Chapter 32

Optimising and enhancing human performance through nutrition

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CHAPTER CONTENTS

1 INTRODUCTION

Good nutrition plays an important role in allowing athletes to achieve training and competition goals. A well-chosen eating plan is needed to maximise the success of the training programme and prepare the athlete for competition. Strategies for fluid and fuel intake before, during and after exercise can reduce fatigue and promote optimal performance. These strategies are particularly important in the competition arena. Despite the degree of expertise that underpins the majority of guidelines for sports nutrition, many athletes do not appear to follow good nutrition practices. The aim of this chapter is to provide an overview of the different ways in which nutrition can contribute to sporting success.

2 EATING TO OPTIMISE TRAINING

The major role of the everyday diet is to supply the athlete with fuel and nutrients needed to optimise the adaptations achieved during training, and to recover quickly between workouts. The athlete must also eat appropriately to stay in good health and to achieve and maintain an optimal physique.

2.1 Energy needs for training and the ideal physique

The body continuously expends energy to maintain physiological functions, for the biosynthesis of macromolecules such as proteins, glycogen and triacylglycerols, and for muscular work. However, most of this energy is lost as heat (~80%; Chapter 19), due to the inefficiency of the metabolic pathways. This relationship means that energy expenditure can be assessed from the direct measurement of heat production (calorimetry). Since there is a close relation between energy metabolism and oxygen uptake, steady-state oxygen uptake can also be used to estimate energy expenditure (indirect calorimetry).

Total daily energy expenditure is composed of three components: the basal (or resting) metabolic rate, the thermic effect of food and physical activity (Figure 32.1).

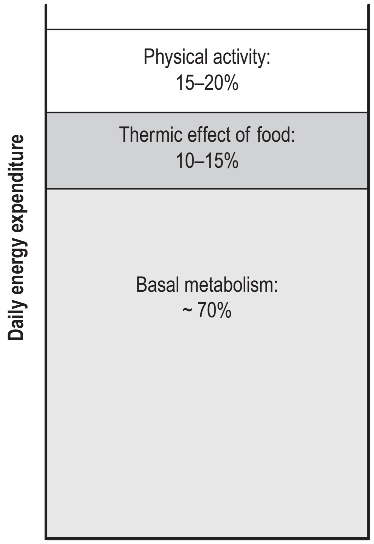

Figure 32.1 The three principal components of total daily energy expenditure in an inactive individual. PA, physical activity; TEF, postprandial thermogenic effect of food; BMR, basal metabolic rate.

A certain amount of energy is required to support life, and the rate at which a resting organism oxidises its own stored fuels is called the basal metabolic rate which is measured under standardised conditions (awake, supine rest (at least 30 min), comfortably warm environment, after 10–12 h fast). A distinction exists between the basal and resting metabolic rates. However, these may be considered similar if measurements are performed in the postabsorptive state.

Basal metabolic rate appears to vary with age when normalised to body mass, height or surface area, since muscle mass declines beyond ~50 years. However, when normalised to the summed mass of the most metabolically active tissues (heart, kidney, brain, liver), which retain mass, basal metabolic rate is stable with ageing (Chapter 16). Fat cells are metabolically active but contain a substantial amount of inert fat, making adipose one of the least active tissues. Thus, the larger fat mass of an average woman results in a lower basal metabolic rate, when normalised to body mass but not when normalised to the fat-free mass.

Basal metabolic rate is also dependent on body size, physiological status (growth, pregnancy) and hormonal status. Most women show cyclic variations in basal metabolic rate (±5%), which is lowest in the late follicular phase of the menstrual cycle, increasing at ovulation and peaking in the late luteal phase.

Besides the basal metabolic requirements, energy is also required for processing food (eating, digestion, absorption, transportation, storing) – the thermic effect of food or dietary-induced thermogenesis (Figure 32.1). The remaining energy expenditure is accounted for by daily physical activity, over and above the resting state.

Due to the often huge training volume (intensity and frequency) of many athletes, energy expenditure can be several fold higher compared to a sedentary person, resulting in a large increase in energy needs. Since athletes also vary in size (female gymnast versus heavy male rower), and body size influences energy expenditure, then body mass influences the energy requirement.

Growth is another important factor to consider. An individual is in a state of energy balance if energy intake equals energy expenditure. For growth to occur, intake must exceed output (energy turnover). This does not only apply to young athletes, since an increased body mass may also be of importance to athletes in sports where a high power output is desired. However, whenever energy intake and expenditure are not equal, a change in body mass will occur; a negative energy balance results in the use of stored energy (protein, glycogen, fat), while a positive energy balance results in energy storage (primarily as fat). If performance is to be maintained during periods of intense training, high energy expenditures must be matched by equivalent energy intakes.

Experimental data indicate that most athletes are in energy balance. Despite this, numerous cross-sectional observation studies have repeatedly reported a negative energy balance, primarily in female, but also in male athletes. In male athletes, this is often related to weight classification sports. Furthermore, most but not all studies have reported that amenorrhoeic athletes consume even less energy than eumenorrhoeic athletes (Chapter 11), when matched for training level, size and body mass (Loucks 2004). Of interest, however, is that despite apparent negative energy balance, body mass often remains stable. Although these studies collectively imply that female athletes, in particular amenorrhoeic athletes, are chronically exposed to a state of low-energy availability, many studies have been questioned due to the under-reporting of energy intake, undereating during the registration period and methodological difficulties in measuring energy intake and expenditure.

However, low energy intake in female athletes is reported from various laboratories, especially in aesthetic sports and in those where low body mass is desired. This may suggest that, in some situations, energy balance is not maintained on a daily basis, and that many athletes might be energy deficient during their heavy exercise training programmes. Repeatedly low energy intake may indicate an adaptation to a lower energy balance in these individuals.

There is considerable evidence that a low energy availability can have serious consequences for the hormonal, immunological and health status of athletes. This is exemplified in females who develop the female athlete triad: low energy availability, impaired menstrual function and reduced bone mineral density (Loucks 2004). Many female athletes develop metabolic, reproductive and bone disruptions because they excessively restrict energy intake. Among these is an increased plasma cortisol concentration, a catabolic hormone that accelerates protein degradation during states of energy deprivation and especially carbohydrate deprivation. Furthermore, plasma growth hormone concentrations will increase, while plasma insulin, insulin growth factor type I, triiodothyronine, leptin and glucose concentrations decrease with energy deficiency (Loucks et al. 1998).

In a non-human primate model, Williams et al. (2001) demonstrated that the induction of amenorrhoea was a product of the volume of calories consumed during training, which decreased the energy availability for reproductive and other necessary metabolic functions. Furthermore, prospective studies in a primate model, examining the short-term effect of low energy availability on circulating reproductive hormone levels, provide evidence that the energy cost of exercise can result in a suppression of reproductive hormone secretion, when exercise-induced increase in energy expenditure is not offset by supplemental caloric intake.

When young lean men were exposed to an average energy deficiency of $4\ MJ{\cdot}d^{-1}$ for 8 weeks in multi-stress environments (heat, cold, sleep deprivation; Friedl et al. 2000), they reduced fat mass by 51% and fat free mass by 6%. Reductions in metabolic substrates and hormones were similar to those reported by Loucks et al. (1998) for women. But interestingly, Friedl et al. (2000) demonstrated that, after 1 week of refeeding, the plasma concentrations of growth hormone, insulin growth factor type I and triiodothyronine were restored, despite the continuation of exercise and other stresses (Friedl et al. 2000). In accordance with this, Williams et al. (2001) found that induced amenorrhoea in primates was reversed by supplementing energy intake, but without modifying training volume. Thus, these data indicate that it is the decreased energy availability, resulting from a failure to increase energy intake to match expenditure (and carbohydrate utilisation), and not exercise per se that leads to a disruption of metabolic and reproductive functions.

