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Innovations in athletic preparation: Role of substrate availability to modify training adaptation and performance

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Abstract
World records for athletic events continue to improve and in the search for superior methods to gain a competitive edge, coaches and athletes are constantly searching for the latest "magic bullet". Although it is assumed that optimal adaptation to the demands of repeated training sessions requires a diet that can sustain muscle energy reserves, this premise does not consider the unsolved longstanding question of whether it is a lack or a surplus of a substrate that triggers the training adaptation. As such, recent scientific enquiry has re-focused attention on the role of substrate availability before, during, and after training to amplify the training adaptation. There has also been a resurgence of interest in the potential for protein ingestion to improve performance and/or promote training-induced adaptations in skeletal muscle. Altitude training (real or simulated) is now an accepted part of competition preparation for many athletic events, and such interventions attract their own nutritional issues. These and other diet–training interactions with the potential to alter training adaptation and performance are discussed.

Keywords: *Branch-chain amino acids, AMP-activated protein kinase, endurance training, protein, resistance training, training adaptation*

Modification of the training stimulus–response by dietary manipulation: A molecular perspective

The acute metabolic responses associated with a single exercise bout and the subsequent training-induced adaptations have been reviewed previously (Coffey & Hawley, 2006; Hawley, 2002a; Hawley & Spargo, 2007; Hawley, Tipton, & Millard-Stafford, 2006; Stepto, Martin, Fallon, & Hawley, 2001). Of interest is the extent to which acutely or chronically altering substrate availability might modify the training stimulus. Changes in dietary intake that alter the concentration of blood-borne substrates and hormones have the potential to cause large perturbations in the macronutrient storage profile of skeletal muscle and other insulin-sensitive tissues. Accordingly, nutrient status exerts profound effects on resting energy metabolism and subsequent fuel use during training and competition (Arkinstall *et al.*, 2004a; Burke *et al.*, 2000; Hawley *et al.*, 2000; Stepto *et al.*, 2002), as well as on many of the acute regulatory processes underlying gene expression

(Arkinstall, Tunstall, Cameron-Smith, & Hawley, 2004b; Cameron-Smith *et al.*, 2003; Churchley *et al.*, 2007; Creer *et al.*, 2005) and cell signalling (Wojtaszewski *et al.*, 2003).

When a cellular/molecular view on training is taken, any exercise- or diet-induced adaptation is a consequence of accumulation of specific proteins (Hansen *et al.*, 2005). As such, the altered gene expression that allows for these changes in protein concentration becomes pivotal to the subsequent training adaptation. Many studies have reported transient changes in mRNA levels of both myogenic (Churchley *et al.*, 2007; Coffey *et al.*, 2006; Willoughby & Nelson, 2002; Yang, Creer, Jemiolo, & Trappe, 2005) and metabolic genes (Cluberton, McGee, Murphy, & Hargreaves, 2005; Koval *et al.*, 1998; Kraniou, Cameron-Smith, Misso, Collier, & Hargreaves, 2000; Pilegaard, Ordway, Saltin, & Neufer, 2000; Pilegaard *et al.*, 2002; Pilegaard *et al.*, 2003; Yang *et al.*, 2005) after an acute bout of exercise in humans. Of note is evidence to suggest that commencing selective exercise sessions with low muscle glycogen content enhances the transcription

rate of several genes involved in the training adaptation (Febbraio et al., 2003; Pilegaard et al., 2002). This information underpins the recent hypothesis that a "cycling" of muscle glycogen stores may be desirable to optimize the training response/ adaptation (Chakravarthy & Booth, 2004). Although it has generally been assumed that the optimal adaptation to the demands of repeated training sessions requires a diet that can sustain muscle energy reserves (Coyle, 2000), this premise does not consider the unsolved longstanding question of whether it is a lack or a surplus of a substrate that triggers the training adaptation.

Carbohydrate availability and the training response: Train-low, compete-high?

