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Nutrition for the sprinter

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Abstract
The primary roles for nutrition in sprints are for recovery from training and competition and influencing training adaptations. Sprint success is determined largely by the power-to-mass ratio, so sprinters aim to increase muscle mass and power. However, extra mass that does not increase power may be detrimental. Energy and protein intake are important for increasing muscle mass. If energy balance is maintained, increased mass and strength are possible on a wide range of protein intakes, so energy intake is crucial. Most sprinters likely consume ample protein. The quantity of energy and protein intake necessary for optimal training adaptations depends on the individual athlete and training demands; specific recommendations for all sprinters are, at best, useless, and are potentially harmful. However, if carbohydrate and fat intake are sufficient to maintain energy levels, then increased protein intake is unlikely to be detrimental. The type and timing of protein intake and nutrients ingested concurrently must be considered when designing optimal nutritional strategies for increasing muscle mass and power. On race day, athletes should avoid foods that result in gastrointestinal discomfort, dehydration or sluggishness. Several supplements potentially influence sprint training or performance. Beta-alanine and bicarbonate may be useful as buffering agents in longer sprints. Creatine may be efficacious for increasing muscle mass and strength and perhaps increasing intensity of repeat sprint performance during training.

Keywords: *Muscle hypertrophy, training adaptations, net muscle protein balance, creatine, power-to-mass ratio*

Introduction

The sprint events cover distances from 60 to 400 m. These running and hurdle events rely primarily on the development of power through anaerobic energy, the phosphocreatine (shorter events, e.g. 100 m and 200 m), and glycolytic (longer events, e.g. 400 m) systems for energy. A sprint consists of an all-out effort for a short period of time; performance is determined by the ability to achieve maximal velocity and to limit the loss of power as the sprint progresses. Biomechanical, neuromuscular, and metabolic factors all influence performance.

Nutritional support for athletic performance is a popular and widely covered topic. However, most sports nutrition research has focused on endurance performance. Relatively little has been written about nutrition for sprinting performance. Although the potential for an effect is arguably somewhat less than for endurance sports, especially during an event, nutritional choices and strategies will contribute to adaptations to training and to performance in sprinters.

Early work demonstrated that elite sprinters have muscles composed predominantly of fast-twitch fibres (Costill *et al.*, 1976). Thus, success requires large, powerful muscles. Accordingly, the major role for nutrition may be to modulate muscle hypertrophy from training. In this review, we focus on the role of nutrition for increasing muscle mass and strength, as well as the potential for nutritional choices to influence competition day performance.

Nutritional support for athletes is often considered for two general situations: training and competition. Important considerations for the sprinter are:

- maintaining energy levels during training;
- quick recovery from training;
- optimizing training adaptations with nutrition;
- achieving a high power-to-weight ratio, thus maximizing muscle mass and maintaining low body fat;
- staying focused, sharp and maintaining concentration during competition days;
- improved reaction times.

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Nutrition for sprint training

Sprint training is focused on developing lean body mass capable of generating the power necessary to carry the athlete as rapidly as possible. Adaptations to training are specific to the mode, intensity, and duration of the exercise. These adaptations stem primarily from the exercise stimulus on the muscle fibres, but may be influenced by nutritional factors. Nutrition most certainly will influence muscle hypertrophy and this aspect of nutrition is usually the focus for sprinters. Besides specific sprint training, weight training with the goal of developing muscle mass is the primary form of training throughout the year. However, it is important to recognize that optimum mass may not equal maximum mass for a sprinter. At some point, the power-to-mass ratio may begin to decline with extra mass regardless of composition. Some aspects of the nutritional influence on training adaptations are also covered in other reviews in this issue (Hawley, Gibala, & Bermon, 2007; Houtkooper, Abbot, & Nimmo, 2007).

Recent evidence on the molecular and metabolic levels indicates that training adaptations occur as protein levels change due to the response to each bout of exercise (Hawley, Tipton, & Millard-Stafford, 2006). Muscle mass is determined by changes in protein levels, particularly myofibrillar proteins. Increased myofibrillar proteins result from net positive balance of myofibrillar synthesis and breakdown over a given period. The primary changes occur in response to exercise plus nutrition.

