Nutrition and Equine Performance

HAROLD F. HINTZ
Cornell University, Ithaca, NY 14853-4801

ABSTRACT Some aspects of energy, protein and vitamin E nutrition of the performance horse are discussed. The amount, dietary source and time of ingestion of energy before exercise can influence performance. In 1989 the National Research Council (NRC) increased their estimates of energy required by racehorses. Recent studies indicate that the increase was reasonable. Many factors, however, can influence energy requirements. Therefore, the best measure would be body weight and composition of the horse. A proper balance of soluble carbohydrate, fiber, fat and protein is essential. Some guidelines are presented. The amount and type energy source given before exercise can influence level of plasma glucose and free fatty acids during exercise, but the effects of these changes in the concentration of metabolites remains to be determined. There is no evidence that increased dietary concentrations of protein are needed and, in fact, may impair performance. Supplemental histidine (to enhance carnosine levels) or carnitine appear to be of limited value for horses fed conventional diets. Dietary concentrations of vitamin E less than the 80 IU/kg recommended by NRC seem to adequately protect against exercise-induced peroxidation. The NRC value may be justified on the basis of immune response, but further studies are needed. Vitamin E has been shown to be involved with familial equine degenerative myeloencephalopathy and may be involved with equine motor neuron disease, a condition considered to be similar to amyotrophic lateral sclerosis in humans. J. Nutr. 124: 2723S-2729S, 1994.

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• horses • energy • vitamin E • protein

Energy

Energy is the nutritional factor most influenced by exercise. The amount of energy, source of energy and...
time of giving the supply of energy before exercise is discussed.

**Amount.** The amount of energy required depends on the type and duration of activity. The National Research Council (NRC) (1989) suggested the energy requirement for intense work such as racing is about twice that for maintenance. The maintenance requirement for a 500-kg horse is 68.6 MJ of digestible energy (DE). Thus, a 500-kg horse at hard work requires ~137 MJ of DE per day. The NRC (1989) estimate was increased ~27% above the previous NRC value (NRC 1978). The increase was based on studies by Anderson et al. (1983) and Pagan and Hintz (1986) and on surveys taken at race tracks (Burton and MacNeil 1985, Ignatoff and Hintz 1980, Nash and Hintz 1981, Winter and Hintz 1981). Recent surveys indicate the 1989 estimates are not unreasonable for energy intakes of horses doing intense work (Gallagher et al. 1992a, Gallagher et al. 1992b, Glade 1983, Schils and Jordan 1989, Southwood et al. 1993, Zmija et al. 1991). The NRC guidelines should also be reasonable for smaller equines. Eaton et al. (1991) reported that the energy expenditure in ponies and horses (per unit of body weight) is similar over a range of submaximal speeds. Many factors, however, such as condition and training of the horse, ability of rider or driver, environmental temperature and temperament of the horse, can influence the utilization of energy. Therefore, NRC and similar estimates should only be used as guides. Body weight and body condition are better indices of adequacy of energy intake. Excess body fat is obviously detrimental to athletic performance. Lawrence et al. (1992) studied body composition of 38 horses in a 150-mile 2-d endurance event. Sixty-one horses started but only 25 horses finished. Body fat content of top finishers and nonfinishers was estimated by measuring rump fat thickness with ultrasound. The top finishers had 6.5% body fat, whereas the nonfinishers had 11% body fat.

On the other hand, if the horses are not fed adequate energy, it would seem that glycogen storage and hence performance would not be maximized. Lim (1980) stated that optimal body weight of race horses had a high correlation with performance. Furthermore, he concluded that the optimal body weight for a horse had a range of only plus or minus 1.5% of body weight. He also stated, “Performance appears to be less affected above rather than below optimal weight.”

