Energy and Vitamin Nutrition of the Equine Athlete

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Nutrition of the performance horse has been the subject of several recent reviews (Meyer, 1987; Frape, 1988; Glade, 1988; Jackson, 1988; Pagan, 1988a). When reviewing the literature several points become obvious 1) There is much to be learned about nutrition of the equine athlete 2) Energy nutrition is usually considered to be of greatest concern and 3) There is great interest in vitamin nutrition.

Energy

There is considerable interest in the determination of the best dietary sources of energy. Should the dietary sources of energy be changed depending on the type of work? What is the effect of dietary energy sources on the relative amounts of cellular fuels and storage of energy.

Soluble carbohydrates such as starch from grain are readily digested and are excellent energy sources. In addition to supplying glucose, it has also been suggested that high carbohydrate intake may increase glycogen storage in horses.

Glycogen loading in humans has been associated with improved performance in athletes competing in endurance or multiple-event competitions. In the past, glycogen loading in humans was often accomplished by first depleting the muscle glycogen while eating high protein, high fat diets and then doing heavy work. When the athlete rested and ate only carbohydrates, increased glycogen storage took place. Adverse side effects such as digestive upsets can occur with such an approach. The American Dietetic Association now recommends that athletes avoid the high fat, high protein phase but rather eat a high carbohydrate diet throughout training and then begin a tapered rest approximately 7 days prior to the event and have complete rest the day before the event.

An increase in muscle glycogen can be accomplished in horses by dietary means, however the increase is not as dramatic as in humans. Pagan et al. (1987) concluded "Muscle glycogen loading appears to be most successful when high carbohydrate diets are fed to horses following intensive exercise bouts which deplete large amounts of glycogen from the muscle. The usefulness of this practice, however is questionable." Snow and Vogel (1987) concluded that muscle glycogen is not limiting in horse races of 1 to 2 miles. Furthermore, it is often stated that glycogen loading procedures in horses could have harmful side effects, such as predisposition to founder, endotoxemia, exertional myopathy, (Frape, 1988; Pagan, 1988b). The classical azoturia or Monday morning disease in draft horses was considered to be the result of resting horses on Sunday but still giving them the same amount of grain they would have received had they been working. Such a procedure may have increased glycogen stores.

On the other hand, others feel that increased glycogen stores could be beneficial to racehorses. Potter as quoted by Sellnow (1987) suggested

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that horses with glycogen stores of less than 10 mg/g of wet muscle may suffer from onset of fatigue sooner than horses with higher levels of glycogen, even those performing sprint type exercise; Potter also opined that many Thoroughbreds are glycogen deficient at the start of the race events. Potter also suggested that horses with greater stores (20 to 30 mg/g wet tissue) were able to mobilize more glycogen and therefore more effectively support anaerobic metabolism.

The amount of fibrous carbohydrate must also be considered. A diet high in fiber could limit energy intake because of the bulk. The lower digestibility of fiber would increase gut fill. The effect of gut fill on performance has not been studied but it seems logical that extra weight could have an influence.

Meyer (1987) suggested that racehorses should be fed 4-6 kg of hay daily but that endurance horses should be fed larger amounts of roughage (6-8 kg daily) because the fibrous ration would dilate the intestine and increase the intestinal water and electrolyte reserve.

The feeding of fat to horses has received a great amount of attention recently (Meyers, et al., 1987; Scott et al., 1987; Worth et al., 1987). Many fat supplements and feeds containing as much as 10-15% fat have appeared on the market. There is no doubt that the addition of fat increases the digestible energy content of a diet. Fat is highly digestible and contains about 2.25 times as much gross energy per gram as does starch. One benefit of feeding fat may be simply providing more calories. There may also be extra-caloric effects. Earlier studies indicated that diets containing 8% fat fed to horses doing endurance work do not promote glycogen storage but may aleviate the decline in blood glucose (Slade, et al., 1975; Hintz et al., 1978; Hambleton et al., 1980). One theory for the aleviation of glucose decline is that feeding of fat during training-stimulates the enzymes needed to metabolize fat. When the animal draws on body fat reserves during an endurance event, the fat can be utilized more effectively because the mechanisms have been more extensively developed.

Dietary fat also might be used to increase the caloric intake with less danger of laminitis than if soluble carbohydrate was used.

