



FC Barcelona Sports Nutrition Guide

The evidence base for FC Barcelona

Sports Nutrition Recommendations

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EXECUTIVE SUMMARY

Futbol Club Barcelona (FCB) is a centre of sporting excellence where it is recognized that appropriate nutrition is essential to health and performance. The Gatorade Sports Science Institute (GSSI)'s mission is to help athletes improve their health and performance through research and education in hydration and nutrition science. This document is the result of a partnership between FCB and GSSI and aims to provide FCB with world class and evidence based sports nutrition information. More specifically, this documents contains the evidence base of sports nutrition recommendations at FCB. A following document will turn these theoretical, literature based recommendations into practice.

In brief, daily carbohydrate intake should reflect the intensity, duration and frequency of the athletes' exercise activity and may also vary between individuals. The quantity and type of carbohydrate ingested before, during (and after) exercise should be determined by the type of activity in which the athlete participates. Dehydration can negatively impact on physical and skill performance. Fluid losses as a consequence of sweating during exercise will vary significantly between individuals, and depend on the intensity of exercise as well as environmental conditions.

For optimal adaptation it is recommended to consume sufficient quantities (20-25g) of high quality protein at regular intervals during the day, equating to approximately 1.5-2.0 g/kg of an athletes body mass. Very few nutrition supplements have an evidence base and the use of dietary supplements should be under the direction and monitoring of an appropriate sports nutrition/medicine/science practitioner. Thus, individualised drinking and fueling plans before, during and after exercise are recommended with supplementation where necessary. It is also recommended that the nutrition intake is guided by factors such as goals, environment, type and intensity of exercise.

Because there are many variables that determine the specific nutritional needs of each athlete it is important to develop strategies that take these needs into account. It is generally acknowledged that personalized sports nutrition will become the norm in elite sport.





1. INTRODUCTION

Futbol Club Barcelona (FCB) is a centre of sporting excellence where it is recognized that appropriate nutrition is essential to health and performance of athletes and staff. The Gatorade Sports Science Institute (GSSI)'s mission is to help athletes improve their health and performance through research and education in hydration and nutrition science. This document is the result of a partnership between FCB and GSSI and aims to provide FCB with world class and evidence based sports nutrition information. This document will inform all practical implementation of sports nutrition at FCB and is the manual for all staff, who want to know more about sports nutrition. The document will make sure that nutrition information shared across FCB is uniform and up to date.

FCB comprises of professional, amateur and youth athletes. There are over one hundred professional players involved in football, basketball, futsal, handball and roller hockey. Amateur sections include volleyball, field hockey, ice hockey, athletics, rugby and figure skating, involving over 250 high performance athletes. Finally, for each sport at FCB there is youth development, with the aim of nurturing new and exciting talent through to senior teams. FCB is renowned for its specialism in team sports and specifically football. Team sport performance is characterised by high intensity efforts such as sprints, changes of direction and jumping, interspersed with periods of low, to moderate level activity. High performance is expected in all categories at FCB, however, it is recognised that each sport and athletic population has its own unique demands and challenges. For example, the energy demands of exercise and the optimal physique of the athlete will vary significantly between sports. To achieve excellence in their sport, athletes must complete the hours of training required to develop the high levels of skill proficiency and physical capabilities, which are superior to their opponents. To this end, the aim of sports nutrition is to assist the athlete with the preparation, execution and recovery from the demands of their sports training and competition.

At FCB the different sports have varied competition programmes, with seasons of varying durations and matches played at various times of year. Furthermore, team programmes are often sporadically modified by additional competitions, such as knockout cups, continental tournaments and national team fixtures. The co-ordination of nutritional strategies would





therefore seem difficult to implement between sports. However, it is important to note that the sports nutrition required to support performance is not specific to the sport but specific to the individual athlete. Thus, similar nutrition principles can be applied to individuals across a spectrum of sporting activities.

At present there is much misinformation on nutrition, especially with the ingestion of food acutely round exercise and the use of nutrition supplements. **This document will be available to all staff at FCB and provides the evidence base on which practical recommendations at FCB are based.** The most effective way to promote a culture of good nutrition practice is to adopt a multi-disciplinary approach, empowering all people who are “touch points” for players to reinforce nutrition messages. This document will be the source of a consistent message with regard to nutrition education, both internally at FCB and also to the wider global football and sporting community.

The food options available to athletes at FCB may be dependent on current nutrition partnerships or commercial contracts. Regardless, the use of any foods or products will be founded on rigorous criteria and supported by appropriate scientific research. Thus, the present document will provide the facts which underpin current nutrition guidelines. As new research and evidence is published, recommendations will be modified accordingly. To achieve this, the present document will be reviewed and updated bi-annually.

This document will be the source document for sports specific nutrition advice which will translate scientific findings, which often use complicated terminology and less tangible outcomes, into simple and practical food equivalents. This document has been prepared by the medical team at FCB and the Gatorade Sports Science Institute (GSSI). The FCB medical team are world leaders in their field and the GSSI are a global authority on sports nutrition, leading to a potent collaboration. All information is based on peer reviewed research and summaries are a consensus view of both FCB and the GSSI.



2. ENERGY BALANCE

Body composition can be an important element of performance and health and energy balance is an important determinant of body composition. The aim of this chapter is to provide an overview of our current understanding of energy balance and its impact on body composition.

Energy

Energy is provided to the athlete via all the foods and beverages in their diet. The energy ingested via food is used by the athlete in several fundamental processes including cellular maintenance, thermoregulation, growth, reproduction, immunity and locomotion (Loucks et al., 2011). When athletes train or compete, energy expenditure is increased significantly, which increases the need for additional energy to be ingested through the diet.

Energy availability to the athlete has been defined as the amount of energy intake minus the energy expended during exercise. Thus, energy availability is the energy available to input to the body's physiological systems. If the total energy expenditure of the athlete, including exercise-related expenditure and the energy required to support daily physiological function, exceeds that of energy intake, the athlete is said to be in negative energy balance.

Alternatively, if energy intake exceeds total energy expenditure, the athlete will be in positive energy balance.

Energy-related definitions

- EA = energy availability
- EI = energy intake
- EEE = energy expended in exercise
- EB = energy balance
- TEE = total energy expenditure

Energy-related equations

- $EA = EI - EEE$
- $EB = EI - TEE$



As athletes work towards sport-specific goals they will engage in different diet and exercise behaviours that will impact on EA. When athlete's modify training to expend more or less energy, dietary modification becomes an important part of the nutritional strategy to increase or reduce EA and help in the management of body composition (Loucks et al., 2011). The main factors governing the energy expended during exercise are the intensity and duration of training/matches.

Furthermore, the composition of the athlete's body and their metabolism will also influence energy expenditure. For example, athletes with more muscle expend more energy, even when at rest. To this end, the energy requirements will vary depending on the sport and individual athlete involved. The diet and training programs need to be carefully managed to modify body composition, achieve performance goals and avoid ill health.

The challenge for athletes at FCB is to make appropriate adjustments to their diet to compensate for energy expended during the participation in their sport. In team sports like football, the average calorie expenditure of a single match/training session has been reported to be approximately 1000-1500 Kilocalories (Kcal) (Bangsbo et al., 2006). As a general guide, for healthy young adults the diet should contain approximately 45 kcal / kg FFM per day (Loucks et al., 2011). In a "typical" 70 kg athlete, body fat percentage body fat is approximately 10% (~7 kg of fat), bone mineral content is 3-4% (3 kg), with the remaining 86 % (~60 kg) assumed to be lean mass. Therefore, EA without counting physical activity would be more than 30 kcal / kg lean mass (1800-2000 kcal). Research suggests that when EA is less than 30 kcal / kg of lean muscle mass per day, problems such as tiredness, fatigue, reduced performance and compromised immune function may follow (Burke 2003; Loucks 2004). Of note, athletes engaging in sports with an emphasis on low body fat, typically increase their training hours and make more frequent attempts to lose weight. It is known that the energy intakes of these athletes can be very low (Erp van-Baart et al., 1989; Dahlstrom et al., 1990). As a consequence, these athletes are reported to be twice as likely of upper respiratory tract infections in comparison to athletes maintaining EB (Hagmar et al., 2008). Excessive training, chronic low energy, low nutrient intake and psychological distress are often involved in fat loss strategies and may cause long-term damage to health, well-being and consequently performance. Therefore, training periodisation in sport requires a



corresponding periodisation of EA to meet the objectives of the training and to support adaptation.

70 kg athlete: $45 \text{ kcal} \times 70 \text{ kg} = 3150 \text{ kcal/day}$

1000 Kcal can be used in training or matches

2150 kcal /day for other energy needs = “energy availability”

Studies suggest that an athlete’s appetite and *ad libitum* intake of food is an unreliable indicator of energy requirements. Athletes engaging in intensive or prolonged training should be advised to eat by discipline i.e. eat specific quantities of particular foods at designated times (Loucks et al., 2011).

Finally, research suggests that an athlete’s key influencer is their coach. With that regard, implementing nutritional strategies should not be isolated to the “club nutritionist”. Instead, behaviour modification will be most effective when all staff surrounding an athlete understands the importance of EA and nutritional strategies. This can be achieved by small-group nutrition workshops and specifically designed handbooks i.e. to target coaches. Furthermore, involving the coach or having coaching staff present at routine athlete weight monitoring can have a large impact on an athlete’s adherence to a nutrition/exercise program.

Body Composition

Physical characteristics, such as height, body mass (BM), muscle mass and body fat levels can all play a role in the performance of sport. Athletes often want to change their BM in an attempt to improve performance. An athlete’s physique is determined by inherited characteristics, as well as the conditioning effects of their training program and diet.

A report recently approved by the International Olympic Committee’s Medical Commission detailed concerns that athletes engaging in inappropriate strategies to reduce body fat such as severe food restriction are at risk of damaging their health (Meyer et al., 2013). Often, “ideal” physiques for individual sports are set, based on a rigid set of characteristics of successful athletes. However, this process fails to take into account that there is considerable variability in the physical characteristics of sports people, even between elite athletes within the same sport. Research has reported that the typical body fat percentage of elite male football players can range between 7 and 19% (Wittich et al., 2001; Reilly et al., 2009). It is





discouraged to establish rigid prescriptions for individuals. Instead, it is advised to nominate a range of acceptable values for body fat and BM for individuals within each sport. Routine monitoring of BM provides a quick and non-invasive method to monitor the player's health and also generates values that may be related to performance. In elite athletes, body composition should be monitored at regular intervals during the season. Seasonal trends reflect an increase in body fat levels during the off season, which are then reduced during the preseason, where training volume is highest (Carling & Orhant 2010). It is also common for lean (muscle) mass to be reduced during heavy training volumes in some players, if adequate energy is not ingested.

The methods of assessing body composition will differ greatly depending on the sport and the resources at the team's disposal. Regardless of the protocol, standardisation of the procedure is fundamental to tracking meaningful changes. A position statement on body composition was published in 2012 under the auspices of the International Olympic Committee's Medical Commission, providing a guide for practitioners working in sport (Ackland et al., 2012). In summary, anthropometry (skinfolds) provides a reliable 'in the field' measurement, allowing the practitioner to measure skinfold and muscle girths. . Nevertheless, education of players and coaches is required when feeding back results as a 'sum of 8', as opposed to the often preferred body fat percentage.

Dual-Energy X-Ray Absorptiometry (DXA) is now commonly used at elite clubs. Dual-Energy X-Ray Absorptiometry provides a relatively precise estimate of fat mass, bone mineral content and lean mass. The popularity of DXA is most likely due to scans being relatively quick and easy to perform, whilst providing precise and attractive data to feedback to the players. It is important to note that DXA also has limitations and strict standardization procedures should be adhered to if meaningful changes are to be identified (Clarys et al., 2010; Nana et al., 2014). The error in DXA measurement of fat mass and muscle mass is approximately 1 kg. As a consequence, small differences of interest to the practitioner and athlete during routine monitoring are difficult to detect (Clarys et al., 2010; Nana et al., 2014). Thus, despite adhering to strict standardization procedures, errors will be present independent of method used. Therefore, all body composition results need to be interpreted with care and, typically, comparison with different methodologies should be avoided.





Novel methods of body composition assessment continue to be proposed, of these ultrasound shows some promise. Although ultrasound research is in its infancy, the method may offer a valid and more accurate alternative to skinfolds and more expensive measures of assessment (Pfeiffer et al., 2010; Meyer et al., 2013).

It is important to recognise populations known to be most at “risk” of experiencing the detrimental effects of a negative EB. Recently the “female athlete triad”, the co-existence of disordered eating, disturbed menstrual function and sub-optimal bone density has received considerable publicity (Loucks 2003; Birch 2005; Beals & Meyer 2007). Expert advice from sports medicine professionals, including dietitians, psychologists and physicians, is important in the early detection and management of problems related to body composition and nutrition.





3. CARBOHYDRATE REQUIREMENTS FOR ATHLETES

Fuel provided from ingesting carbohydrate plays a major role in the performance of many types of exercise and sport. Carbohydrate and fat are the fuels, which provide the player with the energy required for training and matches. The relative contribution of these fuels during exercise will depend on several factors, including the pre-exercise carbohydrate stores (muscle and liver glycogen), the exercise intensity and duration, and the training status of the player (Jeukendrup 2003). Unfortunately, total body carbohydrate stores are limited and are often substantially less than the fuel requirements of the training and competition sessions undertaken by athletes. The depletion of body carbohydrate stores is a cause of fatigue or performance impairments during exercise, particularly during prolonged (90 min or longer) sessions of sub-maximal or intermittent high-intensity activity. This fatigue may be seen both in the muscle (peripheral fatigue) and in the central nervous system (central fatigue) (Noakes 2000; Nybo 2003). Therefore, strategies for athletes include consuming carbohydrate before, during and in the recovery period between exercise bouts.

Many studies have shown that exercise is improved by strategies that enhance or maintain carbohydrate status during exercise (Bergström et al., 1967; Bergstrom & Hultman 1967; Hultman 1967) (for detailed reviews see (Hawley et al., 1997; Jeukendrup & Jentjens 2000; Jeukendrup 2004)). Recent studies have provided preliminary evidence that there are additional benefits to be gained from carbohydrate eating strategies apart from increasing fuel stores for exercise. For example, there are a growing number of investigations that have reported performance benefits when carbohydrate is consumed before and during high intensity exercise of ~1 hour (Anantaraman et al., 1995; Below et al., 1995; Jeukendrup et al., 1997; Carter et al., 2003; Carter et al., 2005; Rollo & Williams 2009). In these situations, the athlete's body carbohydrate stores should already be sufficient to fuel the event. Thus, it is not clear why additional carbohydrate would provide an advantage. It is possible that carbohydrate intake improves the function of the brain and central nervous system, reducing the perception of effort during the exercise task. In line with this, infusion of glucose has been shown not to affect performance (Carter et al., 2004), but a mouth rinse with a carbohydrate solution (without swallowing the solution) has been reported to improve both running (Rollo et al., 2010) and cycling performance (Carter et al., 2004). The potential





activation of brains areas involved with exercise regulation and reward through stimulation of receptors in the mouth, by the rinsed carbohydrate, has been cited as the potential mechanism; highlighting the metabolic and central benefits of carbohydrate.

Carbohydrate intake during and after exercise also appears to assist the immune response to exercise (Gleeson 2000; Gleeson 2006; Nieman & Bishop 2006; Nieman 2007). Cellular immune parameters are often reduced or compromised after a prolonged workout. It is reasonable to assume that acute improved immune status, via carbohydrate ingestion, may result in less sick days experienced by the athlete. However, this is still to be determined.