When energy availability was restricted for exercising women and men to $<126\ kJ{\cdot}kg^{-1}$ fat-free mass per day (over 5 days), the pulsatile secretion of luteinising hormone was depressed in both genders, especially with low carbohydrate availability (Loucks et al. 1998). In exercising women, glucoregulatory hormones do not maintain normal plasma glucose concentrations below an energy availability of $126\ kJ{\cdot}kg^{-1}$ fat-free mass per day (Loucks & Nattiv 2005). It is noteworthy that energy balance normally occurs in young adults at an energy availability of about $190\ kJ{\cdot}kg^{-1}$ fat-free mass per day. Some athletes, especially amenorrhoeic athletes, have caloric intakes of only $67\ kJ{\cdot}kg^{-1}$ fat-free mass per day, even when training (Loucks & Nattiv 2005).

In summary, while the measurement of low dietary energy intake in athletes, especially in female athletes, has been questioned, evidence is accumulating that some athletes restrict, whilst others in endurance sports fail to modify, caloric intake to compensate for increased energy expenditure. Such caloric deficits increase the risk of hormonal dysfunctions, impaired menstrual status, poor bone health, immune function and growth. For those athletes who are on fat loss programmes, it is recommended that caloric intake should not fall below $126\ kJ{\cdot}kg^{-1}$ fat-free mass per day.

2.2 Strategies to reduce mass and body fat

An adequate energy intake is critical for optimal physical performance. However, some athletes want to reduce body mass to improve their aesthetic appearance, while others want to lose body fat (increase fat-free mass) to optimise performance. In addition, athletes involved in weight category sports must achieve fixed mass targets prior to competition. The similarity between these athletes is that they often try to reduce body mass well below that considered normal for their stature. For example, athletes engaged in weight category sports often compete in a class 5–10% below their usual body mass, but competition is often followed with periods of unrestricted food intake and mass gains. This often leads to unhealthy nutritional practices including deliberate vomiting, overexercising, voluntary dehydration and the use of diet pills and diuretics. These practices may result in severe health consequences, such as delayed maturation, impaired growth, menstrual irregularities, increased rate of infections, eating disorders and depression. However, is there an optimal strategy for reducing body mass without experiencing adverse health implications or reducing performance?

To lose weight a negative energy balance must be elicited, and to maintain lost body weight, a lower energy balance must be maintained. Negative energy balance can be achieved by restricting energy intake, increasing energy expenditure by additional training or through simultaneously modifying both sides of the equation.

When body mass is reduced through energy restriction alone, a large percentage of this loss can be accounted for by reductions in lean body mass and total body water. To what extent lean mass is lost depends on the extent of the energy restriction and the duration of that restriction. For instance, caloric restriction with rowers reduced body mass by 2–8 kg over 2–4 months but muscle mass loss accounted for 32–87% of the mass change (Koutedakis et al. 1994, Slater et al. 2006a).

There is some evidence that resting metabolic rate is largely related to the lean body mass, since, in adults <50 years, muscle mass accounts for about 40% of the body mass. Thus, whilst the resting metabolic rate of muscle ($54\ kJ{\cdot}kg^{-1}{\cdot}d^{-1}$) is only a small fraction of that of the heart ($1842\ kJ{\cdot}kg^{-1}{\cdot}d^{-1}$), its mass-specific energy consumption is more than twofold greater. Reduced thyroid function most certainly contributes to this metabolic reduction. Repeated episodes of mass loss and gain (weight cycling) have also been associated with reduced resting metabolic rate, altered patterns of body fat distribution and increased rates of mass gain. These changes will make subsequent mass loss more difficult, since a greater energy restriction is required to achieve a negative energy balance. Most knowledge on this aspect, as it relates to athletes, has been obtained from studies of wrestlers. The data indicate that, during the season, when body mass was lowest, resting metabolic rate was significantly reduced (Melby et al. 1990).

The effect of rapid mass loss on performance appears to depend on how the loss is achieved, its magnitude and the type of exercise performed. Absolute maximal oxygen uptake may decrease after mass losses. Some would suggest this may affect endurance performance (Fogelholm 1994). However, this needs to be viewed cautiously, particularly in weight-bearing activities, where a mass reduction equates with a reduced energy expenditure to move at a constant velocity. That is, a reduction in the size or number of cells is only detrimental if it impinges upon one's ability to perform, which, at elite levels, is often unrelated to slight changes in aerobic power (Chapter 10.2).

Burge et al. (1993) observed that a 5.2% decrease in body mass over 24 h resulted in a 22 s increase in a simulated 2000 m ergometer performance in competitive rowers. However, this cannot be due to the loss of metabolically active cells, but to dehydration and perhaps some muscle glycogen depletion. Such performance decrements are entirely predictable (Viitasalo et al. 1987). The effect of such acute mass losses is reduced or eliminated when repeated over several days (Slater et al. 2006b). In a study of three heavyweight rowers, who were prepared to compete as lightweight rowers (16 weeks), body mass decreased 2.0–8.0 kg, with muscle mass accounting for a large proportion of this change (32–85%). Two athletes maintained performance. However, performance was compromised in the athlete who experienced the greatest mass loss (Slater et al. 2006a).

Can dietary strategies modify physiological adaptation to energy restrictions and prevent a decrease in performance? Horswill et al. (1990) found that performance was unimpaired after a mass loss of 6% over 4 days, if a relatively high carbohydrate (energy) consumption was achieved. In addition, low carbohydrate intake was suggested to explain impaired performance obtained in judo athletes after energy restriction in combination with intense training that caused mass losses (Degoutte et al. 2006).

A high carbohydrate diet during energy restriction seems mandatory for maintaining physical performance, probably because it may prevent glycogen depletion that might otherwise compromise performance (Chapter 7). Moreover, for athletes who are on fat loss programmes, energy availability should not be below 126 $kJ \cdot kg^{-1}$ fat-free mass per day (Loucks & Thuma 2003). Thus, to avoid performance and training decrements and also health complications, mass losses should not exceed ~0.5–1.0 $kg \cdot wk^{-1}$ or an energy deficit of 2–4 MJ. Hence, acute mass losses should be avoided and weight loss should slowly be achieved over several weeks, and be planned well in advance.

Evidence from wrestlers has shown that while resting metabolic rate was significantly reduced during the season, their off-season body masses returned to normal, as did resting metabolic rate (Melby et al. 1990). Thus, participating in numerous cycles of mass loss and gain did not permanently lower resting metabolic rate in these athletes. On the other hand, a recent study from a cohort of 1838 male athletes engaged in international competitions from 1929 to 1965, including 370 males engaged in boxing, weight lifting and wrestling, indicated that repeated cycles of mass loss and gain appeared to enhance subsequent mass gains and may predispose to obesity later in life (Saarni et al. 2006).

Thus, when mass or fat reductions are required, these should be achieved by a programme of eating and exercise that allows the athlete to perform well, stay healthy and remain free of unreasonable stress, in both the short and long term. Many athletes need assistance to plan dietary programmes that meet goals for adequate energy intake, and sufficient protein and micronutrients consumption. Although it is difficult to get reliable figures on the prevalence of eating disorders among athletes, there appears to be a higher risk of problems among female athletes and athletes in sports that require specific mass targets or lower body fat levels than in the general population (Beals & Manore 1994, Sundgot-Borgen 2000).

2.3 Requirements for growth and gaining lean body mass

Lean body mass represents several tissues but for athletes, the focus is muscle mass. The metabolic foundation for changes in muscle mass is the difference between muscle protein synthesis and breakdown (net muscle protein balance, nitrogen balance or protein turnover), which occur continually and concurrently.

Accretion of muscle proteins results when protein turnover, particularly the balance of myofibrillar proteins, is positive (net muscle protein synthesis). Exercise and nutrition have an immense influence on protein turnover which, on a daily basis, can be either positive or negative, depending on feeding and exercise situations. The length and duration of these periods of positive and negative balance determine the net loss or gain of muscle mass. Consequently, in healthy, mass-stable adults, periods of positive and negative turnover will be equal and no growth occurs. Muscle growth only results when a cumulative positive protein turnover prevails.

A clear relationship between energy intake and muscle growth should not be surprising. Nevertheless, athletes most often focus on protein intake when muscle growth is desired. However, it is not clear that increasing protein intake above habitual levels should be the primary objective. First and foremost, the athlete must at least maintain energy balance, and most likely a positive energy turnover.

After all, protein accretion is an energetically expensive process; the deposition of 1 g of protein consumes ~100 kJ of energy.

