nutrition plays a major role in the induction of the condition in foals (*Jeffcott* 1991).

Carbohydrate overload

Over-nutrition as a predisposing factor in the production of osteochondrosis in the horse was originally based on the work of *Hedhammar* et al (1974) in the dog. A study by *Glade* and *Belling* (1986) who fed young Thoroughbred horses ~ 130% of the *National Research Council (NRC)* (1989) recommendations for both digestible energy (DE) and protein appeared to induce lesions of osteochondrosis and physeal dysplasia. *Glade, Gupta* and *Reimers* (1984) postulated that the deleterious effects seen in cartilage of horses overfed with carbohydrate were mediated endocrinologically. Thyroxine is required for the maturation of chondrocytes (*Glade* 1992) and possibly collagen and proteoglycan synthesis. Horses fed high carbohydrate diets may become temporarily hypothyroxemic post-prandially, which could have adverse effects on the cartilage. However, if this were completely accurate it would be expected that young horses fed high amounts of lipid (fat/oil) may be protected and this is not true (*Savage, McCarthy* and *Jeffcott* 1993a). A series of controlled experiments showed that diets with 128% *NRC* (1989) DE requirements, composed of both carbohydrate and corn oil components were instrumental in the induction of osteochondrosis. Control foals in these experiments were fed a diet based on 100% of *NRC* (1989) requirements for weanlings growing at an average daily gain (ADG) of

0.65–0.85 kg/day (*Savage* 1992; *Savage* et al. 1993a). It is conceivable that the feeding of excessive DE in some way causes endocrinological alterations especially through mediation of local cartilaginous factors.
The high energy diet does not manifest its effects through increases in ADG, as no significant increases occurred in foals fed the high energy diet compared to those fed the basal control diet (Savage et al 1993a). Although this seems paradoxical it has been reported in numerous studies in which foals have been fed high energy or control diets (*Knight* et al. 1992). However, *Leach* (1990) found that 2 year old horses fed ad libitum were 13% heavier than those fed a diet approximating *NRC* (1989) recommendations .

Superimposed effects of exercise

Controversy surrounds the subject of exercise and the subsequent effects on the prescence and severity of osteochondrosis lesions. Exercise in the study by *Savage* et al was restricted purposefully, however, *Leedle, Raub, Anderson* and *Wilson* (1995) fed weanling Quarterhorses diets of low or high energy/protein content and exercised these weanlings to different extents. They saw a trend of increased incidence in macroscopic osteochondrosis in non-exercised foals fed 110% of *NRC* (1989) DE and protein recommendation compared to exercised foals on this diet; or exercised foals that received 135% of *NRC* (1989) DE and protein recommendation. However, no histological evaluation was performed and therefore objective interpretation is difficult. *Bruin* and *Creemers* (1994) showed the effect of varying levels of exercise on the incidence of foals maintained on either low or high energy diets. The foals on the high intensity exercise programme fed low energy actually had a higher level of osteochondrosis (13%) than foals fed a high energy diet during high intensity exercise. Foals fed the low energy diet whilst on the low intensity exercise regime also had a low incidence of osteochondrosis. Like findings by *Savage* et al. (1993a), foals fed high energy and provided with little exercise had a high incidence of osteochondrosis. These findings may indicate that increased exercise may be beneficial when high energy diets are chosen, but detrimental if lesions are already present, as purported in the group in which foals were fed a low energy diet, but exercised more frequently and intensively.
Another possible reason that foals fed excessive energy may develop osteochondrosis is that copper absorption may be decreased, or in cases in which high fats are fed, the calcium absorption may be increased (*Atteh* and *Leeson* 1984) inducing a relative phosphorus excess.

Excess dietary protein

Diets excessive in protein have been proposed as a cause of DOD (*Pool* 1987; *Stashak* 1987), however, in a controlled study only 2/6 foals fed high protein had single abberations consistent with osteochondrosis (*Savage* et al.1993a). When these foals' metaphyses and articular/epiphyseal cartilage complexes were examined histologically 3/6 had minor alterations in a metaphyseal physis and 1/6 had a small core of retained cartilage in the caudal humeral head. These lesions were minor and similar to those seen in the control group. When the number of foals with histological lesions of the articular epiphyseal or the metaphysis were compared statistically there were no significant differences between the high protein group and the control group or between the high protein group and the high energy group (although the high energy group had a significantly ($p<0.05$) greater incidence of osteochondrosis compared to the control foals). There was a lack of statistical power in this study and if more foals were used and fed high protein it is likely that they would have a lower incidence of osteochondrosis. (*Savage* 1992; *Savage* et al. 1993a). It is the opinion of the authors that diets high in protein are unlikely to cause osteochondrosis, even though excessive protein can be converted to DE. *Meakim* et al. (1981) did not show that excessive protein caused deleterious effects on growth or calcium metabolism. This contrasts with findings that combined high intakes of protein and DE increased the rate of skeletal growth, but decreased the deposition of mineral into bone (*Thompson, Baker* and *Jackson* 1988), although this is not necessarily due to the increased protein component of the diet (*Savage, McCarthy* and *Jeffcott* 1993b). A possible mechanism by which excess protein could cause DOD is associated with the hypercalciuric effect which occurs when there is excessive intake of sulphur-containing amino acids with subsequent overload of the renal buffering capacity of sulphuric acid. The subsequent acidotic state produced may inhibit renal mineral reabsorption and calcium is lost in the urine (*Glade* et al 1985). *Schryver* et al. (1987) were unable to confirm these results when they fed foals varying levels of protein. They postulated that horses can acclimatize to the excess protein and the associated hypercalciuria is diminished. It is not known whether low calcium levels over long periods of time or relative excesses in phosphorus are important in the induction of osteochondrosis.

