ABSTRACT: In recent years, research investigating strategies to reduce exercise-induced muscle damage have become popular, with acute carbohydrate-protein supplementation gaining interest. The results of these studies are equivocal. A review of published peer-reviewed articles in reference to acute carbohydrate-protein supplementation and their impact on alleviating exercise-induced muscle damage is provided, in addition to an overview of the exercise-induced muscle damage process and rationale for their use. It can be concluded that there is potential for acute carbohydrate-protein supplementation to reduce some symptoms of exercise-induced muscle damage. Primarily, there is evidence of reduced increases in intramuscular proteins in serum and attenuated reductions in concentric muscle actions. However, there is little evidence of muscle soreness being alleviated. There are also substantial gaps in the literature, with information lacking in: (i) optimal dosage; (ii) optimal timing of supplementation; (iii) the effect on all paradigms of muscle function; and (iv) make-up of supplement(s), although whey protein concentrate and milk-based protein appear to provide benefits. Due to the conflicting results and the lack of studies conducted in this area it is difficult to provide definitive advice to the exercising individual. However, consuming carbohydrate-protein supplements would be recommended as they have demonstrated potential for reducing exercise-induced muscle damage and may be beneficial for other aspects of recovery.

KEYWORDS: Carbohydrate, Protein, Muscle Damage

INTRODUCTION

Exercise-induced muscle damage (EIMD) occurs following unaccustomed physical activity or activity of a high intensity or prolonged duration (Tee et al., 2007). It is commonly accepted that physical activity involving a high component of eccentric muscle actions is the primary initiating event stimulating EIMD (Proske and Morgan, 2001; Proske and Allen, 2005). In this respect, EIMD has been shown to occur following a number of different physical activities including resistance exercise with an eccentric component (Byrne and Eston, 2002a), vertical jumps (Twist and Eston, 2009), downhill running (Green et al., 2008), marathon running (Kobayashi et al., 2005) and endurance cycling (Saunders et al., 2007). The processes subsequent to the exercise bout may be different depending on the mode of exercise; however, there is wide acceptance with regards to the consequences of it. Evidence of EIMD manifests itself as increases in serum levels of intramuscular proteins (Sorichter et al., 2001a), increases in localised muscle soreness, commonly referred to as delayed onset of muscle soreness (DOMS) (MacIntyre et al., 2001), decreased muscle function (for review see Byrne et al., 2004), and changes in metabolism (for review see Tee et al., 2007). The extent of these perturbations is initiated by exercise insult, can be exacerbated over time and can last for several days, which has implications for subsequent exercise sessions. Therefore, any intervention that can potentially alleviate the negative consequences of EIMD may be beneficial to a range of exercising populations (Howatson and Van Someren, 2008).

Many studies have investigated interventions to attenuate the negative effects of EIMD, including pharmaceutical, therapeutic and nutritional methods, which have been extensively reviewed elsewhere (Howatson and Van Someren, 2008). Nutritional interventions are commonly investigated with protein-carbohydrate (CHO-P) supplements recently gaining interest (Wojcik et al., 2001; Saunders et al., 2004; Seifert et al., 2005; Baty et al., 2007; Saunders et al., 2007; Cockburn et al., 2008; Green et al., 2008; Rowlands et al., 2008; Valentine et al., 2008; White et al., 2008; Betts et al., 2009). The purpose of this review is to provide the reader with a current overview of the literature examining the effects of acute CHO-P supplementation following muscle damaging exercise.

PROCESS OF EIMD

There has been substantial research investigating the mechanisms underlying EIMD, but despite this the findings...
remain inconclusive. Many researchers have reviewed the mechanisms of EIMD (Armstrong, 1990; Armstrong et al., 1991; Clarkson and Sayers, 1999; Lieber and Friden, 1999; Proske and Morgan, 2001). Four stages have been proposed (Armstrong, 1990): initial, autogenic, phagocytic and regenerative. The purpose of this article is not to provide the reader with an extensive review of EIMD but an overview of the processes (figure I). This review simplifies these stages into two main events: primary and secondary.

**Primary Event**

There are two competing hypotheses for the primary event stimulating the process of EIMD: mechanical and metabolic. Mechanical stress is the most widely accepted proposal (Tee et al., 2007), and focuses on the distinguishing aspects of eccentric muscle actions, compared to concentric and isometric muscle actions (Armstrong et al., 1991). Theories relating to mechanical stress have primarily centred around excessive sarcomere strain during eccentric muscle actions due to the existence of sarcomere inhomogeneities (Julian and Morgan, 1979) on the descending limb of the length-tension curve. It is proposed that this leads to some sarcomere’s ‘popping’ as they are unable to maintain the tension (Morgan, 1990). This may lead to disrupted sarcomeres distributed throughout the muscle (Talbot and Morgan, 1996) with others remaining intact (Friden et al., 1981a; Newham et al., 1983; Nurenberg et al., 1992). Alternatively, rather than damage to the sarcomere occurring through mechanical stress it has recently been suggested that the primary event is due to excitation-contraction (E-C) coupling failure (Warren et al., 2001). Evidence for this hypothesis comes from measures of intracellular Ca$^{2+}$, however, increases could be secondary to mechanical change in the fibre (Yeung and Allen, 2004). Furthermore, E-C coupling failure as the primary event is more difficult to envisage as it is difficult to account for why disruption of the t-tubules (Takekura et al., 2001) is the primary site for damage and why it only occurs beyond optimum length (Proske and Morgan, 2001).

