When talented, motivated and highly trained athletes meet for competition the margin between victory and defeat is usually small. When everything else is equal, nutrition can make the difference between winning and losing. Although the primary concern of many athletes is to supplement the diet with protein, vitamins and minerals, and a range of more exotic compounds, key dietary issues are often neglected. Athletes must establish their nutritional goals, and must also be able to translate them into dietary strategies that will meet these goals. Athletes are often concerned with dietary manipulations in the period around competition, but the main role of nutrition may be to support consistent intensive training which will lead to improved performance. Meeting energy demand and maintaining body mass and body fat at appropriate levels are key goals. An adequate intake of carbohydrate is crucial for maintaining muscle glycogen stores during hard training, but the types of food and the timing of intake are also important. Protein ingestion may stimulate muscle protein synthesis in the post-exercise period, promoting the process of adaptation in the muscles. Restoration of fluid and electrolyte balance after exercise is essential. If energy intake is high and a varied diet is consumed, supplementation of the diet with vitamins and minerals is not warranted, unless a specific deficiency is identified. Specific strategies before competition may be necessary, but this requirement depends on the demands of the sport. Generally, it is important to ensure high pre-competition glycogen stores and to maintain fluid balance. There is limited evidence to support the use of dietary supplements, but some, including perhaps creatine and caffeine, may be beneficial.

The recreational athlete can improve performance by various strategies, the most obvious of which is to increase the training load. At the highest levels of competitive sport, however, there are fewer options open. All the competitors are likely to be training at a level close to the limit of what can be sustained, and the margin between victory and defeat is often vanishingly small. In the 1989 Tour de France, for example, the winning margin was 8 s, after more than 87 h of racing over a total distance of 3285 km. It is hardly surprising, therefore, that athletes seek to explore all opportunities for improving performance. Considering too the enormous financial rewards that are now open to the successful athlete, the attraction of any means of performance enhancement, however unlikely, is also obvious.

Athletes are often concerned to seek an advantage by employing novel nutritional strategies before or during competition, but nutritional interventions may have their biggest impact on performance by supporting consistent intensive training and thus promoting the physiological and biochemical adaptations that will, in turn, lead to improved performance. Nutritional goals associated with training should include:

- maintaining energy supply to the working muscles and other tissues;
- promoting tissue adaptation, growth and repair;
- promoting immune function and resistance to illness and infection;
- rehearsal and refinement of competition strategies.

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In competition some of the goals will be the same, but the requirements and the strategies adopted will vary greatly depending on the nature of the event. A sprinter aiming to run 100 m inside 10 s faces very different problems from the Tour de France cyclist who faces the challenge of racing over a total distance that may exceed 4000 km spread over about 22 d. Challenges and opportunities for nutritional intervention immediately before and during competition include:

- enhancing energy supply to delay the onset of fatigue;
- minimising the negative effects of dehydration;
- central nervous system effects.

The task for the nutritionist involved in competitive sport is to identify the nutritional goals of the athlete and to translate them into dietary strategies that take account of individual circumstances. The same general principles apply to all sports, but the strategies adopted may be very different because of the different demands of training and competition. The present review will focus primarily on issues relating to nutritional support for training, and will provide only a brief overview of issues relating to competition.

**Nutrition for training**

Any form of physical activity will increase the rate of energy expenditure, and unless this increase is compensated for by a reduction in energy expenditure elsewhere in the day, energy intake must be increased accordingly. The energy demands of sportsmen and women in training vary greatly, depending primarily on body mass and on the training load. There may be times when the athlete must be in negative energy balance to reduce body mass (for example, the individual who competes in a weight-category sport) or body fat levels (for example, the team games player returning to training after the off-season). Hard physical exercise on an energy-restricted diet can have a number of negative consequences, including an increased rate of protein catabolism (Lemon, 1991) and an increased release of cortisol and catecholamines which can lead to compromised immune function (Pedersen et al. 2000). In sports where there is an advantage in maintaining a low body mass, and especially a low body fat content, individuals may follow a pattern of restrained eating for very prolonged periods. There is a clear division between sports where performers have a low body fat but the energy turnover is high, such as marathon running, and those where the energy turnover is low, including perhaps gymnastics and the jockey in horse racing; it is important to recognise that not all athletes have high levels of energy expenditure. An inverse relationship between energy intake and body fat content has been demonstrated in runners (Maughan & Piehl Aulin, 1997). This apparently contradictory relationship was the result of a positive association between energy intake and training volume (expressed as distance run per week) and a negative relationship between body fat content and training volume.

