Optimal Nutrition for Athletic Performance, with Emphasis on Fat Adaptation in Dogs and Horses[1,2]

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ABSTRACT Four mathematical approaches are proposed to determine optimal ranges of nutrients for specified purposes. For exercise, the diet must provide optimal mixtures of fuels, also optimal amounts of nutrients conducive to a sound structure, a desired power/weight ratio, a water-electrolyte system that resists dehydration and buffers hydrogen ions, a tolerance to the cumulative stress of repetitive competition and tractable attitude. The nutritional strategy of carbohydrate loading risks a variety of abnormalities in dogs and horses. An alternative strategy of fat adaptation (the combination of fat feeding and training) was found to improve aerobic performance in dogs and horses and to spare glycogen utilization and reduce lactate accumulation. Surprisingly, improved anaerobic performance has also been confirmed in fat-adapted horses that have been sprint trained. Fat adaptation increased the blood lactate responses to incremental tests and repeated sprints. Blood lactate accumulation during repeated sprints was affected synergistically by the combination of fat adaptation and sodium bicarbonate supplementation. Fat adaptation in horses appears to facilitate metabolic regulation to achieve power needs, with glycolysis decreasing during aerobic work but increasing during anaerobic work and with blood lactate changes following accordingly. Interactions between fat adaptation and dietary cation–anion balance need further investigation. J. Nutr. 124: 2745S–2753S, 1994.

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• *dogs* • *horses* • *optimal nutrient ranges* • *exercise* • *stamina* • *sprinting* • *heat tolerance*

Certain breeds of dogs and horses have been selected for athletic performance. Study of their nutrition has direct application in their use for sport and recreation. These animals also serve as models for research, because of the extreme nature of several adaptations to strenuous exercise. Compared with other species, dogs and horses have greater aerobic capacities and oxidative enzyme activities and greater increases in hematocrit, heart rate, cardiac output and blood flow in skeletal muscle during strenuous exercise (Snow 1985).

Horses and dogs also are good models for research because they are tractable and readily learn behavior routines. They instinctively perform to fatigue with humane positive encouragement; hence, unlike rodents, they need no electric grids on their treadmills. Horses and dogs are large enough to allow multiple sequential sampling of blood and other tissues, which facilitates study of the timecourse of metabolic changes during exercise and recovery. These species are not susceptible to atheromatous heart disease, a condition that has deterred chronic studies of fat-rich diets in human athletes. Instead, horses and dogs experience digestive and metabolic limitations to high grain diets, which reflect their evolution on diets relatively low in soluble carbohydrates (Clarke et al. 1990, Kronfeld 1973, Sprouse et al. 1987, White et al. 1993).

In contrast to dogs and horses, human athletes adapt readily to high intakes of soluble carbohydrates, >50% of metabolizable energy (ME). Common nutritional recommendations for human athletes reduce simply to observance of the recommended dietary allowances (RDAs) for body weight (Food and Nutrition Board 1989), with carbohydrate loading before an event and supplementation of carbohydrates and water as required during and after prolonged strenuous exercise (Costill 1985, Williams 1993).

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Early studies of muscle fatigue differentiated between central (mental and emotional) and peripheral (neural and muscular) components; both appear influenced favorably in human athletes by ingestion of soluble carbohydrates. Muscle glycogen depletion and blood lactate accumulation are delayed in humans by the provision of simple carbohydrates as drinks or candy bars (Williams 1993). Carbohydrate ingestion also maintains blood concentrations of glucose and the ratio of branched-chain amino acids to free tryptophan, thereby delaying central fatigue (Davis 1993).

Feeding regimens for canine and equine athletes also used to be simple. Dietary changes for increasing work that were intended to increase nutrient density also improved digestibility, thereby reducing residual bulk in the large bowel. For more work, horses were simply fed less hay (fiber) and more grain (soluble carbohydrate), whereas most dogs were fed less grain and more meat (protein and fat).

The exception among dogs has been the racing Greyhound (Kohnke 1983). The 30- to 35-kg sprinter has traditionally been fed 700–800 g/d lean meat as a highly digestible source of food energy, 150–200 g/d kibble and up to 100 g/d of further supplements, most secret and many bizarre. The Greyhound remains in the grip of this tradition, but the nutrition of race horses and working dogs, especially racing sled dogs, has been an active field of research for the last two decades (Grandjean and Paragon 1993, Hintz 1994, Kronfeld and Downey 1981, Meyer 1987).