The bulk of the response of net muscle protein balance occurs following, rather than during, exercise (Durham *et al.*, 2004); increased muscle protein synthesis, rather than decreased breakdown, is responsible for the increased balance (Tipton & Wolfe, 2004). Elevation of muscle protein synthesis is delayed after exercise (Pitkanen *et al.*, 2003), presumably due to inhibition of translational pathways due to elevations in AMPK (Bolster *et al.*, 2003; Koopman, Zorenc, Gransier, Cameron-Smith, & van Loon, 2006). Once activated, muscle protein synthesis and net muscle protein balance remain elevated for up to 48 h after the exercise bout (Phillips, Tipton, Aarsland, Wolf, & Wolfe, 1997); however, protein balance does not become positive without provision of exogenous amino acid sources (Biolo, Tipton, Klein, & Wolfe, 1997).

Consumption of a source of amino acids following exercise increases the response of muscle protein synthesis in an additive manner resulting in positive muscle protein balance (Biolo *et al.*, 1997; Borsheim, Tipton, Wolf, & Wolfe, 2002). The response of net muscle protein balance is due primarily to the essential amino acids (Borsheim *et al.*, 2002; Tipton, Ferrando, Phillips, Doyle, & Wolfe, 1999). Thus, consumption of a source of essential amino acids, be it an intact protein or a free amino acid mixture, will stimulate uptake of amino acids for synthesis of muscle proteins necessary for muscle growth. The optimum amount of amino acids has yet to be determined, but it is clear that a relatively small amount of exogenous amino acids (about 12 g) results in positive protein balance (Borsheim *et al.*, 2002). However, it remains to be seen if chronic consumption of a small amount of amino acids capable of stimulating a transient metabolic response is enough to stimulate muscle hypertrophy in the long term.

It is clear that the metabolic and thus phenotypic responses to exercise and thus training adaptations are mediated by intracellular signalling. Recent reviews have examined the response of these pathways to exercise and nutrition in detail (Rennie, 2005; Tipton & Sharp, 2005). Signalling aspects of muscle adaptation to training are also discussed elsewhere in this issue (Hawley *et al.*, 2007).

Both translational (Bolster, Jefferson, & Kimball, 2004) and transcriptional (Creer *et al.*, 2005; Psilander, Damsgaard, & Pilegaard, 2003) mechanisms are stimulated by resistance exercise, but the bulk of the increase in muscle protein synthesis is translational (Chesley, MacDougall, Tarnopolsky, Atkinson, & Smith, 1992). Rates of muscle protein synthesis are increased with no increase in total RNA, suggesting that it is the efficiency of translation – that is, an increase in synthesis per molecule of RNA – that is increased by resistance exercise (Chesley *et al.*, 1992). Resistance exercise increases phosphorylation of many translation initiation pathway components (Coffey *et al.*, 2006; Karlsson *et al.*, 2004; Koopman *et al.*, 2006). Adaptations that influence muscle growth also stem from transcriptional regulation (Coffey *et al.*, 2006; Williamson, Gallagher, Harber, Hollon, & Trappe, 2003); transcriptional activity of genes for muscle growth factors and myosin heavy chain is stimulated by resistance exercise (Psilander *et al.*, 2003; Raue, Slivka, Jemiolo, Holon, & Trappe, 2006). At this juncture, it is somewhat difficult to discern detailed influences of these pathways due to differences in study design and methods among the studies. Much work needs to be done to address the gaps in our knowledge.

Nutrition has a clear effect on the signalling pathways related to muscle protein synthesis. Previous reviews have detailed the response of intracellular signalling to exercise and nutrition (Kimball, Farrell, & Jefferson, 2002; Tipton & Sharp, 2005). The influence of amino acids on muscle protein synthesis is primarily through the mTOR signalling pathways (Kimball *et al.*, 2002; Tipton & Sharp, 2005). Leucine is particularly effective in stimulating

initiation of translation following exercise (Anthony *et al.*, 2000; Gautsch *et al.*, 1998). In humans, the administration of branched-chain amino acids after resistance exercise increased phosphorylation of p70s6k 3.5-fold above the increase due to exercise when measured at 1 and 2 h after exercise (Karlsson *et al.*, 2004). However, the time course of signalling events relative to muscle protein synthesis has yet to be examined in humans and much has yet to be determined.