The metabolic hypothesis focuses on metabolic deficiencies in the working muscle due to exercise (Tee et al., 2007). Metabolic muscle damage has been proposed to occur from insufficient mitochondria respiration, the production of free radicals and high intramuscular temperatures, which have been reviewed elsewhere (Armstrong et al., 1991). These may render the muscle vulnerable to further damage via mechanical stress. However, there appears to be very little scientific research in support of this theory. Specifically, eccentric muscle actions have a lower metabolic cost than both concentric and isometric muscle actions when working at the same absolute loads, yet these latter types of muscle actions do not induce EIMD (Newham et al., 1983).

There are a number of theories relating to the primary event and the exact mechanisms remain inconclusive, although there is stronger support for mechanical stress. However, it is unlikely that either type of stress to the muscle occurs in isolation since all forms of exercise will incorporate a degree of both components (Tee et al., 2007). The contribution of each may be dependent upon the physical activity used to induce EIMD (Tee et al., 2007).

**Secondary Event**

Following the primary event there appears to be mechanisms occurring in the muscle that exacerbate the evidence of damage over time via autogenic and phagocytic processes. The most commonly accepted event is the disturbance of Ca$^{2+}$ homeostasis, which consequently initiates a number of proteolytic and lipolytic pathways. Increases in intracellular Ca$^{2+}$ may be due to damage to the sarcoplasmic reticulum (SR), failure of the SR to resequester Ca$^{2+}$, Ca$^{2+}$ uptake via stretch activated channels (SAC) and/or diffusion of extracellular Ca$^{2+}$ through the damaged sarcolemma (Evans and Cannon, 1991; Clarkson and Sayers, 1999; Yeung and Allen, 2004).

A number of proteolytic and lipolytic pathways have been implicated in the process of EIMD, which include the non-lysosomal proteasome calpain, phospholipases, lysosomal degradation via prostaglandin E$_2$ (PGE$_2$) production and the ubiquitin-proteasome (Ub-P) pathway (Armstrong et al., 1991). The autogenic phase involves the activation of calpain and phospholipases. Calpain cleaves protein structures including the cytoskeleton (desmin, α-actinin, titin, nebulin), myofibrillar (troponin and tropomyosin) and membrane proteins (Gissel, 2005). Furthermore, calpain may render the muscle fibre vulnerable to further damage via the Ub-P pathway and may also trigger further proteolytic pathways by attracting neutrophils to the site of damage (Belcastro et al., 1998; Raj et al., 1998). Phospholipases, specifically PLA$_2$, may result in the degradation of membrane phospholipids (Jackson et al., 1984) which may lead to increased permeability of the sarcolemma (Gissel and Clausen, 2001) allowing the efflux of intracellular proteins.

The phagocytic phase is primarily known as the inflammatory response and is characterised by increased infiltrating neutrophils, cytokines and macrophages (Tidball, 2005), which release reactive oxygen species (ROS) and proteases that can potentially cause further damage to the muscle (Malm et al., 1999; MacIntyre et al., 2001). The activation of ROS can lead to the lysis of the muscle cell membrane (Tidball, 2005), via further lipid peroxidation (Close et al., 2005). PGE$_2$ production and the Ub-P pathway may be
involved at this stage, with evidence demonstrating a role of the Ub-P pathway in the loss of myofibrillar protein (Thompson and Scordilis, 1994; Willoughby et al., 2003b; Willoughby et al., 2003a).

The process of EIMD involves a number of pathways with research suggesting it is bi-phasic. The primary event may cause direct damage to the muscle with the secondary event exacerbating the damage via increased protein degradation or change in the myofibrillar protein metabolism rate (Trappe et al., 2002). The extent to which each is involved in the process remains to be determined.