The majority of evidence supports the contention that a high carbohydrate intake during training promotes a superior training response (Hawley et al., 2006). However, recent data pertaining to nutrient–gene interactions and the acute responses of several of the cellular signalling pathways that promote muscle adaptations to training suggest that, under certain conditions, a lack of muscle substrate may be desirable for promoting the training response (Hansen et al., 2005). Accordingly, it has been proposed that training in the face of low muscle glycogen stores will improve the subsequent training adaptation (Febbraio et al., 2003). To test this hypothesis, Hansen et al. (2005) had untrained males complete an intensive training programme of leg knee extensor exercise 5 days a week for 10 weeks. Each of the participants' legs was trained according to a different schedule, but the total amount of work undertaken by each leg was the same. Participants trained one leg twice a day, every second day (LOW), whereas the contralateral leg was trained daily (HIGH). On the first day of each 5-day training cycle, both legs trained simultaneously for 1 h at 75% of (one-leg) maximal power output. Following 2 h of recovery, the LOW leg trained again for a further 1 h at 75% of maximal power output. On the second day, only the HIGH leg trained. Muscle biopsies were taken from both legs before and after 5 and 10 weeks of training. Submaximal and maximal exercise testing was performed before and after training. Resting muscle glycogen content before training was similar for both conditions but was only increased for the LOW condition after training. The maximal activities of several metabolic enzymes increased in both legs after training, although the magnitude of increase was generally greater in the LOW than HIGH condition. Exercise performance (time to exhaustion at 90% of post-training maximal power output) was similar for both legs before training but the magnitude of increase after training

was twice as great for the LOW than the HIGH condition. These results demonstrate that under specific experimental conditions, the training adaptation may be augmented by a lack of substrate availability. Thus it is reasonable to suggest that there may be some benefit in implementing diets and training programmes that intentionally deplete muscle fuel stores, at least in the short term, as was originally proposed by Hawley and Hopkins (1995). However, coaches and athletes should exercise caution when drawing practical consequences from the results of that study with regard to training current practices. First, the participants in the investigation of Hansen et al. (2005) were untrained: it is not currently known whether chronic perturbations in glycogen stores translate into an improved training adaptation in well-trained athletes. Second, the training sessions undertaken by the participants in that study were "clamped" at a fixed (submaximal) intensity for the duration of the training programme: competitive athletes typically periodize training programmes to incorporate a "hard–easy" pattern to the overall organization of training, as well as progressive overload (Hawley, 2000). Finally, the mode of training (one-legged kicking) and the exercise "performance" task bears little resemblance to the training regimens undertaken by most competitive athletes. Notwithstanding these limitations, the results are intriguing and clearly there is urgent need for further investigations into the effect of muscle glycogen status on training adaptation using trained athletes and more appropriate training and testing regimens.

While the effects of chronically altering glycogen availability on training adaptation and performance are unresolved, there is strong evidence that muscle glycogen content is a potent modulator of several intracellular signalling proteins that are thought to play an obligatory role in adapting skeletal muscle to repeated exercise bouts (for a review, see Hawley & Zierath, 2004). One of these signalling proteins is the $5'$-AMP-activated protein kinase (AMPK), which is turned on by cellular stresses that deplete ATP (and consequently elevate AMP) either by accelerating ATP consumption (e.g. muscle contraction) or by inhibiting ATP production (e.g. hypoxia, ischaemia). Aside from manipulating muscle glycogen concentrations, another way to alter carbohydrate availability during exercise is to ingest glucose or other sugars. Most evidence strongly supports the notion that carbohydrate (CHO) feeding is ergogenic during athletic events lasting ≥ 1 h, which has led sports nutrition organizations to recommend consuming $30 – 60$ g $CHO \cdot h^{-1}$ to help optimize performance (Burke, 2003). However, carbohydrate ingestion during and after training could inhibit some of the chronic adaptations to training (Åkerstrom, Wotjaszewski, Plomgaard, & Pedersen, 2005;

Febbraio *et al.*, 2003). To test this hypothesis, Åkerstrom *et al.* (2005) determined the effects of glucose ingestion (or placebo) in untrained individuals on substrate metabolism, training responses, and performance during 10 weeks of supervised endurance training (2 h a day, 5 days a week). There were large training-induced improvements in performance for both groups. However, increasing glucose availability during training did not alter patterns of substrate utilization or a variety of markers of training response in muscle.

