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COMPREHENSIVE REVIEW

Dietary strategies to recover from exercise-induced muscle damage

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Abstract

Exhaustive or unaccustomed intense exercise can cause exercise-induced muscle damage (EIMD) and its undesirable consequences may decrease the ability to exercise and to adhere to a training programme. This review briefly summarises the muscle damage process, focusing predominantly on oxidative stress and inflammation as contributing factors, and describes how nutrition may be positively used to recover from EIMD. The combined intake of carbohydrates and proteins and the use of antioxidants and/or anti-inflammatory nutrients within physiological ranges are interventions that may assist the recovery process. Although the works studying food instead of nutritional supplements are very scarce, their results seem to indicate that food might be a favourable option as a recovery strategy. To date, the only tested foods were milk, cherries, blueberries and pomegranate with promising results. Other potential solutions are foods rich in protein, carbohydrates, antioxidants and/or anti-inflammatory nutrients.

Keywords

Food, inflammation, nutrients, oxidative stress

History

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Introduction

Exhaustive or unaccustomed intense exercise can cause muscle damage, which results in muscle soreness, temporary decrease in muscle force, oedema, inflammation and an increase of intramuscular proteins in blood (Howatson & Van Someren, 2008; Smith et al., 2008). It has been described that the combination of a novel type of exercise with eccentric contractions leads to the occurrence of a higher degree of damage (Howatson & Van Someren, 2008), and its severity is influenced by the type, intensity and duration of training (Schoenfeld, 2012). Although the energy cost is lower for eccentric contractions compared with concentric ones, for the same power output, the former can cause a large degree of muscle damage (Evans, 2000; Newham et al., 1983). Eccentric contractions have also been considered more damaging than isometric ones (Clarkson & Hubal, 2002). It is believed that this is due to the increased generation of tension as muscle lengthens, resulting in a higher load distributed amongst the same number of fibres that causes a higher load per fibre ratio (Clarkson & Hubal, 2002; Enoka, 1996).

One of the most undesirable consequences of exercise-induced muscle damage (EIMD), especially in practical athletic terms, is its negative impact on muscle function, namely the decrease in muscle force-generating capacity, which is seen particularly after exercises involving eccentric contractions (Cheung et al., 2003; McGinley et al., 2009). Injury-induced strength loss, due to eccentric contractions, starts immediately after the end of the exercise and, depending on the severity of damage, it may persist from several days (McGinley et al., 2009) to 5–6 weeks (Howell et al., 1993). Muscle strength may decline up to 40%–50% after the exercise (Howell et al., 1993; Ingalls et al., 1998), leading to a large deleterious impact on athletic performance. On the other hand, pain, tenderness, swelling and stiffness, typically appear only within the first 24–48 h after eccentric exercise, being its duration also related to the extent of the damage (Allen, 2001; McGinley et al., 2009). Given the delayed nature of these symptoms, they are altogether often called ‘‘delayed onset muscle soreness’’ (DOMS) (Allen, 2001). Although EIMD can have detrimental effects, it has also been proposed that the associated inflammation and increased protein turnover are essential for hypertrophic adaptation (Evans & Cannon, 1991). Schoenfeld (2012), in his recent review, concluded that there is theoretical rationale supporting that EIMD may enhance the accretion of muscle proteins, although it seems that muscle growth can also occur in the relative absence of muscle damage. Furthermore, there may be a threshold beyond which damage does not have further effect on hypertrophy (Komulainen et al., 2000), and that excessive damage, particularly due to its induced force loss, can impair athletes' ability to train, which would consequently have an detrimental impact on muscle growth (Schoenfeld, 2012).

Nevertheless, due to its consequences, EIMD can hinder the adherence to an exercise training programme (Howatson & Van Someren, 2008). So, the study of interventions that may help to reduce the negative impact of EIMD, in order to accelerate the recovery process, may play a significant role for the sports population. The most common strategies used to prevent and treat EIMD are nutritional, pharmacological, stretching, massage, electrical therapy, cryotherapy and exercise (Howatson &

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Van Someren, 2008). Regarding the nutritional approach, the existing review literature is almost null and not focused on practical recommendations.

Thus, the aims of this review are to briefly summarise the muscle damage process, focusing on oxidative stress and inflammation, and to describe how nutrition may be positively used to help recovering from EIMD. Although nutrition is believed to provide prophylactic and therapeutic effect in reducing EIMD (Howatson & Van Someren, 2008), this review will focus especially on its therapeutic effects, particularly during the recovery period. For this review, databases PubMed and Scopus were used and searches were performed up to March 2013. Combinations of the following keywords were used as search terms: ''muscle damage'', ''recovery'', ''oxidative stress'', ''inflammation'', ''exercise'', ''food'', ''antioxidants'', ''proteins'', ''carbohydrates'' and ''omega-3 fatty acids''. References of retrieved articles were used whenever they were considered relevant. Additionally, the book Nutrition (Insel et al., 2007) was used to search nutritional contents of food.

Muscle damage

Although the exact mechanisms responsible for muscle damage remain unclear, it is believed that both mechanical and metabolic pathways are involved (Torres et al., 2012) and that the magnitude of damage is influenced by the mode, intensity and duration of exercise (Bowtell et al., 2011). A damage model, divided into two general phases, has been proposed: (i) a primary damage that occurs during the exercise, involving mechanical and metabolic alterations (Ebbeling & Clarkson, 1989; Tee et al., 2007); and (ii) a secondary damage associated with the inflammatory response (Howatson & Van Someren, 2008). EIMD involves, therefore, a complex interaction of events (Trombold et al., 2011), which seems to include sarcomere disruption due to the high mechanical tension on the myofibril (Proske & Morgan, 2001), impaired excitation–contraction coupling related to altered intracellular calcium homeostasis (Warren et al., 2001), oxidative stress (Favero, 1999) and inflammation (Peake et al., 2005). These events will lead to structural damage of the skeletal muscle cells and degradation of cell membrane, resulting in fibre necrosis and, ultimately, in fibre remodelling (Howatson & Van Someren, 2008). The possible sources of oxidants and the inflammatory process associated with EIMD will be further developed in the next sections.

Oxidative stress

It is largely accepted that exercise can create an imbalance between oxidant and antioxidant levels, known as oxidative stress (Leeuwenburgh & Heinecke, 2001). This phenomenon is caused by the production of reactive oxygen species (ROS) both acutely, i.e. during exercise, and throughout the long-term response to the EIMD (Powers & Jackson, 2008). ROS play an important role as mediators of EIMD (Finaud et al., 2006; Sacheck & Blumberg, 2001). Although it is widely recognized that ROS lead to the increment of markers of lipid, protein and DNA oxidation, ROS actions regarding EIMD seem to be associated with the oxidation of critical redox-sensitive sites within skeletal muscle – see Powers & Jackson (2008) for comprehensive review. Beyond their negative impact, ROS can also lead to positive outcomes, as contraction-induced adaptive responses of muscle fibres and regulation of gene expression (Powers & Jackson, 2008; Powers et al., 2011b).