**Evidence of Damage**

Research examining direct evidence of EIMD has provided data demonstrating myofibrillar damage, evident as Z-disk disruption, the loss of desmin, disorganisation of the thick and thin filaments, loss of myofibrillar band registry and disturbances of the regular titin lattice (Friden et al., 1983; Newham et al., 1983; Friden and Lieber, 1996; Lieber et al., 1996; Friden and Lieber, 1998). The damage is observed to develop to more widespread areas in the 24-48 h following EIMD (Newham et al., 1983), possibly due to the progression of damage via the secondary event. There is also evidence of E-C coupling damage evident as structural changes involving the t-tubules and abnormal membrane systems with damage increasing 24-72 h post eccentric muscle actions (Takekura et al., 2001). The damage appears to be more focussed in fast twitch fibres (Friden et al., 1981b; Friden et al., 1983; Lieber et al., 1991; Vijayan et al., 2001), although some research demonstrates damage to slow twitch fibres (Sorichter et al., 2001b).

More commonly, however, indirect markers of EIMD such as intramuscular proteins in serum, muscle soreness and muscle function, are used to draw conclusions about damage to the muscle. Increases in intramuscular proteins in serum are commonly used as evidence of increased permeability or breakdown of the muscle cell membrane (Friden and Lieber, 2001). Increases in creatine kinase (CK) (Lee et al., 2002; Shahbazpour et al., 2004), myoglobin (Mb) (Cockburn et al., 2008) and lactate dehydrogenase (LDH) (Betts et al., 2009) measured in serum have been demonstrated following exercise with an eccentric component.

Muscle soreness, evident as tenderness and stiffness (Gulick and Kimura, 1996; Lieber and Friden, 2002) has been shown to increase from 24 – 96 h following muscle damaging exercise (Harrison and Gaffney, 2004; Twist and Eston, 2005; Twist et al., 2008). The use of muscle soreness as evidence of the magnitude of EIMD should be used with caution (Nosaka et al., 2002). The mechanisms underlying DOMS are out with the scope of this article but have been reviewed elsewhere (MacIntyre et al., 1995; Cheung et al., 2003), with the inflammatory response a commonly cited theory (Smith, 1991; MacIntyre et al., 1995).

Finally, but most importantly for the exercising individual, decrements in a variety of muscle function paradigms have been observed in the days following muscle damaging exercise. Isometric (Behm et al., 2001; Sayers and Clarkson, 2001; Harrison and Gaffney, 2004; Shahbazpour et al., 2004), concentric and eccentric (Eston et al., 1996; Behm et al., 2001; Byrne and Eston, 2002b; Twist et al., 2008) maximum voluntary contractions (MVC’s) are the most commonly used measure of muscle function and have been shown to significantly decrease following muscle damaging exercise. Countermovement, squat and drop jumps (Byrne and Eston, 2002b; Harrison and Gaffney, 2004; Marginson et al., 2005; Garcia-Lopez et al., 2006), peak power output (Byrne and Eston, 2002a; Twist and Eston, 2005), 10m sprints times (Twist and Eston, 2005) and endurance performance (Marcora and Bosio, 2007; Davies et al., 2009; Twist and Eston, 2009) have also all been shown to be negatively affected following muscle damaging exercise.

The mechanisms underlying reduced muscle performance include peripheral (myofibrillar and/or E-C uncoupling), central and impaired metabolism theories (Byrne and Eston, 2002b; Twist and Eston, 2005; Twist et al., 2008), with a complex interaction of these mechanisms most likely (Warren et al., 2002). Peripheral damage is the most common cited mechanism. Initial decrements in muscle function are thought to occur from E-C uncoupling and in the days following muscle damaging exercise, the degeneration of force transmitting and generating structures are implicated (Warren et al., 2002). However, it must be noted that most of the evidence is taken from animal models, therefore, caution must be taken when generalising to humans (Warren et al., 2002).

**ACUTE CHO-P SUPPLEMENTATION: EFFECTS ON EIMD**

Muscle pain, stiffness and the reduced capacity to exercise at optimal levels can affect subsequent exercise sessions. Therefore, interventions to alleviate these perturbations may be of benefit to the exercising individual. Acute CHO-P supplementation is one such intervention that has recently gained interest.

Eccentric exercise results in transient decreases in rates of skeletal muscle protein synthesis and enhanced rates of protein degradation (Fielding et al., 1991; Lowe et al., 1995). These changes in protein metabolism may be a causal factor of the ultrastructural damage observed (Lowe et al., 1995), leading to myofibrillar disruption, contractile protein loss and reduced cell membrane integrity. As a consequence intramuscular proteins in serum and muscle soreness may be elevated, and muscle function reduced. However, to the author’s knowledge a direct relationship between these perturbations and changes in protein metabolism has not been demonstrated, although a relationship between muscle performance and the percentage of desmin negative fibres has been observed (Lieber et al., 1994). It is possible that those fibres lacking desmin had undergone a degree of proteolysis (Lieber et al., 1994).