Attention has been given in racing dogs and horses not only to fuels (mainly fats vs. carbohydrates) but also to the influence of feeding regimens on other aspects:

- Body weight (including bulk in intestines), composition (especially unnecessary fat and water), condition (fat/lean ratio) and resistance to mechanical break down;
- Respiratory capacity (lowering carbon dioxide production by oxidizing fatty acids and, perhaps, by lowering body weight);
- Acid-base status (influenced by fats, buffers and the cation–anion balance of the diet);
- Coping with stress (aided by protein and ascorbic acid);
- Ergogenic supplements, prerace intravenous "jugs" (more potentially lethal, in our view, than potentially ergogenic);
- Practical feeding plans during conditioning and for an event.

This review illustrates certain lines of thinking that are motivating research in this field, with emphasis on conceptual approaches to optimal ranges of nutrients for growth, physical soundness and athletic performance and to nutritional strategies for stamina, sprinting and performing in ambient heat, with emphasis on fat adaptation.

Optimal ranges of nutrients

Practicing nutritionists usually recommend diets (feed composition) and allow trainers to determine rations (daily intakes) of racing dogs and horses. The diets have specified nutrient goals, set within tentative optimal or desired ranges. These goals and ranges express personal experiences and judgments as well as relevant nutritional science, which is usually inadequate in relation to athletic performance. Because strenuous exercise involves damage to tissues, which then undergo repair and remodeling, the common guides are the nutrient requirements for growth rather than maintenance.

The dog and the horse have nutrient requirements specified by the National Research Council (NRC) that are mean minimal values for specified purposes (NRC 1985, NRC 1989). These minimums are a far cry from human RDAs (Food and Nutrition Board 1989), which are two standard deviations above respective mean minimums. A nutrient profile for dog foods has been proposed for regulatory purposes by the Association of American Feed Control Officials (Dzanis 1994), but it has no systematic basis, like the RDA, and does not purport to represent an optimal diet for a racing dog.

Mathematical models have been used to define optimal ranges of nutrients for specified purposes. The following models reveal the kind of data needed to derive an optimal range. One was proposed in 1912 as *Bertrand's rule* (Mertz 1981). With increasing nutrient intake (or dietary content), a measure of desired performance rises to a plateau and then falls (**Fig. 1**). The plateau or optimal range is broad because the measure is not precise, a population has variation and an individual has homeostatic regulation. A paradigm, that the optimal range becomes more narrow under more demanding conditions, has been illustrated for dietary protein in dogs (Kronfeld and Downey 1981); the range is broad for maintenance, less broad for growth and narrow for stress in a racing sled dog (Fig. 1).

The Bertrand model may be approximated by fitting a parabolic curve. Peak nitrogen retention was found at 43% protein (ME basis) in growing beagles (Kronfeld and Banta 1989), and peak weight gain was found at 41% protein in German shorthair pointers (Dumon et al. 1994). In contrast, traditional statistical approaches to determination of the minimum protein requirement for growth indicate 10.5% ME for dogs (NRC 1985).

In horses, an optimal range has been defined for vitamin A during growth (Donoghue et al. 1981). Parabolic curves fitting eight variables peaked at 2–10 times the NRC minimum of 2000 IU/kg dry matter (DM) vitamin A for growth and work (NRC 1989). Bone lesions were found histologically at 1 and 100 times the NRC minimum for vitamin A.

A high muscle glycogen concentration has promoted speed over 600 and 1600 m in horses (Harkins et al.

