# Nutritional interventions for reducing the signs and symptoms of exercise-induced muscle damage and accelerate recovery in athletes: current knowledge, practical application and future perspectives

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**Keywords**
Damage, muscle, supplementation, nutrition, recovery

**Abbreviations**

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| EIMD | Exercise-induced muscle damage |
| ROM | Range of motion |
| CK | Creatine kinase |
| LDH | Lactate dehydrogenase |
| MYO | Myoglobin |
| CRP | C-reactive protein |
| ACTN3 | Gene that encodes the α-actinin-3 protein |
| TNF | Tumour necrosis factor |
| IL-6 | Interleukin-6 |
| IGF2 | Insulin-like growth factor 2 |
| ECM | Extracellular matrix |
| RBE | Repeated bout effect |
| E-C | Excitation contraction |
| NGF | Nerve growth factor |
| GDNF | Glial cell line-derived neurotrophic factor |
| $PLA_2$ | Phospholipase $A_2$ |
| FFA | Free fatty acids |
| EPA | Eicosapentaenoic acid |
| DHA | Docosahexaenoic acid |
| CMJ | Countermovement jump |
| RSI | Reactive strength index |
| BCAA | Branched chain amino acids |
| CMJ | Counter movement jump |
| AST | Aspartate aminotransferase |
| ALT | Alanine aminotransferase |
| 1-RM | One-repetition-maximum |
| MDA | Malondialdehyde |
| PC | Protein carbonyls |
| MSM | Methylsulfonylmethane |
| RONS | Reactive oxygen and nitrogen species |
| MVC | Maximal voluntary contraction |
| PNS | Polar-nonpolar-sandwich |
| VAS | Visual analogic scale |
| TBARS | Thiobarbituric acid reactive substances |

**Abstract**

**Purpose** This review provides an overview of the current knowledge of the nutritional strategies to treat the signs and symptoms related to EIMD. These strategies have been organized into the following sections based upon the quality and quantity of the scientific support available: 1) interventions with a good level of evidence; 2) interventions with some evidence and require more research; and 3) potential nutritional interventions with little to-no-evidence to support efficacy.

**Method** Pubmed, EMBASE, Scopus and Web of Science were used. The search terms 'EIMD' and 'exercise-induced muscle damage' were individually concatenated with 'supplementation', 'athletes', 'recovery', 'adaptation', 'nutritional strategies', hormesis'.

**Result** Supplementation with tart cherries, beetroot, pomegranate, creatine monohydrate and vitamin D appear to provide a prophylactic effect in reducing EIMD. β-hydroxy β-methylbutyrate, and the ingestion of protein, BCAA and milk could represent promising strategies to manage EIMD. Other nutritional interventions were identified but offered limited effect in the treatment of EIMD; however, inconsistencies in the dose and frequency of interventions might account for the lack of consensus regarding their efficacy.

**Conclusion** There are clearly varying levels of evidence and practitioners should be mindful to refer to this evidence-base when prescribing to clients and athletes. One concern is the potential for these interventions to interfere with the exercise-recovery-adaptation continuum. Whilst there is no evidence that these interventions will blunt adaptation, it seems pragmatic to use a periodised approach to administering these strategies until data are in place to provide and evidence base on any interference effect on adaptation.

## Introduction

Exercise-induced muscle damage (EIMD) is a transient event caused by unaccustomed exercise (Harty et al. 2019) or strenuous high intensity or long duration exercise (Jang et al. 2018; Leeder et al. 2014). Moreover, EIMD can be evident after isometric contractions at a long muscle length, even at a low intensity (Chen et al. 2012). Muscle damage is characterized by a primary response from a mechanical challenge during the exercise that results in myofibrillar disruption (Yu et al. 2013) and a secondary inflammatory response that results in cell infiltration into the damaged tissues (Deyhle et al. 2015; Harty et al. 2019) to initiate subsequent tissue repair and remodelling (Peake et al. 2017). Beyond myofibre damage, the extracellular matrix (ECM) of skeletal muscle is also subjected to degradation and remodelling following muscle-damaging exercise that can be directly observed histologically (Hyldahl et al., 2015). Indirect evidence of ECM damage also exists, with a marked increase in the circulation of collagen-specific amino acids, most notably hydroxyproline, in the days following muscle-damaging exercise (Clifford et al. 2019).

Although the exact mechanisms responsible for skeletal muscle damage remain unclear, a damage model has been proposed (Figure 1). The damage model can be briefly summarized into two general phases: an initial or *primary phase*, which occurs during the exercise bout (Armstrong 1984; Ebbeling and Clarkson 1989; Belcastro et al. 1998; Verburg et al. 2005; Belcastro 1993) that result in an increase in cell membrane permeability causing further influx of $Ca^{2+}$ (for more details see Gissel 2005). The *secondary phase* which is linked with the inflammatory response and increased oxidative stress that further affect cell structure and function (Jackson et al. 1984; Howatson and van Someren 2008; Baumert et al. 2016; Owens et al. 2019; Hody et al. 2019). Thus, muscle damage can be considered a tightly regulated process of degradation and repair, where several cell types (i.e. neutrophils, macrophages, cytokines) interact during the proinflammatory and inflammatory stages of muscle damage with the aim of restoring tissue homeostasis.

Most of the signs and symptoms of EIMD (i.e. muscle soreness, stiffness, tenderness, pain, impaired muscle function) are evident within a few hours following the exercise insult and can persist for many days following the initial bout, however the magnitude and duration can vary greatly between individuals. Of primary concern is a temporary decrease in muscle function (strength, power, speed, economy of movement) that is accompanied by reduced range of motion (Jamurtas et al. 2005; Howatson et al. 2008; Damas et al. 2016; Qamar et al. 2019). Furthermore, there is often evidence of muscle swelling, muscle soreness, decreased pain threshold, increase of oxidative stress markers (Childs et al., 2001; Lee et al., 2002; Sacheck et al., 2003) and rise of various inflammatory interleukins, C-reactive protein (CRP), tenascin C, which can be accompanied by elevated intramuscular proteins (creatine kinase; CK, myoglobin; MYO, lactate dehydrogenase; LDH, for example); these can be present for several days following the damaging bout of exercise (Byrne et al. 2004; Hyldahl et al. 2014; Warren et al. 1999; Harty et al. 2019; Qamar et al. 2019; Baumert et al. 2016). Delayed-onset of muscle soreness (DOMS), a common symptom of EIMD, typically appears between 8 and 24 hours after the muscle-damaging exercise, peaks between 24 and 48 hours and usually subsides within 96 h (Damas et al. 2016; Jones et al. 1987). The magnitude and time course of signs and symptoms of EIMD and their subsequent impact on performance are variable and depend on the extent of initial muscle damage, which in turn is influenced by the duration, muscle group targeted, mode of exercise, intensity, and volume of the damaging exercise (Bowtell et al. 2011; Peake et al. 2017) and the individual's susceptibility to the damaging stimulus (Owens et al. 2019). Age, sex and physiological training status of the individual (Plowman and Smith 2013), along with genetic variability where certain genotypes are thought to be more susceptible to damage (Baumert et al. 2016), are additional factors that could play a contributing factors in the magnitude to EIMD and hence might require a longer recovery period.

Research indicates that inflammation and oxidative stress is evident following muscle damaging exercise (Jamurtas 2018). However, it is widely accepted these processes are necessary for the adaptive remodelling process and

hence critical for the resolution of function (Owens et al. 2019). Consequently, there is a dilemma when planning nutrition strategies for interventions that target the post-exercise inflammatory and oxidative stress response. If the principal purpose is to maximize the training stimulus, athletes and practitioners might need to consider a periodised approach to supplementation to adequately support training and competition demands to maximize the balance between the potential for adaptation and optimising recovery. This notion can be applied with the hormesis concept, whereby the biological systems respond in a bell-shaped fashion, where a positive adaptive response is experienced when exposed to an increase in training stimulus (Hawley et al. 2011). However, when the stimulus exposure becomes too great; for example, when EIMD impairs muscular functions for an extended period or the need to accelerate recovery is greater than the need for an adaptive response, a need to intervene to negate the negative effects exists. Consequently, the aim of a recovery intervention is highly dependent upon the purpose of the session and whether it is more important adapt or accelerate recovery of muscle function, ameliorate soreness in preparation for subsequent training or competition (Figure 2).

Optimal nutrition plays an important role during post-exercise recovery, where replenishment of endogenous substrate stores and facilitation of muscle-damage-repair process are the main goals (Beelen et al., 2010). The fundamentals of nutrition are extremely important, so ensuring adequate hydration, carbohydrates and protein availability are critical, but these fundamental aspects of nutrition are not covered here. Thus, the aim of this review is to provide an overview of the current evidence of nutritional supplementation strategies to ameliorate the signs and symptoms related to EIMD beyond the fundamentals of good hydration and macronutrient intake. Specifically, we focus on nutritional strategies that have been explored to accelerate the recovery process after damaging exercise. These strategies have been organized into the following sections based on level of available evidence: 1) interventions with good level of research investigating their efficacy; 2) interventions that have a modest evidence base; and 3) nutritional interventions with little or no evidence that might prove worthwhile avenues for future research.

## Supplements with good level of evidence

### Vitamin D

It is widely recognized that vitamin D plays an important role of bone health of athletes (Larson-Meyer et al. 2019), maintaining a healthy mineralized skeleton (Charoenngam et al. 2019) and regulating the processes of skeletal muscle regeneration (Owens et al. 2015). Vitamin D also has immunomodulatory effects, with increased vitamin D shown to reduce inflammation (Willis et al. 2008); furthermore, there is a strong association between sufficient vitamin D and optimal muscle function (Shuler et al. 2012). Vitamin $D_3$ (cholecalciferol) can be synthesized endogenously in the skin as a result of exposure to ultraviolet B radiation from sunlight, or obtained from dietary intake in foods that include fatty fish, eggs, and fortified foods (i.e. milk, citrus juice, breakfast cereals), while vitamin $D_2$ (ergocalciferol) is found only in plants and fungi such as mushrooms (Wilson et al. 2017) and algae (Göring 2018). Dietary sources of vitamin D are thought to have only a small effect on serum 25(OH)D concentration (an index of vitamin D levels) and the combination of limited dietary sources and several others factors (i.e. season, sport, body composition, training location, altitude, air pollution, geographical latitude, athlete's skin pigmentation) could be responsible for the increased risk of vitamin D deficiency in athletes (Larson-Meyer et al. 2019).

A previous randomised, placebo-controlled trial has shown the role of vitamin D for muscle recovery, and the interplay between serum vitamin D status and the magnitude of exercise-induced muscle damage (Owens et al., 2015) in 20 moderately active, adult males with deficient vitamin D (serum 25(OH)D). Following a six week oral vitamin $D_3$ supplement (4000 IU/day) or a placebo, volunteers completed a damaging bout of exercise. Serum 25(OH)D was

increased in the vitamin $D_3$ group and had improved recovery of muscle function in the days following the damaging exercise compared to the placebo control. Conversely, Shanely et al. (2014) found no effects of vitamin $D_2$ supplementation (600 IU per day, for 6 weeks) on recovery of lower limb muscle function or circulating markers of muscle damage following 1.5 h high-intensity intermittent exercise. The association between vitamin D status, exercise-induced muscle pain and weakness was investigated in 48 young healthy males and females (Ring et al. 2010). Following a damaging bout of eccentric exercise to the elbow flexors (3 x 12, 75% MVC) vitamin D status was not able to predict changes in muscle strength loss or pain outcomes. In contrast, an observational study reported that pre-exercise vitamin D status was influential on muscular weakness after eccentric exercise (Barker et al. 2013a). The study showed a transient increase in circulatory 25(OH)D immediately after the exercise, followed by a decrease. The authors suggested that serum 25(OH)D is temporally sensitive to intense exercise and that a multitude of biomarkers (i.e. cytokines, ALT, AST, albumin) contribute, in part, to the 25(OH)D fluctuations (Barker et al. 2013a) and hence provide some evidence of the interplay between eccentric exercise, circulating vitamin D and the possible role in recovery.

Following exercise-induced muscle damage (EIMD), induced by eccentric exercise, Barker et al. (2013b) demonstrated that supplemental vitamin $D_3$ (4000 IU/d) for 35 days increased serum 25(OH)D concentrations and was associated with a faster restoration of peak force during recovery. Peak force was approximately 8% superior in the supplemented group compared to the placebo at 24 h after the damaging exercise, although recovery between the groups was similar thereafter. Other biomarkers of exercise stress, such as circulating aspartate amino-transferase (68 h post-exercise) was attenuated by 29%, while alanine amino-transferase (24 h post-exercise) was attenuated by 40%, compared to placebo. In contrast, Nieman et al. (2013) showed that vitamin $D_2$ supplementation in professional race car pit-crew had no effect on muscle function tests following 90 min eccentric exercise.

