

Advances in Equine Nutrition Volume II

J.D. Pagan



GLUCOSE INTOLERANCE AND DEVELOPMENTAL ORTHOPEDIC DISEASE IN FOALS-A CONNECTION?

SARAH L. RALSTON

Cook College, Rutgers University, New Brunswick, NJ

Large foals that are growing rapidly are considered to be at increased risk of developmental orthopedic disease (DOD). A multifactorial problem, DOD includes problems such as osteochondritis dissecans (OCD), epiphysitis, flexural and angular limb deformities and perhaps wobbler syndrome (McIllwraith et al. 1991; Jeffcott, 1991; Jeffcott, 1996). Of these, OCD is probably the biggest problem in the equine industry in terms of lost revenue (Jeffcott, 1996). Genetics, nutrition and exercise all play a role in the incidence of DOD in horses. Recently a connection between high insulin concentrations, especially after feeding a meal of concentrates, and OCD has been reported (Ralston, 1996; Ralston et al., 1998). In this paper I will explore both the old and new theories regarding the causes of DOD and OCD in foals and provide recommendations, based on the most recent information, for feeding young horses.

General Factors

Hereditary predisposition to at least OCD is well documented in Standardbred and Swedish warmblood horses, with the incidence as high as 45% in some bloodlines (Grondahl, 1991; Schougaard et al., 1990). However the genetic defect that causes the growth associated problems in the horses has not been identified. Breeds selected for rapid growth maturation are at increased risk of developing problems, but it is not growth rate alone that causes the problem (Jeffcott, 1991). It is not always the most rapidly growing foal that develops DOD, but often the one with the most erratic growth rate (Jeffcott, 1991; Lewis, 1996).

Trauma due to excessive concussion, due either to obesity or forced exercise, may increase the incidence of DOD (Jeffcott, 1991; Lewis, 1996). Other reports, however, revealed that restriction of exercise adversely impacted bone growth and development in young horses. Turning the foals out in as large an area (either pasture or paddock) as possible for as long as possible is highly recommended. Ideally they should get 24 hour turnout. However, strenuous forced exercise, especially longeing in circles, should be avoided. Foals should not be allowed to become obese.

Nutritional Factors

MINERALS

Mineral imbalances have been well documented to cause DOD (Jeffcott, 1991; Lewis, 1996). Deficiencies of calcium, phosphorus and/or copper all result in defective bone maturation. Zinc toxicity and perhaps deficiency have also



397

398 Glucose Intolerance and Orthopedic Disease

resulted in lesions, though the effects of simple zinc deficiency are not well documented. The optimal intakes of copper and zinc for young horses have not been well defined. My current recommendations for acceptable ranges of mineral content of rations for foals less than 1 year of age are given in Table 1.

 Table 1. Recommended concentrations of minerals* in rations fed to rapidly growing young horses.

Mineral	Range
Calcium	0.8 to 1.0%
Phosphorus	0.4 to 0.6%
Copper	10 to 15 mg/kg feed
Zinc	40 to 60 mg/kg feed

* Other minerals such as manganese, magnesium, selenium and iron

are probably important, but there are no data available on requirements

of young horses for these nutrients. There are no data on vitamin

requirements either, though over 10 times the recommended level of vitamin A

resulted in weakened bones in ponies (Donoghue et al., 1981).

PROTEIN

Excessive protein (greater than 16%) was incriminated as a cause of DOD in the 1970s but subsequent studies have not revealed a direct relationship between high protein rations and DOD (Lewis, 1996). Weanlings fed rations deficient in protein (less than 12%) had reduced growth rates and poor bone mineralization compared to weanlings fed rations that were higher in protein. Restricting protein in a rapidly growing foal's ration will not result in improved bone growth and may actually be detrimental to the animal.

CARBOHYDRATES

Rations providing over 100% of the National Research Council's recommended amounts of energy for rapid growth in foals may cause an increased incidence of DOD, especially if the ration contains more than 50% sweet feed (grain mix plus molasses) or other high sugar concentrate by weight (Lewis, 1996; Glade, 1986; Glade and Belling, 1986). High carbohydrate rations such as sweet feeds may contribute to the appearance of DOD, possibly related to the high blood glucose and insulin and low blood pH they cause for up to 4 hours after feeding (Glade, 1986; Glade and Belling, 1986). It is interesting that foals between 3 and 12 months of age have been documented to be relatively insulin resistant (Ralston, 1996; Krusic et al., 1997), since this is the period during which OCD lesions most commonly develop (McIllwraith et al., 1991; Jeffcott, 1996; Jeffcott, 1991). Pelleting and extrusion of concentrates appear to affect the availability of carbohydrates (Ralston, 1992; Ralston 1995-1999). Pelleted or extruded concentrates may cause lower glucose and insulin changes than textured feeds with the same basic formulation (Ralston, 1992; Ralston, 1995-1999). There may be a correlation between OCD and glucose intolerance (abnormally high blood



glucose and insulin after a meal of concentrates) in foals that are genetically predisposed to the problem (Ralston, 1996; Ralston et al., 1998). At Rutgers we are currently developing a patented glucose challenge that can be easily administered to foals < 3 mo old in order to identify those potentially at risk. These foals could then be fed specially formulated rations to reduce the risk of lesions appearing, since diet composition can dramatically affect glucose/insulin responses (Garcia and Beech 1986; Lawrence et al., 1993; Ralston et al., 1979).