Carbohydrate Prior to Exercise

The daily intake of carbohydrate should be proportionate to the estimated fuel cost of the training session or match. It is unlikely that players complete matches or high-intensity sessions on a daily basis, especially during the season. Therefore, current guidelines recommend that for low intensity, recovery or skill-based training players ingest 3-5 g of carbohydrate/ kg BM/ day. When the intensity or duration of training is increased i.e. when players complete moderate training, approximately 1 h a day, carbohydrate intake of 5-7 g/kg BM/ day is recommended (Burke et al., 2011). Intake requirements above this range are less common in team sport athletes, however, during pre-season when the need to condition the players is of importance, the daily carbohydrate range may be 6-10 g/kg BM/ day to support 1-3 hours of training (Burke et al., 2011). Such intakes though should be considered along with player-specific body composition changes, which are often desired and seen in pre-season (Table 1).

Carbohydrate stores in the muscle and liver should be well-filled prior to exercise. This becomes particularly important for intense training or competition where the athlete wants to perform at their best. The key factors in glycogen storage are dietary carbohydrate intake, and in the case of muscle stores, tapered exercise or rest. In the absence of muscle damage, muscle glycogen stores can be returned to normal resting levels (to 350-500 mmol/kg dry weight muscle) with 24-36 h of rest and an adequate carbohydrate intake (7-10 g /kg BM/ day) (Bussau et al., 2002). Normalized stores appear adequate for the fuel needs of events of less or equal to 60-90 min in duration – for example, a football game, a half-marathon or basketball game.



Although strategies to promote carbohydrate availability have been shown to enhance performance and recovery after a *single* bout of exercise, it has been difficult to demonstrate that a *long-term* period of high carbohydrate eating will promote better training adaptations and long-term performance than a moderate carbohydrate diet. Theoretically, inadequate carbohydrate intake during repeated days of exercise will lead to gradual depletion of muscle glycogen stores and impairment of exercise endurance (Costill et al., 1971). Contrary to this, recent investigators have suggested that training in a low glycogen state may have some beneficial effects on the adaptation of the muscle (Rauch et al., 1995; Hawley 2011; Philp et al., 2011). However, further research is required to identify situations both in health and performance where training with low glycogen levels may be beneficial. Thus, athletes and coaches need to consider the advantages and disadvantages of training with low carbohydrate to support a periodised training program.

Dietary guidelines for the general population make recommendations for carbohydrate intake as a percentage of dietary energy intake (for example, to increase carbohydrate to greater than 55% of total energy intake). However, athletes undertaking strenuous exercise have daily carbohydrate requirements based primarily on muscle fuel needs which are quantifiable according to the size of the athlete and the duration/intensity of their exercise program. Therefore it makes sense, and it is advisable, to be consistent in describing daily carbohydrate intake goals in terms of grams per kg of the athlete's body mass. This allows the athlete and practitioner to quickly calculate the carbohydrate requirement for a given situation - for example 7g/ kg BM for a 70 kg athlete equates to 490 g. Meals and menus can then be constructed using information on food labels or in food composition tables to achieve this carbohydrate target.

Carbohydrate loading is a nutrition strategy that aims to maximize or "supercompensate" muscle glycogen stores up to twice the normal resting level (e.g. ~500-900 mmol/kg dry weight). Ingesting a diet rich in carbohydrate for several days has been shown to result in a "supercompensation" of muscle glycogen stores and prolonged the cycling time to exhaustion (Sherman et al., 1981). Specifically, this model consists of a 3-4 day "depletion" phase of hard training and low carbohydrate intake, followed by a 3-4 day "loading" phase of high carbohydrate intake and exercise taper (i.e. decreased amounts of training) (Sherman





1983). More recent studies suggest that a high carbohydrate diet combined with rest can result in “supercompensated” muscle glycogen in as little as a 24-36 h (Bussau et al., 2002). Furthermore, the trained muscle appears to have the ability to store more glycogen in comparison to untrained muscle and thus be more susceptible to “supercompensation” protocols (McInerney et al., 2005). Studies undertaken on trained subjects have produced a “modified” carbohydrate loading strategy (Sherman et al., 1981). The muscle of well-trained athletes has been found to be able to “supercompensate” its glycogen stores without a prior depletion or “glycogen stripping” phase. For well-trained athletes at least, carbohydrate loading may be seen as an extension of “fuelling up” – involving rest/taper and high carbohydrate intake over the 3-4 days leading up to a contest. The modified carbohydrate loading protocol offers a more practical strategy for competition preparation, by avoiding the fatigue and complexity of the extreme diet and training protocols associated with the previous depletion phase. Typically, carbohydrate loading will postpone fatigue and extend the duration of steady state exercise by ~ 20%, and improve performance over a set distance or workload by 2-3% (Hawley et al., 1997). However, it is important to note that no “supercompensation” of muscle glycogen concentrations have been reported 48 h after a football match, a typical response reported following prolonged running or cycling exercise. Thus, this will have implications for when athletes engaging in this type of activity are next ready to perform.

Pre-exercise meals

The ingestion of food and fluids consumed in the 4 h prior to an event has several important objectives. First, if glycogen stores are not fully restored after the last exercise, carbohydrate ingestion can help continue to fill muscle glycogen stores and to restore liver glycogen. This is especially important for events undertaken in the morning where liver stores are depleted from an overnight fast. Second, the ingestion of fluid ensures that the athlete is well-hydrated and also helps to prevent feelings of hunger. Finally, it is the food ingested in the 4 h prior to exercise that becomes engrained into an athlete’s preparation, impacting on psychology and superstition. Players should practice any pre-exercise nutrition strategies in training before adopting in competition, to determine personal tolerances and minimise adverse effects.





Consuming carbohydrate-rich foods and drinks in the pre-exercise meal is especially important in situations where body carbohydrate stores have not been fully recovered and/or where the event is of sufficient duration and intensity to deplete these stores. The intake of a substantial amount of carbohydrate (~200-300 g) in the 3-4 h before exercise has been shown to enhance various measures of exercise performance compared to performance undertaken after an overnight fast (Sherman et al., 1989; Wright et al., 1991; Schabert et al., 1999). For example, the importance of glycogen to football performance has resulted in the widely utilised “pre-match meal” strategy. The focus of the pre-match meal is to ingest an easy to digest high-carbohydrate meal 3-4 hs before exercise, to increase resting levels of glycogen in the muscle and liver. On match day the relative gains in endogenous glycogen stores achieved with carbohydrate feedings will be dependent upon starting concentrations and the training status of the muscle. However, as a guide, after an overnight fast, ingesting a meal containing 2.5 g of carbohydrate per kg of the player’s body mass has been reported to increase muscle glycogen by 11-15% and liver glycogen by 33%, 3 h after ingestion (Taylor et al., 1996; Wu & Williams 2006).

Immediately prior to the warm up or match (depending on individual preference) players may ingest carbohydrate (25-30g) to blunt the release of glucose from the liver, thus sparing the hepatic store of glycogen (Howlett et al., 1998). The role of liver glycogen is the regulation of blood glucose concentrations (euglycaemia: 4-5.5 mmol·l⁻¹). At the onset of a match, muscular contraction will cause an increased uptake of glucose from the blood. In concert, liver glycogenolysis will be activated by the actions of glucagon and adrenaline. Interestingly, blood glucose has been reported to be elevated during repeated sprint activity and is rarely observed to decrease to concentrations that may impact on performance (Krustrup et al., 2006). These findings would suggest that the rate of glucose release from the liver is sufficient to compensate for the use of blood glucose throughout 90 min of football activity, in well fed players. In fact, during football blood glucose is only lowered during the “half time” period. This is most likely a consequence of the continued uptake of glucose by the previously active muscle and a reduction in liver glycogenolysis, via a lowered catecholamine level during this period of recovery (Krustrup et al., 2006).

It is important to note that during prolonged match play, i.e. into extra/over time and penalties, blood glucose concentrations will fall and if not replenished may result in





hypoglycaemia (Foskett et al., 2008). Symptoms of hypoglycaemia include sub-optimal functioning of the central nervous system, which might have implications for physical and technical skill performance (Vergauwen et al., 1998; Nybo 2003; McRae & Galloway 2012). The preservation of blood glucose concentrations appears preferential when executing complex skills that require high levels of central nervous system activation, particularly during high intensity intermittent activity (McMorris & Graydon 1997; Winnick et al., 2005). Therefore, it is reasonable to conclude that maintaining blood glucose would maintain “skill execution” under circumstances of fatigue and or hypoglycemia.

However, it has also been suggested that carbohydrate intake before exercise may have *negative* consequences for performance, especially when it is consumed in the hour prior to exercise. Carbohydrate intake causes a rise in plasma insulin concentrations, which in turn lowers plasma glucose concentration and suppresses the availability and oxidation of fat as an exercise fuel. The final result is an increased reliance on carbohydrate oxidation at the onset of exercise – leading to faster depletion of muscle glycogen stores and a further decline in plasma glucose concentration (rebound hypoglycemia) (Koivisto et al., 1981). There has been considerable publicity surrounding one study from the 1970s, which found that subjects performed *worse* after consuming carbohydrate in the hour before exercise than when they cycled without consuming anything (Foster et al., 1979). This has led to warnings that carbohydrate should not be consumed in the hour before exercise. However, a far greater number of studies have shown that any metabolic disturbances following pre-exercise carbohydrate feedings are short-lived or unimportant (Hargreaves et al., 2004). These studies show that carbohydrate intake in the hour before exercise is associated with a neutral performance outcome (Jentjens et al., 2003; Jentjens & Jeukendrup 2003; Moseley et al., 2003).

Nevertheless, there may be a small subgroup of athletes who experience a true fatigue, associated with a decline in blood glucose levels (Jentjens & Jeukendrup 2002), if they start to exercise within the hour after consuming a carbohydrate snack. This problem can be avoided or diminished by a number of dietary strategies:

- Consume carbohydrate 5-10 min before the start of the exercise or incorporate this into a warm-up. By the time insulin starts to rise, the exercise has already started and insulin release will be suppressed by catecholamines.





- Consume a substantial amount of carbohydrate (> 75 g) rather than a small amount, so that the additional carbohydrate more than compensates for the increased rate of carbohydrate oxidation during the exercise.
- Choose a carbohydrate-rich food or drink that produces a low Glycemic Index (GI) response (that is, a low blood glucose and insulin response) rather than a carbohydrate source that has a high GI (producing a large and rapid blood glucose and insulin response).
- Consume carbohydrate throughout the exercise session.

The type, timing and quantity of pre-event meals should be chosen according to the athlete's individual circumstance, experience and preference. Foods with a low-fat, low-fibre and low-moderate protein content are the preferred choice for the pre-event meal since they are less likely to cause gastrointestinal upsets (Jeukendrup & Killer 2010).

Carbohydrate intake during exercise

Numerous studies show that the intake of carbohydrate during prolonged sessions of moderate-intensity or intermittent high-intensity exercise can improve endurance (i.e. prolong time to exhaustion) and performance. There is some evidence that increasing carbohydrate availability causes glycogen sparing in slow-twitch muscle fibres during running (Tsintzas et al., 1995), while alternative mechanisms to explain the benefits of carbohydrate feedings during prolonged exercise include the maintenance of plasma glucose concentration (sustaining brain function) and the provision of an additional carbohydrate supply to allow the muscle to continue high rates of carbohydrate oxidation (Coyle et al., 1986; Jeukendrup 2004). The precise mechanism remains subject to debate, however, it is likely to involve a combination of factors incorporating the maintenance of plasma glucose levels and carbohydrate oxidation rates, as well as the protection of muscle and liver glycogen stores (see Cermak and van Loon, 2013, for a review).

Most carbohydrates (glucose, sucrose, maltose, maltodextrins, amylopectin) are oxidized at relatively high rates whereas other carbohydrates (i.e., fructose, galactose, trehalose) are oxidized at slightly lower rates (Leijssen et al., 1995). Carbohydrate consumed during exercise is oxidized in small amounts during the first hour of exercise (~ 20 g) and thereafter reaches a peak rate of ~60 g/h. Even ingestion of very large amounts of carbohydrate will not





result in higher oxidation rates (Jeukendrup & Jentjens 2000; Jeukendrup 2004). In general, and for exercise of 2-3 h duration, a carbohydrate intake of 60 g/h is recommended with carbohydrate feedings starting well in advance of fatigue/depletion of body carbohydrate stores. Ingestion of more than 60 g/h will not have an additive effect and may even cause gastro-intestinal distress, while for exercise between 1-2 h a carbohydrate intake of 30 g/h may be more appropriate (Jeukendrup & McLaughlin 2011).

Several years ago it was discovered that exogenous carbohydrate oxidation is most likely limited by gastro-intestinal absorption (for review and detailed discussions see (Jeukendrup 2004)). Thus, strategies to improve absorption could provide more energy to the working muscle. It is likely feeding of a single carbohydrate source (for example glucose or maltodextrin) at high rates results in saturation of sodium dependent glucose transporters (SGLT1). Once these transporters are saturated feeding more of that carbohydrate will not result in greater absorption or increase oxidation rates. It was suggested by intestinal perfusion studies that the ingestion of carbohydrates that use different transporters might increase total carbohydrate absorption (Shi et al., 1995). In dual tracer studies, the oxidation of glucose and fructose mixtures during exercise were investigated and, therefore, the roles of SGLT1 and GLUT5 transporters. It was found that the ingestion of glucose at a rate of 1.2 g/min resulted in oxidation rates around 0.8 g/min. Ingesting more glucose (1.8 g/min) did not increase the oxidation. However, by ingesting glucose (1.2 g/min) and fructose (0.8 g/min) the total exogenous carbohydrate oxidation rate increased to 1.26 g/min, an increase in oxidation of 45% compared with a similar amount of glucose (Jeukendrup & Moseley 2008).

In the following years different combinations and amounts of carbohydrates were tested in an attempt to determine the maximal oxidation rate exogenous carbohydrate (Jentjens et al., 2003; Jentjens et al., 2004; Jentjens et al., 2004; Jentjens et al., 2004; Jentjens & Jeukendrup 2005; Jentjens et al., 2005; Jentjens et al., 2006; Jeukendrup et al., 2006; Wallis et al., 2007). It was observed that very high oxidation rates were reached with combinations of glucose+fructose, maltodextrin+fructose and glucose+sucrose+fructose. The highest rates were observed with a mixture of glucose and fructose ingested at a rate of 2.4 g/min. With this feeding regimen exogenous CHO oxidation peaked at 1.75 g/min. This is 75% higher





than what was previously thought to be the absolute maximum and is a result of the two types of carbohydrates having different intestinal transporters.

The increased oxidation of ingested multiple transportable carbohydrates on performance is still in its infancy and is currently limited to exercise of relatively long duration i.e. >2.5 h. From a laboratory study in which subjects cycled for 5 h with water, glucose or glucose+fructose there are some indications that drinks with multiple carbohydrates could improve performance (Jeukendrup et al., 2006). In this study carbohydrate was ingested at a rate of 90 g/h. The first indication of improved performance was that the ratings of perceived exertion (RPE) tended to be lower with glucose+fructose compared with glucose, which in turn was lower than water placebo.

Another important observation is that the oxidation efficiency of drinks containing multiple transportable carbohydrates is greater than drinks containing a single source of carbohydrate (Currell et al., 2008). The amount of carbohydrate remaining in the intestine is therefore smaller and osmotic shifts and malabsorption may be reduced. This probably means that drinks with multiple transportable carbohydrates are less likely to cause gastro-intestinal distress. Interestingly, this is a consistent finding in studies that have attempted to record gastro-intestinal discomfort during exercise (Jentjens et al., 2003; Jentjens et al., 2004; Jentjens et al., 2004; Jentjens et al., 2004; Jentjens & Jeukendrup 2005; Jentjens et al., 2005; Jentjens et al., 2006; Jeukendrup et al., 2006; Wallis et al., 2007). In general, subjects tended to feel less bloated when a glucose+fructose drink had been consumed versus a glucose only drink. A larger scale study into the effects of drinks with different types of carbohydrates on gastro-intestinal discomfort has not yet been conducted.

