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ORIGINAL ARTICLE

# Effect of volume of milk consumed on the attenuation of exercise-induced muscle damage

**Emma Cockburn · Paula Robson-Ansley · Philip R. Hayes · Emma Stevenson**

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**Abstract** Exercise-induced muscle damage (EIMD) leads to decrements in muscle performance, increases in intramuscular proteins and delayed-onset of muscle soreness (DOMS). Previous research demonstrated that one litre of milk-based protein–carbohydrate (CHO) consumed immediately following muscle damaging exercise can limit changes in markers of EIMD possibly due to attenuating protein degradation and/or increasing protein synthesis. If the attenuation of EIMD is derived from changes in protein metabolism then it can be hypothesised that consuming a smaller volume of CHO and protein will elicit similar effects. Three independent matched groups of 8 males consumed 500 mL of milk, 1,000 mL of milk or a placebo immediately following muscle damaging exercise. Passive and active DOMS, isokinetic muscle performance, creatine kinase (CK), myoglobin and interleukin-6 were assessed immediately before and 24, 48 and 72 h after EIMD. After 72 h 1,000 mL of milk had a likely benefit for limiting decrements in peak torque compared to the placebo. After 48 h, 1,000 mL of milk had a very likely benefit of limiting increases in CK in comparison to the placebo. There were no differences between consuming 500 or 1,000 mL of milk for changes in peak torque and CK. In conclusion, decrements in isokinetic muscle performance and increases in CK can be limited with the consumption of 500 mL of milk.

**Keywords** Carbohydrate · Protein · Milk · Muscle damage · DOMS · Muscle performance

Communicated by Susan A. Ward.

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## Introduction

Exercise-induced muscle damage (EIMD) is a common phenomenon associated with activities involving eccentric muscle actions. EIMD leads to the disruption of muscle structures and is associated with a number of symptoms. The delayed-onset of muscle soreness (DOMS) is commonly reported (MacIntyre et al. 2001; Semark et al. 1999), and there is a decreased ability to produce force (Byrne and Eston 2002; Harrison and Gaffney 2004; Twist and Eston 2005). There is also an increase in intramuscular proteins measured in the plasma (Seifert et al. 2005; Sorichter et al. 2001). DOMS and reduced force production can limit an athlete's ability to train and perform.

The use of protein–carbohydrate (CHO) supplements has been extensively studied (Baty et al. 2007; Betts et al. 2009; Cockburn et al. 2008, 2010; Gilson et al. 2010; Green et al. 2008; Saunders et al. 2004, 2007; Seifert et al. 2005; Valentine et al. 2008; White et al. 2008; Wojcik et al. 2001) with equivocal results. Research observing benefits have speculated that they are due to the combination of carbohydrate and protein, which alters protein metabolism (Borsheim et al. 2004; Tang et al. 2007). This may then limit the breakdown and/or increase the repair of muscle protein structures limiting myofibrillar disruption and the loss of cell membrane integrity. As a consequence, increases in intramuscular protein release and decrements in muscle performance would be limited.

The authors have previously demonstrated that acute supplementation with 1,000 mL of milk or milk-based protein–CHO consumed immediately following damaging exercise attenuates EIMD (Cockburn et al. 2008, 2010). These studies provided participants with 1,000 mL of the supplement, equating to 34 g protein, 118 g CHO and 707 kcal for the milk-based protein–CHO supplement, and 34 g protein, 49 g CHO and 480 kcal in milk. This volume is quite high, especially in terms of caloric content. Furthermore, consumption of this high volume following exercise may lead to stomach fullness and discomfort. Other investigations demonstrating a positive impact of protein–CHO supplementation have provided participants with varying amounts of CHO and protein (Baty et al. 2007; Valentine et al. 2008; Wojcik et al. 2001). Moore et al. (2009) demonstrated that muscle protein synthesis reaches maximal stimulation after the consumption of 20 g high-quality intact protein, suggesting an upper limit for the incorporation of amino acids into proteins. Consuming protein in greater amounts than this leads to no further increase in protein synthesis, with additional amino acids oxidised (Moore et al. 2009). If the benefit of protein–CHO supplementation is derived from changes in protein metabolism then it can be hypothesised that consuming a smaller volume of CHO and protein can elicit similar effects. A lower volume of milk may also be more practical for the athlete in terms of reduced caloric intake, and less stomach fullness and discomfort. By decreasing the volume of supplement ingested it may be hypothesised that the same benefits to changes in muscle performance and intramuscular proteins will be observed. There have not been any other studies investigating changes in the volume of protein–CHO supplementation on recovery from EIMD. Therefore, the aim of the current study is to determine if a reduced volume of milk, which will be more practical for an athlete, ingested immediately following muscle damaging exercise reduces indices of EIMD.