Several mechanisms have been proposed as potentially liable parties of ROS increment during exercise. The electron transport associated with the mitochondrial respiratory chain has been considered one of the major intracellular source of ROS during exercise (Di Meo & Venditti, 2001). It is known that around 0.15% of the consumed oxygen is not completely reduced to water in the respiratory chain (St-Pierre et al., 2002), being converted into superoxide ion ($O_2^{\bullet-}$) (Boveris et al., 1972) primarily in complexes I and III of the mitochondrial electron transport chain (Finaud et al., 2006). Another alternative cause for ROS production could be ischemia-reperfusion (Finaud et al., 2006). During exhaustive exercise, working muscles are the primary tissue to be supplied with blood, while other tissues may undergo partial ischemia due to the reduced blood flow (Vollaard et al., 2005). Additionally, during exercise performed at intensities at or above maximal oxygen consumption ($\dot{V}O_{2max}$), muscle fibres may experience hypoxia since oxygen supply is beneath the energy demand (Packer, 1997). The ischemia conditions trigger the conversion of xanthine dehydrogenase to xanthine oxidase, which upon reoxygenation of the hypoxic tissue produces $O_2^{\bullet-}$ (Gomes et al., 2012). However, this mechanism has been shown to happen only in few studies (Gomes et al., 2012). Other processes that may be involved in ROS production during exercise include: (i) auto-oxidation of haem proteins, namely haemoglobin and myoglobin (Mb) (Finaud et al., 2006), (ii) the activity of the enzyme nicotinamide adenine dinucleotide phosphate oxidase (NADPH oxidase) (Powers & Jackson, 2008), (iii) the phospholipase A_2-dependent processes (Powers & Jackson, 2008; Powers et al., 2011b), and (iv) nitric oxide (NO) synthase (Powers & Jackson, 2008). Additional exercise-related changes that might be involved in ROS production are: (i) the increase in catecholamines which can lead to ROS release during their metabolic inactivation (Clarkson & Thompson, 2000), (ii) the production of lactic acid that is able to convert $O_2^{\bullet-}$ into hydroxyl radical ($OH^{\bullet}$) (Clarkson & Thompson, 2000), (iii) the rise in muscle temperature and (iv) the increase in carbon dioxide (CO_2) (Arbogast & Reid, 2004).

Although ROS may mediate cell damage, exercise-induced cell damage can also stimulate ROS production, since during the inflammatory response to EIMD the infiltrated leukocytes (neutrophils and macrophages) can release ROS (Leeuwenburgh & Heinecke, 2001). Their oxidative burst involves the production of $O_2^{\bullet-}$ that can be rapidly removed by reaction with other free radicals or due to conversion to hydrogen peroxide (H_2O_2) by superoxide dismutase (SOD) (Hampton et al., 1998). Furthermore, neutrophils can convert H_2O_2 into hypochlorous acid, a highly potent oxidant, via myeloperoxidase (Vollaard et al., 2005). Myeloperoxidase, an enzyme expressed primarily by neutrophils, has been shown to increase following exercise, and may remain elevated for days (Childs et al., 2001). Additionally, it was demonstrated that the level of myeloperoxidase activity per neutrophil is increased by exercise (Suzuki et al., 1996). Moreover, activated macrophages are a rich source of nitric oxide (NO) and they can lyse muscle cells *in vitro* through nitric-oxide-dependent mechanisms (Nguyen & Tidball, 2003). In addition, the presence of muscle cells seems to induce a higher NO production by macrophages and their cytolytic capacity appears to be increased by the presence of neutrophils (Nguyen & Tidball, 2003). Similar to neutrophils, macrophages are also thought to play a major role in promoting muscle damage after muscle injury (Tidball, 2005). However, recent findings demonstrated that macrophages recruited by damaged skeletal cells exhibit a phagocytic and pro-inflammatory profile, which is rapidly converted to an anti-inflammatory phenotype (Arnold et al., 2007). This phenotype is associated with myogenesis and muscle growth (Arnold et al., 2007), suggesting that macrophages do not contribute to secondary damage. Other aspects of the inflammatory phenomenon resultant from EIMD will be developed in the subsequent section.

Inflammation

Intense physical exercise, especially eccentric exercise, triggers a rapid and sequential invasion of muscle by inflammatory cells which can persist for days to weeks (Leeuwenburgh & Heinecke, 2001; Tidball, 2005). White blood cells (WBC) are the major cellular mediators of inflammation (Cannon & St. Pierre, 1998) and their increased concentrations after EIMD are believed to be mainly due to the rise of neutrophils and monocytes/macrophages (Evans, 2000; Malm et al., 1999; Sacheck & Blumberg, 2001; Urso & Clarkson, 2003). Lymphocytes are also recruited during strenuous exercise but their count declines immediately after the end of the exercise (Pendersen & Toft, 2000). The inflammatory process is believed to be mediated (i) by neuroendocrinological factors, such as adrenaline, noradrenaline, growth hormone and cortisol (Pendersen & Toft, 2000) and (ii) by cytokines, namely pro-inflammatory cytokines as tumour necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β), and the inflammation-responsive cytokine IL-6 (Pedersen & Hoffman-Goetz, 2000). Although it is generally accepted that cytokines have a central role in the inflammatory process, the exact mechanism of action of each one remains unclear (Smith et al., 2008) (for detailed review see Cannon & St Pierre (1998)). Endothelial cells also play a significant role in regulating the inflammatory response, namely (i) by expressing leukocyte's adhesion molecules, which are determinant for the influx of neutrophils and monocytes, (ii) possibly by producing NO, which is vasodilator, and (iii) by secreting several cytokines as IL-1α and -1β, IL-6 and IL-8 (Cannon & St. Pierre, 1998). The local inflammatory process is afterwards accompanied by a systemic response known as acute-phase response, similar to what happens in an infection (Evans, 2000; Pedersen & Hoffman-Goetz, 2000).

