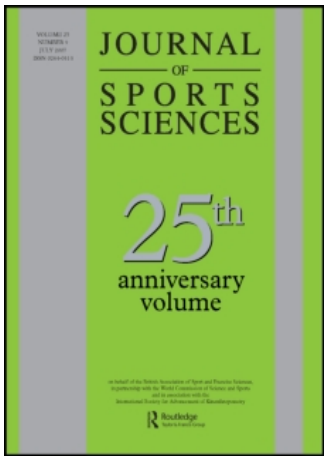

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Nutritional strategies for football: Counteracting heat, cold, high altitude, and jet lag

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Nutritional strategies for football: Counteracting heat, cold, high altitude, and jet lag

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Abstract
Environmental factors often influence the physical and mental performance of football players. Heat, cold, high altitude, and travel across time zones (i.e. leading to jet lag) act as stressors that alter normal physiological function, homeostasis, metabolism, and whole-body nutrient balance. Rather than accepting performance decrements as inevitable, well-informed coaches and players should plan strategies for training and competition that offset environmental challenges. Considering the strength of scientific evidence, this paper reviews recommendations regarding nutritional interventions that purportedly counterbalance dehydration, hyperthermia, hypothermia, hypoxia, acute or chronic substrate deficiencies, sleep loss, and desynchronization of internal biological clocks.

Keywords: *Metabolism, carbohydrate, glycogen, fat, protein, hydration*

Background

The energy requirement for competitive football is large and is influenced by many factors. The mean rate of aerobic energy production for elite players is 70–80% of maximal aerobic power ($\dot{V}O_{2max}$) during the course of a 90 min match. The energy cost of an entire match is 5–6 MJ (1195–1434 kcal), depending on the total distance covered (~10–11 km) and the style of play (Bangsbo, 1994; Ekblom, 1986). These values reflect environmental, physiological, tactical, and technical factors (Kuzon *et al.*, 1990; Mohr, Krustrup, & Bangsbo, 2003; Shephard, 1999).

The metabolic responses and substrate utilization that occur during football are difficult to study because play involves intermittent exercise, varied intensities, and rest periods. In attempts to reproduce match-play, some investigators have designed laboratory simulations, whereas others have conducted controlled studies involving intermittent exercise based on match characteristics (Bangsbo, Norregaard, & Thorsoe, 1991). In general, these research studies indicate that football places great demands on both aerobic and anaerobic energy-producing systems (Balsom, 1995; Shephard, 1999). For example, the liver must mobilize stored glycogen to maintain blood glucose during a match. Also, a pronounced utilization of stored muscle glycogen occurs during a match, indicating that (at a high exercise intensity) substrate availability for anaerobic energy production may be a limiting factor for performance (Balsom, 1995), as evidenced by frequent reports of peak blood lactate concentrations in the range of $4-10\,mmol\cdot l^{-1}$ (Bangsbo, 1994). Regarding lipid metabolism, the concentration of plasma free fatty acids rises (probably due to effects of increased catecholamines and suppressed insulin) during the later stages of a contest, with only a minor increase of plasma glycerol (Shephard, 1999). This suggests a large uptake of glycerol by various tissues, likely as a precursor of gluconeogenesis (Bangsbo, 1994). The roles of intramuscular lipolysis, circulating ketone bodies, and protein as energy sources are not well described (Bangsbo, 1994; Essen, 1978).

Further complicating scientific investigations, football is played in a variety of extreme environments around the world, including heat, cold, and high altitude (Askew, 1995). As athletes move into these environments and travel across time zones, metabolism and substrate utilization change, as does human performance (Armstrong, 2000; Committee on Military Nutrition Research, 1996, 1999a, 1999b; Shephard, 1999; Wilbur, 2004). Today, physiologists and dietitians view nutrition as a key factor to

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offset the physical and cognitive performance decrements that occur in stressful environments (Askew, 1996). Therefore, the purposes of this review are to: (a) describe the effects of four environmental factors on exercise metabolism, whole-body nutrient balance, and performance; (b) review nutritional interventions that may counteract these effects; (c) evaluate the strength of evidence regarding nutritional interventions; and (d) recommend directions for future research. This review does not consider pharmacological interventions or banned substances (FIFA, 2004).

Environmental influences on metabolism

Previous publications have described the metabolic changes induced by living in stressful environments. For example, two classic studies utilized animals to explore the effects of heat ($>35^{\circ}$C), cold ($<5^{\circ}$C), and high-altitude (>5500 m, 25°C) stresses on metabolism. After being exposed to each environment for 4 weeks, rats exhibited changes of food and water intake, as well as altered excretion rates for electrolytes and nitrogen (i.e. representing protein and non-protein compounds) (Mefferd, Hale, & Martens, 1958), compared with a control group. The rate of food intake and the rate of waste excretion were considerably greater in the cold-exposed group than the heat-exposed group, reflecting an elevated metabolic rate (i.e. shivering) in the cold. Furthermore, none of the animals had normal weight gains, in comparison to the control group, suggesting that their metabolism did not adapt to these environmental extremes within 4 weeks (Mefferd *et al.*, 1958).

Table I presents the four environmental factors that are considered in this review, and the human physiological outcomes that result from each. The primary threats to homeostasis are dehydration, hyperthermia, hypothermia, hypoxia, acute or chronic substrate deficiencies, sleep loss, and desynchronization of internal biological clocks. Table I also describes the specific effects of heat, cold, high altitude, and transmeridian air travel on metabolism and performance.

Dehydration affects metabolism and performance

The four environmental factors in Table I encourage dehydration. Heat exposure is the most obvious, due to sweat losses. Exposure to cold and high-altitude environments stimulates diuresis (i.e. transient increase of urine production), predisposing athletes to dehydration. Also, during lengthy airline flights, dry cabin air and restricted access to food and water result in mild-to-moderate dehydration.

In recent years, numerous studies led to a theory regarding the role that cell shrinkage (i.e. dehydration) plays in cellular metabolism and hormonal responses. This theory states that, when cells shrink, metabolism becomes predominantly catabolic and, when cells swell, metabolism becomes anabolic. Thus, cell shrinkage and swelling lead to opposite effects on protein, carbohydrate, and lipid metabolism. For example, measurements involving humans and animals (Ritz *et al.*, 2003) have shown that cell shrinkage signals the cleavage of glycogen, lysing of proteins, and a temporary halt to the formation of both glycogen and protein. This makes glucose and amino acids available for alternative metabolic pathways. Changes in cell size also theoretically mediate lipolysis, via the hormonal effects of insulin, glucagons, and catecholamines (Keller, Szinnai, Bilz, & Berneis, 2003). If these concepts are supported by future research, the dehydration that often occurs in hot, cold, and high-altitude environments (and the resultant cell shrinkage) may be viewed as an important aspect of metabolic regulation.

