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Nutrition: Key factor in exercise recovery.

Nutrição: Factor fundamental na recuperação do exercício.

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Monografia

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Índice

Lista de Abreviaturas

VO_2max: Maximal oxygen uptake;

EIMD: Exercise induced muscle damage;

AOX: Antioxidant

CH: Carbohydrates;

GLUT: Glucose Transporter Carrier Proteins;

GI: Glycemic Index;

P: Protein;

CHP: Carbohydrate and Protein;

DOMS: Delayed Onset Muscle Soreness;

CK: Creatine Kinase;

LDH: Lactate Dehydrogenase;

AA: Amino Acids;

IL-6: Interleukin-6;

BCAA: Branched-Chain Amino Acids;

HMB: β-Hydroxy-β-Methylbutyrate;

ROS: Reactive Oxygen Species;

RNS: Reactive Nitrogen Species;

SOD: Superoxide Dismutase;

CAT: Catalase;

TBARS: Thiobarbituric Acid Reactive Substances;

TAS: Total antioxidant status

Resumo

A recuperação pós exercício é um desafio muito importante para os atletas, especialmente para aqueles que treinam ou competem várias vezes ao dia. Esta recuperação envolve vários processos que ajudam a restaurar a homeostasia. Alguns deles abrangem um conjunto complexo de questões nutricionais.

No final do exercício é normal ocorrer um certo grau de desidratação, por isso os atletas devem repor completamente os fluidos perdidos antes de iniciarem outro exercício. Está reportado na literatura, que para se encontrar um balanço de fluidos, é necessário ingerir uma quantidade de fluidos entre a 125 e 150% do peso do peso perdido. É também necessário que estes contenham uma quantidade significativa de sódio. Os factores que interferem com a ingestão de fluidos também estão retratados na literatura. O glicerol tem a capacidade de promover a retenção de fluidos, processo este investigado por alguns autores. O álcool e a cafeína exercem um efeito diurético, sendo por isso desaconselhado o seu consumo durante o período de recuperação.

O glicogénio muscular é a principal fonte de energia durante o exercício, sendo por isso um dos factores mais importantes na recuperação pós exercício e possivelmente o parâmetro mais bem estudado. Como os hidratos de carbono são o principal substrato para a síntese do glicogénio vários estudos investigaram a influência da quantidade, tempo, forma física, tipo e interacções com outros nutrientes. Alguns estudos avaliam a relação entre performance e o conteúdo muscular em glicogénio.

O dano muscular derivado do exercício é muito frequente, existindo na literatura vários trabalhos que investigam os efeitos de vários nutrientes na redução ou

prevenção do dano muscular e da dor bem como na manutenção da função muscular. Os nutrientes melhor estudados são os hidratos de carbono, as proteínas, os aminoácidos de cadeia ramificada e alguns antioxidantes como a vitamina C e E.

O exercício físico pode levar a uma diminuição da capacidade imunitária possivelmente devido a um aumento dos níveis da hormona do stress (cortisol), sendo por isso teorizado que estratégias nutricionais capazes de reduzir a resposta do cortisol ao exercício poderiam limitar a extensão da disfunção imunitária induzida pelo exercício. Os hidratos de carbono, os aminoácidos de cadeia ramificada, o glutamato e a vitamina C são os principais nutrientes abordados neste capítulo.

A hidratação e a regeneração do glicogénio muscular são os parâmetros mais abordados na recuperação nutricional do exercício. No entanto, alguns aspectos na recuperação do dano muscular e sistema imunitário continuam pouco conhecidos. Para além disso algumas estratégias nutricionais podem atrasar a recuperação, retardar a adaptação ao estímulo de treino e podem potencialmente ter efeitos negativos a longo prazo.

A recuperação nutricional do exercício continua a ser um tema de interesse para a comunidade científica. Futuras investigações são necessárias para compreender alguns tópicos de interesse nesta área.

Palavras-Chave: Hidratação; Glicogénio; Dano muscular; Imunidade; Hidratos de carbono; Proteínas; Antioxidantes.

Abstract

Exercise recovery is an important challenge, especially to athletes who train or compete multiple times per day. Recovery involves an array of processes that helps to restore homeostasis and some of them encompass a complex range of nutritional issues.

At the end of exercise, a degree of dehydration is normal and athletes should fully restore fluid losses before the next exercise bout. The literature report that, to achieve a fluid balance, is necessary drink a fluid amount equivalent to 125 to 150% of the body mass loss with a significant amount of sodium. The factors that influence fluid intake are also addressed in the literature. Due to the capacity of promote a fluid retention, glycerol is also addressed in the literature. Alcohol and caffeine have diuretic effects, so the consumption of these substances is discouraged during the recovery period.

Muscle glycogen is the principal energy source in exercise; due to this, its replacement is one of the most important issues in exercise recovery and this is possibly the most well studied parameter. Because carbohydrates are the mainly substrate for glycogen regeneration several investigations have studied the amount, timing, physical form, type and its interactions with other nutrients. Some studies have investigate the relationship between performance and muscle glycogen content,

Exercise-induced muscle damage is frequent. A lot of papers have investigated the effects of various nutrients in reduce or prevent muscle damage and soreness and maintain muscle function. The most well studied nutrients are carbohydrate, proteins, branched-chain amino acids and some antioxidants like vitamin C and E.

Exercise can lead to immune impairment in athletes, possibly due to an elevation of stress hormone (cortisol). It was hypothesized that nutritional strategies that effectively reduce the stress hormone response to exercise should limit the degree of exercise-induced immune dysfunction. Carbohydrates, branched-chain amino acids, glutamate and vitamin C are the principal nutrients addressed in this chapter.

Rehydration and restoration of muscle glycogen are the most addressed issues in nutritional exercise recovery. However, some aspects in the recovery from muscle damage and immune system remain somewhat nebulous. Furthermore some nutritional strategies may delay recovery, retard adaptations to training stimulus and can potentially have negative effects on long-term.

The issue of nutritional exercise recovery remains a topic of interest to the scientific community and further investigation are necessary to understand some interesting topics in this area.

Key-Words: Rehydration; Glycogen; Muscle damage; Immunity; Carbohydrate; Protein; Antioxidants.

Introduction

Successful athletic performance is a combination of favorable genetics, desire, proper training and a sensible approach to nutrition. Whether an athlete is recreational or elite, young or mature, the importance of nutrition as a contributing factor to success in training and competition has been recognized for decades [(1)]. The relationship between nutrition and sports has been established for a long time. For example, the ancient Greeks believed that high protein intakes were important for athletes, and, therefore, these athletes consumed diets that contained excessive amounts of meat. One case reported in the existent literature is Milos, a Cretan wrestler, who won gold medals at five Olympic Games and whose daily meat intake was reported to be 10 kg per day [(2-3)]. Such ideas still pervade today, especially with resistance based sports where athletes consume diets that have more than double the recommended levels of dietary protein. Research has clearly demonstrated that such excessively high protein intakes are not necessary to promote the adaptations that occur with resistance training. More recently, literature suggests that the timing of nutritional intake is also very important in optimizing the adaptations and recovery from both resistance and endurance exercise [(3)].