Eccentric exercise results in a greater rise of neutrophil counts compared with concentric exercise, where both circulating and skeletal muscle neutrophils increase (Evans, 2000). Exercise causes demargination of neutrophils, increasing the circulating populations (Tidball, 2005). Within 1 h of increased muscle loading, neutrophils invasion begins and their concentrations can remain elevated for periods as long as 5 days (Fielding et al., 1993). The mobilization of neutrophils seems to depend on exercise intensity and to be mediated by the secretion of stress hormones, such as catecholamines, cortisol and growth hormone (Pendersen & Toft, 2000; Sacheck & Blumberg, 2001). Their function after infiltration in the damaged muscle is to phagocytise the necrotic myofibers and help to degrade cellular debris (Cannon & St. Pierre, 1998), by releasing proteases and oxygen radicals, as explained in the previous section, which can damage muscle even further and also other healthy surrounding tissues (Evans & Cannon, 1991; Leeuwenburgh & Heinecke, 2001; Tidball, 2005). This process is known as secondary damage (Smith et al., 2008). In fact, evidence has shown that administration of an antibody that blocks the respiratory burst and degranulation of neutrophils prior to a single eccentric contraction, led to a significant decrease in muscle damage (Brickson et al., 2003). Moreover, neutrophils are thought to be one of the most important players in the secondary damage since they are the immune cells that predominate in the injured tissue at the time when secondary damage occurs (Smith et al., 2008). Neutrophils may also magnify the inflammatory process via the release of inflammatory cytokines as IL-6 (Smith et al., 2008), IL-1β and TNF-α (Cannon & St. Pierre, 1998).

The inflammatory cell pattern changes within the first 24 h, with the number of neutrophils starting to decrease and the macrophages count beginning to increase (Smith et al., 2008). Thus, macrophages are evident in the damaged muscle around 1 day after exercise and their counts may remain high up to 7–14 days post-exercise (Round et al., 1987). There is a poor understanding of macrophages' functions during exercise-induced inflammation and their role in muscle damage is complex, since they secrete various growth factors, cytokines and free radicals, and act as antigen-presenting cells, regulating the cellular immune response (Tidball, 2005). Some of the cytokines released by macrophages include IL-1β, TNF-α and IL-6, which magnify the inflammatory process and coordinate the various elements of the systemic acute-phase response (Sacheck & Blumberg, 2001; Smith et al., 2008). As neutrophils, they also have the ability to phagocytize damaged tissue and both seem to play a key role in muscle repair and remodelling (Tidball, 2005). The discussion of the mechanisms by which these inflammatory cells contribute to muscle repair and remodelling is beyond the scope of this review; for that, other review papers (Smith et al., 2008; Tidball, 2005) are suggested.

Nutritional strategies

Due to the heavy sport's schedule of athletes, training or competing more than once within a single day is oftentimes their routine. Therefore, maximising and accelerating the recovery processes is crucial to potentiate their performance (Betts & Williams, 2010). Some interventions have been proposed to reduce the negative effects associated with EIMD, like nutrition, pharmacological strategies, electrical and manual therapies, cryotherapy and active exercise (Howatson & Van Someren, 2008; Torres et al., 2012). With training programmes becoming more demanding any possible help should be considered, and nutrition is an area that obviously can make a difference (Maughan et al., 2004). Given the fact that feeding is a mandatory physiological demand, it is of countless interest to potentiate the athletes' food intake in order to maximise their training programme. Recovering faster and more efficiently will allow athletes to train more and to respond to training more positively, leading to the expected performance improvements. It has been widely stated that during post-exercise recovery, optimal nutritional intake is essential to facilitate muscle repair and regeneration (Beelen et al., 2010). Some nutrition interventions have been considered capable of assisting recovery after EIMD.

Proteins

Proteins alone

Few studies have been conducted that have examined the role of protein supplementation in preventing or alleviating symptoms associated with EIMD (Howatson et al., 2012; Jackman et al., 2010; Matsumoto et al., 2009; Nosaka et al., 2006; Shimomura et al., 2006), with the majority of them using branched chain amino acids (BCAA). It has been concluded that the ingestion of amino acids seem to be able to reduce muscle damage – measured by creatine kinase (CK) (Howatson et al., 2012; Matsumoto et al., 2009; Nosaka et al., 2006), aldolase (Nosaka et al., 2006), Mb (Nosaka et al., 2006), lactate dehydrogenase (LDH) (Matsumoto et al., 2009), granulocyte elastase (Matsumoto et al., 2009) or muscle soreness (Howatson et al., 2012; Jackman et al., 2010; Matsumoto et al., 2009; Nosaka et al., 2006; Shimomura et al., 2006) – decrease sensation of fatigue (Matsumoto et al., 2009; Shimomura et al., 2006), and accelerate the functional recovery process (Howatson et al., 2012). However, in one study (Jackman et al., 2010), no differences were found for CK, Mb, IL-6, maximal isometric strength and low-frequency fatigue compared with placebo.

It is widely accepted that a positive muscle protein balance is necessary to facilitate the muscle repair and adaptation from EIMD (Hawley et al., 2006). Regarding muscle protein synthesis

(MPS) and net protein accretion, it has been concluded that they can be promoted by an early post-exercise protein consumption (Phillips, 2011). For optimal stimulation of muscle protein synthesis, recent data suggest an intake of 20–25 g protein following resistance exercise (Moore et al., 2009; Phillips, 2011). Essential amino acids (EAA) seem to be the primarily responsible for the stimulation of muscle protein synthesis (Volpi et al., 2003) and leucine seems to be a key metabolic regulator of MPS (Crozier et al., 2005). The amount of protein mentioned above ($\approx$20 g) corresponds approximately to 8.5 g of EAA or 1.5 g leucine (Phillips, 2011), which is approximately the amount shown to maximally stimulate protein synthesis (Cuthbertson et al., 2005). Considering this, Phillips (2011) in his recent review suggests an ingestion of at least 25 g high-quality protein containing not less than 8–10 g EAA, delivered as soon as possible post-exercise, for maximal stimulation of MPS.

Proteins plus carbohydrates

Although it has been demonstrated that the administration of CHO alone has little or no effect in attenuating signs and symptoms of muscle damage (Howatson & Van Someren, 2008), the combined intake of CHO with proteins seems to be beneficial. There is already a strong body of scientific evidence showing that the simultaneous ingestion of CHO and protein may attenuate muscle damage (Baty et al., 2007; Bird et al., 2006; Cockburn et al., 2008, 2010; Doyle et al., 1993; Luden et al., 2007; Pritchett et al., 2009; Romano-Ely et al., 2006; Samadi et al., 2012; Saunders et al., 2004, 2007, 2009; Skillen et al., 2008; Valentine et al., 2008), suggesting that the combination of these two macronutrients can be a valuable strategy. However, some studies (Breen et al., 2010; Green et al., 2008; White et al., 2008; Wojcik et al., 2001) do no support these findings. The possible reasons for these discrepancies are (i) the inherent inter-individual variability for indirect systemic markers of muscle damage, namely CK (Betts & Williams, 2010), which was the only blood parameter used to assess muscle damage in the four studies that did not find positive results, and (ii) the different exercise protocols applied.