Increased insulin levels resulting from carbohydrate intake are clearly a major controller of translation initiation pathways (Kimball *et al.*, 2002). Thus, carbohydrates seem to play a role in the response of muscle protein synthesis to feeding after exercise. Insulin stimulates these translation signalling pathways primarily through phosphoinositol-3-kinase (PI3k), Akt, and mTOR (Kimball *et al.*, 2002). Interestingly, post-exercise hyperinsulinaemia only minimally stimulates muscle protein synthesis above normal post-exercise levels (Biolo, Williams, Fleming, & Wolfe, 1999). Moreover, Anthony *et al.* (2000) demonstrated that the response of translation initiation to amino acids is mediated by insulin. Taken together, these results suggest that combining carbohydrates and proteins may be the best strategy for stimulation of anabolic pathways. Indeed, utilization of ingested amino acids for synthesis of muscle proteins is greatest when carbohydrates are ingested concurrently with an amino acid source (Tipton & Witard, 2007). Furthermore, leucine has been demonstrated to be an effective insulin secretagogue (Koopman *et al.*, 2005).

Carbohydrate intake may be important for muscle anabolism in other ways as well. Depletion of muscle glycogen is possible, given the multiple sprints common to a sprint training session (Gaitanos, Williams, Boobis, & Brooks, 1993). Thus, carbohydrate intake should be sufficient to maintain glycogen levels, not only to prevent fatigue development and maximize training potential, but also perhaps to optimize muscle anabolism. Low glycogen availability may influence the adaptive response to training (Churchley *et al.*, 2007; Creer *et al.*, 2005), suggesting that the maximal anabolic response to resistance exercise may not be possible when exercise is initiated with low glycogen levels. Thus, sprinters should consume sufficient carbohydrate to maintain glycogen during training.

Clearly, protein intake is important for muscle hypertrophy, but the amount of dietary protein necessary for a sprint athlete to optimize muscle gains and performance is difficult to determine. A high protein intake is often thought to be critical for muscle growth, repair, and enhancement of training adaptations, and a huge supplement industry has been built upon this assumption. The scientific evidence for the efficacy of high protein intakes for increasing muscle mass is, at best, equivocal, and has been extensively debated in the scientific community. The reader is referred to previous reviews for discussions of the merits of increased protein requirements in athletes (Phillips, 2006; Rennie, Wackerhage, Spangenburg, & Booth, 2004; Tipton & Witard, 2007; Tipton & Wolfe, 2004).

It is likely that the disparity of opinions regarding overall protein needs arises primarily from two sources: methodological limitations (nitrogen balance and leucine oxidation) and, perhaps more fundamentally, a lack of consideration for the principal reason athletes ingest protein (Tipton & Witard, 2007). Rather than the attainment of nitrogen balance, the amount of protein that optimizes muscle hypertrophy and maximizes performance is the most salient factor. Attainment of nitrogen balance is unlikely to concern sprinters; rather, they endeavour to consume the amount of protein necessary to optimize muscle mass and power.

For many, if not most athletes, including sprinters, the point may be inconsequential. Most athletes ingest enough protein to cover even the higher estimates of $\sim 1.2-1.5\ g \cdot kg^{-1} \cdot day^{-1}$ (Phillips, 2006; Tarnopolsky, 2004). Thus, recommending increased protein intake would not be necessary for the majority of athletes consuming a well-chosen diet that meets energy needs. Moore *et al.* (2007) recently demonstrated that increased nitrogen balance and lean body mass result from 12 weeks of intense resistance training while consuming $1.4\ g \cdot kg^{-1} \cdot day^{-1}$. Thus, habitually high intakes, often greater than $2\ g \cdot kg^{-1} \cdot day^{-1}$, appear to be unnecessary for muscle hypertrophy and increased strength and power. It is likely that protein intake in excess of $1.7\ g \cdot kg^{-1} \cdot day^{-1}$ is simply oxidized (Tarnopolsky, 2004). Of course, it should be noted that these results come from previously untrained individuals and it is arguable that years of training may change these responses.