The ingestion of a milk protein concentrate immediately post muscle damaging exercise has been shown to attenuate decrements in isometric MVC and peak power output, without parallel attenuations of serum CK and DOMS (Etheridge et al., 2008). Reasons for alleviation of decrements in muscle function were reasoned to have been due to altered protein metabolism, with an increase in myofibrillar protein synthesis rate possibly leading to a greater production of contractile proteins (Phillips et al., 1997).
and therefore, recovery of muscle force (Etheridge et al., 2008). In contrast, although CHO intake is important for other aspects of recovery from exercise (glycogen re-synthesis), its consumption provides no benefit for alleviating EIMD in comparison to a control/placebo supplement (Wojcik et al., 2001; Cockburn et al., 2008; Green et al., 2008; Valentine et al., 2008).

A combination of protein/amino acids (AA) and CHO has been shown to increase protein synthesis and inhibit elevations in protein degradation that occur from exercise (Rasmussen et al., 2000; Bird et al., 2006; Tang et al., 2007). Therefore, the potent stimulatory effect of protein and CHO consumed together may augment muscle repair following acute muscle damage by increasing protein turnover (Biolo et al., 1997; Phillips et al., 1997). This may preserve and/or restore the structural integrity and function of skeletal muscle (Betts et al., 2009), and alleviate increases in intramuscular proteins and soreness, and decrements in muscle function. This has recently led to investigators examining the effect of combined CHO and protein ingestion on the attenuation of EIMD.

The results of the studies investigating acute CHO-P supplementation and EIMD (table I) are equivocal. Some studies report no benefit (Wojcik et al., 2001; Green et al., 2008; White et al., 2008; Betts et al., 2009) and others demonstrate reductions in markers of EIMD (Saunders et al., 2004; Seifert et al., 2005; Baty et al., 2007; Saunders et al., 2007; Cockburn et al., 2008; Rowlands et al., 2008) following acute CHO-P supplementation.

The studies that have been conducted have utilised different methodologies (model of EIMD; markers of EIMD and when measured; study design; type of CHO and protein in supplement; amount of supplement; timing of consumption; matching of supplements). This makes it difficult to provide those exercising with definitive advice and is likely why the results are equivocal. The primary aim of this article is to review the research conducted, discussing reasons for the conflicting results and provide recommendations for exercising individuals and future research. This section will consider human studies only. Two studies have been eliminated from this review as the supplement provided anti-oxidants in addition to CHO-P (Romano-Ely et al., 2006; Luden et al., 2007).

Current Research

The majority of studies demonstrating a benefit of acute CHO-P supplementation have centred on indirect markers of EIMD following endurance cycling (Saunders et al., 2004; Saunders et al., 2007; Rowlands et al., 2008; Valentine et al., 2008). These studies have demonstrated attenuations in increased CK between 15 – 24 h (Saunders et al., 2004; Saunders et al., 2007; Valentine et al., 2008) and Mb at 9 h (Valentine et al., 2008) following consumption of CHO-P before, during and/or after endurance cycling in comparison to CHO or a control supplement. However, the applicability of these studies to all forms of EIMD is questionable. Endurance cycling is largely concentric (Tee et al., 2007). The results of the studies investigating acute CHO-P supplementation and EIMD (table I) are equivocal. Some studies report no benefit (Wojcik et al., 2001; Green et al., 2008; White et al., 2008; Betts et al., 2009) and others demonstrate reductions in markers of EIMD (Saunders et al., 2004; Seifert et al., 2005; Baty et al., 2007; Saunders et al., 2007; Cockburn et al., 2008; Rowlands et al., 2008) following acute CHO-P supplementation.