Potential reasons for these discrepancies across research might be related to outcome measures, populations, mode/type, length and dose of supplementation and follow-up or different experimental protocols, including the muscle group(s) exercised, damaged protocol, contraction modalities, volume, duration, intensity of the exercise protocol. In addition, differences between studies exist with respect to baseline levels of 25(OH)D. More work is necessary to clarify the benefit of vitamin D for athletic muscle recovery. Athletes should aim to maintain appropriate vitamin D status through regular sun exposure, supplementation, and/or diet. The target level of 25(OH)D has not been identified for an endpoint of muscle repair; however, it seems prudent for athletes to aim for the clinical cut-off for sufficient vitamin D status, which is 30-50 nmol/L (Owens et al., 2015). The available data suggest vitamin D might play a role in the muscle repair and recovery process (Heaton et al. 2017) but based on the available evidence there is no clear benefit in improving recovery following damaging exercise.

**Omega-3 polyunsaturated fatty acids**

Omega-3 fatty acids (eicosapentaenoic acid or EPA and docosahexaenoic acid or DHA) are essential nutrients; there is no mechanism in humans for producing these fats however, they can be consumed through oily fish, selected vegetables, nuts and seeds. It is widely known that omega-3 are effective for improving cardiac function, depression, cognitive function and a relationship with improved endurance performance (Ochi and Tsuchiya 2018). Omega-3 polyunsaturated fatty acids (PUFA) are able to modulate inflammation, decrease swelling, and reduce sensitivity to pain (Jouris et al. 2011). Moreover, EPA and DHA have been shown to play a role in the regulation of skeletal muscle protein synthesis and immune function (Heaton et al. 2017). Many studies have highlighted that intake of omega-3 PUFA could modulate EIMD outcomes, though results are generally equivocal because of high variability in study designs, participants, dosing and timing protocol exists in the literature (Tartibian et al. 2009; Philpott et al. 2018; Tsuchiya et al.,

2016). For example, some studies have reported attenuated the increase in plasma IL-6 up to 72 h after strenuous exercise (DiLorenzo et al. 2014; Phillips et al. 2003; Tartibian et al. 2011) and others have not (Lenn et al. 2002; Nieman et al. 2009).

Lenn et al. (2002) examined the effect of 30 days fish oils (1.8 g of EPA and DHA per day) supplementation on delayed onset of muscle soreness following a damaging bout of exercise. The authors reported that fish oils were not effective in ameliorating DOMS in comparison to a placebo or isoflavones (120 mg of soy isolate) supplementation. Similarly, others (Bloomer et al., 2009) found no effect of 6 weeks of omega-3 PUFA (2224 mg EPA and 2208 mg DHA per day) on attenuation of oxidative stress and inflammation induced by a 60-min treadmill climb, in 15 well-trained participants. In contrast, Tartibian et al. (2009) showed a beneficial treatment effect of omega-3 (32 days of 1.8 g of EPA and DHA per day versus a placebo) on perceived pain and a minor decrement of range of motion 48 h after damaging exercise (40 min of bench stepping exercise) in 27 healthy untrained males. In a subsequent randomized, double-blinded study (Tartibian et al., 2011), 45 healthy untrained men completed an eccentric exercise program (40 minutes of bench stepping) following 30 days supplementation (324 mg of EPA and 216 mg of DHA per day, which continued 48 h after exercise. The authors showed a reduction in the plasma TNF-α, PGE2, LDH, IL-6, CK immediately, 24, and 48 h after the damaging exercise compared to the placebo (soybean/corn oil mixture) and control group. A recent double-blind, placebo-controlled, parallel design study showed that daily supplementation for 8 weeks of fish oil rich in omega-3 (EPA; 600mg and DHA; 260 mg) has a positive role in inhibiting muscle stiffness after eccentric contractions and was able to inhibit the loss of muscle strength, reduce ROM, development of DOMS, and to reduce muscle swelling in comparison to a placebo (Tsuchiya et al. 2019). Likewise, Ramos-Campo et al. (2020) reported that the ingestion of 2.1 g of DHA and 240 mg of EPA every day for 10 weeks, promoted lower concentrations of IL1β, IL6, CK, LDH and muscle soreness after a damaging eccentric exercise in endurance athletes, when compared to placebo treatment (olive oil).

In eleven healthy men and women, Jouris et al. (2011) tested the effects of 7 days of EPA and DHA supplementation, in a ratio 2:1 (2000 mg of EPA and 1000 mg of DHA) on inflammation induced by two sets of eccentric elbow flexion exercise (120% 1RM) and showed a reduction of post-exercise soreness at 48 h post exercise. In contrast, Houghton and Onambele (2012) investigated the effects of a dose of 360 mg of EPA supplementation per day for 3 weeks on muscle soreness and basal inflammation induced by acute and chronic resistance exercise in a three-week, double-blind placebo-controlled study in 20 participants. Data showed no effect in reducing DOMS and attenuating increases in IL-6 levels in comparison to a placebo. The authors speculated that these results might have been associated by increased EPA-mediated contractile capacity, resulting in increased glucose metabolism, glycogen depletion and hence elevations in the IL-6 concentrations. Conversely, in a study by DiLorenzo et al. (2014), 41 untrained volunteers received 28 days (2 g/day) of DHA or a placebo, before a 17-day resistance exercise program. The DHA supplemented group showed a reduction of CK and IL-6 response in the first 4 days of the program, while no differences in muscle strength and soreness were observed between groups. Similarly, Gray et al. (2014) examined the effect of a six-week fish oil supplementation (1.3 g EPA, 0.3 g DHA and 45 I.U. d-α tocopherol) or a placebo on exercise induced muscle soreness and oxidative stress markers in 20 trained males following eccentric exercise. There were no differences between groups for indices of muscle damage and soreness. However, the fish oil group showed lower oxidative stress markers (thiobarbituric acid reactive substances; TBARS and $H_2O_2$ stimulated DNA damage) at 48 and 72 h after the exercise bout of eccentric exercise. Nevertheless, it is important to treat these results with caution, because TBARS and $H_2O_2$ have methodological limitations and should be considered surrogate measures, at best. In another study (Lembke et al. 2014), 63 healthy male and female college students were supplemented for 30 days with omega-3 (2.7 g per day) or placebo (sunflower oil) prior to forearm extension eccentric exercise. Results showed a decrease in muscle soreness at 72 h and 96 h after damaging exercise in

the supplemented group compared to placebo. A more recent double-blind, placebo-controlled, parallel-group trial (Tsuchiya et al. 2016), conducted on 24 healthy Japanese men, concluded that EPA and DHA supplementation (600 mg EPA and 260 mg DHA per day) for eight weeks prior to, and 5 days after maximal unilateral isokinetic elbow flexor exercise was more beneficial than a placebo to attenuate strength loss and limit reductions in ROM during the recovery period. Jakerman et al. (2017) first examined the effect of an acute intake of two fish oil supplements, with different concentrations of EPA and DHA in a double-blind, placebo-controlled study conducted in 27 physically active males. Immediately following a damaging bout of exercise (100 drop jumps), volunteers ingested either a low-EPA fish oil (150 mg EPA, 100 mg DHA), high-EPA fish oil (750 mg EPA, 50 mg DHA) or a placebo. Results showed that acute ingestion of high-EPA fish oil was beneficial on some markers of performance compared to low-EPA fish oil and placebo.

Some have reported beneficial effects of omega-3 supplementation in females. In 42 healthy women, Corder et al. (2016) examined the effect of DHA supplementation (7 days before and 2 days after exercise with 3000 mg per day) on DOMS and local muscle inflammation after an eccentric elbow flexion exercise. Results showed DHA supplementation reduced muscle soreness and stiffness after 48 h, but no effects on swelling compared to a placebo. Later, Tinsley et al. (2017) supplemented both EPA and DHA in 17 non-resistance trained females. Participants were randomized into fish oil (6 g/d; 5:1 EPA:DHA) or placebo groups and took the supplement for one week prior to, and one week after a damaging elbow flexion and extension exercise. Results showed that at 48 h post-exercise, the supplemented group reported less muscle soreness in the lower body than the placebo group.

In some recent investigations the benefits of combining omega-3 with other nutrients have been examined. For example, Philpott et al. (2018) added 1100 mg EPA and 1100 mg DHA to a ready-to-drink beverage (15 g whey protein, 1.8 g leucine and 20 g carbohydrate) and supplemented this blend in ten male soccer players each day for 6 weeks. The other two groups (10 participants per group) ingested a beverage containing 15 g whey protein, 1.8 g leucine and 20 g carbohydrate, or a beverage containing only carbohydrate (20g). After 6 weeks of supplementation, an eccentric damaging exercise was performed, and muscle soreness and blood markers of muscle damage were measured. Notable results showed that muscle soreness, during the 72 h recovery, and blood CK was less in participants that ingested the beverage with EPA and DHA, compared to other two groups. No differences in muscle function, soccer performance or blood CRP were observed between groups. In a further recent double-blind study, Black et al. (2018) investigated the effectiveness of consuming, twice daily, 551 mg EPA and 551 mg DHA added to a protein-carbohydrate supplement (15 g protein, 14.5 g carbohydrate and 8.4 g fat) or placebo (15 g protein, 14.5 g carbohydrate and 8.4 g fat, without omega-3) in 33 professional Rugby Union players during 5 weeks of pre-season training. Reduced muscle soreness and fatigue and a better maintenance of jump performance was reported in athletes that consumed the enriched-omega 3 beverage compared to placebo group during all time points. The efficacy of a dietary supplement that contained, in part, omega 3 PUFA to modulate inflammation following EIMD was evaluated in 40 healthy, untrained males (Phillips et al., 2014). Researchers supplemented a blend consisting of 300 mg mixed tocopherols, 800 mg DHA and 300 mg of flavonoids (100 mg hesperetin and 200 mg quercetin) or placebo (high-oleic sunflower oil) for 14 days. Although blood indices of damage (LDH and CK) was not different between groups, the supplemented group showed a reduction in IL-6 and CRP after eccentric exercise, compared to the placebo. Although these show some promise, the blended supplement makes it difficult to attribute solely to omega-3 PUFA.

The divergent results in this section could be explained, in part, by differences in supplement dose, type of fatty acid ingested, fitness level of participants (trained vs untrained) duration of supplementation and intensity and/or modality of exercise. Albeit there is a potential for omega-3 PUFA supplementation to improve muscle adaptation and muscle recovery, supplementation might yet prove to be effective to attenuate muscle damage (Hennigar et al. 2017) and further

study in to the optimal dose of EPA:DHA ratio, the synergistic effect with other nutrients and the timing areas for further enquiry.

**Tart Montmorency cherries (*Prunus cerasus*)**

Montmorency tart cherries (*Prunus cerasus L.*) have been proposed as a recovery supplement because of their high concentrations of phytochemicals, and in particular, the flavonoids anthocyanins and other phenolic compounds (Bell et al. 2015; Coelho et al. 2015; Howatson et al. 2010). These phenolic compounds have been shown to reduce oxidative stress and inflammation by inhibiting the action of cyclooxygenase to a similar extent as nonsteroidal anti-inflammatory drugs (Seeram et al. 2001; Wang et al., 1999). These findings have led to numerous lines of enquiry that applied Montmorency cherries in exercise recovery (Connolly et al., 2006; Howatson et al., 2010; Kuehl et al., 2010; Bowtell et al. 2011; Bell et al., 2014).

The first study (Connolly et al., 2006) examined the efficacy of a tart Montmorency cherry juice blend in preventing the symptoms of exercise induced muscle damage in a randomised, placebo-controlled trial. Results showed that 12 oz of tart Montmorency cherry juice taken twice a day for 4 d before and 4 d after heavy eccentric contractions of the elbow flexors, reduced pain and limited post-exercise strength loss (4% versus 22%) compared to a placebo. In a subsequent applied study, Howatson et al. (2010) use the same cherry juice blend used previously (Connolly et al., 2006) and showed improved recovery of strength and reduced inflammation (IL-6 and C-reactive protein) and oxidative stress (TBARS) following a marathon run. This placebo controlled, parallel group study supplemented with 8 fl oz of cherry juice twice a day, ingested for 5 consecutively days before, the day of race and 2 days after running a marathon or a placebo group (8 fl oz of fruit flavoured cordial). A further study using a randomized, double blind, placebo-controlled trial design (Kuehl et al., 2010) in a cohort of 54 runners, examined the effect of tart Montmorency cherry juice following a strenuous trail running event. They showed that ingestion of 335 mL tart Montmorency cherry juice consumed for 7 days prior to, and during, the event reduced post-run muscle pain compared to placebo. Collectively, these studies indicate that the intervention is effective to reduce pain and restore function in damaging running activity.

Bowtell et al. (2011) showed that consumption of a tart Montmorency cherry concentrate (30 mL twice per day for 10 days) in ten well-trained males reduced symptoms of EIMD. Force recovery was greater in the cherry juice group after a damaging eccentric knee extensor exercise compared to an isoenergetic fruit concentrate placebo. This study illustrated the potential application in a well-trained cohort following strength-based training. In a further study (Kastello et al. 2014) using a double-blind, placebo controlled, cross-over design, the effects of tart cherry supplementation (freeze dried powder tablets – one tablet, twice per day for 16 days prior to, and 3 days following) was examined in an untrained mixed sex cohort (10 females and 4 males) following damaging exercise. Findings suggested showed cherries to reduce C reactive protein levels, range of motion and perceived pain more than a placebo (Kastello et al. 2014).

