Diet and exercise performance in the horse

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The rate of energy expenditure in maximal physical activity is fifty times that occurring at rest. Thus, a consideration of the manipulation of diet to influence physical performance should enquire into dietary energy and its metabolism. It is self-evident that a failure to meet the horse’s energy requirements, as estimated by INRA (1990) and National Research Council (1989), will result in a chronic loss of body mass, debility and a decline in physical ability. In a survey of racing Standardbreds (STB) (Gallagher et al. 1992a) the daily intake of digestible energy (DE) was found to exceed the National Research Council (1989) estimate by 27%, and so compliance with chronic energy needs is generally assessed by degree of fatness.

Thoroughbred (TB) horses of moderate fatness apparently use depot fat as a source of energy more effectively than thin or fat horses, as indicated by reduced glycogen expenditure during exercise at a heart rate of 200 beats per min (bpm) (Scott et al. 1992). Nevertheless, it is unclear how athletic ability is affected by an abnormally large dietary energy intake for a brief period, when fat deposition will change little. Thus, a distinction is drawn between acute and chronic effects of diet on athletic response (Frape, 1988). Changes in this response are hard to demonstrate because they are difficult to replicate on the track. Even using a standardized treadmill Plummer et al. (1991) found a mean coefficient of variation of 9·6%. Moreover, differences exist between relative solo-run times and a run time in competition, which has a potent influence on performance of TB (Harkins & Kamerling, 1992). Therefore, despite greater precision of laboratory measurements, performance in competition is not reliably predicted. Yet non-competitive work is an important function that can justify laboratory measurements to unravel the role of diet in work output.

POSTPRANDIAL DIGESTION AND FERMENTATION

Processing of cereals can influence digestibility and, therefore, the proportions digested and fermented. Processing of roughage can accelerate rate of passage, reduce gut fill, but decrease hindgut utilization (Wolter et al. 1975, 1977, 1978). The extent to which feed is fermented will influence the weight of ingesta. Slade (1987) measured the speed of Quarter horses galloping at up to 19·6 m/s (44 mph) over 137 m or 229 m from a running start. Speeds differed according to the digestibility of feeds, as reflected by differences in gut fill.

The extent to which cereal starch provides glucose, or volatile fatty acids (VFA), depends on its precaecal, and even its pre-ileal digestibility. Kienzle et al. (1992) reported that the pre-ileal digestibility of oat starch was higher than that of maize starch, with similar degrees of processing. Grinding of whole grain led to high pre-ileal digestibility for oats amounting to 98·1% and for maize 70·6%; rolling, or breaking, had little effect. Starch gelatinization enhances its small intestinal digestion, but only at moderate or high rates of intake. At low intakes most sources of starch are digested in the small intestine (Potter et al. 1992a).
Energy for muscle contraction is derived from creatine phosphate (CP) and ATP. TB possess a high proportion of phosphocreatine (PCr)-rich fast-twitch fibres in skeletal muscles (Harris & Hultman, 1992), so that loss of adenine nucleotides (AN) during intense exercise is greater than in man. After repeated gallops a 50% loss of ATP was recorded (Harris et al. 1991c, 1992; Sewell & Harris, 1991), associated with a decrease in running speed and fatigue (Harris et al. 1991b), and with lower glycolytic rates and muscle ATP:ADP. The accumulation of ADP stimulates AN degradation to inosine monophosphate (IMP) with an increase in plasma NH₃ concentration. There is a critical pH below which ADP rephosphorylation declines, PCr acting both as an intracellular buffer and as a reservoir for this rephosphorylation. Supplementation of human subjects with creatine monohydrate has increased muscle total creatine and PCr contents (Harris et al. 1992). The energy reserves for this synthesis depend on training, diet and the inherent characteristics of the horse. ATP resynthesis fuelled by fatty acid (FFA) oxidation is slow, so inadequate glycogen storage in active muscle fibres causes early fatigue despite an abundance of FFA. With increasing rates of energy expenditure the preference for glucose as a substrate increases, although the proportions of glucose and FFA used change rapidly with distance (Harris et al. 1987). Glycogen stores apparently do not limit sprint performance following training, which can increase glycogen content of limb muscles by 39% (Guy & Snow, 1977). Fatigue during extended exercise partly results from a decline in blood glucose concentration, and the rate of this decline is affected by velocity, by the extent of glycogen stores, by dietary manipulation that spares glycogen mobilization and by training that promotes a greater use of both fat and glycogen during maximal exercise (Snow & Mackenzie, 1977).