CHO ingestion after exercise has been shown to improve net protein balance by attenuating the exercise-induced increment in muscle protein breakdown, which has been attributed to a rise in plasma insulin (Beelen et al., 2010; Børsheim et al., 2004). However, when an ample dose of protein was administrated, the co-ingestion of CHO and proteins did not seem to further improve protein synthesis and protein breakdown (Koopman et al., 2007; Staples et al., 2011). Still, it is important to note that the amount of CHO used in those studies was considerably low: 0.15 or 0.6 g/kg/h (Koopman et al., 2007) and 50 g (Staples et al., 2011). Although conflicting data still exists regarding whether or not the ingestion of CHO plus proteins has an undoubtedly advantage versus protein alone, it seems clear that it is not a disadvantage combining these two macronutrients during the recovery time. Additionally, the palatability of a CHO-protein solution has usually a better acceptance than one with proteins only. Moreover, low glycogen levels have been shown to possibly have a negative impact on MPS (Churchley et al., 2007; Creer et al., 2005; Wojtaszewski et al., 2003) and to promote muscle protein breakdown (Lemon & Mullin, 1980). It is important to note that a high volume of resistance exercise can lead to a decrease in muscle glycogen stores (MacDougall et al., 1999; Robergs et al., 1991) and that muscle glycogen re-synthesis is impaired by EIMD (Costill et al., 1990; O'Reilly et al., 1987; Seifert et al., 2005). It is known that CHO feeding during the recovery period can stimulate greater rates of muscle glycogen re-synthesis than when no CHO are ingested at all (Betts & Williams, 2010). Furthermore, it has already been shown that a high CHO intake (8.5 g CHO/kg/d) after an eccentric exercise leads to a higher increase in intramuscular CHO storage compared to a lower amount (4.25 g CHO/kg/d) (Costill et al., 1990). Moreover, a recent review paper (Beelen et al., 2010) suggested that the co-ingestion of 0.2–0.4 g/kg/h protein with 0.8 g/kg/h CHO (compared to the recommended amount of 1.2 g/kg/h CHO alone), in addition to provide proteins that are essential to stimulate MPS, seems to result in optimal muscle glycogen-repletion rate (Beelen et al., 2010; Van Loon et al., 2000). This phenomenon may be due to the synergetic influence of CHO and protein on insulin secretion (Van Loon et al., 2000).

Therefore, and regarding the existing evidence, it seems that ingesting 0.8–1.2 g CHO/kg/h and 0.2–0.4 g protein/kg/h, preferably in the early recovery period, with a minimum content of 20 g high-quality protein, may enhance the recovery after EIMD. Some discussions have been raised recently regarding the importance of nutrient timing consumption (Aragon & Schoenfeld, 2013) and if CHO and proteins really need to be consumed as soon as possible after the exercise. Even though it is not yet certain that a real advantage for the early feeding after exercise exists, certainly a quick nutrient delivery after a demanding effort will not be a disadvantage. Furthermore, from a practical point of view, the promotion of an early nutritional recovery strategy, preferably carried out within the sports context, may enhance the adherence to that strategy and ensures a correct and proportional feeding.

Antioxidant supplementation

Whether or not athletes benefit from the use of antioxidant supplements remains a hot topic and it is still controversial. Powers and collaborators, in a recent review about this topic (Powers et al., 2011a), highlighted the arguments that have been used for and against antioxidant supplementation. Briefly, the most commonly used arguments to support antioxidant supplementation are: (i) the fact that exercise leads to an increase in ROS production and that increased levels of antioxidants could counteract the ROS, preventing or reducing damage and, therefore, muscle pain (Urso & Clarkson, 2003), (ii) that some antioxidants shown to improve endurance performance (Kelly et al., 2009) and to delay fatigue, and (iii) that some athletes may not achieve the nutritional recommendations for antioxidant intake just with food (Machefer et al., 2007; Palazzetti et al., 2004; Rankinen et al., 1998). On the other hand, some arguments have been used against antioxidant supplementation, namely: (i) the fact that regular exercise leads to an increase in enzymatic and non-enzymatic antioxidants in muscle fibres (Powers et al., 2011b), (ii) that antioxidant supplementation may impair muscle function or delay some adaptations induced by exercise (Coombes et al., 2001; Teixeira et al., 2009), by interfering with cell signalling functions of ROS, affecting muscular performance (McGinley et al., 2009), (iii) that antioxidant supplementation does not seem to lead to better outcomes, compared with placebo, regarding muscle function, inflammation (Beaton et al., 2002) and redox status (Theodorou et al., 2011) after eccentric exercise; (iv) that antioxidant supplementation may contribute to increase muscle damage and oxidative stress (Childs et al., 2001), and (v) that some studies do not support the concept that antioxidant supplementation is beneficial to human health (Bjelakovic et al., 2007) and doubts have been placed about the long-term effects of antioxidant supplementation in high doses (McGinley et al., 2009). Moreover, it has been reported that the protective effect of a diet, with natural sources of antioxidants, is not equivalent to the protective effect of supplementation (Halliwell, 2000). Given these facts, it is currently suggested (Peternelj & Coombes, 2011; Powers et al., 2011a) that due to the limited evidence to recommend antioxidant supplements, athletes should rather focus on consuming a well-balanced and energetically adequate diet, which can provide antioxidant-rich foods.

Antioxidant and/or anti-inflammatory nutrients in food

Keeping in mind that high doses of antioxidants seem to have detrimental consequences, the use of antioxidant-rich foods seems to be the best option. These foods can provide an amount of antioxidant within the physiological range, while nutritional supplements usually provide supra-physiological doses. Therefore, this review will only focus on the key antioxidants found in food and a brief explanation of their main mechanisms will be given below. Some of these compounds also seem to have an anti-inflammatory action, which may have further benefits on the recovery from EIMD. Given that, most studies regarding muscle damage used nutritional supplements rather than food, reference will be made not only to the studies that used nutrient-dense foods but also to those that utilized the isolate substances in humans. It is important to mention that the level of evidence regarding the cause and health effect relationship, for the majority of these substances, is not yet sufficient for the European Food Safety Authority (EFSA) to consider making health claims. Therefore, more research in this area, especially clinical trials with human beings, will help to understand the possible relationship between the intake of these substances and their possible effects.

Vitamin C and/or vitamin E

Vitamin C, or ascorbic acid, is a potent water soluble vitamin, present in the cytosolic compartment of the cells (Evans, 2000). It is found mainly in citrus fruits, with sweet peppers, strawberries, cruciferous and leafy vegetables, being also good sources (Gerber, 2003). This vitamin exerts its functions by scavenging ROS and reactive nitrogen species, as well as regenerating other antioxidant molecules from their radical species, namely vitamin E, β-carotene and glutathione (Carr & Frei, 1999).