Dehydration (i.e. due to heat, cold, or high-altitude exposure) need not be severe to alter mental and physical performance (Maughan, 2003b). Mild dehydration (i.e. equivalent to 1–2% of body mass), for even a brief period, leads to (a) a reduction of the subjective perception of alertness and ability to concentrate, (b) an increase of self-reported tiredness, and (c) an increase of headache pain. Mild dehydration also impairs high-intensity endurance exercise performance (Armstrong, Costill, & Fink, 1985; Maughan, 2003b) and intermittent supramaximal running performance (Maxwell, Gardner, & Nimmo, 1999), although maximal muscle strength and power appear to be relatively unaffected (Maughan, 2003b; Watson *et al.*, 2005).

Nutrition for football in all environments

Three nutritional strategies, to optimize performance and minimize fatigue, are recommended for football players in any environment. First, at least 3 h before a contest and during the half-time of a football match, provide 100–300 g of carbohydrate (Williams & Serrasota, 2006). Ample dietary carbohydrate intake approximately doubles normal muscle glycogen reservoirs (Shephard, 1999) and can result in a 5–6% increase of the ability to perform multiple sprints after 45 min of simulated football (Bangsbo, Norregaard, & Thorsoe, 1992). When left without guidance, most players fail to ingest the necessary quantity and type of carbohydrates during the hours after exercise (Shephard, 1999). As long as a 70 kg person consumes 500–600 g of carbohydrate, glycogen resynthesis is similar whether eaten in two meals or several small meals (Costill *et al.*, 1981); both simple and complex

Table I. Effects of four environmental factors on metabolism and performance.

Environmental factors	Physiological outcomes of environmental stress[a]	Effects on human metabolism and performance[b]
Heat	• increased skin and core body temperature • increased cardiovascular strain[c] • increased sweating may lead to fluid-electrolyte deficit (i.e. sodium)	• increased anaerobic metabolism • increased plasma lactate accumulation • increased rate of glycogen depletion • reduced $\dot{V}O_{2max}$ • reduced endurance, strength, and power performance • increased perceived exertion • increased resting metabolic rate[d]
Cold	• decreased skin and core body temperature	• increased heat production (1 – 4 times resting metabolism) due to shivering • increased metabolism without shivering[e] • increased appetite • decreased utilization of free fatty acids • increased utilization of plasma glucose and muscle glycogen • increased plasma lactate concentration • increased diuresis • reduced maximal aerobic power ($\dot{V}O_{2max}$) • reduced endurance exercise performance • reduced muscular strength and power when muscle temperature decreases[f] • reduced memory and cognitive function
High altitude	• low barometric pressure results in arterial blood hypoxia (reduced partial pressure of oxygen) • increased resting ventilation • breathlessness during exercise or activities • reduced cardiac output (rest and exercise) • reduced oxygen saturation of haemoglobin and reduced oxygen delivery to tissues • increased incidence of sleep disturbance and sleep apnoea	• reduced maximal aerobic power ($\dot{V}O_{2max}$) • reduced endurance performance[g] • reduced reaction time and motor coordination • aerobic metabolism is unable to provide adequate energy; this is partially offset by increased anaerobic metabolism • increased carbohydrate utilization • increased plasma lactate accumulation • increased water loss via diuresis and respiratory water loss (acute); decreased muscle and fat mass (chronic)[h] • impaired mood and appetite • reduced cognitive performance (i.e. memory, decision making, calculations) • reduced vigour and increased fatigue • increased oxidative stress (i.e. free radical formation)[i]
Jet lag	• travel across time zones disrupts the coordination of environmental cues (i.e. light – dark cycle, social activities) with internal biological clocks • disrupted sleep cycle, sleep loss	• disturbed mood • increased daytime fatigue • reduced daytime alertness • disturbed gastrointestinal function • altered schedule of eating and drinking

Note: This table was compiled from the following references: Aerospace Medical Association (1996), Ahlers, Thomas, Schrot and Shurtleff (1994), Armstrong (2000), Committee on Military Nutrition Research (1996), Doubt (1991), Ferretti (1992), Fulco and Cymerman (1988), Galloway and Maughan (1997), Normand, Vargas, Bordachar, Benoit and Raynaud (1992), Sawka and Wenger (1988), Stephenson and Kolka (1988), Toner and McArdle (1988), Wenger (1988), Wilbur (2004), Young (1988), Youngstedt and Buxton (2003).

[a]8 – 14 days of acclimatization to heat, cold and altitude alters these effects favourably, in response to specific stresses imposed on the body.

[b]These effects increase as environmental stress increases.

[c]Due to simultaneous needs to supply blood flow to exercising muscle to support metabolism, and skin to dissipate internal heat.

[d]In a hot environment ($>35^{\circ}C$), resting energy expenditure increases up to 5%, due to the energetic cost of sweating, hyperventilation, circulatory strain, or the Q_{10} effect (Consolazio & Schnakenberg, 1977).

[e]Probably because of secretion of hormones (i.e. adrenal catecholamines, thyroxine).

[f]Muscle temperature may be normal or elevated, even in very cold air, depending on clothing insulation and exercise-induced heat production (Bergh & Ekblom, 1979; Blomstrand, Bergh, Essen-Gustavsson, & Ekblom, 1984).

[g]The results of studies regarding anaerobic performance are equivocal.

[h]Chronic body mass loss may be due to a combination of increased resting metabolic rate, increased activity level, decreased appetite and food intake.

[i]Due to the electron transport chain, hypoxia and ultraviolet radiation.

carbohydrates are effective (Coyle, 1995). Even when recovery periods are brief (i.e. 4 h), players can benefit from consuming carbohydrate (Burke, Coyle, & Maughan, 2003; Williams, 1995). Interestingly, one study demonstrated that a carbohydrate-protein solution (7.75% carbohydrate and 1.94% protein) enhanced continuous cycling endurance performance beyond that of a 7.75% carbohydrate solution alone (Ivy, Res, Sprague, & Widzer, 2003). Although the effects of a carbohydrate-protein solution on high-intensity, intermittent exercise are unknown, this project provides a testable hypothesis for future investigations.

Second, because low intramuscular glycogen becomes an important cause of fatigue as a game progresses, players should optimize muscle glycogen resynthesis after exercise (Bangsbo, 1991; Saltin, 1973) by consuming 8–11 g of carbohydrate per kilogram of body mass per day (Sherman, 1992). Third, ensure that total energy, carbohydrate, and protein levels are adequate, as provided by a well-balanced daily diet. After reviewing numerous studies, Shephard (1999) provided the following dietary recommendations for male football players: daily energy intake, $14-15\,\mathrm{MJ}\cdot\mathrm{day}^{-1}$ ($3346-3585\,\mathrm{kcal}\cdot\mathrm{day}^{-1}$); carbohydrate, $8\,\mathrm{g}\cdot\mathrm{kg\ body\ mass}^{-1}\cdot\mathrm{day}^{-1}$; protein, $1.5\,\mathrm{g}\cdot\mathrm{kg\ body\ mass}^{-1}\cdot\mathrm{day}^{-1}$. Further information regarding nutritional practices can be found in Williams and Serratosa (2006) and Burke, Loucks and Broad (2006).