It has been estimated that ~30% of the variation in protein turnover may be attributed to differences in energy balance (Pellet & Young 1992). As much as 100 years ago, Chittenden (1907) demonstrated that athletes gain strength and maintain muscle mass even during periods of low protein intake, provided energy intake is sufficient. In a series of classical studies, Butterfield & Calloway (1984) and Todd et al. (1984) established that maintenance of a balanced protein turnover during training is not possible if energy balance is negative, regardless of protein intake. More recently, studies have shown that additional energy intake results in greater gains in lean body mass than additional protein intake during resistance training (Gater et al. 1992, Rozenek et al. 2002). Clearly, energy intake is critical for protein accretion and muscle growth.

3 PROTEIN NEEDS FOR MUSCLE GAIN, TRAINING ENHANCEMENT AND REPAIR

Most athletes feel that high protein intake is critical for muscle growth, repair and enhancement of training adaptations, and a huge supplement industry has been built upon this assumption (Chapter 34.1). The scientific evidence for high protein intakes in athletes is, at best, equivocal and is extensively debated in the scientific community (Phillips 2004, Rennie & Tipton 2000, Tarnopolsky 2004, Tipton & Wolfe 2004).

Generally, there is much disagreement as to the protein needs for athletes and exercising relative to sedentary individuals. Well-controlled studies have demonstrated that nitrogen balance is generally greater for athletes than in sedentary controls (Lemon et al. 1992, Tarnopolsky et al. 1988, 1992). Increased protein needs are likely to stem from increased amino acid oxidation during exercise (McKenzie et al. 2000, Phillips et al. 1993), and the growth and repair of muscle. During recovery from both endurance (Carraro et al. 1990, Tipton et al. 1996) and resistance training (Biolo et al. 1995, Phillips et al. 1997), muscle protein synthesis is elevated. Thus, increased protein intake may provide amino acids for the elevated synthesis for repairing damaged protein, muscle growth and mitochondrial biogenesis.

On the other hand, many authors maintain that protein needs for active individuals, even those involved in heavy training, are not increased (Rennie & Tipton 2000, Tipton & Wolfe 2004). This argument is supported by the fact that the efficiency of amino acid utilisation is increased by exercise (Todd et al. 1984), perhaps due to increased efficiency of reutilisation of amino acids from muscle protein breakdown (Phillips et al. 1999). Whole-body protein balance decreases following training, indicating that protein requirements would actually be less with regular training (Hartman et al. 2006).

The disagreement possibly originates from two sources: methodological limitations (nitrogen balance and leucine oxidation) and, perhaps more fundamentally, a lack of consideration for the primary reason why athletes need protein. Rather than the attainment of nitrogen balance, the amount of protein that optimises training and maximises performance is the important consideration. Each athlete will have a unique protein intake that optimises adaptations to training and performance. Thus, a single numerical value indicating the protein requirement for all athletes, or even relatively arbitrary divisions for endurance and strength athletes, seems illogical.

For many, if not most, athletes, the point may be inconsequential. Most athletes ingest enough protein to cover even the higher estimates of ~1.2–1.5 $g \cdot kg^{-1} \cdot d^{-1}$ (Phillips 2004, Tarnopolsky 2004). Thus, recommending increased protein intake would not be necessary for the majority of athletes consuming a well-chosen diet that meets energy needs. In fact, for some athletes, extra protein may be detrimental. For example, if carbohydrate intake is reduced to make room for protein, given a limited energy intake, performance may be impaired (Macdermid & Stannard 2006).

On the other hand, there are undoubtedly athletes for whom increased protein intake may be beneficial. If increased muscle mass and strength is the primary goal, there seems little reason to limit protein intake, provided the requirements for other nutrients are satisfied. However, given the high energy intakes necessary to support increased muscle mass, habitual protein consumption is likely to assure maximum muscle accretion. Certainly, no evidence exists to suggest the anabolic response to protein from food sources is inferior to that from commercially available supplements (Elliot et al. 2006, Phillips et al. 2005).

Athletes desiring mass reduction, and who are in negative energy balance, may benefit from higher protein consumption (Layman & Walker 2006). Use of a risk/benefit approach may offer some insights. If risk is minimal and there is a rationale for potential benefit, then there is no reason to recommend against increasing protein intake. Health problems have often been touted as reasons for avoiding protein intake, particularly kidney damage. While the relationship between high protein intake and chronic diseases has not been established, it should be noted that individuals with pre-existing problems, particularly kidney disease, should not consume high-protein diets (Zello 2006).

A further complication to assigning specific amounts of daily dietary protein to all athletes, or even groups of athletes, is that amino acids utilisation and the metabolic response to ingested protein are variable among individuals, depending on the circumstances in which the protein was ingested (Tipton & Wolfe 2004). For instance, Phillips and colleagues (2005) reported that amino acid uptake from proteins into muscle is greater for milk than soy proteins, following resistance training. However, unlike whole-body amino acid uptake at rest (Dangin et al. 2001), the use of individual milk proteins by muscle following exercise cannot be distinguished (Tipton et al. 2004).

Similarly, the anabolic response to protein ingestion will vary with other concurrently ingested nutrients. Following exercise, ingestion of carbohydrates increases amino acid use by muscle (Borsheim et al. 2004, Miller et al. 2003), an effect likely to be mediated by the insulin response (Biolo et al. 1999). Interestingly, recent evidence implies that amino acids from protein ingestion may be used to a greater extent when fat is simultaneously ingested (Elliot et al. 2006). However, the mechanism for increased amino acid use with fat ingestion is unclear, and these observations require more systematic investigation before firm conclusions may be drawn. Nonetheless, taken together, it is clear that ingesting a given amount of protein results in differential muscle use (Figure 32.2), and thus training adaptations may depend less on the amount of protein ingested and more on the type of proteins ingested, and other nutrients ingested in the same meal.

The anabolic response of muscle is determined not only by nutrients but also by timing the ingestion of protein in relation to the exercise bout (Roy et al. 1997, 2000, Tipton et al. 2001). It seems there is an interaction between the type of amino acid source, nutrients ingested concurrently and timing in relation to exercise (Tipton et al. 2001, 2007), and the complexities of assessing the relationship of the anabolic response should be readily apparent. Indeed, muscle mass may be maintained, and even increased, on a wide range of protein intakes. The anabolic response is not determined solely by protein ingested, but varies depending on

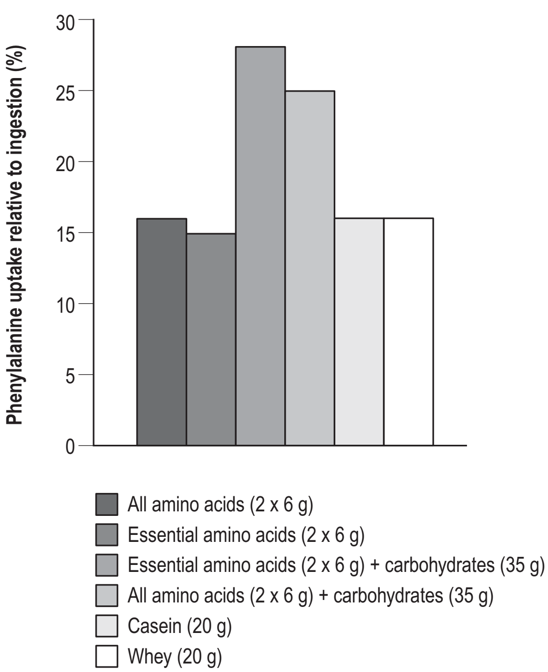

Figure 32.2 Amino acid uptake by leg muscle from ingested amino acids or proteins following exercise.

other nutritional factors associated with consuming the protein. Undoubtedly, future work will uncover more regarding these interactions.