Vitamin E is the most important lipid-soluble antioxidant vitamin and it is virtually found in all cell membranes (Evans, 2000). Its main sources are vegetable oils, especially sunflower, safflower and nuts (Gerber, 2003). It exists in eight different isomers: α-, β-, γ- and δ-tocopherol and α-, β-, γ- and δ-tocotrienol (Gülçin, 2012), being the α-tocopherol the most important biologically active form (McGinley et al., 2009). Vitamin E is known for its ability for stopping the progression of the lipid peroxidation chain reaction and also for acting as a scavenger of superoxide, hydroxyl and lipid peroxyl radicals (McGinley et al., 2009).

An exhaustive review (McGinley et al., 2009) about the effects of supplementation with these two antioxidants, alone or combined, concluded that there is little evidence to support its protection against muscle damage, although there is evidence showing that both can reduce indices of oxidative stress. Moreover, the typical large supplementation dosages can even has a detrimental effect on the adaptive and recovery processes since it may interfere with the signalling functions of ROS (McGinley et al., 2009). Studies after that review continue to show contradictory results, (i) some showing positive outcomes of supplementation concerning muscle soreness (Silva et al., 2010), muscle damage (measured trough LDH (Silva et al., 2010) and CK (Nakhostin-Roohi et al., 2008)), oxidative status (Nakhostin-Roohi et al., 2008; Silva et al., 2010) and inflammation (Silva et al., 2010), (ii) and others showing no effect on muscle damage using CK (Theodorou et al., 2011), muscle soreness (Theodorou et al., 2011), muscle function by isometric peak torque (Theodorou et al., 2011), inflammation (Nakhostin-Roohi et al., 2008; Silva et al., 2010), and oxidative status (Theodorou et al., 2011).

Polyphenols

Polyphenols are the biggest group of phytochemicals and are known for being strong antioxidants (Tsao, 2010). Flavonoids are the largest group of polyphenolic compounds with more than 4000 identified varieties distributed among fruits, vegetables, nuts, tea and wine (Cabrera et al., 2006; González-Gallego et al., 2010; Marzocchella et al., 2011). It has been suggested by a large number of publications that these compounds have immunomodulatory, antioxidant and anti-inflammatory properties (González-Gallego et al., 2010; Marzocchella et al., 2011). Flavonoids seem to exert their antioxidant activity by scavenging ROS (García-Lafuente et al., 2009) and by impairing ROS production by inhibiting NADPH oxidase, xanthine oxidase and myeloperoxidase (Cotelle, 2001). In addition, they also have the ability to inhibit lipid peroxidation, chelating redox-active metals, activating antioxidant enzymes and reducing α-tocopherol radicals (Heim et al., 2002). Some mechanisms have been proposed in order to explain the anti-inflammatory effects of flavonoids, as the reduction of the activities of the arachidonic acid metabolizing enzymes (phospholipase A2, cyclooxygenase, lipoxygenase), inhibition of NO synthase, inhibition of pro-inflammatory molecules (IL-1β, IL-2, IL-6, TNF-α, among others), and modulation of pro-inflammatory gene expression (Marzocchella et al., 2011).

The flavonoid quercetin has been used in some studies (Nieman et al., 2007a,b; O'Fallon et al., 2012) in the context of EIMD. Apart from a diminished post-exercise expression of leukocyte IL-8 and IL-10 mRNA in one of the studies (Nieman et al., 2007b), quercetin, contrary to the expected, failed to positively influence muscle strength, muscle damage, inflammation and plasma cytokine, and hormone levels. Another flavonoid, the epigallocatechin gallate (EGCG) that is a catechin found in high concentrations in green tea, was also investigated along with muscle damage (Kerksick et al., 2010). Its supplementation resulted in reduced muscle soreness compared to placebo. No differences were seen for the other tested parameters (peak torque production, LDH, CK, serum cortisol, neutrophil counts, neutrophil:lymphocyte ratio and markers of apoptosis). Regarding food, cherries and berries, known to be rich in various polyphenol compounds especially in anthocyanins (another class of flavonoids) and the flavonol quercetin (McCune et al., 2011; Szajdek & Borowska, 2008), have been used as treatment in studies related to EIMD (Bowtell et al., 2011; Connolly et al., 2006; Howatson et al., 2010; McLeay et al., 2012). Impressively, all studies showed positive results for muscle force recovery; regarding markers of muscle damage, most of the studies did not found any differences between the tested groups and the results for oxidative stress and inflammation markers varied among the studies (Table 1). Pomegranate and the respective extract are especially rich in the polyphenols ellagitannins (Medjakovic & Jungbauer, 2013), and have also been used in research concerning muscle damage. In a study (Trombold et al., 2010), pomegranate extract showed to reduce muscle force loss but had no impact on CK, Mb, IL-6 and CRP. The same research group conducted another study (Trombold et al., 2011) with pomegranate juice in which they concluded that the supplementation attenuated the force loss and reduced soreness of the elbow flexor muscles; however, no differences from placebo were found for the knee extensor muscles. The general positive results in these studies using food as the supplementation, strengthens the possible positive effect

Table 1. Studies that used food as the strategy to recover from EIMD in humans.

		Food			Muscle damage		Oxidative stress		Inflammation marker		Force/Performance recovery	
Study	Subjects	Dosage	Duration	Exercise protocol	Marker	Treatment effect	Marker	Treatment effect	Marker	Treatment effect	Test	Treatment effect (speed)
(Connolly et al., 2006)	14 males college students	2 × 355 mL cherry juice[a]	3 d pre-ex	2 × 20 single-elbow ECC flexion	Muscle soreness	↓					Isometric	↑
(Cockburn et al., 2008)	24 active males	500 mL semi-skimmed milk[b]	Immediately and 2 h after ex	6 × 10 ECC-CON actions knee flexors 1.05 rad.s^{-1}	CK DOMS Mb	↔ ↔ ↔					PT Total work	↔ Non-dominant leg ↑ Dominant leg ↔ Non-dominant leg ↑ Dominant leg
(Pritchett et al., 2009)	10 males regional-level cyclists and triathletes	Low-fat chocolate milk to achieve 1.0 g/kg[a]	Immediately and 2 h after ex	6 × (5 min cycling at 60% $\dot{V}O_2$ max + 3 × 10 s Wingate sprints)	CK Muscle soreness	↔ ↔					Cycling time at 85% $\dot{V}O_2$ max to exhaustion	↔
(Bowtell et al., 2011)	10 well-trained males	2 × 30 mL cherry juice[a]	7 d pre-ex	10 × 10 single-knee extension 80% 1RM	CK Nitrotyrosine PPT	↔ ↔ ↔	TAS PC	↔ ↓	hsCRP	↔	MVC	↑
(Howatson et al., 2010)	20 (13 male) recreational marathon runners	2 × 237 mL cherry juice[a]	5 d pre-ex	marathon run	CK LDH DOMS	↔ ↔ ↔	TAS TBARS PC	↑ ↓ ↔	CRP IL-6 Uric acid	↓ ↓ ↓	MVIC	↑
(Trombold et al., 2011)	17 trained males	2 × 250 mL pomegranate juice[a]	7 d pre-ex	3 × 20 single-elbow ECC flexion 0.7 rad.s^{-1} + 6 × 10 single-knee extension 110% 1RM	Muscle soreness	↓ Elbow flexors ↔ Knee extensor					Isometric	↑ Elbow flexor ↔ Knee extensor
(McLeay et al., 2012)	10 healthy females	1 blueberry smoothie: 200 g blueberries, 1 banana, 200 mL apple juice[a]	5 and 10 h pre-ex, immediately, 12 and 36 h after ex	3 × 100 single-knee ECC flexion 30°.s^{-1}	CK Muscle soreness	↔ ↔	PC ROS-GC FRAP	↔ ↓ ↑	IL-6	↔	Isometric ECC CON	↑ ↔ ↔
(Cockburn et al., 2013)	14 healthy males	500 mL semi-skimmed milk[a]	Immediately after ex	6 × 10 ECC-CON knee flexion 1.05 rad.s^{-1}	Passive DOMS Active DOMS CK Mb	Unclear Unclear Unclear Unclear					CMJ height Reactive strength index 5 m sprint 10 m sprint 15 m sprint Agility LIST	Unclear Unclear Unclear ↓ ↓ ↓ Unclear