Nutritional intake is important for optimizing sports and exercise performance. Furthermore, good nutrition is important in optimizing adaptations to training [(3)]. Exercise recovery is an important challenge to athletes, especially to those who have strenuous training programs or compete multiple times per day [(4-5)]. Recovery from exercise depends on the nature of exercise, its intensity, duration and the time available for recovery [(6)]. Recovery involves a complex array of desirable processes that help to restore homeostasis or allow the body to adapt

to physiological stress. Some of these processes encompass a complex range of nutritional related issues including replacement of fluid and electrolytes, restoration of muscle and liver stores, regeneration, repair and adaptation processes following the catabolic stress and damage caused by the exercise. Replacement of fluid and electrolytes lost in sweat and restoration of glycogen stores are well studied but the repair and adaptation process following the catabolic state and damage remains somewhat nebulous [4, 7]. The same occurs with the recovery of the immune and antioxidant systems, important factors in exercise recovery, but less well documented [7].

Exercise is associated with high rates of metabolic heat production [8]. Depending upon the environmental conditions (temperature, humidity, sun, wind exposure) and clothing worn, exercise can induce significant elevations in body (core and skin) temperatures [9]. When body temperature is increased, the sweating response is invoked to promote evaporative heat loss. Sweat evaporation provides the primary avenue of heat loss during vigorous exercise in warm or hot environment. Sweat losses can be substantial, exceeding 2 liters per hour for prolonged periods. Besides containing water, sweat contains electrolytes that are lost and the resulting water and electrolyte deficit has major implications for exercise performance and thermoregulatory capacity. Besides dehydration can develop an adverse impact on the individual's exercise performance, it can inclusively result in a life-threatening situation [8-10]. Athletes who incur in substantial fluid deficits may experience some performance decrements associated with dehydration and result in degraded physical and mental performance, increased cardiovascular strain, changes in metabolism, and decreased heat tolerance [10-12]. Some individuals undertaking training twice a day,

or prolonged daily sessions of exercise in hot conditions, may also carry a fluid deficit from their previous workout into the next, because sweat loss generally exceeds fluid intake during exercise so a hypohydrated state at the end of exercise is normal [9, 13]. Ideally, the athlete should fully restore fluid losses and replenish energy substrate stores before the next bout of exercise is undertaken to minimize the detrimental effects on physiological function and subsequent exercise performance [7-8, 13].

An important goal of the athlete's everyday diet is to provide the muscle with substrates to fuel the training program that will achieve optimal adaptations and performance enhancements. Body fat and glycogen stores provide the major sources of exercise fuel. However, whereas fat sources are relatively plentiful, glycogen sources (plasma glucose and muscle glycogen) are limited [14]. Glycogen is a fuel of major importance for the support of the energy demands of muscle during intense physical activity [15-16]. It is commonly stated that muscle glycogen becomes depleted after 2 or 3 hours of continuous exercise performed at moderate intensities (60 to 80% VO_2max) [17-18]. Although this is true, it is not usually appreciated that muscle glycogen can also become depleted after only 15 to 30 minutes of intermittent exercise performed at very high intensities (90 to 130% VO_2max) [17-18]. These patterns of exercise are typical of many individual and team sports, so it is not uncommon for athletes to become somewhat glycogen-depleted by the half-time of a game or after a hard practice [17-18]. Post-exercise muscle glycogen repletion forms the most important factor in determining the time needed to recover and it is important to maintain performance capacity in athletes who train twice daily or those who need to compete on several consecutive days [16, 19].

When an individual performs unaccustomed exercise, particularly if the exercise involves high intensity or large amounts of eccentric contractions, considerable muscle soreness or damage can occur [20]. Consequently, exercise-induced muscle damage (EIMD) can have a profound effect on the ability to perform subsequent bouts of exercise and therefore adhere to an exercise training program [21]. A variety of interventions have been used prophylactically or therapeutically in an attempt to reduce the negative effects associated with EIMD and it has been suggested that nutritional interventions during the immediate recovery period following endurance exercise might attenuate muscle damage and soreness [5, 21]. This could have a profound impact for previously inactive individuals who become discouraged because of the discomfort that accompanies the initiation of an exercise program [5].

A single bout of exercise provides stress to the body's immune and antioxidant (AOX) status, with chronic exercise promoting beneficial adaptations in these systems [7, 22]. Unlike the situation with the previously presented issues in post-exercise recovery, specific nutritional strategies to promote or preserve optimal AOX and immune function in athletes are not well described. This is unfortunate, since illness is likely to have a marked effect on performance of many sports events [7].

The aim of this review is to provide summary of the evidence based nutritional strategies in promoting an optimal exercise recovery. For this purpose, we made an exhaustive review of the existent literature in the fields addressed in this paper.

Fluid and electrolyte replacement

Water is the key to life. It is the body's transport medium for nutrients, gases and waste products, and all biochemical reactions take place in an aqueous environment. The physicochemical properties of water assist thermal homeostasis by thermal conductance and latent heat of vaporization. It aids joint lubrication and gives the body structure and form [10, 23].

The metabolic heat generated by exercise must be dissipated to maintain body temperature within narrow physiological limits. When ambient temperature exceeds skin temperature, heat loss can occur only by evaporation of sweat from the skin surface. Significant rates of sweat production will also occur in a cool environment if the work rate is high. Sweat rates exceeding 2 liters per hour can be maintained for many hours by trained and acclimated people exercising in warm, humid conditions [8-9, 23-24].

The sensation of thirst indicates the desire to drink and therefore is important in the control of fluid intake and balance. Although thirst is a poor indicator of acute hydration status in humans, the overall stability of the total water volume indicates that the desire to drink is a powerful regulatory factor over the long term [23], so the thirst mechanism would not be sufficient to replace the fluids losses by sweat, leading to involuntary dehydration [25-28]. Even when fluids are available, the volume ingested is seldom sufficient to match the rate of sweat loss, and some degree of fluid deficit normally accompanies exercise. If not appropriately replaced, water and electrolytes imbalance can develop and adversely impact on the individuals exercise performance and perhaps health[9, 28]. Replacement of these losses must be achieved in the recovery period before the next bout of exercise is undertaken [8]. This is difficult when there is a moderate to

high fluid deficit (2 to 5% of body mass or greater) and the rehydration period is less than 6 to 8 hours [7].

Free exchange of water among body fluid compartments ensures that the water content of sweat is derived from all compartments. The distribution is influenced by sweat rate, sweat composition and total water and electrolyte losses. At low levels of body water loss, the water loss is derived largely from the extracelular space, as the extent of water loss increase, a greater percentage of the loss come from the intracellular space. The longer time taken to achieve the higher levels of sweat loss will have resulted in some redistribution of body water [12, 23, 29].

The major electrolytes lost in sweat are sodium and chloride, and it is clear that replacement of these ions, especially sodium, should be a priority. The concentrations of sodium lost in sweat is typical of about 40 to 60 mmol per litter, compared with about 4 to 8 mmol per litter for potassium [8, 23, 28-29]. Given the higher sodium loss and the distribution of these cations between the body water compartments, the primary water loss is likely to be from the extracelular space [23]. The ingestion of sodium during the fluid replacement period has some theoretical benefits and poses little or no risk. In fact, the addition of sodium to drinks in certain concentrations improves taste while also stimulating thirst, which appears to increase voluntary drinking and minimize the involuntary dehydration [28]. Compared with sodium and chloride, the sweat concentrations of other electrolytes are low and there is no good evidence for the inclusion of any other electrolyte in fluid replacement drinks [28, 30].