## Method

### Participants

Twenty-four healthy male participants (age 21 ± 3 years; stature 181.4 ± 6.5 cm; body mass 79.7 ± 9.3 kg) who regularly competed in a variety of sports (team and individual) volunteered to take part in the study. After institutional ethical approval, the experimental procedures and the associated risks and benefits were explained; the participants then gave their written informed consent. Participants were fully familiarised with all testing procedures prior to commencing the study, and had no prior experience in the bout of muscle damaging exercise. Participants were instructed to maintain their habitual diet throughout the study and to record their food intake in the food diary provided. There were no differences between groups in total energy intake or macronutrient content of the diets. Participants were required to arrive at the laboratory in a rested state, having avoided strenuous physical activity, caffeine, alcohol and anti-inflammatory drugs for at least 48 h and having not taken any nutritional supplements in the previous 6 months. Participants were tested in the morning, following an overnight fast, to minimise diurnal variation.

### Procedures

#### *Design*

Participants were assigned to 1 of 3 independent groups: (1) 500 mL milk, (2) 1,000 mL milk, or (3) a placebo (1,000 mL of water). Participants in each group were equally matched on the basis of concentric knee flexion peak torque, recorded from 6 knee extension–flexions during preliminary testing. A one-way analysis of variance (ANOVA) revealed no group differences in baseline participant characteristics (age, stature and body mass), or peak torque values used for group allocation ($p < 0.05$).

All participants were required to attend the laboratory on four consecutive days. Prior to baseline tests, height and body mass were recorded. On each day prior to any exercise, a venous blood sample was collected for analysis, and participants rated passive muscle soreness on a visual analogue scale. Participants completed a standardised warm-up, consisting of 5 min of cycling at 60 W on a cycle ergometer (Monark 824 E, Stockholm, Sweden), carried out isokinetic muscle performance measures and completed a visual analogue scale for active muscle soreness when conducting knee flexions. Following baseline testing, participants completed a bout of exercise designed to induce acute muscle damage. Upon completing the exercise bout, they immediately consumed their allocated supplement in the relevant volume. At 24, 48, and 72 h after muscle damaging exercise, participants repeated baseline testing.

#### *Nutritional supplement*

Participants were provided with semi-skimmed milk (Rock Farm Dairy, Durham, UK), which provides whey and casein protein, and CHO in the form of lactose. Previous research has shown that 1,000 mL of this supplement results in the significant attenuation of decreases in isokinetic muscle performance, and increases in creatine kinase (CK) (Cockburn et al. 2008). Table 1 details the caloric and macronutrient content of the supplement in each volume.