The Japan Racing Association (JRA) provides information on body weight and body weight changes on the racing program. It was thought that perhaps a study of the weight changes as presented in the JRA racing programs might indicate if the Japanese trainers believed information on body weight changes were of value. Data were summarized for 565 horses (Hintz 1993). The average body weight change between race days was only +0.6 kg, 25.3% of the horses had no weight change, 54.3% had changes of ≤2 kg, 86.9% had changes of ≤0.8 kg and 95% had changes of ≤10 kg. Only 3.8% of the horses lost >6 kg and only 1.2% lost >10 kg, whereas 9.2% gained >6 kg and 3.4% gained >10 kg. The results could suggest that Japanese trainers try to maintain body weight of the horses and might not race the horses if weight loss is greater than expected. Unfortunately, it was not possible to obtain weights of the horses that were not raced.

**Source.** The primary sources of dietary energy for horses are carbohydrates (soluble and fiber), fats and protein. Protein is not considered to be an efficient source of energy [Miller and Lawrence 1988]. Thus, most of the following discussion is about carbohydrates and fats. Hargreaves (1991) pointed out that during prolonged strenuous exercise in human athletes, muscle glycogen and blood glucose are major substrates for contracting skeletal muscle and fatigue is often associated with glycogen depletion. Thus, human athletes in endurance events used an exercise–diet regimen known as glycogen loading that involved depletion of glycogen stores and then ingestion of large amounts of soluble carbohydrates to greatly increase preexercise glycogen concentrations.

It has been stressed that the practice of repeated glycogen loading in humans can have undesirable side effects, such as depression, lethargy, loss of muscle tissue, chest pains, abnormal electrocardiograms, cramps, overweight feeling and early fatigue [American Dietetic Association 1987]. Fortunately in recent years, it has been shown that suitable glycogen storage can be attained in trained human athletes by simply tapering of training and increasing dietary carbohydrates to 60–70% of the total energy [Hargreaves 1991].

Glycogen depletion has also been associated with fatigue of horses in endurance events (Hodgson et al. 1983), but glycogen loading does not seem to be the answer. Radical dietary changes in equine diets, such as feeding high intakes of starch to stimulate glycogen loading, are not widely recommended. Snow (1992) concluded that 1) a normal high energy diet is all that is required to produce adequate glycogen storage, 2) the trained horse normally stores glycogen in high amounts, 3) excessive soluble carbohydrate intake could cause digestive upsets and laminitis and 4) rate of depletion of glycogen is not greatly improved by supplements of soluble carbohydrates.

On the other hand, inadequate soluble carbohydrate intake could result in decreased glycogen storage [Pagan 1992]. Thus, although glycogen loading by dietary manipulation is not recommended, the diet must contain adequate amounts of soluble carbohydrate. NRC (1989) suggests that a 65:35 ratio of concentrate to hay is reasonable for horses performing intense work. A certain amount of roughage is needed to maintain normal digestive function and to satisfy the horse’s need for chewing [Meyer 1987]. NRC (1989) acknowledged the importance of roughage in the ration.
but did not establish a requirement. It has been suggested that intakes of 0.5 kg hay/100 kg body weight could be satisfactory but that an intake of ≥1 kg hay/100 kg body weight would be preferred (Hintz 1983, Meyer 1987). Decreasing the concentrate-to-hay ratio to <50:50 could limit energy intake.

Many studies have been conducted on the feeding of fat to horses since the report of Slade et al. (1975) in which it was concluded that the addition of fat to the diet of horses could enhance endurance. Fat supplementation was thought to enhance endurance because of a glucose-sparing effect due to greater mobilization of free fatty acids (Hintz et al. 1978, Slade et al. 1975). Despite the many studies since 1975, there is still controversy about the effect of supplemental fat on performance and metabolism. Lawrence (1990) reviewed several studies and concluded that there is little consistency in the results of the various studies on the effect of dietary fat on changes in glycogen, glucose and lactate during exercise. Her conclusion was “to date the information supporting a positive effect of supplemental fat on fuel metabolism is inconclusive.” Snow (1992) also questioned the glycogen-sparing effect of fat. He concluded that fat supplementation might be of value for horses involved in prolonged activity because of the high energy density of fat. Increased density could cause an increase in total energy intake or it could provide the same amount of energy in less feed and therefore resulting in less gut fill.