In an investigation conducted by Nose *et al.* [31] the results indicate that when plain water was consumed, it took three times longer for the restoration of

plasma volume following exercise-induced dehydration compared to consumption of same amount of fluid with 77 mmol per litter sodium chloride. The results indicate that, compared with the saline solution, plain water was less effective in restoring hydration status; not only did plain water reduce the stimulus to drink but the free water clearance was also increased, mainly due to the loss of electrolytes during dehydration. Therefore, it has been suggested that rehydration after exercise can only be complete and rapid if the sodium lost in sweat during exercise is replaced as well as the water. These results corroborate the results from Maughan *et al.* [32] and Merson and collaborators [33], where they investigate how moderate sodium chloride concentrations affect rehydration after exercise and subsequent exercise capacity. After exercise dehydration (1.98 ± 0.1% body mass loss) the participants ingested an amount equivalent to 150% of body mass loss of one of four drinks with different sodium contents. The results show that the addition of 40 or 50 mmol/l of sodium chloride to the rehydration beverage reduces the subsequent urine output, thereby providing a more effective rehydration than a sodium-free drink. Since these studies, we can say that if sodium losses are not replaced, a substantial fraction of the ingested fluid is lost as urine, because in order to replace body water, extracellular sodium losses also need to be replaced [11]. The ingestion of plain water in the post exercise period results in a rapid fall in the plasma sodium concentration and osmolality. Both these factors are important in determining fluid balance and have the effect of reducing the stimulus to drink (thirst) and of stimulating urine output [8].

Large-volume fluid intake immediately following activity increases urine production, whereas spacing the fluid intake in portions over long periods improves rehydration [11]. Because sweating and obligatory urine losses continue

during the rehydration phase, athletes must replace more than their post exercise fluid deficit to achieve fluid restoration. Shirreffs *et al.* [34] investigated the relation between the volume and the composition of fluids ingested in terms of rehydration effectiveness. They combined two different sodium concentrations (23 mmol/l and 61 mmol/l) with four different volumes (50%, 100%, 150% and 200% of body mass loss). The results found in this investigation suggested that both sodium concentration and volume ingested interacts to affect the rehydration process. They concluded that, during the recovery period, drink a greater volume of fluid than sweat loss must be necessary to restore fluid balance. On the other hand, if the sodium content of the beverage is not sufficiently high, it only results in an increased urinary output. Typically, a volume of fluid equivalent of 125 to 150% of the deficit must be consumed to compensate for these ongoing losses and to ensure that fluid balance is achieved over the first 4 to 6 hours of recovery [7, 34]. It is well demonstrated that important factors for rehydration purpose after exercise are the consumption of both an adequate volume of fluid, greater than the net deficit of the sweat volume lost, and the amount of sodium. Without both of these, rehydration will be neither rapid nor complete and maintained [30].

Milk is a potential candidate for an effective post-exercise solution, given its naturally high electrolyte content. Shirreffs *et al.* [13] compared the effects of milk with a sports drink and water and concluded that it can be effectively used as a post-exercise rehydration drink. A similar study done by Watson and collaborators [35] corroborate these results, reinforcing the concept that milk can be a rehydration drink for use after exercise by everyone except by those who have lactose intolerance. These studies show us that rehydration can be achieved with a drink that can commonly be found at home [13] and the same occurs with whole foods

and water [24]. Sproles *et al.* [36] and Maughan *et al.* [37] demonstrated an improved rehydration when the same amount of fluid was ingested from whole foods and water in comparison with a carbohydrate-electrolyte beverage and with water only. These improved results in rehydration can be attributed to the presence of additional nutrients and ingredients in foods and the value of a particular food in promoting hydration is likely the result of the interaction between its water content and the amounts of other constituents that affects the absorption, distribution and retention of water [24], for instance proteins.

Another important factor in the rehydration beverage is its palatability. It is imperative that the drink is palatable in order to ensure that large amounts of fluid can be consumed willingly. Individuals tend to drink more of beverages that find more palatable, like flavored drinks [38-39], when rehydrating after exercise [40]. The palatability can be profitably influenced by the presence of carbohydrate (CH) and these macronutrients accelerate water absorption in the small intestine by stimulating carrier-mediated water movement [41-42]. On the contrary, very high sodium concentrations can turn the beverage unpalatable [33]. The temperatures of drinks also affect the intake: very cold drinks may be regarded as the most pleasurable, and cool beverages are more likely to be consumed quickly and in large quantities [7].

Generally, it is believed to be useful to drink isotonic fluid that contains such sodium, potassium and chloride at concentrations near to those in body fluids. It has also been suggested that the intake of hypotonic fluid may exert a similar or more rapid effect [10, 43] on replenishment of body water because it is rapidly absorbed from the small intestine along an electrochemical gradient, and also the sodium concentration and osmolality of sweat is lower than the extracellular fluid

[10]. However, other study [44] reported no differences between hypertonic or hypotonic fluids in restoring fluid balance.

Glycerol is a naturally occurring 3-carbon alcohol that plays an important role in fat metabolism [45]. Its intake has been shown to increase total body water, improve thermoregulation during exercise in hot environment and prolong exercise endurance as result of a reduced dieresis [46]. It has been proposed that this altered fluid balance was result from a direct effect on the kidney, because the reabsorption of glycerol, in the proximal and distal tubes, occurs with simultaneous water reabsorption, similar to the aldosterone-Na^+ mechanism [46-47]. Surprisingly, although there is a vast amount of information on the role of glycerol as hyperhydration agent, very limited information is available on its effect in rehydration [47]. The first who investigate glycerol effects on rehydration was Scheett *et al*. [48]. They promoted a 3.1% exercise-induced dehydration in volunteers exercising in a hot environment and observed that, following glycerol-induced rehydration, time to exhaustion was significantly greater in the subsequent exercise bout and significantly increased plasma volume restoration when compared to water. However, urine output and total bodyweight were not different between treatments at any time during the 3-hour rehydration. These results corroborate the results from Kavouras *et al*. [47]. Interestingly, in this study the authors reported that glycerol had little or no major effect on fluid-regulating hormones. Magal *et al*. [49] compared the effects of glycerol *versus* placebo in rehydration after exercise-induced dehydration with a previously hyperhydration. They reported that at the end of the rehydration phase, plasma volume was greater in the glycerol trial, resulting in a greater fluid retention. According to the acceded literature, glycerol seems to improve rehydration; however more research

is needed to explain the mechanisms underlying fluid retention. Headaches and gastrointestinal complaints are not uncommon after consumption of glycerol and any benefits must be weighed against possible ergolytic effects [50].

Alcohol is commonly found in beverages that can be consumed by hypohydrated athletes [50-51]. Alcohol has a diuretic action by inhibiting vasopressin secretion and thereby promotes free water clearance and increased urine production despite a body water deficit [50, 52]. The degree of dieresis is proportional to the amount of alcohol consumption [52]. Because of this known effect, the consumption of alcohol during recovery period is discouraged, but there appears to be little evidence to support this. To our knowledge, only one study [52] has investigated the effects of alcohol consumption in the fluid replacement after exercise-induced dehydration. The authors administrated 4 drinks with different amounts of alcohol (0, 1, 2 and 4 % in a volume equivalent to 150% mass loss) in the recovery period. They concluded that drinks containing 4% alcohol tend to delay the recovery process. In a study [53], designed to evaluate the effects of alcohol in blood rheological variables during exercise recovery, was reported an increase in plasma viscosity and the authors hypothesized that may be related to alcohol-induced dehydration.

