Nutritional Strategies to Support Adaptation to High-Intensity Interval Training in Team Sports

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Abstract
Team sports are characterized by intermittent high-intensity activity patterns. Typically, play consists of short periods of very intense or all-out efforts interspersed with longer periods of low-intensity activity. Fatigue is a complex, multi-factorial process, but intense intermittent exercise performance can potentially be limited by reduced availability of substrates stored in skeletal muscle and/or metabolic by-products associated with fuel breakdown. High-intensity interval training (HIT) has been shown to induce adaptations in skeletal muscle that enhance the capacity for both oxidative and non-oxidative metabolism. Nutrient availability is a potent modulator of many acute physiological responses to exercise, including various molecular signaling pathways that are believed to regulate cellular adaptation to training. Several nutritional strategies have also been reported to acutely alter metabolism and enhance intermittent high-intensity exercise performance. However, relatively little is known regarding the effect of chronic interventions, and whether supplementation over a period of weeks or months augments HIT-induced physiological remodeling and promotes greater performance adaptations. Theoretically, a nutritional intervention could augment HIT adaptation by improving energy metabolism during exercise, which could facilitate greater total work and an enhanced chronic training stimulus, or promoting some aspect of the adaptive response during recovery, which could lead to enhanced physiological adaptations over time.

Introduction
Team sports are characterized by intermittent high-intensity activity patterns. For a given player, the metabolic demands will vary depending on many factors including position, playing style and game strategy. Typically however, play
consists of short periods of very intense or all-out efforts interspersed with longer periods of low-intensity activity, and training programs are at least partly designed to simulate this activity pattern. Much of our knowledge regarding the metabolic demands of training and competition in team sport comes from studies conducted on soccer players [1]. For example, top players can perform 150–250 brief intense sprints over 10–15 m while covering a total of 10–12 km during the course of a match. The intermittent nature of this type of activity requires a high capacity for both aerobic (oxidative) and anaerobic (non-oxidative) energy provision, with skeletal muscle glycogen being a predominant fuel source [2].

**Metabolic Factors Limiting Performance during Intense Intermittent Exercise**

Fatigue is a complex, multi-factorial process, but intense intermittent exercise performance can potentially be limited by reduced availability of substrates stored in skeletal muscle (e.g. glycogen) and/or metabolic by-products associated with fuel breakdown [3]. Almost half the individual muscle fibers examined after a standard soccer match were either completely or nearly completely emptied of glycogen, suggesting that fiber-specific depletion may have impaired force-generating capacity and contributed to the reduced sprint performance observed after the second half of play [2]. Another potential mechanism related to glycogen metabolism is the metabolic acidosis that can occur during intense muscle contraction, owing to increased hydrogen ion accumulation in conjunction with lactate production. Intense intermittent exercise can cause significant decreases in muscle pH that are associated with impaired metabolic and contractile processes [3], although the acute change in muscle pH reported after a soccer game was modest and unrelated to the decline in sprint performance observed [2]. Nonetheless, a high buffering capacity has been associated with enhanced high-intensity exercise performance [4].

**Skeletal Muscle Adaptation to High-Intensity Interval Training**

High-intensity interval training (HIT) is infinitely variable, and the specific physiological adaptations induced by this form of training are determined by numerous factors including the precise nature of the exercise stimulus, i.e. the intensity, duration and number of intervals performed as well as the nature and duration of the recovery periods [5]. Interval intensity is a critical variable that
can be quantified in various ways, but HIT generally refers to repeated efforts that correspond to ≥90% of maximal heart rate or ≥85% of peak oxygen uptake (VO\textsubscript{2peak}). Numerous short-term HIT protocols – mainly cycling or running models – have been shown to induce adaptations in skeletal muscle that enhance the capacity for both oxidative and non-oxidative metabolism [6–9]. As little as sessions of HIT over 2 weeks can increase the content of mitochondrial enzymes (fig. 1), alter substrate metabolism (such that muscle glycogen is used more ‘efficiently’ during exercise), and improve buffering capacity [6, 7]. Much of this work has been conducted on recreational athletes, and while short-term HIT can also improve performance in high-trained subjects, the precise mechanisms responsible are less clear [9]. It has been suggested that training-induced changes in Na\textsuperscript{+}/K\textsuperscript{+} pump activity may help to preserve cell excitability and force production, thereby delay fatigue development during intense exercise [8].