The differences among studies reported by Lawrence (1990) may be due to many factors such as type of exercise, conditioning of the animal and ratio of fat to carbohydrate in the diet. Pagan (1992) concluded that the excessive intakes of fat could, in fact, decrease glycogen storage because of the increased intake of dietary carbohydrate. Studies of the glycogen-sparing effect can also be complicated by the variance in glycogen content between different sampling sites in the muscle (Snow and Harris 1991).

Glycogen sparing is thought to be of much less benefit to horses working ≤2 miles than to endurance horses because most studies have indicated that only 25–33% of prerace glycogen is used during the shorter events (Snow and Harris 1991). Nevertheless, supplemental fat has been reported to improve performance of horses during anaerobic activities (Harkins et al. 1992, Oldham et al. 1989, Webb et al. 1987). Harkins et al. (1992) pointed out, however, that even though they found the addition of fat improved the time to travel 1600 m by 2.4 s, there were inherent problems when conducting such studies. They concluded “More research is needed before an ergogenic benefit from fat can be claimed.” Mechanisms other than glycogen sparing could be involved for horses doing anaerobic work. Scott et al. (1993) suggested that fat supplementation could enhance anaerobic performance by decreasing thermal load.

Changes in acid-base balance could be involved, but the role of fat is not clear. Pagan et al. (1993) reported that feeding fat could decrease lactate during intense exercise, whereas Taylor et al. (1993) reported feeding fat increased lactate levels.

**Timing.** When should the energy be fed? Meyer (1987) wrote “No experimental data are available about the optimal feeding for race horses and trotters on the day of the race.” Surveys indicate feeding practices vary among trainers. One survey indicated that about half the trainers at four standardbred tracks fed no hay on race day and the other half greatly decreased hay on race day (Ignatoff and Hintz 1980). Schils and Jordan (1989) surveyed thoroughbred trainers at one track and found that 96% fed less hay on race day, 33% fed less grain, 63% fed the same amount of grain and 4% fed more grain. Meyer (1987) concluded that based on studies of rate of passage and studies on hormonal responses, horses should be fed no less than 4 h before the event. In human athletes, glucose or starch 1–4 h before exercise can result in a decrease in blood glucose concentrations during competition (Willcuts et al. 1988). Lawrence et al. (1993) studied the effect of providing no feed (control) or 1, 2, or 4 kg of corn to horses ~2.5–3 h before exercise. The design was a 4x4 Latin square with the horses working on a high speed treadmill. There was a warming-up phase and then a high intensity work bout for 1600 m at 11 m/s. The responses to feeding 1, 2, or 3 kg of corn were similar, but there were significant differences between the fed animals and the controls. The control animals had a steady plasma glucose concentration throughout warm-up and exercise. The fed animals started with a higher glucose concentration than the control animals but had a steady decline, and by the end of the exercise bout had blood glucose levels ~1 mmol/l lower than the control horses. Free fatty acid (FFA) concentrations were significantly higher in the control horses than in the fed animals. Plasma lactate concentrations and heart rate were increased by exercise but were not influenced by dietary treatment.

Zimmerman et al. (1992) fed corn or alfalfa to horses 2 h before exercise. The horses fed alfalfa had a lower starch intake than those fed corn and had a higher FFA level during exercise. The lower FFA found in the horses fed corn is consistent with the study of Lawrence et al. (1993) in which the corn-fed horses had lower FFA than the controls.

It is concluded that the time of eating before exercise could cause changes in metabolites but that the effects of the changes of metabolites on performance of the horse remain to be determined.