Nutritional interventions for specific environments

Physiologists and dietitians have prescribed specific nutritional interventions and supplements that purportedly diminish the effects of environmental stressors (Table II). Although these recommendations have not been tested for high-intensity, intermittent exercise, they are supported by reports of dietary deficiencies among German and Dutch football teams (Tiedt, Grimm, & Unger, 1991; Van Erp-Baart *et al.*, 1989). The decision to utilize the recommendations in Table II may be difficult or even controversial because some health care professionals discourage the use of all ergogenic aids; most professionals suggest that they be used with caution and only after careful examination of safety, efficacy, potency, and legality (ACSM, 2000; FIFA, 2004). Therefore, column 3 of Table II provides guidance regarding the strength of scientific evidence for each.

Table III considers other nutritional supplements and strategies. Although not specifically recommended for heat, cold, high altitude, and jet lag, these may enhance performance or alter metabolism favourably. The strength of supporting scientific evidence is presented in column 3 of Table III.

Further complicating our understanding of nutritional interventions, female football players may experience subtle changes of exercise metabolism at different phases of the menstrual cycle, as shown by recent investigations that administered exogenous hormones. These data (D'Eon *et al.*, 2002) suggest that estrogen alone reduced total carbohydrate oxidation during exercise by decreasing the use of both blood glucose and glycogen. Administration of progesterone further reduced blood glucose use but increased glycogen utilization. These findings indicate that substrate utilization across the menstrual cycle is dependent on the relative changes of both estrogen and progesterone. However, their effects on the magnitude and direction of these changes, on mental and physical performance, or their interactions with environmental factors (Table I), are not known.

Heat exposure

High ambient temperatures ($>35^{\circ}\mathrm{C}$) increase the strain that an athlete's body experiences. This strain is observed as an increased core body temperature, decreased cardiac stroke volume, increased heart rate, and increased perceived exertion. Dehydration *per se* also increases cardiovascular strain, in an additive manner, and increases muscle glycogen utilization (i.e. versus a euhydrated state) (Shirreffs, 2005). Endurance exercise performance declines when the body water deficit reaches approximately 2–3% of body mass (Armstrong *et al.*, 1985; Cheuvront, Carter, & Sawka, 2003; Shirreffs, 2005). Regarding muscular strength, power, and sprint performance, authorities disagree on the exact level of dehydration that elicits a performance decrement; this threshold apparently occurs at a loss of 5–8% of body mass (Sawka & Pandolf, 1990; Watson *et al.*, 2005). Thus, a body water deficit of 1% or 2% during a football match in the heat is tolerable and ordinarily unavoidable (Maughan, Shirreffs, Merson, & Horswill, 2005).

Field observations of sweat losses and body temperatures during football matches and training sessions provide additional insights. These indicate that the mean sweat losses of elite footballers range from 1.06 to 2.65 litres (mean = 1.69 litres) during a 90 min practice session in cool air ($5^{\circ}\mathrm{C}$, 81% relative humidity), from 1.67 to 3.14 litres (mean = 2.91 litres) during a 90 min practice session in warm air ($33^{\circ}\mathrm{C}$, 20% relative humidity), and from 1.48 to 3.93 litres (mean = 2.32 litres) during Olympic qualifying matches ($26^{\circ}\mathrm{C}$, 78% relative humidity and $33^{\circ}\mathrm{C}$, 40% relative humidity) (Maughan *et al.*, 2005; Mustafa & Mahmoud, 1979; Shirreffs *et al.*, 2005). By consuming fluids during exercise and rest periods, these players replaced 25%, 45%, and 9–31% of

Table II. Recommended nutritional interventions that counteract environmental stressors, as they appear in publications. Most studies (column 4) did not involve football specifically.

Environmental factors	Nutritional interventions and associated effects on performance	Strength of scientific evidence[a]	References
Heat	• Water and carbohydrate-electrolyte replacement counteract the detrimental effects of dehydration	A	Armstrong and Maresh (1996b), Armstrong *et al.* (1997), Coyle (1991, 1995), Hawley *et al.* (1994), Shirreffs *et al.* (1996)
	• Sodium chloride supplementation offsets a whole-body sodium deficiency	D	
	• Post-exercise rehydration fluids should contain sodium; tvolume should equal 150% of the fluid deficit incurred during exercise	A	
Cold	• Tyrosine[b] reduces cognitive deficits, adverse moods, and performance impairments due to cold exposure	B	Ahlers *et al.* (1994), Baker-Fulco *et al.* (2001), Bandaret and Lieberman (1989), Berglund and Hemmingsson (1982), Doubt (1991), Fulco *et al.* (1989), Lieberman (1994), Lieberman and Shukitt-Hale (1996), Reynolds (1996), Schmidt *et al.* (2002)
	• Caffeine improves endurance performance at low (300 m), moderate (2900 m), and high (4300 m) altitudes	B	
	• Vitamins E, C, β-carotene, pantothenic acid, zinc, selenium, and other antioxidants reduce the oxidative stress of individuals with low initial antioxidant status	D	
High altitude[c]	• Tyrosine[b] reduces cognitive deficits, adverse moods, performance impairments, and the symptoms of altitude illness	B	Baker-Fulco *et al.* (2001), Bandaret and Lieberman (1989), Committee on Military Nutrition Research (1996), Fulco *et al.* (2005), Hoyt and Honig (1996), Lieberman (1994), Lieberman and Shukitt-Hale (1996), Simon-Schnass (1996), Wright, Klawitter, Iscru, Merola and Clanton (2005), Zuo and Clanton (2005)
	• Easily consumed liquid or solid carbohydrate foods maintain performance and macronutrient balance[de]	B	
	• Consume ample energy (MJ, kcal) each day to maintain body mass	B	
	• Ensure that iron status is normal and that the recommended daily allowance for iron is consumed	B	
	• Supplement diet with vitamins C, E, and other antioxidants if altitude exposure is prolonged	D	
Jet lag	• Caffeine temporarily reverses sleepiness and cognitive deficits due to sleep deprivation	B	Aerospace Medical Association (1996), Penetar (1994), Reilly, Atkinson and Waterhouse (1997c)
	• Alter, timing, size, and composition of meals to reduce the negative effects of jet lag	C	
All environments	• Consume a high-carbohydrate diet; maximize pre-exercise glycogen levels in muscle and liver[f]; enhances performance[g] during brief high-intensity, repeated-sprint, and endurance exercise[h]	A	Armstrong and Maresh (1996a), Bemben and Lamont (2005), Branch and Williams (2002), Coyle (1991), Fulco *et al.* (2005), Hawley & Burke (1997), Ivy (1994), Maughan (2000), Maughan (2002), Shepard (1999), Sherman (1992), Wagenmakers (1999)
	• Consume a fluid-electrolyte replacement beverage during training and competition[g]; provides carbohydrates; sodium supports extracellular (i.e. plasma) volume; flavouring encourages drinking and reduces dehydration	A	

(*continued*)

Table II. (*Continued*).