On the other hand, a relatively high protein intake is unlikely to be detrimental, even if not entirely necessary. Given the high energy intakes necessary to support increased muscle mass, habitual protein consumption is likely to ensure maximum muscle accretion. Although protein and amino acid supplements may be convenient sources of essential amino acids, no evidence exists to suggest the anabolic response to protein from food sources is inferior to commercially available supplements (Elliot, Cree, Sanford, Wolfe, & Tipton, 2006; Phillips, 2006).

It is not possible to make a broad recommendation for a specific amount of protein for all sprinters based on current scientific evidence. Any such recommendations are based on the presumption that muscle

protein accretion is linear in relation to the amount of protein ingested (Tipton & Witard, 2007). In fact, much of the evidence casts doubt upon such broad recommendations. Metabolic studies clearly demonstrate that the anabolic response to feeding is dependent on many factors, including, but not limited to, the amount of protein ingested. The type of protein ingested will affect the utilization of the amino acids for synthesis of muscle proteins (Wilkinson *et al.*, 2007), the anabolic response to protein ingestion will vary with ingestion of other concurrently ingested nutrients (Borsheim, Aarsland, & Wolfe, 2004a; Elliot *et al.*, 2006), and, finally, the timing of ingestion of an amino acid source will influence the anabolic response of muscle (Tipton *et al.*, 2001, 2006). Taken together, it is clear that ingestion of a given amount of protein may result in a variable metabolic response depending on several factors other than solely the amount of protein. Training adaptations may depend less on the amount of protein ingested, and more on the type of proteins ingested, timing of ingestion, and other nutrients ingested in the same meal.

A risk–benefit approach might offer some insights for recommendations of protein intake for athletes. If risk is minimal and there is a rationale for potential benefit, then there is no reason to recommend against increasing protein intake. Health problems, such as kidney damage and bone loss, have often been given as reasons for avoiding high protein intakes. Whereas these issues are theoretically associated with high protein intake, to date there is no evidence for kidney damage from high protein in those with no predisposing kidney maladies (Zello, 2006). A major component of bone is protein and, in fact, synthesis of bone collagen responds similarly to muscle proteins following ingestion of an amino acid source (Babraj *et al.*, 2005). Thus, it appears that reasonably high protein intakes offer little in the way of health dangers to sprinters and other athletes. The relationship between high protein intake and chronic diseases has not been firmly established, but it should be noted that individuals with pre-existing kidney disease should not consume high-protein diets (Zello, 2006). For many athletes, the primary risk of high protein intakes is a necessary reduction in carbohydrate intake, which may then affect performance (Macdermid & Stannard, 2006). Glycogen depletion is likely during training sessions involving repeated sprints (Balsom, Gaitanos, Soderlund, & Ekblom, 1999; Gaitanos *et al.*, 1993) and so sufficient carbohydrate intake to support these training sessions is clearly necessary. However, sprint athletes habitually consume ample carbohydrate and protein (Burke, Millet, & Tarnopolsky, 2007) to support training.

Protein nutrition is most often the focus of athletes whose goals include increased muscle mass. However, it is clear that energy balance is just as important, if not much more so, for muscle hypertrophy than is protein intake. It is not possible to maintain positive nitrogen balance during an energy deficit (Todd, Butterfield, & Calloway, 1984). Up to about one-third of the variability in nitrogen balance is likely due to energy intake (Pellett & Young, 1992). As early as 1907, Chittenden demonstrated that as long as energy intake is sufficient, athletes will gain muscle mass and increase strength even during periods of low protein intake. More recently, positive energy balance has been demonstrated to be more important than the amount of protein ingested for gains in lean body mass during resistance training (Rozenek, Ward, Long, & Garhammer, 2002).

The arguments rendered above are contingent upon one primary assumption, namely that the acute metabolic response of muscle to exercise and nutrition represents the potential for long-term muscle gain. Clearly, the ideal study to determine the effect of various feeding strategies on sprinters would be to measure changes in muscle mass, strength, power, and speed – ultimately sprint performance – during periods with different feeding strategies. Unfortunately, these studies are virtually impossible to perform, primarily due to difficulties in controlling all variables (Tipton & Witard, 2007; Tipton & Wolfe, 2004). A series of recent investigations suggests that results from acute metabolic studies adequately represent the potential for changes in muscle mass in response to training and nutrition (Paddon-Jones *et al.*, 2004; Tipton, Borsheim, Wolf, Sanford, & Wolfe, 2003; Tipton & Witard, 2007). Results from studies at the molecular level support this contention (Coffey *et al.*, 2006; Hawley *et al.*, 2006; Psilander *et al.*, 2003).