**Macronutrient needs**

The energy requirements of training are largely met by oxidation of fat and carbohydrate. Protein typically constitutes about 12–15 % of the total energy intake, but oxidation of the C skeletons of protein makes only a small contribution to energy supply during prolonged exercise. This contribution normally amounts to ≤ 5 % of the total energy demand, but may be more than this level if the muscle glycogen stores are low (Lemon, 1991). The higher the intensity of exercise, the greater the total energy demand, and the greater the reliance on carbohydrate as a fuel (Coyle, 1991). At an exercise intensity corresponding to about 50 % maximum O2 uptake, approximately two-thirds of the total energy requirement is met by fat oxidation, with carbohydrate oxidation supplying about one-third. At about 75 % maximum O2 uptake, which is closer to the typical training intensity in many endurance sports, the total energy expenditure is increased, and carbohydrate, especially muscle glycogen, is now the major fuel. Recently, it was shown that the response is similar in men and in women (Romijn et al. 2000).

The body stores of carbohydrate are small (Table 1) relative to the amount that can be used during exercise. In prolonged hard exercise carbohydrate can be oxidised at a rate of 3–4 g/min by well-trained athletes; if this level is sustained for ≥ 2 h a very large proportion of the total body carbohydrate content will be exhausted. In intense exercise anaerobic metabolism provides most of the energy demand, and a large proportion of the muscle glycogen is rapidly converted to lactate by anaerobic glycolysis. In a single 6 s sprint the muscle glycogen content may fall by as much as 16 % of its initial value (Boobis, 1987). Nevill et al. (1989) reported a 32 % decrease in muscle glycogen content (from 317 to 215 mmol/kg) after a single 30 s treadmill sprint. Many team games players will include multiple short sprints in their training programmes, but these individuals often do not consider that glycogen depletion is an important factor in their training.

During each strenuous training session, depletion of the glycogen stores in the exercising muscles takes place. In prolonged exercise a substantial part of the liver glycogen reserve will also be mobilised; this process has been demonstrated by both direct biopsy measurements (Hultman, 1981) and by tracer methodologies (Romijn et al. 1993). If these carbohydrate stores are not replenished before the next exercise bout, training intensity must be reduced, leading to

| Table 1. Normal body stores of fat and carbohydrate in a typical 70 kg male athlete and a typical 60 kg female athlete |
|-------------------------------------------------|---------------------|---------------------|
| Male                                           | Female              |
| Carbohydrate stores                            |                     |
| Liver glycogen (g)                             | 90                  | 70                  |
| Muscle glycogen (g)                            | 400                 | 300                 |
| Fat stores*                                    |                     |
| Intramuscular (g)                              | 500                 | 500                 |
| Adipose tissue (kg)                            | 7–10                | 9–20                |

* The body fat content especially can vary greatly, from as little as about 3 % body weight in very lean male individuals to ≥ 50 % in the obese (including the sumo wrestler, one of the few sports where obesity is an advantage).
corresponding decrements in the training response. Any athlete training hard on a daily basis can readily observe this response; if a low-carbohydrate diet, consisting mostly of fat and protein, is consumed after training on 1 d, it will be difficult to repeat the same training load on the following day. A further disadvantage of training on a low-carbohydrate diet is the potential for an increased risk of injury and of increased susceptibility to minor infectious illness (Nieman & Pedersen, 1999).