Abbreviations: Pre-ex, pre-exercise; ECC, eccentric; ex, exercise; ECC-CON, eccentric-concentric; CK, creatine kinase; DOMS, delayed onset muscle soreness; Mb, myoblobin; PT, peak torque; $\dot{V}O_2$ max, maximal oxygen uptake; RM, repetition maximum; PPT, pressure pain threshold; TAS, total antioxidant status; PC, protein carbonyls; hsCRP, high-sensitivity C-reactive protein; MVC, maximum voluntary contraction; LDH, lactate dehydrogenase; TBARS, thiobarbituric acid reactive species; CRP, C-reactive protein; IL, interleukin; MVIC, maximum voluntary isometric contraction; ROS-GC, radical oxygen species-generating capacity; FRAP, ferric reducing anti-oxidant power; CMJ, countermovement jump; LIST, Loughborough Intermittent Shuttle Test.

↓, significant decrease; ↑, significant increase; ↔, no significant change.

[a]Comparison with a placebo/control group;

[b]The study has four groups: milk, milk-based carbohydrate-protein supplement, sports drink and water (control). The results are expressed for milk versus control.

of physiological ranges of antioxidants over the typically used supra-physiological doses.

Carotenoids

Carotenoids are present in plants, algae and microorganisms, and they are divided into two classes: carotenes and xanthophylls (Riccioni, 2009). The major dietary sources of carotenoids are fruits and vegetables (Semba et al., 2007), in which they are the principal pigments responsible for their colour (Gülçin, 2012). The most abundant carotenes in the diet are the β-carotene and lycopene while lutein, β-cryptoxanthin, zeaxanthin and astaxanthin, are the most common xanthophylls (Riccioni, 2009). Carotenoids are efficient antioxidants and comprise an important component of the antioxidant defence system in humans by protecting against oxidative stress, scavenging singlet molecular oxygen and free radicals, and inhibiting lipid peroxidation (Semba et al., 2007).

To date, there is only one study in humans (Djordjevic et al., 2012) using only carotenoids (astaxanthin) as the anti-oxidant supplementation treatment. This study showed a positive effect of the supplementation on CK and on the total antioxidant status (TAS), but not on SOD.

α-Lipoic acid

Dietary α-lipoic acid (LA) can be obtained from both animal and plant sources, but is primarily found in animal-derived foods, namely red meat, liver, heart and kidney (Gorąca et al., 2011). Humans can also obtain LA by *de novo* synthesis from fatty acids and cysteine (Biewenga et al., 1997). The reduced form of LA is known as dihydrolipoic acid (DHLA) and it is this form that predominantly interacts with ROS, although the oxidized form of LA can also inactivate free radicals (Packer et al., 2001). Furthermore, both forms may exhibit antioxidant activity by metal chelating (Packer et al., 2001). DHLA has also the capacity to reduce the oxidized forms of several important antioxidants, such as vitamin C and E, co-enzyme Q_{10}, and glutathione (Bilska & Włodek, 2005; Gorąca et al., 2011; Kozlov et al., 1999). For these reasons, the LA/DHLA redox couple is now being recognized as one of the most powerful biologic antioxidant systems (Gorąca et al., 2011).

Regarding LA, one study (Zembron-Lacny et al., 2009b) showed no differences on muscle damage markers CK and LDH between treatments. LA supplementation, on the other hand, influenced the levels of glutathione (GSH), glutathione reductase (GR) and glutathione peroxidase (GPx), after exercise; although the levels of total thiols, TBARS and protein carbonyls (PC), were positively changed throughout the trial compared with the control group, no different kinetics were seen between conditions with exercise. Moreover, no changes were seen after LA supplementation on the exercise parameters measured by the isokinetic device, namely peak torque, time to reach peak torque, total work, average power and maximal average peak torque. In another study (Zembron-Lacny et al., 2009a) by the same research group, similar outcomes were found: no differences in CK levels compared to control but positive results regarding TAS, total thiols, TBARS, PC and uric acid. In a third study (Fogarty et al., 2013), there was also an increase in blood total antioxidant capacity as a result of LA supplementation while DNA damage, lipid peroxidation and hydrogen peroxide, increased following exercise only in the non-supplemented group.

Co-enzyme Q10

Co-enzyme Q, also called ubiquinones, is a natural lipophilic compound found in every living cell (Pravst et al., 2010). Co-enzyme Q10 (CoQ_{10}) is the most abundant form in humans and most animals (Pravst et al., 2010). In addition to endogenous synthesis, food is also a source of CoQ_{10}, with meat, fish, nuts and certain vegetable oils, being the richest nutritional sources (Pravst et al., 2010). However, its dietary uptake is limited to only a few percentage (Bentinger et al., 2010). CoQ_{10} is a key component of the mitochondrial respiratory chain but it is also known for its antioxidant properties (Littarru & Tiano, 2010). Mostly in its reduced form, CoQ_{10} is an effective antioxidant with capacity to protect against lipid peroxidation, DNA and protein oxidation, and to regenerate vitamin C and E as well (Finaud et al., 2006; Pravst et al., 2010).

