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NUTRITION AND OTHER TOPICS OF INTEREST TO THE HORSE INDUSTRY.

Special Edition

Do Elevated Blood Insulin Concentrations Increase The Risk of OCD?

Recently discussed by equine nutritionists is a theory that high post-feeding blood insulin concentration somehow disrupts the normal bone/cartilage development in the joints of young growing horses, and thereby causes osteochondritis dissecans (OCD). It is further implied that feeds with a lower glycemic response would consistently produce a lower blood insulin concentration. Not surprisingly, this theory has sparked considerable interest. We are pleased to be able to print a review of the scientific evidence by Nerida Richards, Ph.D., an expert in this field.

Dr Richards is the principal nutritionist in her own independent equine nutrition consultancy based in Australia. Dr Richards provides consultation services and decision support systems in the field of equine nutrition to feed manufacturers and horse owners/breeders. Her particular area of expertise is in the digestion and fermentation of cereal grains in the equine gastrointestinal tract. As part of her recent research, Dr Richards has measured and analyzed numerous post-feeding glycemic and insulin responses in horses.

Elevated blood insulin concentrations: Do they increase the risk of Osteochondritis Dissecans (OCD)?

Nerida Richards, Ph.D.

Introduction

While there are many factors that may contribute to the development of osteochondritis dissecans (OCD) in young horses, nutrition plays a major role. Mineral deficiencies, excesses or imbalances; protein deficiencies and excessive dietary energy have all been implicated as causative factors in equine OCD. A recent theory proposed is that elevated post-feeding blood insulin concentrations, which may occur following the consumption of a diet rich in starch and other non-structural carbohydrates such as those found in corn, oats and other cereal grains, are disrupting the normal bone formation process in young horses, resulting in OCD. The following article examines the possible mechanisms of action for insulin to disrupt the bone formation process and reviews the scientific evidence in support and also in contrast to this theory.

What is OCD?

OCD is a disease that occurs during skeletal development. OCD lesions may include subchondral fracture, subchondral cysts, 'wear lines', chondromalacia and osteochondritis, synovitis and cartilage flaps, which may in turn lead to the production of bony fragments within the joints (Jeffcott 1997). OCD may develop at various sites within the skeleton including the fetlock, shoulder, hock and stifle regions and begins as a primary lesion within growth cartilage known as 'dyschondroplasia'. Dyschondroplasia occurs during a failure in the process of endochondral ossification (the process of cartilage formation, degradation and eventual replacement by bone), which results in a small 'retained' core of cartilage within a joint (Jeffcott et al. 1998). These primary lesions of retained cartilage are then susceptible to further damage within the joint, particularly during exercise and training, and can lead to OCD (Jeffcott et al. 1998).

How can insulin cause OCD?

In an *in vitro* (meaning *in glass* or a study conducted in the laboratory) study, Henson and others (1997) demonstrated that insulin has a 'survival' effect on equine cartilage cells. By promoting the survival of cartilage cells, insulin may inhibit the maturation of equine cartilage, thereby slowing the process of bone formation and potentially resulting in the formation of retained cartilage cores as described above.

It is also proposed that elevated post-feeding concentrations of insulin may lower the concentrations of circulating thyroxine (T_4) hormone, causing a temporary status of hypothyroidism (Gavin and Moeller 1983). Thyroxine is crucial for the process of cartilage maturation and bone formation, and thus temporary periods of hypothyroidism may contribute to the development of OCD in horses (Glade et al. 1984; McLaughlin et al. 1986).

What scientific evidence do we have to support this theory?

While genetic predisposition and a horse's environment play a role in determining its susceptibility to OCD, the incidence of OCD in feral herds of horses is far lower (approximately 6%) than that observed in some domestic herds, which may have up to 25% of a population affected (Alvarado et al. 1989; Valentino et al. 1999). These observations suggest that factors in modern day management systems are contributing to the abnormally high incidence of OCD in domestic herds. One such management factor that has constantly been implicated as a causative agent of equine OCD is the feeding of high-energy diets that often contain significant quantities of cereal grains.