The aforementioned studies show that tart Montmorency cherries are able to provide enhanced recovery following exercise with a mechanical stressor where there is myofibrillar disruption from high intensity eccentric contractions. In an attempt to investigate the possible application in a solely metabolic challenge, Bell et al. (2015) investigated if a tart Montmorency cherries concentrate would facilitate recovery on signs and symptoms of EIMD in 16 well-trained cyclists. In this parallel group design, volunteers were given the cherry concentrate or caloric matched placebo for 8 days. On the fifth day of supplementation, a strenuous bout of cycling designed to replicate a road race was completed and indices of inflammation, and muscle damage were taken. Results showed that the Montmorency tart cherry group showed lower levels of IL-6 and hsCRP, and attenuated reductions in muscle function (Bell et al. 2015). In a similar study with the same research group, Bell et al. (2014) examined the effect of a tart Montmorency cherry

concentrate in trained cyclists conducting three repeated days of simulated road racing and showed reductions in inflammation during the 3 days of cycling compared to a placebo. Collectively, these data show that tart Montmorency cherries can support recovery following both mechanically and metabolically challenging exercise (Bell et al., 2014).

Levers et al. (2015) investigated 10 days supplementation (480 g/d) with tart cherry skin powder on indices on EIMD. After 7 days, of supplementation, resistance trained males conducted a damaging bout of resistance training followed by 3 days supplementation for three days. They showed reduced creatinine, total protein and perceived soreness in comparison to a placebo in the 48 h after the exercise insult. In a successive study (Levers et al. 2016), using the same powdered tart cherries, examined in 27 endurance-trained runners or triathletes (18 males and 9 females) following a half marathon. The groups received a similar 7 day loading phase and dose (480 g/d) followed by two days of supplementation or a placebo. The dried cherry skins attenuated post-run muscle soreness, urea/blood urea nitrogen, total protein, and cortisol compared to the placebo group (Levers et al. 2016). In a related study to Howatson et al. (2010), Dimitriou et al. (2015) investigated the effects of Montmorency cherry juice on muscle inflammation, immunity and stress. Using the same cohort and supplementation to the aforementioned study (Howatson et al., 2010), the cherry juice group showed no upper respiratory symptoms in the period following the marathon compared to the placebo group indicating some potential for immune function.

Montmorency tart cherry supplementation was also studied in sixteen semi-professional male soccer players in a double blind, placebo-controlled design with independent groups following prolonged intermittent sprint exercise (Bell et al. 2016). Participants were supplemented with 30 mL twice per day of tart Montmorency cherry concentrate or caloric matched placebo for seven consecutive days. This study showed an attenuation in performance declines and a reduction of muscle damage indices, for example IL-6 at 24 and 48 h post exercise. In addition, Brown et al. (2019) investigated the effect of tart cherry in twenty female dancers in a randomised, double-blind, placebo-controlled study. Participants were allocated tart Montmorency cherry concentrate (30 mL twice a day) or placebo (fruit flavoured concentrate) for eight days. After 4 days of supplementation, participants completed a damaging repeated sprint exercise protocol. Results showed that dancers that ingested tart cherry juice had a faster recovery of muscle function, as well as a lower reported DOMS compared to the placebo group. Likewise, Quinlan and Hill (2019) reported that male and female team-sport players who ingested tart Montmorency cherry juice concentrate accelerated recovery after an intermittent shuttle run test, compared to the placebo group. Collectively, these data show the application of tart Montmorency cherries in repeated sprint activity like soccer and other field sports for both males and females.

Tart cherry was investigated in different doses, formulation and different sports and trained and untrained athletes. Overall, studies have typically reported beneficial effects on signs and symptoms of EIMD, however, some work has shown no benefit (Beals et al. 2017; McCormick et al. 2016). Beals et al. (2017) saw no beneficial effect of supplementation with a beverage containing freeze-dried tart cherry powder on the recovery of DOMS or muscle strength in recreationally active men and women following damaging eccentric exercise. Similarly, McCormick et al. (2016) found no effect of supplementation with tart cherry juice on DOMS and markers of inflammation and oxidative stress in well-trained male water polo players who undertook a simulated game of water polo. Furthermore, in a recent study (Lamb et al. 2019) the supplementation with tart cherry juice (2 x 250 mL) for 9 days did not enhance recovery in non-resistance trained men after maximal eccentric elbow flexor exercise. Likewise, Abbott et al. (2019) have reported that academy soccer players who ingested tart cherry juice at the dose of 30 mL before and 30 mL after a 90-minute match and at 12 and 36 hours after failed to hasten recovery of muscle function, when compared who ingested an isocaloric cherry-flavoured control drink, although this study was not able to control diet and did not include a loading phase which might explain the null effects. In many of the cases, tart Montmorency cherries reduce the inflammatory response following

exercise, which is an important signally cascade for the adaptative response. Whilst there is no evidence that any functional foods affect the adaptive response, it is clear that some inflammatory processes could be modulated. One caveat to this is that the inflammatory responses are not absent, but rather reduced by the intervention, so some inflammatory signalling is still present; the question for researchers and practitioners is "how much inflammation is enough?". Pragmatically, once the athletes are in a condition where further adaptations are not a priority, and restoration of function is the focus, tart Montmorency cherries have a beneficial role (Vitale et al. 2017).

**Creatine monohydrate**

Beyond the well-known effects of creatine monohydrate to improve exercise performance such as power (Kim et al. 2015), body composition, anaerobic, aerobic and strength performance (Fernandez-Landa et al. 2019), several studies suggested that creatine supplementation might also reduce muscle damage and/or enhance recovery following intense exercise (Kreider et al. 2017; Kim et al. 2015). Creatine supplementation has been shown to play an important role in muscle damage; one mechanism could be through cell membrane stabilization. The molecular structure of phosphocreatine, allows it to bind to the phospholipid heads of the cell membrane and stabilize the membrane, thereby reducing membrane fluidity and thus potentially decrease the degradation associated with muscle damage (McKinnon et al. 2012). Vierck et al. (2003) and Olsen et al. (2006) demonstrated that creatine supplementation might influence post-exercise muscle recovery by enhancing muscle satellite cell proliferation. Since satellite cells are myogenic stem cells that fuse with damaged muscle fibres to affect repair (Olsen et al. 2006), increased satellite cell proliferation could result in an increase recovery in muscle fibre integrity (McKinnon et al. 2012). Other potential mechanisms provide some rationale for the use of creatine on EIMD, including modulation of the inflammatory response (Santos et al. 2004; Bassit et al. 2008; Deminice et al. 2013), oxidative stress (Lawler et al. 2002; Deminice and Jordao 2012) and regulation of calcium homeostasis (Beaton et al., 2002; Cooke et al., 2009). According to a human study by Rahimi (2011) ingesting 20 g/day of creatine for 7 days decreased oxidative stress (malondialdehyde; MDA and 8-hydroxy-2-deoxyguanosine; 8-OHdG) after resistance exercise compared to a placebo. Regulation of calcium homeostasis is another candidate mechanism of creatine supplementation. Cooke et al. (2009) and Korge et al. (1993) reported that creatine assists in maintaining the sarcoplasmic reticulum calcium pump function by phosphorylating ADP to ATP, which decreases cytosolic calcium levels. Minajeva et al. (1996) suggested that increasing muscle Phosphocreatine levels accelerates ATP homeostasis, leading to reduced secondary damage due to an increase of calcium concentration in sarcoplasmic reticulum. However, the evidence for this idea in a muscle damage paradigm is thin and requires greater evidence.

In early work in this area, Willoughby and Rosene (2003) gave creatine to 22 untrained males (6 g/day for 12 weeks) followed by a bout of unaccustomed exercise. Results showed that the perception of muscle soreness was reduced in the supplemented group. It was speculated that creatine helped to direct gene transcription and upregulate protein synthesis. In a later study, Cooke et al. (2009) investigated the effects of five days of creatine supplementation (21 g per day) and carbohydrate (84 g day) or only carbohydrate in 14 untrained healthy males on muscle function and muscle damage indices after intense eccentric exercise. They showed that plasma CK levels were lower (by an average of 84%) in the days following the exercise in the creatine group; furthermore, this group showed a greater isokinetic (+10%) and isometric (+21%) knee extension strength following EIMD than carbohydrate group. Rosene et al. (2009) showed that both shorter and longer-term creatine supplementation (20 g/day for the first 7 days followed by 6 g/day for 23 days) improves maximal strength after exercise in 20 physically active males. Veggi et al. (2013) also showed that ingesting 20/day of creatine for 6 days between exercise bouts attenuates rises in CK, improved range of motion and decreased muscle soreness.

Santos et al. (2004) also studied the effects of creatine supplementation (20 g/day for 5 days prior to the 30 km race) on inflammatory markers and muscle soreness in experienced marathon runners. Results showed creatine supplementation attenuated the increase of prostaglandin E2 (-60.9%), TNF-α (-33.7%), CK (-19%) and abolished the increase of LDH levels after the run, compared to controls. Further, Bassit et al. (2010) in a small cohort (8 triathletes) randomized into a placebo or creatine monohydrate supplementation group (20 g/day for 5 days). The authors reported that creatine supplementation decreased plasma creatine kinase, LDH and aldolase, and prevented the rise of glutamic pyruvic acid transaminase and glutamic oxaloacetic acid transaminase, when compared to placebo group. Likewise, Deminice et al. (2013) supported previous data showing that seven days of creatine supplementation (0.3 g/kg of body mass/d) in 25 healthy, well-trained male soccer players reduced the increase of TNF-α, CRP and LDH, after a repeated sprint exercise protocol. In contrast, in a double-blind study where creatine supplementation (0.6 g/kg of body mass/d for 7 days) or placebo was examined in sedentary individuals following damaging resistance exercise, no difference between groups was observed (Machado et al. 2009).

Although several studies reported that creatine attenuated indices of EIMD (Willoughby and Rosene 2003; Santos et al. 2004; Bassit et al. 2010, Cooke et al. 2009; Rosene et al. 2009; Deminice et al. 2013; Veggi et al. 2013) other studies have reported that creatine had no effect (Rawson et al. 2001; Rawson et al. 2007; Mckinnon et al. 2012). For example, Rawson et al. (2001) investigated the effects of creatine supplementation (20 g/day taken for 5 days before and after exercise) in 23 non-weight-trained men, following eccentric contractions and showed no differences in muscle damage indices the creatine and placebo groups. In a later study, Rawson et al. (2007) examined the effects of short-term creatine supplementation on markers of muscle damage in 22 healthy, resistance-trained males. Participants consumed creatine (0.3 g/kg of body mass for the first 5 days and 0.03 g/kg for the next 5 days) or a placebo. Following the initial 5 days of supplementation, subjects performed damaging resistance exercise. There were no differences between groups suggesting that oral creatine supplementation did not reduce indices of EIMD. In further support of the null findings, Mckinnon et al. (2012) reported that ingesting creatine (loading dose: 40 g split into two servings/day for the first 5 days, followed by a maintenance period of 10 g/day split into two servings/day next 5 days) did not reduce indices of muscle damage or speed recovery following eccentrically induced muscle damage. Finally, a recent study (Fernández-Landa et al. 2020) failed to find any positive effect of combined creatine monohydrate and β-hydroxy β-methylbutyrate consumption (creatine 0.04 g/day per kg of body mass, plus 3g/day of HMB) in elite male rowers on reducing indirect markers of EIMD. Despite the mixed results, athletes might benefit from creatine supplementation to restore muscle function and assisting to reduce delayed onset muscle soreness.

**Beetroot juice**

Red Beetroot (*Beta vulgaris rubra*) is a functional food rich in nitrate, and other phytochemicals that include bioactive compounds such as betalains, ascorbic acid, carotenoids, phenolic acids and flavonoids. Several published works have reported beneficial effects of beetroot ingestion in managing hypertension, atherosclerosis, type 2 diabetes and dementia (Clifford et al. 2015; Siervo et al., 2018; Gilchrist et al., 2013). In addition, recent investigations have showed that the consumption of beetroot juice after eccentric exercise could reduce the magnitude of EIMD. In the first of a series of studies by the same laboratory, beetroot juice was used to manage signs and symptoms of exercise-induced muscle damage (Clifford et al., 2016a). Using a double-blind, independent groups design, 30 recreationally active males were randomized to consume a higher dose (250 mL per serving), or a lower dose of beetroot juice (125 mL per serving) or an isocaloric placebo (250 mL). Three servings were given immediately following damaging drop jump exercise, and 2 servings after 24 and 48 h. Results showed a faster recovery of pressure pain threshold during a 72-h recovery period

with beetroot juice in comparison to the placebo. However, muscle function was only improved in the higher-dose group compared to placebo. The same group adopted the same supplementation protocol in 29 recreationally actives males that performed two bouts of 100-drop jumps, separated by 14-21 days. In this trial, authors reported that supplementation with beetroot juice did not adversely affect acute adaptations (repeated bout effect) to eccentric exercise (Clifford et al. 2017a). In a further study (Clifford et al. 2016b) beetroot juice was applied as a post-exercise recovery strategy to attenuate EIMD in 20 collegiate team-sports players. In this trial, the effect of 500 mL of beetroot juice or a placebo for 3 days was examined after performing an initial repeated sprint test (20 x 30 m) followed by a second repeated sprint test, performed 72 h later. The supplemented group showed a faster recovery of countermovement jump (CMJ) and reactive strength index (RSI) after the first repeated sprint test. After 72 h following damaging exercise, the supplemented group showed an increased restoration of CMJ and RSI performance (7.6% and 13.8%, respectively) compared to placebo group (Clifford et al. 2016b).