4 FUEL NEEDS FOR TRAINING AND RECOVERY

An important goal of the athlete's diet is the provision of adequate fuel to the muscles to support the training programme. The major fuels for exercise are body fat and carbohydrate stores. Sources of fat, including plasma free fatty acids derived from adipose tissue and intramuscular triglycerides, are relatively plentiful. In contrast, carbohydrate supplies, such as plasma glucose (derived from the liver or carbohydrate intake) and muscle glycogen stores, are limited. In fact, the availability of carbohydrate as a substrate for muscle and the central nervous system becomes a limiting factor in endurance exercise of submaximal or intermittent high-intensity exercise (>90 min; Chapter 7), and plays a permissive role in the performance of brief, high-intensity work (Coyle 1995). As a result, sports nutrition guidelines have focused on strategies to enhance carbohydrate availability. The present section will focus on refuelling from day to day and the challenge of recovering between daily training sessions or multiple workouts, where fuel requirements are likely to challenge or exceed normal body carbohydrate stores.

Muscle glycogen synthesis follows a biphasic response, consisting of a rapid early phase for the first 30–60 min (non-insulin dependent) followed by a slower phase (insulin dependent), lasting up to several days (Ivy & Kuo 1998). Maximal rates of muscle glycogen storage reported during the first 12 h of recovery are within the range of 5–10 mmol·kg wet weight^{-1}·h^{-1} (Jentjens & Jeukendrup 2003) and 20–24 h of recovery are required to normalise muscle glycogen levels (100–120 mmol·kg wet weight^{-1}) after depletion through prolonged or intense exercise (Coyle 1991). However, since the training and competition schedules of many athletes often provide considerably less time than this, many athletes commence a training session with some degree of muscle glycogen depletion.

Data from a recent study show that training with low glycogen concentrations might be advantageous for training adaptation (Hansen et al. 2005). In this study, untrained subjects achieved greater increases in muscle enzyme content and endurance in the leg that trained with a protocol promoting glycogen depletion than the contralateral leg that undertook the same volume of training in a glycogen-recovered state. However, other chronic studies of diet and training interventions in well-trained athletes show that higher carbohydrate intakes that allow greater glycogen recovery are associated with fewer symptoms of overtraining during high-volume periods (Achten et al. 2004; Chapter 29) and greater training adaptations (Simonsen et al. 1991). It is likely that elite athletes optimise training outcomes by periodising training and diet, so that some sessions are undertaken with relative glycogen depletion, while highquality performance sessions occur following complete refuelling.

The major dietary factor involved in postexercise refuelling is the amount of carbohydrate consumed. As long as total energy intake as adequate (Tarnopolsky et al. 2001), increased carbohydrate intake promotes increased muscle glycogen storage, until the threshold for glycogen synthesis is reached. A recent update of the guidelines for sports nutrition of the International Olympic Committee recommended two changes in the recommended carbohydrate intake of athletes (Burke et al. 2004). The first change recognised that guidelines should be scaled according to the training load undertaken by athletes, while the second recommended that guidelines be expressed as grams of carbohydrate relative to the size of the athlete, rather than an arbitrary percentage of the athlete's total energy intake. A summary of some of these revised guidelines is presented in Table 32.1.

Athletes have been advised to enhance recovery by consuming carbohydrates as soon as possible after training. The highest rates of muscle glycogen storage occur during the first hour after exercise, and the intake of carbohydrate immediately after exercise potentiates this effect (Ivy et al. 1988). However, the most important consideration is that an absence of carbohydrate intake leads to very low rates of glycogen restoration. Therefore, an early carbohydrate intake following strenuous exercise is most valuable because it provides an immediate source of substrate to the muscle cell and maximises effective recovery. Although early feeding may be important when there is only 4–8 h between sessions (Ivy et al. 1988), it may have less impact over a longer recovery period (Parkin et al. 1997). Overall it appears that, when the interval between exercise sessions is short, the athlete should begin to consume carbohydrate as soon as possible to maximise the effective recovery time. In addition, there may be some advantages in the first couple of hours of recovery to achieving a target of 1 $g{\cdot}kg^{-1}$ body mass each hour, spread into a series of small snacks (Burke et al. 2004). However, when longer recovery periods are available, the athlete can choose their preferred meal schedule as long as total carbohydrate intake goals are achieved.

The type of carbohydrate consumed may have some effect on glycogen restoration rate. For instance, moderate and high glycaemic index, carbohydrate-rich foods and drinks appear to promote greater glycogen storage than meals based on low glycaemic index carbohydrates (Burke et al. 1993). However, the mechanisms may include factors such as the malabsorption of low glycaemic index carbohydrate, rather than differences in the glycaemic and insulinaemic responses (Burke et al. 1996).

Early research indicated that glycogen synthesis was enhanced by adding protein to carbohydrate snacks consumed after exercise, an observation that was explained by the protein-stimulated enhancement of the insulin response (Zawadzki et al. 1992). However, this has been refuted by other studies (Burke et al. 2004), and the current consensus is that the co-ingestion of protein or amino acids with carbohydrate does not clearly enhance glycogen synthesis (Burke et al. 2004). Benefits to glycogen storage are limited to the first hour of recovery (Ivy et al. 2002) or to situations where protein is added to carbohydrate intakes below the threshold for maximal glycogen synthesis. Of course, the intake of protein within carbohydrate-rich recovery meals may allow the athlete to meet other nutritional goals including the enhancement of net protein balance after exercise.

Table 32.1 Revised guidelines for the carbohydrate intake of athletes (adapted from Burke et al. 2004)

- Aim to achieve carbohydrate intakes that meet fuel requirements of training while optimising muscle glycogen between workouts. Recommendations should be fine-tuned with consideration of total energy needs, specific training needs and performance feedback:
  - Immediate recovery (0–4 h): 1–1.2 $g{\cdot}kg^{-1}{\cdot}h^{-1}$ consumed at frequent intervals
  - Daily recovery: moderate-duration, low-intensity training: 5–7 $g{\cdot}kg^{-1}{\cdot}h^{-1}$
  - Daily recovery: moderate-heavy endurance training: 7–12 $g{\cdot}kg^{-1}{\cdot}h^{-1}$
  - Daily recovery: extreme programme (4–6 h+ per day): 10–12 $g{\cdot}kg^{-1}{\cdot}h^{-1}$
- Adequate energy intake is important for optimal glycogen recovery.
- Guidelines for carbohydrate (or other macronutrients) should not be provided in terms of percentage contributions to total dietary energy intake.

5 EATING TO MINIMISE ILLNESS AND INJURY

Athletes must be able to train hard and compete without the interruptions of illness and injury. Eating well to achieve nutrient needs is also important for general health and well-being. However, there are several health challenges that are specific to sport and exercise. These include the risk of iron depletion, immunosuppression that is known to accompany prolonged and strenuous training, and the disturbances of the athlete's endocrine function, with potential implications for illness and bone integrity.

5.1 Calcium, bones and the female athlete triad

Since exercise provides a major stimulus for bone formation and bone health, it seems ironic that many female athletes are reported to suffer from compromised bone health, from frank osteopenia to a failure to achieve an optimal peak bone density. Poor bone health can reduce an athlete's potential by increasing the risk of injury, including stress fractures. Long-term problems include an increased risk of osteoporosis (Chapter 13). Initially, an awareness of poor bone health was identified as the female athlete triad syndrome (Otis et al. 1997), and is a disorder cluster involving disordered eating, amenorrhoea and osteopenia. The focus was directed to the prevalence of menstrual disturbances in females, with the recognition that disruptions to reproductive hormone have a negative effect on bone formation and remodelling. Much debate centred on the cause of the menstrual dysfunction, with theories including low body fat and high training volumes. We now know that the common thread to impairment of menstrual status and other hormonal systems is low energy availability (Loucks 2004).

This syndrome has now been updated (American College of Sports Medicine 2007) to target energy availability, menstrual health and bone density. The new message is that each of these issues involves a continuum between optimal health and frank disorder, and the athlete must be alert to changes in her status of any issue. Athletes must be educated about the benefits of early diagnosis and treatment of problems and the likelihood that negative outcomes occur at a much earlier stage than previously considered. Recent research has shown that low energy availability directly impairs bone formation and resorption (Ihle & Loucks 2004), and problems may also be seen in male athletes.