**POSTPRANDIAL FITNESS AND GLYCOGEN LOADING**

Glucose solutions given before prolonged exercise could increase glycogen degradation rate by raising plasma insulin thereby lowering FFA mobilization. Glucose given during hard exercise does not stimulate insulin secretion, but hyperinsulinaemia is protracted in TB following a meal (Stull et al. 1987; Frape, 1989); thus, timing of meals before extended exercise may be critical. Ponies given each day after exercise a fluid providing 5-4 MJ DE exhibited lower heart rates and blood lactate concentrations during and following subsequent exercise (Lindner et al. 1991).

Glycogen loading is the practice of elevating muscle glycogen by first depleting muscle glycogen, through hard anaerobic exercise, then repleting while the horse is at rest. Harris & Hultman (1992) found no difference between diets in glycogen loading, but it probably occurs more effectively with a high-carbohydrate diet, than with a high-fat diet, following intense exercise that depletes type II muscle fibres of glycogen (Pagan et al. 1987a). Glycogen loading after aerobic work is ineffective and generally may cause poorer performance (Topliff et al. 1985, 1987; Pagan et al. 1987a) and an increased risk of exertional rhabdomyolysis (tying-up). Hodgson (1993) speculates that horses pre-disposed to tying-up exhibit a temporary failure in the control of intracellular Ca²⁺.

**DIETARY FAT AND EXERCISE**

Carbohydrate-rich diets increase the risk of colic and laminitis, but fat-rich diets do not; whereas fats are not subject to microbial fermentation and they yield less CO₂ per mol
Fig. 1. Relationship of respiratory quotient (RQ) to velocity of horses given high-starch, high-protein or high-fat diets. At high velocity there is no difference in RQ because high rate of energy expenditure demands glycolysis. At low–moderate velocity, fat and protein may be used.

Fig. 2. Relationship between respiratory quotient (RQ) and time during aerobic work (low–moderate velocity, 4-6 m/s) in horses given high-starch, high-fat, or high-protein diets. On high-fat or high-protein diets fat mobilization becomes predominant earlier, thereby conserving muscle glycogen.

ATP generated. Dietary oils and fats are well utilized (McCann et al. 1987; Hollands & Cuddeford, 1992; Potter et al. 1992b) and, as well as reducing the risk of colic and laminitis, they may promote intramuscular and hepatic fat metabolism. Hard training (Hambleton et al. 1980) and fat supplementation with anaerobic (Pagan et al. 1993) and extended aerobic (Pagan et al. 1987c) exercise are followed by an elevation in plasma FFA, but resting FFA are lowered by supplementation (Harkins et al. 1992). Thus, there may be stimulation of β-oxidation, or of both fat mobilization and metabolism (Figs. 1 and 2), sparing glycogen. Oils especially rich in polyunsaturated fatty acids (PUFA) have no notable benefit for ponies given a PUFA-deficient diet for 7 months (Sallmann et al.)
Table 1. *Resting muscle glycogen as affected by added dietary fat (g/kg diet) in diets of differing energy densities but generally given to horses to equalize digestible energy (DE) intake*

<table>
<thead>
<tr>
<th>Added dietary fat (g/kg diet)</th>
<th>0</th>
<th>20–30</th>
<th>50–60</th>
<th>80–100</th>
<th>140–150</th>
<th>SE</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat source</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Animal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Hintz et al. (1978)</td>
</tr>
<tr>
<td></td>
<td>81</td>
<td>—</td>
<td>—</td>
<td>78</td>
<td>—</td>
<td></td>
<td>Meyers et al. (1989)</td>
</tr>
<tr>
<td></td>
<td>94</td>
<td>—</td>
<td>109</td>
<td>143</td>
<td>—</td>
<td>10.5</td>
<td>Oldham et al. (1990)</td>
</tr>
<tr>
<td></td>
<td>88</td>
<td>—</td>
<td>—</td>
<td>127</td>
<td>—</td>
<td>2.6</td>
<td>Scott et al. (1992)</td>
</tr>
<tr>
<td></td>
<td>93</td>
<td>—</td>
<td>—</td>
<td>145</td>
<td>—</td>
<td>2.1</td>
<td></td>
</tr>
<tr>
<td>Vegetable oil†</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Hambleton et al. (1980)</td>
</tr>
<tr>
<td></td>
<td>680</td>
<td>—</td>
<td>255</td>
<td>292</td>
<td>240</td>
<td>—</td>
<td>Pagan et al. (1987b)</td>
</tr>
<tr>
<td></td>
<td>198</td>
<td>229</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>12.0</td>
<td>Harkins et al. (1992)</td>
</tr>
</tbody>
</table>

