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THE SUPPLEMENTATION OF CREATINE, MALTODEXTRIN AND LACTATE PRODUCTION DURING SPORT

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ABSTRACT
The aim of this study was to examine the metabolism of lactate. During high intensity exercise, the largest tract of ATP supply are the breakdown of creatine phosphate and degradation of muscle glycogen to lactic acid. Thus, reduction of glycogen and creatine phosphate contribute to the decrease in the anaerobic energy production and exercício.A fatigue performance is a decrease in muscle strength to maintain the power generation and the rate of relaxation, inducing alterations in the contractile characteristics of muscle and changes in electrical properties that generate malfunctions in the human neuromuscular system. Fatigue is extensively researched, but the exact mechanisms that lead to the changes it causes are still unclear. However, this fatigue is followed by a number of physiological and metabolic changes. It is concluded that blood lactate once again proves to be the best option for control of training. Moreover, the reduction of lactate demonstrates the use of smaller and larger glycolytic ATP-CP system via providing better performance.

KEYWORDS: glycolytic ATP-CP.

INTRODUCTION

During high intensity exercise, the major routes of ATP delivery are the breakdown of creatine phosphate and the breakdown of muscle glycogen to lactic acid. Thus, the reduction of creatine phosphate and glycogen contributes to the decline of anaerobic energy production and exercise performance (CALFEE & FEDALE, 2006). Fatigue is the decrease of the muscular capacity to maintain the generation of strength and the speed of relaxation, induction of changes in the contractile characteristics of the muscle and changes in the electrical properties that generate dysfunctions in the human neuromuscular system. Fatigue is highly researched, but the exact mechanisms that lead to the changes caused by it are still unclear. However, this fatigue is accompanied by a number of physiological and metabolic changes (SHAO & HATCHCOCK, 2006) During a race, at each step given by a competitor, a quantity of about 1019 molecules of ATP are converted to ADP adenosine diphosphate, with the corresponding transfer of energy to the muscle work; in a way, this would mean that a marathon runner could spend the equivalent of 75 kg of ATP during the race. As the athlete could not meet this demand for ATP, creatine phosphate (CP) promotes rapid regeneration of the molecule from ADP and inorganic phosphate. With increased creatine stores due to supplementation, ATP resynthesis can be facilitated by delaying fatigue and delaying glycolysis for a few seconds, which can mean much in competitive terms (GLAISTER et al., 2006). Creatine is a cheap and affordable option for most of these athletes. In this context, several studies seek to verify changes in muscle power (DOBGENSKI, 2006), in the various physical valences (SASAKI, 2005) and also in body composition (VOLEK, 2004) with creatine supplementation. Supplementation can improve muscle performance in three different ways: Increasing phosphocreatine (PCr) stocks, the most important form of energy for ATP regeneration in the first few seconds of intense exercise; optimizing PCr resynthesis during recovery and also by depression of adenine degradation (VOLEK, 2004). Consumption of some ergogenic resources may have been positive for doping tests, so they are vetoed by the International Olympic Committee (IOC). Creatine is not prohibited by the IOC, so its consumption is not considered as "doping". In sports, this substance was popularized at the 1992 Olympic Games in Barcelona, when British runner Linford Christie, who won the 100m gold medal, credited his victory to creatine consumption (HAWES, 1998).

Literature Review Metabolism of Lactate

Glucose accounts for approximately 99% of all sugars circulating in the blood, originating from the digestion of carbohydrates or from the breakdown of hepatic glycogen. The synthesis of glycogen occurs from the glucose in a process determined as glycogenesis. This glycogen is then stored in the liver or muscle and is used