The detection, prevention and management of this triad, or individual elements within it, require expertise and, ideally, the teamwork of sports physicians, dieticians, psychologists, physiologists and coaches (Beals & Manore 2002). Dietary intervention is important to correct factors that underpin menstrual dysfunction, as well as those that contribute to suboptimal bone density. Prevention or early intervention is clearly the preferred option, since it is not always certain that damage to bone strength can be overturned, particularly when it is long term (Drinkwater et al. 1996). Dietary goals include adequate energy and calcium intakes. In the latter case, daily calcium requirements may be increased to 1200–1500 $mg \cdot d^{-1}$ in athletes with impaired menstrual function. Where adequate calcium intake cannot be met through dietary means (e.g. low-fat dairy foods or calcium-enriched soy alternatives), a calcium supplement may be considered (Kerr et al. 2006).

5.2 Iron depletion

An inadequate iron status is the most likely micronutrient deficiency among athletes and the general community. Exercise affects many measures of iron status, due to changes in the plasma volume or the acute-phase response to stress. Therefore, conventional haematological standards are often inappropriate for diagnosing the true prevalence of problematic iron deficiency in athletics.

Inadequate iron status can reduce performance via suboptimal haemoglobin concentrations and oxygen delivery (Chapters 1 and 10.1), and perhaps also via reduced myoglobin and iron-related enzymes (Hood et al. 1992). However, it is often difficult to detect the stage of iron deficiency at which impairments to exercise performance are observed. Despite initial conflict in the literature, it now appears that iron depletion, in the absence of anaemia (reduced serum ferritin concentrations), may impair exercise performance (Deakin 2006). In addition, athletes with reduced iron stores complain of feeling fatigued and failing to recover between training sessions (Chapter 29). Since low ferritin concentrations may become progressively lower and eventually lead to iron deficiency anaemia, it makes sense to monitor athletes who are at high risk of iron depletion, and to intervene as soon as iron status appears to decline substantially or to symptomatic levels.

The evaluation and management of iron status in athletes should be undertaken by a sports physician. It is tempting for fatigued athletes to self-diagnose iron deficiency and to self-medicate with iron supplements. However, there are dangers in self-prescription or long-term supplementation in the absence of medical follow-up. Iron supplementation is not a replacement for medical and dietary assessment and therapy, since it typically fails to correct underlying problems that have caused iron drain (i.e. factors causing iron requirements and losses to exceed iron intake). Chronic supplementation with high doses of iron carries a risk of iron overload, especially in males for whom the genetic traits for haemochromatosis are more prevalent.

A diagnosis of iron deficiency requires multiple sources of information which assess the presence of risk factors for low iron status and determine whether this has lead to a functional outcome. These include clinical signs and symptoms suggestive of iron deficiency or anaemia (e.g. unexplained fatigue, reduced recovery, recurrent infections, pallor), a dietary assessment which indicates an inadequate intake of bioavailable iron and the presence of other factors that may predict an increase in iron requirements or loss. Haematological evidence of iron deficiency is the presence of pale (hypochromic) and small (microcytic) red blood cells on a blood film, and a plasma haemoglobin concentration below the laboratory reference range (12 $g \cdot 100\ mL^{-1}$ (females); 14 $g \cdot 100\ mL^{-1}$ (males)). These parameters will remain normal with iron deficiency without anaemia. Although iron deficiency in the general population is normally denoted by reduced serum ferritin concentrations below the reference range (12 $ng \cdot mL^{-1}$), in athlete populations thresholds of 20 $ng \cdot mL^{-1}$ (Nielsen & Nachtigall 1998) or 30 $ng \cdot mL^{-1}$ (Fallon 2004) are often applied. Plasma measurements of soluble transferrin receptors have been described as a new marker of iron status, but this needs to be confirmed in athletic populations (Pitsis et al. 2004).

Changes to iron status parameters that occur with acute or chronic training include haemodilution, due to increased plasma volume that accompanies endurance training (Chapter 27.3), and heat adaptation (Chapter 21). These changes do not impair exercise capacity. Alternatively, an increase in serum ferritin (an acute-phase reactant) can be expected in response to a single strenuous bout of exercise, inflammation or infection, without any true change in iron status. Therefore, haematological and biochemical tests undertaken in athletes should be administered in a way that standardises these effects. For example, all tests should be completed in the same laboratory, after a light training day and before any exercise is undertaken for that day (Deakin 2006). Serial monitoring of athletes may help establish normal ranges over which such parameters vary for each athlete over the training and competition year, thereby helping to identify changes that may impair health, function or performance.

Although iron supplementation may play a role in the prevention and treatment of iron deficiency, the management plan should be based on long-term interventions to reverse iron drain, reducing excessive iron losses and increasing dietary iron. Dietary interventions should increase total iron intake and increase the bioavailability of this iron. The haem form of iron found in meat, fish and poultry is better absorbed than the organic (non-haem) iron found in plant foods such as fortified and wholegrain cereals, legumes and green leafy vegetables (Hallberg 1981, Monsen 1988). However, iron bioavailability can be manipulated by matching iron-rich foods with dietary factors promoting iron absorption (e.g. vitamin C and other food acids, 'meat factor' found in animal flesh) and reducing the interaction with inhibitory factors for iron absorption (e.g. phytates in fibre-rich cereals, tannins in tea; Hallberg 1981, Monsen 1988). Finally, changes to iron intake should be achieved with eating patterns that are compatible with the athlete's other nutritional goals.

5.3 Nutrition for the immune system

Nutrition is an important component of proper immune function (Gleeson 2006, Gleeson et al. 2004; Chapter 29). High-intensity exercise is associated with an increased incidence of infection (Nieman et al 1990) and immunosuppression (Gleeson 2006, Gleeson et al. 2004). The prevailing notion is that exercise of sufficient intensity and duration results in high plasma concentrations of stress hormones (cortisol, catecholamines), and immunosuppression ensues. Immune system depression for several hours following strenuous exercise increases the opportunity for infection (the open window hypothesis). However, despite ample evidence of the acute and chronic impact on immune function, there is little direct evidence of a link with increased illnesses (Gleeson 2006).

Nutritional deficiencies can have a profound impact on immune function, with immune dysfunction linked with severe energy restriction, which is quickly corrected with refeeding (Walrand et al. 2001). Severe energy restriction is not common among athletic populations, but subclinical eating disorders in athletes are associated with increased infection rates (Beals & Manore 1994). It is clear that insufficient protein intake (Daly et al. 1990) and deficiencies of micronutrients may lead to immunosuppression (Gleeson 2006). However, athletes who consume sufficient energy to support training demands should not be in danger of deficiencies leading to immune impairment.

Since the exercise-induced depression of the immune system is linked to increased stress hormones, nutritional manipulations that ameliorate this rise should effectively limit immune dysfunction. Carbohydrate intake may be used to minimise the immune impairments associated with prolonged exercise (Nieman 1998) and glutamine, vitamin C and zinc have also been implicated in the immune response. However, evidence for the efficacy of these supplements is equivocal, and excesses of several nutrients result in depression of immune responses (Gleeson 2006). Thus, it is clear that modulation of the immune system results from heavy exercise but there is much to be investigated about these interactions and nutrition.

5.4 Vitamins, minerals and the antioxidant system

Vitamins, minerals and the antioxidant system, as regulators of metabolism, are integral parts of nutritional considerations for all involved in regular exercise. Deficiencies of these nutrients clearly impair performance, but scientific evidence does not necessarily support vitamin and mineral supplementation to improve performance.

The main issues concerning vitamins and minerals for athletes seem to be whether regular, rigorous exercise increases their requirements and whether supplementation increases performance. Since vitamins and minerals are integral for many metabolic processes, there is ample rationale to expect exercise to impact upon nutrient requirements. Certainly, deficiencies will impair performance (Lukaski 2004, Manore 2000). However, most athletes consume ample vitamins and minerals to support training and performance, but observations are often complicated by the uncertainty of assessing vitamin and mineral status (Lukaski 2004). An obvious point of concern is for athletes with restricted energy intakes (e.g. making weight or those in body image sports) in whom low vitamin and mineral intakes would not be surprising.