In an attempt to understand the role of dietary nitrate in the beetroot juice, a later study investigated the effects of a nitrate only drink (sodium nitrate), beetroot juice or placebo drink on indices of muscle damage caused by exercise. In this trial, Clifford et al. (2017b) recruited 30 recreationally active males that were equally randomized in to the 3 groups and ingested the supplement immediately, 24 and 48 h (3 x 250 mL servings the first day, 2 x 250 mL second and third day) following 100 drop jumps. Pressure-pain threshold was attenuated in the beetroot juice group, which was 104±26% of baseline values at 72 h post exercise, compared to sodium nitrate (94±16%) and the placebo (91±19%) suggesting that beetroot was more effective than sodium nitrate for attenuating exercise-induced muscle pain; thus, any analgesic effect could be attributable to phytonutrients other than nitrate, or interactions between nitrate and other constituent compounds. Similarly, Montenegro et al. (2017) supported previous results of Clifford et al. (2016a; 2016b; 2017b) reporting that a betalain-rich beetroot concentrate, not containing nitrates (50 mg twice a day, before breakfast and evening, per 7 days), attenuated the increase of creatine kinase after a 10 km running time trial in triathletes. Most recently, these previous beneficial effects of beetroot juice supplementation were mirrored by Daab et al. (2020) in a randomized double-blind cross-over design in thirteen soccer players. Seven days of beetroot juice supplementation (2 servings of 150 mL per day for 3 days pre-exercise, day of test and 3 days after damaging exercise) was able to reduce muscle soreness and attenuate the decrease of MVC and CMJ. One contrasting study (Clifford et al., 2017c) reported no effect of beetroot juice consumption (3 x 250 mL servings/day for 3 days) on muscle damage indices exercise-induced, in 34 well trained runners who completed a marathon.

Existing evidence has shown some potential beneficial effects of beetroot supplementation on preserving muscle function and reducing exercise-induced pain; however, it should be made clear that any benefit appears to be very small and is largely around perceptual measures. Whilst an optimal dosing strategy does not currently exist, beetroot is easily applied for those who can tolerate the taste.

**Pomegranate juice**

Pomegranate (*Punica granatum* L.) is categorized as a fleshy berry that is rich in polyphenols and has shown some potential for promoting recovery from EIMD. It contains malic and citric acid, flavanols, anthocyanins, hydrolysable and condensed tannins (Kandylis and Kokkinomagoulos, 2020), in particular ellagitannins has anti-inflammatory and antioxidant properties (Danesi and Ferguson, 2017) and precursors of urolithin A, which has been associated with mitophagy and increased muscle function (Martinez-Sanchez et al. 2017). Consequently, a number of studies have suggested that consumption of pomegranate juice could be an intervention to manage the effects of exercise-

induced muscle damage.

Trombold et al. (2010) investigated the effects of ellagitannin extract (from pomegranate) on recovery of muscle strength after an eccentric exercise. In a crossover, placebo-controlled trial, 16 recreationally active males were randomized either pomegranate extract (480 mL twice a day) or a placebo for 9 days. Participants ingesting the extract had greater preservation of muscle function following damaging eccentric exercise compared to an isocaloric placebo. The same group (Trombold et al. 2011) supported these data in a subsequent study conducted in 17 resistance-trained subjects. Participants were assigned either 250 mL/day of pomegranate juice or a placebo for 15 days. On the eighth day of supplementation, damaging eccentric exercise of the elbow flexors and knee extensors was performed. Findings showed that pomegranate supplementation attenuated upper body strength loss and soreness compared to a placebo. No effect was reported on lower body and the researchers hypothesised that knee extensors are more active during activities of daily living and hence more accustomed to the exercise stimulus. It would, be prudent to consider the results arising from cross-over design studies in EIMD paradigms, because of potential issues relating to the contralateral RBE (Howatson and van Someren 2007), which show that the magnitude of the damage response on the contralateral limb can be significantly attenuated and hence mask possible effects of an intervention.

Other work (Machin et al., 2014) determined if there was a dose response effect of pomegranate supplementation on muscular strength recovery following a bout of combined upper and lower body eccentric exercise. Forty-five non-resistance trained volunteers were randomized into: (1) once-daily (30 mL of pomegranate juice concentrate); (2) twice-daily (60 mL of pomegranate juice concentrate), or (3) placebo, consumed for 8 days. After 3 days of supplementation, subjects performed 20 minutes of downhill running and 40 eccentric contractions of the elbow flexors. During the 4-day recovery period, both lower and higher doses facilitated the recovery of elbow flexion and knee extension strength when compared to the placebo. Other positive responses were also observed with pomegranate juice by Ammar et al. (2016) in professional male weightlifters. Subjects that consumed pomegranate juice had lower soreness during the 48-h recovery period and smaller increases in CK and LDH post-exercise, compared to a placebo.

In contrast, Lamb et al. (2019) observed that resistance trained men who ingested 500 mL/day of tart cherry juice or pomegranate juice, for 5 days before and 4 days after a damaging exercise, failed to enhance recovery from high-force eccentric exercise of the elbow flexors. In addition, Torregrosa-Garcìa et al. (2019) failed to show significant differences for force restoration after pomegranate extract supplementation (225 mg/day of prunicalgins) or placebo in 26 amateur cyclists. Recent evidence suggested that pomegranate juice showed promise as a strategy to improve muscle recovery and reduce indirect biomarkers of exercise-induced muscle damage.

**Interventions with a modest evidence base**

**L-citrulline and Watermelon juice**

L-citrulline is a colourless, water-soluble non-essential amino acid (Yang et al. 2019), an endogenous precursor of L-Arginine and an indirect precursor of nitric oxide (Martinez-Sanchez et al. 2017). Furthermore, L-citrulline is an essential component of the urea cycle in the liver and kidneys, being responsible for detoxification of ammonia via conversion to urea (Martinez-Sanchez et al. 2017).

Pérez-Guisado and Jakerman (2010) investigated the acute effects of citrulline on muscle soreness in 41 men. Participants who ingested l-citrulline (8 g of citrulline malate) 1 hour before the performing 8 sets of bench press exercise to fatigue at 80% 1RM. They showed a decrease of 40% in muscle soreness at 24 and 48 hours after the damaging session,

compared to placebo or isocaloric drink. A further study (Tarazona-Diaz et al., 2013) investigated the effect of watermelon juice containing 1.17 g or 6 g of L-citrulline or a placebo on muscle soreness in 7 men. Subjects who ingested the placebo 1 h prior to exercise session (8 x 30 s, with 1 min rest of cycle interval sprints) had greater soreness after 24 h of recovery period compared to participants that consumed watermelon juice. After these initial studies, Martinez-Sanchez et al. (2017a) reported that healthy male runners who consumed watermelon juice (3.45 g of L-citrulline per 500 mL) 1 h prior to a half-marathon race, showed an attenuation of muscle soreness during 24-72 h of recovery and preserved muscle function compared to the placebo. The same research group (Martinez-Sanchez et al. 2017b) investigated the effects of two watermelon juices with different concentration of L-citrulline (0.5 g and 3.3 g per serving, respectively) or a mix of watermelon juice and a concentrate of pomegranate enriched in L-citrulline (3.3 g per serving) and ellagitannins (22.0 mg per serving) compared to a placebo beverage in nineteen healthy males. Subjects consuming a serving of watermelon juice with a concentration of 3.3 g of L-citrulline or the beverage mix containing 3.3 g L-citrulline and 22.0 mg ellagitannins, prior to a half-squat exercise (8 sets of 8 repetitions) reported a reduced muscle soreness during 48 h of recovery, exhibited lower increase of myoglobin and showed a significant maintenance of muscle function compared to the placebo group.

In contrast to these promising results with watermelon juice enriched with L-citrulline other studies have reported no benefit (Shanely et al. 2016; da Silva et al. 2017; Chappell et al. 2018). For example, Shanely et al. (2016) reported no effect of watermelon puree consumption (980 mL/day for 14 days) on markers of inflammation in trained cyclists after a 75 km time trial. However, it is important to indicate that cycling (a predominantly concentric contraction sport) is not considered to be a damaging *per se*. Similarly, da Silva et al. (2017) found no effect of supplementation with 6 g of citrulline malate on muscle soreness or serum CK during the 72 h recovery period after a leg press and hack squat exercise when compared to placebo control. Finally, a more recent study (Chappell et al., 2018) reported that acute citrulline malate supplementation (4.21 g citrulline and 3.79 g malate) actually *increased* total muscle soreness during the 72-h period after isokinetic knee extensions (10 sets of 10 repetitions, 70% MVC) compared to placebo (6 g of citric acid) in 5 females and 13 males resistance-trained. This is one of the very few examples where a functional food has been shown to have a negative effect as opposed to a positive or null effect. On balance, L-citrulline and watermelon show some benefit to decreasing muscle soreness and preserving muscle function after EIMD.

**β-hydroxy β-methylbutyrate (HMB)**

β-hydroxy β-methylbutyrate (HMB) is a metabolite of the amino acid leucine and a potent stimulus for translation initiation and protein synthesis (Fitschen et al. 2013). Research during the last 20 years has investigated the effects of HMB supplementation on muscle soreness, indirect markers of muscle damage (Knitter et al. 2000; Paddon-Jones et al. 2001; Hoffman et al. 2004; van Someren et al. 2005; Nunan et al., 2010; Wilson et al. 2009; Wilson et al. 2013; Wilson et al. 2014) and circulating levels of pro-inflammatory cytokines (Portal et al. 2011; Gonzalez et al. 2014; Hoffman et al. 2016; Arazi et al. 2018b; Arazi et al. 2019) and showed mixed results. The first study (Knitter et al., 2000) to investigate the effects of HMB on EIMD used six weeks supplementation (3 g/day) prior to a 20 km run. The HMB supplementation was shown to reduce CK and LDH response after a prolonged run. Further work (van Someren et al., 2005) showed that HMB supplementation (3 g/day) associated with alpha ketoisocaproic acid (0.3 g/day) in eight male volunteers, for 14 d prior a damaging exercise bout of biceps curls at 70% of 1-RM, resulted in less muscle soreness at 24 h post bout, lower CK increases and overall in a greater muscle function, compared to a placebo. Similarly, beneficial effects of supplementation with the free acid form of HMB (3 g per day) was shown by Wilson et al. (2014) over a 12 weeks period. In addition, Hoffman et al. (2016) showed that 23 days of HMB supplementation in combat soldiers attenuated the