In the world of sport, athletes consume carbohydrate during exercise using a variety of foods and drinks, and a variety of feeding schedules. Carbohydrate is oxidised effectively, whether it is provided in solid, i.e., in bars, chews, semi-solid gels, or in a drink (Pfeiffer et al., 2010). Thus, strategies to provide approximately 30- 60- 90 g of carbohydrate / h (depending on the exercise duration) can be modified according to player preference and in the context of other nutritional requirements such as the fluid needs of the player. Sports drinks (commercial solutions providing 4-8% carbohydrate – 4-8 g carbohydrate /100 mL -, electrolytes and palatable flavours) are particularly valuable since these allow the athlete to replace their fluid





and carbohydrate needs simultaneously (Table 2). For sports of more than 60 min in duration, in which fatigue may occur, athletes are encouraged to develop a personalised exercise nutrition plan to combined their needs with their preferences (Burke et al., 2011). Each sport or exercise activity offers particular opportunities for fluid and carbohydrate to be consumed throughout the session - whether from aid stations, supplies carried by the athlete, or at formal stoppages in play such as time outs or half-time breaks. The athlete, coach and medical staff should be reminded to make use of these opportunities.

Post-exercise carbohydrate ingestion

Restoration of muscle glycogen concentrations is an important component of post-exercise recovery and is challenging for athletes who train or compete more than once each day or play multiple competitive matches in a week. The main dietary issue in glycogen synthesis is the amount of carbohydrate consumed, with an optimal intake for glycogen storage reported as 7-10 g/ kg BM /day (Jentjens & Jeukendrup 2003). There is some evidence that moderate and high GI carbohydrate-rich foods and drinks may be more favourable for glycogen storage than some low GI food choices (Jentjens & Jeukendrup 2003). Glycogen storage may occur at a slightly faster rate during the first couple of hours after exercise (Ivy 2001). However, the main reason for encouraging an athlete to consume carbohydrate-rich meals or snacks soon after exercise is that effective refuelling does not start until a substantial amount of carbohydrate (~ 1 g/kg BM) is consumed. When there is limited time between workouts or events (e.g. hours or less) it makes sense to turn every minute into effective recovery time by consuming carbohydrate as soon as possible after the first session. However, when recovery time is longer, immediate carbohydrate intake after exercise is unnecessary and the athlete can afford to follow their preferred and practical eating schedule as long as goals for total carbohydrate intake are met over the day. Under these circumstances muscle glycogen can be restored to resting levels, 24 h after exercise (Parkin et al., 1997).

Interestingly, recent studies have suggested that the rate of muscle glycogen re-synthesis may be slowed following competitive high intensity intermittent exercise i.e. football match.

Glycogen stores were reported to be lower than pre-match concentrations 48 h post match, despite the ingestion of a high carbohydrate diet (Bangsbo et al., 2006; Krstrup et al., 2011). Football-specific activities, such as frequent changes in direction and decelerations from sprints, have a high eccentric component. Eccentric contractions in combination with contact





between players results in muscle damage, which in turn may impair glycogen synthesis (Krustrup et al., 2011). This phenomenon is not alleviated by a diet high in carbohydrate and whey protein (Gunnarsson et al., 2013), despite certain amino acids having a potent effect on the secretion of insulin, which is a stimulator of glycogen re-synthesis (van Loon et al., 2000). For this reason the effects of adding amino acids and proteins to a carbohydrate solution have been investigated. One study compared glycogen re-synthesis rates after ingestion of carbohydrate, protein or carbohydrate plus protein (Zawadzki et al., 1992). As expected, very little glycogen was stored when protein alone was ingested, and glycogen storage was increased when carbohydrate was ingested. But most interestingly, glycogen storage was further increased when carbohydrate was ingested together with protein. However, other studies have shown that if the amount of ingested carbohydrate is sufficient, addition of protein or amino acids has no further effect (van Hall et al., 2000; van Loon et al., 2000; Jentjens et al., 2001). Nevertheless, recovery goals also include attention to the immune system, muscle building and injury repair. Therefore, it may be useful to eat nutrient-rich forms of carbohydrate foods and drinks during the recovery period to provide a range of valuable nutrients (Betts & Williams 2010).

In an attempt to speed player recovery and reduce muscle soreness, cryotherapy “ice baths” have become a common strategy adopted by many sports. Due to the vasoconstrictive consequence of cryotherapy, concerns have risen as to whether glycogen re-synthesis would be impaired due to reduced availability of substrate as a consequence of a reduction in blood flow to the muscle. However, one study investigated that effect of 10 min lower limb cold water immersion (8 °C), following exhaustive exercise, and found no impairment in glycogen restoration, in comparison to being seated at rest, when appropriate quantities of carbohydrate were ingested (Gregson et al., 2013).





Table 1. Daily carbohydrate needs for fuel and recovery

Daily guide for carbohydrate intake in relation to exercise requirements. Adapted from Burke et al. (2011). These recommendations should be refined with individual considerations of total energy needs, specific training needs and feedback from training / competition performance.

	Situation	Carbohydrate Targets g /kg BM/ day	Type and timing of Intake
Light	Low intensity, recovery or skill based activities	3- 5	Timing of intake should promote speedy replenishment of glycogen, or to provide carbohydrate intake around the training sessions of the day. As long as carbohydrate needs are provided, the pattern of intake can be governed by convenience and individual choice. Protein and other nutrient rich foods or meal combinations will allow the athlete to meet other acute or chronic sports nutrition goals.
Moderate	Moderate exercise program 1 h per day	5-7	
High	Endurance program or moderate to high intensity exercise 1-3 h per day	6-10	Athletes may choose compact carbohydrate-rich sources that are low in fibre/residue and easily consumed to ensure that fuel targets are met.
Very high	Extreme commitment to moderate to high intensity exercise >3 h per day	8-12	The athlete should practice to find a carbohydrate plan that suits their individual goals including hydration needs and gut comfort. Products providing multiple transportable carbohydrates (glucose:fructose mixtures) will achieve high rates of oxidation of carbohydrate consumed during exercise.



Table 2. Acute carbohydrate intake strategies

These guidelines are designed to promote high carbohydrate availability to promote optimal performance in key training sessions and competition (Burke et al. 2011).

	Situation	Carbohydrate Targets g / kg BM/ day	Type and timing of Intake
General preparation	Preparation for events up to 90 min in duration	7-12	Athletes may choose compact carbohydrate-rich sources that are low in fibre/residue and easily consumed to ensure that fuel targets are met, and to meet goals for gut comfort or lighter “racing weight” There may be benefits in consuming small regular snacks
Carbohydrate loading	Preparation for events greater than 90 min continuous/ intermittent exercise	36-48 h of 10-12	Can be achieved by increasing meal portion sizes of carbohydrate and snacking on carbohydrate rich foods between meals.
Pre-event meal	Before exercise >60 min	1-4 g 1-4 h before exercise	The timing, amount, and type of carbohydrate foods and drinks should be chosen to suit the practical needs of the event and individual preferences/ experiences. Choices high in fat/protein/fibre may need to be avoided to reduce risk of gastrointestinal issues during the event. Low GI choices may provide a more sustained source of fuel for situations where carbohydrate cannot be consumed during exercise
During short duration exercise	<45 min	None required mouth rinse	Athletes may benefit from rinsing their mouths with carbohydrate solution



During continuous high intensity exercise	45-60 min	Small quantity needed / mouth rinse	
During endurance exercise including “stop and start” sports	1.0-2.5 h	30-60 g / h	<p>Opportunities to consume foods and drinks vary according to the rules and nature of each sport.</p> <p>A range of drinks and sports products can provide easily consumed carbohydrate.</p> <p>A range of everyday dietary choices and specialised sports products ranging in form from liquid to solid may be useful.</p> <p>The athlete should practice carbohydrate intake during training to before adopting in competition. This will help determine their individual goals and preference considering hydration needs and gut comfort.</p>
During ultra-endurance exercise	>2.5-3 h	Up to 90 g / h	<p>Higher intakes of carbohydrate are associated with better performance.</p> <p>Products providing multiple transportable carbohydrates (glucose:fructose mixtures) will achieve high rates of oxidation of carbohydrate consumed during exercise.</p>
Quick refuelling	< 8 h recovery between two demanding sessions or playing two competitive games a week.	1.0-1.2 g in the 4 h post exercise, then resume daily carbohydrate needs	The athlete should practice to find a fuelling plan that suits their individual goals including hydration needs and gut comfort.



4. PROTEIN REQUIREMENTS FOR ATHLETES

In the human body all protein is functional. Proteins are synthesised from amino acids during the process of translation, where the unique chain of amino acids is determined by the nucleotide sequence of the specific gene encoding its formation. The diversity of protein functionality and specialization is vast. For example, there are thousands of proteins located amongst the body's cells and fluids involved in structural tissues, transport systems, hormones and metabolic processes, all of which are essential for life.

Protein is not stored in the human body; all protein is integral and functional. An average 70 kg male contains approximately 12 kg of protein and 220 g of free amino acids (Avril et al., 2003). Any unused protein is degraded and the liberated amino acids either oxidised or metabolised to fatty acids or glucose. Thus, the ingestion of an appropriate quantity and quality of protein from food is required to routinely deliver amino acids to the peripheral circulation. This pool of plasma amino acids then provide a metabolically available source of amino acids to be utilised and incorporated into proteins as required.

In muscle the majority of amino acids are incorporated into tissue proteins, with a small pool of free amino acids. This pool undergoes turnover receiving free amino acids from the breakdown of protein and contributing amino acids for protein synthesis. Protein breakdown in skeletal muscle serves two main purposes:

1. to provide essential amino acids when individual amino acids are converted to acetyl co A or TCA cycle intermediates.
2. to provide individual amino acids that can be used elsewhere in the body for the synthesis of neurotransmitters, hormones, glucose and proteins.

Clearly, if protein degradation rates are greater than the rates of synthesis, there will be a reduction of protein content; conversely, muscle protein content can only increase if the rate of synthesis exceeds that of degradation.

Increased protein requirements

There is still considerable debate about how much dietary protein is required for optimal athletic performance (Philips 2013). Muscle accounts for 30-50% of all protein turnover in the body. Both the structural proteins that make up the myofibrillar proteins and the proteins





that act as enzymes within a muscle cell change as an adaptation to exercise training. Indeed, muscle mass, muscle protein composition and muscle protein content will change in response to training. Exercise (especially endurance exercise) results in increased oxidation of the branched chain amino acids (BCAA), which are essential amino acids and cannot be synthesized within the body. Therefore, increased oxidation would imply that the dietary protein requirements are increased. Some studies, in which the nitrogen balance technique was used, showed that the dietary protein requirements for athletes involved in prolonged endurance training were higher than those for sedentary individuals. Whether requirements are really higher remains somewhat controversial though (for review see (Phillips 2006; Tipton & Witard 2007)).

It has been estimated that protein may contribute up to about 15% to energy expenditure in resting conditions. During exercise this relative contribution is likely to decrease because energy expenditure is increased and most of this energy is provided by carbohydrate and fat. During very prolonged exercise, when carbohydrate availability becomes limited, the contribution of protein to energy expenditure may amount up to about 10% of total energy expenditure. Thus, although protein oxidation is increased during endurance exercise, the relative contribution of protein to energy expenditure remains small. Protein requirements may be increased somewhat but this increased need may be met easily by a moderate protein intake. The research groups that advocate an increased protein intake for endurance athletes usually recommend a daily intake of 1.2-1.8 g/kg BM. This is about twice the level of protein intake that is recommended for sedentary populations (Moore et al., 2014) .

There are reports of increased protein breakdown after resistance exercise (Biolo et al., 1994). The suggested increased dietary protein requirements with resistance training are related to increased muscle bulk (hypertrophy) rather than increased oxidation of amino acids. Muscle protein breakdown is increased after resistance training, but to a smaller degree than muscle protein synthesis, while these elevations in protein degradation and synthesis are transient. Protein breakdown and synthesis after exercise are elevated at 3 and 24 h after exercise but return to baseline levels after 48 h. These results seem to apply both to resistance exercise and high intensity dynamic exercise (Areta et al., 2014).





There is controversy as to whether strength athletes need to eat larger amounts of protein. The nitrogen balance studies that have been conducted on such athletes have been criticized because they generally have been of short duration, where a steady state situation may not be established (Tipton & Witard 2007). The recommendation for protein intake for strength athletes is, therefore, generally 1.6-1.7 g /kg BM / day. Again, this seems to be met easily with a normal diet and no extra attention to protein intake is needed. Protein supplements are often used but are not necessary to meet the recommended protein intake. There is also no evidence that supplements would be more effective than normal foods.

Protein in recovery

Exercise will increase both muscle protein breakdown and muscle protein synthesis. However, in the absence of protein in the diet, net protein balance will remain negative. Therefore, protein is a key ingredient after matches and hard training sessions in order to achieve a positive net protein balance. In addition to general effects of exercise, most “stop and go” sports involve many decelerations (eccentric contractions) and contact between athletes, and as a consequence muscle damage may occur. Protein ingestion is, therefore, advised to aid with the repair of muscle tissue and other potential injuries (Res 2014).

After resistance exercise, muscle protein synthesis in response to a meal has been shown to be elevated for up to 24 h (Burd et al., 2011). Still, protein intake should best be commenced directly after exercise for optimal recovery, especially if limited time is available until the next match or important practice. Muscle protein synthesis decreases over time if blood amino acids are continuously high. Thus, for optimal recovery, meals containing protein should be eaten about every three hours, with a last snack containing protein just before bed (Res et al., 2012; Areta et al., 2013).

After exercise, the ingestion of different quantities of foods can deliver the similar quantity of total amino acids. However, the speed at which amino acids are delivered and the profile of BCAA and leucine in particular will differ (Burke et al., 2012). The optimal dose of protein to maximally stimulate muscle protein synthesis appears to be around 20-25 g, or about 0.3 g/kg BM (Moore et al., 2009; Witard et al., 2014). Animal protein contains more of the amino acid leucine, which is believed to be a main trigger for increases in muscle protein synthesis (van Loon 2012). Whey protein can be quickly digested and absorbed and contains



a high proportion of leucine. For example, approximately 2.5 g of leucine is contained in 20 g of whey. Whey has also been shown to elicit superior muscle protein synthesis compared to soy or casein, when taken in isocaloric amounts (Tang et al., 2009). Plant-based proteins contain less leucine compared to whey, therefore, comparatively more plant-based protein may need to be ingested to maximize muscle protein synthesis. Whey protein (or good sources of) is, therefore, considered the preferential protein to ingest directly after exercise.

After the initial protein intake directly post-exercise, players should continue maximizing their muscle protein synthesis. During the day, players should be encouraged to ingest protein from a variety of foods such as fish, meat, poultry and dairy, but also from vegetable sources like legumes, nuts, rice, corn or wheat. Casein has been shown to be beneficial for a pre-bedtime snack, as it is a slowly digesting protein that will be available during a longer portion of the night (Res et al., 2012).