Recovery of the muscle and liver glycogen stores after exercise is a rather slow process, and complete recovery may not be achieved until 24–48 h after the end of exercise (Piehl, 1974). The rate of glycogen resynthesis after exercise is determined largely by the amount of carbohydrate supplied by the diet (Ivy, 2000), and the amount of carbohydrate consumed is of far greater importance for this process than the type of carbohydrate. The training diet, therefore, should be high in carbohydrate, with perhaps ≥60 % of the total energy intake coming from carbohydrate. This suggestion conforms with the recommendations of various expert committees (for example, see Department of Health, 1991) that carbohydrates provide >50 % of the dietary energy intake. It may not be helpful to athletes and those who advise them, however, for the carbohydrate requirements to be expressed as a percentage of the total energy intake; energy intake varies greatly between individuals, and a high carbohydrate intake may be achieved at very high energy intakes with a relatively low proportion of the total food intake consisting of carbohydrate. It may be better to think of an absolute requirement for carbohydrate, consisting of the amount used in training plus the amount used by body tissues during the remainder of the day, rather than relating the need to total energy intake. A daily dietary carbohydrate intake of 500–600 g may be necessary to ensure adequate glycogen resynthesis during periods of intensive training, and for some athletes the amount of carbohydrate that must be consumed on a daily basis is even greater (Coyle, 1991). Although the carbohydrate requirement is determined primarily by training volume and intensity, body size is also an important factor, so expressing the requirement on a g/kg body mass per d basis might be the best option. A daily requirement of about 8–10 g/kg body mass is likely in periods of hard training (Williams, 1998).

Professional athletes can organise their days around training, resting and eating, but the athlete who also has to work or study faces practical difficulties in meeting the demand for energy and carbohydrate, especially when training two or three times daily. Most athletes find it difficult to train hard for at least 3 h after food intake, and the appetite is also suppressed for a time after hard exercise. In this situation it is particularly important to focus on ensuring a rapid recovery of the glycogen stores between training sessions. This recovery is best achieved when carbohydrate is consumed as soon as possible after training, as the rate of glycogen synthesis is most rapid at this time. It is normally recommended that at least 1–2 g/kg body mass (a total of 50–100 g carbohydrate) should be consumed in the first hour, and a high carbohydrate intake continued thereafter (Ivy et al., 1988; Coyle, 1991). There is clearly a maximum rate at which muscle glycogen resynthesis can occur, and there appears to be no benefit in increasing the carbohydrate intake to levels in excess of 100 g every 2 h. The type of carbohydrate is less crucial than the amount consumed, but there may be some benefit from ingesting high-glycaemic index foods at this time to ensure a rapid elevation of the blood glucose level. Initial results suggesting that the addition of protein might be effective in stimulating faster rates of glycogen synthesis (Zawadzki et al. 1992) have not been supported by later studies, where the energy content of meals has been more closely matched (van Hall et al. 2000a,b).

In practical terms these high levels of carbohydrate intake are difficult to achieve without consuming large amounts of simple sugars and other compact forms of carbohydrate, as well as increasing the frequency of meals and snacks, leading to a ‘grazing’ pattern of eating. Similar rates of replenishment of muscle glycogen have been observed in response to ingestion of a fixed amount of carbohydrate, ingested as numerous small meals or a small number of large meals (Costill et al. 1981; Burke et al. 1996). Athletes may find that sugar, jam, honey and high-sugar foods such as confectionery, as well as carbohydrate-containing drinks, such as soft drinks, fruit juices and specialist sports drinks, can provide a low-bulk, palatable and convenient addition of carbohydrate to the nutritious food base (Clark, 1994). There is no evidence that this pattern of eating is in any way harmful, although attention to dental hygiene should, of course, be emphasised. Many carbohydrate-containing commercial sports drinks have the potential to promote dental erosion, but no more so than most of the alternatives (Milošević, 1997).

**High-fat diets**

If carbohydrate is not available, or is available in only a limited amount, the intensity of the exercise must be reduced to a level where the greater part of the energy requirement can be met by fat oxidation. Although it seems obvious that the ability to train at high intensities will be impaired if a high-carbohydrate diet is not consumed, there is limited experimental evidence from studies on human subjects to support this premise (Williams, 1998). Studies where subjects have trained on high-fat diets have been more convincing, showing that a high-carbohydrate diet during a period of training brings about greater improvements in performance, even when a high-carbohydrate diet is fed for a few days after the high-fat diet to allow normalisation of the muscle glycogen stores before exercise performance is measured (Kiens & Helge, 1998). It must be recognised, though, that these short-term training studies usually involve relatively untrained individuals, and may not reflect the situation of the highly trained elite endurance athlete, where the capacity of the muscle for oxidation of fatty acids will be much higher (Henriksen & Hickner, 1998). For the athlete with very high levels of energy expenditure in training, the exercise intensity will inevitably be reduced to a level where fatty acid oxidation will make a substantial contribution to energy supply and fat will provide an important energy source in the diet. Once the requirements for protein and carbohydrate are met, the balance of energy intake can be in the form of fat.
Protein synthesis and tissue remodelling after training