when its breakdown forms glucose-1-phosphate through glycogenolysis (ROBERGS et al., 2004). Glycolysis begins when glucose-1-phosphate forms glucose-6-phosphate, a glucose molecule is degraded through enzyme-catalyzed reactions to release 2 molecules of pyruvate each. Among the many reactions that occur, the oxidation of glyceraldehyde-3-phosphate requires a hydrogen receptor - the NAD + coenzyme. Reduction of NAD + causes the release of reduced NADH coenzyme. However, the NADH formed in this step of glycolysis needs to be reoxidized, otherwise glycolysis would soon stop for lack of NAD +. Cells contain limited amounts of this oxidized coenzyme, and the inability to regenerate NADH in NAD + would leave the cell lacking the electron acceptor for glyceraldehyde-3-phosphate oxidation and the glucose energy release reactions would cease (CABRERA et al., 1999). The NADH generated by glycolysis in anaerobiosis can not be reoxidized by O2. NAD + therefore needs to be regenerated through other reactions, such as the reduction of pyruvate to lactate. The fate of 2 molecules of pyruvate under anaerobic conditions produces 2 molecules of lactate, catalyzed by lactate dehydrogenase. As the reduction of 2 molecules of pyruvate in 2 molecules of lactate regenerates 2 molecules of NAD +, the overall process balances and can continue indefinitely. The lactate formed can still be recycled, for example, in the liver, where it is converted to glucose again through the Cori Cycle (gluconeogenesis) (LEHNINGER et al., 2002). During the anaerobic glycolysis that occurs in type IIb skeletal muscle, when the amount of oxygen is limited, lactate is formed from pyruvate, to allow NAD + oxidation to occur and glycolysis to continue in the generation of ATP. This reduction of pyruvate to lactate is catalyzed by the enzyme lactate dehydrogenase. PIRUVATO + NADH + H + ⇓ ----- LDH -------- ◊ L-LACTATE + NAD + In an all-out sprint effort of 1 to 2 minutes, the glycolytic system is highly prompted by increasing the concentrations of lactic acid of 1 mmol / kg at rest to values greater than 25 mmol / kg. Concomitant to this occurs the acidification of muscle fibers that inhibits the degradation of glycogen by impairing the function of the glycolytic enzyme, and making it difficult to bind with the calcium of the fibers, preventing normal muscle contraction (ROBERGS et al. Lactic acid and lactate are not the same component. Lactate is any salt derived from lactic acid (C3H6O3), when the latter releases H +. Anaerobic glucose produces lactic acid, but it quickly dissociates and the salt formed is called lactate. During exercise with steady rhythm, oxygen is supplied to and used by the active muscles. Under these conditions the lactic acid does not exceed the rest values. However, if aerobic metabolism is insufficient, anaerobic glycolysis contributes to energy demands and forms lactic acid. Almost all of the lactic acid generated during anaerobic metabolism is buffered in the blood by sodium bicarbonate and the carbon dioxide released in this buffering reaction is expelled into the atmosphere as the venous blood enters the lungs (CABRERA et al. This metabolic acidosis is called the anaerobic threshold, that is, it is the maximum capacity of oxygen absorption during loads of resistance (WEINECK, 1989). According to the same author the threshold can be identified by an increase in blood lactic acid and corresponding reduction in blood pH. The anaerobic threshold may also be related to an increase in the ratio of respiratory changes (R) due to the release of excess carbon dioxide in the buffering process. This is due to a deviation in the linearity of the relationship between oxygen consumption and ventilation due to the powerful ventilatory stimulus provided by both the increase in acidity and by a release of carbon dioxide through the buffer. The lactate threshold occurs between 50 and 60% of the maximum oxygen consumption capacity (VO^2max) in sedentary or non-conditioned individuals. In the well-conditioned athletes, the anaerobic threshold is above 70% of the VO^2max. According to MAGLISCHO (1999) in 100m swimming tests (40s to 60s for young adult swimmers) the relative participation of each phase of energy metabolism occurs as follows: 25% allele anaerobic, 65% anaerobic lactic and 10% aerobic, being the distance of greater percentage of the lactic anaerobic metabolism. The percentage of fat metabolism for this distance is, according to the author, negligible. Thus, activities involving the anaerobic system may have benefits from creatine supplementation (KREIDER, 2003). The way this occurs is the fact that creatine supplementation raises both phosphocreatine and free creatine body stores, aiding in ATP resynthesis (WILLIAMS & BRANCH, 1999).

