



MUSCLE ENERGETICS DURING EXPLOSIVE ACTIVITIES AND THE POTENTIAL EFFECTS OF NUTRITION

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- High-intensity exercise performance is dependent on anaerobic energy production.
- Limitations of anaerobic energy provision are primarily related to the amount of energy that can be produced from high-energy phosphates and glycolysis.
- Anaerobic capacity can be increased if the contracting muscle mass is increased through training regimes that induce hypertrophy or through training regimes that distribute the work to more muscles and/or muscle fibers.
- Studies demonstrate that high-intensity interval training increases muscle buffer capacity and high-intensity exercise performance.
- It is well documented that increasing the content of creatine and phosphocreatine ("creatine loading") in skeletal muscle increases anaerobic performance especially during short periods of interval exercise. The improved performance is related to increased muscle contents of the high-energy phosphate phosphocreatine and increased muscle buffer capacity.
- Studies demonstrate that bicarbonate loading can improve performance during exhaustive exercise lasting between 1-7 min. Gastrointestinal distress has limited the use of bicarbonate, but repeated doses over several days prior to competition may reduce the problems.
- Studies demonstrate that muscle buffer capacity and high-intensity exercise performance can be improved by beta-alanine supplementation and increased muscle carnosine content.
- The improved performance after interventions that increase buffer capacity demonstrates that the acidosis associated with high glycolytic rates is an important factor in fatigue.

INTRODUCTION

Many sports include explosive activities where high-intensity exercise (HIE) occurs during a sustained period (e.g., sprint and middle distance events) or as interval exercise with short bursts of repeated HIE with intervening periods with low-intensity exercise (e.g., ice hockey, soccer, handball, basketball, etc.) (Sahlin, 2014). The rate of energy utilization is high during HIE and exceeds what can be derived from aerobic processes (Harris et al., 1977; Sahlin et al., 1976; Sahlin, 1978). Energy must, therefore, also be derived from anaerobic processes which have a higher power of ATP production than the aerobic processes. Anaerobic energy utilization will result in depletion of high-energy phosphates (mainly phosphocreatine) and accumulation of byproducts such as creatine, inorganic phosphate (Pi) and lactic acid (Harris et al., 1977; Sahlin et al., 1976). The mechanism of fatigue in these situations is multifactorial and is still a hotly debated topic (Fitts, 2016; Westerblad, 2016). However, there is strong evidence that metabolic factors limit performance during HIE and that energy deficiency and/or acidosis, related to high glycolytic activity and lactate accumulation, are involved (Debold et al., 2016). There are several links between acidosis and energy deficiency, and the relative importance of these factors in fatigue is therefore difficult to separate. Regardless, performance during HIE can be improved with interventions (training and/or nutrition) that improve cellular energy production and/or ameliorate the acidotic state in the muscle.

IMPROVING MUSCLE ENERGY STATE WITH TRAINING

Aerobic energy processes are mainly limited by the rate at which ATP can be produced (i.e., aerobic power or $\dot{V}O_2$). In contrast, anaerobic processes are mainly limited by the amount of ATP that can be produced (i.e., anaerobic capacity). Utilization of phosphocreatine accounts for about 30% of anaerobic capacity, whereas glycolytic activity with lactate production accounts for the remaining 70%. The proportion covered by phosphocreatine will be higher during interval exercise, especially when the recovery periods are short (< 4 min). This is explained by the rapid resynthesis of phosphocreatine during rest or low activity periods, but slower removal of muscle lactate during the recovery period (Harris et al., 1976; Sahlin et al., 1979).

Several studies have shown that muscle content of ATP and phosphocreatine (/g of muscle) is not changed by training (Nevill et al., 1989; Perry et al., 2008). However, increasing the contracting muscle mass by resistance training will increase the total amount of ATP-Phosphocreatine that can be used during exercise. Similarly, sprint training has the potential to alter muscle recruitment and/or fiber recruitment, thus increasing the contracting muscle mass (Johansen & Quistorff, 2003). A training-induced increase in the working muscle mass will also increase the distribution volume of lactate and thus enhance the amount of ATP that can be produced through glycolysis. Larger working muscle mass, due to hypertrophy or altered muscle/fiber recruitment, will thus increase anaerobic capacity and improve performance during high-intensity exercise.

The rate of phosphocreatine resynthesis is rapid with half of the utilized phosphocreatine resynthesized within 30 sec (Sahlin et al., 1979). Phosphocreatine resynthesis is dependent on muscle oxidative capacity and can be improved by training protocols that stimulate mitochondrial biogenesis. Several studies have documented improved rates of phosphocreatine resynthesis after endurance training (Johansen & Quistorff, 2003). Speeding up the rate of phosphocreatine resynthesis will increase the capacity of anaerobic energy production during interval exercise with short rest periods.