Recommendations

- Nursing foals should be introduced to concentrates when they are 1 to 1. 2 months of age. The concentrate should contain 14 to 18% protein and have added calcium, phosphorus, copper and zinc (see Table 1) in a formulation designed specifically for growing horses. The higher percentages of protein and minerals should be used if only grass hay is available. The lower percentages can be used with legume or legume/ grass mix hays. Concentrates should be fed at the rate of 0.50% to 1.0% of body weight, with the emphasis on maintaining lean body condition (ribs not visible but can be felt with mild pressure over the flank; loin, croup and neck have smooth outlines without creases or visible bony structures). If the lower amounts are fed (less than 0.5%) body weight), addition of a balanced calcium/phosphorus/trace mineral mix may be necessary to maintain the proper mineral intake. Ideally the foals should be fed regulated amounts that are inaccessible to their dams or other foals twice a day. The mares should be fed the same concentrate if the foal has access to the mare's feed.
- 2. Don't let the foal get obese (obvious crease down the back, ribs cannot be easily felt) or excessively thin (ribs easily visible, hip bones prominent, hair coat dull and shaggy). If group feeding foals and mares, monitor their condition daily and feed any excessively thin or fat foals separately. Since pelleted and extruded feeds may cause lower glucose and insulin responses than do sweet feeds, the former two types of concentrate may be preferable to textured sweet feed mixes, especially in foals from bloodlines potentially predisposed to OCD.
- 3. Weanlings should be fed the same type of concentrate as when they were nursing and at the same rate as above and monitored carefully for signs of excessive weight gain or loss and DOD. From 0.25 to 1.0% body weight of a properly formulated concentrate divided into two or three meals a day, with free choice access to good quality mixed leume/grass hay or pasture, will maintain optimal growth rates of most light horse breeds while reducing the risk of DOD. The goal is to maintain steady growth, avoiding sudden increases or decreases, and to maintain good, but not fat or thin, body condition. Plain white or trace mineral salt and a good, clean source of water should be available free choice at all times.



400 Glucose Intolerance and Orthopedic Disease

- 4. If signs of epiphysitis (enlargement at the growth plates above the fetlocks and/or knees associated with lameness and reluctance to exercise) or other deformities (contracted tendons, angular deformities) appear, the ration is probably not properly balanced. The amount of concentrate fed should be TEMPORARILY reduced while the total ration's nutrient content is assessed. Any deficits or excesses should be corrected and a properly balanced ration reintroduced as soon as possible. Starving foals (feeding only grass hay and oats for a prolonged period of time, resulting in weight loss, poor growth and roughlooking hair coats) will not correct the problem on a long-term basis.
- 5. Yearling rations can be reduced to 12%-14% protein with lesser concentrations of minerals, but still above that usually found in mixes formulated for adult horses. Maintaining yearlings on the weanling rations will not hurt and may help, especially if horses are still growing fairly rapidly.

References

- Donoghue S, Kronfeld DS, Berkowitz SJ, Copp RL. 1981. Vitamin A nutrition of the equine: Growth, serum biochemistry, and hematology. J. Nutr. 111:365-372.
- Garcia MC, Beech J. 1986. Equine intravenous glucose tolerance test: Glucose and insulin responses of healthy horses fed grain or hay and of horses with pituitary adenomas. Am J Vet Res 47:570-572.
- Glade MJ. 1986. The control of cartilage growth in osteochondrosis: a review. J Eq Vet Sci 6:175-187.
- Glade MJ and Belling TH. 1986. A dietary etiology for osteochondrotic cartilage. J Eq Vet Sci 6:151-155.
- Grondahl AM. 1991. The incidence of osteochondrosis in the tibiotarsal joint of Norwegian Standardbred trotters. J Eq Vet Sci. 11:273-274.
- Jeffcott L.B. 1991. Osteochondrosis in the horse searching for the key to pathogenesis. Equine Veterinary Journal 23: 331-338.
- Jeffcott LB. 1996. Osteochondrosis-An international problem for the horse industry. J Eq Vet Sci 16:32-37.
- Krusic L, Krusic-Kaplja A, Cestnik V, Snoj T, Pogacnik A, Pangos S, Gatta D, Moni P. 1997. Insulin response after oral glucose application in growing Lipizzaner foals. Proc 15th Eq Nutr Physiol Symp. (Fort Worth, TX):397-403.
- Lawrence LM, Soderholm LV, Roberts A, Williams J, Hintz H. 1993. Feeding status affects glucose metabolism in exercising horses. J Nutr 123:2152-2157.
- Lewis LD. 1996. Feeding and Care of the Horse, 2nd edition. Williams and Wilkins, Philadelphia.
- McIlwraith CW, Foerner JJ, Davis DM. 1991. Osteochondritis dissecans of the tarsocrural joint: results of treatment with arthroscopic surgery. Eq Vet J 23:155-162.
- Ralston SL. Effect of soluble carbohydrate content of pelleted diets on postprandial glucose and insulin profiles in horses. Pferdeheilkunde(September, 1992):112-115, 1992.
- Ralston SL. Hyperglycemia/hyperinsulinemia after feeding a meal of grain to young



horses with osteochondritis dissecans (OCD) lesions. Pferdeheilkunde (May, 1996):320-322, 1996.

- Ralston SL. 1995-1999. Unpublished data. Ralston SL, Van den Broek G, Baile CA. 1979. Feed intake patterns and associated blood glucose, free fatty acid and insulin changes in ponies. J. Anim. Sci. 57: 815-821.
- Ralston SL, Black A, Suslak-Brown L, Schoknecht PA. Postprandial insulin resistance associated with osteochondrosis in weanling fillies. J Animal Science 76 (supplement 1):176, 1998.
- Schougaard H, Falk-Ronne J, Philipsson J. 1990. A radiographic survey of tibiotarsal osteochondrosis in a selected population of trotting horses in Denmark and its possible genetic significance. Equine Vet J. 22:288-289.



402 Glucose Intolerance and Orthopedic Disease