Since the balance between synthesis and breakdown ultimately determines the amount of each protein in muscle, a decrease in muscle protein breakdown rates should contribute to increased muscle mass. Thus, nutritional strategies aimed at decreasing rates of muscle protein breakdown following exercise are often recommended. Amino acid ingestion following resistance exercise can reduce muscle protein breakdown (Biolo *et al.*, 1997; Tipton *et al.*, 1999). Utilization of amino acids from ingested amino acid sources by muscle has been shown to increase when carbohydrates are ingested simultaneously (Borsheim *et al.*, 2004a, 2004b). The effect of carbohydrates is presumably due to the associated insulin release. Insulin increases net muscle protein balance following resistance exercise primarily by blocking the rise in

muscle protein breakdown (Biolo *et al.*, 1999). Thus, it is often recommended to consume protein plus carbohydrates after exercise to maximize net muscle protein balance and increase muscle mass.

Recent data suggest that the role of protein breakdown for muscle hypertrophy may not be as clear. After a resistance exercise bout, rates of muscle protein breakdown increase and are associated with the increased rates of synthesis (Biolo, Maggi, Williams, Tipton, & Wolfe, 1995; Phillips *et al.*, 1997). Furthermore, transient changes in gene expression and mRNA levels of both myogenic (Psilander *et al.*, 2003) and atrogenic (Churchley *et al.*, 2007; Jones *et al.*, 2004) genes following resistance exercise suggest that increases in both the synthesis and breakdown of proteins are important for changes in protein levels. These data suggest that muscle protein breakdown may play an important role for accretion of muscle. Clearly, more research on the effect of nutrient intake on specific muscle degradative pathways is warranted.

Nutrition for racing

The acute influence of nutritional intake for sprinting is not likely to be as great as for endurance events. The length of the race alone prevents a large influence from acute intakes. Although sprint events only last seconds, competition can be rather drawn out. A typical competition day involves a number of heats and finals with variable amounts of waiting around in between. For example, at the World Championships in Osaka in 2007, the schedule for the 100 m is:

- Day 1, 12:10 h, men's 100 m heats
- Day 1, 20:15 h, men's 100 m quarter-finals
- Day 3, 20:10 h, men's 100 m semi-finals
- Day 3, 22:20 h, men's 100 m final

On day 1, there are 8 h between rounds 1 and 2, but on the third day only 2 h between the semi-finals and final!

During the time in between heats, athletes should stay hydrated but avoid over-drinking, maintain blood glucose levels, and avoid behaviours, including feeding, that may contribute to discomfort, particularly gastrointestinal discomfort. Careful consideration of what *not* to eat is probably more important than what to eat. It is likely that there is no common way to achieve these goals in every athlete. There is almost certainly large individual variation and preference in ways to achieve these goals. Experimenting in training is therefore essential to develop a good routine on race day.

Supplements for the sprinter

There are a few supplements that should be addressed in relation to sprint performance. We will concentrate primarily on supplements for which there is sufficient evidence of their efficacy. Others that are often considered to be important for aspects related to sprint performance (e.g. hydroxymethylbutyrate, ribose) will not be included in this discussion. The results from studies on most supplements are equivocal and it is difficult to recommend usage by sprinters at this time. More detail on these and other supplements can be found in another review in this issue by Maughan and colleagues (Maughan, Depiesse, & Geyer, 2007) and in previous reviews (Hespel, Maughan, & Greenhaff, 2006; Maughan, King, & Lea, 2004).

When maximal exercise is performed for more than 30 s, most of the energy is derived from anaerobic glycolysis. These high rates of glycolysis have been associated with increased muscle acidity and this may eventually impair muscle contraction. Increasing the buffering capacity is theoretically a way of improving performance in such events (>30 s up to about 7 min; 400-m running may just be long enough to benefit from an increased buffering capacity).