Savage and others (1993) reported that 100% of foals fed a diet that provided 129% of the NRC dietary energy recommendations developed dyschondroplasia lesions, while only 17% of foals on a diet which supplied 100% of the NRC dietary energy recommendations developed lesions. Similarly, Glade and Belling (1984) found that cartilage taken from horses fed a high-energy diet, supplying 130% of the NRC dietary energy recommendations, was thickened in comparison to horses fed 100% of NRC dietary energy recommendations. Cartilage from the 'overfed' horses also showed characteristics typically displayed in OCD affected joints. Namely, the cartilage remained unpenetrated by blood capillaries and no longer displayed the 'normal' columnar organisation of cartilage cells. A majority of modern day high-energy diets are based on cereal grains that may contain between 30 and 75% starch. Diets containing starch are known to elevate both blood glucose and insulin concentrations following consumption (Glade et al. 1984; Pagan 1999; Richards 2003). Thus, it has been theorised that high-energy diets are increasing the incidence of OCD in domestic horse populations via an insulin mediated mechanism.

Ralston (1996) observed that compared to 11 'normal' horses with no OCD lesions, four young horses with OCD lesions had significantly higher plasma insulin concentrations following the consumption of a textured sweet feed. This observation led her to conclude that hyperinsulinemia may be related to the development of OCD in young horses.

Pagan (2003) observed during a field study involving six Thoroughbred breeding farms in central Kentucky, that plasma insulin concentrations in foals with clinical OCD were significantly higher 2 hours post-feeding than those observed in 'unaffected' foals. The insulin response to a farm's feed was also positively related to the incidence of OCD on that farm (i.e. as insulin response increased, the number of OCD affected foals increased). Thus, the studies of Ralston and Pagan lend some support to the theory that elevated blood insulin concentrations are disrupting the normal bone formation process in the young horse and contributing to OCD.

There is scientific evidence contrary to this theory

It has been well accepted that high-energy diets increase the risk of OCD in horses. It is further proposed that these high-energy diets do this via an insulin mediated mechanism. However, Glade and others (1984) were able to demonstrate that insulin responses to a low energy diet (supplying 80% of the NRCs energy recommendations) and a high energy diet (supplying 160% of the NRCs energy recommendations) were virtually identical. The insulin response to the low energy diet actually peaked slightly higher and remained elevated longer than the insulin response observed in horses on the high energy diet. In a separate experiment, Glade and others (1984) showed that insulin responses to diets containing 1.22, 1.62 or 2.11 grams of starch per kilogram of body weight (equivalent to approximately 436, 579 and 754 grams of corn for a 250 kg weanling) initiated virtually identical post-feeding insulin responses in weanlings. The results demonstrate again that high energy diets do not necessarily have more exaggerated insulin responses than low energy diets (Glade 1987). These studies suggest therefore, that the increased incidence of OCD in horses consuming high-energy diets may not be a direct effect of elevated post-feeding insulin concentrations.

Interestingly, in both of the studies reported above, Glade and others observed that post-feeding thyroxine (T_4) hormone concentrations decreased sharply following the feeding of the higher energy diets. Given that thyroxine is essential for normal bone maturation and that bone lesions caused by hypothyroidism are very similar to those seen in OCD, it is possible that short periods of post-feeding hypothyroidism, independent of insulin response, are contributing to the increased incidence of OCD in horses on high-energy diets.

The studies of Ralston and Pagan also leave many questions unanswered with respect to insulin's role in the development of OCD. For studies aiming to establish a new causative factor of OCD, it is essential that other possible causative factors are first eliminated.

Both studies neglected to detail the energy intakes and mineral composition of the diets consumed by the horses. Since both excessive energy consumption and sub-optimal mineral intakes are well-established causes of equine OCD, it is possible that either or both of these factors played a role in these studies, leaving us unable to draw any sound conclusions with respect to insulin's role in the development of the disease.

A multitude of factors affect a horse's insulin response following the consumption of a meal. One such factor is a horse's body condition score or body fatness. A horse with a higher body condition score will tend to display an exaggerated post-feeding insulin response, possibly due to an insulin insensitivity or insulin resistance that develops as the horse gets fat (Jeffcott et al. 1986).

In the study of Pagan (2003) the horses on the farm with the highest incidence of OCD were 15% heavier than the average of 350 central Kentucky weanlings, whereas weanlings on the farm with a 0% incidence of OCD were 3% lighter than the average of 350 central Kentucky weanlings. So a confounding issue arises in that the horses with OCD may have had higher body condition scores than the 'normal' horses. This would implicate excessive energy intake, rapid growth rates and trauma as the causative factors of OCD. It also suggests that the elevated insulin responses observed in the horses with OCD may be merely a symptom of their excessive body weight and fatness and not a cause of OCD as it has been suggested.