In contrast to the lack of effect on training adaptation when carbohydrate was ingested during a prolonged training block, Åkerstrom *et al.* (2006) reported that oral glucose ingestion during a single bout of endurance exercise (one-legged kicking) induced an attenuation of the exercise-stimulated muscle AMPK $\alpha2$ activity. However, others (Lee-Young *et al.*, 2006) have reported that carbohydrate ingestion does not alter skeletal muscle AMPK signalling during prolonged (2 h) cycling exercise. While a key role of AMPK in inducing metabolic adaptations of skeletal muscle to exercise -training has been hypothesized (Aschenbach *et al.*, 2004; Coffey & Hawley, 2006; Jorgensen, Richter, & Wojtaszewski 2006; Reznick & Shulman, 2006; Scarpulla, 2002), few studies at present have provided conclusive results to support this contention. Further investigations that manipulate muscle glycogen content and glucose availability in trained athletes during periods of intense training, and determine the effects of training adaptation and performance outcomes, are urgently needed.

Fat availability and the training response

The effects of acute and chronic fat supplementation on metabolism and exercise performance have been reviewed elsewhere (Burke & Hawley, 2002, 2006; Hawley, 2002b; Hawley, Brouns, & Jeukendrup, 1998). Stepto *et al.* (2002) found that well-trained cyclists could undertake intense interval training during short-term (5 days) exposure to a high fat diet. Indeed, performing such training in the face of a high fat diet was associated with rates of fat oxidation that are among the highest ever reported. However, compared with an isoenergetic high carbohydrate diet, training on the high fat diet was associated with increased ratings of perceived exertion. In that study (Stepto *et al.*, 2002), exercise intensity was "clamped" and it is unlikely that athletes on the high fat diet would have selected to train as intensely given the choice.

Increasing fat availability by acute feeding, intra-lipid infusion or chronic diet manipulation (i.e. "fat adaptation") results in large shifts in substrate metabolism in favour of lipid oxidation, with a concomitant "sparing" of muscle glycogen (Hawley, 2002b; Hawley *et al.*, 1998). Even when carbohydrate availability is increased, by the restoration of muscle and liver glycogen stores and the provision of exogenous carbohydrate during exercise, the enhanced capacity for lipid oxidation following a chronic high fat diet persists (Burke *et al.*, 2002). Yet despite creating metabolic conditions that should, in theory, enhance endurance capacity, performance of prolonged (2–5 h) continuous exercise (Burke *et al.*, 2000; Carey *et al.*, 2001) or submaximal exercise that includes maximal intermittent sprints (Havemann *et al.*, 2006) is not improved by fat-adaptation strategies. This is probably because the muscle glycogen "sparing" observed in studies of fat adaptation (Burke *et al.*, 2000; Carey *et al.*, 2001) is more likely to be an impairment of glycogenolysis due to a down-regulation of the multi-enzyme complex pyruvate dehydroegenase (Stellingwerff *et al.*, 2006). Such conditions would not be favourable for athletes whose training requires repeated bouts of intermittent, maximal sprinting or lifting.