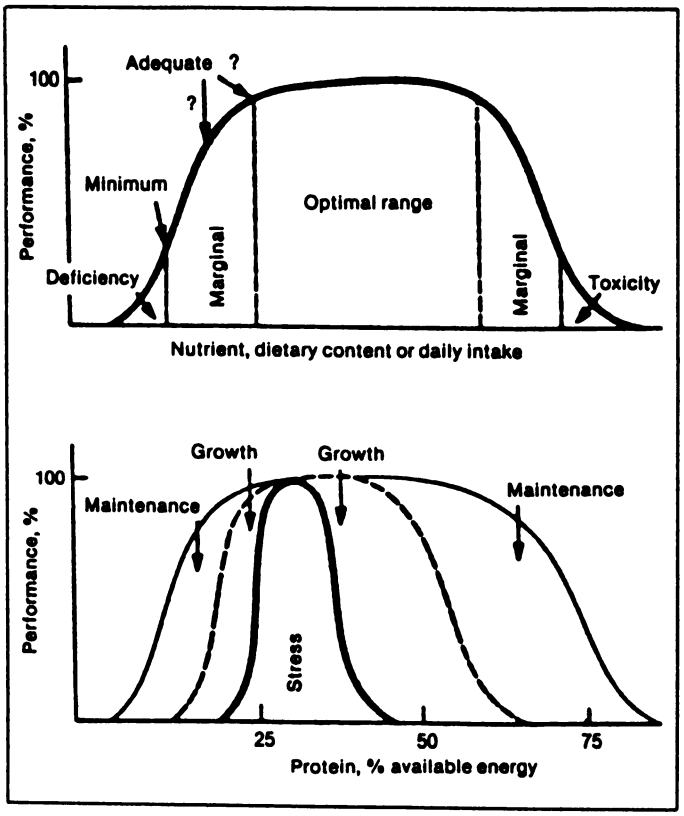

FIGURE 1 Influence of dietary content or nutrient intake on a measure of desired performance rises to a plateau, the optimal range, and then declines (top). The optimal range is broad under undemanding conditions, such as maintenance, but becomes narrower under more demanding conditions, such as the stress of competitive athletics (bottom). Modified with permission from Kronfeld, D. S. (1989) vitamin & Mineral Supplementation for Dogs and Cats, Veterinary Practice Publishing Co., Santa Barbara, CA.

1992, Oldham et al. 1990). Muscle glycogen was measured in horses trained for 3 wk and fed 4, 8, 12 or 16% fat (Hambleton et al. 1980). Fitting a parabolic curve indicates a maximum value for muscle glycogen at 11.6% dietary fat (**Fig. 2**).

Upper and lower limits of an optimal range may also be set by undesired indicators, such as clinical signs or lesions pertaining to deficiency or intoxication, shown here as two frequency distributions in a population of dogs (**Fig. 3**). This hypothetical example, which relates dietary calcium to bone development during growth, is based mainly on studies on Great Dane pups (Hazewinkle et al. 1985, Hazewinkle et al. 1991). Osteoporosis, including fractures, was observed in pups fed a diet containing calcium at 0.55% DM, which is close to the NRC minimum of 0.59% calcium for growth (NRC 1985). Osteochondrosis developed in pups fed a diet containing 3.3% calcium.

Following the example of the human RDAs, one might apply the coefficient of variation (CV) for energy requirement to define the RDA of a nutrient when the CV for that nutrient is unknown. The CV for energy

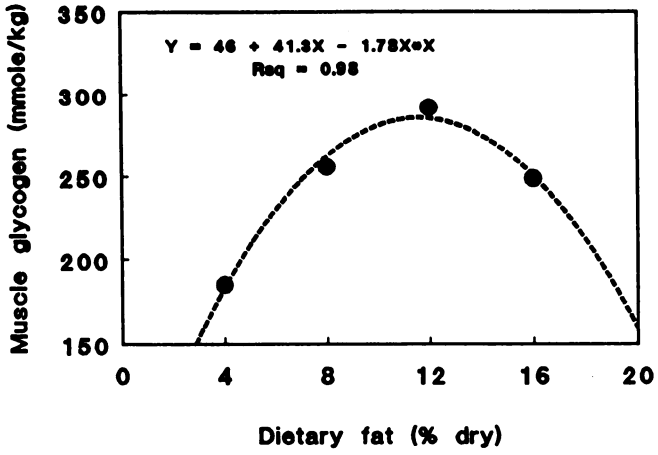

FIGURE 2 Influence of dietary fat on muscle glycogen content in aerobically trained horses. The two-term polynomial equation defines the dietary fat content, 11.6%, yielding the maximum muscle glycogen content. An optimal range giving 95% of peak muscle glycogen (280 mmol/kg dry tissue) would be 10–13%.

requirement is ~15% for adult humans and ~20% for dogs (Kendall et al. 1983). Thus, to cover 98% of the population, twice the CV is added to the mean minimum, determined for a deficiency sign, to yield the RDA for a human, and one might do the same for a dog, using the larger CV for this species. Similarly, twice the CV might be subtracted from the mean minimum for an intoxication. The result would be an optimal range for 98% of the population.