Environmental factors	Nutritional interventions and associated effects on performance	Strength of scientific evidence[a]	References
	• Creatine enhances anaerobic performance (maximal force or strength) for events lasting less than 4 min, with no ergogenic effect for endurance exercise	A	

[a]Modification of an evidentiary model (National Heart, Lung, & Blood Institute, 1998) designed to evaluate the strength of scientific evidence. *Level A*: randomized controlled trials (rich body of data) with a substantial number of well-designed studies, substantial number of participants, and consistent pattern of findings. *Level B*: randomized controlled trials (limited body of data) that include post-hoc field studies, subgroups, or meta-analyses; participant populations differ from the target population (i.e. animals vs. humans). *Level C*: evidence arises from uncontrolled/non-randomized trials, clinical observations, or case studies. *Level D*: expert judgement that is based on a synthesis of published evidence, panel consensus, clinical experience, and/or laboratory observations; used when guidance is needed but the published literature is lacking. *Level E*: studies suggest that this intervention may be appropriate for football, but the amount of scientific evidence (i.e. randomized controlled trials) is small.

[b]Increased catecholamine release may counteract various environmental stressors and tyrosine is a catecholamine precursor (i.e. agonist).

[c]Each effect may occur at a different altitude.

[d]Body weight and nitrogen balance are maintained when the energy requirement is met and the diet provides the recommended daily allowance for macronutrients (Butterfield *et al.*, 1992).

[e]Consumed as a fluid (10% carbohydrate solution; 14 g per serving for a male weighing 80 kg; 6 – 8 servings per trial) (Fulco *et al.*, 2005).

[f]The timing of replacement is important, with optimal muscle glycogen and protein synthesis occurring 1 – 3 h after exercise (Baker-Fulco *et al.*, 2001; Sherman, 1992).

[g]Endurance performance in a cold environment may not be improved by consumption of a carbohydrate-electrolyte replacement fluid (Galloway & Maughan, 1998; Galloway *et al.*, 2001).

[h]Endurance exercise that is continuous and more than 50 – 60 min at $>70\%$ $\dot{V}O_{2max}$.

sweat losses, respectively. This exemplifies voluntary dehydration, a well-known phenomenon that occurs among athletes, labourers, and military personnel. Independent of weather conditions, fluid replacement lags behind fluid lost as sweat by at least 50% (Burke & Hawley, 1997).

Physiological research provides useful fluid consumption guidelines for athletes, to ensure that they begin each morning well hydrated (Shirreffs, Armstrong, & Cheuvront, 2004). These guidelines arise from rehydration studies that evaluated several combinations of fluid volume and composition (Shirreffs, Taylor, Leiper, & Maughan, 1996). First, players should consume a volume of fluid equal to 150% of the sweat lost during exercise throughout the remainder of the day. Water retention will be greater if the fluid or food contains a moderately high sodium content (i.e. $60\,mmol \cdot l^{-1}$). After encountering a 2% body weight loss, these recommendations will result in normal hydration status within 6 h.

Regarding body temperature, Ekblom (1986) reported an average rectal temperature of 39.5°C in players at the end of a Swedish First Division football match. The corresponding average for players of a lower division was 39.1°C, reflecting a slower overall pace of play. Maughan and Lieper (1994) summarized similar data from six other publications, indicating that rectal temperatures reached 39.2 – 39.6°C at the end of 90 min games contested in environmental conditions ranging from 12 to 38°C.

Because football players may experience large sweat losses, especially when engaged in two training sessions per day, electrolyte losses should also be considered. A whole-body deficit of sodium chloride predisposes players to heat cramps and heat exhaustion (Armstrong, 2003). Two of the previously mentioned studies measured salt losses in the sweat of elite male footballers. The sweat sodium concentration averaged $42\,mmol \cdot l^{-1}$ in cool air (5°C) (Maughan *et al.*, 2005) and $30\,mmol \cdot l^{-1}$ in warm air (33°C) (Shirreffs *et al.*, 2005). These salt losses totalled 73 and 67 mmol per 90 min, respectively. Given the fact that competitive athletes consume an average of 231 mmol of sodium per day (Kies, 1995), and active college students consume 91 – 205 mmol of sodium per day (Armstrong *et al.*, 2005; Kies, 1995), it is unlikely that a whole-body sodium deficit will occur in most footballers. Indeed, Maughan, Leiper and Shirreffs (1996) compared post-exercise rehydration via a meal and a commercial fluid-electrolyte replacement beverage. Although the quantity of water was identical for both methods, a single meal provided considerably more electrolytes (63 mmol sodium, 21 mmol potassium) than the sports drink, and approximated the total salt losses calculated above. A similar conclusion can be made for potassium (data not shown).

Table III. Selected nutritional supplements. Although not recommended for football specifically, these may enhance football performance or alter metabolism favourably. The scientific studies in column 4 generally were conducted in non-stressful environments.

Nutritional supplement/ strategy	General effect on performance or metabolism (all environments)	Strength of scientific evidence[a]	References
Caffeine	Enhances performance during intense aerobic exercise, but not brief power events of less than 90 s. Improves perception of effort, alertness, wakefulness, vigilance, and mood. When consumed in moderation ($3-6\,mg \cdot kg$ body $mass^{-1}$), caffeine does not dehydrate the body	A	Armstrong (2002), Armstrong *et al.* (2005), Ivy (1994), Magkos and Kavouras (2005), Maughan (2002), Nehlig and Debry (1994), Penetar (1994), Spriet (2002)
Iron	When iron deficiency anaemia[b] exists, iron supplements are necessary to maintain exercise performance and health; the immune system is sensitive to iron deficiency	A	Burke *et al.* (2003, 2006), Committee on Military Nutrition Research (1999a), Gleeson and Bishop (2000)
Calcium	Important for healthy bones, especially in adolescent and female athletes; not known to affect exercise performance when whole-body balance is normal	A	Armstrong and Maresh (1996a), Burke *et al.* (2003, 2006)
Carbohydrate and protein-rich diet or solutions[d]	Encourages recovery (i.e. protein synthesis and muscle glycogen resynthesis) after strenuous exercise[c]. Carbohydrates and protein induce different endocrine responses (i.e. plasma insulin, glucagon, growth hormone); when consumed together, they encourage an anabolic state	B	Chandler, Byrne, Patterson and Ivy (1994), Earnest and Rudolph (2001), Ivy (1994), Ivy *et al.* (2003), Miller *et al.* (2002)
Choline	Improves endurance performance time in individuals whose plasma choline levels are reduced (i.e. due to prolonged exercise or environmental stress). When choline is available in near-physiologic concentrations, choline supplementation increases acetylcholine release from nerves, enhancing neurotransmission	C	Wurtman (1994), Zeisel (1994)
Tryptophan	The brain neurotransmitter serotonin, which theoretically is involved in central fatigue, may be modulated by diet or plasma levels of its precursor tryptophan, an amino acid; evidence for this effect is limited	E	Maughan (2002), Newsholme and Castell (2000), Wilson and Maughan (1992)
Vitamin C, vitamin E, selenium, and β-carotene	Although these antioxidants do not enhance performance, they may offer protection (to individuals who exercise strenuously) from intracellular free radical damage, optimize recovery of skeletal muscle, and enhance health in general	D	Committee on Military Nutrition Research (1999a), Gleeson and Bishop (2000), Kalman (2002)
High-fat diet	A few studies have indicated that several weeks of adaptation to a high-fat diet enhances endurance exercise performance, with or without phases of high carbohydrate intake. However, for athletes who participate in high-intensity exercise, there is little support for this approach	E	Coleman and Steen (2002), Coyle (1995), Maughan (2002), Williams (1995)
Glutamine	A glucose precursor that may encourage recovery after intense training, especially in support of the immune system	E	Coleman and Steen (2002), Earnest and Rudolph (2001), Gleeson and Bishop (2000), Wagenmakers (1999)
Sodium bicarbonate	Buffers intramuscular and blood pH, blunting the acidity produced during intense anaerobic exercise lasting 30 s to a few minutes. Effects on exercise performance are equivocal	E	Maughan (2002), Webster (2002)

(*continued*)

Table III. (*Continued*).