Some authors have reported that dietary fat may improve glycogen storage or spare glycogen in horses doing intense work and therefore improve performance under certain conditions (Duren et al., 1987; Webb et al., 1987). Pagan (1988b), however, in a review on feeding fat to horses concluded that it is highly unlikely that feeding fat will increase muscle glycogen stores in horses. He concluded that the diet of racehorses should provide an adequate energy supply and a source of energy which will not cause excessive glycogen storage and will not result in high levels of blood glucose and hence increased insulin release. He stated that the greatest benefit of feeding fat may be in reaching the latter two goals. Feeding diets rich in carbohydrates in several small meals would also decrease insulin secretion peaks during post-feeding.

If fat is used, it appears that it should be added at a rate of at least 0.2 to 0.4 kg per day in order to obtain a reasonable increase in digestible energy content of the diet. Smaller amounts however, particularly if unsaturated, may improve coat condition.
One of the most important aspects of feeding may be to keep athletes in positive energy balance. It is recommended that horses be weighed routinely and maintained within 10 kg of their optimal weight.

**Vitamins**

The vitamins receiving the most attention recently in performance horses are perhaps those with antioxidant activities - vitamin E and vitamin C.

For many years it was often assumed by horse nutritionists that if the diet contained good quality forage and adequate levels of selenium there was little likelihood of Vitamin E deficiency. The NRC (1978) estimate of the Vitamin E requirement was only 15 mg of Vitamin E/kg of diet. Hay might be expected to contain 50 to 90 mg/kg. Grains might contain 20 to 30 mg/kg.

Several studies, however, indicate that the NRC 1978 estimate was too low. Liu et al., (1983) reported degenerative myelopathy (which perhaps could be called Equine Degenerative Myeloencephalopathy (EDM) in six Przewalskii horses in the Bronx Zoo. Plasma alpha-tocopherol levels were measured in five of the horses and were found to be 0.4 ± 0.1 µg/ml.

Mayhew et al. (1987) suggested that EDM is a Vitamin E-responsive disorder with a possible familial predisposition. Mayhew and co-workers studied horses on two farms with a high incidence of EDM. Thirteen ataxic foals had an average serum Vitamin E concentration of 0.62 ± 0.13 µg/ml. A level of 1.5 µg/ml or less was considered to be deficient. Supplements of 1250-2500 mg of Vitamin E per horse per day increased the blood levels. On one farm the incidence of EDM was reduced from 40% to less than 10% the year following Vitamin E supplementation. A genetic-nutrient interaction was indicated because a high incidence of the disease was evident in offspring of two particular sires. The low Vitamin E intake was attributed to the lack of fresh green roughage and the feeding of poor quality hay.

Roneus et al., (1986) fed a poor quality hay containing 11 to 19 mg of Vitamin E per kg and oats that were heated in an oven to reduce the Vitamin E content from 21 to 33 mg of Vitamin E per kg to 9 to 10 mg to 12 horses for 2½ months. The horses were then divided into four groups receiving various supplemental levels of Vitamin E. The results led Roneus to suggest that in order to ensure nutritional adequacy, horses at maintenance fed diets containing low levels of Vitamin E should be given supplements providing 600 to 1800 mg of DL-alpha tocopherol acetate which corresponds to 1.5 to 4.4 mg/kg of bodyweight. Nutritional adequacy was defined according to plasma and tissue Vitamin E levels. The NRC level of 15 mg/kg of feed would provide only .22 mg/kg of body weight for horses fed at maintenance. A concentration of 100 mg/kg of feed could provide about 1.5 mg/kg of body weight at maintenance.

Baalsrud and Overnes (1986) fed horses a diet of hay and oats that provided 18 or 80 mg of alpha-tocopherol. The humoral immune response to novel antigens such as tetanus toxoid and equine influenza virus was increased in groups receiving either Vitamin E.

The effect of vitamin E on the athlete has received considerable attention. There is no doubt that a deficiency can cause severe problems. Jackson et al., (1983) reported that skeletal muscles from vitamin E-deficient rats and mice are more susceptible than normal muscles to contractile damage. The exercise endurance of vitamin E-deficient rats was found to be 40 percent.
lower than that of controls (Quintanilha and Packer, 1983). But does supple-
mentation above the requirement (megadoses) have a beneficial effect? Some
trainers apparently think so. Snow and Frigg (1987a) reported that one of
the leading trainers in England was providing 11.5 g of alpha tocopherol
daily to his horses.