Caffeine is a drug that enjoys social acceptance and widespread use around the world [54]. The popularity of caffeine, as an ergogenic aid, has increased dramatically over the last decade [55]. Because of its widespread use as ergogenic aid and its ubiquitous availability, caffeine is no longer on the banned substance list of the international list of the International Olympic Committee [56]. Because of its diuretics effects, caffeine ingestion is discouraged during the rehydration process [50-51, 57]. González-Alonso *et al*. [58] reported that the ingestion

of a caffeinated diet cola was less effective in whole body rehydration when compared with water or a 6% carbohydrate-electrolyte solution. More recently Dias *et al*. [59] reported that hydration status 16 hours after dehydration did not change with chronic caffeine ingestion. However, these findings may result from the low levels of dehydration achieved with this protocol.

Restoration of muscle glycogen stores and performance

The competitive nature of sports today requires many athletes to cross-train and train multiple times per day. Moreover, many athletes may be required to compete in several different contests over subsequent days or even in the same day [60].

At low to moderate intensity exercise, the major source of fuel is the body fat (plasma free fatty acids derived from adipose tissues and intramuscular triglycerides) which is relatively plentiful. As the intensity of exercise increases the utilization of CH (plasma glucose derived from the liver or CH intake and muscle glycogen stores) as fuel source also increases, and at high intensities glycogen is the principal energy source [1, 14, 61]. The major source of fuel used by skeletal muscle during prolonged strenuous aerobic exercise is muscle glycogen and its importance as a fuel source cannot be overstated [16, 60]. In fact, the availability of CH as a substrate for muscle and central nervous system becomes a limiting factor in performance of prolonged sessions [14]. It is well demonstrated that muscle glycogen becomes depleted after 2 to 3 hours of continuous moderate intensity exercise, although glycogen can become depleted after only 15 to 30 minutes of intermittent high intensity exercise [17-18]. The restoration of muscle glycogen after heavy training or competition is the most important factor in determining the time needed to recover between intense bouts of exercise [16-19].

It has been demonstrated that aerobic endurance is directly related to the initial muscle glycogen stores, that perception of fatigue during prolonged intense exercise parallels the decline in muscle glycogen, and that after substantial glycogen depletion is necessary to terminate or reduce the exercise intensity [60, 62-64]. Furthermore, the increase in aerobic endurance after training is related to an increased muscle glycogen storage capacity, as well as its more efficient use [63, 65].

One of the most important aspects of recovery that can be influenced by nutrition is the synthesis of muscle glycogen to replace stores lost during exercise [66-67]. Because aerobic performance is tightly related to the content of muscle in glycogen, nutritional strategies have been developed to enhance glycogen synthesis during short term recovery in preparation for additional exercise [5-6, 60, 63, 68-69].

Glycogen re-synthesis begins immediately after exercise and occurs in a biphasic manner. The process of glycogen replacement after exercise is characterized by an enhanced glucose uptake into muscle, which early on appears to be noninsulin dependent, followed by a later period of increased insulin sensitivity. The first step in the pathway of muscle glycogen re-synthesis is the transport by facilitated diffusion of glucose across the muscle cell membrane, through the glucose transporter carrier proteins (GLUT), in particular the GLUT-4. Insulin binds to the receptor of GLUT-4, which is located intracellularly, promoting its translocation to the cell membrane. This translocation can also be promoted directly by muscle contractions, independently of insulin action [5, 66, 70].

This initial period is a rapid phase of glycogen synthesis which can proceed without insulin, and therefore is called insulin-independent phase, lasting only 30

to 60 minutes. The mechanism that contributes to muscle glycogen synthesis in this phase is the enzyme glycogen synthase, which is activated by the reduction in muscle glycogen, muscle contractions and insulin [5, 66]. Insulin is a stronger activator of muscle glycogen synthesis; this activation can occur first by increasing the rate of muscle glucose transport, providing substrate for glycogen synthesis, and secondly by activating glycogen synthase, which is the rate limit enzyme in the glycogen synthesis pathway [71].

Following this phase, muscle glycogen synthesis occurs in a much slower rate, called slower phase or insulin dependent phase, which can last for several hours and if high insulin levels and CH are available. This slow phase is characterized by a marked increase in sensitivity of muscle glucose uptake and glycogen synthesis to insulin [5, 66]. This increased insulin sensitivity is the result of exercise bouts. In this phase, it is suggested that the presence of insulin contributes to glycogen re-synthesis by extending the time period over which GLUT-4 are active, recruit more transporters and stimulating the activity of the glycogen sinthase [6].

As stated above, muscle glycogen content is closely related with aerobic performance, so for practical reasons is more interesting to study the effects of muscle glycogen repletion on performance. However, evidence that enhanced muscle glycogen repletion from optimal post-exercise nutrition unequivocally translates into performance benefits is surprisingly limited in the prevailing literature [5]. A study from Haub *et al*. [72] failed to prove that muscle glycogen repletion enhances performance. In this study, after 1 hour of recovery, the time to complete the 100 kj cycling test, in the second ride, was significantly increased in the placebo treatment, when compared with the first ride, but not in the CH

treatment. In a later research Haub and collaborators [73] concluded that CH ingestion after maximal exercise does not appear to influence subsequent short-duration maximal effort exercise in competitive cyclists. A study from Wong *et al*. [74] reported that ingesting a CH-electrolyte drink is more effective in restore the endurance capacity compared with the same amount of a placebo even though complete rehydration was achieved between treatments. However, the placebo was electrolyte free and glycogen replacement was not analyzed, so it cannot be said that the performance enhancement is related to muscle glycogen. These results are consistent with the results of a previous research from Fallowfield and collaborators [75].

The CH requirement of an athlete during recovery from a training session or race depends primarily on the duration and intensity of the previous event and when the subsequent workout will occur [76]. Muscle glycogen re-synthesis following prolonged exercise has, accordingly, been well investigated with respect to the amount [77-81], timing [64, 82-83], physical form [84] and type [85] of the ingested CH.

The amount of CH consumed is an important factor that affect muscle glycogen replenishment and it is not surprising that consuming CH immediately after exercise helps to re-synthesizes muscle glycogen more rapidly than when food intake is restricted [83, 86]. In order to maximize glycogen re-synthesis, is recommended that athletes consume, at least, 1 to 1.5 grams of CH per kg of body weight within 30 minutes and at 2 hours intervals after exercise up to 6 hours [57]. These feeding patterns have been shown to experience a greater rate of muscle glycogen re-synthesis than when supplementation is delayed by 2 hours [83]. Blom *et al*. [80] found that supplementing with 0.35 g of glucose/kg of body

weight per hour appeared to maximize muscle glycogen storage because they did not find differences between supplementing with 0.35 or 0,7g of glucose/kg of body weight. In a later research from Ivy *et al*. [81] the results suggest that, in order to maximize the glycogen storage, is necessary to supplement with 0.6 to 0.7 g of glucose/kg of body per hour. However, a more recently study by van Loon *et al*. [77] showed that when the rate of CH ingestion was increased from 0.8 to 1.2 g/kg of body weight per hour, resulted in higher muscle glycogen synthesis rates. In this study, they provide the CH supplement at intervals of 30 minutes and in the others studies [80-81] the supplement was provided at 2 hours intervals. The differences in these studies suggests that providing CH supplements at 2 hours intervals may not adequately increase and maintain blood glucose and insulin levels for 2 hours [83]. The most important findings of this study is that failure to consume CH in the immediate phase of post exercise recovery leads to very low rates of glycogen re-synthesis until feeding occurs [79]. These findings demonstrate the importance of nutrient timing in post exercise recovery.