Nutritional supplement/ strategy	General effect on performance or metabolism (all environments)	Strength of scientific evidence[a]	References
D-Ribose	Observed to preserve body pools of ATP in rats; theoretically, this could maintain maximal functional capacity in humans, but controlled human studies indicate that ribose supplementation has no effect on anaerobic exercise capacity and maximal intermittent exercise performance	E	Brault and Terjung (1999), Coleman and Steen (2002), Kreider *et al.* (2003), Op't Eijnde *et al.* (2001), Tullson and Terjung (1991)
Branched-chain amino acids	May limit fatigue at the level of the central nervous system by increasing brain serotonin (i.e. a neurotransmitter that modulates central fatigue). Human performance studies are equivocal	E	Coleman and Steen (2002), Newsholme and Castell (2000), Wagenmakers (1999), Williams (1995)

[a]Modified from the evidentiary model described in Table II (National Heart, Lung, and Blood Institute, 1998).
[b]Iron deficiency anaemia is defined as a serum ferritin concentration of less than $12\,\mu g \cdot ml^{-1}$ in combination with a haemoglobin concentration of less than $120\,g \cdot l^{-1}$.
[c]Includes amino acid supplements mixed with carbohydrates.

The few players who have both a high sweat rate and a high sweat sodium concentration (i.e. >3 litres $\cdot h^{-1}$ and >60 mmol Na^+/L) should receive individualized nutritional guidance (i.e. sodium, potassium, and fluid intake), and should be monitored regularly by the sports medicine staff (Bergeron, 2003). Some authors recommend salt supplements (i.e. $8-10\,g \cdot day^{-1}$ by adding salt to food) for athletes with a history of heat illness, and for all individuals during the initial days of chronic heat exposure (Wenger, 1988).

Heat acclimatization is important for every athlete who competes in warm or hot ambient temperatures. This process requires 8–14 days of exposure to heat, and results in several physiological adaptations that make exercise in the heat easier to perform (Wenger, 1988). Three interesting acclimatization facts are seldom appreciated. First, dehydration reduces or nullifies the benefits of heat acclimatization, in a progressive manner, as dehydration becomes more severe (Cadarette, Sawka, Toner, & Pandolf, 1984). Second, heat acclimatization affects sweat glands by increasing the amount of sweat that is produced (Wenger, 1988). Although beneficial (i.e. it ensures wet skin and evaporative cooling), this adaptation also increases the water requirement of an athlete exercising in the heat. Both of these facts reinforce the goal of minimizing body water loss (see above). Third, in studies conducted among South African miners, ascorbic acid (vitamin C) supplements of $250-500\,mg \cdot day^{-1}$ were given during a 10 day ($4\,h \cdot day^{-1}$) laboratory heat acclimation protocol (Strydom, Kotze, Van der Walt, & Rogers, 1976). As a result, vitamin C reduced rectal temperature and total sweat loss. However, the miners in this study may have been deficient in vitamin C due to their poor dietary habits (Askew, 1995). Although it is unclear whether supplementation benefits individuals who are adequately nourished, this study suggests that adequate dietary vitamin C (and perhaps other antioxidants) is important for normal heat acclimatization to occur.

The foregoing paragraphs point to the primary nutritional needs in hot environments: fluid, carbohydrate, and electrolytes. In hot environments, therefore, footballers should ensure that muscle and liver glycogen are optimal by reducing the volume and intensity of training in the 48–72 h before an important contest and by consuming up to 10 g of carbohydrate per kilogram of body mass per day (Sherman & Wimer, 1991). Before, during, and after a contest, they should consume carbohydrates in a systematic way to maintain exercise metabolism and performance. Specific details are available in the review of this topic by Hawley, Dennis and Noakes (1994). There is no evidence to indicate an increased requirement for protein or fat during exposure to a hot environment (Committee on Military Nutrition Research, 1999b).

Contracting skeletal muscle produces more free radicals when muscle temperature exceeds 42°C, and high free radical production contributes to muscle fatigue (Zuo *et al.*, 2000). Similarly, studies of isolated rat livers, perfused at normal and elevated temperatures (Bowers *et al.*, 1984), demonstrated that leakage of transaminase enzymes did not occur until the perfusion temperature reached 42°C. At this temperature, structural integrity degraded and signs of membrane destabilization occurred; both are consistent with the liver damage that occurs with human and animal heat stroke (Hubbard & Armstrong, 1988). These observations suggest that (a) heat stress stimulates intracellular and extracellular superoxide production,

which may contribute to the physiological responses to severe exercise and hyperthermia, and (b) antioxidant intake may some day be shown to protect cells from the stress and damage that hyperthermia induces.

A glycerol-water solution has been shown to be an effective hyperhydrating agent at rest. Although some researchers have demonstrated that ingesting 1.0–1.5 g of glycerol per kg body mass, together with a large volume of water (e.g. 20 ml · kg body mass^{-1}), significantly increases temporary water retention and cycling time to fatigue, and decreases circulatory and thermal strain (Anderson, Cotter, Garnham, Casley, & Febbraio, 2001), others have observed no difference in physiological responses or performance (Shirreffs *et al.*, 2004). None of these studies involved high-intensity, intermittent exercise (i.e. similar to a football match), and several reported unwanted side-effects in a small percentage of participants (Latzka & Sawka, 2000). When considered collectively, these studies suggest that the efficacy of pre-exercise hyperhydration with glycerol is uncertain, especially if hydration is maintained during exercise (Latzka & Sawka, 2000; Shirreffs *et al.*, 2004). The present scientific literature does not support a recommendation for the use of glycerol in football.

Cold environments

Although not widely recognized by players and coaches, chronic exposure to very cold air ($<5^{\circ}$C) can lead to dehydration (i.e. 2–5% of body mass) due to cold-induced diuresis that is accompanied by reduced blood and plasma volumes. Sweating, respiratory water loss, reduced intake of fluids (Committee on Military Nutrition Research, 1996), and diminished thirst (Freund & Sawka, 1996) also contribute to this dehydration Respiratory water loss has been estimated as 0.9 litres · 24 h^{-1} in 0°C air and 1.0 litres · 24 h^{-1} in -20°C air. Sweat loss for moderate-to-heavy exercise has been estimated as 0.9–1.9 litres · 24 h^{-1} in air at 0°C and 0.4–1.9 litres · 24 h^{-1} in air at -20°C (Freund & Sawka, 1996). This dehydration may contribute to the changes of performance, appetite, and emotions that are observed when humans are exposed to very cold environments (see section headed ''Dehydration affects metabolism and performance'').