In our case, the optimal range for dietary calcium for 98% of Great Danes would be 0.77–1.98% DM. A narrower range, using three times the CV to cover 99.9% of the population, would be 0.9–1.3% calcium.

Undesired performance indices are used for the lower and upper limits of the optimal range of dietary

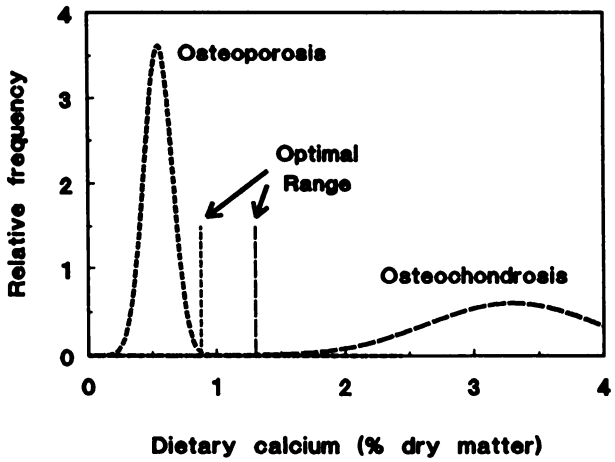

FIGURE 3 Influences of two undesired effects that define the lower and upper limits of an optimal range of dietary calcium is illustrated by bone abnormalities in growing Great Danes fed various levels. The frequency distribution in the population approximates the normal distribution.

protein for the racing sled dog, shown here as cumulative frequencies (**Fig. 4**). The lower limit is reasonably well defined by the progressive development of stress anemia below 32% ME protein (Kronfeld et al. 1989). The upper limit is described less precisely by a presumed progressive loss of stamina as protein displaces dietary fat, which promotes stamina (Downey et al. 1980). In this model, 100% of dogs will walk when fed a diet containing 15% protein and 5% fat. Those few sled dogs that become competitive athletes are sustained better by a diet providing 30% protein and 60% fat.

Optimal ranges of nutrients are not well established for racing dogs and horses; none are better than the above examples. Thus, opportunity abounds for advances in this field.

Strategies for stamina

Carbohydrate loading. The combined training and dieting regimen of carbohydrate loading was introduced by Swedish physiologists (Bergstrom et al. 1967). Initially, it was designed to maximize muscle glycogen content before an endurance event by depletion and then overcompensatory repletion. Desired muscle glycogen levels are reached through conditioning in most horses and many human athletes without the depletion phase, which has been deemphasized in contemporary carbohydrate loading. Moreover, glycogen conservation and repletion is achieved by carbohydrate ingestion during and after exertion (Costill 1985).

Serious side effects in humans, such as lesions of heart muscle, skeletal muscle and kidney, were apparently infrequent and did little to discourage the acceptance of carbohydrate loading. More attention was given to side effects in dogs and horses, species that did not evolve on high grain diets. Attempts at carbohydrate loading led to tying up, a mild form of exertional rhabdomyolysis in racing sled dogs (Kronfeld 1973). Increasing soluble carbohydrate in the diet of the horse appears to excede the hydrolytic capacity of the small intestine and lead to excessive rapid fermentation in the large bowel, with several attendent disorders, such as founder and colic (Clarke et al. 1990, Sprouse et al. 1987, White et al. 1993). As an alternative to carbohydrate loading, studies were made of increasing dietary fat for endurance horses and sled dogs.