In one study (Díaz-Castro et al., 2012), the CoQ_{10} supplementation decreased oxidative markers, increased antioxidant markers and also had a positive effect on inflammatory markers. However, no markers of muscle damage or muscle performance were measured. In another study (Östman et al., 2012), the supplementation with CoQ_{10} had no effect on markers of oxidative stress and CK compared with placebo. In a third one (Kon et al., 2008), supplementation with CoQ_{10} decreased CK, Mb and lipid peroxide (LPO), and had no influence on changes in neutrophil counts after exercise. In a last one (Malm et al., 1996), anaerobic work capacity was impaired and CK levels were increased at various points in the CoQ_{10} group whereas during placebo trial there were no changes. It is worth mentioning that the American College of Sports Medicine (Rodriguez et al., 2009) classified CoQ_{10} as an ergogenic aid that does not perform as claimed.

n-3 Polyunsaturated fatty acids

The *n*-3 polyunsaturated fatty acids (PUFAs), namely the long-chain *n*-3 PUFA eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are extensively associated with anti-inflammatory and immunomodulatory properties (Galli & Calder, 2009). Rich sources of PUFA include fatty fish, such as salmon, tuna and mackerel, fish oil and nuts (Insel et al., 2007; Ros & Mataix, 2006).

Typically, the phospholipids of immune cells contain proportionally more arachidonic acid (AA), an *n*-6 PUFA, than other long-chain fatty acids, including *n*-3 PUFAs (Calder et al., 1994; Kew et al., 2004). Therefore, AA is usually the major substrate for eicosanoids synthesis, which is a key mediator and regulator of inflammation (Calder, 2009). The AA-derived eicosanoids, namely prostaglandin (PG) E_2 and 4-series of leukotrienes (LTs), are generally assumed to be pro-inflammatory, although it has been recently discovered that PGE_2 can also have anti-inflammatory actions (Calder, 2009). Nevertheless, it has been shown that the EPA and DHA content of the immune cells membrane can be altered through oral administration of these fatty acids (Calder, 2007). This manipulation results in a decreased production of AA-derived eicosanoids, a rise in alternative substances, such as PGE_3 (less potent then PGE_2) and resolvins (potent anti-inflammatory mediators), and also in an altered gene expression by a direct effect of *n*-3 fatty acids on signalling pathways (Calder, 2009). Through these mechanisms, *n*-3 PUFAs intake seems to have the ability to affect phagocytosis, T-cell signalling and antigen presentation capability (Calder, 2007) and to lead to a decrease in cytokines and ROS production, and in the expression of adhesion molecules (Calder, 2006).

For these reasons, *n*-3 PUFAs may be of a useful nutritional help to modulate the exercise-induced inflammation and immune dysfunction resultant from EIMD. A recent review about *n*-3 fatty acids and physical performance (Mickleborough, 2013) concluded that supplementation with *n*-3 PUFAs may help to alleviate DOMS resulting from muscle damage, possibly due to the ability of these fatty acids to increase blood flow. However, the author concluded that the evidence from human data is inconclusive to show a beneficial effect of n-3 PUFA in attenuating the inflammatory and immunomodulatory response to exercise.

Can food be an adequate alternative to supplements?

Most work, in the area of nutritional recovery from exercise, focus on the use of nutritional supplements rather than on foods. The few studies done using food – milk, cherries, blueberry or pomegranate – to recover from EIMD are described in Table 1. Although the number of studies is scarce and they used different methodologies, their results seem to indicate that food might be a favourable option as a recovery strategy. Moreover, given the issues related to potential contamination resulting in inadvertent doping (Burke et al., 2009), it is a safer option for athletes if they rely on food rather than on nutritional supplements.

Milk

Cow's milk and its derivatives represent a very good source of protein, lipids, amino acids, vitamins and minerals (Roy, 2008). Milk has several characteristics that make it an interesting recovery drink. One of its advantages is that it contains both casein and whey proteins in a ratio of approximately 3:1, which results in sustained elevations of blood amino acid concentrations (Bos et al., 2003; Roy, 2008). Therefore, milk has both fast dietary proteins (whey) that stimulate protein synthesis, and slowly absorbed ones (casein) which suppress muscle protein breakdown (Boirie et al., 1997; Dangin et al., 2003). Another advantage is that whey proteins contain a large proportion of BCAA, which are important in muscle metabolism and protein synthesis (Roy, 2008).

Milk was used in three (Cockburn et al., 2008, 2013; Pritchett et al., 2009) of the eight studies mentioned in Table 1. Essentially, the positive results were found regarding force/performance recovery: one study (Cockburn et al., 2008) found a positive effect of semi-skimmed milk on force recovery and total work for the dominant leg, and the second one (Cockburn et al., 2013) found a positive effect of the same type of milk on limiting increases in sprint time and agility. However, the third study (Pritchett et al., 2009), found no differences on the measured recovery parameters between low-fat chocolate milk and a carbohydrate replacement beverage, despite the previously published data suggesting it has an effective recovery aid (Karp et al., 2006). The three studies found no differences between the milk group and the respective comparison group for the muscle damaged markers, namely CK (Cockburn et al., 2008, 2013; Pritchett et al., 2009), DOMS (Cockburn et al., 2008, 2013; Pritchett et al., 2009) and Mb (Cockburn et al., 2008, 2013).

Cherries

Cherries are known to have a high content in numerous phytochemicals possessing antioxidant properties, with anthocyanins and quercetin playing a special role (McCune et al., 2011). These fruits also contain vitamins C and E and some carotenoids, especially β-carotene (Ferretti et al., 2010). In addition to the antioxidants effects, the consumption of cherries has also been associated with anti-inflammatory effects, namely through inhibition of the activity of the cyclooxygenase II (Ferretti et al., 2010; Seeram et al., 2001), and with pain inhibition (in animal studies) (Tall et al., 2004).

Three (Bowtell et al., 2011; Connolly et al., 2006; Howatson et al., 2010) of the eight studies using food as the intervention to attenuate the consequences associated with muscle damage, used cherries as the treatment. The results seen in these studies (Table 1) are, as a whole, positive and promising. For all the three studies, the cherry supplementation enhanced the performance recovery. One of the studies (Connolly et al., 2006) showed a decrease in DOMS, while the other two did not find any difference in the muscle damage markers. Howatson & collaborators (2010) found a positive outcome for inflammation (CRP, IL-6 and uric acid) and oxidative stress (TAS and TBARS) markers except PC, whereas Bowtell et al. (2011) only found a positive effect for PC (and not for TAS or high-sensitivity CRP).

Berries

Berries, specifically blueberries, are fruits particularly rich in antioxidants (Ehlenfeldt & Prior, 2001; Proteggente et al., 2002; Wang et al., 1996). It is believed that the phenolic compounds – including phenolic acids, tannins, namely ellagitannins and flavonoids as anthocyanins, flavonols and flavanols – are mainly responsible for their antioxidant properties (Basu et al., 2010; Szajdek & Borowska, 2008). Other substances, as β-carotene and other carotenoids and ascorbic acid, may also contribute to these properties but in smaller proportions (Szajdek & Borowska, 2008). Similar to cherries, berries also seem to have anti-inflammatory properties. They have been shown to reduced TNF-α induced up-regulation of inflammatory mediators in human micro-vascular endothelial cells (Youdim et al., 2002), to attenuate inflammatory gene expression in mice (DeFuria et al., 2009), and also to positively influence the NO metabolism (Basu et al., 2010; Pergola et al., 2006).