Several vitamins and minerals, including vitamins C, E, A (as β-carotene), selenium, zinc, iron, copper and manganese, as well as other dietary components (e.g. flavonoids), play a role as part of antioxidant defences. Muscles produce free radicals and other reactive oxygen species during exercise (Davies et al. 1982, Jackson et al 1998), and the type of activity is likely to determine the pattern and magnitude of free radical production (Patwell & Jackson 2004). These radicals may contribute to oxidative damage and perhaps fatigue (Powers et al. 2004, Urso & Clarkson 2003). However, cells are protected by a complex antioxidant defence mechanism, to which dietary components contribute, thereby providing the rationale for antioxidant supplementation for athletes during heavy training loads (Powers et al. 2004, Urso & Clarkson 2003).

However, it is not clear at this time that supplemental antioxidants are beneficial for performance, but supplementation may play a role in scavenging free radicals and possibly preserving cell structure and function (Powers et al. 2004). Nevertheless, it is not certain that performance is impacted, and the interpretation that athletes need antioxidants to protect against oxidative damage is based primarily on studies that measure cellular and extracellular damage (Alessio 1993, Mastaloudis et al. 2001), leaving the question open. Ingestion of antioxidants may reduce markers of damage, but due primarily to a lack of well-designed studies, there is no consensus on the efficacy of antioxidants for exercise performance. On the contrary, there is clear evidence that reactive oxygen species regulate gene expression through stimulation of signalling pathways (Jackson et al. 2002). Thus, it is conceivable that antioxidant supplementation may interfere with the adaptive process to training.

6 EATING FOR COMPETITION PERFORMANCE

To achieve optimal performance, the athlete should identify nutritional factors that are likely to cause fatigue during their event, and undertake strategies before, during and after the event that minimise or delay the onset of this fatigue. Potential factors include dehydration (Chapters 19, 33 and 34.2), depletion of glycogen stores (Chapter 7), low blood glucose concentrations and other disturbances of the central nervous system (Chapter 5), gastrointestinal distress and hyponatraemia (Chapters 33 and 34.2). These nutritional challenges present according to the length and intensity of the event, the environment and factors that influence opportunities to eat and drink during the event or recovery. Of course, practical considerations are important, including the availability of suitable foods or drinks, gastrointestinal challenges to eating or drinking while exercising, and finding access to food supplies when competition takes place away from home.

6.1 Making weight to meet competition weight targets

In weight class sports, it is common practice for athletes to train at a higher body mass, before rapidly reducing mass to qualify at a lower class division against smaller, weaker opponents. There are many different practices used to achieve this reduction, but most involve severe restriction of food intake and dehydration (Steen & Brownell 2000). However, the use of diuretics and other pharmacological agents, as well as dehydration, should be avoided.

Rapid mass losses reduce lean body mass as well as fat mass, and may decrease performance (Horswill 1993) and result in health problems and even death (CDCP, 1998). A more reasonable approach may be to select the weight class that optimises each athlete's performance. In other sports where weight loss is prevalent, an optimal mass/fat level must be determined and appropriate dietary strategies should be used to achieve and maintain these goals. These strategies should maximise the opportunity to meet all nutrition goals, but without undue food-related stress.

6.2 Fuelling for competition

A key part of the preparation for competition is to ensure that muscle fuel stores are adequate for the demands of the event. Resting muscle glycogen concentrations of trained athletes (100–120 mmol·kg wet weight^{-1}) appear adequate for events lasting up to 60–90 min (Hawley et al. 1997). Such stores can be achieved by 24 h of rest and an adequate carbohydrate intake (7–10 g·kg^{-1}·d^{-1}; Costill et al. 1981), unless there is severe muscle damage. For some athletes, glycogen restoration can be achieved with everyday eating plans. However, athletes following restricted diets may need to increase carbohydrate (and energy) intake over the day before competition, and make fuelling up a higher priority than body mass concerns.

Carbohydrate loading describes practices that aim to maximise muscle glycogen stores prior to longer events and loading protocols evolved in the 1960s. These typically involved a 6-day strategy, starting with glycogen depletion (3 days on low carbohydrate diet and training) followed by glycogen supercompensation (3 days with tapered training and high carbohydrate intake; Bergstrom & Hultman 1966). This strategy was shown to boost muscle glycogen stores to ~150–250 mmol·kg wet weight^{-1}.

In the 1980s, it was found that well-trained athletes did not need to include the depletion or glycogen-stripping phase (Sherman et al. 1981). More recent studies show that maximal glycogen storage can be achieved by well-trained athletes in as little as 36–48 h following the last exercise session, proving the athlete rests and consumes an adequate carbohydrate intake (Bussau et al. 2002).

Theoretically, carbohydrate loading could enhance the performance in sports that would otherwise be limited by glycogen depletion (e.g. >90 min; Hawley et al. 1997). Increased pre-event glycogen stores prolong the duration for which moderate-intensity exercise can be undertaken before fatiguing, and may enhance the performance of steady state by ~20% and time-trial performance or the completion of a set amount of work by 2–3%, by preventing the decline in work output (pace) that would otherwise occur (Hawley et al. 1997).

Such preparation of fuel stores may enhance performance in prolonged distance events, but may also be useful for athletes in prolonged intermittent sports, who may otherwise incur fatigue from depleted glycogen reserves. The benefits of carbohydrate loading may be specific not only to the sport but also to the athlete, depending on the requirements of their position or style of play. Of course, the logistics of competition in many sports, where games may be played every day or every second day, might prevent pre-event optimisation of glycogen stores. Indeed, a recent study showed that it is not possible to supercompensate muscle glycogen stores several times within a short time period, although performance can be restored between several bouts of prolonged exercise by high carbohydrate eating (McInerney et al. 2005). An example of an eating plan for carbohydrate loading is provided in Table 32.2.

6.3 Fat adaptation and glycogen restoration strategies

Different dietary strategies have been used to improve endurance performance and especially focusing on optimising muscle glycogen stores. In the classical studies of Christensen & Hansen (1939) and Bergstrom and co-workers (1967), it was shown that a 3–5 day diet consisting primarily of carbohydrates was superior to a fat-rich diet for improving endurance time during exhaustive exercise.

On the other hand, endurance-trained athletes have a high capacity for fat oxidation, and it has been hypothesised that if fat availability to muscle cells was enhanced through the diet, it would increase fat oxidation during exercise, thereby sparing muscle glycogen. Several studies have tested this hypothesis, using both pharmacological and dietary interventions to acutely increase plasma fat availability (Hawley 2002, Kiens & Helge 2000). In most of these studies, plasma fatty acid concentration was only increased slightly relative to baseline, but in studies where the fatty acid concentration was successfully elevated, no clear enhancement of exercise performance was observed. In addition, it is evident from studies including brief high-fat diets lasting less than 7 days that endurance performance was impaired (Bergstrom et al. 1967, Galbo et al. 1979). Longer-term adaptations to a high-fat diet in combination with exercise training might, on the other hand, induce metabolic and morphological skeletal muscle adaptations, which could influence performance and the capacity for fat oxidation during exercise. Training-induced skeletal muscle adaptations include increased capillarisation and enhanced activity of the oxidative enzymes (Henriksson 1977, Kiens et al. 1993), and these all play a significant role in elevating the fat oxidative capacity of muscle. Accordingly, there has been interest in the impact of the combined adaptations to a high-fat diet and endurance training on performance.