It is clear that a prolonged period of training will cause substantial changes in the structural and functional characteristics of skeletal muscle and other tissues. Although major changes are not apparent in response to single exercise bouts, these changes must take place between training sessions. There is good evidence of adaptive changes in muscle structure and function taking place in response to only a few exercise sessions (Green et al. 1991). These changes are different from those commonly observed to occur in untrained subjects after a single bout of intense exercise, when the observed responses are largely catabolic in nature and evidence themselves as muscle damage involving the efflux of cellular components into the extracellular space and are accompanied by a subjective sensation of soreness (Clarkson, 1997). Nonetheless, there must be adaptive changes involving synthesis of new proteins in response to each training stimulus. It is likely that the methods currently available are simply inadequate to measure these changes with a sufficient extent of reliability.

In the recovery period muscle glycogen synthesis is a priority, but synthesis of new proteins should perhaps be seen as being of equal or even greater importance. Since little attention has been paid to this area until recently, it is not at present apparent what factors may be manipulated to influence these processes. The supply of essential amino acids and the hormonal environment are the two obvious factors that may be important. Nutritional status can influence the circulating concentration of a number of hormones that have anabolic properties, the most obvious and important example being insulin. The diet can also supply amino acids for incorporation into proteins. A fall in the intracellular amino acid concentration will restrict the rate of protein synthesis, and there is some evidence that the muscle amino acid concentration does fall after exercise (Tipton & Wolfe, 2001). Ingestion of protein or amino acids immediately after exercise can prevent this fall and promote muscle protein synthesis (Biolo et al. 1997), but no long-term studies have yet been conducted to establish if these effects result in an improved adaptation of the muscle to the training stimulus (Tipton & Wolfe, 2001).

In addition to these effects of nutrient intake, it is increasingly being recognised that cell volume is an important regulator of metabolic processes (Waldeger & Lang, 1997; Lang et al. 1998), and there may be opportunities to manipulate the cell volume after exercise to promote synthesis of proteins and of glycogen. During and after exercise there may be large changes in cell volume, secondary to osmotic pressure changes caused by metabolic activity, hydrostatic pressure changes, or by sweat loss. Alterations in cell volume induced by changes in osmolality are well known to alter the rate of glycogen synthesis in skeletal muscle (Low et al. 1997a). Amino acid transport into muscles is also affected by changes in cell volume induced by manipulation of the trans-membrane osmotic gradient; skeletal muscle uptake of glutamine is stimulated by cell swelling and inhibited by cell shrinkage (Low et al. 1997b), and the intracellular glutamine concentration appears to play an important role in a number of processes, including protein and glycogen synthesis (Rennie et al. 1998).

The full importance of these recent findings for the post-exercise recovery process and the roles they play in adaptation to a training programme remain to be established. Manipulation of fluid and electrolyte balance and the ingestion of a variety of osmotically active substances or their precursors offers potential for optimising the effectiveness of a training regimen. A number of commercial products consisting of combinations of carbohydrates with protein and/or amino acids are now available, but there is no evidence that these products are likely to be more effective than normal foods. A sandwich made with ham, cheese, tuna or even jam might be just as good at supplying both carbohydrate to replenish the muscle glycogen stores and amino acids to stimulate protein synthesis.

It must, of course, be recognised that many laboratory studies are conducted in highly unusual conditions. Subjects in most metabolic studies are fasted for some time (typically 6–12 h) before the experiment begins, and perhaps for much longer than this interval before the crucial measurements are made. Few athletes would abstain from food for more than a few hours before training, so the normal response to exercise may not be a fall in the muscle amino acid pool during the post-exercise period, and further increasing the availability of amino acids may have no effect on protein metabolism. It may be that the effects of food eaten in the pre-exercise period will make manipulation of intake after exercise irrelevant, but at present the answer is not known.