Although the optimal air temperature for endurance exercise is about 11°C (versus 4, 21, and 31°C) (Galloway & Maughan, 1997), it appears that the severity of dehydration and the nature of the exercise performed (i.e. mode, intensity, duration) determine whether physical performance will be affected by cold exposure. For example, a research group led by McConnel (McConnel, Stephens, & Canny, 1999) reported no effect of 1.9% dehydration on cycling exercise (45 min at 80% $\dot{V}O_{2max}$ + 15 min sprint) in a 21°C environment, whereas Cheuvront *et al.* (2003) observed a significant decline in performance (30 min at 50% $\dot{V}O_{2peak}$ + 30 min sprint) in a 20°C environment subsequent to 3% dehydration. Kenefick, Mahood, Hazzard, Quinn and Castellani (2004) incorporated a 4°C environment, brisk treadmill walking (60 min at 50% $\dot{V}O_{2peak}$), and 4% dehydration but observed no differences in performance, thermoregulatory responses, or cardiovascular strain versus a mild 25°C environment. Regarding muscular power, deep muscle temperature influences performance. When muscle temperature falls, power output declines by virtue of the effects of cold on the rate of adenosine triphosphate (ATP) hydrolysis and/or resynthesis (Ferretti, 1992).

Living outdoors in a very cold environment increases the resting energy requirement by 2–10% above that measured in a mild environment, largely due to muscular shivering. However, the utilization of fats and glucose as fuels differs during shivering and exercise (Tipton, Franks, Meneilly, & Mekjavic, 1997). This suggests that the metabolic substrates utilized by two football players may be quite different during a game in a cold/wet environment, depending on whether they are actively competing or playing a passive part in a game. Furthermore, exercise in cold air requires more energy than exercise performed in a thermoneutral environment. This has been attributed to the increased energy demands of thermoregulation, the preferential use of carbohydrate as a substrate (see below), and the restrictive effect of multilayered clothing (Armstrong, 2000; Committee on Military Nutrition Research, 1996; Gray, Consolazio, & Kark, 1951; Welch, Buskirk, & Iampietro, 1958). It is not known if footballers will expend more energy in cold versus neutral or mild environments, in that they live in climate-controlled buildings and experience one or two daily exposures to cold air (i.e. 1–3 h each).

Chronic exposure to cold air stimulates cold acclimatization. The patterns of response are unique, depending on the duration of exposure, insulation of the clothing, the amount of skin exposed to the air, and extent of core body cooling. Physiological adaptations include altered heat production, skin vasoconstriction (i.e. reduced flow in skin blood vessels), muscle blood flow, and a change of the preferred metabolic substrate. Once cold acclimatization has been established, an athlete uses less of the available muscle glycogen stores in response to a given exercise cold exposure (Shephard, 1993). This change is important because exercise metabolism is fuelled primarily by endogenous carbohydrate (i.e. muscle

glycogen) during continuous voluntary exercise in a cold environment (i.e. 84–103 min at 80% $\dot{V}O_{2max}$; 10°C) (Galloway & Maughan, 1998). Exogenous carbohydrate, supplied in fluids, has little or no influence on performance in the cold (Galloway & Maughan, 1998; Galloway, Wooten, Murphy, & Maughan, 2001). If ample carbohydrate is not consumed during periods of prolonged exercise, glycogen resynthesis during recovery will be reduced. Thus, if footballers consume a diet that is high in dietary fat and low in carbohydrate, they may experience decreased mental and physical performance in the cold because their pre-exercise muscle glycogen stores are low (Phinney, Bistrian, Wolfe, & Blackburn, 1983).

The requirement for protein is not increased by chronic exposure to a cold environment *per se* (Committee on Military Nutrition Research, 1999b), and the volitional preference for macronutrients (i.e. carbohydrate versus fat in the diet) does not change when humans freely select foods. For players who consume a well-balanced diet, are healthy, and live in a temperate climate, there is no evidence to suggest that vitamin and mineral supplementation will enhance mental or physical performance (Armstrong & Maresh, 1996b). However, if exposure to cold is prolonged (i.e. one 12 h period or several hours on repeated days) and severe, players may benefit from supplementing their diets with selected antioxidants (Table II) to counteract oxidative stress (Reynolds, 1996).

High-altitude environments

The 1986 Football World Cup was contested at various sites in Mexico, with elevations to 2607 m (8554 ft) above sea level. During acute exposure to such high altitudes, human appetite decreases and food preferences change (Westerterp-Plantenga *et al.*, 1999; Wilbur, 2004). However, chronic body mass loss can be avoided if adequate energy is consumed (Butterfield, 1996; Butterfield *et al.*, 1992).

Most studies have reported that the absolute and relative dietary carbohydrate intake increases, at the expense of fat and protein, with both acute and chronic altitude exposure (Butterfield, 1996; Gill & Pugh, 1964; Rose *et al.*, 1988). Measurements of arterial-venous substrate differences have also shown that altitude acclimatization decreases free fatty acid consumption in the legs, while glucose uptake increases, during rest and exercise (Roberts *et al.*, 1996). Endurance exercise performance is adversely affected when dietary composition is manipulated to decrease carbohydrate intake at altitude (Butterfield, 1996). Thus, adequate carbohydrate intake is an important nutritional objective for players who train and compete at high altitude.

Interestingly, women may not adjust their substrate oxidation in the same manner as men. Carbohydrate utilization decreases when women are exposed to high altitude acutely and chronically (Beidleman *et al.*, 2002; Braun *et al.*, 2000). Similarly, the blood glucose response to a meal is lower for women than for men at high altitude (Braun *et al.*, 1998). Although the mechanism responsible has yet to be identified, it is possible that women are more sensitive to insulin at high altitude, or that glucose output from the liver is suppressed in women (i.e. suggesting greater reliance on non-glucose fuel sources). Despite these unique responses, the strong influence that exercise intensity exerts on utilization of carbohydrate, as the limiting substrate during football training and competition, suggests that adequate carbohydrate intake is a sound nutritional objective for female players.

Total daily water turnover increases by 1 litre (i.e. from about 2.9 to 3.9 litres $\cdot$ day^{-1}) by moving from a sea-level training site to one at high altitude (Pugh, 1962). Because respiratory water losses average about 600 ml $\cdot$ day^{-1} (Westerterp, Kayser, Brouns, Herry, & Saris, 1992) and increase at very high altitudes (Simon-Schnass, 1996), and because sweat losses range from 1.3 to 2.8 litres per 90 min training session in a 24–29°C environment (Maughan, Merson, Broad, & Shirreffs, 2004), provision of adequate fluids remains a primary nutritional goal in football.