Early feedings may be important when there is only 4 to 8 hours between exercise sessions, although it may have less impact over a longer recovery period [14]. In a study of Parkin *et al*. [64] no differences were found in the glycogen re-synthesis after 8 or 24 hours of recovery when a CH-rich feeding began immediately after exercise or was delayed for 2 hours. The results of Ivy and co-workers [83] show that the ingestion of CH immediately after exercise results in a great muscle glycogen re-synthesis when compared with the ingestion of the same amount and type but delayed for 2 hours. Several studies [77, 87-89] have reported high glycogen synthesis when large amounts of CH (1 to 1.85 g/kg/h) were consumed at frequent intervals of 15 to 60 minutes during a 3 to 4 hours recovery

period corroborating the results of van Loon *et al*. [77] stated above. Overall, it would appear that when the interval between exercise sessions is short, the athletes should maximize the effective recovery time by ingesting CH as soon as possible. However, when longer periods are available they can begin the ingestion of CH later as long as the total amount of CH reaches the recommendations of 8 to 10 g of CH per kg of body weight in the first day [14, 90].

The physical form of carbohydrate intake does not appear to affect glycogen synthesis [7]. In a study of Keizer *et al*. [91], the effect of liquid or solid CH-rich feedings on re-synthesis of glycogen after depletion was investigated. The results showed no differences in glycogen repletion after 22 hours of recovery. Furthermore, this result was corroborated by a later study from Reed *et al*. [84].

The type of CH [85, 89, 92] is also a well studied parameter because of the diverse abilities of different CH types to enhance blood glucose availability or increase insulin concentrations [7]. Conlee *et al*. [92] investigated the effects of glucose or fructose feeding on glycogen repletion and concluded that fructose is a poorer nutritional precursor for rapid glycogen repletion after exercise.

CH-rich foods diverge in functional, metabolic and nutritional characteristics which makes it difficult to establish a single recommendation for food choice [93]. The glycemic index (GI) is one characteristic of CH-rich foods that provides effective information in planning the appropriate nutritional strategies on CH supplementation in exercise [17, 19, 93]. Since glycogen storage is influenced by both insulin and a rapid supply of glucose substrate, it has been proposed that high-GI CH sources might enhance post exercise refueling at a greater extent that low-GI CH foods [67, 93]. Numerous studies have examined the influence of GI of CH on muscle glycogen during the post exercise recovery period [19]. Kiens and

collaborators [94] appear to be the first to study CH foods and muscle glycogen replenishment based on glycemic response, in the early 90's, and report that the consumption of a high GI-diet results in a greater glycogen replacement, during the first 6 hours of recovery, than a low GI-diet. A later study from Burke *et al*. [95] supports these findings. They observed that after a depletion trial, greater glycogen storage can be achieved during 24 hours of recovery by consuming a high-CH diet based in high-GI foods in comparison with the same amount of CH provided from a diet based on low-GI foods. An interesting result from this study is that the increases in plasma insulin and glucose concentrations were lower, over the 24 hours, than the magnitude of the increase in glycogen storage, so the authors could not explain the results based in alterations of glucose or insulin responses [93]. In a follow-up study, the authors [96] simulated the flattened glucose and insulin responses of low-GI diets by feeding the original high-GI recovery diet in a pattern of small frequent amounts. In this study, subjects received identical high-GI CH-rich foods during 24 hours of post exercise recovery. On one occasion the food was consumed in four large meals and on the other as 16 small snacks. Despite marked differences in glucose and insulin response over the recovery period, there was no difference in glycogen storage between treatments. It is possible that diets may have caused different rates of glycogen storage in the early phases of recovery and that both diets, providing 10 g CH/kg/day, reached or exceeded the threshold for glycogen storage [93]. The authors suggest that a mechanism other than lowered blood glucose and insulin concentrations needs to be found to explain the reduced rate of muscle glycogen storage reported in the previous investigation [95], after the consumption of low GI foods [96]. Furthermore, this data suggests that when total CH intake is adequate, manipulations of glucose

and insulin levels within physiological ranges are not critical in long-term or daily glycogen storage [93].

Research on including protein (P) with CH feedings during recovery in some ways parallels the research investigating the influence of high GI and low GI on recovery. Some works [71, 87, 97-98], but not all [82], reported higher insulin responses when a combination of CH and protein (CHP) was consumed than when CH was consumed alone [67]. Beets *et al*. [97] investigated the effects of CHP mixtures in recovery of endurance running capacity and reported that CH and CHP restored the running capacity with equal effect, despite the greater insulinemic response elicited by the ingestion of CHP. The null effect on performance, in this study, can be explained by the results of a previously research from Jentjens *et al*. [87] in which great levels of plasma insulin during 3 hours recovery did not result in a great glycogen repletion. Different results were found by Zawadzki and co-workers [71] which found that the consumption of CHP resulted in a greater plasma insulin response and higher rate of muscle glycogen storage when compared with the ingestion of CH alone. However, the treatments used in these studies were not isocaloric and research was criticized for that.

To overcome the limitation of studies with non-isocaloric treatments, some studies investigated the effect of CHP isocaloric supplements [99-103] matched for CH content [69, 104]. Ivy *et al*. [69] compared the effects of CHP supplements with an equal CH content and an isocaloric supplement in the muscle glycogen replenishment 4 hours after exercise. There were no significant differences in performance between treatments; however, muscle glycogen was significantly greater in the CHP treatment compared with the other treatments. These results were consistent with the results from another study [104] were the performance

after recovery was not affected by the ingestion of a CHP supplement or a matched CH content supplement neither by a isocaloric CH treatment. This study failed to investigate the glycogen replenishment and the treatments did not result in differences on insulin responses. Niles and collaborators [103] investigated the ergogenic effects of isocaloric CH (152,7 g CH) and CHP (112 g CH with 40,7 g P) drinks ingested after a glycogen lowering diet and exercise. At 90 minutes of recovery the CHP drink results in great insulin levels compared to CH drink. Furthermore, the run time to exhaustion was longer during the CHP trial compared with the CH trial. The authors concluded that a CHP drink following glycogen depleting exercise may facilitate a greater rate of muscle glycogen re-synthesis than a CH beverage, and improve exercise endurance during a second bout of exercise performed on the same day. However they do not have assessed the muscle glycogen repletion. A more recent study from Howarth and co-workers [105] reported that insulin response and muscle glycogen re-synthesis were not different between the ingestion of supplement with 1.6 g of CH/kg/hour or the ingestion of a supplement with 1.2 g of CH plus 0.4 g of P/kg/hour. Many studies [100-102, 106], but not all [99], reported an augment in performance due to CHP supplement during recovery. This augment was measured at different manners and at different times.