<table>
<thead>
<tr>
<th>Study</th>
<th>Supplement</th>
<th>Exercise Protocol</th>
<th>Effect Post Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wojcik et al., (2001)</td>
<td>CHO-P (0.875 g/kg CHO, 0.375 g/kg PRO) immediately and 2 h post exercise; Protein: skim milk</td>
<td>10 x 10 eccentric actions of quadriceps at 120% 1RM</td>
<td>CK &lt; + + +</td>
</tr>
<tr>
<td>Saunders et al., (2004)</td>
<td>CHO-P (26g CHO, 6.5g PRO per 355 ml consumed during (1.8 ml/kg every 15 min) and within 90 min (10 ml/kg) post exercise; Protein: whey</td>
<td>Cycle to fatigue at 75 % VO2peak</td>
<td>CK ↓ at 15 h</td>
</tr>
<tr>
<td>Seifert et al., (2005)</td>
<td>CHO consumed before, during and after downhill skiing (total 58 g CHO, 24 g PRO); Protein: whey</td>
<td>Downhill skiing (8 – 12 runs)</td>
<td>CK ↓ at 2 h, Mb ↓ at 2 h</td>
</tr>
<tr>
<td>Baty et al., (2007)</td>
<td>CHO-P (6.2 % CHO, 1.5 % PRO) 30 min before (355 ml) immediately before (177 ml), during (177 ml) and post (155 ml) exercise; Protein: whey</td>
<td>2 x 8 reps at RM resistance exercise, 3rd set as many reps as possible</td>
<td>Mb ↓ at 6 h, CK ↓ at 24 h, DOMS ↓ at 24 h</td>
</tr>
<tr>
<td>Saunders et al., (2007)</td>
<td>CHO-P gel (0.146 g/kg BM CHO, 0.0385 g/kg BM PRO) consumed during (2 ml/kg BM every 15 min) and post (5 ml/kg BM exercise)</td>
<td>Cycle to fatigue at 75 % VO2peak</td>
<td>CK ↓ at 15 h</td>
</tr>
<tr>
<td>Cockburn et al., (2008)</td>
<td>CHO-P (138.2 g CHO/1 L, 33.4 g PRO/1 L) or MILK (49 g CHO/1 L, 34 g PRO/11) consumed immediately and 2 h post exercise; Protein: both semi-skimmed milk</td>
<td>6 x 10 reps eccentric actions of hamstrings at 1.05 rad/s</td>
<td>CK ↓ at 48 h, Mb ↓ at 48 h, DOMS ↓ + +, Peak Torque ↑ at 48 h, Total Work of Set ↑ at 48 h</td>
</tr>
<tr>
<td>Green et al., (2008)</td>
<td>CHO-P immediately, 30 min (1.2 kg BM CHO, 0.3 kg BM PRO) and 60 min (0.6 kg BM CHO, 0.15 kg BM PRO) post exercise; Protein: whey</td>
<td>30 min downhill running (-12 %) at 80 mph</td>
<td>CK + + +, DOMS ↓ + +, Isometric Peak Torque + + +</td>
</tr>
<tr>
<td>Rowlands et al., (2008)</td>
<td>Protein enriched recovery meal (1.6 g/kg FFMI/h CHO, 0.8 g/kg FFMI/h PRO) consumed post exercise; Protein: milk based</td>
<td>2.5 h interval cycling at relative intensities of 90 %</td>
<td>CK possible ↓ over 60 h, Mb ↓ at 9 h, Concentric leg extensions to fatigue at 70 %1RM ↑ at 24 h, DOMS ↓ + + +</td>
</tr>
<tr>
<td>Valentine et al., (2008)</td>
<td>CHO-P (77.5 g/h CHO, 19.4 g/h PRO) consumed during exercise; Protein: whey</td>
<td>Cycle to fatigue at 75 % VO2peak</td>
<td>CK ↓ at 24 h, Mb ↓ at 24 h, DOMS ↓ + +, Isometric MVC ↓ + +</td>
</tr>
<tr>
<td>White et al., (2008)</td>
<td>CHO-P (75 g CHO, 23 g PRO) consumed immediately before or post exercise; Protein: whey</td>
<td>50 eccentric actions of quadriceps at 1.05 rad/s</td>
<td>CK + + +, DOMS ↓ + +, Isometric peak torque hip and knee extensors and flexors + + +</td>
</tr>
<tr>
<td>Betts et al., (2009)</td>
<td>CHO-P (1.2 g/kg BM CHO, 0.4 g/kg BM PRO) before, during and for 4 h post exercise; Protein: whey protein isolate</td>
<td>90 min LST</td>
<td>CK + + +, Mb ↓ + +, LDI ↓ + +, R-L ↓ + +, DOMS ↓ + +, Isometric peak torque hip and knee extensors and flexors + + +</td>
</tr>
</tbody>
</table>

3-MH = 3-methylhistidine; BM = body mass; CHO = carbohydrate; CK = creatinine kinase; CRP = C reactive protein; DOMS = delayed onset muscle soreness; FFMI = fat free mass; I = interosseus; LDH = lactate dehydrogenase; LST = long b Sarah intermittent shuttle test; MVC = maximum voluntary contraction; PRO = protein; RM = repetition maximum; TNF-α = tumor necrosis factor α; ↑ = indicates higher values; ↓ = indicates lower values; + + + indicates no difference between groups; + + compared to control/placebo; + compared to CHO-P.
Evidence that indirect markers of EIMD can be independently or muscle soreness (Etheridge et al., 2008). This study provides may explain the equivocal results. Of muscle soreness is subjective (Rodenburg et al., 1993), which lower CK and Mb levels with supplementation. The measurement Valentine et al., (2008) found DOMS was not reduced despite and after alpine skiing reduced CK and Mb 2 h post exercise (Seifert et al., 2005).

Taken together these results would appear to suggest that the consumption of CHO-P before, during and/or after exercise causing muscle damage reduces EIMD in comparison to a CHO or control supplement. This may be via an improved protein balance possibly through the ingestion of CHO-P increasing the extracellular AA concentration and driving protein synthesis whilst inhibiting increases in protein degradation.