588 inflammatory response (IL-8, IL-10, TNF-alpha, granulocyte colony-stimulating factor, fractalkine and interferon-γ) to
589 intense military training, and preserved muscle quality. This suggested that a longer duration supplementation period
590 might be beneficial to influence inflammatory indices. Promising data also comes from the acute consumption of the free
591 acid form of HMB (3 g) that when ingested prior to a maximal eccentric exercise to attenuate LDH levels (Wilson et al.
592 2009), and reducing the increase of CK and improved the perceived readiness to train after a muscle-damaging resistance-
593 training (Wilson et al. 2013). More recent investigations have suggested that free acid form of HMB has greater
594 bioavailability in a shorter time when compared with calcium salt form of HMB (Arazi et al. 2019) and its
595 supplementation might represent a useful strategy to modulate exercise-induced muscle damage (Asadi et al. 2017) and
596 attenuate exercise-induced oxidative stress (Arazi et al. 2018b; Arazi et al. 2019).
597 In contrast to the aforementioned beneficial effects of HMB ingestion, Paddon-Jones et al. (2001) reported no
598 effects of 6 days of HMB supplementation (40 mg/kg body mass per day) prior to a bout of maximal isokinetic eccentric
599 contractions of the elbow flexors in non-resistance trained subjects. Additionally, Hoffman et al. (2004) studied the effects
600 of 10 days of HMB supplementation (3 g/day) and revealed that short duration of HMB supplementation did not provide
601 any benefit in reducing indirect markers of muscle damage (CK and MYO) during a preseason football training camp. In
602 a later study, Nunan et al. (2010) did not find positive effects of 14 days (11 days before and 3 days after a damaging
603 exercise) of 3 g/day of HMB associated with 0.3 g/day of alpha ketoisocaproic acid on muscle function, CK levels or
604 DOMS following a 40-min bout of downhill running, in fourteen untrained male subjects. This contradicted their previous
605 work (van Someren et al. 2005) that did show some benefit using the same supplement in an isolated muscle group.
606 Further, Portal et al. (2011) reported that seven-week consumption of HMB (3 g/day) did not change serum levels of IL-
607 6 and IL-1 during the early phase of training in elite volleyball players. Similarly, to those find null effects, Gonzalez et
608 al. (2014) showed that the acute administration of the free acid form of HMB (3 g) in recreationally resistance trained
609 males did not attenuate post-exercise markers of muscle damage, inflammation or performance following lower body
610 resistance exercise, when compared to placebo or a combination of cold water immersion and HMB-FA or placebo plus
611 cold water immersion. Studies have shown that both acute and chronic supplementation of HMB prior to exercise might
612 improve functional and biochemical markers of EIMD, however, it should be noted that this conclusion is based on a
613 limited number of studies, which are largely done in untrained individuals who are unaccustomed to eccentric exercise,
614 so the applicability to well-trained athletes is not clear.
615
616 **Protein and amino acids**
617 It is well known that protein intake plays a crucial role in the regulation of muscle protein turnover, particularly
618 in response to exercise (Phillips and Van Loon 2011), and that adequate intake should never be compromised in the diet.
619 However, there are doubts about the role of protein and amino acids supplementation in the prevention and treatment of
620 EIMD (Wojcik et al. 2001; Tipton et al. 2003; Blacker et al. 2010, Tipton 2015, Eddens et al. 2017). A systematic review
621 (Pasiakos et al. 2014) of the effects of protein ingestion on EIMD-related outcomes concluded that protein
622 supplementation had little effect on muscle damage which was attributable to large variations in study design and the
623 biomarkers selected. In addition, Poulios et al. (2019) confirmed there was no clear evidence to support a relationship
624 between changes in muscle damage indices with protein-based supplementation. Nevertheless, other studies have
625 suggested that hydrolysate whey protein (25 g) ingested immediately after the completion of maximal eccentric
626 contractions could alleviate muscle damage and enhances recovery of force following damaging exercise in sedentary
627 males (Buckley et al. 2010) cyclists (Eddens et al., 2017) and physically active females (Brown et al. 2018), or after
628 ingestion of isolated soy protein (25g) from boxers and cyclists (Shenoy et al. 2016). More recently, Ma et al. (2020)

reported that young runners who ingested 'immune protein' (10 g/day over a period of 2 months) obtained from cows immunised against specific antigens, had reduced inflammatory markers (TNF-α and IL-1β) post exercise, when compared to placebo treatment.

A systematic review by Fouré and Bendahan (2017) concluded that at least 200 mg per kg of body mass per day of branched chain amino acids (BCAA) for at least 10 days could be effective prophylactic to combat moderate muscle damage. In addition, a recent meta-analysis indicated that BCAA supplementation reduced delayed onset muscle soreness following exercise training (Fedewa et al. 2019). Likewise, Rahimi et al. (2017) reviewed randomized clinical trials and concluded that BCAA supplementation, before and during recovery days after damaging exercise, for at least one week, is able to attenuate muscle strength and muscle power loss. Previously, Nosaka et al. (2006) suggested that the ingestion of 9 essential and 3 non-essential amino acids before and after damaging exercise (7.2 g/day and for 5 days) attenuated indices of EIMD. Similar results were shown (Howatson et al., 2012) where BCAA supplementation for a total of 12 days (10 g twice a day) before and following damaging resistance exercise reduced indices of muscle damage in resistance-trained males. Similarly, Waldron et al. (2017) investigated the acute supplementation of BCAA (0.087 g/Kg of body mass, prior exercise) or placebo on EIMD outcomes in resistance trained athletes and showed improved rates of recovery in muscle function indices compared to placebo. Osmond et al. (2019) also reported the beneficial effect of BCAA (10 g twice a day per 11 days enriched with 5 g of additional leucine) on recovery following 100 drop jumps and afford no recovery benefits.

Collectively, despite the heterogeneity in study design and biomarkers, athletes might benefit from a BCAA supplementation (ratio 2:1:1 of l-leucine, valine, isoleucine) and hydrolysate whey protein to combat signs and symptoms of EIMD. However, further research is required to better understand the implications of different dosing and timing strategies on EIMD in addition to gaining a greater understanding of such interventions in well trained athletes.

**Milk**

Cows' milk contains ~80% casein and ~20% whey protein, thus providing a good balance of slow and fast digested amino acids to the muscle, which some researchers have hypothesized would have a beneficial effect on muscle recovery. Milk is the combination of a protein-carbohydrate beverage that has the potential to resolve EIMD (Cockburn et al., 2013). In a series of studies (Cockburn et al., 2008; 2010; 2012; 2013) showed that milk ingestion consumed post damaging exercise attenuated signs and symptoms of EIMD, such as muscle soreness and muscle function in team and individual sports. A further study (Rankin et al., 2015), male and female team sport athletes ingested 500 ml of milk immediately following a damaging eccentric-concentric exercise and showed smaller increases in soreness and serum CK and skeletal muscle troponin I compared to an energy-matched carbohydrate solution. Some athletes are intolerant to lactose and/or the A1 beta-casein protein, but A2 milk addresses these issues by offers an alternate, but otherwise identical milk (Kirk et al. 2017). Similarly, to whey hydrolysate protein and BCAA, milk could represent a potentially efficacious intervention, although the mechanism(s) underlying its positive effects is not fully understood; critically, milk is readily available to most individuals and is inexpensive,

**L-glutamine**

Glutamine is the most abundant amino acid in skeletal muscles and plasma and is an important fuel for immune system cells (Street et al. 2011). It has beneficial antioxidant and anti-inflammatory properties, so supplementation is considered potentially useful for athletes; for example, and recent research suggested that L-glutamine ingestion can restore plasma concentrations of glutamine (Nemati et al. 2019). In the setting of EIMD, L-glutamine was supplemented

for a short period, using a dosing protocol of 0.3g/Kg/day for up to 7 days, with beneficial effects on muscle function (Street et al. 2011) and on muscle soreness (Legault et al. 2015). Using higher doses (1.5g/kg/day for 7 days), Nakhostin-Roohi et al. (2016) showed beneficial effects on the CK response following strenuous exercise. However, at lower doses taken for longer periods (0.1g/kg/day for 28 days) L-glutamine did not improve muscle function or ameliorate muscle soreness (Rahmani et al., 2013). Although some rationale exists for the use this supplement to sustain immune function, further studies are needed to understand the application of recovery from EIMD.

**L-Carnitine**

L-Carnitine has the potential to ameliorate muscle damage signs and symptoms. In a recent review and meta-analysis, Yarizadh et al. (2020) described that L-carnitine supplementation resulted in significant improvements in muscle soreness following damaging exercise in both resistance-trained and untrained populations, when compared to a placebo. An earlier study (Giamberardino et al., 1996) showed that supplementation of L-carnitine (3g/day) alleviated pain, tenderness and appearance of CK in blood, indicating the potential role of the nutrient in reducing tissue disruption and leakage of cytosolic proteins. Other studies (Spiering et al., 2008; Kraemer et al., 2003; Volek et al., 2002; Spiering et al. 2007) also reported the ability of L-carnitine to reduce indices of muscle damage. For example, a daily intake of 2 g of L-carnitine compared to a placebo was accompanied by a reduction in released CK, MDA as well as hypoxanthine and xanthine oxidase (Spiering et al., 2008; Volek et al., 2002). Spiering et al. (2007) reported that both 1 g/day and 2g/day of L-carnitine supplementation, provided comparable benefits to modulate muscle soreness and indirect markers of muscle damage. Kraemer et al. (2006) investigated the effect of L-carnitine and L-tartrate supplementation over a period of 3 weeks and showed an increase of the level of androgen receptors on muscle cells, thus improving protein signalling that is necessary for recovery after exercise. Further, Parthimos et al. (2008) showed one month of L-carnitine supplementation (2 g/day) was able to maintain total antioxidant status, but it was unaltered post-exercise in basketball players. Similarly, Parandak et al. (2014) reported that 2g/day of L-carnitine over 14 days increased total antioxidant capacity before and 24 h post-exercise, when compared to a placebo, but no differences in muscle damage markers and lipid peroxidation were observed. Although these promising benefits, the exact mechanism(s) through L-carnitine improve outcomes of EIMD remain unknown and further investigations are warranted.

**Curcumin**

Curcumin is a natural compound and bioactive polyphenol found in turmeric (2%-5% by weight). It could represent a promising and beneficial strategy for attenuating EIMD because of the potential antioxidant and anti-inflammatory effects (Fernández-Lázaro et al. 2020). In line with this, some studies (Gaffey et al. 2017; Harty et al. 2019) have shown the antioxidants and anti-inflammatory effect of curcumin. It has can also attenuate muscle damage from thermal shock (Dunsmore et al. 2001) and reduce the increased activity of biomarkers of muscle damage such as CK (Kocaadam and Sanlier 2017). Supplementation of curcumin on sport performance and EIMD has been applied in elite rugby players (Delecroix et al. 2017), in moderately active subjects (Sciberras et al. 2015; Drobnic et al. 2014; Jäger et al. 2019; Nicol et al. 2015; Basham et al. 2019) and relatively inactive participants (Tanabe et al. 2019a; Tanabe et al. 2015; Nakhostin-Roohi et al. 2016; Tanabe et al. 2019b; McFarlin et al. 2016), but there are inconsistencies in findings across these studies.

Nicol et al. (2015) described that curcumin supplementation (5 g/day) taken 48 h before, and 72 h after eccentric exercise, reduced muscle pain (VAS scale -1.4 to -1.7) and blunted the CK activity (-22-29%) following damaging exercise (7 sets of 10 eccentric single-leg press repetitions), although paradoxically the post-exercise (at 0-h) IL-6 was

elevated relative to placebo (+31%). McFarlin et al. (2016) reported that CK, TNF-α and IL-8 responses were blunted (-48%, -25%, -21%, respectively) in subjects who consumed curcumin (400 mg/day for 2 days) prior to, and for 4 days after, damaging eccentric leg-press exercise; however, there were no differences in muscle soreness between supplemented and placebo groups. A similar response (Tanabe et al. (2015) of curcumin (150 mg 1 h prior to and 12 h after damaging eccentric elbow exercise) was observed, where declines in MVC were lower and recovery was faster (e.g., 4 days post-exercise: -31 ± 13% vs. -15 ± 15%) and peak CK was also smaller (peak:7684 ± 8959 IU/L vs. 3398 ± 3562 IU/L) than the placebo condition. Two studies from the same group (Tanabe et al. 2019a; Tanabe et al. 2019b) showed a significant reduction in muscle pain measured after 3-4 days (VAS scale) in subjects that ingested curcumin (90 mg twice daily) when administered four (Tanabe et al. 2019a) and seven (Tanabe et al. 2019b) days after eccentric exercise. In addition, compared to a placebo group, MVC torque and ROM were higher 3-7 days and 2-7 days after exercise, respectively (Tanabe et al. 2019b). These findings were supported by Jäger et al. (2019). who showed that 200 mg of curcumin was effective in preventing the decrease in MVC at 1 and 24 h after damaging exercise and showed less muscle soreness compared to placebo or lower dose of curcumin (50 mg). Similarly, Nakhostin-Roohi et al. (2016) showed that 150 mg of curcumin taken immediately after exercise reduced muscle pain at 48 and 72 h after eccentric exercise and was able to lower maximum CK, AST and ALT. Further evidence (Basham et al., 2019) for curcumin supplementation (1.5 g/day for 28 total days) abating EIMD exists, where 19 males completed 15 min of continuous sitting with one leg (a total of 225 repetitions). They showed that curcumin blunted CK (199.62 U/L vs287.03 U/L) and DOMS (2.88 vs 3.36) compared to the placebo. Most recently, Amalraj et al. (2020) reported that oral consumption of curcumin (Cureit$^{TM}$), which was developed based on the restructuring of the turmeric matrix with active curcumin using polar/nonpolar-sandwich (PNS) technology (Amalraj et al., 2017), decreased the pain VAS score (-59.77%) and the CK levels (-15.55%) following eccentric exercise. The PNS technology could be a strategy to overcome the limitations of curcumin bioavailability by increasing concentrations of free curcuminoids in the blood plasma and hence be beneficial in managing pain and DOMS (Jude et al., 2018).