Many studies have investigated the effects of adding protein to carbohydrates but the results are conflicting, possibly due to the different methodologies used. John Ivy reported, in a review [60], that co-ingestion of P with CH will increase the efficiency of muscle glycogen storage when supplementing at intervals greater than 1 hour apart or when the amount of CH ingested is below the threshold for maximal glycogen synthesis.

Recovery from muscle damage and soreness

Exercise-induced muscle injury frequently occurs after unaccustomed exercise, particularly if the exercise involves a large amount of eccentric contractions [20]. Delayed onset muscle soreness (DOMS) has been described as damaged muscle tissue membranes combined with a secondary inflammatory condition [107]. Typical symptoms associated with DOMS are loss of strength, pain, muscle tenderness, stiffness, swelling and elevated blood levels of markers of muscle injury, like creatine kinase (CK) and lactate dehydrogenase (LDH) [107-108]. Symptoms can vary from mild muscle tenderness to severe debilitating pain. DOMS is a universal symptom familiar to most athletes and athletic performance is typically impaired when an athlete is sore [107].

Recovery is defined as the return to normal physiological conditions after fatigue [109] and including periods to recover between training bouts is important for subsequent muscle performance because appropriate recuperation improves muscle strength and prevents muscle fiber damage [110]. There are several modalities often used in attempt to mitigate soreness and inflammation, including rest, massage, stretching, ice baths, and, of course, nutrition [108]. It has been suggested that nutritional interventions during the immediate recovery period following endurance exercise might attenuate muscle damage and soreness. This could have a profound impact for previously inactive individuals who become discouraged because of the discomfort that accompanies the initiation of an exercise program [104].

Some studies [106, 111-112] investigated the effects of CH supplementation on muscle damage and soreness. Close *et al*. [112] compared the effects of high-CH and low-CH diets adopted for 48 hours on 30 minutes of downhill running. The

authors did not find differences in muscle soreness, CK, muscle function and other markers, despite the high-CH diet demonstrated a trend to provoke higher soreness and CK 24 and 48 hours post-exercise. Miles and collaborators [111] found similar results with a similar protocol. In another investigation Miles *et al*. [106] investigated the effects of CH intake during recovery from eccentric exercise on Interleukin-6 (IL-6) and muscle-damage markers. The results showed that the ingestion of CH did not influence the markers of muscle damage. However this investigation has some limitations and results have to be analyzed with caution. The positive effects of CH in attenuating muscle damage have been refuted by another investigation of Nelson and co-workers [113]. It seems that CH ingestion have little or no effect in attenuating signs and symptoms of muscle damage [21].

Several studies [114-118] documented an attenuation of indirect markers of EIMD when adding P or amino acids (AA) to a CH supplement. However, this findings have not been verified [119] with a protocol explicitly designed to elicit muscle damage [120]. Green *et al*. [120] failed to prove that CHP attenuates muscle damage, while other study [116] with the same timing of ingestion showed a reduction in the markers of muscle damage. Other studies [114-115, 117-118], where the consumption of the supplements occurs during and after exercise showed an attenuation of muscle damage markers. The moment of the ingestion of the CHP supplement was investigated by Whyte and collaborators [121] and they did not find significant differences between ingestion CHP before and after eccentric exercise.

As stated above, CH have little or no effect in preventing muscle damage, so the results of investigations using CHP can be attributed to P. The effectiveness of AA or protein supplementation on attenuating muscle function loss and elevation in muscle soreness and blood muscular proteins seems somewhat

equivocal at present [122]. Positive effects of AA supplementation have been claimed to be related to the branched chain amino acids (BCAA) [123], so the recent interest has focused on the effectiveness of BCAA in reducing the negative symptoms associated with exercise [122]. A study from Nosaka *et al*. [124] showed that an AA supplement was effective in reducing DOMS and muscle protein efflux; however these results only happened when the supplement was ingested in a fasted state 30-minute prior to exercise, immediately after exercise and over a four day recovery. A study from Greer *et al*. [125] investigated the effects of BCAA supplementation on markers of muscle damage after endurance exercise in untrained college-age men and reported attenuation in these parameters. To our knowledge, only two studies [122, 126] have investigated BCAA supplementation and recovery from intense eccentric exercise. The first was conducted by Shimomura and co-workers [126] and reported a reduction in muscle soreness after squat exercise with supplementation of only 5 g of BCAA. However, this research was conducted only in females and the BCAA supplement was ingested only once. A later study from Jackman and collaborators [122] investigated the effects of the ingestion of BCAA in the muscle damage and soreness, in males, after eccentric exercise. The results show that BCAA supplementation may attenuate muscle soreness. Further studies are necessary to elucidate the mechanisms responsible for the effects of BCAA supplementation in muscle soreness. However, a possible explanation is that BCAA may attenuate exercise-induced protein breakdown, while leucine may stimulate protein synthesis [126].

To investigate the effects of CH and P in the muscle recovery many investigators have used supplements prepared at laboratory. However, this is a non-natural situation and the results of these investigations have to be

extrapolated with caution to real life situations where food is ingested. A recent study that overcomes this situation was published by Gilson *et al*. [127] where the efficacy of chocolate milk as a recovery beverage following an increased training duration was assessed in intercollegiate soccer players. The subjects completed 1 week of training followed by 4 days of an increased training duration. After each day of increased training duration subjects received chocolate milk or a high-CH recovery beverage. The two treatments provide similar effects on markers of post exercise recovery over the 4 days period of increased training duration. However, serum CK was lower with chocolate milk treatment compared with high-CH treatment. The authors concluded that, despite the attenuated levels of CK in the chocolate milk treatment, other measures of muscle recovery remains similar and there were no differences in exercise performance between treatment beverages.

β-Hydroxy-β-Methylbutyrate (HMB) is a metabolite of the BCAA leucine and its ketoacid, α-ketoisocaproate. It has recently gained popularity as a dietary supplement, particularly among strength athletes because it has been proposed to have prophylactic effect in preventing muscle protein degradation and EIMD during intense physical training [21]. The evidence suggests that supplementation with HMB for more than 6 days before exercise may attenuate symptoms of EIMD [128-130]. This statement is based on studies examining subjects who are unaccustomed to eccentric exercise and there is some evidence that these benefits are not observed in well trained individuals [131].

It is alleged that frequent performance of high intensity or exhaustive physical exercise increases the susceptibility to injuries, promotes chronic fatigue and overtraining, likely due to the high synthesis of reactive oxygen species (ROS) [132-133] and reactive nitrogen species (RNS) [134]. Because exercise can increase

oxygen consumption by 10 to 15-fold, it has been hypothesized that long-term exercise produces a constant “oxidative stress” on the myocit and other cells [135-136]. This relationship between an apparently healthy act - exercise, and a series of reactions that can, among other things, damage various organ systems has been troublesome to many researchers [136]. The so-called “oxygen paradox” evoked by exercise, and prompted by Jenkins [137], state that “elemental and gaseous oxygen presents a conundrum in that it is essential for and potentially destructive to human life”.