* Tissues were: *gluteus medius, biceps femoris* or *quadriceps femoris.*
† Fat added replaced maize grain giving diets of different energy densities but constant daily energy and protein intakes.
‡ Fat added diet contained less roughage but more fat, starch and protein and was fed to provide equal DE intakes.

1992). Nevertheless, fatty acid chain length and degree of unsaturation may influence the exercise response (Pagan et al. 1993). The increased energy density achievable with dietary fat could be a necessary attribute (Worth et al. 1987). When fat amounting to 100 g/kg diet replaced starch, heat production fell from 77% to 66% of DE and net energy (NE) rose from 16% to 36% of DE during work (Scott et al. 1993), reducing thermal stress (McCann et al. 1987).

Several reports indicate no difference in resting muscle glycogen concentration between high-starch and high-fat diets (Hintz et al. 1978; Pagan et al. 1987a; Topliff et al. 1987), whilst two other reports suggest that fat supplementation lowers muscle glycogen (Pagan et al. 1987b; Greiwe et al. 1989). Most reports describe increased resting muscle glycogen following inclusion of vegetable, or animal, fat at about 100 g/kg diet to provide equal DE or metabolizable energy (ME) intakes (Hambleton et al. 1980; Meyers et al. 1987, 1989; Oldham et al. 1990; Harkins et al. 1992; Jones et al. 1992; Scott et al. 1992; Table 1). Effects of fat on hepatic glycogen capacity, which is 10% of that in skeletal muscle, are equivocal (Hambleton et al. 1980; Pagan et al. 1987b).

Respiratory quotient (RQ) rises with increasing speed (Pagan et al. 1987b), is lowered by training (Meyers et al. 1987) and is either not affected by dietary fat (Meyers et al. 1989), or is lowered by additional protein, or fat, during submaximal exercise (Pagan et al. 1987b) (Table 2, Figs. 1 and 2). RQ is positively correlated with muscle glycogen reserves during mild aerobic exercise and it declines as submaximal exercise progresses (Pagan et al. 1987b) (Fig. 2), indicating a sparing of glycogen. Higher stores of muscle glycogen achieved by fat supplementation accelerate mobilization of muscle glycogen during anaerobic exercise (Oldham et al. 1990; Jones et al. 1992; Scott et al. 1992). However, no clear picture emerges that fat would particularly benefit exercise in which
extended aerobic metabolism dominated (Hintz et al. 1978; Pagan et al. 1987b; Greiwe et al. 1989; Figs. 3 and 4). Metabolic adaptation to a high-fat diet may take 6–11 weeks (Custalow et al. 1993) and failure to take this, and other complications, into account may be responsible for some of the inconsistencies between different studies. Since fat yields energy only by oxidation, minimal glycogen sparing would be expected during maximal aerobic exertion. Some workers report similar (Worth et al. 1987), or lower (Meyers et al. 1989), blood glucose concentrations, in fat-supplemented horses during aerobic exercise; but most (Hintz et al. 1978; Hambleton et al. 1980; Webb et al. 1987a; Oldham et al. 1990; Harkins et al. 1992; Scott et al. 1992; Custalow et al. 1993) observed higher values during and after exercise of all types (Fig. 3, Table 3), even with increased work effort (Webb et al. 1987a; Harkins et al. 1992).