Beta-alanine (β-alanyl-L-histidine)

Beta-alanine is a non-essential amino acid that is common in many foods, especially meats. Beta-alanine is believed to be the rate-limiting substrate for synthesis of carnosine, which is an important intracellular buffer (Dunnett & Harris, 1999). Carnosine is found primarily in type IIa and type IIx fibres in skeletal muscle and contributes to intracellular buffering of H^+. Thus carnosine attenuates the decrease in intracellular pH associated with anaerobic metabolism. Interestingly, carnosine concentrations in athletes, such as sprinters, appear to be higher than those of marathoners or untrained individuals (Abe, 2000; Tallon, Harris, Boobis, Fallowfield, & Wise, 2005). Furthermore, intense physical training may increase muscle carnosine concentrations (Hill *et al.*, 2007). Four weeks of beta-alanine supplementation increased muscle carnosine by 59% and 10 weeks of supplementation increased it by 80% (Hill *et al.*, 2007).

In theory, increasing skeletal muscle carnosine levels (via beta-alanine supplementation or intense training) should increase buffering capacity, delay fatigue, and increase exercise performance. Higher carnosine concentration in muscle was associated with higher mean power from a 30-s maximal sprint on a cycle ergometer (Suzuki, Ito, Mukai, Takahashi, & Takamatsu, 2002). Beta-alanine

supplementation has been demonstrated to increase muscle carnosine content and to be associated with increased performance (Stout *et al.*, 2007).

Future studies need to confirm the limited results that are available and examine the combined effect of beta-alanine supplementation and training on muscle carnosine in highly trained athletes.

Sodium bicarbonate

The primary buffers in the muscle are phosphates and tissue proteins. The most important buffers in the blood are proteins, including haemoglobin, and bicarbonate. During intense exercise, the intracellular buffers (including carnosine) are insufficient to buffer all the hydrogen ions formed. The efflux of H^+ into the circulation increases, and bicarbonate has a role in buffering these H^+ ions. Bicarbonate ingestion (in the form of sodium bicarbonate) is the traditional method of increasing the extracellular buffering capacity, although sodium citrate is often used as well. The mechanism by which bicarbonate supposedly exerts its action is through this buffering of H^+ in the extracellular fluid. This increases the H^+ gradient and increases efflux of H^+ from the muscle.

Reviews of the available literature suggest a dose–response relationship between the amount of bicarbonate ingested and the observed performance effect (Horswill, 1995). A dose of 200 $mg \cdot kg^{-1}$ body mass ingested 1–2 h before exercise seems to improve performance in most studies, but 300 $mg \cdot kg^{-1}$ body mass appears to be the optimum dose (with tolerable side-effects for most athletes). Doses of less than 100 $mg \cdot kg^{-1}$ body mass do not affect performance. Intakes of more than 300 $mg \cdot kg^{-1}$ body mass tend to result in gastrointestinal problems. Most of these studies, however, used exercise lasting longer than 1 min and in most the exercise intensity and duration were comparable to middle-distance running not sprints.

No studies have shown an effect on performance in high-intensity exercise lasting less than 1 min. Therefore, a window for efficacy of bicarbonate has been identified between approximately 1 and 7 min and sprint events are not likely to be affected. Nevertheless, the use of bicarbonate is common in 400-m running, with anecdotal support for its efficacy.

Creatine

Creatine is a natural guanidine compound that occurs in meat and fish in concentrations between 3 and 7 $g \cdot kg^{-1}$ (Walker, 1979). Synthetic creatine supplements exist as creatine monohydrate or various creatine salts, such as creatine citrate or creatine pyruvate. The latter are soluble and stable in solution and thus can be included in sports drinks or gels. On the other hand, creatine monohydrate must be consumed soon after it is brought into solution.

The effects of creatine intake on strength and power, primary determinants of sprint running performance, have been investigated extensively since Harris and co-workers first reported that a few days of high-dose oral creatine supplementation can increase muscle creatine content (Harris, Soderlund, & Hultman, 1992). However, to the best of our knowledge, only two well-controlled studies have looked specifically at performance in well-trained sprint athletes. Skare *et al.* (2001) investigated the effect of creatine intake on sprint velocity during a 100-m sprint followed by 6 × 60-m sprints in locally competitive sprinters. Creatine intake (20 $g \cdot day^{-1}$ for 5 days) marginally increased running velocity in the initial 100-m sprint and in five of the 60-m sprints. Conversely, performance in sprinters at national level was not increased by creatine intake (0.35 $g \cdot kg^{-1}$ body mass for 7 days) in a 6 × 40-m (2-min rest intervals) intermittent exercise test (Delecluse, Diels, & Goris, 2003). Other studies utilizing various types of athletes provide equivocal results (Glaister *et al.*, 2006; Mujika, Padilla, Ibanez, Izquierdo, & Gorostiaga, 2000). Importantly, none of the available studies has reported impaired performance.