In those studies where the dietary period lasted between 1 and 4 weeks, the aerobic fitness level of subjects used

Table 32.2 A carbohydrate loading menu providing carbohydrate intakes of ~10 $g \cdot kg^{-1} \cdot d^{-1}$ for a 65 kg male runner; scale this intake up or down according to body mass

Day	Diet plan (~650 $g \cdot d^{-1}$ carbohydrate)
Day 1 Focuses on carbohydrate-rich foods; other foods can be added to balance the meal. An exercise taper should accompany this menu to optimise glycogen storage. Glycogen supercompensation can be achieved in 2 days with this diet in well-trained runners who can arrange a suitable taper	Breakfast: 2 cups flake cereal + cup milk + banana 250 mL sweetened fruit juice Snack: 500 mL bottle soft drink 2 slices thick toast + jam Lunch: 2 large bread rolls with fillings 200 g carton flavoured yoghurt Snack: Coffee scroll or muffin 250 mL sweetened fruit juice Dinner: 3 cups cooked pasta + ¾ cup sauce and 2 cups jelly Snack: 2 crumpets and honey 250 mL sweetened fruit juice
Day 2	Breakfast: 2 cups flake cereal + cup milk + cup sweetened canned fruit 250 mL sweetened fruit juice Snack: 500 mL fruit smoothie Lunch: 3 stack pancake + syrup + 2 scoops ice-cream 500 mL soft drink Snack: 100 g dried fruit 250 mL sweetened fruit juice Dinner: 3 cups rice dish (fried rice, risotto) Snack: 2 cups fruit salad + 2 scoops ice-cream
Day 3 Many like to increase the focus on low-fibre/low-residue eating on day before competition, thus reaching the line feeling light, rather than with gastrointestinal fullness	Breakfast: 2 cups cereal (low fibre) + cup milk + banana 250 mL sweetened fruit juice Lunch: 4 white crumpets + jam Dinner: 2 cups white pasta + small amount of sauce Over day: 1 L liquid meal drink or 1 L sports drink + 3 sports gels 200 g jelly confectionery

varied from untrained subjects to endurance-trained athletes. Furthermore, the fat content of diets varied from 35 to 80 energy-% and the methods to measure performance varied across studies (Helge et al. 1998, Lambert et al. 1994, Muoio et al. 1994, Phinney et al. 1983). Nevertheless, the effect of these dietary strategies on performance was negligible (Helge et al. 1998, Lambert et al. 1994, Phinney et al. 1983), and improved endurance was only obtained in endurance athletes after 7 days on a semi-high fat diet (Muoio et al. 1994), relative to a carbohydrate-rich diet. In untrained subjects following supervised training through a 4-week intervention while on a high-fat diet, time to exhaustion was increased to a similar extent as when subjects consumed a high-carbohydrate diet (Helge et al. 1998). However, when a fat-rich diet was consumed beyond 4 weeks, impaired performance was evident compared to a carbohydrate-rich diet (Figure 32.3; Helge et al. 1996).

These studies clearly demonstrate that a habitual fat-rich diet used for a short period (up to 4 weeks), and consumed in association with regular endurance training, is not superior to a carbohydrate-rich diet for improving performance. However, when high-fat, low-carbohydrate eating continues beyond 4 weeks, an impairment of training adaptation is evident, relative to the consumption of high-carbohydrate, low-fat diets (Helge et al. 1996).

Other combinations of dietary strategies have been suggested. Such strategies include a short-term high-fat, low-carbohydrate diet, followed by a high-carbohydrate diet to restore muscle glycogen. Such a combination of dietary strategies would seem the perfect preparation for the athlete, simultaneously restoring carbohydrate stores while maximising the capacity for fat oxidation during exercise. Consistent and robust findings are available that a higher total

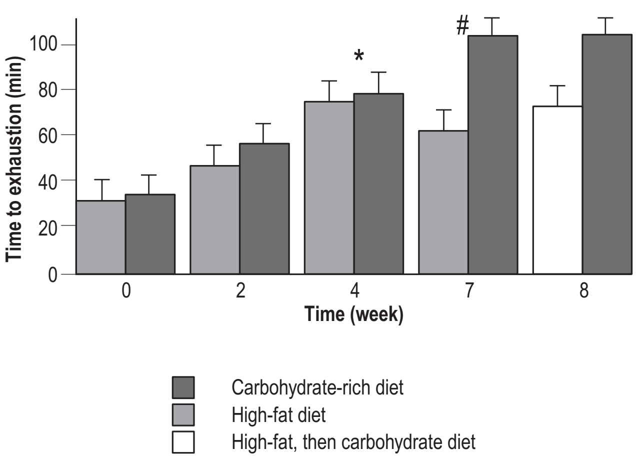

Figure 32.3 Endurance performance (cycle ergometer) after 2–7 weeks on a high-fat diet (65 energy-%) and a carbohydrate-rich diet. After 7 weeks on the high-fat diet, these subjects switched to a high-carbohydrate diet*, significantly different from week 0, significantly different between carbohydrate diet (week 4) and fat plus carbohydrate diet (week 7). Adapted from Kiens & Helge (2000) with permission.

fat oxidation during prolonged exercise is achieved in as little as 5 days training, when using a high-fat diet (65–69 energy-%; Burke et al. 2000, 2002, Carey et al. 2001, Goedecke et al. 1999). A reduction in muscle glycogen stores was also achieved after 5 days on the high-fat diet, and consuming a high-carbohydrate diet for 1 day of rest restored muscle glycogen content but only to its initial levels (Burke et al. 2002, Carey et al. 2001).

To test performance following such dietary strategies, most experiments include a prolonged exercise trial (2–4 h), followed by a time trial. Despite higher fat oxidation during the prolonged exercise trial after the high fat-diet, relative to the high-carbohydrate diet, time-trial performance was not significantly different, and was even slower in some investigations (Havemann et al. 2006). Moreover, after 6 days on a high-fat diet followed by 1 day carbohydrate loading, 1 km sprint power output was significantly lower compared with a high-carbohydrate (only) diet (Havemann et al. 2006).

Interestingly, when carbohydrate loading was extended to 1 week, after high-fat adaptation for 7 weeks, muscle glycogen content was not only restored but was supercompensated, and significantly larger than resulting from a high-carbohydrate (only) diet (Helge et al. 2002). Following the high-fat, low-carbohydrate diet for 7 weeks, an impaired response to training was observed, despite supercompensation of the muscle glycogen stores. In addition, endurance performance only increased slightly and was still impaired compared to the high-carbohydrate, low-fat diet which was consumed during the entire 8 weeks of exercise training (Figure 32.3; Helge et al. 1996).

Thus, what was initially viewed as glycogen sparing after adaptation to a high-fat diet may be a downregulation of carbohydrate metabolism. Accordingly, after long-term fat adaptation followed by 1 week carbohydrate loading, skeletal muscle glucose uptake was impaired, despite a high plasma glucose concentration (Helge et al. 2002). Moreover, despite supercompensation of muscle glycogen, exhaustion occurred when only 37% of the muscle glycogen had been used (Helge et al. 2002). It has also been shown that fat adaptation/carbohydrate restoration strategies were associated with a reduced pyruvate dehydrogenase activity during 20 min of steady-state cycling (Stellingwerff et al. 2006).

Therefore, there is now evidence that such dietary strategies may result in a downregulation of carbohydrate metabolism or glycogen impairment during exercise. Moreover, adaptations to high-fat diets also increase heart rate (Havemann et al. 2006, Helge et al. 1996) and sympathetic activation, as measured by plasma epinephrine concentration (Helge et al. 1996, Sasaki et al. 1991) during submaximal exercise, and these trends persisted despite restoring muscle glycogen to supercompensated levels (Helge et al. 1996). Accordingly, it seems that fat adaptation and fat-loading strategies cannot be considered as valuable methods (Burke & Kiens 2006).

6.4 Pre-event eating (1–4 h)

It is well known that exercise metabolism and performance are influenced by the composition and amount of energy in the diet. The pre-event meal provides the athlete with a final opportunity to address fluid and carbohydrate needs for competition, whilst avoiding gastrointestinal problems during competition (balancing feelings of hunger against gastrointestinal discomfort, vomiting or diarrhoea). Therefore, much emphasis has been on the timing, the dietary composition, the types of carbohydrates and the total energy of the pre-exercise meal, either before training or competition.

The ingestion of a carbohydrate-rich meal can result in a rapid and large increase in plasma glucose and insulin concentrations. The plasma insulin elevation facilitates glucose uptake and decreases lipolysis, possibly resulting in increased glucose and glycogen use during exercise. These metabolic perturbations can persist for up to 6 h after carbohydrate ingestion (Montain et al. 1991).

With regard to timing of the pre-exercise meal, when comparing the effect between a meal ingested 3–4 h before exercise and an overnight fast on endurance performance, no differences in a 10 km time trial were found when participants had breakfast containing 250 g of carbohydrates, 4 h before, when compared with no meal (Whitley et al. 1998). On the other hand, endurance was significantly greater after breakfast (100 g of carbohydrates and milk) ingested 3 h before steady-state exercise (70% maximal oxygen uptake) to exhaustion, when compared with an overnight fast (Schabort et al. 1999). Similarly, Casey et al. (2000) found that a meal 3–4 h before exercise improved performance, relative to exercise in the fasting state. This is mainly due to an optimisation of liver glycogen stores, as these are substantially reduced after an overnight fast. Based on this, a carbohydrate meal 3–4 h before exercise is better than exercising in the overnight fasting state.