Lower heart rates, more rapid recovery of resting rates (Meyers et al. 1987), lower blood lactic acid concentrations during and after submaximal and strenuous exercise (Pagan et al. 1987a,c, 1993; Webb et al. 1987a; Meyers et al. 1989; Table 3), and higher lactate speed threshold (Custalow et al. 1993; Pagan et al. 1993) possibly reflect a slightly lower RQ (Fig. 1). The effects of high-protein diets on blood lactic acid may be more prominent than those of high-fat diets (Pagan et al. 1987a,c). Observations in human subjects indicate that high-protein high-fat diets increase the activity of skeletal muscle lipoprotein lipase (EC 3.1.1.34; LPL), whereas high-carbohydrate diets reduce that activity (Jacobs, 1981). Increased energy generation from fat oxidation with high-fat, high-protein diets may stem from the combined effects of increased muscle LPL hydrolysis of plasma triacylglycerols (TAG) and increased use of plasma FFA (Figs. 2 and 3), accounting for lower plasma lipids during aerobic standardized exercise test (Meyers et al. 1987). It is suggested that high-fat diets increase activity of muscle LPL (and possibly of TAG lipase), reducing adipose tissue LPL activity, in contrast to the effects of starch (Fig. 5). A dietary increase in either protein or fat normally results in decreased starch, reducing ‘heating’, anxiety, heart rate and excitability. The potential advantages and disadvantages of fat are proposed in Table 4.

Strenuous exercise causes increased plasma thiobarbituric acid-reacting substances (TBARS) and breath n-pentane per kg body weight (McMeniman & Hintz, 1992).
Fig. 3. Generalized relationships, with time, of blood glucose and muscle glycogen concentration in horses of moderate fatness during extended aerobic work.

Fig. 4. Generalized relationship between glycogen stores and time-interval during intense anaerobic work (>600 m/min, >190 beats/min).
Table 3. Effect of fat supplementation on blood plasma lactate and glucose concentrations during exercise at constant velocity (C) or at uncontrolled velocity (UC) and after postexercise rest

(Values averaged over the sources used for each comparison of carbohydrate control and added fat)

<table>
<thead>
<tr>
<th>Added dietary fat (g/kg)</th>
<th>Velocity</th>
<th>Plasma lactate (mmol/l)</th>
<th>Plasma glucose (mmol/l)</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Work</td>
<td>Rest</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>2.30</td>
<td>1.98</td>
<td>Hambleton et al. (1980), Meyers et al. (1987, 1989), Webb et al. (1987a), Worth et al. (1987)</td>
</tr>
<tr>
<td></td>
<td>100</td>
<td>1.89</td>
<td>1.47</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>9.90</td>
<td>—</td>
<td>Pagan et al. (1987)</td>
</tr>
<tr>
<td></td>
<td>100</td>
<td>6.70</td>
<td>—</td>
<td>Hintz et al. (1978), Harkins et al. (1992)</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>2.31</td>
<td>2.25</td>
<td>Webb et al. (1987a), Oldham et al. (1990), Scott et al. (1992)</td>
</tr>
<tr>
<td></td>
<td>100</td>
<td>2.12</td>
<td>2.09</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>15.79</td>
<td>7.30</td>
<td></td>
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<tr>
<td></td>
<td>100</td>
<td>18.61</td>
<td>14.70*</td>
<td></td>
</tr>
</tbody>
</table>

Data from Webb et al. (1987) only.

Fig. 5. Proposal for reactions stimulated (→), not measurably influenced (↔), or suppressed (←) by high-protein or high-fat diets during equine exercise of moderate intensity in the postabsorptive state. Reactions proposed to be stimulated by dietary fat (□) and by dietary protein (□). GPT, glutamic–pyruvic transaminase (EC 2.6.1.2); LPL, lipoprotein lipase (EC 3.1.1.34); IMP, inosine monophosphate.
Table 4. Provisional conclusions on effects of high-fat diets given to exercising horses compared with diets of normal fat concentration that provide similar amounts of dietary fibre, protein and digestible energy, but more starch