Daily protein intake for athletes has been advised to be in the range of 1.3-1.8 g/kg BM (Phillips & Van Loon 2011). However, these recommendations are based on nitrogen balance studies and thus protein synthesis needs. There may be other potential benefits of protein ingestion involving different mechanisms, which are largely unknown. For example, in extreme situations, daily protein intake far above general recommendations has proven to be beneficial beyond enhancing muscle protein synthesis. In an offshore race, fatigue and memory loss were attenuated after increased protein intake (Portier et al., 2008). Furthermore, when protein intake was elevated from 1.5 g/kg BM to 3 g/kg BM, tolerance to intensified training was increased (Witard et al., 2011) and immune function was better maintained, resulting in less upper respiratory tract infections (Witard et al., 2013). In addition, elevated daily protein intake in the range of 2.3 g/kg BM has been shown to better maintain muscle mass in the face of an energy deficit (Mettler et al., 2010). Despite these studies advocating the benefits of very high protein intakes, current guidelines for daily protein intake for a 70 kg player are to consume about 120 grams of protein divided over 6 meals, interspersed by about 3 h, with each meal containing approximately 20 g of protein (Phillips and Van Loon 2011). However, it is important to note that in cases of extreme physical demands or energy deficit, protein needs might be even higher.





Amino acid versus protein intake

In the past the amino acid needs of the body were primarily met by ingestion of whole proteins in the diet. However, over the last few years supplementation of individual amino acids has become increasingly popular. This is the result of technological advances that have made it possible to manufacture food-grade ultrapure amino acids, but also reflects the general interest in the pharmacological and metabolic interactions of free-form amino acids in various areas of clinical nutrition. Here, individual amino acids are used to reduce nitrogen losses and improve organ functions in traumatized and critically-ill patients (Roth 1985). The results of these studies have been applied to populations of athletes and healthy individuals, where intake of separate amino acids is claimed to improve exercise performance, stimulate hormone release and improve immune function amongst a variety of other positive effects. In pursuit of increasing the protein intake of the diet the ingestion of whey protein has become prevalent in sport. It is also important to note that the athlete should be reminded at all times to check the quality and source of the whey protein supplements, as these too have been reported to be a potential source for inadvertent doping (Maughan 2013).

However, amino acid metabolism is very complex. One amino acid can be converted into another and amino acids may influence nerve impulse transmission as well as hormone secretion. Composition of specific amino acid mixtures or even high protein diets may lead to nutritional imbalances because overload with one amino acid may reduce the absorption of other amino acids. The addition of the BCAA leucine has been well documented to improve muscle protein synthesis in *in vitro* studies and in animal (primarily rodent) models (Mitchell et al., 2010; Tanaka et al., 2011; Barclay et al., 2012; Maughan et al., 2012; Maughan & Shirreffs 2012). To date, the efficacy of leucine consumption on human skeletal muscle synthesis *in vivo* is still to be determined. Nevertheless, the potential use of leucine as a stimulator of muscle synthesis in populations with greater muscle degradation such as athletes engaging in regular resistance and/or endurance exercise or populations susceptible to muscle loss i.e. the elderly or injured athletes warrants further investigation.





Table 3. Protein guidelines

Protein recommendations should be refined with individual considerations of total energy needs, specific training needs and feedback from training / competition performance.

Occasion	Protein Targets	Principle
Daily intake	1.5-2.0 g / kg BM/ day	Meals containing 20-25 g protein ingested routinely (3h intervals) during the day
Exercise	20-25 g high quality protein containing 2-3g of leucine	Ingest immediately post exercise to support adaptation
Sleep	30 g casein	Ingest prior to sleep



5. FLUID REQUIREMENTS FOR ATHLETES

Water has many important functions in the human body. The total water content of the human body is between 30-50 litres, which is approximately 55-60% of BM. Every day, water is excreted in the form of sweat, urine and evaporative losses, while water intake usually matches this. Water turnover can be very high in some conditions but the total body water content is remarkably constant and rarely exceeds variations of 1 litre. In order to maintain fluid balance, water intake may vary from 1 litre to up to about 12 litres per day. However, during exercise and especially during exercise in hot conditions, sweat rates (and thus water losses) may increase dramatically and dehydration may occur (i.e. the body is in negative fluid balance). Dehydration can have a substantial impact on physical and mental function and increase the risk of heat illness. Even mild dehydration can result in reduced exercise capacity (Cheuvront et al., 2010).

Exercise (muscle contraction) causes an increase in heat production in the body, with muscle contraction during most activities only about 15-20% efficient. Therefore, intense exercise is associated with a high level of metabolic heat production, as ~75-80% of energy is converted to heat in the working muscles (Shirreffs et al., 2005). For every litre of oxygen consumed during exercise approximately 16 kJ (4 kcal) of heat is produced and only 4 kJ (1 kcal) is actually used to perform mechanical work. If this heat is not dissipated athletes would soon overheat. Furthermore, when well-trained individuals are exercising at 80-90% VO_2 max, the body's heat production may be more than 1000 W (i.e. 3.6 MJ or 900 kcal per hour). This could potentially cause body core temperature to increase by 1 °C every 5-8 min if no heat could be dissipated. As a result, body core temperature could approach dangerous levels in less than 20 min.

There are several mechanisms to dissipate this heat and to maintain body core temperature in a relatively narrow range: 36-38 °C in resting conditions and 38-40 °C during exercise and hot conditions. The most important cooling mechanism of the body is sweating, although radiation and convection can also contribute. Sweat must evaporate from the body surface in order to exert a cooling effect and evaporation of one litre of water from the skin will remove 2.4 MJ of heat from the body. Although sweating is a very effective way to dissipate heat, it may cause dehydration if sweat losses are not replenished (Cheuvront et al., 2013; Cheuvront





& Kenefick 2014). This may cause further problems for the athlete: progressive dehydration impairs the ability to sweat and therefore, to regulate body temperature. Body temperature rises faster in the dehydrated state and this is commonly accompanied by a higher heart rate during exercise (Coyle & Gonzalez-Alonso 2001). The most dramatic consequence of dehydration-induced hyperthermia during exercise is a 25-30% reduction in stroke volume that is not generally met with a proportional increase in heart rate; this results in a decline in cardiac output and in arterial blood pressure (Gonzalez-Alonso et al., 1995).

Fluid losses are mainly dependent on five factors:

- 1) the ambient environmental conditions (temperature, humidity)
- 2) the exercise intensity
- 3) the duration of exercise and the duration of the heat exposure
- 4) the clothing and protective equipment worn by the athlete
- 5) the individual sweat rate of the athlete.

The environmental heat stress is determined by the ambient temperature, relative humidity, wind velocity and solar radiation. The relative humidity is the most important of these factors, since a high humidity will severely compromise the evaporative loss of sweat. Often sweat will drip off the skin in such conditions, rather than evaporate. This means that heat loss via this route will be less effective. Furthermore, it is important to note that problems of hyperthermia and heat injury are not restricted to prolonged exercise in a hot environment: heat production is directly proportional to exercise intensity, so that very strenuous exercise - even in a cool environment - can cause a substantial rise in body temperature.

To maintain water balance, fluid intake must compensate for the fluid loss that occurs during exercise. Fluid intake is usually dependent on thirst feelings but thirst (or the lack of thirst) can also be overridden by conscious control (Sawka & Greenleaf 1992). It is important to note, however, that thirst is a poor indicator of fluid requirements or the degree of dehydration. In general, the sensation of feeling thirsty is not perceived until a person has lost at least 2% of BM (McKenna & Thompson 1998). As already mentioned, even this mild degree of dehydration is sufficient to impair exercise performance (Cheuvront et al., 2003). It has also been shown that athletes tend to drink too little even when sufficient fluid is





available. Runners for instance will seldom drink more than 0.5 L/h whilst sweat rates can be much higher than this.

The effects of dehydration on exercise performance

As the body becomes progressively dehydrated, a reduction in skin blood flow and sweat rate may occur. A high humidity may limit evaporative sweat loss which will lead to further rises in core temperature, resulting in fatigue and possible heat injury to body tissues. The latter is potentially fatal (Byard & Riches 2005).

Several studies have shown that mild dehydration, equivalent to the loss of only 2% BM, is sufficient to significantly impair exercise performance (Cheuvront et al., 2003; American College of Sports et al., 2007). In addition, it is often reported that greater losses in BM will result in greater reductions in performance (Baker et al., 2007). Even very low intensity exercise (i.e. walking) is affected by dehydration. The capacity to perform high intensity exercise which results in exhaustion within only a few minutes has been shown to be reduced by as much as 45% by prior dehydration (2.5% of BM) (Walsh et al., 1994). Although there is little opportunity for sweat loss during such short-duration, high-intensity events, athletes who travel to compete in hot climates are likely to experience acute dehydration, which can persist for several days and can be of sufficient magnitude to have a detrimental effect on performance in competition. Although dehydration has detrimental effects, especially on performance in hot conditions, such effects can also be observed in cool conditions. Both decreases in maximal aerobic power ($VO_2\text{max}$) and decreases in endurance capacity have been reported with dehydration in temperate conditions (McConnell et al., 1997), although not all studies found such an effect (Maughan et al., 1989; Robinson et al., 1995).

Although some individuals may be more or less sensitive to dehydration, the level needed to induce performance degradations approximates $>2\%$ decrease in BM (Sawka & Noakes 2007). Muscle strength and anaerobic performance are less likely to be affected by dehydration (Ali & Williams 2013; Cheuvront & Kenefick 2014). Some authors argue that reductions in BM (i.e., dehydration) during a weight-bearing activity, such as football, might be advantageous to force production and vertical jump height (Viitasalo et al., 1987). However, there is no evidence to support this notion. For example, in one study, a diuretic-induced reduction in BM by 2.5% had no effect on sprint and power performance (Watson et





al., 2005). Likewise, there was no correlation between the reduction in BM and vertical jump height (Watson et al., 2005), suggesting that dehydration provides no advantage for weight-bearing activities like football.

McGregor and colleagues (1999) were the first to test the effects of dehydration on football-specific performance. In this study, RPE were higher toward the end of the 90-min Loughborough Shuttle Running Test (LIST) (13-20 °C, 57% relative humidity) when no fluid was given to the players (resulting in 2.5% dehydration) compared to when fluid was provided (resulting in 1.4% dehydration). Likewise, 2.5% dehydration slowed sprint time at the end of LIST in comparison to 1.4% dehydration. This study also showed that football specific skill performance (i.e. dribbling skill) decreased by 5% from pre- to post-LIST with 2.5% dehydration, but was maintained with 1.4% dehydration. Altogether, the results of this experiment suggested that dehydration of 2.5% BM deficit increases the perception of effort and impairs sprinting and football dribbling skills toward the end of 90-min intermittent high-intensity exercise. However, 2.5% dehydration had no impact on football players' mental concentration test scores at the end of LIST (McGregor et al., 1999).

There are several reasons why dehydration results in decreased exercise performance. First of all, a fall in plasma volume, a decreased blood volume, and increased blood viscosity and a lower central venous pressure can result in a reduced stroke volume and maximal cardiac output. In addition, during exercise in the heat, the dilation of the skin blood vessels reduces the proportion of the cardiac output that is devoted to perfusion of the working muscles. Dehydration also impairs the ability of the body to lose heat. Both sweat rate and skin blood flow are lower at the same core temperature for the dehydrated compared with the euhydrated state. This means that body temperature rises faster during exercise when the body is dehydrated (Montain & Coyle 1992). Finally, the larger rise in core temperature during exercise in the dehydrated state is associated with an increased rate of muscle glycogen breakdown (Hargreaves et al., 1996). Depletion of these stores could also result in premature fatigue in prolonged exercise. In addition to the effects of dehydration on endurance there are also reported negative effects on co-ordination and cognitive functioning (Baker et al., 2007). This is likely to impact on all sports where skill and decision making is involved.





Heat illness

Dehydration also poses a serious health risk in that it increases the risk of cramps, heat exhaustion and life-threatening heat stroke. Early symptoms of heat injury are excessive sweating, headache, nausea, dizziness, a reduced consciousness and mental function. When the core temperature rises to over 40 °C, heat stroke may develop, characterized by hot dry skin, confusion and loss of consciousness (Seraj et al., 1991) . There are several anecdotal reports of athletes and army recruits who died because of heat stroke. Most of these deaths have been explained by exercise in hot conditions, often with insufficient fluid intake. These problems do not only affect highly trained athletes, but also less well trained people participating in sport. Although well-trained individuals will generally exercise at higher intensities and therefore produce more heat, less well trained individuals have less effective thermoregulation during exercise, and work less economically. Furthermore, overweight, unacclimated and ill individuals are at higher risk of developing heat stroke (Aarseth et al., 1986).

Hyponatremia

Sweat loss results not only in the loss of water but also in a loss of electrolytes, with sodium the most important ion lost in sweat. An electrolyte imbalance commonly referred to as “water intoxication” that results from *hyponatremia* (low plasma sodium) due to excessive water consumption has occasionally been reported in endurance athletes (Speedy et al., 2000). Hyponatremia is the dilution of serum sodium from normal levels of 135 to 145 mEq/L to levels below 130 mEq/L. This may result in intracellular swelling, which in turn can alter central nervous system function. During prolonged exercise, serum sodium can be diluted by either excessive fluid intake or excessive sodium losses in sweat or both. The symptoms of hyponatremia are almost identical to those of dehydration, exertional heat exhaustion, and exertional heat stroke, and may include nausea, confusion, disorientation, headache, vomiting, aphasia, impaired coordination, muscle cramps, and muscle weakness. Severe hyponatremia can result in complications such as cerebral and pulmonary edema, which in turn can cause seizure, coma, and cardiorespiratory arrest.

Hyponatremia appears to be most common among slow runners in marathon and ultramarathon races and probably arises due to a loss of sodium in sweat coupled with very high and unnecessary intakes of water (Speedy et al., 2000; Speedy et al., 2000). This means





that there can be a danger of misdiagnosis of this condition when it occurs in individuals participating in endurance races. The usual treatment for dehydration is administration of fluid intravenously and orally. If this treatment were to be given to a hyponatremic individual, the consequences could be fatal (Noakes 2000).

Fluid intake strategies

Fluid intake during exercise can help maintain plasma volume and prevent the adverse effects of dehydration, endurance performance and co-ordination. Elevating blood volume just prior to exercise by various hyperhydration (greater than normal body water content) strategies has also been suggested to be effective in enhancing exercise performance (Goulet et al., 2008). When there is only little time in between two exercise bouts, rapid rehydration is crucial and drinking regimens need to be employed to optimize fluid delivery. As such, strategies for fluid replacement before, during and after exercise will be discussed in the following sections. It is important to note that body water gain occurs through metabolic water production and dissociation of water from glycogen. While the relatively small BM changes due to respiration and metabolism can be estimated, for practical purposes, in most studies it is typically assumed that 1 kg of BM loss represents ~1 L of water loss (Maughan et al., 2007).

Pre-exercise hyperhydration

Since even mild dehydration has been shown to result in reduced exercise capacity, it has been hypothesized that hyperhydration could improve heat dissipation and exercise performance in the heat by expanding blood volume and reducing plasma osmolarity (Montain & Coyle 1992). Studies have reported higher sweating rates, lower core temperatures and lower heart rates during exercise after hyperhydration (Latzka et al., 1998), while others have reported improvements in exercise performance (Goulet et al., 2008). However, these results must be interpreted with caution. Some of these studies used a dehydrated state as a control condition and, therefore, it is impossible to conclude from these studies that hyperhydration improves thermoregulation and performance compared to the euhydrated state (Sawka et al., 2001). This issue was addressed in a study carried out by Latzka and colleagues (1997), in which thermoregulatory responses to exercise in the heat were compared in a series of euhydrated and hyperhydrated trials. No differences between hyperhydration and euhydration exercise trials were reported for core and skin temperature





response, heart rate response or sweat response. It was concluded that hyperhydration achieved by increased pre-exercise fluid ingestion (~1.8 litres before and ~2.2 litres during exercise) offered no extra thermoregulatory advantage to exercise in a hot environment compared to euhydration (~2.2 litres during exercise) (Latzka et al., 1997).