These two objectives agree well with the recommendation for a high-carbohydrate, low-fat, liquid supplement as the preferred ration for individuals who live in high-altitude environments (Cymerman, 1996). Also in concert with these two objectives, Hoyt and Honig (1996) recommended that a specialized diet be consumed during the first 3 days of high-altitude exposure, which is rich in carbohydrates and low in sodium chloride. This diet discourages water and salt retention, which is believed to be a key aetiological factor in acute mountain sickness, high-altitude cerebral oedema, and high-altitude pulmonary oedema (Committee on Military Nutrition Research, 1996). Thus, if ample water, salt-free fluid, tea, and coffee are consumed, physiologic natriuresis will be fostered. Unfortunately, exercise at high altitude favours retention of water and salt (Anand & Chandrashekhar, 1996). This suggests that athletes may be more susceptible to acute mountain sickness if they exercise during the first 3 days of exposure to high altitude; it also suggests that they should limit dietary intake of salt. At low or moderate altitudes, these precautions may not be necessary. For athletes who experience symptoms of acute mountain sickness, a bland, low-fat diet (i.e. crackers, bread, cookie bars, mashed potatoes, rice, cereals, pudding) is generally tolerated well when eaten in small portions every 2 h; dietary fat is not tolerated

well (Baker-Fulco, Patton, Montain, & Lieberman, 2001). After the initial days of altitude acclimatization, typical sea-level diet and exercise programmes may be resumed (Hoyt & Honig, 1996).

Although the daily total protein requirement is not increased (Committee on Military Nutrition Research, 1999b) in a high-altitude environment (> 5500 m), limited evidence indicates that adults oxidize leucine and excrete proteins at a slightly greater rate than at sea level (Srivastava & Kumar, 1992). This change of protein metabolism (Cymerman, 1996) suggests that protein and amino acids are utilized as energy sources, although the magnitude of this contribution may be small in comparison to carbohydrates and fats. Furthermore, animal research suggests that free radical production within skeletal muscle is increased in a high-altitude environment, probably because of hypoxia (Zuo & Clanton, 2005). Future research may determine that antioxidant supplements offset the stress of exercise at high altitude.

Two controlled, double-blind studies involving outdoor cross-country skiing trials (at 300 and 2900 m; Berglund & Hemmingsson, 1982) and cycling performance (79 – 85% of altitude-specific $\dot{V}O_{2max}$) within a high-altitude field laboratory (2 week residence at 4300 m; Fulco *et al.*, 1989) found that submaximal exercise was maintained significantly longer after consuming caffeine (6 mg and 4 mg · kg body mass^{-1}, respectively). The mechanism for this effect is unknown, but theoretically may involve (a) increased lipid mobilization and utilization, (b) stimulation of the central nervous system resulting in altered perception of effort, (c) increased cardiac output, (d) enhanced motor unit recruitment, (e) decreased metabolic products that produce fatigue, (f) altered ion movements (Na^+, K^+, Ca^{2+}) into and within skeletal muscle, (g) an increased sensitivity to the effects of caffeine at altitude, or (h) altered ventilatory characteristics that influence oxygen delivery (Berglund & Hemmingsson, 1982; Spriet, 2002). The measurements recorded during the cycling performance study (Fulco *et al.*, 1989) ruled out mechanisms (a), (b), and (c) above. However, tidal volume (i.e. the volume of air inhaled at each breath during normal breathing), not breathing frequency, increased (versus placebo) during the caffeine experiments at altitude but not at sea level. This paradox was probably due to the small advantage of increased tidal volume at sea level and the large advantage at an elevation of 4300 m (i.e. greater oxygen saturation of haemoglobin and greater oxygen delivery to skeletal muscles). Interestingly, repeated tests indicated that the acute improvement in performance (54% after 1 h exposure; 22.0 to 35.0 min) decreased after chronic exposure (24% after 2 weeks; 30.8 to 38.5 min), in concert with a decrease of the magnitude of the caffeine-stimulated increase in tidal volume (Fulco *et al.*, 1989).

Jet lag: Transmeridian travel

Regular biological rhythms have been observed in animals, plants, and unicellular organisms. These rhythms are expressed as oscillations in physiological systems with durations that range from minutes to months. Circadian rhythms last approximately 24 h (derived from the Latin phrase *circa diem*, ''about a day'') and are synchronized to the Earth's light – dark cycle, social interactions, and various other environmental factors. The following physiological processes exhibit circadian rhythms: breathing, heart rate, body temperature, oxygen consumption, blood plasma volume, plasma protein concentration, sweat rate, flexibility, grip strength, muscular endurance, physical work capacity, neuromuscular coordination, reaction time, growth hormone, and cortisol (Luce, 1970; Winget, DeRoshia, & Holley, 1985). These and other rhythms are regulated by the hypothalamus in coordination with the brain neurotransmitter serotonin and the pineal gland hormone melatonin (Reilly, Atkinson, & Budgett, 1997b).

Of great relevance to football, circadian rhythms exist for all body systems that respond to exercise training, including metabolism, central nervous system arousal, circulation, body temperature, muscular performance, and endocrine function. Systematic studies of these sport-significant rhythms have shown that organ function and performance generally peak at similar times each day (Winget *et al.*, 1985). For example, simple measurements recorded throughout the day indicate that grip strength, flexibility, and exercise tolerance are greatest between 14.00 and 18.00 h. Sport-specific mental factors (i.e. vigour, mood, speed of psychomotor responses) peak between 12.00 and 15.00 h each day.

Because circadian rhythms are intimately linked to the light – dark cycle that results from the Earth's rotation, it may be possible to desynchronize the body's rhythms, reduce organ function, and decrease performance by altering an athlete's normal light – dark cycle and daily schedules. Although other environmental stressors may also disrupt the body's normal rhythms, crossing time zones (jet lag) is the most common means by which this occurs. Jet lag reflects a temporary desynchronization of the traveller's ''biological time'' based upon the point of departure and the local time (i.e. light – dark cycle) at the destination. The symptoms of jet lag include periodic tiredness during the day, disturbed concentration, increased irritability, loss of vigour, and irregular sleep at night (Reilly *et al.*, 1997b). The number of time zones crossed, cumulative sleep loss,

and the intensity of environmental cues are the most important modulators of the severity of jet-lag symptoms in humans (Aerospace Medical Association, 1996).

Although at least one research team believes that jet lag does not affect athletic performance (Youngstedt & Buxton, 2003), British Olympic squad members exhibited impairments of several performance measures over 5 days, following travel across five time zones from London to Tallahassee, Florida (Reilly *et al.*, 1997a). American football players experienced similar effects due to jet lag – when travelling eastward, performance was suppressed more than when travelling westward (Jehue *et al.*, 1993); this directional tendency was not observed in other studies with similar experimental designs (Youngstedt & Buxton, 2003).

Because food components affect the central nervous system in various ways, nutritional interventions have been proposed to counteract the effects of jet lag. Reilly *et al.* (1997b) described several dietary strategies involving meal timing and macronutrient composition. They concluded that the scientific literature does not allow inferences to be formed regarding these practices. The Aerospace Medical Association (1996) advises that small meals before and during flights are better tolerated than large meals, and that caffeine and physical activity may be used strategically at the destination to help control day sleepiness.

Sleep loss

Jet lag often results in sleep loss. Two human studies have reported that sleep loss mildly reduces the body's ability to regulate core temperature (Kolka & Stephenson, 1988; Sawka *et al.*, 1984). Both investigations involved cross-over experimental designs in which participants were tested after periods of normal sleep and sleep loss. The latter experiments demonstrated a loss of thermosensitivity (i.e. effector response per degree rise of oesophageal temperature) of forearm sweating and blood flow, without a change of the hypothalamic threshold for the onset of these thermoregulatory responses. These findings suggest that heat storage may be slightly greater, for a specified amount of exercise, after sleep loss. The effects of jet lag *per se* on thermoregulation have not been studied in footballers.