In contrast with previous data, other researches failed to demonstrate benefits of curcumin supplementation (Delecroix et al. 2017; Sciberras et al. 2015; Drobnic et al. 2014) on EIMD outcomes. For example, Drobnic et al. (2014) reported a moderate reduction in pain at 48 h after downhill running (total score: 23.3 ± 7.9 vs. 30.6 ± 7.9, curcumin and placebo group, respectively) with 200 mg curcumin supplementation taken 48 h before and 24 h after exercise. Delecroix et al. (2017) showed that curcumin (6g/day) and piperine (60 mg/day) supplementation (48 h before and 48 h after exercise) in elite rugby players had a limited effect on power loss during the one leg 6 s sprint, without any effect on other aspects of muscle damage. Sciberras et al. (2015) showed that curcumin supplementation failed to produce differences in inflammatory markers after 2 hours of endurance cycling. The authors suggested that the lack of significance could be related to the sample size, mode and intensity of exercise or dose of curcumin used.

The mixed findings in studies could be the result of several limitations, including functional tests used, study settings and timing of supplement ingestion. Importantly, curcumin has poor aqueous solubility, has low absorption volume from the gut, is rapidly metabolised, and is excreted quickly (Anand et al., 2007). While conjugated curcumin achieves greater plasma concentrations around 1 hour after ingestion, a substantial amount of conjugated curcumin (up to a third) can be detected in plasma 24 hours post administration compared to only trace levels of non-conjugated curcumin (Asai and Miyazawa 2000). Although studies support that curcumin conjugates maintain similar molecular properties as the parent curcumin (Sandur et al. 2007; Pfeiffer et al. 2007; Kim et al. 1998; Pari and Murugan 2006), whether the magnitude of their bioactivity is comparable to parent curcumin remains unclear. Importantly, curcumin, like many other polyphenols, has poor bioavailability that can be improved with piperine co-ingestion. Piperine is the major

component of black and long peppers and has been shown to inhibit enzymatic conjugation of curcumin allowing greater levels of unconjugated curcumin to be absorbed into blood (Shoba et al. 1998) and increase curcumin tissue retention time. Notwithstanding, it appears that curcumin has good potential as an intervention to attenuate EIMD, particularly if metabolites are bio-available in a 24 h window.

**Bromelain and Proteases**

Bromelain is a mixture of proteases obtained from the stems of the developing pineapple fruit. Castell et al. (1997) showed that bromelain is absorbed and remains active following digestion with an estimated half-life of 6-9 h and that supplementation in small doses with other proteases has the potential to attenuate reductions in muscle function after damaging eccentric exercise (Buford et al. 2009; Miller et al. 2004). Miller et al. (2004) investigated the effect a combined protease supplement (4.2 g per day, one day and 4 days) prior to a 30 min downhill run. They reported a faster recovery of muscle function and reduced muscle soreness during the 72 h recovery period compared to a placebo group. Similarly, Beck et al. (2007) showed that strength loss immediately following eccentric elbow flexor contractions was reduced following acute protease supplementation. In addition, Buford et al. (2009) showed that protease supplementation (5.8 g per day for 21 days before downhill running and 3 days after) was able to attenuate decrements in MVC, a rise in basophil and eosinophil count and lower pro-inflammatory cytokines (IL-6, IL-12), cyclooxygenase-2. Conversely, Udani et al. (2009) found that a protease-containing supplement (258 mg of a proteolytic enzyme blend that included bromelain and other proteases) for 30 days prior eccentric exercise in untrained subjects did not modulate CK, CRP or cytokine activity. However, it did result in less tenderness 24 h post-exercise and less pain after 48 h of recovery compared to placebo. A more recent study by Shing et al. (2016) examined if markers of muscle damage and testosterone was influenced by acute bromelain in fifteen competitive cyclists that participated in a 10-stage cycle race over six consecutive days. Participants who ingested 1000 mg of bromelain (1500 GDU) twice a day for 6 days, reported lower sensations of fatigue and tended to maintain serum-free testosterone concentration across the race period compared to a placebo group. The exact mechanism(s) underlying the potential ergogenic effect of protease supplementation is poorly understood, but it might be related to reduced inflammation and hence improve recovery of strength loss after exercise (Beck et al. 2007). Thus, bromelain in isolation might have a limited effect, but there could be benefit when used in combination with other protease inhibitors. However, additional research is needed to understand the effects of proteases on EIMD in athletes and the underlying physiological mechanisms.

**Caffeine**

The effects of caffeine in reducing fatigue and increasing wakefulness, alertness (Burke 2008) and modulating pain perception through the action on adenosine receptors is well documented (Baratloo et al. 2016); however, limited research has examined caffeine's effects on exercise-induced muscle damage. Hurley et al. (2013) reported that caffeine consumption (5 mg/kg) 1 h before a damaging elbow flexion and extension exercise resulted in lower muscle soreness at 48 h and 72 h of a 5-day recovery period in resistance-trained males when compared to placebo group. This analgesic effect was later supported by Caldwell et al. (2017) with a lower dose of caffeine (3 mg/kg body mass) in males and females cyclists involved in a 164 km cycle ride. Furthermore, previous work (Chen et al. 2019) showed that acute caffeine (6 mg/kg body mass) facilitated recovery and attenuated muscle soreness after 30-min downhill running in a mixed sex cohort of college athletes.

Conversely, a series of studies conducted by Machado et al. (2009a; 2009b) found no effect following caffeine ingestion (5.5 mg/kg body mass) on biomarkers of muscle damage (serum CK and LDH) in professional soccer players.

The same group (Machado et al. 2010) reported that ingestion of caffeine at 4.5 mg/kg body mass, before exercise, did not reduce markers of muscle damage or leukocytes following moderate intensity resistance exercise session. Previously, Vimercatti et al. (2008) reported no benefit of caffeine supplementation on immune responses or muscle cell integrity. In this study, physically active males performed 60-min of treadmill exercise at 65% of $VO_{2max}$ following caffeine ingestion (4.5 or 5.5 mg/kg body mass 1 h before exercise) or placebo. There were no differences in blood biomarkers of damage and immune function between groups.

These data suggest caffeine could have an analgesic effect through the inhibition of adenosine on the central nervous system (CNS) by blocking the pain perception from the peripheral nerves to the CNS via adenosine receptors (Hurley et al. 2013). Thus, caffeine ingestion could be an intervention for individuals who are particularly susceptible to muscle soreness following training sessions or competitions. Although some recent studies demonstrated that caffeine ingestion was able to modulate others indices of EIMD such as muscle soreness (Hurley et al. 2013; Karabalaeifar et al. 2013, Maridakis et al. 2007) and performance (Hurley et al. 2013; Maridakis et al. 2007), there is a need for a greater understanding of the effects of time of day ingestion, habituation of use, training status and prolonged use of caffeine on post-exercise recovery.

**Taurine**

Taurine is a β-amino acid classified as a conditionally essential nutrient found in most human cells, with levels particularly high in excitable tissues such as skeletal and cardiac muscle (Schaffer and Kim 2018; De Carvalho et al. 2017). This amino acid is commonly known for its effects as an energizer and anti-fatigue properties; beyond this, it can exert other physiological functions that including membrane stabilization, osmoregulation and cytoprotection (De Luca et al. 2015). Regarding supplementation, taurine consumption is linked to diminished body mass index and reduced inflammation in obese women (Schaffer and Kim 2018). It is also reported to have antioxidant effects *in vivo* and a regulatory effect of calcium homeostasis on ion channel function in cardiac and skeletal muscle (De Carvalho et al. 2017). Recently, it has been reported that the ingestion of taurine might improve muscle function and reduce indices of EIMD. Specifically, Ra et al. (2013) investigated whether a combination of taurine and BCAA could attenuate indices of muscle damage in untrained males. The authors reported no effect of taurine supplementation alone (2 g/day, three times daily, for 14 days prior to, and for the 3 day recovery period), but when taurine was combined with BCAA (2 g/day and 3.2 g/day three times per day, respectively) a reduction of soreness and other damage indices were reduced in comparison to a placebo. Although the authors reported a synergic effect between taurine and BCAA, based on the evidence, it might be that 9.6 g of BCAA ingested per day could expedite signs and symptoms EIMD. However, to reinforce the potential use of taurine alone, Ra et al. (2015) showed that (2 g/day, three times daily, for 14 days) attenuated muscle soreness after an eccentric elbow flexion exercise during the 4-days recovery period. These data were supported by McLeay et al. (2017) who reported a faster recovery of eccentric torque in healthy active males who consumed taurine supplement (0.1g/kg body mass per day, 30 min prior exercise and during 3 days after exercise) after a bout of damaging elbow flexors exercise. These data concur with da Silva et al. (2014) who employed taurine a daily dose of 50 mg/kg body mass for 14 days before damaging exercise and for 7 days after, in 21 males. Finally, De Carvalho et al. (2017) administered a combination of taurine (3 g) plus 400 mL of chocolate milk ingested post exercise for 8 weeks. Results of this study indicated decreases in oxidative stress markers in triathletes, compared to a placebo. Taurine could be a useful tool, but data are far from conclusive. Notwithstanding, taurine might be a useful synergist supplement to other nutrients, but requires further investigation.

**Ginger**

Ginger (*Zingiberofficinale Roscoe; Zingiberaceae*) is a popular tropical spice used as a dietary supplement and food ingredient (Sellami et al. 2018). Several scientific reviews and meta-analyses have examined the use of ginger for the treatment of vomiting and nausea, cancer prevention, pain management and inflammation (Wilson 2015). Although the exact analgesic mechanism(s) is unclear, it seems that ginger blocks the activity of COX enzymes, leukotriene and prostaglandin synthesis. In addition, ginger inhibits the release of proinflammatory cytokines that play a role in exacerbating EIMD (Wilson 2015).

Black et al. (2010) showed in untrained participants, who ingested 2 g raw or heat-treated ginger per day for 11 days, less pain during the 24 h after eccentric elbow flexor exercise, relative to placebo. Matsumura et al. (2015) showed beneficial effects of ginger powder supplementation (4 g/day for 5 days) on a faster recovery of elbow flexor 1-RM strength during the 48 h recovery period after a damaging exercise, compared to placebo. Hoseinzadeh et al. (2015) reported that untrained females who completed 20 minutes of stepping exercise following 60 mg of ginger extract 1 h before showed a reduction of pain and IL-6 during the 48 h recovery period, when compared with consuming ginger immediately after the exercise and a placebo. This suggested that the timing of the ginger supplementation is an important consideration. Furthermore, Wilson et al. (2015) evaluated the effects of ginger root supplementation (2.2 g/day for 3 days before, the day of, and the day after exercise) on muscle function following a 20-22 mile training run, among 20 college students. Although median muscle soreness during jogging at 24 h after the training run was lower in the ginger root group compared to placebo, muscle function recovery was not different between groups.

Conversely, Black and O'Connor (2008) reported that an acute dose of ground ginger (2 g) ingested 30 minutes before 30 minutes cycling at 60% $VO_2$ peak in untrained females and males showed no differences in pain perception between treated and placebo groups. A subsequent study by the same authors (Black and O'Connor, 2010) noted that an acute dose of 2 g of ginger consumed by untrained participants 24 h after eccentric elbow exercise did not induce any improvements on pain intensity, range of motion, arm volume and metabolic rate the following day, relative to placebo. Overall, the limited evidence indicates no clear benefit of ginger on indices of EIMD.

**Ginseng**

There are several species of ginseng (Asian, Korean, Chinese, Canadian, Siberian and American (Sellami et al. 2018) with varying levels of active phytochemical compounds, the most important of which, is probably ginsenosides. Ginsenosides are thought to modulate immune function, glucose metabolism, enhance cognitive performance and affect oxidative stress (Bach et al. 2016). Recent evidence suggested that ginseng might have an important role in modulating markers of EIMD. Hsu et al. (2005) examined the influence of American ginseng (1.6 g/day for 4 weeks) on a damaging exercise bout of treadmill running in active college males. The authors showed lower levels of CK, compared to placebo, in the days after exercise. Likewise, Jung et al. (2011) showed that post exercise CK and IL-6 were reduced in male college students who ingested Korean red ginseng extract (20 g per 3 times/day for seven days prior, and for four days after eccentric exercise) compared to placebo group. Further, Pumpa et al. (2013) had well trained males complete intermittent downhill running treadmill exercise (5 bouts of 8 min running at -10% gradient) following consumption of 4g of Chinese ginseng 1 h before plus another 4g immediately after the exercise bout. They showed that muscle function was preserved and muscle soreness and pro-inflammatory cytokines (IL-6 and TNF-α) were reduced with a ginseng. Finally, Caldwell et al. (2018) investigated the effects of two doses of ginseng on perceived muscle soreness after resistance exercise in males and females. The authors showed that both lower (160 mg/day) and higher doses (960 mg/day) of Korean ginseng ingested 14 days prior to strenuous resistance exercise reduced changes in muscle soreness

at 24 post exercise. Whilst these preliminary data are promising, this strategy warrants further investigation.