It is also worth mentioning that evidence suggests that ingestion of larger than normal volumes of fluid prior to exercise can have negative effects. Labourers working under conditions of heat stress may experience an increased gastric discomfort associated with consuming large volumes of water just prior to such work (Wagner et al., 1972).

Furthermore, extra water obtained through traditional methods of simply ingesting more water is transitory as the kidneys quickly remove any excess body water resulting in an increased urinary production (Maughan 1997; Maughan et al., 1997).

As a consequence, glycerol supplementation in conjunction with hyperhydration may be more appropriate as it results in increased water retention and a reduction in urinary output. However, its use in sport is prohibited and, as such, its use is not recommended for athletes. In addition, and as with hyperhydrating with water alone, there exists significant evidence that hyperhydrating with glycerol provides no additional benefit to exercise in the heat compared to euhydration (Cheung et al., 2000; Sawka et al., 2001). While the large volume of accompanying water required, subsequent increased discomfort and the reported side effects (nausea, light headedness and gastric complaints), make it inappropriate for athletic use (Latzka et al., 1997).

Fluid intake during exercise

In order to avoid dehydration during prolonged exercise fluids must be consumed to match the sweat losses. By regularly measuring BM before and after a training session it is possible to get a good indication of fluid loss. Ideally, the weight loss would be compensated by an equal amount of fluid intake, although it may not always be possible to prevent dehydration completely. Sweat rates during strenuous exercise in the heat can amount up to two-to-three litres per hour. Such large volumes of fluid are difficult, if not impossible to ingest and even one litre may feel quite uncomfortable in the stomach. Therefore, it is often not practically possible to achieve fluid intakes that match sweat losses during exercise.





Another factor that can make the ingestion of large amounts of fluid difficult is the fact that in some sports or disciplines the rules or practicalities of the specific sport may limit the opportunities for drinking during exercise. Fluid intake may be useful during exercise longer than 30-60 min but there appears little advantage during strenuous exercise of less than 30 min duration. During such high intensity exercise gastric emptying is inhibited and the drink may cause gastro-intestinal distress with no performance benefit. Therefore, the athlete's goal should be to minimise fluid losses during exercise of an hour or more to $\leq 2\%$ starting BM (assuming the athlete started in a euhydrated state). This goal becomes even more important with exercise in the heat (Shirreffs & Sawka 2011).

Practice drinking during training

Although it is often difficult to tolerate the volumes of fluid needed to prevent dehydration, the volume of fluid that is tolerable is trainable and can be increased with frequent drinking in training. Training to drink will accustom athletes to the feeling of exercising with fluid in the stomach, whilst helping to optimise the delivery of carbohydrate if sports drinks are chosen (Jeukendrup 2011). It also gives the opportunity to experiment with different volumes and flavourings to determine how much fluid intake they can tolerate and which formulations suit them best.

Composition of sports drinks

Numerous studies have shown that regular water intake during prolonged exercise is effective in improving performance (for review see (Shirreffs & Sawka 2011)). Fluid intake during prolonged exercise, of course, offers the opportunity to provide some fuel (carbohydrate) as well. The addition of some carbohydrate to drinks consumed during exercise has been shown to have an additive and independent effect in improving exercise performance (Below et al., 1995). The ideal drink for fluid and energy replacement during exercise is one that tastes good to the athlete, does not cause gastrointestinal discomfort when consumed in large volumes, is rapidly emptied from the stomach and absorbed in the intestine, provides energy in the form of carbohydrate and a source of electrolytes. Therefore, sports drinks typically have three main ingredients: water, carbohydrate and sodium. The water and carbohydrate provide fluid and energy respectively, while sodium is included to aid water absorption and retention.





Although carbohydrate is important, a too concentrated carbohydrate solution designed to provide more fuel for the working muscles may decrease the amount of water that can be absorbed due to a slowing of gastric emptying. Water is absorbed into the body primarily through the small intestine, but the absorption of water is decreased if the concentration of dissolved carbohydrate (or other substances) in the drink is too high. In this situation, water will actually be drawn out of the interstitial fluid and plasma and into the lumen of the small intestine by osmosis (Gisolfi et al., 1992). So long as the fluid remains hypotonic with respect to plasma, the uptake of water from the small intestine is not adversely affected. In fact, the presence of small amounts of glucose and sodium tend to slightly increase the rate of water absorption compared with pure water (Gisolfi & Duchman 1992; Shi & Passe 2010). It must be emphasized here that the addition of sodium and other electrolytes to sports drinks is to increase palatability, maintain thirst (and therefore promote drinking), prevent hyponatremia and increase the rate of water uptake, rather than to replace the electrolyte losses through sweating. Replacement of the electrolytes lost in sweat can normally wait until the post-exercise recovery period.

Rehydration after exercise

When there is little time for recovery in between exercise bouts, the replacement of fluid and electrolytes in the post-exercise recovery period is of crucial importance. In the limited time available the athlete should strive to maximize rehydration. The main factors influencing the effectiveness of post-exercise rehydration are the volume and composition of the fluid consumed.

Rehydration is an important part of the post-exercise recovery process. If players have accrued a body mass deficit, they should aim to completely replace fluid and electrolyte losses prior to the start of the next training session or match. If dehydration is severe (>5% of BM) or rapid rehydration is needed (e.g. < 24 h before next practice or match) the recommendation is to drink ~1.5 L of fluid for each 1 kg of BM deficit (Shirreffs & Sawka 2011). This is because some of the ingested fluid will be excreted in urine and studies indicate that ingestion of 150% or more of weight loss is required to achieve normal hydration within 6 hours following exercise (Shirreffs et al., 1996). In most other situations, water and sodium can be consumed with normal eating and drinking practices with no





urgency. Drinking a beverage with sodium or eating sodium-containing snacks/foods helps replace sweat sodium losses, stimulate thirst and retain the ingested fluids (Shirreffs & Sawka, 2011).

Ingesting a beverage containing sodium not only promotes rapid fluid absorption in the small intestine, but also allows the plasma sodium concentration to remain elevated during the rehydration period and helps to maintain thirst while delaying stimulation of urine production (Maughan et al., 1996). The inclusion of potassium in the beverage consumed after exercise would be expected to enhance the replacement of intracellular water and thus promote rehydration, but currently there is little experimental evidence to support this. The rehydration drink should also contain carbohydrate (glucose or glucose polymers) because the presence of glucose will also stimulate fluid absorption in the gut and improve beverage taste. Following exercise, the uptake of glucose into the muscle for glycogen re-synthesis should also promote intracellular rehydration (Gisolfi et al., 1992; Gisolfi et al., 2001).

Thus, plain water is not the ideal post-exercise rehydration beverage when rapid and complete restoration of body fluid balance is necessary and where all intake is in liquid form. Ingestion of water alone in the post-exercise period results in a rapid fall in the plasma sodium concentration and the plasma osmolarity. These changes have the effect of reducing the stimulation to drink (thirst) and increasing the urine output, both of which will delay the rehydration process. Plasma volume is more rapidly and completely restored in the post-exercise period if sodium chloride (77 mmol/L or 450 mg/L) is added to the water consumed (Maughan & Leiper 1995). This sodium concentration is similar to the upper limit of the sodium concentration found in sweat, but is considerably higher than the sodium concentration of many commercially available sports drinks, which usually contain 10-25 mmol/L (60-150 mg/L).





Table 4. Fluid guidelines

Fluid recommendations should be refined with individual sweat rates and specific training intensities. Feedback from training / competition performance is required. Fluid needs will increase during exercise in warmer environments.

Occasion	Targets	Principle
Daily intake	Adjust to daily levels physical activity	Fluid can be ingested via beverages, fruits and vegetables.
Prior to Exercise	5-7 ml /kg BM	Drink 3-4 h before exercise
During exercise	Routine ingestion of fluid	Base intake on individual fluid losses and adjust for intensity and environment of exercise
Post Exercise	Drink 150% of body mass losses Avoid alcohol	Replace fluid losses as a consequence of sweating during exercise. The addition of sodium to the recovery beverage will help with fluid retention and maintain the drive to drink.



6. MICRONUTRIENT REQUIREMENTS FOR ATHLETES

The daily requirement for some vitamins and minerals is increased beyond population levels in people undertaking a strenuous exercise program (Gleeson 2013). The potential reasons for this increased requirement are increased losses through sweat, urine and perhaps faeces, and through increased production of free radicals. Unfortunately, at present we are unable to quantify the additional micronutrient requirements of athletes. The key factors ensuring an adequate intake of vitamins and minerals are a moderate to high energy intake and a varied diet based on nutritious foods (Williams 2005). Dietary surveys show that most athletes report dietary practices that easily supply vitamin and minerals in excess of Recommended Daily Allowances (RDAs) and are likely to meet any increases in micronutrient demand caused by training (van der Beek 1991). However, not all athletes eat varied diets of adequate energy intake and some may need help to improve both the quality and quantity of their food selections.

Studies of the micronutrient status of athletes have not revealed any significant differences between indices in athletes and sedentary controls (Volpe 2007). The results suggest that athletic training, *per se*, does not lead to micronutrient deficiency. These data should, however, be interpreted very carefully since most indices are not sensitive enough to detect marginal deficiencies. Overall, generalized vitamin and mineral supplementation for all athletes is not justified. Furthermore, studies do not support an increase in performance with such supplementation except in the case where a pre-existing deficiency was corrected (Clarkson 1991). The best management for the athlete with a high risk of suboptimal intake of micronutrients is to provide nutrition education to improve the variety and quantity of food intake. However, a low dose, broad range multivitamin/mineral supplement may be useful where the athlete is unwilling or unable to make dietary changes, or when the athlete is travelling to places with an uncertain food supply and eating schedule.

Anti-oxidant vitamins

Exercise has been linked with an increased production of free oxygen radical species capable of causing cellular damage (Powers et al., 2010). A sudden increase in training stress (such as an increase in volume or intensity) or a stressful environment (training in hot conditions or at





altitude) is believed to increase the production of these reactive oxygen species leading to an increase in markers of cellular damage. Supplementation with anti-oxidant vitamins such as vitamin C or vitamin E is often suggested to increase anti-oxidant status and provide protection against this damage (Taghiyar et al., 2013).

However, the literature on the effects of antioxidant supplementation on anti-oxidant status, cellular damage and performance is complex and confusing. Some, but not all, studies show that acute supplementation during periods of increased stress may provide bridging protection until the athlete is able to adapt his or her own anti-oxidant status to meet this stress. It is possible that subtle benefits occur at a cellular level, which are too small to translate into detectable performance benefits. Whether ongoing supplementation is necessary or even desirable for optimal training adaptations and competition performance of athletes is also unknown. Recently it has been suggested that the increase in free radical production during a period of non-damaging exercise may act as signal for adaptation and therefore ingesting anti-oxidant vitamins may actually interfere with the desired adaptation (McArdle & Jackson 2000).

Interesting data is emerging regarding “anti oxidants” and other food components that may indirectly influence the recovery process. Vigorous (eccentric) exercise has been shown to increase muscle damage, inflammation, delayed onset muscle soreness and reduced muscle function (Bowtell et al., 2011). This response is potentially triggered by inflammatory cytokines (Davis et al., 2007). This is a healthy process to some extent, but it might overshoot and limit recovery. In that case, food components that modulate the inflammatory process might be helpful in the acute recovery process (Nedelec et al., 2013). Studies have shown some beneficial effects of omega-3 fatty acids (Tartibian et al., 2009), curcumin (Davis et al., 2007), tart cherry juice (Connolly et al., 2006; Howatson et al., 2010) and N-acetyl cysteine (Michailidis et al., 2013) in the recovery process due to their anti-inflammatory and/or anti-oxidant effects. Although these data show promising results, it should be noted that not all results were obtained from human experiments, effects on functional outcomes are not always clear and long-term effects have not been evaluated. In any case, anti-inflammatory and anti-oxidant supplementation should be carefully dosed, as the inflammatory process and redox reactions trigger exercise adaptations. Thus, chronic high or mis-timed dosages of anti-oxidant supplementation may be detrimental to long-term



training (Baar 2014). Furthermore, it is important to note that training up-regulates anti-oxidant and anti-inflammatory defences (Gomez-Cabrera et al., 2008). Thus, the anti-inflammatory effects of food and supplementation are likely to be less in well-trained athletes. The use of functional foods or food ingredients to enhance recovery is an exciting new area of research, but clearly, more research should be done to be able to determine optimal timing, ingredients, dose and judging long-term effects.

Iron

Inadequate iron status can reduce exercise performance via sub-optimal levels of hemoglobin, and perhaps iron-related muscle enzymes. Reductions in the hemoglobin levels of distance runners first alerted sports scientists to the issue of the iron status of athletes (Ricci et al., 1988). However, more recent research has raised the problem of distinguishing true iron deficiency from alterations in iron status measures that are caused by exercise itself. Low iron status in athletes is over-diagnosed from single measures of low hemoglobin and ferritin levels (Taniguchi et al., 1991). A major problem is the failure to recognize that the increase in blood volume that accompanies training will cause a dilution of all the blood contents. This hemodilution, often termed “sports anemia”, does not impair exercise performance (Balaban 1992; Sacirovic et al., 2013).

Nevertheless, some athletes are at true risk of becoming iron deficient. The causes are essentially the same as for members of the general community: a lower than desirable intake of bioavailable iron and/or increased iron requirements or losses. Iron requirements may be increased in some athletes due to growth needs, or to increased losses of blood and red blood cell destruction. However, the most common risk factor among athletes is a low energy and/or low iron diet, with females, “restricted” eaters, vegetarians and athletes eating high carbohydrate/ low meat diets being likely targets (Mann et al., 2002).

Iron is found in a range of plant and animal food sources in two forms: heme iron is found only in flesh or blood containing animal foods; whereas organic iron is found both in animal foods and plant foods. Whilst heme iron is relatively well absorbed from single foods and mixed meals (15-35% bioavailability), the absorption of non-heme iron from single plant sources is low and variable (2-8%). The bioavailability of non-heme iron, and to a lesser extent, heme iron is affected by other foods consumed in the same meal. Factors that enhance





iron absorption include Vitamin C, peptides from fish and meat, alcohol and food acids, while factors that inhibit absorption include phytates, polyphenols, calcium and peptides from plant sources such as soy protein (Lane & Richardson 2014). The absorption of both heme and non-heme iron is increased as an adaptive response in people who are iron-deficient or have increased iron requirements. While the iron bioavailability studies from which these observations have been made have not been undertaken on special groups such as athletes, it is generally assumed that the results can be applied across populations of healthy people.

The assessment of total dietary iron intake of athletes is not necessarily a good predictor of their iron status; the mixing and matching of foods at meals plays an important role by determining the bioavailability of dietary iron intake. Low iron status, indicated by serum ferritin levels lower than 20 ng/mL, should be considered for further assessment and treatment. Present evidence does not support that low iron status without anemia reduces exercise performance. However many athletes with such low iron stores, or a sudden drop in iron status, frequently complain of fatigue and inability to recover after heavy training. Many of these respond to strategies that improve iron status or prevent a further decrease in iron stores (DellaValle & Haas 2014).

Evaluation and management of iron status should be undertaken on an individual basis by a sports medicine expert. Prevention and treatment of iron deficiency may include iron supplementation, with a recommended therapeutic dose of 100 mg/day of elemental iron for 2-3 months. However the management plan should include dietary counselling to increase the intake of bioavailable iron, and appropriate strategies to reduce any unwarranted iron loss. Many athletes self-prescribe iron supplements. However, this practice does not provide the athlete with the opportunity for adequate assessment of iron losses and expert dietary counselling from a sports dietician. Dietary guidelines for increasing iron intake should be integrated with the athlete's other nutritional goals such as a need for high carbohydrate intake or reduced energy intake.