Conclusions of these studies have, however, been based primarily on measures of intramuscular proteins in the serum, with CK frequently used. CK is a highly variable marker of EIMD, which is problematic when using an independent groups design (Batts et al., 2009). Few studies concluding benefits of CHO-P supplementation, based on measures of CK, utilised an independent design (Seifert et al., 2005; Baty et al., 2007). Although cross-over designs may be methodologically stronger when using CK as the only marker of EIMD, there use may be problematic in EIMD studies due to the repeated bout effect (McHugh, 2003). There are other issues with the use of CK. Vigorous exercise has been associated with the release of CK from sources other than muscle (Piper et al., 1984). Furthermore, poor correlations between CK and changes in Z-band streaming have been reported (Fielding et al., 1993; Malm et al., 2000; Beaton et al., 2002). However, it should be noted that a lack of correlation may be due to other factors: CK data is obtained from venous blood were as muscle histology such as Z-band streaming is assessed by muscle biopsies and there is high inter-individual variability of CK, both of which may reduce correlations (Saunders, 2005). Due to these factors it is difficult to conclude with certainty from these studies that acute CHO-P supplementation alleviates EIMD.

From an applied perspective, the exercising individual needs to know if acute CHO-P supplementation will alleviate soreness, stiffness and the reduced capacity to exercise. From the studies discussed only two assessed muscle soreness (Baty et al., 2007; Valentine et al., 2008). Baty et al., (2007) reported reduced DOMS at 24 h following muscle damaging exercise. However, Valentine et al., (2008) found DOMS was not reduced despite lower CK and Mb levels with supplementation. The measurement of muscle soreness is subjective (Rodenburg et al., 1993), which may explain the equivocal results.

Changes in biochemical markers are also likely to be functionally irrelevant. The consumption of a milk protein concentrate following muscle damaging exercise has been found to attenuate decrements in muscle function, without parallel reductions in CK or muscle soreness (Etheridge et al., 2008). This study provides evidence that indirect markers of EIMD can be independently affected, likely due to the different degradative pathways responsible for changes in these variables (figure I). Lipolytic pathways (phospholipase and PGE2) are predominantly implicated in the release of intramuscular proteins with proteolytic pathways (Ub-P, calpain) responsible for myofibrillar degradation and thus decrements in muscle function.

Valentine et al., (2008) investigated muscle function by measuring leg extensions to fatigue at 70 % of participants 1 repetition maximum (1RM) and found more repetitions were complete 24 h following muscle damaging exercise with CHO-P compared to CHO consumption. However, it is difficult to state with certainty that these results were due to a reduction in EIMD. The improved muscle function with CHO-P ingestion may have been due to an improvement in glycogen re-synthesis due to the addition of protein. Using measures of peak torque, Cockburn et al., (2008) demonstrated attenuated reductions in peak torque at 48 h post exercise following CHO-P consumption in comparison to a control and CHO supplement. Both of these studies measured biochemical markers and muscle soreness, demonstrating reduced CK and Mb but no benefits for muscle soreness. Together these studies provide evidence that acute CHO-P supplementation is beneficial for alleviating some perturbations of EIMD and provide an indication that subsequent exercise could take place at closer to optimal levels, which would be of benefit to the exercising individual.

Measuring muscle function in conjunction with a variety of other indirect markers (CK, Interleukin-6 (IL-6), 3-methylhistadine (3-MH), DOMS, isometric peak torque) no beneficial effects of CHO-P consumed post eccentric exercise were observed compared to CHO and a control group (Wojcik et al., 2001). However, eccentric actions of the quadriceps to induce EIMD were completed following a bout of glycogen depleting exercise. Temporary damage of the muscles in a glycogen depleted state may have confounded results. More importantly from an applied perspective, it could be argued that exercising individuals do not train in a glycogen depleted state and therefore, this does not reflect practices that could be generalised to applied settings. 3-MH was assessed and found to increase 24 h post when compared to 1 day prior to eccentric exercise with no effect of nutrient intake. This is in contrast to the observed reduction in 3-MH excretion 48 h post resistance exercise with consumption of a CHO/essential AA supplement (Bird et al., 2006). 3-MH values on day 1 may not have been reflective of baseline samples, as participants had completed the bout of muscle glycogen depleting exercise.