Overtraining, infection and illness

Moderate exercise levels seem to be associated with a reduced risk of minor illness and infection, but there is some evidence that athletes in hard training are at increased risk of opportunistic infections, especially those of the upper respiratory tract (Niemann, 1996). Although generally trivial in themselves, these infections may be sufficient to interrupt training. Failure to recover fully between training sessions also leads to a condition of chronic fatigue, and although this condition is not well defined, it is well recognised and is characterised by underperformance (Budgett, 1999). Damage to tissues caused by an increased level of free radical generation during and after exercise (in part from increased rate of aerobic metabolism and in part from release by neutrophilic leucocytes during phagocytosis) has been proposed as one of the factors in incomplete recovery, leading to suggestions that an increased dietary intake of antioxidant nutrients may confer some protection.

A variety of nutritional interventions have been proposed to enhance immune function, to increase the antioxidant defence mechanisms and to improve the resistance of the athlete to illness and excessive fatigue. Many of the commercial products on sale are based on herbal preparations and have not been fully evaluated for efficacy or purity, relying instead on anecdote and endorsement by successful athletes. There has been sufficient evaluation of some of the potential nutritional interventions, however, to allow the athlete to make a reasonable assessment of the potential costs and benefits.
Glutamine is used as an energy source by the cells of the immune system, and hard exercise causes a fall in the plasma glutamine level; from here it is only a short step to propose that glutamine supplementation may enhance immune function (for a review of the background to this hypothesis, see Walsh et al. 1998). Unfortunately, the available evidence does not support this suggestion at the present time (Pedersen et al. 1999). A more effective dietary strategy to enhance immunity may be to ensure an adequate dietary carbohydrate intake before exercise and regular carbohydrate ingestion during exercise. This strategy has the effect of minimising any rise in plasma levels of stress hormones (cortisol, catecholamines and growth hormone) known to have a negative effect on immunity, and is likely to be the most successful nutritional strategy (Nieman & Pedersen, 1999). If exercise is continued to the point of exhaustion, however, providing carbohydrate during exercise can extend the exercise time, but has little effect on the hormonal and immune cell responses (Bishop et al. 2001). This last finding is interesting and underlines the physiological mechanisms involved, but it is not clear whether there are practical implications for the athlete engaged in intensive training. Maintaining an adequate level of dietary carbohydrate intake remains a priority.

Although there is good evidence that the delayed-onset muscle soreness and damage that are experienced after hard exercise may be mediated at least in part by the release of free radicals, it is less clear whether supplementation of the endogenous defence mechanisms with an increased intake of antioxidant nutrients will have any effect on these processes. Supplementation with antioxidants may reduce some of the markers of free radical-mediated muscle damage, but this factor alone is not sufficient justification to recommend the use of dietary supplements, and there is no evidence of performance benefits for athletes (Packer, 1997).

**Dietary supplements and ergogenic aids**

The use of dietary supplements is widespread in sport. These supplements range from the daily multivitamin tablet, that is seen as some sort of health insurance policy, to the more exotic supplements to be found on the shelves of health food stores. A report of supplement use among 100 Norwegian national-level competitors from various sports revealed that 84 % used some form of micronutrient supplementation (Bishop et al. 2001). If exercise is continued to the point of exhaustion, however, providing carbohydrate during exercise can extend the exercise time, but has little effect on the hormonal and immune cell responses (Bishop et al. 2001). This last finding is interesting and underlines the physiological mechanisms involved, but it is not clear whether there are practical implications for the athlete engaged in intensive training. Maintaining an adequate level of dietary carbohydrate intake remains a priority.

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The creatine phosphate content of muscle is small, but it is an important energy source for muscle during intense exercise. The normal daily intake is less than 1 g, but the estimated daily requirement for the average individual is about 2 g. The body has a limited capacity to synthesise creatine from amino acid precursors (arginine and glycine), mainly in the liver, kidneys and pancreas, and in other tissues, but the primary site of synthesis in man is the kidney (Greenhaff, 2000). Endogenous synthesis supplies the amount required in excess of the dietary intake, and is also the only way in which vegetarians can meet their requirement. It is interesting to note that many endurance athletes adopt a vegetarian diet, but there seem to be no reports of vegetarian athletes succeeding at the highest level in strength events.