**Table 1** Nutritional content of semi-skimmed milk

| | 500 mL | 1,000 mL |
|---|---|---|
| Energy (kcal) | 240 | 480 |
| Protein (g) | 17 | 34 |
| Casein (g) | 13.6 | 27.2 |
| Whey (g) | 3.4 | 6.8 |
| Carbohydrate (g) | 24.5 | 49 |
| Fat (g) | 8.5 | 17 |

*Muscle damaging exercise*

Muscle damage was induced in the hamstrings. Participants completed 6 sets of 10 repetitions, with 90 s rest between sets, of unilateral eccentric–concentric knee flexions at a speed of 1.05 rad $s^{-1}$, using an isokinetic dynamometer (Cybex Norm, Cybex International, New York, NY, USA). This was conducted on one side of the body and then repeated on the contralateral leg. This protocol lasted approximately 30 min. Participants were instructed to expend maximal effort during the eccentric phase of each knee flexion. During the concentric phase, participants were instructed to return their leg to the starting position with minimal effort. This protocol has been previously used in similar studies (Cockburn et al. 2008, 2010), and has been shown to induce significant increases in serum CK, myoglobin (Mb), and DOMS, and decreases in peak torque and total work of the set (Cockburn et al. 2008).

*Muscle soreness measurement*

Participants were required to rate both passive and active DOMS on a visual analogue scale. Participants were required to rate the level of soreness, combined for both legs, that they perceived to have in their hamstrings when standing (passive) and when conducting maximal knee flexions on the isokinetic dynamometer (active), from 0 (no pain-soreness) to 10 (pain-soreness as bad as it could be). This scale has been previously used to determine changes in passive and active muscle soreness (Cockburn et al. 2008, 2010).

*Peak torque*

Peak torque of the best repetition for six concentric maximal-effort knee flexion repetitions were measured sequentially on both legs, at a test speed of 1.05 rad $s^{-1}$, using an isokinetic dynamometer (Cybex Norm, Cybex International). Participants were required to maximally extend and flex their leg through their maximum range of motion for six repetitions. Coefficients of variation, determined from reliability trials conducted in our laboratories, for this protocol are reported at 4.5–4.9%.

*Blood sample collection and analysis*

Serum CK and Mb concentrations were determined from blood samples collected via venipuncture from a forearm vein into a serum gel monovette (7.5 mL). Plasma IL-6 concentration was determined from $K_3$EDTA treated venous blood. The samples were centrifuged at 3,000 rpm for 10 min (Allegra X-22 Centrifuge, Beckman Coulter, Bucks, UK), aliquoted and stored at −80°C for later analysis.

Total CK activity was analysed using high sensitivity procedures (Advia 2,400, Seimens Healthcare Diagnostics, UK). This method is adapted from the International Federation of Clinical Chemistry (IFCC) reference method. Myoglobin was analysed using an assay kit (Myoglobin Enzyme Immunoassay Test Kit, Oxford Biosystems Ltd., Wheatley, Oxon, UK). Absorbance was read using an Anthos 2010 Microplate reader (Anthos Labtec Instruments, Salzberg, Germany). Plasma IL-6 concentrations were analysed using an enzyme linked immunoabsorbent assay (R&D Systems, Minneapolis, USA).

Intra-assay and inter-assay coefficients of variation for CK were 0.5–0.8 and 0.9–1.6%, respectively. For Mb assays, they were 3.9–6.6 and 5.2–11.8%, respectively. Coefficients of variation for IL-6 were less than 5.8 and 9.6%. All coefficients of variation were reported from the manufacturers own precision tests.

*Statistical analysis*

The current study used statistical analysis that reports uncertainty of outcomes as 90% confidence intervals (CI), making probabilistic magnitude based inferences about true values of outcomes using methods described by Batterham and Hopkins (2006). The authors have previously used this method to determine the effect of the independent variable on the dependent variables (Cockburn et al. 2010). Each dependent variable was analysed using a published spreadsheet (Hopkins 2006) to determine the effect of the independent variable as the difference in the change between each group. The analysis of dependent variables were conducted on log-transformed values to overcome heteroscedastic error (Nevill and Lane 2007), except muscle soreness data. This variable was not log-transformed as it is inappropriate due to interval scaling (Nevill and Lane 2007). Participant descriptive data and muscle soreness data are presented as absolute means ± standard deviations (SD). Means derived from the analysis of log-transformed variables were back transformed to provide mean percentage change and percentage SD, except intramuscular protein values, which were reported as factors due to the large percentage changes (Hopkins 2003).