Similarly, other investigations have observed no alleviation of EIMD using functional, biochemical and subjective markers (Green et al., 2008; White et al., 2008; Betts et al., 2009). Aspects of methodology were similar to other investigations observing benefits; however, there were also distinct differences. Primarily, the studies that observed no benefit used isometric MVC’s as a measure of muscle function (Wojcik et al., 2001; Green et al., 2008; White et al., 2008; Betts et al., 2009), where as those observing a benefit used concentric muscle actions (Cockburn et al., 2008; Valentine et al., 2008). This may imply that reductions in muscle actions
other than isometric can be attenuated with acute CHO-P supplementation. However, there are other methodological differences between the studies. For example, both White et al., (2008) and Cockburn et al., (2008) used a similar model of EIMD, an independent group design and assessed similar markers at the same time points following EIMD, with contrasting results. Differences in the type of protein consumed, participants recruited, muscle group damaged and amount of supplement provided may be implicated in the different outcomes. Similarly, using the same bout of damaging exercise (downhill running) and assessing similar indirect markers at the same time points to Etheridge et al., (2008), no beneficial effects of acute CHO-P supplementation were observed by Green et al., (2008). Differences may be attributed to contrasting designs (independent v cross-over), type of protein, timing of supplementation and gender of participants. In the context of EIMD and acute CHO-P supplements only Green et al., (2008) has recruited female participants. It has been suggested that females may demonstrate faster recovery rates than males following EIMD (Sayers and Clarkson, 2001), which may have influenced results.

Methodological differences can confound results for a number of reasons and this is a likely reason for why there is inconsistent information regarding the benefits of acute CHO-P supplementation for alleviating EIMD. Firstly, the accurate interpretation of the effectiveness of CHO-P intake is difficult because timing of ingestion differs between studies, occurring before, during and/or after exercise. If supplements are to be investigated to improve recovery then ingestion at different time points may confound findings as it is difficult to determine if the positive effects are due to a reduction in the initial damage, enhanced recovery or a combination of both (Green et al., 2008). Furthermore, if benefits are a result of altered protein metabolism, then CHO-P intake at different time points may alter the response (Tipton et al., 2001).

Secondly, a number of studies have assessed indirect markers of EIMD at one time point. It is accepted that the histological evidence of damage is exacerbated over a number of days (Newham et al., 1983). Therefore, to assess the attenuation of EIMD, markers of EIMD should be assessed over days to provide an accurate picture of the change occurring. Furthermore, by assessing only one time point, potential benefits of supplementation may be missed.

Thirdly, using different models of EIMD may affect results due to the different type (metabolic v mechanical) of muscle stress. Prolonged endurance exercise is likely to primarily induce metabolic stress, which may influence the level of activation and types of degenerative pathways. The degree of damage may also affect results. The majority of studies providing evidence in support of the use of CHO-P show low to moderate levels of muscle damage (Baty et al., 2007; Saunders et al., 2007; Rowlands et al., 2008; Valentine et al., 2008), as assessed by CK levels. Investigations demonstrating no positive effect primarily produce high degrees of damage (Wojcik et al., 2001; Green et al., 2008; White et al., 2008; Betts et al., 2009). This may imply that acute CHO-P supplementation alleviates EIMD, when damage is not severe. However, Saunders et al., (2004) and Cockburn et al., (2008) both demonstrated benefits of CHO-P intake with severe damage. However, using CK to determine the magnitude of damage is problematic as it does not provide this information (Friden and Lieber, 2001). Furthermore, using CK to assess differences between studies should be done with caution as CK has a high inter-individual variance and it may not have been measured at standardised temperatures (Betts et al., 2009).

The effects of CHO-P on EIMD have been examined by comparing to CHO matched for CHO content (Saunders et al., 2004; Saunders et al., 2007; Green et al., 2008; Valentine et al., 2008; Betts et al., 2009), energy (Wojcik et al., 2001; Valentine et al., 2008) or volume (Cockburn et al., 2008) and a control or a control only (Seifert et al., 2005; Baty et al., 2007; White et al., 2008). Although using isovolumetric comparisons may be more practically relevant, it is difficult to state with certainty that differences are due to the addition of protein. The greater caloric content or macronutrient composition may contribute to differences. Furthermore, by matching for CHO only then the extra energy content is not accounted for and may impact on results.

Lastly, studies have used both different types of CHO and protein in the supplement. CHO intake increases insulin, which has been linked to the inhibition of increases in protein degradation following exercise (Biolo et al., 1997). Therefore, different types of CHO may affect results if the mechanisms underpinning the attenuation of EIMD are explained by altered protein metabolism. However, this is unlikely as postprandial insulin response is not significantly affected by different types of CHO co-ingested with protein following resistance exercise (Kreider et al., 2007), and insulin does not play a role in the regulation of myofibrillar protein degradation (Bird et al., 2006).