Schubert (1987) reported that horses given an additional 1000 mg of
vitamin E per day placed in 48.8% of their starts compared to 29.9% for horses
not given additional vitamin E.

Although there is much to be learned about the vitamin E nutrition of
the horses NRC (1989) increased the estimates of the requirements of Vitamin
E. It was stated that "Until this issue is clarified, it may be prudent
to ensure that diets contain 80 to 100 IU of vitamin E/kg dietary dry matter
in the total diet for foals pregnant and lactating mares and working horses."

The reduced form of Vitamin C, ascorbic acid, is required for the hydroxyl-
ation of proline and lysine to form hydroxyproline and hydroxyllysine. Hydroxy-
proline is a major constituent of collagen. Many of the signs of scurvy
(the classical deficiency of Vitamin C) such as failure of wound healing,
brittle bones and internal bleeding can be attributed to lack of normal collagen.

Vitamin C can also function as an antioxidant and, therefore, protects
lipids, proteins and membranes from radical induced oxidative damage. It
also interacts with Vitamin E. Vitamin C scavenges oxygen radicals in the
aqueous phase, whereas Vitamin E scavenges oxygen radicals within the membranes.
Vitamin C may also regenerate Vitamin E by reducing Vitamin E radicals formed
when Vitamin E scavenges the oxygen radicals (Niki et al., 1987).

Vitamin C is a dietary requirement for human beings, subhuman primates,
guinea pigs, bats, shrimp, some species of birds such as the red-vented bulbul
and blackheaded oriole and some species of fish such as the minnow, catfish,
trout and salmon. These species lack L-gulonolactone oxidase, the last in
a series of four enzymes needed for the biosynthesis of Vitamin C from glucose.

Cattle, dogs, cats, swine, sheep, chickens, ducks, turkeys and horses
have L-gulonolactone oxidase, and Vitamin C is not considered to be a dietary
requirement for these species. For example, Stillions et al (1971) fed geldings
a semi-purified diet without ascorbic acid for 6 months with no adverse effects
on health or plasma ascorbic values noted.

However, some scientists have suggested that even in those species which
have L-gulonolactone oxidase dietary sources of Vitamin C may be needed during
periods of stress. For example, Pardue and Thaxton (1986) concluded that
poultry are capable of ascorbic acid synthesis and dietary supplementation
is not necessary when the animals are managed properly. They suggested,
however, that stressful conditions such as adverse temperatures or the presence
of certain pathogens might increase the requirement for ascorbic acid beyond
the capacity of the body for synthesis.

Ralston reported that geriatric horses (20 to 27 years old) had lower
levels of plasma Vitamin C than did young animals (6 to 7 years old) under
the same dietary and environmental conditions. It was suggested that the
lower values were due to chronic stress or decreased synthesis in the older
animals. Loscher et al. (1984) reported that decreased ascorbic acid serum
levels in horses may be associated with wound infections, epistaxis, strangles, rhinopneumonia and decreased performance. They suggested that IV injection of ascorbic acid was the only satisfactory route of administration if supplementation is required because oral administration did not increase the serum levels. However, both subcutaneous and intramuscular administration resulted in marked local irritation.

Snow and Frigg (1987a) reported that several leading trainers of Thoroughbreds in England give high doses (as much as 20 g per day) orally to their horses. They found that a single dose of 20 g of ascorbic acid did not result in any increase in plasma concentrations, but daily oral doses of either 4.5 or 20 g caused significant increases in plasma concentrations.

Snow and Frigg (1987b) reported that the form in which the vitamin is administered could influence the response. They found that weekly oral doses of ascorbyl palmitate resulted in higher plasma ascorbic acid concentrations than did oral doses of crystalline ascorbic acid. The compounds were given on an isomolecular basis (20 g for ascorbic acid and 47 g for ascorbyl palmitate).

Snow and Gash (1987) pointed out that neither the need -nor nor an appropriate dose of ascorbic acid for supplementation in the horse is yet to be ascertained. They concluded that no deleterious effects can be attributed to a dose rate of 20 g of ascorbic acid per day over several years of administration.

REFERENCES