Calcium

Weight-bearing exercise is considered to be one of the best protectors of bone health. Therefore, it is puzzling to find reports of low bone density in some female athletes, notably





distance runners. However, a serious outcome of menstrual disturbances in female athletes is the high risk of either direct loss of bone density, or failure to optimize the gaining of peak bone mass during early adulthood. Individually, or in combination, the problems involved in the female athlete triad (disordered eating, menstrual dysfunction and reduced bone status) can directly impair athletic performance. Significantly, they will reduce the athlete's career span by increasing their risk of illness and injury, including stress fractures (Okamoto et al., 2010). Long-term problems may include an increased risk of osteoporosis in later life.

Optimal nutrition is important to correct factors that underpin the menstrual dysfunction, as well as those that contribute to sub-optimal bone density. Adequate energy intake and the reversal of disordered eating or inadequate nutrient intake are important. A team approach involving sports physician, sports dietician, psychologist and/or psychiatrist, coach and family may be needed to treat the athlete with disordered eating or eating disorders. Adequate calcium intake (~1000 mg / d) is important for bone health, and requirements may be increased to 1200-1500 mg/day in athletes with impaired menstrual function. Again, strategies to meet calcium needs must be integrated into the total nutrition goals of the athlete. Where adequate calcium intake cannot be met through dietary means, usually through the use of low-fat dairy foods or calcium-enriched soy alternatives, a calcium supplement may be considered.

Vitamin D

Vitamin D is a hormonal precursor that plays a well-documented role in supporting bone health and immune function. However, the discovery of the vitamin D receptor in human skeletal muscle has led to increased research on the potential role of vitamin D in regulating muscle protein synthesis (MPS) and muscle function, thus having obvious implications for training adaptations (Morton 2014). The study of vitamin D is particularly relevant given that many athletes, including professional football players, exhibit vitamin D deficiency in the winter months (Morton et al., 2012; Close et al., 2013). At this time, there is no UV radiation of appropriate wavelength for cutaneous production of pre-vitamin D₃ to occur (Webb & Holick 1988; Webb et al., 1988).

To account for the seasonal variation in ultraviolet-B radiation during winter, it has therefore become common practice to supplement with vitamin D₃ (cholecalciferol) in order to





promote vitamin D synthesis. To this end, daily supplementation with 5,000 IU appears a safe and tolerable dose to restore circulating 25[OH]D concentrations to sufficient levels within six weeks i.e. approximately 100 nmol/L (Close et al., 2013). Although not conclusive, preliminary evidence also suggests that vitamin D supplementation in those athletes who exhibit severe deficiency (i.e. 25[OH]D < 12.5 nmol/L) can improve sprint and jump performance in a cohort of youth professional football players (Close et al., 2013).

In an attempt to promote training adaptations as well as maintain both bone and immune health, it is therefore recommended that football players correct any deficiency with appropriate supplementation strategies during the winter period when exposure to natural sunlight is likely negated. Furthermore, because of the clinical implications of both vitamin D deficiency but also toxicity, it is highly advised that individuals' baseline serum 25 (OH)D levels are suitably assessed using reliable and valid techniques (such as tandem mass spectrometry) prior to intervening with any supplement strategy. The latter point is particularly pertinent given that the magnitude of increases in serum 25 (OH)D is inversely proportional to basal levels (Close et al., 2013) and hence, a one-size-fits-all approach to supplementation is not appropriate. Indeed, high dose supplementation in individuals exhibiting high basal levels may also increase the risk of toxicity. Despite uncertainty with optimal dosing strategies, recent data suggests that weekly supplementation of 40 000 IU per week for six weeks is superior to 20,000 IU in terms of magnitude of elevation, though it is noteworthy that six weeks of continued supplementation with 40 000 IU induces no further elevations beyond 100 nmol.L⁻¹ (Morton 2014). At present, daily supplementation of 5000 IU therefore appears a safe and practically relevant dose though players should also seek medical advice. In those athletes where a deficiency is not present, subsequent supplementation is unlikely to provide a health or performance benefit and may lead to toxicity and, therefore, is not recommended.





Table 5. Dietary supplementation for nutrient deficiencies

The following dietary supplements should be used in specific situations in sport using evidence-based protocols. They should be used by some athletes to directly contribute to optimal performance. The supplements should be used in individualised protocols under the direction and monitoring of an appropriate sports nutrition/medicine/science practitioner.

Medical supplements for nutrient deficiencies	Supplement
Medical supplements are used to treat clinical issues, including diagnosed nutrient deficiencies. Requires individual dispensing and supervision by appropriate sports medicine/science practitioner.	Iron
	Calcium
	Multivitamin/mineral
	Vitamin D





7. DIETARY SUPPLEMENTATION FOR ATHLETES

Supplements are commonly used amongst athletes and coaches in pursuit of enhanced performance, improved recovery and enhanced general health. It is important to note that there is rarely a need to supplement if the diet of the athlete is healthy, varied and balanced. There are exceptions where supplements can help performance or recovery but in any case they should be consumed to “supplement” a healthy balanced diet, not as a replacement. In this context a supplement is defined as a product intended for ingestion that contains a "dietary ingredient" intended to add further nutritional value to (supplement) the diet (Finley et al., 2013). A "dietary ingredient" may be one, or any combination, of the following substances: a vitamin, a mineral, a herb or other botanical, an amino acid or a dietary substance for use by people to supplement the diet by increasing the total dietary intake with a concentrate, metabolite, constituent or extract. Sports nutrition products such as sports drinks and recovery/protein drinks are not considered supplements (Morton 2014). A common misconception is that “supplementation” can compensate for poor day-to-day food choices. This is not the case.

Nevertheless, as training and competition become ever more demanding, every possible advantage must be seized and nutrition is an obvious area that can make a difference (Maughan 2013). Athletes, coaches, parents and sport science staff are often overwhelmed when faced with the challenge of developing a practical and evidence-based supplement strategy that supports training and competition. The single most important factor, if the decision is made to “supplement”, is that the chosen approach should adhere to the World Anti Doping Association (WADA) code of conduct. Specifically, it should be ensured that all supplements are free from prohibited substances. There are thousands of supplements available to athletes and the present chapter provides an evidence-based review of those supplements that may be considered suitable for practical use for athletes’ training and competition. Table 2 provides details of other supplement products, which are categorised as either “medical”, “performance” or “recovery”. Readers are referred to a comprehensive review of dietary supplements by Maughan et al. (2011).





Caffeine

Caffeine (chemical name 1,3,7-trimethylxanthine) is unique as it is found in a variety of drinks and foodstuffs (e.g. tea, coffee, cola, chocolate, etc.) and is perhaps the most widely studied and research-proven of all ergogenic aids (a supplement taken to improve performance). Indeed, caffeine has been consistently shown to improve both cognitive and physical performance across a range of endurance sports such as running, cycling, rowing and swimming (Burke 2008). However, numerous data suggest that caffeine also improves the physical and technical elements of performance that are inherent to most team competitions. For example, caffeine can enhance repeated sprint and jump performance (Gant et al., 2010), reactive agility (Duvnjak-Zaknich et al., 2011) and passing accuracy (Foskett et al., 2009) during intermittent-type exercise protocols. The ergogenic effects of caffeine are typically achieved with ingestion of 2-6 mg/kg BM (Burke 2013). Given that plasma caffeine levels peak approximately 45-60 min after ingestion (Graham & Spriet 1995), it is recommended to consume caffeinated drinks, capsules or gels (depending on players' preferences) within the warm-up period prior to kick-off.

Although the precise ergogenic mechanisms are still considered elusive, most researchers agree that the ability of caffeine to modulate the central nervous system (CNS) is the predominant mechanism (Meeusen 2014). Indeed, caffeine is readily transported across the blood-brain barrier and can act as an adenosine antagonist, thereby opposing the action of adenosine. As such, caffeine can increase concentrations of important neurotransmitters such as dopamine (Fredholm 1995), the result of which manifests itself as increased motivation (Maridakis et al., 2009) and motor drive (Davis et al., 2003). In addition to its effect on the CNS, recent data suggest that caffeine may also exert its ergogenic influences during high-intensity intermittent exercise through an additional mechanism related to maintenance of muscle excitability. Indeed, Mohr et al. (2011) observed improved performance on the Yo-Yo Intermittent Recovery Test 2 following caffeine supplementation that was associated with reduced muscle interstitial accumulation of potassium (K^+) during intense intermittent exercise. The latter observation is consistent with the notion that extra-cellular accumulation of K^+ is a contributing cause of fatigue during very high-intensity exercise (Mohr et al., 2011).

In contrast to competition days when specialised caffeinated sports products are typically





consumed, players may achieve ergogenic effects on training days by consuming caffeine in the form of tea or coffee with their breakfast meal prior to training (Morton 2014). Indeed, this strategy seems appropriate given recent evidence that pre-exercise coffee ingestion induces similar performance benefits to that of anhydrous caffeine ingestion (Hodgson et al., 2013). Finally, it has been suggested that post-training caffeine ingestion may help promote recovery and performance during a subsequent training session undertaken on the same day. Indeed, post-exercise muscle glycogen re-synthesis was enhanced when caffeine (8 mg/kg BM administered as 2 x 4 mg/kg BM doses at 2-h intervals) was co-ingested with post-exercise carbohydrate feeding (Pedersen et al., 2008). It is noteworthy, however, that not all researchers have observed that post-exercise caffeine ingestion enhances muscle glycogen re-synthesis (Beelen et al., 2012).

Despite the substantial evidence base supporting caffeine ingestion for exercise performance, it is highly recommended that players initially experiment with caffeine in training sessions. Caffeine can have a number of adverse 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 (Maughan et al., 2011). Indeed, not all individuals display performance enhancements after acute caffeine ingestion, and large doses (i.e. especially > 6 mg/kg BM) may often induce negative symptoms such as increased heart rate, irritability, tremor, confusion, reduced concentration and shortness of breath (Graham & Spriet 1995), many of which would have implications for skill-based performance. Furthermore, consuming high doses of caffeine prior to or during night competition can also be problematic given that sleep quality can be negatively affected (Drake et al., 2013).

Creatine

Like caffeine, creatine is one of the most widely researched supplements and has a strong supporting evidence base. Creatine is a guanidine compound that it is synthesized in the liver and kidneys from the amino acids arginine and glycine. From a dietary perspective, the predominant sources of creatine are fish and red meat. For example, 1 kg of fresh steak contains about 5 g of creatine (Maughan et al., 2011). The largest store of creatine in the body is skeletal muscle (Wyss & Kaddurah-Daouk 2000), where approximately 60-70% is stored as a phosphorylated form known as phosphocreatine (PCr). Creatine supplementation has traditionally been associated with strength and power athletes, such as weightlifters and





sprinters, given the role of PCr hydrolysis in regenerating ATP during the initial seconds of supra-maximal activity. However, in the context of high intensity intermittent team sports, creatine supplementation is also of particular interest given that PCr stores exhibit significant declines during football match play (Krustrup et al., 2006). Accordingly, creatine supplementation improves repeated sprint performance during both short duration (Casey et al., 1996) and prolonged intermittent exercise protocols (Mujika et al., 2000), likely due to increased resting muscle PCr stores as well as improved rates of PCr re-synthesis in the recovery periods between successive sprints (Casey et al., 1996). In both studies cited here, sprint performance improvements following creatine supplementation (and compared to a placebo) were in the range of 1-4%. In addition to augmenting repeated sprint performance, players may also wish to consume creatine with the goal of augmenting training-induced improvements in muscle mass, strength and power (Branch 2003).

Harris and colleague provided the initial evidence that creatine supplementation (using a loading protocol of 20 g/d for 5 days) increased (in the magnitude of 20%) both total creatine and PCr stores in skeletal muscle (Harris et al., 1992). As such, the conventional creatine dosing strategy is to undertake a loading protocol (usually involving 4 x 5 g doses/d for 5-7 days) followed by a daily maintenance dose of 3-5 g/d (Hultman et al., 1996). However, given that player adherence to such a protocol may be limited, it is noteworthy that daily consumption of a lower dose over a longer period (i.e., 3 g/d for 30 days) will eventually augment muscle creatine to a similar level as that observed with classical loading protocols (Hultman et al., 1996). Upon cessation of supplementation, the elevated muscle creatine stores tend to return towards basal levels within 5-8 weeks. To maximize creatine storage, it is also recommended that creatine is consumed post-exercise and in conjunction with carbohydrate and/or protein feeding, given that contraction and elevated insulin are known to increase muscle creatine uptake (Robinson et al., 1999). In a practical context, this means ensuring creatine provision before and after training periods in conjunction with other sports nutrition products containing carbohydrate (and/or protein) or with whole food provision at the main meals of breakfast, lunch and dinner. Prior loading with creatine may also enhance post-exercise muscle glycogen re-synthesis rates (Robinson et al., 1999). Considering the difficulty of replenishing post-game muscle glycogen stores even with sufficient carbohydrate and protein intakes, this strategy appears relevant during those periods of intense fixture schedules when multiple games are played with limited recovery time.





It is important to note that not every individual will respond similarly to creatine supplementation in terms of both augmentation of muscle creatine stores and subsequent improvements in performance. Indeed, the magnitude of elevation of muscle creatine to a given dose of creatine supplementation is highly variable and appears to be largely determined by the initial level of muscle creatine concentration prior to supplementation, the latter likely determined by habitual diet (Hultman et al., 1996). In general, individuals with lower muscle creatine stores exhibit greater increases in total muscle creatine during supplementation compared with those individuals who already exhibit high concentrations of muscle creatine. Accordingly, creatine-induced improvements in intermittent exercise performance are greater in those individuals who exhibited larger increases in muscle (especially Type II fibres) creatine and PCr (Casey et al., 1996).

Acute creatine supplementation (i.e. loading) can also induce a 1-1.5 kg gain in BM, an effect that is greater in men compared with women (Mihic et al., 2000). Such increases in BM are confined to fat free mass and are likely due to an increase in intra-cellular water accumulation. For this reason, not all players may choose to supplement with creatine given the perception that they feel heavier and slower, an effect that may be especially relevant for those who rely on speed and agility as key physical attributes athletes (such as strikers and wide midfielders in football). Additionally, creatine supplementation is also often perceived to have negative health effects in terms of liver and kidney function. It is noteworthy, however, that prospective studies demonstrate no adverse health effects in healthy individuals who were long-term creatine users (Kim et al., 2011).

In general, the available evidence supports a beneficial effect of creatine on short term high intensity and repeated sprint exercise. Creatine has several key roles within skeletal muscle as a temporal energy buffer, energy carrier and maintaining ATP/ADP ratios (Greenhaff 2001). Given that it takes weeks for creatine stores to return towards basal levels upon the cessation of supplementation (hence ergogenic effects should still occur), it may be prudent for athletes to “cycle” creatine supplementation at specific stages of the season (e.g. pre-season, congested fixture schedules) and/or training goals (e.g., strength / hypertrophy goals) (Morton 2014).





β -alanine

In skeletal muscle cells, β -alanine combines with L-histidine to form the dipeptide β -alanyl-L-histidine, the latter more commonly known as carnosine. Carnosine is of particular reference for high-intensity exercise performance given that it can act as an intracellular buffer to hydrogen ions (H^+) due to its imidazole ring having a pKa of 6.83 whilst also being present in muscle at fairly high concentrations (e.g. 10-60 mmol/kg d.w) (Hobson et al., 2012). Given the repeated sprint nature of football match play, muscle pH declines to levels that may impair the capacity to generate ATP through glycolytic metabolism (Krustrup et al., 2006). As such, it has become common practice for football players to consume daily β -alanine supplements (as the rate-limiting determinant of carnosine synthesis) to increase muscle carnosine stores and hence, potentially improve high-intensity exercise performance. Indeed, in relation to the former, daily β -alanine supplementation has been consistently shown to elevate skeletal muscle carnosine concentration by approximately 50% in both type I and II human skeletal muscle fibres (Harris & Sale 2012). Furthermore, in recent meta-analyses, Hobson et al. (2012) concluded likely ergogenic effects of β -alanine supplementation during high-intensity sports lasting in duration from 1- 6 min such as track and field events, cycling, rowing and swimming.