The addition of protein to CHO appears to be the key nutrient for influencing EIMD and therefore, the type of protein consumed may be important. The investigations conducted in this research area have used either whey or milk-based protein. Using milk based protein the majority of studies (Cockburn et al., 2008; Etheridge et al., 2008; Rowlands et al., 2008) have demonstrated potential benefits. Only Wojcik et al., (2001) did not support these results, but as previously discussed this is the only study to damage muscles in a glycogen depleted state. Furthermore, skimmed milk was used and the fat content may be important for inflammatory factors. Using whey protein the results are more equivocal. It is difficult to ascertain why this is. The majority of studies demonstrating a reduction in EIMD with whey protein have used whey protein concentrate (Saunders et al., 2004; Baty et al., 2007; Saunders et al., 2007; Valentine et al., 2008). Betts et al., (2009) who reported no reduction in EIMD used whey protein isolate, whilst White et al., (2008) who had similar results did not provide this information. These results may imply that the type of protein is important with milk based or whey protein concentrate being the most beneficial. However, Green et al., (2008) used whey protein concentrate and observed no difference in EIMD with a CHO-P supplementation. Factors other than protein type may have confounded results, such as the use of female participants.
**Practical Applications**

In terms of providing exercising individuals with advice, there is evidence suggesting a potential benefit of acute CHO-P supplementation. There is potential for acute supplementation to attenuate reductions in concentric muscle actions and reduce increases in intramuscular proteins in the serum. Alleviating decrements in muscle function would allow the exercising individual to continue exercising at closer to optimal levels on subsequent days. However, there does not appear to be a benefit for alleviating muscle soreness.

Due to the lack of literature and varied methodologies, there is little information regarding when CHO-P should be consumed around an exercise bout and how much. However, the majority of research conducted has used commercially available products, which makes it easier for the exercising individual to apply some of the information from the research to their own practices. As well as potential benefits in alleviating EIMD the consumption of certain CHO-P supplements may have benefits for hydration (Shirreffs et al., 2007) and glycogen re-synthesis (Karp et al., 2006). Therefore, the use of CHO-P supplements should be included in nutritional strategies for recovery.

**CONCLUSION**

Based on the current knowledge, there is potential for favourable results with acute CHO-P supplementation in the context of EIMD. Of the research conducted there are vast methodological differences and this makes it difficult to conclude with certainty on a number of factors: (i) timing of consumption; (ii) amount of supplement; (iii) effects on different paradigms of muscle function; and (iv) the type of supplement(s). However, regarding the type of supplement, there is evidence to suggest that milk based protein and whey protein concentrate may have to be included in the CHO-P supplement to alleviate EIMD. Milk based protein and whey protein concentrates are both commercially available, therefore, supplements of this nature may provide the most beneficial option when including CHO-P supplements in nutritional recovery strategies.

It is apparent that there are substantial gaps in the literature and that future research is warranted. This should focus on addressing areas that may impact on exercising individuals and conflicting methodologies:

1. Potential mechanisms of reduced EIMD: Very few studies investigating the effects of acute CHO-P supplementation on EIMD have included measures of protein degradation. It is postulated that the benefits of CHO-P are via altered protein metabolism; therefore, future studies investigating the use of CHO-P on attenuating EIMD should assess protein synthesis and degradation in conjunction with indirect markers of EIMD. More studies have measured cytokines (Wojcik et al., 2001; Rowlands et al., 2008; Betts et al., 2009) with no studies reporting changes in these variables due to supplementation, despite reduced EIMD (Rowlands et al., 2008). Theoretically, nutritional supplements would minimise secondary damage (Bloomer and Goldfarb, 2003), which is characterised by the inflammatory response. Although research has not demonstrated changes in markers of inflammation, future research should investigate this further.

2. Standardised timing of supplementation: Future investigations should focus on providing CHO-P at a set time point or comparing timing of supplementation of which only one study has done (White et al., 2008).

3. Measures and timing of assessment: Investigations should ensure that a variety of biochemical, subjective and functional measures are assessed and measured at a variety of time points over a number of days. This is to ensure that a complete picture of EIMD and acute supplementation is obtained. With regards to functional measures, the majority of studies have used isometric (Wojcik et al., 2001; Green et al., 2008; White et al., 2008; Betts et al., 2009) or concentric (Cockburn et al., 2008; Valentine et al., 2008) MVC’s as measures of muscle function. Since these assessments have limited external validity, future studies should make an effort to measure dynamic muscle function that can be applied to the exercising individual.

4. Type of supplement: For applied reasons, researchers should use supplements that are readily available so that if benefits are observed the exercising individual is able to purchase the supplement easily and cheaply. The type of protein in the supplement should also be considered. Researchers should also consider the rationale for studying CHO-P supplementation and use this to match supplement comparisons.

5. Statistical methods: Each study reviewed except one (Rowlands et al., 2008) utilised traditional null hypothesis testing based on a p value. If research investigating CHO-P supplementation is to be used in the applied setting then the use of magnitude based inferences (Batterham and Hopkins, 2006) should be considered. This method defines the smallest biological or practical effect, allowing the researcher to quantify the probability of a worthwhile effect with inferential descriptors to aid interpretation (Rowlands et al., 2008). Magnitude based inferences recognise sample variability (Rowlands et al., 2008) and provide scientists, support staff and athletes with an indication of the meaningfulness of the results.

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