Fat adaptation. An alternative nutritional strategy for stamina is fat adaptation, training animals fed a high fat diet with the intention of enhancing fatty acid oxidation, hence sparing utilization of muscle glycogen and blood glucose (Kronfeld and Downey 1981). The impetus was a clinical experience with tying up in a champion team of racing Alaskan Huskies that was subjected to carbohydrate loading (Kronfeld

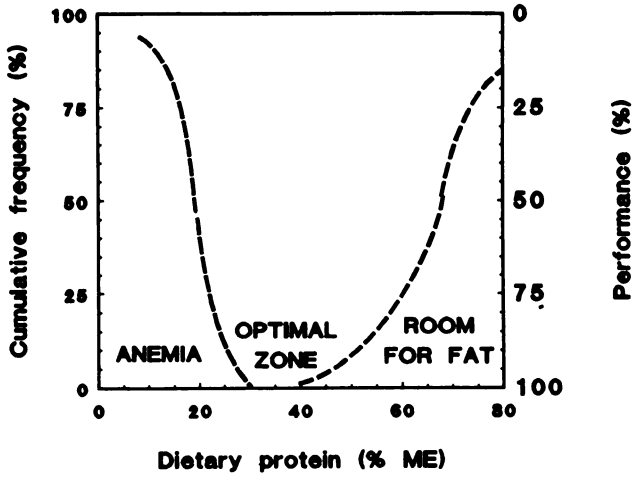

FIGURE 4 Influences of two undesired effects on the optimal range is illustrated as cumulative frequencies, in contrast to the relative frequencies in Figure 3. The lower limit is defined well by the development of anemia in highly stressed sled dogs. The upper limit is created by the need for as much fat as possible in the diet to promote stamina.

1973); their performance improved step-wise as carbohydrate was replaced by protein and fat.

Field studies on this team suggested that fatty acid utilization during a race was increased by training on a high fat diet; the better performing dogs, selected by the trainer/driver at the start of the study, had higher plasma free fatty acid concentrations following a hard run (Hammel et al. 1977). A treadmill study on Beagles fed four diets showed that fatigue time was related positively to dietary fat and negatively to dietary carbohydrate (Downey et al. 1980).

Field trials on endurance horses suggested that fat adaptation sustained blood glucose concentration and reduced dehydration (Hintz et al. 1978, Slade et al. 1975). A treadmill study demonstrated muscle glycogen sparing in horses trained aerobically and fed 10% added fat (Griewe et al. 1989).

Metabolic regulation. Carbohydrate loading promotes glycogen synthesis. Massive carbohydrate ingestion releases insulin and increases muscle cell uptake of glucose to form glucose-6-phosphate, the allosteric activator of glycogen synthase (Ivy et al. 1983). Exhaustive exercise also may activate glycogen synthase (Costill 1985).

Fat adaptation spares glycogen utilization. Training dogs fed a high fat diet was proposed to increase fatty acid oxidation and hence to spare the use of glucose and glycogen (Kronfeld and Downey 1981). Adaptive increases in fatty acid oxidation had been shown previously to develop during training. It was associated with an increase in the number of mitochondria in trained muscles and with a decline in utilization of muscle glycogen and blood glucose (Holloszy 1990). The observations on dogs suggested that the increase in fatty acid oxidation by training was augmented by

a high fat diet (Kronfeld and Downey 1981). Subsequently, muscle glycogen sparing during aerobic exercise was found to be increased by training while consuming higher fat diets in humans, rats and horses (Griewe et al. 1989, Phinney et al. 1983).

Numerous metabolic controls integrate the utilization of fatty acids and glucose/glycogen during exercise. Suppression of glucose and glycogen utilization has been attributed to inhibition of hexokinase and phosphorylase by glucose-6-phosphate, which accumulates when phosphofructokinase is inhibited by citrate, produced by fatty acid oxidation (Newsholme 1983).

The relatively slow transport of fatty acids into cells and mitochondria was proposed to be limiting for the rate of ATP generation from fatty acid oxidation (McGilvery 1973). Thus, power generated from fat as a fuel was thought to be about half that from using glucose aerobically, which, in turn, was about half that of using glucose anaerobically. Fat was regarded as a likely fuel for long-distance low intensity work, probably at rates <25% of maximal oxygen uptake (Newsholme 1983).