IMPROVING MUSCLE BUFFER CAPACITY WITH TRAINING

Lactate production is limited by the extent of the lactate-induced acidosis, i.e., the decrease in muscle pH. Muscle buffer capacity will ameliorate changes in muscle pH and increases in muscle buffer capacity (induced by training or nutrition) will increase the amount of lactate that can be accumulated in the muscle and thus increase anaerobic capacity and HIE performance. A number of studies have shown that high-intensity training can improve muscle buffer capacity in both untrained (Bishop et al., 2004; Sharp et al., 1986) and endurance trained subjects (Weston et al., 1997), whereas other studies show no effects (Bishop et al., 2009; Sahlin & Henriksson, 1984). High altitude training has also been shown to improve muscle buffer capacity (Mizuno et al., 1990). Muscle buffer capacity is determined by several components, of which the major ones are: phosphocreatine-Pi, protein, bicarbonate-CO₂ and carnosine (Sahlin, 1978). It is not known which component is influenced by training. Several methods are available to determine muscle buffer capacity but, due to the complexity of the measurements, none are free from criticism.

CREATINE INGESTION AND HIGH-INTENSITY EXERCISE PERFORMANCE

In a classic study, Harris et al. (1992) demonstrated that the muscle content of creatine and phosphocreatine could be increased by creatine supplementation. Oral creatine intake increases the blood creatine concentration and a portion is taken up by the muscle. Subsequently, a fraction of the creatine taken up by the muscle is transformed to phosphocreatine. The average total creatine content (TCr = creatine + phosphocreatine) in skeletal muscles is 120-125 mmol/kg dry muscle (Harris et al., 1992; Hultman et al., 1996). The increase in TCr following creatine loading is ~20% and the increase in phosphocreatine is ~10% (Hultman et al., 1996). However, there are large differences in the response between subjects with a more pronounced effect in subjects with low initial muscle content of TCr (e.g., vegetarians) and absent in subjects with already high initial contents of TCr. Moreover, there appears to be a ceiling for maximal TCr in human skeletal muscle of ~150-160 mmol/kg dry muscle.

It is well documented that creatine supplementation increases performance during HIE, especially during interval exercise (Balsom et al., 1993; Greenhaff et al., 1993; Harris et al., 1993). Due to the ergogenic effect of creatine, supplementation prior to or during a training

period can increase the training load and thus enhance the training adaptation. This may explain the increased gain in muscle strength when resistance training is combined with creatine loading (Maganaris & Maughan, 1998).

Mechanisms of the Ergogenic Effect of Creatine

The obvious ergogenic mechanism of creatine loading is that the increased muscle content of phosphocreatine increases the anaerobic capacity. During sustained HIE the 10% increase in phosphocreatine would, on average, increase the anaerobic capacity by ~3% (10% increase x 0.3 of anaerobic ATP capacity). The effect will be larger during interval exercise where phosphocreatine utilization becomes the more dominant process of anaerobic energy production. The reduced catabolism of adenine nucleotides during high-intensity exercise after creatine loading gives experimental support for an improved energetic status (Balsom et al., 1993).

Another mechanism, by which creatine loading can increase performance, is related to the important role of phosphocreatine-Pi in muscle buffering where phosphocreatine-Pi accounts for more than 50% of the total muscle buffer capacity. Muscle buffering is the first line of defense against the negative effects of acidosis. It can be calculated that the improved muscle buffering after creatine loading, together with the knowledge that glycolysis accounts for 70% of the anaerobic ATP production, can increase the anaerobic capacity by 3.5% (10% increase x 0.5 of muscle buffering capacity x 0.7 anaerobic ATP capacity). The combined effect of increased high-energy phosphate content and increased buffer capacity could on average increase anaerobic capacity by about 6-7%. However, due to the heterogenic response between subjects to creatine loading, one would expect that some subjects would benefit more, whereas others would not have any effect at all (creatine non-responders).

Increased Body Mass after Creatine Loading

Creatine loading is associated with a body mass gain of about 1 kg. Although it has been suggested that creatine loading might stimulate protein synthesis and muscle growth and account for this increase, the evidence is not convincing. The increased body mass after creatine loading is probably related to increased tissue water content due to an osmotic effect of increased contents of phosphocreatine and creatine. In subjects with otherwise stable body weight, the increased body mass may be used as a rough marker of the effect of creatine loading. The increased body mass may negatively affect performance in running and cycling and other sports where body mass influences energy demand.

Optimizing Creatine Supplementation

The recommendations for creatine loading were outlined in the original articles (Harris et al., 1992; Hultman et al., 1996). The first five days (4-6) include a loading phase with 20 g of creatine monohydrate ingested each day, distributed in 4 x 5 g doses dissolved in water, taken 4-5 h apart. The loading phase is followed by a maintenance phase with 2 g of creatine monohydrate ingested each day, which is sufficient to

maintain the elevated muscle creatine level (Hultman et al., 1996). The muscle uptake of creatine can be enhanced by exercise and by combining creatine supplementation with glucose. The basis for this is that elevated insulin stimulates muscle creatine uptake and that exercise increases the blood flow to the working muscles. About 30% of the ingested creatine during the first two days of loading is retained in the muscle and the remaining part is excreted in the urine as creatinine. Commercial products contain a variable content of creatine and in many cases the creatine content is much less than that stated on the package, making it wise to use products where the creatine content has been measured and verified by an independent accredited laboratory.