<table>
<thead>
<tr>
<th>Advantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Lower RQ during submaximal exercise (promoting fat catabolism) potentially extending endurance</td>
</tr>
<tr>
<td>2. Possible decrease in adipose tissue LPL activity and increase in muscle LPL activity</td>
</tr>
<tr>
<td>3. Increase in muscle glycogen stores, more glycolytic energy and possible delay in glycogen exhaustion during extended aerobic exercise</td>
</tr>
<tr>
<td>4. Increased, or sustained, blood glucose concentrations during extended exercise</td>
</tr>
<tr>
<td>5. Possibly delayed lactic acid accumulation during anaerobic exercise (lactic acid accumulation is proportional to the rate of glycogen expenditure, when other conditions are constant)</td>
</tr>
<tr>
<td>6. Reduction in heat of fermentation and in gut fill, which may benefit work at &gt;200 bpm, but which may compromise endurance</td>
</tr>
<tr>
<td>7. Reduced excitability of hot blooded horses and a possible reduction in risks of colic and laminitis in all types of horse.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. High cost of high grade fat</td>
</tr>
<tr>
<td>2. Wide availability of poor-quality feed grade fat and difficulty of assessing quality</td>
</tr>
<tr>
<td>3. Lack of stability of large fat supplements in mixed feed and practical problems of administration to the horse</td>
</tr>
<tr>
<td>4. Refusal of high-fat diets, or delay in acceptance of equivalent intakes, i.e. lower palatability</td>
</tr>
<tr>
<td>5. Lower large intestinal fluid reserves for endurance events</td>
</tr>
</tbody>
</table>

RQ, respiratory quotient; LPL, lipoprotein lipase (EC 3.1.1.34); bpm, beats/min.

However, the peroxidative stress of 30 g maize oil/kg diet was accommodated in exercising ponies given 46-2 mg vitamin E/kg dietary dry matter, through increased plasma glutathione peroxidase (EC 1.11.1.9) and superoxide dismutase (EC 1.15.1.1) activities and ascorbic acid concentration, despite higher muscle TBARS regardless of vitamin E concentration.

**Dietary Protein Requirements and Exercise**

The National Research Council (1989) has concluded that dietary protein requirements of horses are proportional to those for DE. Both extended work (Rose et al. 1980) and exercise in the postabsorptive state following high-protein meals (Miller & Lawrence, 1988) elevate plasma urea. Plasma NH₃ and uric acid concentrations rise and attain maxima during recovery (Harris et al. 1987; Miller et al. 1987; Miller & Lawrence, 1988), signifying an increased rate of AN cycling. A greater demand for ATP is partly met by the myokinase (EC 2.7.4.3) reaction (2ADP→1ATP+1AMP; Fig. 5). Deamination of AMP, which supplementary NaHCO₃ may ameliorate, produces NH₃ and IMP, yielding uric acid (Harris et al. 1987). Additional dietary protein may, or may not, aggravate this.

Protein assimilation occurs during work, following rest (Meyer, 1987). Johnson et al. (1988) detected no increase in N balance of ponies when set to work. Patterson et al. (1985) found that 1.9 g digestible protein per kg body weight⁰.⁷⁵ was adequate for intense exercise, although N balance was not determined. Orton et al. (1985) trotted growing horses for 12 km daily at 12 km/h, on either a 120–140 g protein/kg diet or a 60–80 g protein/kg diet; exercise increased feed intake of horses on the low-protein diet so that protein intake and growth rate equalled that of horses receiving the higher protein diet.
In a survey of racing TB, Glade (1983) found that protein intake was positively correlated with time-period to finish, implying that excess protein depressed speed. There seems little justification for greatly increasing the daily protein intake of exercising horses to meet some putative increase in chronic requirement; the relationship of dietary protein with extreme performance is, however, far from clear. Despite seemingly high National Research Council (1989) estimates, actual intakes are still higher. Yet a 56% excess over the estimates amongst racing STB (Gallagher et al. 1992a) and 21% excess for racing TB (Gallagher et al. 1992b) may reflect the natural protein content of palatable high-energy feeds.

Compared with horses given a control diet, RQ was lower in horses given a high-protein diet and exercised at high speed (Pagan et al. 1987b), implying stimulation of protein or fat metabolism in the postabsorptive state (Fig. 1), increasing urea yield (Frank et al. 1987) and water needs. Apart from this increased need Hintz et al. (1980) observed no detrimental protein effect in horses during distance riding, where dehydration causes fatigue. Compared with horses at about N balance, exercised horses with dietary protein intakes which give well in excess of N balance exhibit increased concentrations of blood urea, but reduced postexercise venous blood NH₃ when untrained. They also show decreased RQ during aerobic exercise and, when exercised at 240–600 m/min, heart rate, concentrations of blood and hepatic lactate and venous lactate:pyruvate are lower (Frank et al. 1987; Pagan et al. 1987b,c; Miller & Lawrence, 1988; Miller-Graber et al. 1991). It is concluded that high-protein diets, above the need for N balance, may confer some metabolic advantages to working horses.