Direct determination of a beneficial effect of creatine supplementation on sprint performance is difficult. First, the performance benefit of creatine intake as a rule is very small, and conceivably within the limits of day-to-day variability in performance, thus the reliability of sprint tests ''in the field'' may be too small to demonstrate a significant effect of creatine in a small sample of individuals. Second, the available data pertain predominantly to very short sprints (15–60 m) where start reaction time as well as running skill (coordination) are major determinants of performance. Creatine intake is unlikely to be beneficial to either of these important factors. Finally, it is important to emphasize that results from well-controlled laboratory studies consistently indicate that creatine supplementation can enhance power output during short maximal exercise (Terjung *et al.*, 2000), in particular during intermittent series (10–30 s) of maximal muscle contractions (Casey, Short, Curtis, & Greenhaff, 1996; Greenhaff *et al.*, 1993) interspersed by 1–2 min rest intervals. Heavy resistance training accounts for an important fraction of the total training volume in elite sprinters. It has been well documented that creatine supplementation can potentiate the gains in fat-free mass and muscle force and power output that accompany resistance training (Hespel *et al.*, 2001; Volek *et al.*, 1997). Thus, creatine supplementation conceivably could contribute to improving sprint performance by enhancing the efficacy of resistance training.

A classical creatine loading regimen consists of an initial loading phase ($15-20\ g \cdot day^{-1}$ for 4–7 days) followed by a maintenance dose ($2-5\ g \cdot day^{-1}$) (Terjung *et al.*, 2000). However, individual responses vary and there are indications that a positive effect on muscle mass may diminish after 8–10 weeks (Derave, Eijnde, & Hespel, 2003). Although solid data are lacking, it may be advisable to add wash-out periods to periods of creatine supplementation for optimum impact. Individuals with a low initial muscle creatine content, such as vegetarians (Burke *et al.*, 2003), respond better to creatine supplementation than others with high a natural muscle creatine content. Therefore, creatine intake probably is an adequate adjuvant to a vegetarian diet in sprinters. Furthermore, ingesting creatine in conjunction with training sessions can stimulate muscle creatine uptake, as exercise is known to facilitate the disposal of ingested creatine into the musculature (Harris *et al.*, 1992). It is probably also worthwhile considering the possibility of ingesting creatine supplements in combination with post-exercise carbohydrate–amino acid–protein supplements in order to enhance muscle creatine retention due to increased insulin concentrations (Steenge, Simpson, & Greenhaff, 2000). Creatine is not on the doping list and its intake is generally found to be safe in healthy adults provided the aforementioned guidelines are followed (Terjung *et al.*, 2000). Creatine intake does result in increased body mass from intracellular water accumulation. Increased water content may be problematic for some athletes, particularly sprinters desiring optimal power-to-mass ratios.

The physiological mechanisms underlying the effects of creatine supplementation are only partly understood. Some indicate that an increased muscle creatine content can facilitate flux through the creatine kinase reaction and thereby prevent net ATP degradation during high-intensity muscle contractions (Casey *et al.*, 1996). This flux could also explain the shortening of muscle relaxation time seen after creatine loading (Van, Vandenberghe, & Hespel, 1999) possibly contributing to increased stride frequency during sprinting with creatine intake (Schedel, Terrier, & Schutz, 2000). Given that the importance of phosphocreatine to energy production (relative to muscle glycogen) increases as the duration of a sprint is shortened, the ergogenic effect of creatine may be more important in the 60- and 100-m sprints than in the longer sprint events (200–400 m). Furthermore, stimulation of muscle phosphocreatine resynthesis may contribute to enhanced recovery between intermittent sprint bouts (Casey *et al.*, 1996; Greenhaff, Bodin, Soderlund, & Hultman, 1994), thus enhancing sprint training.