Effect of protein ingestion during exercise on endurance performance

Several studies have determined whether the addition of small amounts of protein (typically 1.5–2% whey protein) to a carbohydrate-containing sports drink can enhance performance to a greater extent than ingestion of carbohydrate alone (Ivy, Res, Sprague, & Widzer, 2003; Romano-Ely, Todd, Saunders, & Laurent, 2006; Saunders, Kane, & Todd, 2004; van Essen & Gibala, 2006). Both Ivy *et al.* (2003) and Saunders *et al.* (2004) reported that co-ingestion of protein with a sports drink improved endurance capacity (time to fatigue at 75–85% peak oxygen uptake, $\dot{V}O_{2peak}$) by ~30% compared with ingestion of the sports drink alone. Although these findings are intriguing, any recommendations based on the results from these investigations are muted because of issues with research design and methodology. First, the rate of carbohydrate given in the sports drink in the studies by Ivy *et al.* (2003) and Saunders *et al.* (2004) was less than what is recommended to maximize endurance performance (Jeukendrup, 2004). In addition, the "performance" test employed in these studies (Ivy *et al.*, 2003; Saunders *et al.*, 2004) bears little resemblance to typical athletic competition where the goal of an athlete is to compete a pre-determined distance as fast as possible. While this methodological limitation is not uncommon, studies that seek to evaluate the potential ergogenic effects of nutritional interventions should ideally strive to simulate as closely as possible the normal practices of competitive athletes.

In contrast to the results of Ivy et al. (2003) and Saunders et al. (2004), others have reported little or no benefit to the addition of protein to a carbohydrate-containing sports drink. Van Essen and Gibala (2006) had trained participants ingest a carbohydrate solution at a rate sufficient to deliver $60 \text{ g} \cdot \text{h}^{-1}$, and perform an exercise test that closely simulated "real life" race conditions. The participants completed an 80-km cycling time-trial on three occasions and ingested drinks that contained 6% carbohydrate, 6% carbohydrate + 2% protein, or a sweetened placebo at a rate of 1 litre $\cdot \text{h}^{-1}$. The rates of carbohydrate ingestion in the study of van Essen and Gibala (2006) were 25–40% higher than those used by Ivy et al. (2003) and Saunders et al. (2004). Performance times were identical during the carbohydrate-only and carbohydrate plus protein trials, which was significantly faster (4%) than in the placebo trial. Consistent with these results, Romano-Ely et al. (2006) reported no effect on endurance time to fatigue when a drink that delivered $60 \text{ g CHO} \cdot \text{h}^{-1}$ was compared with an isoenergetic protein–carbohydrate drink.

In summary, some studies report that adding protein to sports drinks extends endurance time to fatigue when suboptimal amounts of carbohydrate are ingested and glucose availability during exercise is compromised. However, exercise performance is not enhanced when athletes are permitted to ingest sufficient amounts of carbohydrate and perform exercise tasks that closely mimic real life athletic competition. It is important to note that, aside from simply providing more energy, there is no established mechanism by which protein ingestion during exercise could potentially improve endurance performance. However, protein ingestion during prolonged exercise may attenuate whole-body protein degradation (Koopman et al., 2004) and reduce markers of muscle damage (Romano-Ely et al., 2006; Saunders et al., 2004), which could enhance skeletal muscle recovery following exercise and promote training-induced adaptations (Hawley, Hargreaves, & Zierath, 2006).

Effect of protein ingestion during recovery from endurance exercise on skeletal muscle adaptation and performance

Nutrition during the immediate post-exercise period may benefit an athlete in several ways, including the repair and synthesis of muscle proteins and a more rapid rate of synthesis of muscle glycogen. Skeletal muscle protein turnover is increased after an acute bout of endurance exercise and nutrition can modify the potential for net loss of accretion of skeletal muscle protein by affecting rates of protein synthesis and breakdown (Rodriguez, Vislocky, & Gaine,

2007). While most studies have focused on the nutritional manipulation of protein turnover after resistance exercise (Phillips, Hartman, & Wilkinson, 2005), Levenhagen et al. (2002) reported that ingesting protein with carbohydrate during recovery facilitated leg uptake of amino acids and promoted greater net protein accretion. These authors also suggested that the availability of amino acids was more important than the availability of energy per se for post-exercise repair and synthesis of muscle proteins.