A detailed description of the horses involved in the study of Ralston (1996) was not provided. The study, therefore, was left open to speculation about whether the elevated insulin responses observed in this study were also a symptom of excess energy intake and body condition.

A further inconsistency in the study of Pagan *et al*. (2003) was that, within a farm, it was reported that there was no difference in post-feeding blood glucose concentrations between weanlings with and without OCD lesions. The recommendation at the conclusion of the study, however, was to "feed foals concentrates that produce low glycemic responses". Given that a weanling's post-feeding glycaemic response did not appear to have a role in determining its susceptibility to OCD within a farm, the recommendation to feed low glycaemic response feeds has very limited scientific support. In addition, this recommendation is making the assumption that a weanling's glycaemic and insulin responses are well related. A predictable relationship between glycaemic and insulin response was not reported during this study. Unless a relationship is established, we are unable to assume that a low glycaemic response feed will consistently produce a low post-feeding insulin response in weanlings. Likewise we cannot assume that a feed which produces an elevated post-feeding glycaemic response will also initiate an elevated post-feeding insulin response.

The study of Savage and others (1993) raises further questions with respect to the relationship between elevated post-feeding insulin responses and equine OCD. This study, as previously reported, found a 100% incidence of dyschondroplasia in weanlings fed a high-energy diet. The high-energy diet used consisted of a rice-based pellet and 0.25kg of corn oil. Recent research in horses has demonstrated that when oil is added to a diet based on cereal grain, the observed post-feeding plasma glucose and insulin responses are significantly reduced (Pagan 2001; Richards 2003). It would therefore be reasonable to assume that the post-feeding insulin responses for horses on the high-energy diet in this feeding trial were low, perhaps even lower than those observed in horses consuming the low-energy diet. Thus it is unlikely that the 100% incidence of dyschondroplasia in the horses on this high-energy diet was attributable to high post-feeding blood insulin concentrations.

An unpublished study examining the effect of exercise on the incidence of OCD in Dutch Warmblood foals raises further questions as to the relationship between post-feeding insulin response and OCD. During the study, it was observed that foals on a high-energy diet, undergoing an exercise regime, had a much lower incidence of OCD (5%) than foals consuming an identical high-energy diet on a restricted exercise regime (18%) (Jeffcott 1991). As these two groups of foals were consuming the same diet, it would be reasonable to assume that their post-feeding insulin responses were similar; and yet, the incidence of OCD between the two groups differed markedly. This study suggests that exercise is having a protective effect, perhaps by burning some of the energy that may otherwise have been used for growth, thereby implicating energy intake and growth rate as the causative factors of OCD and not post-feeding insulin response.

Conclusions

Equine OCD is a complex disease with many factors including genetics, trauma and nutrition playing integral roles in its development in young growing horses. Nutrition plays a major role in the incidence of OCD, with excess dietary energy intakes and mineral deficiencies being well documented as causes of OCD in horses.

A recent theory being proposed (Ralston 1996; Pagan 2003) suggests that exaggerated post-feeding insulin responses are capable of contributing to or causing OCD. The scientific evidence presented to support this theory to date is incomplete and appears to be almost circumstantial. The studies of Ralston (1996) and Pagan (2003) have reported that horses with OCD have a tendency to display exaggerated post-feeding insulin responses. However, neither of these studies provided detailed information on the energy intakes of the horses involved, the mineral composition of the diets fed, nor the body condition scores of the horses used. Thus it remains likely that the observed incidence of OCD in these studies was due to excessive energy intakes and or mineral deficiencies and that the exaggerated insulin responses observed were a symptom of their energy intake and body fatness.

Nevertheless, there is currently a push toward the use of high-fibre, high-fat diets that cause a characteristically low post-feeding glycaemic/insulin response in horses. However, until a relationship between post-feeding blood glucose and insulin concentrations is determined and a solid and scientifically proven link between post-feeding insulin responses and equine OCD can be established, a total restructure of the methods used to feed young, growing horses cannot be justified. These high-fibre, high-fat feeds can provide just as much, if not more, dietary energy than a traditional cereal grain diet and from this respect, are equally as capable of causing equine OCD.

To date, sound management practices that reduce the risk and occurrence of bone trauma and ensure optimum energy and mineral intakes by young growing horses, appear to be the most scientifically proven and effective methods of minimising the on-farm incidence of OCD.

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