Lactate and Ph

Blood tests indicate training speeds, monitoring training progress, diagnosing training weaknesses, and comparing athletes. The anaerobic threshold localization tests measure the content of lactic acid after each series of time shifts at progressively faster speeds. To find the individual anaerobic threshold, which is defined when the accumulation of lactic acid is greater than its resynthesis (WILMORE & COSTILL, 2001), one has to find increases in blood lactate concentration where there is a break in linear increase progression. The individual anaerobic threshold does not propose a fixed value for its characterization, and can range from 1.3 to 6.8 mmol / L (McLellan & Jacobs, 1989). Intense muscle activity often results in the production and accumulation of lactate and H +, causing some impairment in energy metabolism and consequently reducing muscle contraction force. Under normal resting conditions, body fluids have more bases (bicarbonate, phosphate and proteins) than acids, leading to a tissue pH ranging from 7.1 in muscle to 7.4 in arterial blood. However, the upper and lower limits in arterial blood are values between 6.9 and 7.5, and these extremes can only be tolerated for a few minutes (PEYREBRUNE et al., 2005). In muscle fibers hydrogen is mainly buffered by phosphates. Any increase in free hydrogen in the blood stimulates the respiratory center, increasing ventilation and facilitating the binding of H + to the bicarbonate that will be removed in the form of

carbon dioxide. Excess H + is removed by kidneys that filter H + along with other metabolic degradation products (LEHNINGER et al., 2002). In speed exercises pH reduction and lactate accumulation occur because these products do not diffuse through the membranes of muscle fibers. During a maximum effort of 60 seconds (approximately 100m freestyle) the resting concentrations only normalize after 5 to 10 minutes, and it can be faster to remove if it is done actively at 50% of the VO2max. due to the greater blood flow helping the removal of these metabolites. Blood pH may remain altered 30 to 40 minutes after extreme exercise and lactate can be elevated up to 2 hours after maximal exertion (WILMORE & COSTILL, 2001). The temperature during exercise also causes changes in lactate concentration, the higher the heat, the greater the use of muscle glycogen and consequently the higher the lactate production, and the faster the fatigue will occur. .

The Supplementation of Creatine, Maltodextrin and lactate Production During Sport