Strategies for sprinting

Fat adaptation and anaerobic work. Surprizing, in view of the low power output or rate of ATP generation from fatty acid oxidation (McGilvery 1973), were reports that fat-supplemented diets improved the performance of quarter horses performing repeated turns (cutting) and thoroughbreds sprinting 600 or 1600 m (Harkins et al. 1992, Oldham et al. 1990, Webb et al. 1987). Muscle glycogen content was elevated in horses fed 10–12% fat (Fig. 2) compared with 2 or 16% fat (Hambleton et al. 1980, Oldham et al. 1990, Harkins et al. 1992). Muscle glycogen depletion and blood lactate accumulation during four repeated 600-m sprints were increased by a 10% added fat diet (Oldham et al. 1990), but muscle glycogen depletion during a 1600-m run was not affected by 12% added fat in another study (Harkins et al. 1992).

In our laboratory, Arabian horses were fat adapted by sprint training for 11 wk while fed a 10% added corn oil diet. They had increased lactate thresholds and higher peak blood lactate concentrations during incremental exercise tests on a high speed treadmill (Custalow et al. 1993). Moreover, during a series of repeated sprints, blood lactates were consistently higher in fat-supplemented horses than in controls (Ferrante et al. 1993). In contrast, blood lactate was lower after an aerobic exercise test in fat-adapted horses that had been aerobically trained (Griewe et al. 1989).

These different blood lactate responses suggest that fat adaptation involving sprint training of horses might diminish glucose oxidation predominently at the level of pyruvate and the pyruvate dehydrogenase (PDH) complex rather than higher up the glycolytic chain at glucose-6-phosphate, hexokinase and phosphorylase, as proposed for aerobic training. High resting muscle glycogen, glycogen sparing, and high blood lactates after sprinting have been observed only in horses subjected to sprint training while fed 10–12% fat diets, so the role of PDH inhibition may be more important in this species than in others.

The PDH complex. Conversion of pyruvate to acetyl-CoA is catalyzed by PDH. Reduced activity of PDH favors the accumulation of lactate. It may come about in several ways during a period of increased fatty acid oxidation. One is inhibition by its reaction products, acetyl-CoA and NADH. Another is indirect: higher ratios of acetyl-CoA/CoA and NADH/NAD^+ activate PDH kinase, which converts PDH to its inactive phosphorylated form (Kerbey et al. 1976).

Acetyl-CoA accumulation during fatty acid oxidation is minimized by the storage of acetyl groups as acetylcarnitine (Constantin-Teodosiu et al. 1991). Acetylcarnitine content of muscle increases during intense exercises in horses (Harris and Foster 1990). High rates of fatty acid oxidation in fat-adapted animals may reduce the availability of carnitine for the storage of acetyl groups, thereby contributing to inhibition of the PDH complex and to the accumulation of lactate.

Vitamin C supplementation has appeared to be beneficial in fat-adapted sled dogs (Donoghue et al. 1987, Donoghue et al. 1993). One possibility is that fat adaptation increases the demand for carnitine, hence ascorbic acid, which is required by a hydrolase in carnitine synthesis. Low blood or plasma ascorbate has been observed not only in sled dogs during the racing season but also overtrained horses (Jaesckhe and Kellner 1978). The possible influences of supplementation with carnitine or ascorbate on lactate accumulation during intense exercise in fat-adapted horses deserves investigation.

Power/weight. For a given total power output, if low power fat oxidation diminishes medium power glucose oxidation, then an even higher power system, such as glycolysis, will need to be recruited. In rough terms, three power units of fatty acid oxidation and one unit of glycolysis may be equivalent to four units of glucose oxidation (McGilvery 1973). In this view, the ability of a fat-adapted animal to sprint would require diminished glucose and glycogen utilization at the pyruvate level rather than at the level of glucose-6-phosphate.

For a given power output, regardless of the mixtures of sources, only one variable can account for a faster speed—lower body weight. Thus, one factor contributing to faster speeds recorded by fat-adapted thoroughbreds over sprint distances of 600 and 1600 m (Harkins et al. 1992, Oldham et al. 1990) is a better power/weight ratio: less weight in the large bowel associated with less undigested fiber, which retains about twice its weight of water. In contrast, a water reservoir

in the large bowel may benefit an endurance horses (Meyer 1987).