**PROTEIN DIGESTIBILITY OR DEGRADATION**

The true digestibility of protein in the small intestine of horses ranges from 45 to 80%. At high rates of protein intake more will be degraded to NH₃ in the large intestines. Utilization of NH₃ by gut bacteria is between 80 and 100% (Potter et al. 1992c). Excessive protein intakes must inevitably increase the burden of unusable N, either in the form of inorganic N, or as relatively unusable bacterial protein. This burden is influenced by feeding sequence. The provision of a concentrate feed 2 h later than roughage, compared with simultaneous feeding, caused higher levels of plasma free, and particularly essential, amino acids, 6 and 9 h later (Cabrera et al. 1992). Plasma urea did not rise with dissociated feeding, but rose continuously for 9 h after the mixed feeding, indicating a large flow of N to the caecum. No corresponding measurements of heat production in the horse are available, but Belko et al. (1986) found that the thermic effect of food in exercising men increased with the protein content, 150–270 min postprandially.

**HEAT PRODUCTION, BLOOD FLOW AND MEAL TIME IN RELATION TO EXERCISE**

Blood volume is important for transport of both O₂ and heat so that a large plasma volume is accompanied by a considerable skin blood flow. This volume can increase by 30% over 2 weeks training (Erickson et al. 1987). During exercise the rise in core temperature is proportional, not to exercise intensity, but to the percentage $V_{O_{2\max}}$, which is increased by training. Most heat is dissipated through the skin, rather than the lungs, accompanied by an increased rate of insensible moisture and electrolyte losses.
Fatigue is delayed by partial replacement of these losses. Dilation of subcutaneous blood vessels during exercise diverts blood from skeletal muscles, contributing to a decrease in work capacity (Webb et al. 1987b). A diet supplemented with 10 g fat/kg reduced the thermal load by increasing NE:DE (Potter et al. 1990; Scott et al. 1993). If circulatory adjustments fail to maintain heat balance, ventilation rate is increased, inducing respiratory alkalosis. Following feed ingestion the amount of blood distributed to the gastrointestinal (GI) tract increases, competing with the redistribution evoked by a rise in heat production. Accommodation is achieved by augmenting cardiac output, redistributing regional blood flow, or by combining these mechanisms (Table 5). Experiments with ponies (Duren et al. 1992) indicated that exercise of 7-8 m/s on a 6-3° incline at 75% of maximum heart rate for 30 min, 1-4 h after feeding, led to higher GI tract and skeletal muscle blood flows than those in fasted ponies and there was an increase in cardiac output, stroke volume and arterial blood pressure. Therefore, work is normally delayed for 5-8 h after feeding to avoid decreased blood glucose (Laurence et al. 1993) and problems of blood flow redistribution; the optimum time interval is influenced by the proportion of diet subject to fermentation.

**FEEDING BEFORE ENDURANCE EXERCISE**

Meyer (1987) concluded that endurance horses should be given large amounts of roughage (6-8 kg/d) to dilate the large intestinal volume and increase water and electrolyte reserves. The maximum postprandial increase in caecal capacity is 8-14 kg/kg dry matter ingested, depending on the fibre content, with 130-135 mmol Na/l flow from the ileum. The average Na content of digesta in the large intestine is 40 mmol/l. Meyer (1987) concluded from previous evidence (Meyer et al. 1982) that the horse should be fed more than 5 h before an endurance race, depending on the feeding sequence, as most of the residue will then have passed the ileo-caecal orifice. Meyer (1987) compared two rations, one contained 2 kg concentrates and 3 kg hay, providing 11-5 g Na and 80 g K, the other 2 kg oats, providing 1 g Na and 10 g K. At 4 h after feeding the retention of water, Na and K were respectively 5-8 kg, 9-3 g and 48 g for the first ration and 0-6 kg, 0-2 g and 1 g for the second ration. In support of Meyer (1987), Ralston (1988) found that horses failing to complete 160 km races had received mixtures containing less hay and more grain, and had been trained for greater distances (83 cf. 61 km/week). The energy intake per km of training was less in those that failed. Roughage has the disadvantage of inducing additional fluid weight and some contain excessive K that can stimulate diuresis, and loss of water. Water and electrolytes could be provided during a ride; but if there has been an iso-osmotic loss in sweat spontaneous drinking may not occur, unless the fall in plasma volume exceeds 6% (Sufit et al. 1985).
ACID-BASE BALANCE AND EXERCISE