Unfortunately, investigations evaluating the effects of β -alanine supplementation during high-intensity intermittent exercise protocols that are applicable to field sports such as football are both limited and conflicting. For example, Saunders and colleagues observed no beneficial effect of four weeks of β -alanine supplementation (6.4 g/d) on sprint performance during the field-based LIST (Saunders et al., 2012). In contrast, the same researchers later observed improved performance during the Yo-Yo Intermittent Recovery Test Level 2 following 12 weeks of daily supplementation with 3.2 g of β -alanine (Saunders et al., 2012). Unfortunately, neither studies measured changes in muscle carnosine stores following supplementation, though it is possible that the enhanced effect observed in the latter study was due to the longer period of supplementation. This hypothesis is especially relevant given that length of β -alanine supplementation is a determinant of increases in muscle carnosine concentration (Hill et al., 2007).

A negative side effect of β -alanine supplementation when administered as single doses >10 mg/kg BM (especially when in solution or as gelatin capsules) is a flushing of the skin and





tingly sensation (Harris et al., 2006), a phenomenon known as paraesthesia. To reduce such symptoms, sustained release formulations have been developed that allow two 800 mg doses to be ingested simultaneously without any symptoms (Decombaz et al., 2012). Although the optimal dosing and delivery strategy of β -alanine supplementation is not currently known, it is noteworthy that a significant linear relationship exists between total β -alanine intake (within the range of 1.6 - 6.4 g/d) and both relative and absolute increases in muscle carnosine (Stellingwerff et al., 2012a). To this end, Stellingwerff and colleagues (2012b) observed that four weeks of supplementation with 3.2 g of β -alanine daily induced 2-fold greater increases in muscle carnosine stores compared with 1.6 g daily. Moreover, these researchers also observed that subsequent daily doses of 1.6 g/d continued to induce further increases despite already high carnosine stores following the four weeks of higher dose β -alanine supplementation (Stellingwerff et al., 2012; Stellingwerff et al., 2012). More recently, it has been reported that following 6 weeks of 3.2 g β -alanine/d a further daily maintenance dose of 1.2 g/d was required to maintain muscle carnosine content elevated at 30-50% above baseline values (Stegen et al., 2014). Indeed, upon cessation of supplementation, muscle carnosine stores typically return towards basal levels within 10-20 weeks (Baguet et al., 2009). On the basis of the above background, it is therefore recommended that where muscle carnosine stores are required to be elevated quickly (perhaps during important stages of competition such as intense fixture schedules), loading with larger doses (e.g. 3-6 g daily for 3-4 weeks) may be initially beneficial followed by daily maintenance doses >1.2 g. To minimize symptoms of paraesthesia, players may benefit from consuming slow-release formulas in a number of doses spread evenly throughout the day. Finally, it has been shown that carnosine loading via β -alanine supplementation is more pronounced in athletes i.e. trained muscle in comparison to untrained individuals (Bex et al., 2014).

Nitrates

In recent years, dietary inorganic nitrate supplementation has received a significant amount of research attention due to the effects of nitric oxide on a variety of physiological functions. Indeed, nitric oxide has well-documented roles in regulating blood flow, muscle glucose uptake and contractile properties of skeletal muscle (Jones 2014). The traditional pathway of endogenous nitric oxide production is recognised as that of L-arginine oxidation, facilitated by the enzyme nitric oxide synthase. However, it is now known that dietary ingestion of





inorganic nitrate can also be metabolized to nitrite and subsequently, nitric oxide, thereby complementing that produced from the L-arginine pathway (Hord et al., 2011). Identification of this biochemical pathway has, therefore, led to a series of studies conducted in the last decade evaluating the effects of inorganic nitrate ingestion on exercise performance.

Nitrates are especially prevalent in green leafy vegetables such as beetroot, lettuce and spinach though the exact content can vary considerably based on soil conditions and time of year. As a means to provide a constant dose of nitrate, most researchers have therefore used standard doses of beetroot juice (0.5 L is equivalent to approximately 5 mmol nitrate) to elevate nitrate and nitrite availability. Using both chronic (ranging from 3-15 days of 0.5 L beetroot juice per day) and/or acute ingestion 2.5 h before exercise, it was collectively demonstrated that nitrate ingestion reduces blood pressure, lowers oxygen consumption for a given workload or velocity during steady-state exercise as well as improving exercise capacity during short-duration high-intensity cycling or running (Bailey et al., 2009; Vanhatalo et al., 2010; Lansley et al., 2011). These initial studies were later supported by experiments demonstrating that acute (Lansley et al., 2011) and chronic beetroot juice ingestion (Cermak et al., 2012) in trained but sub-elite athletes also improved cycling time trial performance in distances ranging from 4 km to 16.1 km (i.e., approximately 5-30 min of exercise). It is noteworthy, however, that the performance-enhancing effects of nitrate are not readily apparent in elite athletes (Wilkerson et al., 2012), likely due to a combination of underpinning differences in the physiology of elite versus sub-elite athletes that collectively render a trained athlete less sensitive to additional nitric oxide availability e.g. higher nitric oxide synthase activity, plasma nitrite values, greater muscle capillarization, higher type I fibres (Jones 2014).

The mechanisms underpinning the reduced oxygen cost of exercise and improved capacity / performance are currently thought to involve improved muscle efficiency and energy metabolism (Jones, 2014). For example, Bailey and colleagues observed that reduced oxygen uptake during exercise (following six days of 0.5 L beetroot juice ingestion per day) was associated with reduced PCr degradation and accumulation of ADP and Pi, thus implying a reduced ATP cost of contraction for a given power output and hence reduced signals to stimulate respiration (Bailey et al., 2010). Larsen et al. (2011) suggested that mitochondrial efficiency might be improved in human skeletal muscle following three days of sodium





nitrate ingestion (0.1 mmol/kg BM),(Larsen et al., 2011). More recently, Haider and Folland (2014) observed that seven days of nitrate loading in the form of concentrated beetroot juice (9.7 mmol/d) also improved in vivo contractile properties of human skeletal muscle, as evidenced by improved excitation-coupling at low frequencies of stimulation as well as explosive force produced by supra-maximal stimulation (Haider & Folland 2014).

The optimal loading dose to facilitate the ergogenic effects of nitrate is also not currently well known, especially in relation to whether acute (i.e. 2.5 h before exercise) or chronic (i.e. several days) loading protocols are required. Nevertheless, in the acute context, Wylie and colleagues (2013) observed that the improved exercise tolerance (relative to placebo) was not different when 8.4 or 16.8 mmol of nitrate was ingested 2.5 h before exercise, but that both were more efficacious than 4.2 mmol. It is noteworthy, however, that the reduction in oxygen cost during exercise associated with nitrate ingestion was greater with the higher dose (Wylie et al., 2013). Such data suggest that the inability to detect physiological effects of nitrate in acute scenarios (especially with elite athletes) may be overcome by using higher pre-exercise dosing strategies and/or longer duration dosing protocols (>3 days).

Despite the data reviewed above, convincing evidence demonstrating ergogenic effects of nitrate ingestion during intermittent exercise protocols relative to football is not yet available. However, using a more aggressive loading dose of concentrated beetroot juice (approximately 30 mmol in a 36 h period), Wylie and colleagues (2013) observed significant improvements in the distance run on the Yo-Yo Intermittent Recovery Test Level 1 when compared with placebo supplementation. Interestingly, these researchers observed reduced plasma glucose during exercise in the beetroot trial, suggesting that muscle glucose increased and that improved performance may be due to muscle glycogen sparing (Wylie et al., 2013). Additionally, improved performance may have been due to maintained muscle membrane excitability given that plasma K^+ was lower during exercise following beetroot juice supplementation. From a practical perspective, the use of an intense 36 h nitrate loading protocol is likely to gain more acceptance amongst football players than the conventional 3-6 day loading approach. Nevertheless, the practical application of nitrate supplementation (even in concentrated form) may be limited due to the taste and palatability issues of the current nitrate products that are commercially available. It is highly recommended that players experiment with nitrate supplementation (perhaps even more so than the supplements





reviewed previously) prior to implementing in high-level competition. Furthermore, and to promote the potential beneficial effects of nitrate supplementation, athletes are also advised to avoid antibacterial mouthwash and chewing gum, as these products diminish the nitrate-nitrite conversion (Jones 2014).

Table 6. Dietary supplementation for performance and recovery

The following dietary supplements should be used in specific situations in sport using evidence-based protocols. They should be used by some athletes to directly contribute to optimal performance. The supplements should be used in individualised protocols under the direction and monitoring of an appropriate sports nutrition/medicine/science practitioner. While there may be a general evidence base for these products, additional research may often be required to fine-tune protocols for individualised and event-specific use.

Performance	Supplement
Performance supplements are used solely to contribute to optimal performance. Should be used in individualised protocols under the direction of an appropriate sports medicine/science practitioner While there may be a general evidence base for these products, additional research may often be required to fine-tune protocols for individualised and event-specific use.	Caffeine
	Creatine
	B-alanine
	Dietary Nitrate
	Bicarbonate

Recovery	Supplement
Food polyphenols — food chemicals which have purported bioactivity, including antioxidant and anti-inflammatory activity. May be consumed in food form or as a concentrate.	Tart cherry juice
	Glutamine
	Fish Oils



8. NUTRITION AND TRAINING ADAPTATIONS

Athletes invest a considerable amount of time training in order to optimize their performance and/or change their physique. The training adaptations are the result of an accumulation of specific proteins. Each training session will cause disruptions in cellular homeostasis which will signal the muscle to increase protein synthesis. These disruptions and the responses are highly specific to the sport and the type of training and, therefore, different types of training will result in vastly different proteins to be synthesized (Nader & Esser 2001; Hildebrandt et al., 2003). For example, strength training will result in an increase in muscle strength and mass whereas endurance training will result in increased mitochondrial mass and endurance. As a result, the different types of training will result in distinctly different phenotypes being expressed (Baar 2014; Baar 2014). It is thought that chronic training adaptations are the result of the cumulative effect of transient events that occur during and after acute bouts of exercise. Evidence is emerging that certain nutrients can be potent modulators of these events that occur during exercise and in the recovery phase. Therefore, nutrition is a major determinant of the adaptations that occur in response to a training program.

At the onset of exercise the rapid changes in the muscle calcium content, the ATP/ADP ratio, Na^+/K^+ ATPase activity and pH (and other factors) can stimulate certain signalling proteins (kinases and phosphatases). The most studied signalling proteins are 5'-adenosine monophosphate-activated protein kinase (AMPK), mitogen-activated protein kinase (MAPK) and the mammalian target of rapamycin (mTOR). The protein AMPK has been described as a “fuel sensing” molecule involved in many metabolic responses in skeletal muscle during exercise. AMPK is activated by disturbances in energy status and will stimulate various processes geared towards ATP production whilst inhibiting ATP consuming processes (Hardie & Hawley 2001). The effects of AMPK are both acute by switching on and off catabolic and anabolic processes (enzymes) and chronic by altering gene expression (for example AMPK increases GLUT4 protein expression and increases mitochondrial density) (Aschenbach et al., 2004; Baar 2014). The MAPK signal transduction pathway has been suggested to be the system responsible for translating contraction-induced biochemical perturbations into appropriate intracellular responses (Widegren et al., 2001). Exercise activates the MAPK pathway and plays a role in the transcriptional regulation of





protein synthesis as well as stimulating the translational stage of protein synthesis. The mTOR pathway plays a role especially in muscle growth (Baar 2014). Both insulin and amino acids are potent stimulators of the mTOR signalling cascade (Deldicque et al., 2005), although activation of AMPK, however, will inhibit mTOR. This will have important practical implication as endurance training may increase mitochondrial biosynthesis but may not result in muscle hypertrophy.

Changes in diet can alter the concentrations of nutrients and hormones in blood and can change the body's energy stores. These factors in turn can have an impact on the initial signalling cascade and various stages of protein synthesis. The availability of macronutrients has been shown to have an impact on gene expression post-exercise. Muscle glycogen content for example is a potent modulator of the resting and contraction-induced AMPK and MAPK responses (Philp et al., 2012). At rest AMPK activity is 2.5 fold higher in a low glycogen versus a high glycogen state. Similarly, increasing glucose availability by feeding carbohydrate during exercise has been shown to blunt the effects on gene expression (Civitarese et al., 2005) and it has been proposed that training in a low glycogen state (Hansen et al., 2005) and without carbohydrate feeding (Civitarese et al., 2005) may result in superior training adaptations. Several years ago untrained participants trained for 10 weeks (Hansen et al., 2005) during this training period one leg was trained in a low glycogen state half of the time whereas the other leg always trained with high glycogen. Both legs performed the same training and it was observed that the "low glycogen leg" had a more pronounced increase in resting muscle glycogen and citrate synthase activity.

However, there are several limitations to the studies performed that make it impossible to translate these findings into practical advice. For example, it is unlikely that in a free living situation the same training would be performed in a low glycogen versus a high glycogen state. In fact, training with low glycogen in some studies (especially when training programs were very strenuous) resulted in a reduced training adaptation (Simonsen et al., 1991) and resulted in more symptoms of overreaching when very hard daily training was performed (Achten et al., 2003). Also, a recent study showed no differences in training adaptation when carbohydrate was ingested during training sessions compared with fasted sessions (Akerstrom et al., 2006). Despite these findings, there is evidence that training in a low glycogen state and with reduced carbohydrate availability will result in enhanced signalling





and gene expression in pathways associated with fat oxidation (Yeo et al., 2010; Camera et al., 2012). Therefore, carbohydrate availability can be manipulated acutely around exercise to promote the adaptation which the training session is aiming to target (Bartlett et al., 2014).

Diets high in fat have been shown to increase the expression of mRNA-encoding proteins that are relevant to fatty acid metabolism (Cameron-Smith et al., 2003) within five-to-seven days. It has therefore been suggested that high-fat diets might give similar adaptations to endurance training (Hawley 2011; Yeo et al., 2011). However, Stellingwerff and colleagues (Stellingwerff et al., 2006) fed their subjects a high fat diet for five days followed by one day of carbo-loading. They observed that the high fat diet actually reduced the activity of pyruvate dehydrogenase (PDH), a key enzyme in glycolysis, and could therefore actually result in an impairment of carbohydrate metabolism. The authors suggested that the observed glycogen sparing effect with high fat diets is probably more a result of impaired carbohydrate metabolism rather than increased fat metabolism.

In addition to insulin and exercise, amino acids stimulate the phosphatidylinositol 3-kinase (PI3K) mTOR signaling pathway. Acute protein ingestion near the time of exercise appears to have the greatest potential on training adaptations (Baar 2014). Ingesting a mixture of carbohydrate and amino acids before or immediately after a training session increases amino acid availability and transport into the muscle, increases protein synthesis and reduces protein breakdown, resulting in a net positive protein balance (Biolo et al., 1997; Tipton et al., 2001). It seems that the effect of post-exercise amino acid ingestion is enhanced by the co-ingestion of carbohydrate (Miller et al., 2003), most likely because of the elevated insulin concentrations. The amount of protein required to elicit this effect is very small (~6 g) (Tipton et al., 1999; Tipton et al., 2001) but these small amounts are far more effective in increasing protein synthesis than the ingestion of carbohydrate only (Borsheim et al., 2004). Little is known about the effects of the type of amino acids or the form in which they are ingested, and the timing of intake on long term training adaptations and future research will focus on these questions.