**Protein**

**Amount.** There is no evidence that performance can be enhanced by feeding diets containing concentrations of protein greater than the 11% (dry matter
Amino acids. The amino acid requirements of growing horses have received some attention. Lysine seems to be the first limiting amino acid in conventional feedstuffs [NRC 1989]. It has been suggested that threonine is the second limiting amino acid for growing horses fed grass forages and corn/oat/soybean meal–based concentrates (Graham et al. 1994). Little attention has been given to the amino acid nutrition of the performance horse. It has been suggested that certain amino acids may influence energy metabolism. Glade (1991) gave an amino acid mixture of leucine, isoleucine, valine, glutamine and carnitine orally via a syringe 30 min before exercise and again within 10 min after completion of exercise. The horses walked on an inclined treadmill for 30 min at a rate of 1.5 m/s. Horses given the amino acid supplementation had lower plasma lactate concentrations during exercise than the controls. Glade suggested that the mixture of amino acids may have improved the oxidative capacity of the horse. Carnitine, one of the compounds in the above mixture, is a low-molecular-weight quaternary amine and should not be classified as an amino acid. It has been suggested the carnitine may have an ergogenic activity in human athletes because of its role in fatty acid metabolism (Snow 1992). Snow (1992) suggested that because it is difficult to increase equine muscle stores of carnitine by dietary supplementation, it is difficult to see how carnitine supplementation could be expected to improve performance in horses. Furthermore, Foster and Harris (1992) reported no detectable loss of total carnitine from the middle gluteal muscle of thoroughbreds that was associated with intense exercise. Studies in other species have also cast doubt on the value of supplemental carnitine. Decombez et al. (1993) studied the effect of carnitine in humans in a glycogen depleted state. They concluded "it seems unlikely that L-carnitine could quantitatively influence fat oxidation during submaximal exercise in healthy individuals."

Carnosine (β-alanyl-L-histidine) is found in high concentrations in equine muscle [Miller-Graber et al. 1990] and is a major physiochemical buffer (Sewell et al. 1992). It has been suggested that increasing the histidine intake may increase the muscle carnosine content. Miller-Graber and Syers (1993), however, found no increase in muscle carnosine in horses given diets containing 0.25 or 0.56% histidine. The concentration of carnosine probably has a genetic basis. Bump et al. (1990) reported that samples from the middle gluteal muscles from quarter horses had significantly greater carnosine concentrations than samples from thoroughbreds or standardbreds. The carnosine content was positively correlated with fast twitch glycolytic fiber percentage. Therefore, it is not expected that carnosine can be increased by dietary means.

Vitamin E

There has been great interest in the vitamin E nutrition of the horse. NRC (1989) increased the estimate...
The effect of vitamin E on the immune system was not surprising because studies with several species have shown that antioxidant supplementation can significantly improve certain immune responses (Bendich 1993). The need to provide more than the NRC 1978 intake to maintain vitamin E blood levels was recently supported by Saastamoinen and Juusela (1993), who suggested that 1500 IU was needed daily to maintain plasma vitamin E in finish horses in the winter. Vitamin E in the horse was also of interest because of reports in other species that exercise induces free radical formation in muscle and liver, resulting in oxidative damage such as lipid peroxidation. It has been reported that the damage can be reduced by antioxidants such as vitamin E (Witt et al. 1992).

Petersson et al. (1990) examined the effect of vitamin E on lipid peroxidation in the horse during exercise. Two groups of horses, one exercised and one control, were fed a diet containing 13 IU of vitamin E/kg for 4 mo and then supplemented with vitamin E. No clinical signs of vitamin E deficiency were noted in either the exercised or unexercised group. It was hypothesized that exercise would increase the indices of lipid peroxidation such as breath pentane production and plasma thiobarbituric reactive substances (TBARS). No differences were noted between the exercised and unexercised groups. Supplementation with vitamin E did not change the response to exercise. However, the expired air, muscle and blood samples were not taken immediately after exercise and thus differences may have gone undetected.

Ponies that had been fed a diet containing 42 IU vitamin E/kg for 10 mo were exercised on a treadmill at a rate of 4 m/s for 20 min at 0 and 7° slope (McMeniman and Hintz 1992). As in the study by Petersson et al. (1990), no signs of vitamin E deficiency were noted. Expired air, venous blood and samples from the middle gluteal muscle were obtained immediately after exercise. Exercise increased heart rate, plasma lactate and breath pentane, but no increases in plasma or muscle TBARS were noted. A second study was conducted in which the ponies were fed a diet of 70% timothy hay, 27% sweetfeed and 3% corn oil for 10 wk. Half of the ponies were fed 100 IU vitamin E/d. After 10 wk the ponies were exercised at a speed of 4 m/s and slope of 10° until fatigued. Exercise increased heart rate, plasma lactate and breath pentane but not TBARS. Vitamin E supplementation increased plasma vitamin E but not muscle vitamin E.