BICARBONATE SUPPLEMENTATION CAN IMPROVE BUFFER CAPACITY

Sodium bicarbonate (Na-HCO_3) has been used as an ergogenic aid for many years and most studies show a positive effect on performance during exhaustive exercise lasting 1-7 min (Linderman & Gosselink, 1994). Most studies have used acute ingestion of bicarbonate 1-3 h prior to competition, but since the effect of bicarbonate lasts at least 24 h, chronic ingestion over several days may be a better strategy to minimize gastrointestinal distress (Mc Naughton & Thompson, 2001). Supplementation with bicarbonate increases blood pH and bicarbonate concentrations but not in muscle, since the cell membrane is impermeable to bicarbonate. The $\text{HCO}_3^-/\text{CO}_2$ system is the major buffer in extracellular fluid and the suggested ergogenic mechanism is that the efflux of lactate and hydrogen ions from muscle is accelerated. As alluded to previously, the major problem, which has limited the use of bicarbonate, is the gastrointestinal distress, including abdominal pain and diarrhea, that is associated with supplementation. The problems may be reduced if the ingestion time is prolonged and if ample volumes of water are provided (Siegler et al., 2012).

BETA-ALANINE SUPPLEMENTATION CAN IMPROVE BUFFER CAPACITY

During recent years beta-alanine supplementation has been used to increase muscle buffer capacity and high-intensity exercise performance. The basic idea put forward by Roger Harris and colleagues was that the formation of muscle carnosine, which is a naturally existing dipeptide with ideal buffering properties, is limited by the availability of beta-alanine (Hill et al., 2007; Sale et al., 2010). Muscle carnosine accounts for about 7% of muscle buffering, and the higher concentration in more fast-twitch glycolytic fibers is consistent with a functional role as a buffer. This is further stressed by the extremely high content of carnosine in animals specialized in anaerobic work (e.g., whales) (Abe, 2000). Oral supplementation with beta-alanine for 10 weeks has been shown to double muscle carnosine content (Hill et al., 2007) and assuming a 7% increase in muscle buffering one would expect a ~5% increase in anaerobic capacity. Several studies have documented that beta-alanine supplementation can improve performance during HIE especially during exhaustive exercise lasting between 1-4 min (Hill et al., 2007; Hobson et

al., 2012). It should also be mentioned that the intake should be divided into at least four doses during the day to minimize side effects, which can include a significant skin flushing and tingling sensation ('paresthesia'), and optimize loading.

SUMMARY

High-intensity exercise performance is limited by anaerobic capacity, which can be improved by training and/or nutrition. Training can increase the contracting muscle mass through both hypertrophy (more muscle) or altered fiber/muscle recruitment (improved use of existing muscles). An increase in contracting muscle mass will increase the energy production through increases in phosphocreatine utilization, and glycolytic activity and lactate production. Training may also improve muscle buffer capacity, which is the first line of defense against lactic acidosis.

Nutritional interventions can add to training-induced improvements. Creatine loading will increase anaerobic capacity by elevating the muscle content of phosphocreatine and improving the muscle buffer capacity. Buffer capacity can also be improved through supplementation with beta-alanine and bicarbonate. The ergogenic effects of creatine, beta-alanine and bicarbonate will enable athletes to increase the training load and optimize the training adaptation, which may be as important as the direct performance effects of increased muscle anaerobic capacity and buffer capacity.

It is well documented that interventions that improve buffer capacity and anaerobic capacity also enhance performance during HIE. There is convincing evidence from studies in exercising humans that dietary interventions that reduce acidosis improve performance, providing strong support for the idea that lactic acidosis is an important factor in fatigue.

PRACTICAL IMPLICATIONS

- Athletes can improve performance and enhance training adaptations during explosive activities with creatine supplementation.
- The recommended guidelines are 20 g of creatine monohydrate, divided into four doses/day for five days, followed by a maintenance dose of 2 g/day. Loading can be improved by exercise and if glucose is administered together with the creatine.
- Bicarbonate ingestion will increase pH and buffer capacity in the blood and has documented positive effects on performance during exhaustive exercise lasting 1-7 min. The recommended dose is 300 mg/kg/day divided into several doses combined with fluid. The timing of bicarbonate ingestion should be > 3 h, or alternately during three to five days, stopping 12-24 h prior to the event. The frequent problems with gastrointestinal distress have limited the use of bicarbonate as an ergogenic aid and athletes are recommended to test the procedure before using it in important competitions.
- The ingestion of beta-alanine increases muscle buffering

capacity and there is evidence of improved performance during explosive activities. Based on the information available, the recommendations are 3-6 g/day for 10 wk. The intake should be divided into at least four doses during the day to minimize side effects, which can include a significant skin flushing and tingling sensation ('paresthesia'), and optimize loading.

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