Recent data suggest that very intense HIT protocols may indeed provide a sufficient stimulus for mitochondrial adaptation in trained individuals. Pnilander et al. [10] reported that a single bout of low-volume HIT (7 × 30-second all-out efforts) stimulated increases in mitochondrial gene expression that were comparable to or greater than the changes after more prolonged efforts (3 × 20-min bouts at ~87% of VO\textsubscript{2peak}) in well-trained cyclists. Notably, of the two interventions, only the 30-second protocol stimulated an increase in mitochondrial transcription factor A, the downstream target of peroxisome-proliferator activated receptor-γ coactivator-1α, which is regarded as the master regulator of mitochondrial biogenesis in muscle [11]. The authors concluded that brief ‘su-

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**Fig. 1.** Maximal activity of cytochrome oxidase (COX) measured in resting muscle biopsy samples obtained before and after 6 sessions of HIT or endurance training (ET) over 2 weeks. *p ≤ 0.05 versus pre-training (main effect for time). Redrawn from Gibala and McGee [6] with permission.
pramaximal’ type interval training might be a time-efficient strategy to promote skeletal muscle adaptation in highly trained individuals. In this regard, Laursen [12] proposed that a polarized approach to training, in which ∼75% of total training volume be performed at low intensities, with 10–15% performed at supra-maximal intensities may be the optimal training intensity distribution for elite athletes who compete in intense endurance events.

**Potential Nutritional Strategies to Alter HIT Adaptation**

Guidelines are available regarding the appropriate selection of food and fluids, timing of intake, and supplement choices [13], including recommendations by sports nutrition experts specifically tailored to team sport players [14, 15] and those who engage in power sports [16]. The general consensus is that athletes should consume a high-carbohydrate diet (6–12 g/kg per day) in order to maximize muscle glycogen availability and meet the energy demands of training and competition. However, there is also evidence to suggest that periodic training with reduced carbohydrate availability may augment HIT-induced adaptations in skeletal muscle [17], including one study that applied the ‘train low’ theory to a team sport model. Morton et al. [18] studied three groups of recreationally active men who performed 6 weeks of high-intensity intermittent running. Two groups of subjects trained twice per day, 2 days per week, such that half of their training was performed in a glycogen-reduced state, whereas the third group trained once per day, 4 days per week, under conditions of high carbohydrate availability. One of the ‘low’ groups received a carbohydrate drink prior to the second training session, whereas the other group received a non-energetic placebo. The most intriguing finding was that training under conditions of reduced carbohydrate availability (i.e. the low group without the carbohydrate drink) provided an enhanced stimulus for skeletal muscle adaptation, such that the increase in oxidative enzymes was superior compared to the two carbohydrate-supplemented conditions. However, the training-induced improvements in a high-intensity intermittent running test were similar among all three groups of subjects. Thus, while periodic training with reduced glycogen availability stimulated greater muscle adaptations, this did not translate into improved performance. In the current volume, Hawley [pp. 1–14] considers in greater detail the potential effects of manipulating carbohydrate availability on training adaptation and performance.

Nutrient availability is a potent modulator of many acute physiological responses to exercise, including various molecular signaling pathways that are believed to regulate cellular adaptation to training [19]. While most studies have
focused on the response to traditional endurance or resistance exercise, several recent investigations have reported that dietary manipulation can alter the acute molecular signaling response to high-intensity exercise. Guerra et al. [20] examined AMP-activated protein kinase (AMPK) signaling after a 30-second all-out cycling effort (Wingate test), which was performed in an overnight fasted state or following ingestion of a 75% glucose drink. Glucose ingestion blunted peak AMPKα phosphorylation 30 min after exercise and generally altered the acute signaling response during recovery, possibly via changes in circulating insulin concentration. Cochran et al. [21] reported that when two sessions of interval exercise were performed on the same day separated by several hours of recovery, manipulating food intake after the first (morning) exercise session influenced the acute skeletal muscle signaling response to the second (afternoon) session. Specifically, exercise-induced phosphorylation of p38 mitogen-activated protein kinase was higher when subjects ingested a non-energetic placebo drink during recovery as compared to a glucose beverage. The results from these two studies suggest that restricting carbohydrate availability can augment some acute signaling responses linked to mitochondrial biogenesis, consistent with the ‘train low’ theory described above. In contrast, Coffey et al. [22] showed that nutrient provision enhanced molecular signaling pathways linked to muscle growth after repeated sprint exercise. Specifically, ingestion of a carbohydrate-protein supplement was associated with coordinated increases in phosphorylation of the Akt-mTOR-S6K-rpS6 anabolic signaling cascade and myofibrillar protein synthesis during recovery, whereas no effect was observed when exercise was undertaken in the fasted state. The practical implication of this work is that manipulating food availability in close temporal proximity to high-intensity exercise can modulate the acute adaptive response of skeletal muscle, but the chronic implications – if any – of these short-term transient effects remain to be elucidated.

Several nutritional supplements have also been shown to acutely alter metabolism and enhance intermittent high-intensity exercise performance [14–16]. However, relatively little is known regarding the effect of chronic interventions, and whether supplementation over a period of weeks or months augments HIT-induced physiological remodeling and promotes greater performance adaptations. Theoretically, a nutritional intervention could augments the adaptation to HIT by (1) improving energy metabolism during acute high-intensity exercise (e.g. enhancing mitochondrial function), which could facilitate greater total work and an enhanced chronic training stimulus; (2) promoting some aspect of the acute molecular response to exercise (e.g. by increasing gene expression in recovery), which could lead to enhanced physiological adaptations over time, or (3) some combination of these two factors. A theoretical model by
which nutritional manipulation could augment adaptation to HIT by ‘optimizing’ the effect of successive training bouts is shown in figure 2.