**Vitamin C and Vitamin E**

Vitamin C (e.g. L-ascorbic acid) and vitamin E (α-tocopherol) are two essential micronutrients, with redox-dependent and independent biochemical functionality (Owens et al., 2019). Several studies have investigated the effects on EIMD indices and save few exceptions (Gabrial et al. 2018; Righi et al., 2020), the general consensus is that both vitamins show limited ability to positively impact the signs and symptoms of EIMD (Thompson et al. 2001; Thompson et al. 2004; Shafat et al., 2004; Connolly et al., 2006; McGinley et al., 2009; Sousa et al., 2014; Candia-Luján et al., 2014; de Oliveira et al. 2019; Owens et al. 2019).

Gabrial et al. (2018) examined the role of vitamin C intake in attenuating markers of muscle damage, oxidative stress and inflammatory responses in 20 recreationally active males that were regularly involved (2-3 times per week) in strenuous recreational exercise. The authors showed that longer term vitamin C ingestion (500 mg/day for 90 days) reduced CRP and uric acid when compared to a placebo group. Most recently, Righi et al. (2020) systematically reviewed the effects of vitamin C supplementation on oxidative stress, indirect markers of muscle damage and musculoskeletal functionality after a single bout of exercise; they reported that vitamin C supplementation attenuated lipid peroxidation and IL-6 elevation following a single session of damaging exercise.

Conversely, de Oliveira et al. (2019) showed no effects of vitamin C (500 mg/day) and vitamin E (400 UI/day) supplementation 7 days before and 7 days after plyometric exercise on EIMD indices. Notably the dosing strategy was much shorter than the aforementioned studies. In addition, a Cochrane review suggested a moderate to low quality of scientific evidence for the use of high dose of "antioxidant" supplements in reducing delayed onset muscle soreness (Ranchordas et al. 2017). Similarly, a previous systematic review reported the low effectiveness of vitamin C and E supplementation in reducing DOMS (Candia-Luján et al. 2014) that was echoed by other work (Connolly et al., 2006) that also found no benefit of vitamin C supplementation (1000 mg/day) ingested 3 days before and 5 days after damaging exercise. Two studies (Thompson et al., 2001; 2004) suggested that supplementation with vitamin C 400 mg/day for 14 days did not affect IL-6 levels after a downhill running exercise (Thompson et al. 2004); nor did a higher dose of vitamin C (1000 mg/day for 14 days prior to, and 2 h before) attenuate variables following 90 min of intermittent shuttle-running test (Thompson et al. 2001). In a recent review, Owens et al. (2019) highlighted the issues of measuring free radical and non-radical species to quantify redox balance in humans and suggested that vitamin C and vitamin E are unlikely to directly scavenge free radicals in the phagosome that interfere with the inflammatory responses. Thus, the use of vitamin E and vitamin C to modulate EIMD outcomes appears to lack support and further well-designed studies are warranted to better understand if lower doses for a longer period or a periodized approach to supplementation with either vitamin C or E alone, or in combination, in addition to a balanced and varied diet, can be used to reduce signs and symptoms of EIMD.

**Nutritional interventions with little or no evidence to support efficacy**

Exercise scientists have identified other potential interventions as solutions to manage the negative effects associated with strenuous physical activity. This section of the review offers a brief overview of a wide assortment of nutritional options with little evidence to support efficacy but that in the next future could attract researchers for additional investigations.

One of the most studied rich sources of polyphenols is green tea. Previous studies showed that green tea extract from *Camelia sinensis* supplementation reduced oxidative stress (Sugita et al., 2016), reduced muscle soreness (Herrlinger et al., 2015), decreased other indirect markers of muscle damage after eccentric exercise (da Silva et al., 2018), and

maintained neuromuscular function after a submaximal bout of exercise (Machado et al., 2018). Some promising beneficial effects were also observed following consumption of flavin-enriched black tea extract (Arent et al., 2010) and mate tea (Panza et al. 2016). Nevertheless, although there are some promising findings that green, black and mate tea polyphenols improve some outcomes of EIMD, research in this topic still is relatively weak and definitive conclusions about efficacy cannot yet be drawn because of the variations in study design, intervention protocols, eligibility criteria, statistical power, and timing of the intervention.

The exogenous antioxidants, Quercetin (Q), a polyphenol present in grapes, berries, tomatoes, apples, onions as well as in some medicinal plants such as *Hypericum perforatum* and *Gingko biloba* have been demonstrated to exert a variety of bioactive effects that are related to its anti-inflammatory and antioxidant properties (Abbey and Rankin, 2011; Malaguti et al., 2013; Bazzucchi et al., 2019; Askari et al., 2012). In addition, Martin-Rincon et al. (2020) investigated competition recovery in males and females following ingestion quercetin (140 mg) in combination with mango leaf extract that was rich in the polyphenol mangiferin (140 mg) one hour before exercise, followed by three additional doses every eight hours and reported an attenuation of muscle pain and a faster recovery of muscle performance. Conversely, other lines of enquiry failed to support the use of quercetin in managing EIMD in trained cyclists (Nieman et al. 2007a; McAnulty et al. 2008), ultramarathoners (Nieman et al., 2007b), healthy men and women (O'Fallon et al. 2012), young men (Patrizio et al. 2018) and male and female runners (Konrad et al. 2011). In summary, quercetin requires further research to clarify the most appropriate dose, timing and implication for longer term supplementation. Astaxanthin is also a powerful antioxidant naturally found in algae, fish and birds, has been shown to be effective against lipid peroxidation and oxidative stress *in vivo* and *in vitro* models. Some authors suggest that astaxanthin has small beneficial effect on managing EIMD (Djordjevic et al. 2012), conversely other studies (Bloomer et al. 2005; Klinkenberg 2013) demonstrated no benefit on muscle function nor antioxidant capacity.

In relation to EIMD, there is a growing interest from scientists and practitioners in the use of foods rich in phytochemicals, for example black chokeberry (Kim et al., 2019), acai (Carvalho-Peixoto et al., 2015; Sadowska-Krępa et al., 2015), grape (Kim and So, 2019; Dalla Corte et al., 2013), trans-resveratrol and polyphenol-enriched extracts (Jo et al., 2019) and cacao (Morgan et al., 2018; Peschek et al., 2013) but to date, there is very limited evidence to support efficacy on EIMD outcomes. Lyall et al. (2009) examined the consumption of a New Zealand Blackcurrant extract (equivalent to 48 g of blackcurrants) in male and female rowers and showed a blunted CK response at 24 h post-exercise and suppressed plasma protein carbonyls levels ($0.9 \pm 0.1$ vs. $0.6 \pm 0.1$ nmol/mg) compared to a placebo. Later, Hutchison et al. (2016) supported these findings and confirmed that consumption of a drink of blackcurrant nectar (16 oz, twice a day), reduced CK levels at 48 and 96 h after a session of eccentric knee extensions in college students, compared to placebo. Similarly, Coelho et al. (2017) reported that females who completed an maximum eccentric exercise of the bicep brachii following consumption of New Zealand blackcurrant extract (7 days before and 4 days following), reported less muscle soreness at 24 and 48 h post exercise, compared to a placebo, but no differences between groups in CK efflux or IL-6. The preliminary data from these studies show that blackcurrants might attenuate indices of EIMD, although the mechanisms have not been clarified. Like blackcurrants, blueberries and bilberries are also known for the anthocyanin and other phenolic compounds (Wu et al. 2004) that could be important modulators of oxidative stress and inflammation (McAnulty et al., 2004; Hurst et al., 2010 McLeay et al. 2012) in an EIMD scenario.

Tomato juice, rich in lycopene, vitamin C and other vitamins and minerals, could represents a protective food against oxidative stress and inflammation (Jacob et al. 2008). Previous studies investigated the effects of tomato juice on LDH, CK, C-RP and homocysteine (Tsitsimpikou et al. 2013) and oxidative stress exercise-induced (Harms-Ringdahl et al., 2012; Samaras et al. 2014) showing potential use of tomato juice as a strategy to modulate the negative outcomes of

EIMD. While tomato juice consumption has proven effective in ameliorating LDH and CK responses to anaerobic training (Tsitsimpikou et al. 2013) and reducing serum levels of 8-oxo-dG (Harms-Ringdahl et al., 2012), further studies must clarify doses, duration and better explain the mechanism(s) of action.

An area of growing interest is diet modulation of gut microbiota and, in particular, probiotics supplementation to influence immune functions (Gleeson et al. 2011), support healthy digestive and prevent respiratory illness or persistent common cold (Mach et al. 2017) in subjects who follow exercise programs. Other studies have shown potential beneficial effects between probiotics use, muscle damage, repair (Jäger 2016a; Jäger 2016b; Gepner 2017; Martarelli et al. 2011), fatigue (Shing et al. 2014) and inflammation (Lamprecht et al. 2012). Although the use of probiotics in athlete populations has attracted interest and is promising, the small number of studies and considerable differences in experimental approaches represents important limitations. There is little doubt that gut health will impact on whole system physiology, but the interaction between alterations in gut health and microbial populations and exercise performance, recovery and adaptation requires a great deal of work (Francavilla et al., 2017).

A growing interest arise for supplements containing collagen-specific peptides, or gelatine (partially hydrolysed collagen), due to its potential role on extra cellular matrix collagen synthesis supporting the remodelling process (Shaw et al. 2017; Li and Wu, 2018), on reduction of muscle soreness (Clifford et al. (2019) and on attenuation of CK activity following muscle-damaging exercise (Lopez et al. 2014). A recently published study showed that ingestion of 5 or 15 g of gelatine augmented bone collagen synthesis following acute mechanical loading (Shaw et al., 2017). As a result, it is tenable that collagen hydrolysate supplementation could attenuate some of signs and symptoms associated with EIMD.

Other nutritional strategies to improve recovery from exercise-induced muscle damage include methylsulfonylmethane (Barmaki et al. 2012; van der Merwe and Bloomer, 2016) selenium (Milias et al. 2006), allicin (Su et al. 2008), lemon verbena extract (Buchwald-Werner et al., 2018), flavanol-rich lychee (Nishizawa et al. 2011) saffron (Meamarbashi and Rajabi 2015), sesame (Barbosa et al. 2017), spinach (Bohlooli et al. 2015) melatonin (Cheikh et al. 2020) and sucrosomial® iron (Bongiovanni et al. 2019) specialized water (Borsa et al. 2013) and the leucine metabolite alfa-hydroxy-isocaproic acid (HICA) studied by Mero et al. (2010) and Teixeira et al. (2019) for its proposed ability to reduce training-induced inflammation and DOMS.

The aforementioned studies have tested the consumption of foods and supplements to reduce exercise-induced outcomes, but other options, as such caloric restriction and/or intermittent fasting, to modulate inflammation and oxidative stress was object of investigations (Dannecker et al. 2013; Pons et al. 2018). Fasting and caloric restriction are intriguing for their potential effects on markers of inflammation and oxidative status (Golbidi et al., 2017) but could they represent a possible application to modulate signs and symptoms of EIMD, particularly in athletic populations? Future studies should attempt to explore this fascinating topic.

**Limitations, practical applications and future perspectives**

Importantly, all supplements that contain antioxidants might be of limited benefit where the athlete's diet has a rich variety of fruits, vegetables, whole grains and nuts. A "food first" approach is highly recommended (Close et al., 2019), rather than processed capsules. Many foods contain sufficient phytochemicals to support the stressors associated with training and competition and aid recovery (Peternelj and Coombes 2011; Myburgh 2014). Exercise-induced muscle damage inflammation and oxidative stress are important elements in the activation of appropriate adaptive pathways that underpin repair of structures and exercise-induced improvements in health and immune function. Indeed, a growing body of evidence indicates that RONS production are a necessary response for exercise adaptations. Therefore supplements (e.g. antioxidant, anti-inflammatory) that completely mitigate or stop oxidative stress and inflammation

could alter these adaptive events. It is only when the exercise stressors might our-weigh the benefits that whole foods can afford that these supplements could be used to facilitate the recovery process. Critical to this mantra, is the need to assess the balance between the benefits these supplements might provide against the potential to interference with exercise-induced adaptations. Consequently, practitioners should consider a periodised approach where identification of higher-stress training and competition needs to be managed to facilitate timely recovery. Equally researchers should attempt to investigate the longer-term effect of the use interventions on the adaptative response and hence the ability to interfere or facilitate long term athlete development. Notwithstanding, there is currently no evidence to suggest that any functional food interventions (e.g., fruits, berries, cherries) affect the adaptive response. However, until this is systematically examined, the aforementioned idea of hormesis should be pragmatically applied to use interventions at an appropriate time (Figure 2).

From an applied perspective, how might this be applied? For example, in a preseason training phase, the best nutritional advice might be to follow a regular and varied diet and allow adaptations to occur naturally without the addition of supplements. However, when recovery is the priority, then the use of carefully planned interventions can be very helpful to restore function and abate soreness. One scenario could be during congested competition phases and busy travel schedules, such as those experienced in professional soccer, ice hockey, rugby, basketball and so on, where the main goal is to restore athletes back to the basal state as quickly as possible.