Fatigue during exercise is associated with a deviation of blood pH from the ideal range, causing metabolic acidosis, or with over-heating, respiratory alkalosis. In lactic acidosis there is increased production of H⁺ ions, owing to an O₂ debt. With overheating there is increased loss of CO₂, resulting from a high respiration volume per min. Mineral nutrition has a role in acid–base balance. Excess base (or acid) in feed may be approximated as the difference between the sums of mineral cations and anions:

\[
\text{base excess (MEQ/g)} = (\text{Na}+\text{K}+\text{Ca}+\text{Mg}) - (\text{Cl}+\text{P}+\text{S}).
\]

Despite dietary variations in these, adaption serves to maintain the pH of body fluids in the normal physiological range. Tissue pH may be altered to the extent that these compensatory mechanisms are not fully effective. Excretion of excess fixed ions requires water as a solvent, increasing water demand. Loading the horse with electrolytes temporarily increases accumulation of electrolytes and water in the large intestine (Slade, 1987) which may act as a reserve for extended work (Coenen, 1992; Meyer, 1992). The daily ileo-caecal flow of water and electrolytes per kg body weight is in the range of 100–140 ml water, 300–420 mg Na⁺, 50–70 mg K⁺ and 100–140 mg Cl⁻. Absorption along with water from the large intestine, during ingesta fermentation, is about 75–95% for Na⁺, over 90% for Cl⁻ and 30–55% for K⁺ (Meyer, 1992). These nutrients can revive tissue depleted of water, Cl⁻, Na⁺, K⁺ and Ca²⁺ through sweating (Rose et al. 1977), especially in horses that are severely dehydrated (Carlson et al. 1976) and are reluctant to drink.

High base excess in the diet (greater than 200 mg/kg) produces beneficial effects on pH, pCO₂ and HCO₃⁻ concentration of blood and on recovery of normal blood glucose (Stutz et al. 1992) and heart rate (Popplewell et al. 1993) after exercise. The benefit from transitory buffering appears to be achieved by feeding 3.5–4.5 h before anaerobic exercise.

Anaerobic exercise causes a rise in plasma K⁺ released from the contracting muscle fibre. If there is inadequate buffering within active muscle fibres, ATP may be less available and the Na–K pump will be inhibited, leading to poor re-uptake of K⁺ (Harris & Snow, 1988, 1992). The loss of intracellular K⁺ leads to an altered transmembrane potential that may contribute to fatigue during exercise. Oral NaHCO₃ reduces the rise in plasma NH₃ by reducing deamination of AMP and possibly by accelerating H⁺ removal (Greenhaff et al. 1990a, 1991). A positive effect of oral NaHCO₃ on performance seems to occur only during exercise of 2–3 min (Lawrence et al. 1987, 1990; Harkins & Kamerling, 1992). The optimum dose and time is about 0.4 g NaHCO₃/kg body weight (in 1 litre water), 2–4 h before work (Greenhaff et al. 1990b; Corn et al. 1993). However, a dose of Na equivalent to 20% of the body’s total exchangeable Na should increase plasma volume and could have an effect on sprint performance contrary to that expected of a buffer. Moreover, the value of the large intestine as a reservoir of Na may be modulated by acetate production (Argenzio et al. 1977), which varies with the timing and nature of the last meal. When supplemental NaHCO₃ and NaCl (1 g/kg body weight) were compared, the NaHCO₃ extended exercise to exhaustion on a treadmill and increased blood lactate, but compared with untreated horses gave poorer endurance, possibly from a higher fluid load (Lloyd et al. 1993). The response to oral NaHCO₃ is complicated, as alkalosis caused by the 1 g/kg dose led to hypercapnia and some hypoxaemia through respiratory compensation. Thus, the dose, method of administration and overall effects of NaHCO₃ and water will bear further examination.
A more enlightened approach to combating the rise in intracellular H$^+$ may be to alter the intracellular concentration of the imidazole dipeptide buffers carnosine (β-alanylhistidine) and its N2-methyl derivative, anserine. Carnosine contributes 30% of the buffering in equine skeletal muscle (Harris et al. 1991a), and in type IIB fibres (prominent in equine muscle) it may account for up to 50%, with a concentration of 188 mmol/kg muscle dry matter (Sewell et al. 1991a,b, 1992b).