Mineral imbalance

Imbalance of mineral homeostasis may have a role in the pathogenesis of osteochondrosis. Numerous aetiologies have been proposed including diets with excess calcium (*Krook* and *Maylin* 1988); excess phosphorus (*Savage, McCarthy* and *Jeffcott* 1993b); and a deficiency in copper (*Hurtig* et al. 1990). In the 1980s a theory existed that foals could be fed excess DE safely if combined with excessive calcium, this was later proven to be unfounded (*Savage* et al. 1993b).

Calcium

Krook and *Maylin* (1988) proposed that diets excessive in calcium (i.e lucerne/alfalfa based or multiple/highly supple-

mented rations, as is commonly found in the US) were responsible for hypercalcitoninism. This was believed to cause disturbances of chondrocytic maturation, disturbance in cartilage's replacement by bone and finally an osteosclerosis, which may have increased the likelihood of pathologic fracture. Thyroid parafollicular cell (C cell) hyperplasia has been documented in dogs fed excess calcium (*Stephens, Norrdin* and *Benjamin* 1985) and also in foetal lambs, whose dams had been fed excess calcium (*Corbellini* et al. 1991). These researchers also showed that there was retarded cartilage differentiation in these foetal lambs, yet studies in weanling foals fed excess calcium (342% of the recommended *NRC* (1989) level for calcium) did not show significant numbers or severity of cartilage lesions (*Savage* et al. 1993b). Two of 6 foals fed high calcium in this study had histological lesions of either articular/epiphyseal cartilage complex or metaphyseal growth plates. One of these had only minor metaphyseal growth plate lesions, which had not been observed at post mortem. The other foal had bilateral lesions of the distal third metatarsal bones, which were found at post mortem.

Knight et al. (1985) reported that young horses from farms with the greatest amount of DOD were on rations with the lowest calcium levels (as well as other nutritional abberations). They recommended that growing horses be fed higher levels of calcium and phosphorus than currently recommended by *NRC* (1989).

Phosphorus

Diets containing excessive phosphorus or a low calcium to phosphorus ratio (i.e 1.3:1 usually considered adequate and reasonable) have been incriminated in causing DOD. Five of 6 weanling foals fed extremely high phosphorus levels (i.e 4.5 X NRC recommendations or 388% of *NRC* (1989) requirement for phosphorus) showed numerous, severe lesions of osteochondrosis, yet no clinical signs of nutritional secondary hyperparathyroidism (*Savage* et al. 1993b). Histomorphometrical studies of these foals revealed a significantly increased cortical bone porosity from wing of ilium biopsies (*Savage* et al. 1993c), which supports the existence of subclinical nutritional secondary hyperparathyroidism. Osteochondrosis lesions were severe, despite a diminished ADG due to the dietary intake being dampened, as the diet appeared unpalatable. This further strengthens the proposal that ADG is not solely responsible for disturbance of endochondral ossification in growth cartilages.

It is possible that diets with excess phosphorus result in acidosis, due to an excess of anions (i.e. PO_4^- and Cl^-) which are normally buffered by cations (i.e. Ca^{2+} and Mg^{2+}). If the buffering capacity of the cations is surpassed, then the strong ion difference is altered and an acidaemia may result. An alkaline pH promotes calcification (*Boulet* and *Marier* 1961) and this is normally present in the hypertrophic zone (*Cuervo, Pita* and *Howell* 1971), due to the presence of carbonic anhydrase (*Ekman* 1990). In broiler chickens fed excess PO_4^- and Cl^-, an increased incidence of tibial dyschondroplasia was seen (*Lilburn* et al. 1989) and this should be further investigated in the foal.