Creatine, phosphate and dimethylglycine. An abstract has described substantial improvements in 300 and 1000 m running times of human athletes subjected to creatine loading (Harris et al. 1993). Megadosage of creatine monohydrate has been found to increase the muscle concentration of creatine and phosphocreatine in humans (Harris et al. 1992). Phosphocreatine acts in two ways: as a donor of high energy phosphate bonds to convert ADP to ATP and as a buffer.

Creatine is not regarded as an essential nutrient, because synthesis from arginine and glycine is adequate for most demands. Also, creatine monohydrate breaks down to creatinine during cooking and in acidic solution, such as in the stomach.

Creatine loading has been compared with carbohydrate loading (Harris et al. 1992). Oral intakes of ~1–2 g/d creatine monohydrate, equivalent to 200–400 g raw meat or fish, are comparable with likely intakes of human ancestors during the social carnivore phase of evolution. Such intakes may require several weeks to raise blood and muscle levels of creatine (R.C. Harris, personal communication). Over shorter periods, larger doses of creatine monohydrate, 20–30 g/d, equivalent to 4–6 kg raw steak a day, are needed (Harris et al. 1992). Given its ergogenic effect, appropriate authorities will have to decide whether creatine monohydrate in specified dosages is to be regarded as a nutrient or a drug.

Phosphate loading also has been proposed to promote creatine phosphate synthesis. In one study, six athletes consumed sodium phosphate or a placebo, 1 g every 4 or 5 h for 4 d (Kreider et al. 1992). Phosphate loading increased maximal oxidative capacity, increased time to anaerobic threshold and improved endurance performance. Phosphate loading may be more hazardous in horses than in most species, because high phosphate intakes reduce calcium retention in the horse (Schryver et al. 1971).

Dimethyglycine (DMG) has been suggested to increase creatine phosphate and, along with dichloroacetate (another component of pangamic acid or so-called vitamin B-15), to activate the PDH complex and reduce lactate production during strenuous exercise. A field study on 20 racing Greyhounds showed decreased times over 300 or 500 m in successive runs with both agents (Gannon and Kendall 1982). This result could have been confounded by training, track or season. On the other hand, these dogs perform with extraordinary consistency and every dog improved ($P < 0.0000001$ by the Wilcoxon sign test). The improvement came after the first 100 m that is, when the PDH complex could be assuming a dominant role in regulating power output.

Another field study on standardbred horses was less convincing (Levine et al. 1982). Mean blood lactate concentrations tended to be lower after training runs when horses were given DMG. Two treadmill trials of DMG have been conducted on horses. Blood lactate accumulation was decreased in one (Moffitt et al. 1985) but not the other (Rose et al. 1989).

Buffers. The energy required for muscle contraction is provided by oxidation, which produces a weak acid (CO_2, pK 6.1), and by glycolysis, which produces a strong acid (lactic, pK 3.7). Accumulations of lactic acid and, more generally, hydrogen ions or protons contribute to metabolic fatigue (Mainwood and Renaud 1985), which might be delayed by the provision of buffers.

Historically, oral supplementation with buffers has had inconsistent effects on athletic performance, which suggests that buffers are effective only under limited conditions. A metaanalysis of sodium bicarbonate administration to human athletes reviewed 19 studies that showed a positive effect and 16 that showed no effect (Matson and Tran 1993). In five studies of sodium bicarbonate supplementation in a total of 66 horses, no improvement was found in three (Greenhaff et al. 1991, Harkins and Kamerling 1992, Lawrence et al. 1987) and a trend ($0.05 < P < 0.10$) toward improvement in two (Kelso et al. 1987, Lawrence et al. 1990).

Statistically significant results have been found in another two studies using 1 g sodium bicarbonate/kg body weight, but the results are conflicting (Hinchcliff et al. 1991, Lloyd et al. 1993). In both studies, sodium chloride was used as an isoosmotic control. A preliminary report of an experiment on four horses subjected to an incremental exercise test indicated that horses supplemented with sodium bicarbonate had increased ($P = 0.046$) running times compared with plain water but not sodium chloride (Hinchcliff et al. 1991). In the other study, horses were warmed up then run at 110% $VO_{2\,max}$ to fatigue (Lloyd et al. 1993). Horses given sodium bicarbonate had decreased ($P < 0.05$) running times than when given plain water, a surprising result, and increased ($P < 0.02$) running times compared with sodium chloride supplementation. This last result is consistent with a greater number of studies in humans, in which sodium chloride and sodium bicarbonate exert opposing effects on blood pH and athletic performance (Matson and Tran 1993).