The only study (McLeay et al., 2012) in the context of EIMD that used blueberries as treatment showed positive results regarding oxidative stress and force recovery, but not for the muscle damaged parameters (CK and muscle soreness).

Pomegranate

Pomegranate was also studied (Trombold et al., 2011) with interesting and positive results. In this study, the ingestion of pomegranate juice was associated with the attenuation of weakness and reduction of soreness in the elbow flexor muscles. Pomegranate is considered a potent and unique polyphenol-rich food, containing mainly ellagitannins and their derived metabolites, which can protect against most types of free radical oxidants (Visioli et al., 2011).

To date, pomegranate's capacity to inhibit oxidative processes, and to accelerate the breakdown and the removal of oxidized lipids, has still been more studied in the health field, namely regarding atherosclerosis development and its consequent cardiovascular events (Aviram et al., 2000, 2004; Visioli et al., 2011).

Other potential solutions

Taking into account the evidence discussed throughout this review, there are some other foods that, although not studied yet, might also have the potential to be considered as an effective solution for EIMD recovery. Therefore, the following examples were chosen due to their nutritional characteristics and may be considered in future investigations.

Meat and fish, although may not be considered as conventional as liquid options, can also be a valuable alternative not only due to their content in proteins with high-biological value (Guigoz, 2011), but also because they are one of the richest sources of some compounds mentioned above, namely LA, CoQ_{10} and PUFAs. Moreover, fatty fishes as salmon, tuna and mackerel may also be a good choice due to their high amounts in n-3 PUFAs (Insel et al., 2007). Beef has already shown to be capable of stimulating MPS from young to old persons (Robinson et al., 2013; Symons et al., 2007); however, to date, no studies regarding muscle damage have been conducted.

Egg may also be a valid option since it has a biologic value of 100, meaning that all the absorbed egg protein is retain by the body (Insel et al., 2007). Egg protein was already used to study MPS (Moore et al., 2009); however, to date, no study has used eggs to investigate recovery from EIMD.

In contrast to the other plant foods, the protein isolated from soya beans provides a complete, high-quality protein equal to the animal protein (Young, 1991). Soya protein is considered a fast protein since it is digested rapidly, leading to a large but transient rise in aminoacidemia (Wilkinson et al., 2007). Yet, compared to fluid milk, it seems to lead to a less acute rise in muscle protein synthesis (Wilkinson et al., 2007). This may be due to the fact that the leucinemia is greater and more prolonged with milk consumption than with soya, probably reflecting the higher leucine content of whey proteins (Phillips, 2011). Nevertheless, soya beverages can be used, for example, when there is a contraindication for milk consumption, such as cow's milk-protein allergy or lactose intolerance.

Typical sources of CHO include bread, pasta, rice, potatoes, beans and fruit. Athletes may choose the type of CHO-rich food to consume according to the glycemic index (GI), the individual goals of each athlete and the timing of ingestion (Mondazzi & Arcelli, 2009). Fruit, in addition to have a high content in minerals, vitamins and antioxidants, is also a rich source of CHO, namely fructose and glucose. It has been shown (Jeukendrup, 2010) that when a combination of several CHO, specially glucose and fructose, is used instead of just one, the CHO absorption could be increased. This phenomenon is due to the utilization of different intestinal transporters for absorption (Jeukendrup, 2010). Particularly, the mixture glucose–fructose, namely the one with a 1:1 ratio, seems to produce one of the highest exogenous carbohydrate absorption rates (Jeukendrup, 2010). Therefore, ingesting a mixture of glucose and fructose seems to provide an optimal balance of dietary CHO for both muscle (Wallis et al., 2008) and liver (Casey et al., 2000) glycogen re-synthesis. Regarding the antioxidant potential of fruits, other berries may also be of interest for future studies, e.g. strawberries, raspberries and blackberries.

Tea is also known for its antioxidant content. Tea is originated from the leaves of *Camellia sinensis* L. and, according to the fermentation process, one can obtain green (not fermented), oolong (partially fermented) and black tea (fermented) (Lin et al., 2003). Green tea is considered an important dietary source of polyphenols, particularly flavonoids (Cabrera et al., 2006), being catechins the main flavonoid present (McKay & Blumberg, 2002). Catechins – especially EGCG – which are found in higher amounts in green tea than in black or oolong, are considered to have strong antioxidant potential, extensively demonstrated by *in vitro* and animal studies (Cabrera et al., 2006), and anti-inflammatory properties (Cabrera et al., 2006). Therefore, as the human clinical evidence is still scarce, future studies are needed in order to define the magnitude of the possible benefits and to establish, if it is the case, safe ranges of consumptions related to its benefits (Chacko et al., 2010).

Along with their high content in vitamins and minerals, unsaturated fatty acids and fibre, nuts enclose several phytochemicals that have been shown to possess a range of bioactive actions, including antioxidant and anti-inflammatory properties (Bolling et al., 2010; Chen & Blumberg, 2008). In fact, the consumption of nuts has been inversely associated with biomarkers of inflammation (Jiang et al., 2006). Regarding the antioxidant properties, it has been attributed mostly to their phenolic compounds, but a limited number of studies are available (Chen & Blumberg, 2008).

Conclusions

Due to the fact that EIMD can impair athletes' ability to train and perform properly, developing strategies that meliorate and accelerate the recovery process after muscle damage are of huge importance for the athletic population. Accelerating this process will result in shorter recovery periods that will allow athletes to return sooner to their normal training routine.

Although there are few studies that relate food and recovery from EIMD, the results available seem promising. Moreover, it is important to bear in mind the current issues related to the potential contamination of the nutritional supplements that athletes often use as a recovery strategy. Therefore, considering food as a potential mean to recover from muscle damage becomes even more important, since it is not contaminated with prohibitive compounds.

Some foods enclose potential to be considered an effective recovery option, especially if combined to ensure the delivery of protein, carbohydrates, antioxidants and anti-inflammatory nutrients. Beyond milk, cherries, blueberries and pomegranates, that were already successfully tested regarding EIMD, other foods are considered to be possible solutions to help in the recovery process from muscle damage. These foods include protein sources as milk, meat, fish, eggs and soy, carbohydrate-rich foods, for instance bread, pasta, rice, potatoes, beans and fruit, and foods with a high content in antioxidant and/or anti-inflammatory nutrients, such as other berries, tea and nuts.

It is clear, therefore, that more studies in this specific filed are needed. It is fundamental to have scientific evidence about which types or combination of foods can improve the recovery from EIMD.

Declaration of interest

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