It was concluded that the exercise caused minimal peroxidative stress when ponies were fed ≥42 IU of vitamin E/kg. The addition of corn oil did not reduce plasma or muscle vitamin E but increased muscle TBARS. It was hypothesized that the addition of corn oil would reduce plasma vitamin E as polyunsaturated fatty acids (PUFA) have been shown to decrease plasma vitamin E in other species. Siciliano and Wood (1993), however, found no evidence that the addition of the oil interfered with vitamin E status in 2-y-old horses fed diets containing 6.4% soybean oil for 90 d. The horses were not exercised. Perhaps if the above trials would be conducted for a longer period or the diets had wider PUFA vitamin E ratios, an influence of vitamin E would have been noted.

The studies by Petersson et al. (1990) and McMeniman and Hintz (1992) do not support the recommendation of 80 IU vitamin E/kg of diet for horses. The lack of interaction of exercise and vitamin E was not expected. Tidus and Houston (1993), however, also recently indicated that the role of exercise on vitamin E nutrition needs further study. They reported that rats fed a vitamin E–free diet for 8 wk and performing either acute or chronic exercise did not exhibit greater peroxidation than controls given vitamin E. Perhaps other antioxidants such as vitamin C responded to the challenge. Goldfarb (1993), however, recently concluded that limited information is available concerning the effects of vitamin E and vitamin C on exercise-induced oxidative stress. He stated that the viability of the vitamins alone and in conjunction with each other in preventing exercise-induced lipid peroxidation requires further investigation.

Of course, the studies of Petersson et al. (1990) and McMeniman and Hintz (1992) should not be used as evidence that the requirement for horses should be <80 IU vitamin E/kg of diet because no measures of immune response were made.

There might also be specific cases for vitamin E supplementation. Equine motor neuron disease (EMD) was first reported by Cummings et al. (1990). Affected horses have generalized weakness, muscle fasciculation, muscle atrophy and weight loss. There is degeneration of the motor neurons in the spinal cord and brainstem, leading to axonal degeneration in the ventral roots and peripheral and cranial nerves. The changes are similar to those described in people with amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig’s disease. Almost all of the affected horses identified thus far have been housed in stables without access to pasture, and very low vitamin E plasma and adipose tissue levels have been reported in the horses (Divers 1993). Further studies are needed to determine the relationship, if any, of vitamin E nutrition to equine motor neuron disease. The recent suggestion by Rosen et al. (1993), however, that mutations in the cytosolic copper/zinc superoxide dismutase gene may be involved with the development of ALS in humans.
lends support to the theory that antioxidant deficiency may be involved in EMD. Rosen et al. (1993) suggested that free radicals may be the pathogenic mechanism for motor neuron death in ALS.

Equine degenerative myeloencephalopathy (EDM) is a diffuse degenerative disease of the spinal cord and brainstem. It is most commonly found in young horses. Affected animals may show various signs such as clumsiness, inability to do complicated movements, malpositioning of limbs at rest or obvious ataxia (Blythe and Craig 1992a). Histologically there can be neuroaxonal dystrophy in brainstem nuclei and throughout the spinal cord (Blythe and Craig 1992a). It has been proposed that it is a familial disease and that a deficiency of vitamin E is involved (Blythe and Craig 1992b). Supplementation with very high levels of vitamin E (6000 IU/d) may reduce the incidence of EDM in young horses from affected families or can cause improvement in some affected animals (Blythe and Craig 1992b). The reason why such high intakes of vitamin E are needed for certain families of horses is unknown, but it has been suggested that such horses are more susceptible to antioxidant deficiencies or have an increased antioxidant requirement during the first year of life (Blythe and Craig 1992b).

There is much to learn about the role of nutrition in the exercising horse. Energy is of prime concern, but many topics not discussed in this review, such as water, electrolytes, acid-base balance, minerals and vitamins, can be of great importance. Advances in equine nutritional science could help increase the well-being of horses, decrease injuries and increase longevity of athletic careers.

LITERATURE CITED