The mechanisms underlying the potential of creatine to stimulate muscle anabolism during resistance training are unclear. Creatine intake may increase the stimulation of satellite cell proliferation (Olsen *et al.*, 2006) or intracellular signalling pathways (Deldicque *et al.*, 2005; Louis, Van, Dehoux, Thissen, & Francaux, 2004). However, direct evidence that creatine can stimulate net protein synthesis in human muscle is lacking (Louis *et al.*, 2003; Parise, Mihic, MacLennan, Yarasheski, & Tarnopolsky, 2001). Alternatively, changes in muscle protein accretion can occur as a consequence of individuals performing more work during high-intensity training programmes while consuming creatine (Kreider *et al.*, 1998).

Caffeine

Caffeine is a popular stimulant used by most individuals, including athletes. Caffeine is contained in coffee, tea, chocolate, and many other caffeinated food sources like cola and so-called "energy-booster" drinks. The primary mechanism of action by which caffeine can beneficially affect performance probably is by enhancing central drive and/or improving muscle fibre recruitment (Graham, 2001). It is well known that small doses of caffeine ($1-2\ mg \cdot kg^{-1}$ body mass) can beneficially influence mental alertness and thereby shorten reaction time (Haskell, Kennedy, Wesnes, & Scholey, 2005), which is obviously crucial to sprint success. However, care must be taken to determine the optimum dose during training because overdosing will have a negative effect on reaction time. Because sprinters typically compete on an empty stomach, caffeine will be very rapidly absorbed, and if ingested during the pre-competition warm-up period, the potential performance benefits may be reduced during the competition to follow (Bell & McLellan, 2002). Caffeine should be ingested in an isolated formulation (capsules or tablets) rather than in the form of strong coffee because the latter is more likely to cause gastrointestinal distress (Tarnopolsky, 1994).

Responses to caffeine intake and withdrawal vary greatly among individuals depending on the degree of habituation (Bell & McLellan, 2002; Magkos & Kavouras, 2004). Therefore, individual tuning of the dosage in the context of training is very important. Frequent high-dose caffeine intake results in rapid desensitization and will require the use of an even higher dose.

The common use of caffeine as a "social stimulant" probably proves that low-dose caffeine intake should be considered to be safe. However, high-dose caffeine intake is well known to be associated with adverse health effects, in particular at the level of the cardiovascular system (Tarnopolsky, 1994). In 2004, the World Anti Doping Agency (WADA) removed

caffeine from the list of banned substances and its use is currently being monitored.

Summary of nutritional guidelines for sprinters

Consensus for:

- Carbohydrate intake should be sufficient ($\sim 5\ g \cdot kg^{-1}$ body mass) to maintain glycogen stores during training.
- Energy intake should be carefully considered: if increased muscle mass is desired, energy intake should be increased; if muscle mass is optimal, energy intake should be maintained and perhaps monitored.
- Protein intake is likely adequate for the majority of sprinters, but if energy intake is increased a portion of this increase could, and perhaps should, be protein.
- Type of protein and timing of protein ingestion should be considered if increased muscle mass is the goal.
- Race day nutrition should be developed individually with the goal of avoiding gastrointestinal distress and dehydration.
- Creatine supplementation may enhance increases in muscle mass and strength, but sprinters must consider the extra weight gain associated with creatine use.

Consensus against:

- A single broad recommendation for protein intake for all sprint athletes is non-sensical.
- Since power/mass is the most critical element of sprinting success, automatically assuming strategies that may increase muscle mass are desirable could be a mistake if increased mass decreases the power-to-mass ratio.
- Use of supplements as sources of protein and amino acids in the belief that they provide superior quality to foods.
- There is insufficient evidence to recommend other supplements (e.g. ribose, hydroxymethylbutyrate, vanadyl sulphate) to sprinters at this time.

Issues that are equivocal:

- Relationship of type of protein and timing of ingestion of proteins for muscle anabolism.
- The role of muscle degradative pathways for adaptation to training.
- The impact of nutrition on muscle degradative pathways for training adaptations.
- Effect of nutrients and the interaction with training on signalling pathways and gene expression in muscle.
- Efficacy of beta-alanine for increasing muscle carnosine in well-trained sprinters.
- Efficacy of bicarbonate in sprinters.

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