**DIETARY ADDITIVES**

Si is a component of bone structure. Sodium zeolite at 18·6 g/kg total diet given to 6-month-old foals for 12 months increased plasma Si and speed and extended work time before leg injury after 18 months of age (Nielsen et al. 1993; Reynolds et al. 1993). Fatigue caused by lactic acid accumulation has been countered by the dietary inclusion of N,N-dimethylglycine (DMG) as a ‘metabolic enhancer’. Mature, conditioned, exercised horses supplemented with 1·6 mg DMG per kg body weight failed to show typical increases in blood lactic acid concentration (Moffitt et al. 1985).

L-Carnitine (β-hydroxy-γ-trimethylaminobutyric acid) is a conditionally essential nutrient, which is present in substantial amounts in diets composed of animal products, but low in feed sources derived from plants. Carnitine facilitates the transport of long-chain fatty acids across inner mitochondrial membranes and it may regulate acetyl-CoA:CoA, by buffering excess acetyl units during intense exercise (Carlin et al. 1990). Supplements of 10 g L-carnitine twice daily for 2 months have doubled plasma carnitine concentration of TB (Snow & Harris, 1989), but there was neither increased content in, nor loss of total carnitine from, middle gluteal muscle, associated with intense exercise (Foster et al. 1988; Foster & Harris, 1989, 1992). The effect of fat on the need for carnitine has not been examined and it is questionable whether the function of carnitine can be enhanced, except in those individuals that have low biosynthetic ability.

**FAT-SOLUBLE VITAMINS AND EXERCISE**

Schubert (1990, 1991) assessed the response of 247 TB to large supplements of vitamin E. Improved performance (significantly more wins and places than controls) was obtained when 1·0 g α-tocopherol was added to a daily ration providing 0·2 g vitamin E. Large doses, of this order, are known to increase muscle α-tocopherol (Ronéus et al. 1986). Yet McMeniman & Hintz (1992) held ponies on a diet containing 46 mg vitamin E/kg dry matter for 10 months but muscle vitamin E concentration remained at 8–10 μg/g. Exhaustive treadmill exercise which led to plasma lactic acid concentrations of 14 mmol/l, caused heart rates in excess of 180 bpm and increased plasma TBARS, evoked no increase in exercise fitness with additional vitamin E. Nevertheless, both pre- and postexercise plasma vitamin E concentrations were negatively correlated with the corresponding plasma TBARS concentration. Vitamin E supplementation (Ji et al. 1990) had little effect on erythrocytes other than to increase glutathione peroxidase activity 30 min after exercise that caused an 18-fold increase in blood lactate.

**WATER-SOLUBLE VITAMINS, TRACE ELEMENTS AND EXERCISE**

Boitin is the only water-soluble vitamin observed to give clinical responses with normal diets. Weak misshapen and crumbly hooves respond to supplementation (Comben et al. 1991).
A greater response has occurred with 15 mg/horse than with 7.5 mg/horse daily (Buffa et al. 1992). These amounts are considerably higher than should normally be adequate. No evidence is available that either dietary ascorbic acid or abnormal amounts of trace elements will improve performance.

CONCLUSIONS

Differences between cereal types and processing methods influence glucose:VFA in products of digestion and fermentation and, therefore, the fluid content of the hindgut. These effects are likely to influence performance.

Factors influencing the optimum form, feeding sequence and timing of a meal before exercise include: fluid content and weight of ingesta, fluid and electrolyte reserve in the large intestine, glycaemia, amino acidemia and insulinaemia, blood distribution and cardiac work.

High-fat (higher energy density) and high-protein diets both may benefit anaerobic and aerobic performance. Muscle glycogen stores, RQ, blood glucose and lactic acid concentrations are influenced and thermal load is decreased by fat supplements. Mechanisms are discussed.

A high dietary base excess, and NaHCO₃ given at a rate of 0.4 g/kg body weight 2–4 h before a 2–3 min sprint, may improve performance; however, larger doses of HCO₃⁻ may expand plasma volume to the detriment of sprint speed, changing tidal volume. A dietary base excess up to 300 meq/kg dry matter, with limited water access, could ameliorate fatigue in sprint events.

The author would like to thank Dr Roger Harris and Ms Catherine Orme, of the Animal Health Trust Newmarket, for useful discussions on the subject.

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