The first study to systematically investigate the effects of supplementation of large amounts of creatine was that of Harris et al. (1992). In a comprehensive study they showed that ingestion of small amounts of creatine (≤ 1 g) had a negligible effect on the circulating creatine concentration, whereas feeding higher doses (5 g) resulted in an
approximately a 15-fold increase. Repeated feeding of 5 g doses every 2 h maintained the plasma concentration at about 1 mmol/l over an 8 h period. Repeated feeding of creatine (5 g four times daily) over a period of 4–5 d resulted in a marked increase in the total creatine content of the quadriceps femoris muscle. An increase in muscle creatine content was apparent within 2 d of starting this regimen, and the increase was greatest in those subjects with a low initial level; in some cases an increase of 50% was observed. Approximately 20% of the increase in total muscle creatine content is accounted for by creatine phosphate. Based on this and other studies, athletes typically take four 5 g doses daily for 4–6 d and 1–2 g daily thereafter.

Since that first study, there have been a large number of published reports; a search of the exercise science literature (SportDiscus, 2001) revealed 427 publications on creatine. A wide range of experimental models and subject groups have been employed, and it is difficult to determine consistent patterns that could account for the effects seen in some studies but not in others. There is strong support for effects on repeated sprints, with less conclusive evidence for effects on single sprints and on muscle strength (Maughan, 1999; Williams et al., 1999). Many studies and much anecdotal information support the suggestion that acute supplementation with creatine is associated with a prompt gain in body mass. This gain typically seems to amount to about 1–2 kg over a supplementation period of 4–5 d, but may be more than this amount. In reviewing those studies where changes in body mass were reported, Clarkson (1998) reported eleven studies where body mass increases occurred and three studies where no change in mass was reported. There is some evidence, which is discussed in detail in the cited reviews, that there may be some stimulation of protein synthesis in response to creatine ingestion, perhaps as a result of cell swelling.

Many concerns have been raised that the effects of long-term use of large doses of creatine are unknown, and that its use may pose a health risk. Concerns seem to focus primarily on the possible effects on renal function, in particular in individuals with impaired renal capacity. Studies on the response to long-term creatine use are in progress at this time, but results are not yet available. There have, however, been no reports of adverse effects in any of the studies published in the literature, or in any athletes taking creatine supplements (Williams et al., 1999).

**Caffeine**

Caffeine is probably the most widely used stimulant drug in the world, being present in many common foods and beverages (Table 2). The effects of caffeine on the central nervous system are relatively mild and it is only weakly addictive. For these reasons and also because of its long-standing and widespread use, it is considered socially acceptable. The use of caffeine is not prohibited in sport, but there is a limit to the amount that may be taken by athletes in competition; any individual whose urine contains caffeine at a level of > 12 mg/l is guilty of a doping offence and is liable to be banned from competition.

<table>
<thead>
<tr>
<th>Foodstuff</th>
<th>Serving size (ml)</th>
<th>Caffeine content (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coffee*</td>
<td>150</td>
<td>50–120</td>
</tr>
<tr>
<td>Tea†</td>
<td>150</td>
<td>15–50</td>
</tr>
<tr>
<td>Hot chocolate†</td>
<td>250</td>
<td>10</td>
</tr>
<tr>
<td>Milk chocolate†</td>
<td>50 g</td>
<td>40</td>
</tr>
<tr>
<td>Soft drinks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coca Cola</td>
<td>330</td>
<td>50</td>
</tr>
<tr>
<td>Pepsi</td>
<td>330</td>
<td>40</td>
</tr>
<tr>
<td>Jolt</td>
<td>330</td>
<td>100</td>
</tr>
</tbody>
</table>

* Values vary widely, depending on the source and method of preparation.
† In addition to its caffeine content, chocolate contains substantial amounts of the related compound theobromine; although this compound is less pharmacologically active, the high content gives it an equivalent effect to that of caffeine.
‡ Coca Cola, The Coca Cola Company, Atlanta, GA, USA; Pepsi, PepsiCo Ltd, New York, USA; Jolt, Wet Planet Beverages, Rochester, NY, USA.

Early studies on the effects of caffeine on endurance performance focused on its role in the mobilisation of free fatty acids from adipose tissue, increasing fat supply to the muscle, which in turn can increase fat oxidation, spare glycogen and thus extend exercise time. In a variety of different experimental models caffeine was shown to increase circulating free fatty acid levels, increase fat oxidation, spare muscle glycogen and improve performance of endurance exercise (Spriet, 1997; Hawley, 1998).