A more contentious issue is whether co-ingestion of protein with carbohydrate enhances muscle glycogen synthesis during the first few hours of recovery from prolonged exercise. In our opinion, most evidence suggests that feeding a high amount of carbohydrate at frequent intervals (e.g. $\geq 1.2 \text{ g CHO} \cdot \text{kg body mass}^{-1} \cdot \text{h}^{-1}$) negates the benefits of added protein. Consistent with this interpretation, a recent review (Burke, Kiens, & Ivy, 2004) concluded that "feeding a high amount of carbohydrate at frequent intervals negates the benefits of added protein... [but] co-ingestion of protein with carbohydrate will increase the efficiency of muscle glycogen storage when the amount of carbohydrate ingested is below the threshold for maximal glycogen synthesis". Thus, similar to the effects on endurance capacity (see above), any beneficial effect of ingesting protein with carbohydrate on glycogen storage may simply be due to higher energy intake per se rather than any known or proven physiological mechanism.

A more practical concern for athletes is whether ingesting protein with carbohydrate during recovery affects subsequent exercise performance. Williams and colleagues (Williams, Raven, Fogt, & Ivy, 2003) reported that, compared with a carbohydrate sports drink, ingesting a carbohydrate–protein drink during recovery from prolonged exercise improved time to exhaustion during a subsequent bout of cycling at 85% $\dot{V}O_{2\text{peak}}$. However, the carbohydrate–protein drink provided almost three times as much energy as the sports drink and thus the improved endurance capacity was likely due to the higher energy intake. In contrast, two recent studies with direct relevance to runners reported no difference in exercise performance after ingestion of a carbohydrate–protein drink versus an isoenergetic carbohydrate beverage (Betts et al., 2005; Millard-Stafford et al., 2005). Betts et al. (2005) examined endurance running capacity at 85% $\dot{V}O_{2\text{peak}}$ 4 h after a 90-min treadmill run at 70% $\dot{V}O_{2\text{peak}}$. In two separate studies, a 9.3% carbohydrate solution or the same solution supplemented with 1.5% protein was used to provide carbohydrate at a rate of 0.8 or $1.2 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ throughout recovery. In both studies, there was no difference between the carbohydrate and

carbohydrate plus protein drinks on run times to fatigue. The results of Betts *et al.* (2005) were supported by Millard-Stafford *et al.* (2005), who found no effect of adding protein to a carbohydrate drink ingested during recovery on subsequent 5-km time-trial performance.

In summary, there is no compelling evidence to suggest that consuming protein with carbohydrate during recovery has a positive effect on subsequent running performance. However, given that a relatively small amount of protein has been shown to promote muscle protein net balance after strenuous endurance exercise (Levenhagen *et al.*, 2002), it seems prudent for athletes to ingest protein with ample carbohydrate as part of their recovery nutrition strategy. No studies have examined whether manipulating protein and carbohydrate intake during recovery between endurance training sessions influences the adaptive response to chronic training, and future work in this area is warranted.

Effect of protein ingestion during recovery from resistance exercise on skeletal muscle adaptation and performance

Many sprinters, jumpers, and throwers engage in heavy resistance training to increase lean body mass and/or strength and facilitate performance in their particular event. As reviewed elsewhere in this issue, the results from many studies have demonstrated that protein ingestion during the hours following an acute bout of resistance exercise improves skeletal muscle protein net balance by increasing the rate of protein synthesis relative to breakdown (Phillips *et al.*, 2005; Tipton & Wolfe, 2004). Essential amino acids and especially the branched-chain amino acids (mainly leucine) appear primarily responsible for the stimulation of muscle protein synthesis after resistance exercise (Blomstrand, Eliasson, Karlsson, & Kohnke, 2006). However, the general response to protein ingestion appears to be qualitatively similar regardless of whether an essential amino acid mixture, whole intact proteins (e.g., whey) or "real" high-quality protein food such as milk is ingested (Tipton & Witard, 2007). Co-ingestion of other nutrients such as carbohydrate may modify the precise magnitude of the response (Tipton & Witard, 2007) and proteins from different sources may differ in their ability to support protein accretion, possibly related to differences in the rate and pattern of hyperaminoacidemia induced (Phillips *et al.*, 2005). In this regard, Wilkinson *et al.* (2007) recently reported that milk-based proteins promote greater skeletal muscle protein accretion than an isoenergetic amount of soy-based proteins when ingested immediately after an acute bout of resistance exercise.