Tissues trauma or inflammation, which results in the activation of phagocytic cells, such as neutrophils, have also been implicated in free radical production via activity of enzymes, such as NADPH oxidase [136]. The initial events caused by unaccustomed eccentric exercise that causes muscle damage elicits an inflammatory response in which phagocytosis and neutrophil respiratory burst results in the production of ROS, thereby imposing an oxidative stress upon the tissue. These ROS have been implicated in the secondary damage that follows the primary mechanical disruption [21]. However, the exact nature of the relationship between ROS production, EIMD and muscle soreness is unclear as highlighted in a review from Close *et al*. [138]. The majority of early studies investigated the potential for eccentric exercise to generate ROS and RNS, but these reactive species are also produced in response to concentric exercise [134].

Oxidative stress occurs due to an imbalance between oxidant production and the AOX capacity of the cell [135]. There are biochemical mechanisms in the body to defend tissues cells against free radical damage. Primarily these defense mechanisms consist of low molecular weight free radical scavengers (α-

tocopherol, ascorbate, β-carotene and glutathione) and enzyme systems (superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase) [139].

Despite the uncertainty surrounding of the exercise-free radical relationship, the relatively consistent findings of an increase in AOX activity in various tissues of trained subjects is highly suggestive of a protective adaptation to the habitual stress of exercise. It is also suggested that a bout of exercise may outstrip the inherent capacity of the protective endogenous AOX enzyme systems, particularly in the non-habituated exerciser, necessitating great protection. The findings of various research studies have suggested that this added protection could come in the form of an exogenous supplement [136].

The most well researched nutritional AOXs in exercising subjects have been the vitamins C and E [21, 136]. However, there are a lot of other nutritional AOXs like β-carotene, other carotenoids, flavonoids, lipoic acid, zinc, copper, selenium and others [57, 133, 140].

Vitamin C is a water-soluble vitamin and is probably the most important AOX in extracellular fluids, but is also effective in cytosol. Vitamin C is more abundant in tissues in which ROS production is more important. This phenomenon is defined as an adaptation against oxidative stress. In fluids vitamin C has the ability to neutralize ROS, inside cells, vitamin C reinforces the action of vitamin E and glutathione by regenerating their active form after they have reacted with ROS. Vitamin C supplementation has often been studied [133]. A number of studies have demonstrated attenuated muscle damage and soreness when high doses are administrated prophylacticaly [141-142]; however, other studies report no effect [143] or negative effects [144-145]. Because vitamin C is primarily located in the plasma and not stored in the active tissue to any great extent, raising plasma

levels of the AOX to coincide with increased ROS production, such as during the inflammatory phase of muscle damage, may potentially offer increased AOX protection at the time of peak oxidant production [146]. However, a study from Thompson *et al*. [147] reported that post-exercise supplementation with vitamin C for 3 days after an intermittent shuttle running failed to attenuate the increase in interleukin IL-6 response and did not affect any indices of muscle damage or lipid peroxidation. Furthermore, a study from Childs *et al*. [148] that provided vitamin C in combination with N-acetylcysteine or a placebo to subjects immediately after 30 eccentric contractions, and for the subsequent 7 days, found evidence of a pro-oxidant effect.

Vitamin E is a fat-soluble vitamin made up of several isoforms. α-Tocopherol is the most active and abundant form. Vitamin E has been called the most important chain breaking AOX because of its abundance in cells and mitochondrial membranes and its ability to act directly on ROS. Vitamin E interacts with numerous AOXs, such as vitamin C and lipoic acid. Those AOXs have the capacity to regenerate vitamin E from its oxidized form. Vitamin E plays an important role in cell membranes because it stops lipid peroxidation. Athletes often use vitamin E supplementation in order to prevent exercise induced ROS muscular damage and fatigue. Some studies [149-150] investigated the prophylactic effects of vitamin E supplementation; however, the results are often contradictory probably because of methodological differences such as vitamin status of subjects before studies, vitamin E supplementation amount, and frequency and duration or training level [133]. It has also been suggested that long-term supplementation or high doses can be pro-oxidative and exert potential negative effects [57, 145].

In a recent study from Teixeira *et al*. [145] the effects of 28 days supplementation with a multi-AOX supplement were investigated in kayakers. The results showed a significantly increase in the plasma α-tocoferol and β-carotene in the supplemented group, whereas the plasma levels of the placebo group remain unchanged. Despite these results, thiobarbituric reactive acid substances (TBARS), a biomarker of lipid peroxidation, and uric acid increased significantly after exercise independently of treatment group. Total antioxidant status (TAS) increased above the pre-exercise values, in supplemented group, after 28 days of supplementation. In both groups the CK activity increased, but decrease from begin to the end of supplementation period in the supplemented group. The authors concluded that multi-AOX supplementation do not confer protection against lipid peroxidation, muscle damage or inflammation and warn that further experimentations are needed to explore the hypothesis that acute AOX supplementation before damaging exercise can alleviate muscle injury and lipid peroxidation, whereas chronic supplementation may delay muscle recovery. According to their findings, they do not recommend AOX supplementation to prevent muscle injury and attenuate exercise induced oxidative stress in already well-trained subjects. This recommendation is made in basis that exercise itself may be already an AOX [151].

Immune system

The immune system protects against, recognizes attacks and destroys elements that are foreign to body. The immune system can be divided into two broad functions- innate immunity and acquired immunity, which works together synergistically. The attempt of an infectious agent to enter the body immediately activates the innate system. This first line of defense comprises three general

mechanisms: physical and structural barriers, chemical barriers and phagocytic cells. Failure of the innate system and the resulting infection activates the acquired system, which aids recovery from infection. Monocytes or macrophages ingest, process and present antigens to lymphocytes. This is followed by clonal proliferation of T and B lymphocytes that possesses receptors that recognize the antigen, engendering specificity and memory that enable the immune system to mount an augmented cell-mediated and humoral response when the host is reinfected by the same pathogen. Critical to the activation and regulation of immune functions is the production of cytokines, including interferons, interleukins and colony-stimulating factors [(152)].

Exercise can have both positive and negative effects on immune function and susceptibility to minor illness. The relationship between exercise and susceptibility to infection has been modeled in the form of a “J” shaped curve [(153-154)]. This model shows that moderate intensity may enhance immune function when compared with inactivity and that large amount of strenuous exercise may impair immune function. It has been reported that there is a 100 to 500% increase in risk of developing an infection following a competitive ultra endurance event [(154)].

A heavy schedule of training and competition can lead to immune impairment in athletes, which is associated with an increased susceptibility to infections, particularly upper respiratory tract infections. This exercise-induced immune dysfunction seems to be mostly due to the immunosuppressive actions of stress hormones such as adrenaline and cortisol [(155)], inhibition of macrophage and T-cell cytokine production, altered heat shock protein expression, and a fall in the plasma concentration of glutamine [(156)].

Nutritional deficiencies can also impair immune function; however, it is also true that excessive intakes of individual micronutrientes can impair immune function and increase the risk of infection [157-158]. Since exercise induced immune function impairment appears mainly to be caused by elevated concentrations of stress hormones, nutritional strategies that putatively reduce the stress hormone response to exercise should limit the degree of exercise-induced immune dysfunction [155].