Growing evidence of a positive effect of caffeine on performance in the absence of any glycogen-sparing effect, and of effects on high-intensity exercise, where glycogen availability is not a limiting factor, has stimulated the search for alternative mechanisms of action (Spriet & Howlett, 2000). Caffeine may affect the activity of Na/K ATPase and the intracellular localisation and binding of Ca in skeletal muscle, it can cause an elevation of the intracellular cAMP level as a result of inhibition of the action of phosphodiesterase, and it may have direct effects on a number of enzymes, including glycogen phosphorylase (Spriet, 1997). Whether all these effects can take place at the tissue concentrations of caffeine that occur after ingestion of moderate doses of caffeine remains unclear.

Effects on the central nervous system, by virtue of its action as an adenosine receptor antagonist, either to modify the perception of effort or on the higher motor centres, have been proposed, but this mechanism of action remains speculation (Graham et al., 1994).

Whatever the mechanism of action, there are a number of studies showing beneficial effects of caffeine ingestion in a variety of laboratory tests of exercise performance. Although the effects on sprint performance are less certain than those on endurance exercise, a positive effect of caffeine can be obtained with caffeine doses that are far below those necessary to produce a positive drug test. Doses of as little as 3 mg/kg body mass can produce ergogenic effects, but there appears to be a wide inter-individual variability in the sensitivity to caffeine (Spriet & Howlett, 2000). The reasons for this variability are not altogether clear, but, perhaps surprisingly, they do not appear to be related to the habitual level of caffeine consumption.
Caffeine has a number of unwanted side effects that may limit its use in some sports or by sensitive individuals; these effects include insomnia, headache, gastrointestinal irritation and bleeding, and a stimulation of diuresis. The diuretic action of caffeine is often stressed, particularly in situations where dehydration is a major issue. This action particularly affects competitions held in hot humid climates where the risk of dehydration is high, and is more important for endurance athletes where dehydration has a greater negative effect on performance. Athletes competing in these conditions are often advised to increase their intake of fluid, but are usually also advised to avoid tea and coffee because of their diuretic effect. It seems likely, however, that this effect is small for those habituated to caffeine use (Wemple et al. 1997), and the negative effects caused by the symptoms of caffeine withdrawal may be more damaging.

**Bicarbonate**

In exercise that causes fatigue within a few minutes, anaerobic glycolysis makes a major contribution to energy metabolism. Anaerobic glycolysis allows higher rates of ATP resynthesis than can be achieved by aerobic metabolism, but the capacity of the system is limited, and fatigue ensues rapidly. The metabolic acidosis that accompanies glycolysis can inhibit key glycolytic enzymes, interfere with Ca transport and binding, and interfere directly with the actin–myosin interaction. Induction of a metabolic alkalosis by ingestion of NaHCO₃ before exercise can increase both the muscle buffering capacity and the rate of efflux of H⁺ from the active muscles, potentially delaying the attainment of a critically low intracellular pH (McNaughton, 2000).

The reason for the conflicting effects on exercise performance reported in the published literature is not altogether clear, but some effects at least are probably due in part to variations in the intensity and duration of the exercise tests used, in the nature of the exercise task, in the dosage of NaHCO₃ administered and in the time delay between bicarbonate administration and the beginning of the exercise test (i.e. in the extent of metabolic alkalosis induced).

Improvements in performance are typically seen in exercise lasting from about 30 s to a few minutes, but several studies have failed to find positive effects, even when they have used exercise of this duration. Effective doses have been rather large, typically about 0·3 g/kg body mass. There are, of course, potential problems associated with the use of such large doses of bicarbonate. Vomiting and diarrhoea are frequently reported as a result of ingestion of even relatively small doses of bicarbonate, and this problem may limit attempts to improve athletic performance by this method. Although unpleasant and to some extent debilitating, these effects are not serious and there are not likely to be long-term adverse consequences of occasional use. Sodium citrate administration, which also results in an alkaline shift in the extracellular fluid, has been reported to improve peak power and total work output in a 60 s exercise test, without adverse gastrointestinal symptoms (McNaughton, 1990).

**Nutrition strategies for competition**

In preparation for competition, and in some events during the competition itself, there is scope for athletes to adopt specific strategies that can improve performance. In view of the diverse nature of sport and the individuals who take part, the strategies adopted vary greatly between sports. Some athletes compete frequently; the typical top-class cyclists, for example, may race on more than 100 d/year (Jeukendrup et al. 2000). These riders face many practical challenges in recovering from one event while preparing for the next, and the disruptions associated with travel and long periods away from the home environment add to these problems. In other sports competition is infrequent, with no more than two or three key competitions per year, allowing each event to be more easily planned.