A key question for athletes is whether the acute, transient changes in skeletal muscle protein turnover induced by nutrient manipulation during recovery between repeated training sessions translates into greater gains in lean mass, skeletal muscle hypertrophy, and/or exercise performance? Several studies conducted over the past several years have examined this issue directly, with equivocal results, likely due in part to differences in study methodology, especially related to the resistance training and nutrient manipulation protocols (Andersen *et al.*, 2005; Beck *et al.*, 2007; Bird, Tarpenning, & Marino, 2006; Candow, Burke, Smith-Palmer, & Burke, 2006; Chromiak *et al.*, 2004; Cribb, Williams, Carey, & Hayes, 2006; Kerksick *et al.*, 2006). Andersen *et al.* (2005) reported hypertrophy of type I and II muscle fibres of the vastus lateralis after 14 weeks of resistance training when a protein supplement was ingested immediately after each training session, but no change versus baseline in a group that ingested an isoenergetic carbohydrate supplement. An unexpected finding from this study, however, was the complete lack of hypertrophy in the carbohydrate-supplemented group, which contrasts with numerous studies that have reported resistance training-induced changes in fibre size despite no specific feeding intervention immediately after exercise (Folland & Williams, 2007). Several other studies have reported greater gains in strength and lean mass after 6–10 weeks of heavy resistance training when a post-exercise protein supplement was ingested rather than carbohydrate (Candow *et al.*, 2006; Kerksick *et al.*, 2006). Another study reported a synergistic effect of protein plus carbohydrate such that the effect of ingesting both on muscle anabolism was greater than that of protein or carbohydrate alone (Bird *et al.*, 2006), while Cribb *et al.* (2006) suggested that training-induced gains in lean mass were higher after whey than casein ingestion. In contrast to those studies that have reported differential effects of nutrient ingestion, other have found no difference in strength or lean mass gains induced by protein versus carbohydrate ingestion after resistance training (Beck *et al.*, 2007; Chromiak *et al.*, 2004).

In summary, there is limited and conflicting information about the effect of protein supplementation during the immediate post-exercise period on training-induced gains in skeletal muscle hypertrophy, lean mass, and strength. Additional work in this area is warranted, especially studies that combine strict exercise training and nutritional controls with sophisticated analytical techniques (e.g. to determine changes in rates of skeletal muscle protein turnover and associated molecular signalling events) and applied performance measurements that are of direct practical relevance to athletes.

Nutrition for altitude training: Special nutritional considerations for training/ competing at altitude

Body mass, energy balance, and appetite

Although body mass loss at high altitude is common, the relative contribution of body fat (Rose *et al.*, 1988) and lean body weight or body fluids (Westerterp, Kayser, Brouns, Herry, & Saris, 1992) to the weight loss is unclear. The amount of body mass lost depends on the altitude attained and the duration of stay (Hamad & Travis, 2006). However, there are very few reports on the amplitude of loss of body mass at altitudes between 1800 and 3000 m. At high to moderate altitude, there is a loss of normal (sea-level) appetite. This suppression may, in part, be explained by an increased concentration of the satiety neuropeptide cholecystokinin (Bailey *et al.*, 2000). This modulation of peripheral cholecystokinin secretion at altitude may also be linked to the metabolic precursors of serotoninergic activity, including non-esterified fatty acid, free tryptophan, and branched-chain amino-acids (Bailey, Davies, Castell, Newsholme, & Calam, 2001). Another explanation is a reduction of appetite through elevated serum leptin concentrations at high altitude (Tschop, Strasburger, Hartmann, Biollaz, & Bärtsch, 1998). Leptin is a key mediator in the neuroendocrine regulation of food intake. During altitude exposure, reduction in energy intake is caused by a change in the appetite profile and in the attitude towards eating. Initially, increased satiety during the course of a meal results in a reduction of meal size, which is partly compensated by an increase in meal frequency. The rapid increase of satiety during a meal is likely to be related to the hypoxic circumstances.