In the most-part, a majority of studies use heterogenous populations and variables, where fitness level, age, gender, sample size, outcome measures, short or longer term period of supplementation and study designs. For example, an important, but nonetheless often-overlooked limitation of some studies, was the use of a cross-over design in a damaging paradigm, which has been shown to confer a contralateral repeated bout effect and hence influence the damage in the contralateral limb (Howatson and van Someren, 2007). In addition, measures of REDOX balance at the systemic level often reveal limited mechanistic insights on the real activity of polyphenols with regard to oxidative stress and defence. Most measures reported in the exercise literature are fraught with limitations and should be treated cautiously to infer what might be occurring at the skeletal muscle level (Cobley et al. 2017).

Furthermore, other important issues to consider include bioavailability and pharmacokinetics: is the dose enough, too much or too little? Is it taken for long enough? When do levels peak in the body? Are the bioactive compounds stored or metabolised quickly? Should it be before or after bout of exercise? With this in mind, it is difficult to univocally translate science into practice. Overall, we classified supplements in three main groups, based on the current level of evidence (Figure 3) and the most frequently studied micronutrients to support EIMD, so called group “good level of evidence”, are summarized in Table 1. Importantly, this group of supplements are suggested to have a good deal research completed on them as opposed to the supplement being unequivocally effective. Despite this, there is still much to be learned regarding these interventions and how they might relate to EIMD, recovery and remodelling.

It is important that further studies be conducted, in male and female recreationally active and elite athletes. Other aspect that must be taken in account is that placebo-controlled, randomized control studies are difficult to design and conduct in elite athletes. Therefore, the importance of high-quality case studies can become increasing vital to inform the evidence base. It is also important that future research replicates how athletes actually utilize supplements and daily dietary programs in a real-world scenario in order to inform practice. Finally, there is a growing need to ensure that professional sports nutritionists receive the right information and education on what to use and when to use it, to correctly translate the evidence into practice.

**Conclusion**

This review provided an overview of EIMD and explored the nutritional strategies that could manage the negative signs and symptoms associated with EIMD. Very often there is little or no evidence-based practice in applying these nutritional interventions and hence it is difficult to ascertain an appropriate strategy to combat the negative effects of EIMD. Further research is warranted to elucidate the most appropriate dose, frequency and intensity of these interventions in order to determine "best evidence-based practice". Importantly, it is unclear how these interventions affect the adaptation process and it might transpire that treatments are being prescribed that attenuate adaptations; therefore, there is a need to ascertain the full implication of these interventions; 1) to reduce the negative effects of EIMD, and 2) understand the impact of these interventions on adaptation. With this in mind, a periodized approach to nutritional supplementation seems a sensible approach to adopt until further evidence is available, particularly for well-trained athletes.

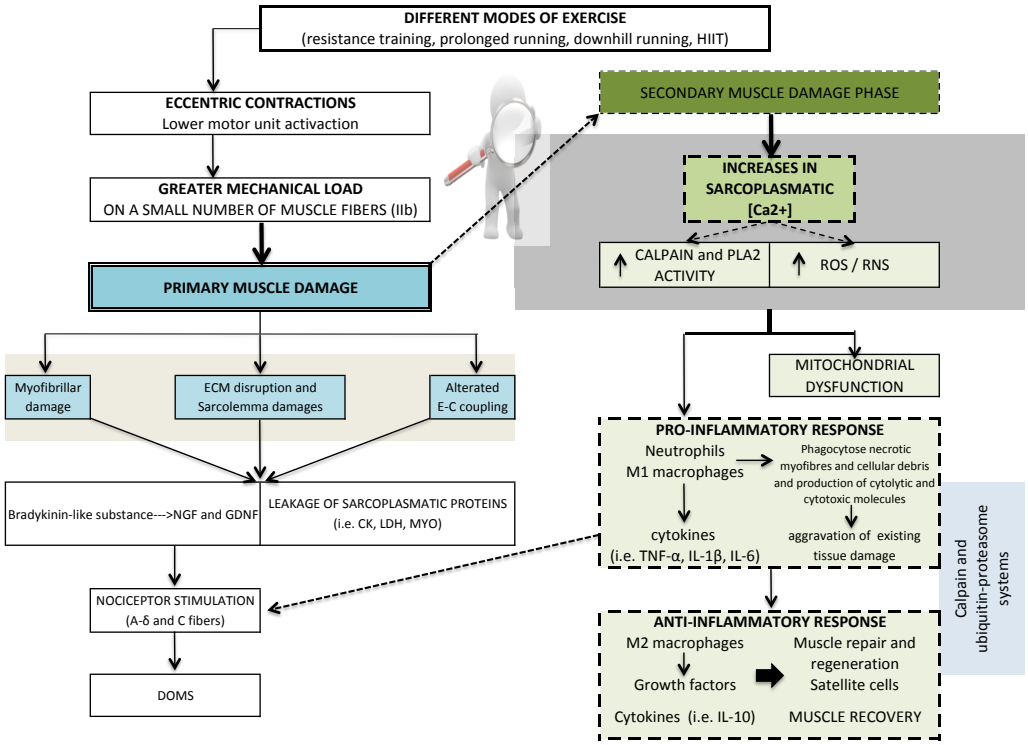


Fig. 1 Proposed mechanisms of Exercise-induced muscle damage

**Fig. 1**

Proposed mechanisms of Exercise-induced muscle damage

The potential damage model can be summarized into two general phases: an initial or *primary phase* (*left-hand side)*, triggered by eccentric contractions, beyond this, an increased permeability of the muscle cellular membrane, causing extracellular influx of $Ca2^{+}$ into the muscle fibre activates different $Ca2^{+}$ -sensitive proteases (calpains). Calpain activation leads to proteolysis of cytoskeletal and costameric proteins. Furthermore, a failure of excitation-contraction coupling also seems to play an important role in strength loss following strenuous exercise. The *secondary phase* (*right-hand side)* is characterized by neutrophils infiltrate to damaged muscle fibres and production of reactive oxygen and nitrogen species to degrade cellular debris. Neutrophils are substituted by macrophages M1 and in the latter stage of muscle damage, a shift from M1 to M2 macrophages is associated with the activation of satellite cells and the subsequent regeneration of muscle fibres. Neutrophils and macrophages also express tumour necrosis factor (TNF), which activates the ubiquitin-proteasome pathway that regulates proteolysis.

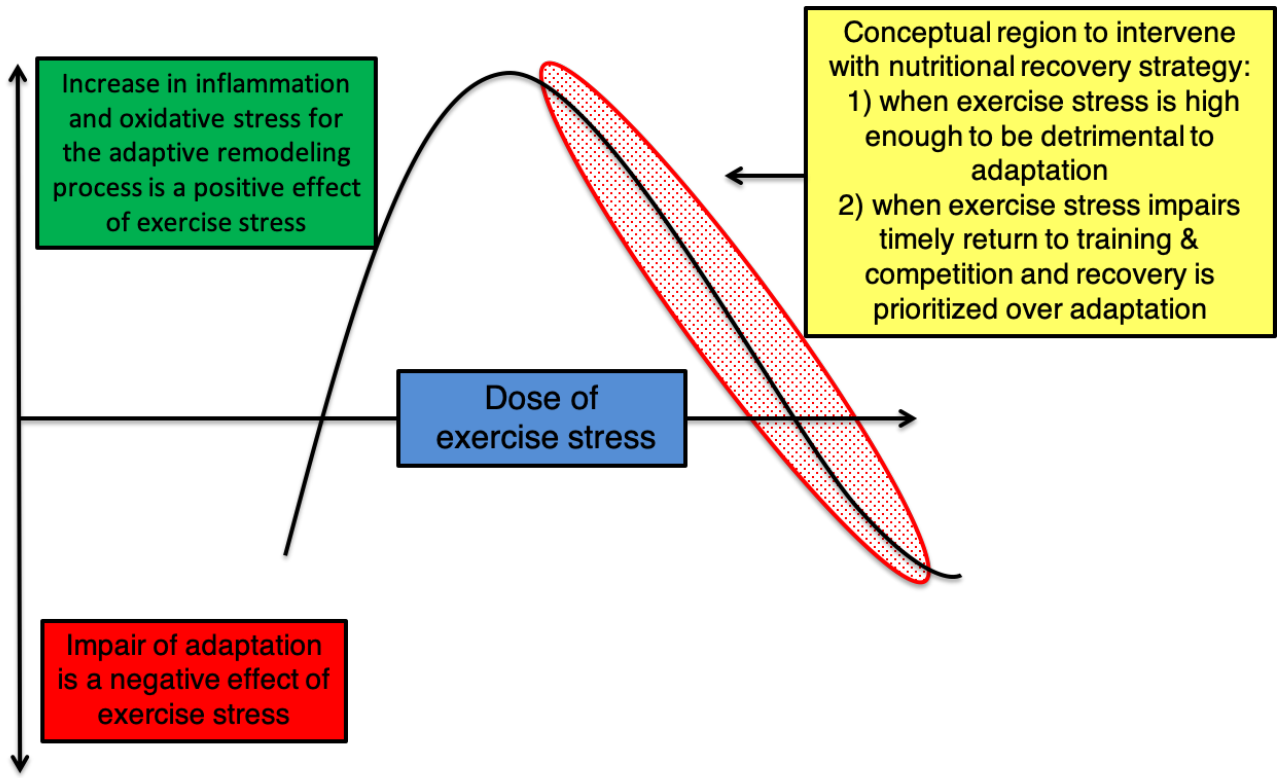


**Fig. 2** Model of hormesis in exercise recovery. Figure adapted from Howatson et al. (2016)

Hormesis theory in the context of nutritional interventions for the management of EIMD. This framework suggests that the adaptive response to EIMD presents as a bell-shaped curve. A positive effect of the exercise stress exists to a point when the exposure becomes too great, thereafter there is an impaired adaptive response. Using this theory, we suggest a conceptual region for intervention (yellow text box) where the exercise stress impairs timely return to training and competition or is detrimental to adaptation.

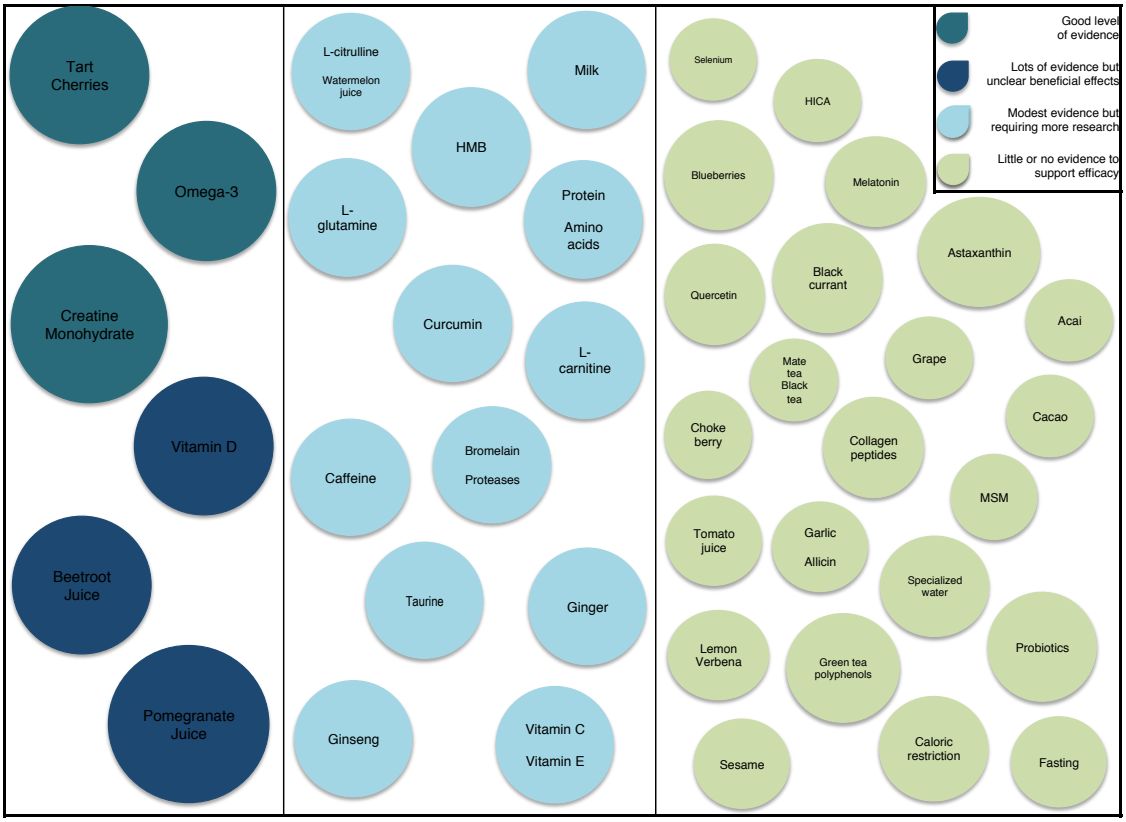


**Fig. 3** Proposed strategies for reducing signs and symptoms of Exercise-induced muscle damage

Overview of nutritional strategies proposed for manage EIMD outcomes and classified based on level of evidence.

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