In the United States alone, $ 100 million was spent on the purchase of creatine in 1998, and it is widely used among weightlifters, tennis players, fighters, swimmers, cyclists, basketball players and soccer players (CALFEE & FEDALE, 2006). However, high-intensity and short-duration physical activities benefit in a greater degree from creatine supplementation, since in these activities, speed and muscular power are decisive and the best performance is also associated with high levels of adenosine triphosphate (ATP) and phosphocreatine (PCr) (WILLIAMS, 1998). Actually the greatest effectiveness reported with creatine intake and athletic performance gain is found in activities that use the ATP-CP system (KREIDER, 2003). JONES et. al. (2004) define that the improvements in performance, due to creatine supplementation, are due to the increase in PCr, optimizing ATP resynthesis. They also point out that depletion of PCr and the reduction in the capacity of force production are closely linked. As for protein synthesis, no relation was found with creatine supplementation. The normal creatine concentration in skeletal muscle is 120 mmol / kg of dry muscle. However, the upper limit can reach 160mmol / kg (HARRIS et al., 1992). This may explain the increase in body creatine concentration with supplementation. Some studies with biopsies, analyzing intramuscular total creatine stores with a supplementation of 20 g of creatine monohydrate daily after 5 consecutive days, showed a 15% increase in phosphocreatine (PCr) and 20% increase in total creatine (TCr) (Greenley, 1996; Casey, 1996). A 20% increase in total muscle creatine concentration was observed after 6 days of supplementation of 20 g / day creatine (HULTAMAN, 1996). Supplementation protocols usually involve a saturation phase with 20g / day for 4 to 6 days, followed by a maintenance phase with 5g / day for 2 to 3 weeks. This regimen would elevate creatine stores to optimal levels, higher doses would be excreted in the urine, and lower doses would require more days to achieve the desired effect (PEYREBRUNE et al., 2005). Intake of creatine and 1g glucose / kg body weight increases creatine uptake by 9% in relation to non-carbohydrate supplementation (PREEN et al., 2003). The improvement in creatine transport occurs due to insulin response to carbohydrate ingestion (THEODOROU et al., 2005). In the case of physical valences, RAWSON & VOLEK (2004) found significant increases in muscle strength (1, 3 and 10 maximal repetitions) in supplemented athletes, 8% more compared to subjects who ingested only placebo (20% and 12% of force increase respectively). Another study conducted by BRANCH (2003) found an increase in lean body mass and muscle strength with creatine supplementation, a result similar to that obtained by NISSEN & SHARP (2003). In intermittent maximal efforts performed on cycloergometro (wingate) with men trained in creatine supplementation may also increase the total work capacity (ALTIMARI et al., 2006). VOLEK et. al. (2004) demonstrated that creatine supplementation can avoid losses in potency and strength capacity in the lower limbs, during a high intensity resistance training over a long period of time. ARCIERO et. al. (1998) reported gains in muscle strength even in the absence of training, demystifying the theory that increased body creatine stock would be linked to strength training. It is likely that individuals exposed to large amounts of training for extended periods and supplemented with creatine also show gains in strength-producing capacity. In an attempt to assert that creatine supplementation may promote improvement in longer exercise, STROUD (1994) tested individuals in 10 km of treadmill with values of 50% to 90% of maximal VO 2. Using 4g for 5 days did not observe significant differences in VO^2 max. and lactate concentration. This result suggests perhaps a low supplementation burden. MONETA (2003) evaluating rowers, through pre and post-test on rowing ergometer, with a 50W increase, with stages of 3 minutes until exhaustion, found that the supplementation of 20g of creatine during 5 days and training only of endurance, significantly increased the load on the lactate threshold compared to the placebo group. In addition, the study showed improvement in time to exhaustion, that is, the supplemented group was able to perform the exercise for a longer time than the control group. THEODOROU et. al. (2005) described comparative differences in performance of swimming athletes supplemented with creatine, but did not observe differences between the supplementation of with creatine. They also found a significant increase in total body mass in the group supplemented with creatine, but no change in muscle mass. Currently studies are going beyond the muscular and athletic abilities that come with creatine supplementation. Some studies point to differences in brain metabolism (PAN & TAKARASHI, 2006), another study reports improvements in some functions in patients with Parkinson's disease (BENDER et al., 2006). In the elderly creatine seems to improve the functional capacity in the lower limbs in the fast movements, possibly avoiding falls (CANETE et al., 2006). Even without limb movement, creatine seems to improve muscular energy

conditions as well as minimize weight loss, which is very useful in fracture immobilization processes, for example (SILVA & CONCELLIERO, 2006). In a recent study OLSEN et. al. (2006) found statistically significant differences in the number of satellite cells in healthy subjects supplemented with creatine compared to the placebo group and the protein-supplemented group who performed strength training for 16 weeks. In the hormonal secretion HOFFMAN et. al. (2006) found significant increases in testosterone with creatine supplementation. SHI (2005) presented significant reductions in the activity of creatine kinase, glucose and urea in athletes supplemented with creatine after competition, and the results were more effective when creatine was given in conjunction with maltodextrin. However OOPIK et. al. (2003), found increase in urea in rats supplemented with creatine. HUSO et. al. (2002) found significant increases in creatinine with standard creatine supplementation. However, they did not observe differences in nitrogenous urea. However, MILLER et. al. (2000) did not observe statistical differences in creatinine and pH when the same type of supplementation was used. In addition, the increase in the concentration of muscle PCr in the hydrolysis consumes H +, increasing approximately 7% in what could reflect in the buffering effect on the musculature avoiding a greater reduction of pH (BALSON, 1995). In Brazil, a study by MENEZES et. al. (2006) demonstrated that creatine supplementation may attenuate the deleterious effect of corticosteroids on body mass in rats. CONCLUSIONS Blood lactate once again proves to be the best choice for training control. Also, through the carbohydrate supplementation, the lactate reduction demonstrates the lower utilization of the glycolytic pathway and higher of the ATP-CP system, allowing better performance.

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