Macronutrient metabolism

The altitude-induced reduction of energy intake is comparable to that observed in chronic obstructive pulmonary disease patients who quickly feel dyspnoeic when feeding (Cochrane & Afolabi, 2004). If this mechanism plays a role at high altitude, this would imply that a low protein diet should be used to reduce diet-induced thermogenesis and thus maintain appetite at high altitude. Thermogenesis values for nutrients are 0 – 3% for fat, 5 – 10% for carbohydrate, but as high as 20 – 30% for protein (Tappy, 1996). Obviously, in the long term, low protein diets will negatively affect protein balance. In addition, there is some evidence that hypoxia may interfere with protein synthesis, whereas a reduction in protein intake and decrease in fat-free mass has mainly been shown over 5000 m. An altitude effect on protein balance would be worsened by a negative energy balance, and thus advice to athletes would be to try and overcome at least the latter by keeping the total energy intake high. To maximize energy intake, fat is preferable for its low thermogenesis, good taste, and higher energy density per gram than carbohydrate. On the other hand, it has been suggested that a greater dependence on glucose rather than fatty acids metabolism would assist in maintaining homeostasis by optimizing the energy yield per unit of oxygen. Compared with fatty acid oxidation, carbohydrate oxidation generates more ATP ($21.1 \ kJ \cdot l^{-1}$) per molecule of oxygen consumed than fat oxidation ($19.6 \ kJ \cdot l^{-1}$). Moreover, carbohydrate can also be metabolized non-oxidatively to yield ATP and lactate. Brooks *et al.* (1991) demonstrated that acclimatization to high altitude (4300 m) resulted in increased utilization of blood glucose both at rest and during submaximal exercise. However, other studies have provided indirect evidence to suggest that after acclimatization there is a greater rate of fat utilization, thereby sparing muscle glycogen stores (Young *et al.*, 1992).

Studies on energy balance at moderate altitude including energy-dense food supplements, which are commonly eaten by professional endurance athletes, are not yet available. However, it is recommended that up to an altitude of 4000 m athletes should be advised to eat a diet high in carbohydrate and fat, according to personal taste, but rather low in protein, aiming at maintaining energy balance.

Micronutrients and "ergogenic compounds"

The most important "erythropoiesis-specific" nutrition factor is iron availability, which can modulate erythropoiesis over a wide range in humans. Adequate iron stores are a necessity for haematological adaptation to hypoxia. However, at moderate altitude, there is a need for rapid mobilization of iron, and even if the stores are normal there is a risk that they cannot be mobilized fast enough for an optimal synthesis of haemoglobin (Berglund, 1992). Thus, all kinds of training aimed at increasing blood mass ("live high – train high" or "live high – train low") should include frequent monitoring of iron stores (e.g. serum ferritin levels) before the onset of the training, and subsequent iron oral supplementation if stores are low and if altitude training or competing is supposed to last more than a month.

Exercise, increased exposure to ultraviolet light (terrestrial altitude), low availability of dietary antioxidants (terrestrial altitude), increases in catecholamine production and auto-oxidation, anoxia/ re-oxygenation (intermittent hypoxic training or exposure), hypoxanthine accumulation, and alteration

of the cell redox potential are all likely contributors to oxidative stress at altitude (for a review, see Askew, 2002). Pialoux *et al.* (2006) reported a significant increase in malondialdehydes and advanced oxidation protein products in response to a 6-week training block at altitude. Moreover, hypoxic training sessions decreased plasma ferric-reducing antioxidant power and the α-tocopherol/triacylglyceride ratio. Providing antioxidant nutrients via the diet or supplements can reduce oxidative stress secondary to altitude exposure (Askew, 2002). Nevertheless, an unanswered question concerning altitude exposure and antioxidant supplementation is when does oxidative stress become potentially damaging enough to merit antioxidant therapy and, conversely, what degree of oxidative stress is necessary to foster the adaptive response of altitude exposure?