In summary, it is thought that chronic training adaptations are the result of the cumulative effect of transient events that occur during and after acute bouts of exercise. Evidence is emerging that certain nutrients can be potent modulators of these events that occur during exercise and in the recovery phase. Little is known about the effects of various nutrients and





exercise on signalling responses, gene expression and protein content. This is an area that is likely to develop rapidly over the next few years as athletes try to increase the effectiveness of their training.





9. NUTRITION FOR THE INJURED ATHLETE

Injuries are an unfortunate aspect of physical activity – regardless of the level of participation. An injury may strike any individual from those exercising for health and enjoyment up to the elite athlete (Tipton 2010). Injuries usually occur when the athlete is engaged in training or competition for their sport. Activities such as running, sprinting, jumping and kicking increase the physical demands placed on the body above that at rest and thus acutely increase the risk of injury.

Activities during exercise lead to both transient in game and post-match fatigue that is linked to a combination of factors, including dehydration, glycogen depletion, muscle damage and neuromuscular fatigue (van Beijsterveldt et al., 2013). The magnitude of competitive exercise-induced fatigue is dependent on intrinsic and extrinsic factors. Extrinsic factors include the result, quality of the opponent, match location and playing surface, whereas intrinsic factors include training status, age, gender and muscle fibre type. Both intrinsic and extrinsic factors have the potential to influence the time course of recovery from fatiguing exercise, making it a complex issue (Nedelec et al., 2012).

In many sports, the demands placed upon professional players are growing because of the increased fixture schedule, resulting in less recovery periods between training and competitive match play and increasing the risk of injury (Dellal et al., 2013). It has been suggested that when the recovery time between two matches is 72 to 96 h, there is sufficient recovery time to maintain the level of physical performance. However, 72-96 h recovery time between completion may not long enough to maintain a low injury rate (Dupont et al., 2010). During periods when the schedule is particularly congested (i.e. two matches per week over several weeks), the recovery time allowed between two successive matches lasts three to four days, which may be insufficient to restore player homeostasis. As a result, players may experience acute and chronic fatigue potentially leading to underperformance and/or injury (Nedelec et al., 2012). For example, in elite European football, players play between 51-78 games a season, averaging 1.6 to 2 matches per week (excluding friendly games). For instance, 80% of the FC Barcelona Professional squad averaged 65 official competitive games throughout seasons 2010-2013. Dupont and colleagues (2010) reported a 6.2-fold higher injury rate in players who played two matches per week compared with those who





played only one match per week. During congested schedules, recovery strategies are commonly used in an attempt to regain performance faster and reduce the risk of injury (Nedelec et al., 2012). The majority of data collected regarding injuries has been collected in football. It is most likely that observations and data from football can extend to other sports which experience reduced recovery time as a consequence of fixture congestion. Thus, this chapter will first focus on common injuries in football before discussing the current nutritional recommendations in response to an injury.

Epidemiology of football injuries

The incidence of outdoor football injuries is among the highest of all sports, particularly for adult male players. An elite football team with 25 players in the squad can expect about 50 injuries each season. Half of the injuries will be minor, causing absences of less than a week, but as many as eight or nine will be severe injuries causing absences of more than four weeks. The incidence has been described to be around 24.6 and 34.8 per 1,000 match hours, and 5.8 to 7.6 per 1,000 training hours (Ekstrand et al., 2011). Almost one-third of all injuries in professional football are muscle injuries. The majority (92%) of injuries affect the four major muscle groups of the lower limbs: hamstrings 37%, adductors 23%, quadriceps 19% and calf muscles 13%. A team of 25 players at elite level can expect about 15 muscle injuries each season with approximately two weeks missed for each injury. Interestingly, the incidence of match injuries has shown an increasing tendency over time in both first and second halves. Some authors have proposed that this is a consequence of insufficient recovery time between games resulting in chronic fatigue (Ekstrand et al., 2011). However, fatigue may also acutely manifest as the duration of a match increases. Thus, specific nutrition strategies to delay fatigue during games may also have an important role in injury prevention (Medina et al., 2014).

Preventative measures

As described previously, muscle injuries represent almost one-third of all injuries in football. Strategies involving hydration, diet, sleep and cold water immersion have been reported to be effective with regard to their ability to counteract the mechanisms associated with muscular fatigue (Nedelec et al., 2012). Recovery strategies aimed at reducing acute inflammation from muscle damage and speeding the rate of inflammation removal are prevalent in professional football settings. Sleep and other methods important for recovery have been





reviewed in detail (Halsen et al., 2003; Halsen 2013). Thus, the focus in this Chapter will be placed on preventive measures, monitoring and recovery of muscle fatigue from a nutritional perspective.

Body composition and injury prevention

Body composition must be assessed at the time of injury, specifically, total BM, lean mass and fat mass. Changes in body composition during injury typically involve increased body fat and decreased lean mass from an early stage. These changes are not always reflected in BM, as BM may increase, decrease or stay relatively constant depending on the ratio of lean- and fat mass change (Peterson et al., 2011).

Being overweight causes more mechanical stress in contact and weight-bearing sporting activities, thereby increasing the risk of injury. Interestingly, abdominal fat (assessed by DXA or abdominal circumference) has been reported as a better predictor of muscular-skeletal injury than body mass index (BMI); this correlation increases with age (Nye et al., 2014). It is important to note that although BMI is frequently used for the general population, players with low body fat and high muscle mass are classified “overweight.” Thus, the use of BMI to monitor body composition is inappropriate for football and athlete-specific recommendations in general.

Body composition is important for elite football and players at professional clubs appear to be a homogenous group with little variation between individuals. The percentage body fat for professional football players has been reported to be $10.6 \pm 2.1\%$ (Sutton et al, 2009). Body composition varies during preseason; a general decrease in abdominal fat mass and increased lean mass in the legs are generally observed. Conversely, during a long period of injury an overall decrease in lean mass is noted, with more marked changes in muscle atrophy and fat deposition in the injured region or segment (Reinke et al., 2009).

Recently, there has been interest in developing injury prevention models based on ratios of different tissues. Schinkel-Ivy and colleagues describe the ratio of components of the lower extremity; showing the ratio between soft and hard tissues, defined as "Tissue mass ratio". The “Tissue mass ratio” differs according to sports and is believed to be optimized by adaptation to the type of stimulus or impact received (Schinkel-Ivy et al., 2014). Thus, this





ratio may be of consideration when planning nutritional interventions and in the prevention and monitoring of injury. Barbat-Artigas et al. (2012) reported that the fat mass:bone mass ratio of a limb correlates inversely with the risk of injury, being lower in non-injured athletes in comparison to those who have suffered injury (Barbat-Artigas et al., 2012). Other indices such as "muscle quality index" correlate the muscle area of a limb and the force or power output (Fragala et al., 2014). This index may be a useful evolutionary parameter when monitoring changes in muscle mass and function in limbs during a period of injury.

Dietary considerations for injury prevention

The energy intake of the athlete should match the daily energy demands (Chapter 1). For example, the energy cost of football is approximately 1,300-1,500 kcal for a 90-min game, depending upon playing position, tactics and body composition of the player. The amount of energy required may be adjusted to reflect the lean body mass (in kg) of the individual athlete. Global positioning satellite technology can be used as a tool to approximate the energy cost of training sessions.

An insufficient energy intake does not cover energy required for performance, training and daily living activities. It has been reported that energy intakes below 30-35 kcal/kg lean body mass (excluding exercise) accentuate fatigue, immune-suppression and the predisposition to injury (Loucks et al., 2011). Furthermore, low-energy diets in which calories are not consumed via a variety of foods typically have low nutritional quality. Insufficient energy intakes combined with poor dietary choices increase the risk of players being deficient in nutrients such as vitamins B or C and minerals like iron, calcium, magnesium, zinc and selenium. Interestingly, inadequate plasma vitamin D concentrations have been observed during the winter months in top-level football players (<30 ng/mL) (Morton et al., 2012). Low vitamin D may affect bone metabolism and has been associated with alterations in strength and muscle components. Therefore, vitamin D status may be a consideration in injury prevention. Unfavourable lipid profiles (pro-inflammatory) due to excesses in the diet of trans-fat, saturated fat and excessive omega-6 fat from vegetable oils should be avoided. Instead, players are encouraged to regularly eat foods, such as oily fish, for a source of omega-3 (Simopoulos, 2007).





Alcohol intake after training and competition reduces rates of myofibrillar protein synthesis even if co-ingested with protein. The suppression of the anabolic response in skeletal muscle will impair recovery and adaptation to training (Parr et al., 2014).

Dupont et al. (2010) reported that the injury rate increases according to hours of football exposure. However, the risk of injury is significantly increased when games overlap training with less than 72 h between them. In this circumstance (a recovery period under 72 h) it is necessary to emphasize optimal nutritional recovery strategies. Specifically, the restoration of muscle glycogen after exercise can be achieved by ingesting approximately 60 g of carbohydrates per hour during the first 2-3 hours (Rollo 2014). Protein intake is recommended immediately post exercise (0.3 g/kg BM, ~20-25 g), together with appropriate volumes of fluid to rehydrate, as covered in Section 5 of the current document (Laitano et al., 2014). Some studies suggest the use of nutritional anti-inflammatory aids such as the flavonoids quercetin or melatonin, while "tart cherry juice" may also be of benefit when recovery time between competitive matches is inadequate. However, evidence is limited and discussion of their application to other aspects of athletic performance is beyond the scope of this current document (Howatson et al., 2010).

Nutrition in different phases of recovery from injury

Nutritional interventions should be coordinated with the different phases of the recovery process to optimize the healing process. From this point of view, injury can be classified in two distinct phases: the immobilisation phase and the functional recovery phase (rehabilitation and re-training). During these phases muscle wasting and atrophy are commonly observed. Therefore, the main objectives are reducing inflammation and increasing anabolic stimuli (Tipton 2010). For those undergoing surgery a "preoperative" phase could also be considered. For example, it has been proposed that whey protein supplementation in the "preoperative" phase can reduce the acute inflammatory response post surgery (Perrone et al., 2011).

Muscle strength loss and atrophy markedly appear within five days of immobilization due to a rapid increase in muscle protein breakdown (MPB) followed by a decrease in muscle protein synthesis (MPS). Around 150 g of muscle mass is lost per day, equivalent to 1





kg/week, with type II muscle fibres being the most susceptible to atrophy (Wall & van Loon, 2013). After ten days muscle loss is mainly caused by MPS inhibition, basal and post-prandial, causing atrophy and functional loss. The decrease in MPS, even post-prandial and known as "anabolic resistance", is conditioned by inactivity and injury. Cytokines and catabolic factors, such as myostatins, block the processes in a similar response to aging-related sarcopenia (Wall et al., 2013). Thus, the effectiveness of protein ingestion is impaired and even in the presence of adequate levels of amino acids, protein synthesis is clearly inferior to the situation of no injury. The key issue seems to be muscle stimuli, since anabolic resistance will remain as long as muscle stimulation is lacking. Of note are methods such as percutaneous electro-stimulation and training the uninjured limb or other muscle groups that can exert some cross effect to diminish the anabolic resistance (Farthing et al., 2009).

From a nutrition perspective, the use of some supplements such as leucine may partially attenuate the decrease in MPS through the activation of mTOR (van Loon 2012). Leucine is an essential amino acid found in greater amounts in proteins of high biological value (i.e. whey protein). The ingestion of three grams of leucine, isolated or contained in protein, is capable of activating MPS in muscle resistant to insulin (Katsanos et al., 2006). Food also offers a good source of leucine; for example, 3 g of leucine can be found in 25-30 g of whey protein, 140 g of chicken or 170 g of fish. The catabolite of leucine, beta-hydroxy beta-methylbutyrate (HMB) ingested at three g per day has also been reported to be an effective supplement in the activation of MPS, although further research is needed before its use can be recommended, especially in consideration for the injured player (Molfino et al., 2013). Finally, the ingestion of four g/day of omega-3 fatty acids may act synergistically with leucine, increasing protein synthesis (Smith et al., 2011; Smith et al., 2011).

Acute injury phase

The acute injury phase is characterized by inflammation and, depending on the injury, immobilisation, reduced weight bearing and rest. The daily energy intake needs to be adjusted to the current needs, which are generally lower than before the injury due to lower activity. It is important to note that some metabolic stress injuries require an increase in energy requirements such as bone fractures or walking with crutches. However, detailed discussion on how energy intake should be adjusted for specific injuries is beyond the scope of the current policy and professional guidance should be sought.





During the acute injury phase, a protein intake of up to 2 g /kg BM is recommended (Tipton 2010). Protein requirements can be achieved by either ingesting food or supplements containing protein of high biological value at regular intervals throughout the day (fractional dose of 25-30 g, (Res 2014)). One strategy is to ingest whey protein between meals at mid-morning or mid-afternoon. Finally, protein intake prior to sleep is also recommended, in this instance the slow-release protein casein is a good choice (Churchward-Venne et al., 2012). Fat intake recommendation should be focussed on omega-3 rich foods such as oily fish, dried nuts, olive oil and avocado, and excess omega-6 intake should be controlled, where possible, as well as other sources of saturated fat. As commented above, supplementing with Omega-3 in doses of four g/ day are also recommended for the injured athlete.

Functional recovery phase

This phase is characterized by progressive hypertrophy and functional recovery. In long-term injuries this phase can be subdivided into regeneration, functional recovery and reconditioning discussed below:

Regeneration phase

In this phase the exercise workout is focused on non-injured muscle groups. General guidelines include adjusting calories to lean mass and controlling carbohydrate intake, choosing low GI foods such as vegetables and legumes. Protein intake is prioritized after exercise (20-25 g/ serving). Interestingly, this phase may benefit from creatine supplementation (Tipton 2010). Creatine has been suggested to help with the recovery of muscle mass after immobilization when supplemented individuals are compared to non-supplemented individuals. An easy way to achieve this is to incorporate creatine into any protein drinks that the player is ingesting at the time (Op 't Eijnde et al., 2001).

Functional Recovery

This phase involves a progressive return of the athlete to their field of play. The greater energy expenditure, as training volume and intensity increases, requires an increase in daily carbohydrate intake to about 3-5 g of carbohydrate/ kg BM (Chapter 3). Appropriately formulated carbohydrate sports beverages are typically ingested during and after exercise, to





help meet player fuel and fluid requirements (Rollo 2014). After exercise, recommendations for protein (Chapter 4) are maintained.

Reconditioning or Alternative Training Phase

In this phase previous recommendations, in line with optimal nutrition practice for the player, should be adapted to ensure, and help support, full recovery (advice on key macronutrients, carbohydrate, fluid and protein for football are discussed in previous chapters of the current policy).

In summary, the demands placed upon professional athletes are growing because of increased fixture schedules with less recovery periods between training and competitive match play, resulting in an increased risk of injury. Appropriate nutrition should be used during exercise to avoid fatigue. Recovery strategies are commonly used in an attempt to regain performance faster and reduce the risk of injury. The assessment of body composition is important for elite athletes, while abdominal fat is a good predictor of musculoskeletal injury and can be used as a monitoring tool during recovery of musculoskeletal injury. Nutrition is among the key recovery strategies in professional sport and interventions for recovery should focus on adequate energy intake to meet the macro and micronutrient needs via foods or appropriate supplementation. During injury, MPS is reduced by inactivity and, as a result, the muscle should be stimulated, in concert with the ingestion of suitable quantities of protein with high biological value.

In summary, during recovery from injury the nutrition principles are not too different from those for optimizing protein synthesis in muscle. Recommendations for protein intake are similar to those discussed for healthy athletes. However, it is important to consider overall energy intake, in relation to energy expended and there may be a role for certain nutrients (fish oils) to manage inflammation.





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