In the only one of the equine bicarbonate studies that presented data on individual horses, running time was improved in eight but was worse in six when supplemented with 0.3 g/kg sodium bicarbonate (Lawrence et al. 1990). A paired t test indicates $P = 0.097$ (our statistic), supporting the report of $P < 0.10$. The distribution of the responses was far removed from a normal curve, however, so a nonparametric test would be preferable; the Wilcoxon sign test indicates $P = 0.79$ (our statistic).

Sodium bicarbonate may be included in the diet to affect the cation–anion balance. Horses consuming

highly cationic diets, supplemented with sodium bicarbonate or potassium citrate, tended to have faster ($P < 0.10$) times in a 1.64-km standard exercise test and recovered more quickly than those fed a highly anionic diet, supplemented with chlorides (Popplewill et al. 1993).

Sodium bicarbonate is a registered feed ingredient, and its use to affect dietary cation–anion balance, and hence performance, is in keeping with this classification. Electrolytes (mixtures of cations and anions) are commonly supplemented in meals of grain concentrates the night before and the morning of endurance events to influence hydration. They also are supplemented as pastes or in applesauce or syrup before, during or after endurance events to compensate for losses and hence maintain acid-base balance. On the other hand, sodium bicarbonate given by nasogastric tube (as a "milkshake") or orally via syringe is prohibited the morning of a race in certain juridictions, as if sodium bicarbonate used in this way becomes a drug, with an ergogenic intention not supported by seven of eight comparative trials, as described above. This prohibition may be contrary to the best interests of the horse, especially when dietary control of its cation–anion balance is compromised by hot ambient conditions.

Strategies for ambient heat

High ambient heat and humidity affect athletic performance over middle and long distances. Relevant nutritional strategies involve energy density, fat adaptation, cations and anions and hydration. Hydration is influenced mainly by dietary sodium, fiber and, of course, water itself (Meyer 1987).

Energy partition studies on fat-adapted thoroughbreds have shown that heat production was 52% of gross energy, compared with 59% in controls (Scott et al. 1993). The authors concluded that substituting fat for soluble carbohydrate lowered heat of fermentation, hence the thermal load. The data, however, showed that digestible energy was 77 and 79% of gross energy in control and fat-adapted horses, respectively. A larger difference was resided in the partition of metabolizable energy into heat increment and net energy, which was 34:66 and 23:77 for control and fat-adapted horses, respectively. In effect, fat adaptation improved the efficiency of utilization of metabolizable energy.

Fat adaptation has abated the increase in venous blood P_{CO_2} in Arabian horses during repeated sprints (Ferrante et al. 1993). This effect reflects in part the low respiratory quotient of fatty acid oxidation (RQ = 0.70) and hence a lower production of carbon dioxide for a given oxygen uptake (or work done) than would occur with glucose oxidation (RQ = 1.00). Thus, fat adaptation may reduce breathing effort and confer an advantage on horses competing in the heat or suffering from lung damage, such as congestion, emphysema or hemorrhage.

Diets containing abundant chloride are needed to compensate for sweating under hot conditions but may have an adverse effect on exercise. A compromise may be to supplement such a high chloride diet with sodium bicarbonate for one or two meals before a race, in the same manner as electrolytes are used before an endurance event. In addition, supplementary sodium bicarbonate may attenuate digestive and metabolic effects of a high soluble carbohydrate diet (Ferrante et al. 1992) and hence reduce the risk of laminitis and colic (Clarke et al. 1990, Sprouse et al. 1987, White et al. 1993) and exertional rhabdomyolysis (Robb and Kronfeld 1986), which is more common under hot conditions. Thus, when hot conditions require a high chloride diet, the use of compensatory bicarbonate before an event is justified on the basis of nutritional physiology.

An unexpected finding in our laboratory was the apparently synergistic effect of the combination of sodium bicarbonate supplementation and fat adaptation on blood lactate accumulation during repeated sprints (Ferrante et al. 1993). Our current experiments are examining further the interactions of fat adaptation and cation–anion balances on exercise in horses.

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