Since altitude exposure has been associated with blood hyperviscosity, it has been hypothesized that omega-3 fatty acids supplementation may be helpful for athletes undergoing altitude training. Whether or not supplementing the normal diet with fish oil increase red blood cell deformability remains controversial (Guezennec, Nadaud, Satabin, Leger, & Lafargue, 1989; Oostenbrug *et al.*, 1997), and data are lacking to recommend fish oil supplementation during medium- or long-term altitude exposures.

As bicarbonate ingestion has proven its ergogenic effects in some running events (Applegate, 1999), athletes competing at altitude might be tempted to use such a strategy to enhance buffering and recovery capacities. Such a supplementation may be detrimental at altitude, since it may counteract the rise in bicarbonate urinary excretion usually observed after altitude ascent, hence possibly promoting the onset of acute mountain sickness (Cumbo, Basnyat, Graham, Lescano, & Gambert, 2002).

Fluid balance

Altitude exposure has profound effects on body fluid balance and the magnitude of perturbation is dependent on both the altitude and the duration of exposure (Sawka, Convertino, Eichner, Schneider, & Young, 2000). At rest, and especially after 12–24 h of exposure to altitude, most individuals generally show a decreased plasma concentration of aldosterone, which results in an increase sodium and water excretion. The early stages of acclimatization to altitude are also associated with a mild hyperventilation and a secondary elimination of bicarbonates and water. Plasma renin as well as arginin vasopressin activities are also decreased under such conditions (Olsen *et al.*, 1992). Accordingly, during the first few days of altitude exposure, there are large water and sodium losses. Moreover, simulating altitude with a hypoxic device may worsen the water losses because

of the possible low hygrometry of the inspired air or mixture. Taken collectively, these findings indicate that water intake should be carefully monitored to avoid hypohydration. This is particularly relevant in athletes living and/or sleeping in real or simulated altitude for several days or weeks, for whom chronic dehydration remains a potential risk.

Data on hydro-electrolytic and hormonal adaptations during exercise below 3000 m are limited. However, it seems that the overall physiological response is similar to that observed at sea level (Meehan, 1986; Olsen *et al.*, 1992). Attention should focus on the thermoregulation aspects of altitude (particularly terrestrial altitude) where increased direct and indirect solar radiation and dry inhaled air may increase water losses (pulmonary and cutaneous) due to evaporation.

Summary: Reaching a consensus

Consensus for:

- Athletes should maximize carbohydrate availability before, during, and after training sessions/competition to enhance the training adaptation/performance.
- Protein ingestion during the early stages (0–3 h) of recovery from acute resistive- and endurance-type exercise favours net skeletal muscle protein accretion compared with ingestion of an isoenergetic amount of food without protein or no feeding.
- Athletes should increase meal frequency to counteract the anorectic effects of high altitude.
- An iron supplement should be provided where indicated by biological measurement.

Consensus against:

- Ingesting protein with carbohydrate during recovery from endurance-type exercise does not appear to enhance muscle glycogen synthesis or improve subsequent exercise performance compared with ingesting an isoenergetic amount of carbohydrate.

Issues that are equivocal:

- Whether chronic perturbations in muscle glycogen content and/or glucose availability impair or enhance training adaptation in competitive athletes who compete in either endurance or strength/power events.
- Adding protein to sports drink may extend endurance time to fatigue when suboptimal amounts of carbohydrate are ingested and glucose availability during exercise is

compromised. However, exercise performance is not enhanced when athletes are permitted to ingest sufficient amounts of carbohydrate and perform exercise tasks that closely mimic real life athletic competition.

- Whether protein ingestion during recovery from acute exercise bout translates into greater skeletal muscle adaptation (e.g. increased size or strength, improved fatigue resistance) or event-specific performance over a period of chronic training.
- Whether athletes training at altitude should increase fat and carbohydrates intake or modify protein intakes. Further research is necessary to determine whether antioxidant supplements and omega-3 fatty acids supplementation are ergogenic for athletes at altitude.

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