Limited evidence suggests that two supplements – sodium bicarbonate and β-alanine – could potentially augment training adaptations by altering muscle buffering capacity. Edge et al. [23] reported that subjects who ingested sodium bicarbonate over an 8-week high-intensity intermittent cycle training program, matched for total volume, experienced greater improvements in time-trial performance compared to a placebo group. Biopsies revealed no differences in several measured metabolites, but the authors posited the sodium bicarbonate group may have experienced greater gains in muscle oxidative capacity. A study conducted on rats showed that chronic bicarbonate ingestion in conjunction with HIT was associated with greater improvements in skeletal muscle mitochondrial mass and mitochondrial respiration, possibly due to reduced hydrogen ion accumulation during training [24]. The chapter by Burke in the current volume [pp. 15–26] reviews in greater detail the practice of bicarbonate loading to potentially improve performance during high-intensity exercise.

β-Alanine is a non-proteinogenic amino acid and rate-determining precursor (along with L-histidine) for the synthesis of carnosine, a dipeptide that functions as a physiologically relevant pH buffer in skeletal muscle [25]. β-Alanine supplementation is an effective means to increase muscle carnosine content, with daily doses of 4.8–6.4 g shown to increase muscle carnosine content by ~50–60% after 4 weeks [25]. Chronic β-alanine supplementation for up to 10

Fig. 2. Theoretical model by which nutritional manipulation could augment adaptation to HIT by ‘optimizing’ the effect of successive training bouts. While nutrient availability is a potent modulator of many acute responses to exercise, at present there is little direct evidence to support the model (i.e. in terms of specific physiological adaptations to HIT that are altered by chronic nutritional manipulation).
weeks has also been reported to improve acute high-intensity cycle exercise capacity [26] and augment resistance training-induced gains in strength [27]. However, no studies have directly investigated the potential for chronic β-alanine supplementation to alter skeletal muscle adaptations to HIT.

**Conclusion**

Several nutritional strategies have been shown to acutely alter metabolism and enhance intermittent high-intensity exercise performance, but little is known regarding the effect of chronic interventions. Nutritional compounds that alter muscle pH such as sodium bicarbonate and β-alanine are potential candidates, but training studies are warranted to determine whether supplementation over weeks or months augments HIT-induced physiological remodeling and/or promotes greater performance adaptations in humans.

**Disclosure Statement**

The author declares that no financial or other conflict of interest exists in relation to the content of the chapter.

**References**

Questions and Answers

Question 1: Dr. Gibala, HIT seems to result in a rapid and fast response of training adaptations. Could you explain the mechanism for that?

Answer: There are a number of different mechanisms. We know though that HIT stimulates many of the same molecular signalling pathways that are activated after endurance type training. So, from a molecular standpoint, much of
the adaptive response appears quite similar. There are some differences and, for example, adaptations in fat metabolism appear to be a little bit slower. Given that HIT relies very heavily on carbohydrate for fuel, perhaps it is not surprising that the adaptations in carbohydrate metabolism are quite fast and the fat metabolism changes are a little more sluggish. That being said, within a few weeks of interval training, even of a very small volume, you can see increases in your capacity to oxidize fats in the muscle.

**Question 2:** Is HIT an exercise type for everybody?

**Answer:** Certainly any serious endurance athlete is already incorporating HIT into their normal training program. We have been looking at other populations including people with type 2 diabetes and metabolic syndrome. Even in these clinical populations, we see that they can perform interval training, and they can benefit. For example, 2 weeks of this type of training in type 2 diabetics lowered their 24-hour blood sugar levels, which we know is associated with positive health outcomes. In applying interval training to different groups, you need to properly monitor it and use some caution, but it can be widely applied to many different groups, and they can benefit.

**Question 3:** Are there any supplements that can further enhance the benefits of HIT?

**Answer:** In theory, any supplement that has been shown to acutely improve high-intensity performance could potentially augment adaptations to HIT, for example sodium bicarbonate, creatine or caffeine. Many of these supplements may be beneficial in theory, but the studies haven’t been done to look at chronic training adaptations. There is a little bit of evidence that sodium bicarbonate ingestion chronically may improve performance even when subjects do work matched-work HIT protocols. So, even though the two groups of subjects did the exact same HIT protocol, chronic sodium bicarbonate ingestion resulted in a further improvement in performance. The mechanisms for that are not clear, but there is some evidence from rat studies that sodium bicarbonate actually improves the function of the mitochondria, and that might be a potential mechanism.

**Question 4:** You told me that you are doing HIT for yourself. Are you also taking supplements or food products?

**Answer:** No, I do not regularly consume supplements except for the caffeine in my morning coffee!