**Enhancing energy supply**

The need for an adequate availability of carbohydrate as a substrate for the working muscles during intense exercise has been discussed earlier. The practical problem of ingesting sufficient carbohydrate to maximise muscle glycogen storage when competitions are close together means that athletes must have a strategy in place to ensure the availability of suitable carbohydrate sources at the right times and in the right amounts. Although nutrition is often perceived by athletes as important, it often has a lower priority than equipment and other issues.

A high-carbohydrate diet is important in training to maximise the intensity of the training load that can be sustained and to reduce the risk of illness and injury, but it is even more critical during competition. An increased store of glycogen in the working muscles has been shown to enhance performance in a number of different exercise models, including prolonged constant intensity effort, simulated race performance, multiple sprint events and also short-duration high-intensity exercise. These studies have been the subject of a number of excellent reviews, including those by Coyle (1991), Hawley & Burke (1997) and Williams (1998), and will not be discussed in detail here.

The debate as to the value of feeding a high-fat diet in preparation for competition continues. There is evidence both for and against the hypothesis that training on a high-fat diet for a period of a few weeks before competition can improve performance by promoting fat use and sparing glycogen stores. Helge et al. (1996) showed that a high-carbohydrate diet during a 7-week period of training resulted in better performance than a high-fat diet fed for the same time, even when a high-carbohydrate diet was fed for a few days to allow normalisation of the muscle glycogen stores before exercise performance was measured. In contrast to these findings, Lambert et al. (2001) have recently reported better performance in well-trained cyclists who consumed a high-fat diet for 10 d before following a high-carbohydrate diet for the last 3 d before an exercise test. Several other studies can also provide evidence to support one or other side of the argument, but differences in diet composition, in the exercise models used and in the training status of the subjects make comparisons between
It is well recognised that dehydration, resulting either from sweat losses or from an inadequate fluid intake, can impair performance in prolonged exercise, but a pre-existing fluid deficit can also reduce the capacity to perform high-intensity exercise. Maintaining body water content at a level close to euhydration is therefore a priority for all athletes in competition. Water is lost from the body by a number of routes; in addition to the major losses in urine and sweat, substantial losses may also occur from the lungs and through the skin, especially at altitude or in air-conditioned environments. Infectious diarrhoea can also cause substantial losses of body water and electrolytes, and may be a particular problem for athletes who travel extensively. Again, these studies have been the subject of recent reviews by a number of authors (Maughan, 2000b; Sawka et al. 2000) and will not be further discussed here.

Central nervous system effects

The use of most of the compounds that act as central nervous system stimulants is specifically prohibited by the doping regulations that govern most sport. Caffeine is a notable exception, but even caffeine is prohibited above a threshold level set by the International Olympic Committee (Spriet & Howlett, 2000). Nonetheless, competitors can gain performance advantages at levels below those that would result in a positive test. The dilemma for the athletes is that individual sensitivity varies greatly, both with regard to the effects on performance and to the urine caffeine concentration that results after ingestion of a fixed dose.

A number of other strategies have been employed in attempts to manipulate brain chemistry and hence function. As most of the centrally acting drugs are not permitted by the governing bodies of sport, the scope is limited, but there has been interest in the use of branched-chain amino acids. Newsholme and colleagues (Newsholme & Castell, 2000) have proposed that fatigue may be mediated in part by the actions of 5-hydroxytryptamine (serotonin) in the brain, and that the synthesis and release of 5-hydroxytryptamine may be modulated by diet. In particular, it is proposed that, because the synthesis of 5-hydroxytryptamine is limited by availability of its precursor tryptophan, and because tryptophan uptake across the blood–brain barrier shares the same transporter as the branched-chain amino acids, an increase in circulating branched-chain amino acid concentration might delay the onset of fatigue (Davis, 2000). While there is some experimental support for the hypothesis of Newsholme & Castell (2000), the evidence of a beneficial effect on exercise performance is limited. Nonetheless, the fact that pharmacological manipulation of 5-hydroxytryptamine activity can alter exercise performance (Wilson & Maughan, 1992) does suggest that nutritional interventions to produce the same effect may also be possible.

References