The amount and type of food may affect the duration of subsequent sleep (Pozos, Roberts, Hackney, & Feith, 1996) by increasing the incidence of indigestion, pattern of food intake, and subjective responses to food (Waterhouse *et al.*, 2005). Also, several animal studies have demonstrated that a high-protein meal triggers the release of somatostatin, which increases rapid eye movement (REM) sleep (i.e. important for a restful night). A meal rich in carbohydrates triggers the release of insulin, which increases the duration of non-REM sleep. Interestingly, these findings contradict a widely publicised strategy that links diet and jet lag; it recommends high-protein meals for breakfast (supposedly to provide substrate for catecholamines) and high-carbohydrate meals for dinner (to furnish substrate for serotonin and hence promote sleep) (Reilly *et al.*, 1997a). Again, the available literature does not allow conclusive statements for or against these strategies.

One non-nutritional strategy to offset the negative effects of jet lag, involving altering the time of football training sessions for a few days before departure to reflect the time of competition in the other time zone, was found to be beneficial (Jehue *et al.*, 1993). Other non-nutritional techniques have been used, with varying success, including mild exercise, bright light therapy, napping, and oral melatonin supplements (Arendt, Aldhous, English, Marks, & Ardent, 1987; Reilly *et al.*, 1997b). Although none of these seems to be effective for all persons in all circumstances, numerous studies support the judicious use of oral melatonin to overcome the negative consequences of jet lag (Cardinali *et al.*, 2002). Reductions in jet-lag symptoms also have been reported when oral melatonin was combined with slow-release caffeine (Piérard *et al.*, 2001) or an altered light–dark schedule (Cardinali *et al.*, 2002). The interactions between jet lag, melatonin, and exercise have not been studied in a sample of athletes.

Counteracting multiple concurrent stressors

Although environmental factors exert unique physiological effects (Table I), the similarities of cold and high-altitude environments (i.e. low ambient temperatures, initial diuresis, increased energy requirements for work and exercise, carbohydrate is tolerated well) (Askew, 1996) suggest that common elements may exist in the responses of the central nervous system to these stressors. Thus, it may be beneficial to seek a single nutritional strategy that serves as an intervention for the adverse effects of two or more environments.

During stressful situations, highly active neurons may require additional precursor so that neurotransmitter synthesis can keep pace with the increased amount of neurotransmitter being released (Bandaret & Lieberman, 1989). Theoretically, the behavioural deficits caused by acute environmental stress (i.e. influencing alertness, anxiety, motor activity) may be reversed by provision of neurotransmitter precursors. Tyrosine, a large amino acid found in dietary proteins, is a precursor of catecholamines (i.e. noradrenaline,

dopamine, and adrenaline). The provision of oral tyrosine in capsule form is known to reverse many adverse effects that are produced by exposure to cold and hypoxia (15°C and 4200–4500 m). The positive effects include improved symptoms (i.e. headache, coldness, distress, fatigue, sleepiness, discomfort), reduced number of adverse emotions (i.e. confusion, unhappiness, hostility, tension), and improved cognitive performance (i.e. pattern recognition, vigilance, choice reaction time) (Bandaret & Lieberman, 1989). Several animal research studies support these findings (Lieberman & Shukitt-Hale, 1996).

Immune function, oxidative stress

Many studies have shown that strenuous exercise suppresses immune function (Pedersen & Bruunsgaard, 1995). Considering Table I and the high-intensity intermittent nature of football, immune system function may be compromised when teams play in stressful environments. This concept is supported by observations of Belgian First Division football club players, who exhibited depressed neutrophil function throughout a season (Bury, Marechal, Mahieu, & Pirnay, 1998). Limited evidence from human and animal studies (Committee on Military Nutrition Research, 1999b; Gleeson & Bishop, 2000) suggests that the following nutrients play a role in the optimal function of the immune system during periods of stress: protein; the minerals iron, zinc, copper, and selenium; vitamins A, C, E, B_6, and B_{12}; the amino acids glutamine and arginine; carbohydrates; and the polyunsaturated fatty acids. Even a mild deficiency of a single nutrient or trace element (i.e. iron, selenium, copper) can result in an altered immune response (Chandra, 1997; Gleeson & Bishop, 2000). Furthermore, it is widely accepted that an inadequate intake of protein impairs host immunity with particularly detrimental effects on the T-cell system, resulting in an increased incidence of opportunistic infections (Gleeson & Bishop, 2000). These observations suggest that future research should focus on the relationship between nutrition and immune function.

Provided that energy intake is adequate, there is no evidence that nutritional supplements improve innate antioxidant protection beyond normal/optimal levels (Butterfield, 1999). However, research suggests that a cold, moderate-altitude environment (i.e. 14–24 days of field training; mean low temperature of −6.9°C; 2546–3048 m altitude) increases oxidative stress, as indicated by six biochemical markers (Chao, Askew, Roberts, Wood, & Perkins, 1999). Authorities believe that this stress may be ameliorated in individuals with low initial antioxidant status, by an antioxidant mixture (i.e. vitamins C and E, selenium, β-carotene) that is consumed as a dietary supplement (Committee on Military Nutrition Research, 1999a; Gleeson & Bishop, 2000; Kalman, 2002; Schmidt *et al.*, 2002). Also, it is reasonable to hypothesize that a deficiency of antioxidants (a) compromises immune function or (b) reduces defences against oxidative stress when intense, intermittent exercise is coupled with environmental stress (Table I). Utilizing this rationale, authors recommend that antioxidants be provided as dietary supplements prophylactically (Committee on Military Nutrition Research, 1999b; Schmidt *et al.*, 2002; Simon-Schnass, 1992) because there appears to be little harm in doing so. However, it is important to recognize that excessive amounts of specific nutrients (e.g. iron, zinc, polyunsaturated fatty acids) may suppress immune function (Gleeson & Bishop, 2000).

Summary

Football performance depends upon many physiological, psychological, tactical, and technical factors. This review focused on the effects of four environmental factors on metabolism, whole-body nutrient balance, and physiological function. The principle that nutritional interventions offset environmentally induced performance decrements (Table II) serves as the foundation of this paper. Pharmacological interventions and banned substances were not considered. In all stressful environments, football players will find these interventions useful: a high intake of carbohydrates, fluid-electrolyte replacement of sweat losses, and creatine. In hot environments, it is important to replace water and sodium deficits. In cold ambient conditions, caffeine and tyrosine may enhance performance. At high altitudes, evidence supports emphasizing total energy intake, iron and tyrosine consumption. When athletes experience jet lag, caffeine consumption and adjustments of meal size/composition may be helpful. Several other nutritional strategies (Table III) may offset performance deficits but cannot be recommended presently because of limited scientific evidence. Because relatively few of the aforementioned published investigations involved footballers, future research regarding any of these strategies and supplements is encouraged.

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