CH supplementation and dietary modifications have been shown to influence cell distribution and in some cases cell responsiveness upon completion and during recovery from exercise. Leukocyte concentration has been reported to be lower upon completion of exercise when CH has been administered and this effect often persists into recovery. CH supplementation has been shown to attenuate the cortisol response to exercise, resulting in a smaller induction of neutrophils [159]. Henson *et al*. [160] reported lower levels of cortisol and lower monocyte and natural killer (NK) cell concentration after intense running and cycling. These results are consistent with the results of a study from Scharhag *et al*. [161] where the authors concluded that a CH beverage can sufficiently attenuate the exercise induced immune response especially in phagocytizing cells by the reduced release of cortisol. Green and co-workers [162] reported that the reduction of the impairment of T-lymphocyte function by CH appears to be independent of cortisol. However, it seems clear that low levels of cortisol results in a smaller induction of neutrophils [159].

NK cells and neutrophils represent one of the body's first lines of defense against invading microorganisms or pathogens. The distribution and function of these cells in response to exercise has been well documented. However, the

influence of CH on NK cell has not yet been confirmed [159]. Nieman *et al*. [163] reported that carbohydrate supplementation versus placebo reduce the concentration of NK cells and cortisol, but did not influence the NK cells activity following 2,5 hours of exercise. The distribution of NK cells during 6 hours of recovery was not different between treatments.

Beside the attenuation of cortisol, consumption of CH during exercise also attenuates rises in plasma catecholamines, adrenocorticotrophic hormone, growth hormone and cytokines (IL-6) [155]. Febbraio and co-workers [164] reported that glucose ingestion during exercise attenuate IL-6 release without decrease intramuscular expression of IL-6 mRNA.

CH feeding during exercise (30 to 60 g/hour) appears to be effective in minimizing some of the immune perturbations associated with prolonged continuous strenuous exercise and appears to be less effective for less damaging intermittent exercise. Pre-exercise feeding of CH does not seem to be very effective in limiting exercise-induced leucocytosis or depression of neutrophil function [155]. Furthermore, a study from Bishop and collaborators [165] concluded that when overall exercise intensity is moderate, and changes in plasma glucose, cortisol and immune variables are relatively small, it would appear that CH has only a minimal influence on the immune response to exercise [157].

BCAA participates in glutamine synthesis and it has been suggested that BCAA supplementation during exercise maintain the plasma glutamine concentration and may modify the immune response to exercise [156]. Bassit *et al*. [166] investigated the effects of supplementation with BCAA in triathletes and long distance endurance runners. The triathletes took the supplement during 30 days before and during 1 week after the competition and runners received the

supplement during 15 days prior to the competition. The results showed that after the exercise, the placebo group presented a decreased plasma glutamine concentration which does not happen in the supplementation group.

Vitamin C positively influences the immune system and reduces the incidence of infection after prolonged exercise by increase the proliferative response in T-lymphocytes and attenuating the stress-induced cortisol response [158, 167-168]. It was originally suggested that vitamin C is co-released with cortisol from adrenal gland or vitamin C must first be released from the adrenal gland in order to permit the synthesis of cortisol [169-170]. Hence, by elevating the plasma vitamin C supplementation could reduce exercise induced oxidative stress and/or reduce the concentration gradient between the adrenals and plasma, consequently reducing adrenal cortisol mobilization [170]. Some studies [171-173] have investigated the effects of vitamin C supplementation for a long period previously to the exercise and report some positive effects. However, only one study [167] has investigated the effect of acute and short term supplementation on cortisol, salivary immunoglobulin-A and upper respiratory tract infection susceptibility following prolonged physical activity. The authors reported no chances in salivary immunoglobulin-A and upper respiratory tract infection susceptibility but vitamin C supplementation can decrease post exercise cortisol. Despite the possible benefits of Vitamin C supplementation, the risks of excessive intake may overweigh any potential benefits. High doses of Vitamin C can cause DNA damage, suggesting a possible link to increased cancer susceptibility and other adverse effects [168].

It has been suggested that exogenous provision of glutamine supplements may be beneficial by maintaining the plasma glutamine concentration and

preventing the impairment of immune function after prolonged exercise [157]. Despite the positive results of the investigation made by Castell *et al*. [174], the results must be viewed with some caution because previous studies have indicated that glutamine supplementation during exercise does not prevent the exercise-induced fall in lymphocyte proliferation [175-176].

Critical analysis

From all nutritional issues related to recovery, rehydration seems to be the best studied. The amount, type, timing, composition, temperature and osmolality of the recovery fluid are all addressed in the current scientific literature, and the physiologic process underlying this issue is very well described. However, it seems that there is lack of information about the importance of other minerals than sodium to complete fluid replacement. The role of potassium in promoting intracellular hydration needs to be further described. It seems that fluid balance can be achieved with food and water, but only few studies have investigated this.

Another important and well-studied factor in exercise recovery is the repletion of muscle glycogen stores. Due to the requirement of glycogen stores to engage in physical activity, several nutritional strategies have been developed during the past years. The amount, timing, physical form and type of ingested CH are well defined and studied. It was hypothesized that augments in insulin result in augments in muscle glycogen restoration. However, some studies do not confirm this theory, so a mechanism must be achieved to explain these inconsistent results. Different methodologies were used to investigate the effects of CH or CHP in muscle glycogen replacement resulting in conflicting results. The results of these researches need to be analyzed with caution. Muscle glycogen content is related to the performance, however great glycogen replacements do not result

always in a greater performance. This is possible explained by an incomplete recovery of all parameters related to performance. Athletes are more interested in performance than in muscle glycogen content, so performance is an important factor that should be always analyzed when investigating nutritional strategies.

Muscle damage and soreness are important factors for athlete's performance. While CH do not seem to have effect on muscle damage and performance, CHP potentially have beneficial effect. However, the results from the literature used in this work are somewhat controversial. Research with similar methodology is needed to evaluate the effects of CHP, AA and BCAA in muscle damage and soreness. Most of the studies that evaluated the muscle damage used CK as a marker of muscle disruption, though this is not the best way to access the real extension of damage.

AOX status and immune function are important for the recovery process. However, the investigation in this area is quite limited. More studies investigating the impact of post-exercise supplementation are needed.

The most part of the studies that investigate nutritional strategies to enhance exercise recovery have some limitations like supplements used are not food, so they consist in an artificial situation and the exercise used in this investigations are simulations and this type of exercise does not correspond to the real sports practice.

Conclusion

It is well demonstrated that exercise recovery is tightly related to nutrition. Actually, an adequate nutritional is a key factor for properly recovery from exercise. The current requirement of the competition, where everybody trains hard

and everyone is good in their modality, nutrition makes the difference between win or lose.

The process of recovery needs nutrients to replenish the depleted stores, repair the damage, respond to the exercise-induced stress and adapt to the exercise stimulus. Timing, type, amounts and form of some nutrients are very important factors in this process. In some cases, there is no effective recovery until nutrients were supplied, and if the begin of the nutritional recovery is delayed it becomes more difficult to achieve an optimal nutritional status for perform another exercise bout.

In the end of this work, there is one question remaining which deserves future investigations: is the stress caused by exercise an unavoidable and undesirable effect or, on the contrary, it is required signal for the body to adapt to the training stimulus and develop protection mechanism? Future research should evaluate the effectiveness of whole floods in promote exercise recovery and restore performance for subsequent exercise bout. Furthermore, nutritional strategies should allow body to adapt to training stimulus, but it is possible that some strategies delay the recovery and more investigation is needed to address this topic.

The issue of exercise recovery nutrition remains a topic of interest to the scientific community and further investigation is necessary to understand some nebulous topics in this area.

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