Copper

Copper (Cu) deficiency has been incriminated as a cause of physeal dysplasia and osteochondrosis in young horses (*Knight* et al. 1985). Copper is an integral portion of the enzyme lysyl oxidase and is therefore essential for the cross-linking of collagen, which is required for the development of normal cartilage and bone matrix. In one study mares were either fed 130 or 350 mg Cu daily through the last trimester of gestation and lactation and their unweaned foals were given access to creep feeds with 15 or 55 ppm (i.e mg/kg feed) Cu respectively. No differences in the rate of growth were documented, yet foals born to dams on the lower Cu diet and supplemented with 15 ppm creep feed had an increase (albeit non-significant) in the number of cartilage lesions. The NRC subcommittee did not consider the data conclusive and only increased the 1989 recommendation to 10 ppm.

Diets fed to 48 foals by *Savage* (1992) contained approximately 11.1–11.7 ppm Cu, which was slightly higher than *NRC* (1989) recommendations, but much lower than the 40–50 ppm suggested for growing horses by *Knight* et al. (1987). These diets (11.1–11.7 ppm Cu) were not associated with an increased incidence of osteochondrosis, provided the DE and phosphorus content were at a basal level (approximately 100% NRC). *Bridges* and *Harris* (1988) used two diets to clarify the effects of Cu deficiency. One contained only 1.7 ppm (i.e. low Cu diet) and the other 14 ppm (i.e adequte control Cu diet), which is similar to the *NRC* (1989) recommended level and the amount used by *Savage* et al. (1993; 1993a; 1993b). The salt-soluble collagen in the foals fed 14 ppm Cu was normal, which contrasted with the greatly increased solubility in foals fed diets containing 1.7 ppm Cu. However, foals fed 7 ppm Cu (i.e as the low Cu diet) had a much higher incidence of macroscopic osteochondrosis than those fed 30 ppm. These changes were found predominantly in the cervical vertebrae, yet many sites commonly associated with lesions of osteochondrosis were poorly represented (*Hurtig* et al. 1990). Copper deficiency in other species has been associated with cartilage and bone changes, primarily due to softening of these structures secondary to a decrease in cross-linking. Typical lesions of osteochondrosis have not been seen in cattle with secondary Cu deficiency, although this may be due to an inappropriate window of exposure. However,

Smith et al. (1975) reported that Cu - deficient cattle had thickened distal metacarpal and metatarsal metaphyses, with "tongues" of uncalcified cartilage and poor provisional calcification present.
Horses, as well as being more tolerant to low Cu diets than cattle (*Hintz* and *Schryver* 1987), are much more tolerant of high Cu levels compared to sheep and cattle. *Smith, Jordan* and *Nelson* (1975) reported that ponies could tolerate up to 791 ppm dietary Cu, although these ponies had decreased ADG and high liver Cu concentrations. Hence, it appears that growing horses could tolerate Cu levels suggested by *Knight* et al. (1985) and *Hurtig* et al. (1990), and this may alleviate some of the signs seen in growing foals due to osteochondrosis.

Zinc and cadmium

Gunson et al. (1982) found generalized cartilage lesions that appeared similar to osteochondrosis in foals accomodated in the vicinity of a smelter. The surrounding environment and pasture had high levels of zinc and cadmium. *Kowalczyk* et al. (1986) also studied the effects of environmental exposure to zinc and cadmium in 5 pregnant pony mares and their foals. Only 2 of the foals had signs of lameness, although all foals had macroscopic lesions resembling osteochondrosis, when they were killed at ages of 2.5 to 18.5 months. Histological examination of cartilage from growing horses in smelter regions does not support a primary pathology in endochondral ossification. *Knight* et al. (1985) postulated that diets with excessive zinc or molybdenum could cause a secondary Cu deficiency, as seen in ruminants. In support of this, *Cymbaluk* et al. (1981) reported an increase in ^{64}Cu in faeces and bile of ponies fed high molybdenum, however, *Strickland* et al. (1987) was unable to show any evidence of persistent, protein-bound thiomolybdates, which are characteristically seen in ruminants. It appears unlikely that molybdenum interferes with Cu metabolism in the horse. It is still possible that diets high in DE may interfere with Cu absorption in the horse.

Conclusions

Osteochondrosis (dyschondroplasia) has a multifactorial aetiology (*Jeffcott* 1991), but nutrition has an important role to play in pathogenesis. This is particularly true in relation to excess DE which appears to be able to induce the condition on its own. However, there are important interactions and interplay with other factors. For example, the intensity and duration of exercise even in the face of excess DE may have a protective effect on the induction of osteochondrosis. The endocrinological response to different dietary regimes appears to be relevant as does the effect of insulin and growth factors on chondrocyte survival. There is still much research to be done, but a clearer picture of the processes involved in chondrocyte metabolism and endochondral ossification will make elucidation of specific pathogenesis much easier.

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