The amount of carbohydrates ingested 3–4 h before exercise seems also to play a role. For instance, while endurance can be improved (Sherman et al. 1989), eating large amounts of carbohydrate 3–4 h before exercise may cause gastrointestinal discomfort in some individuals. Thus, drinking carbohydrate solutions 3–4 h before exercise has been suggested.

Furthermore, not all carbohydrates elicit similar metabolic effects. Thus, when foods with a low-glycaemic index are ingested 3 h before prolonged exercise, increases in plasma glucose and insulin concentrations during the postprandial period and during exercise are smaller. The reduction in fatty acid mobilisation is less and carbohydrate oxidation during exercise is lower, as compared to ingestion of food items of a high glycaemic index (Wee et al. 1999). However, neither high- nor low-glycaemic carbohydrate ingestion (3 h before exercise) appeared to be either detrimental or advantageous to performance (Wee et al. 1999).

Carbohydrates ingested in the hour before exercise will induce a rapid fall in blood glucose concentration during the first period of exercise due to the enhanced plasma glucose and insulin concentrations at onset of exercise, and in some cases hypoglycaemia will last for a considerable time. The degree of metabolic perturbation seems also in this situation to be related to the glycaemic index of the carbohydrates. When a pre-exercise meal consisting of carbohydrates with a low-to-moderate glycaemic index was ingested 30–60 min before exercise, a lower plasma glucose and insulin response was observed before exercise start and hypoglycaemia did not occur compared to when carbohydrates of high glycaemic index were ingested (DeMarco et al. 1999, Kirwan et al. 2001, Thomas et al. 1991) During the following exercise session, the plasma glucose concentration was increased when the low-to-moderate glycaemic index meal was consumed, compared to when the high-glycaemic index carbohydrate meal was consumed (DeMarco et al 1999, Thomas et al. 1991).

Exercise performance (time to exhaustion) is markedly increased after consuming low and moderate glycaemic index pre-exercise meals, compared to high glycaemic index meals (DeMarco et al. 1999, Kirwan et al. 2001, Thomas et al. 1991). However, other studies show no performance change, despite metabolic alterations before and during exercise (Febbraio et al. 2000, Wee et al. 1999). This disparity could be explained by differences in study design, exercise intensities and the training status of subjects, but it may also be due to the extent of the metabolic perturbation caused by the ingested carbohydrates which, in turn, may be related to meal timing, the amount of carbohydrates ingested and the glycaemic index of those carbohydrates. However, data also indicate improved performance after ingesting a low-to-moderate glycaemic index pre-exercise meal, if the meal can maintain euglycaemia during exercise (Burke 2006).

An aspect to consider regarding the composition and timing of the pre-exercise meal is the rapid fall in plasma glucose during exercise, especially after ingesting high glycaemic index carbohydrates <1 h before exercise. This phenomenon is more likely to occur when the exercise intensity is low. Achten & Jeukendrup (2003) showed that when exercising (55%, 77%, 90% maximal oxygen uptake) after the ingestion of a 75 g carbohydrate solution, glucose concentration decreased within the first 5 min, and to a similar extent at all intensities. On average, no evidence of hypoglycaemia was evident. However, on an individual basis, several of the subjects developed hypoglycaemia at each of the three intensities. When the 75 g carbohydrate solution was ingested 15, 45 or 75 min before 20 min of submaximal exercise (65% maximal), followed by a time trial, subjects became hypoglycaemic during the first 10 min of exercise in all situations, but this did not affect performance in the subsequent exercise bout. Thus, some athletes are more prone to developing hypoglycaemia when exercise is performed <1 h after high glycaemic carbohydrate ingestion, and some individuals are also more sensitive to low plasma glucose concentrations, which is important to consider when planning a pre-exercise meal for <1 h before competition.

The athlete should also be conscious of fluid needs and consume enough fluid to ensure that adequate hydration is achieved before competition. This includes restoring losses from previous training or competition, and from intentional dehydration strategies to fine tune body mass (Chapter 33).

6.5 Fuelling during events

When food and fluid consumption can occur during competition, it is important to consider the interaction of strategies undertaken before and during exercise, particularly in relation to fuel metabolism and performance. Carbohydrate consumed during exercise changes the metabolic impact of carbohydrates eaten prior to exercise (Burke et al. 1998). There is also some evidence that the benefits of combining these two strategies to enhance carbohydrate availability for endurance exercise are additive (Chryssanthopoulos & Williams 1997, Wright et al. 1991). However, another study found that ingesting carbohydrates before exercise is only beneficial to time-trial performance late during exercise, when there is further intake of carbohydrate during the session (Febbraio et al. 2000). This deserves further study.

6.6 Postevent recovery

Following a competitive event, the nutrition needs are similar to, if perhaps sometimes more exaggerated than, those following heavy training sessions. The more rapidly an athlete must return to competition or training, the more important it will be to rehydrate, refuel and repair from the first session. Thus, athletes who compete in tournaments, stage races or events involving heats and finals should be directed to follow the prescribed recovery eating strategies described above. Some consideration of the practical issues involved in achieving these nutritional goals may be needed, since many athletes are required to travel interstate or internationally for their most important competitions (Table 32.3).

Table 32.3 Challenges and solution for the travelling athlete

Challenges of travelling	Strategies to cope with the challenges of travelling
• Disruptions to normal training routine and lifestyle • Changes in climate and environment that modify nutritional needs • Jet lag • Changes in the availability of familiar foods • Reliance on hotel, restaurant and takeaway foods instead of home cooking • Exposure to new foods and eating cultures • Temptations of an 'all you can eat' dining hall in an athletes' village • Risk of gastrointestinal illnesses due to exposure to food and water with poor hygiene standards • Excitement and distraction of a new environment	**1. Planning ahead** ○ Investigate food issues on travel routes (e.g. airlines) and at the destination before leaving home. Caterers and food organisers should be contacted well ahead of the trip to let them know meal timing and menu needs. **2. Taking supplies to supplement the local fare** ○ A supply of portable and non-perishable foods should be taken to the destination to replace important items that may be missing. **3. Eating and drinking well en route** ○ Many will turn to boredom eating when confined. They should eat according to real needs, taking into account the forced rest while travelling ○ When moving to a new time zone, the athlete should adopt eating patterns that suit their destination as soon as the trip starts. This will help the body clock to adapt. ○ Unseen fluid losses in air-conditioned vehicles and pressurised plane cabins should be recognised, and a drinking plan should be organised to keep athletes well hydrated. **4. Taking care with food/water hygiene** ○ If the local water supply is unsafe to drink, then drink from sealed bottles, or hot drinks made from well-boiled water. Ice added to drinks is often made from tap water and may be a problem. ○ In high-risk environments, eat only at good hotels or restaurants. Food from local stalls and markets should be avoided. ○ Food that has been well cooked is the safest; it is best to avoid salads or unpeeled fruit that has been in contact with local water or soil. **5. Adhering to the food plan** ○ Choose the best of the local cuisine to meet nutritional needs, supplementing with your own supplies where needed. ○ Be assertive in asking for what is needed at catering outlets (e.g. low-fat cooking styles or an extra carbohydrate choice). ○ The challenge of 'all you can eat' dining should be recognised. Resist the temptation to eat 'what is there' or 'what everyone else is eating'. Follow your own meal plan.

7 CONCLUSION

Various nutrition strategies assist the athlete to train hard in preparation for an event, providing the energy, fuel and nutrient requirements set by their workouts and allowing the athlete to recover between sessions. In some cases, dietary manipulations can assist the athlete to achieve the physique that promotes good performance. For competition, athletes should consider the factors that limit performance or cause fatigue in their event. In many cases, nutritional strategies can be undertaken to reduce or delay the onset of fatigue. Dietary strategies for optimal performance will vary among sports and in some cases, even among athletes in the same sport. Therefore, the athlete should seek professional advice from a sports dietician to determine the strategies that may be of benefit, then experiment to find a nutritional plan that allows optimal training and competition.

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