For calculation of the chances of benefit and harm, the smallest worthwhile or important effect for each dependent

variable was the smallest standardised (Cohen) change in the mean: 0.2 times the between-subject SD for baseline values of all participants (Batterham and Hopkins 2006), which has been used elsewhere in similar investigations (Cockburn et al. 2010; Rowlands et al. 2008). Practical inferences were drawn using the approach identified by Batterham and Hopkins (2006). Quantitative chances of benefit and harm were assessed qualitatively: <1% almost certainly not; 1–5% very unlikely; 5–25% unlikely; 25–75% possibly; 75–95% likely; 95–99% very likely; >99% almost certainly (Hopkins 2002). Due to there being a large number of comparisons that could be reported, only changes from baseline to 48 and 72 h have been reported. Previous research has shown that the point at which milk becomes beneficial is 48 h after muscle damaging exercise (Cockburn et al. 2008). In addition, due to the limitation of not including a fourth treatment group consuming 500 mL of water only comparisons between 500 mL milk and 1,000 mL milk, and 1,000 mL milk and placebo are reported. *p* values for the interaction effect between time and group, determined using a factorial ANOVA with repeated measures on 1 factor (time), have also been stated.

## Results

### Muscle soreness

All groups demonstrated an increase in passive and active muscle soreness that peaked at 48 h and began to return to baseline levels by 72 h. For changes in passive DOMS, all comparisons made were unclear except between 0 and 48 h, which showed a possible benefit for participants consuming 500 mL of milk in comparison to those consuming 1,000 mL of milk for limiting increases in muscle soreness. The *p* value for the interaction effect between time and group was 0.601. For changes in active DOMS, all comparisons made were unclear in both legs (Fig. 1). The *p* value for the interaction effect between time and group was 0.454. A summary of the statistical analysis for DOMS is shown in Table 2.

### Peak torque

Baseline values for peak torque of the dominant leg for the placebo group, the group consuming 500 mL of milk and the group consuming 1,000 mL of milk were 146, 130 and 136 Nm, respectively. Changes in peak torque of the dominant leg between baseline and 48 h for the placebo group, and the groups consuming 500 or 1,000 mL of milk were −19 ± 33, −10 ± 9 and −10 ± 10%, respectively. All effects investigated were unclear. Between baseline and 72 h there was a likely benefit of 1,000 mL (−3 ± 14%) of milk in comparison to the placebo group (−19 ± 28%) for limiting decrements in peak torque of the dominant leg. There were no clear effects of consuming 500 mL (−3 ± 13%) of milk versus 1,000 mL of milk (Fig. 2).

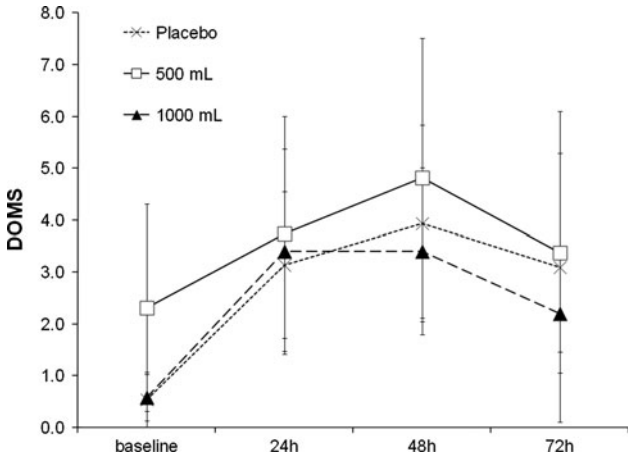


**Fig. 1** Active muscle soreness of the dominant leg in response to exercise-induced muscle damage in the placebo ($n = 8$), 500 mL ($n = 8$) and 1,000 mL ($n = 8$) groups. Values presented as mean ± standard deviation

Baseline values of peak torque of the non-dominant leg were 128, 129 and 136 Nm for the placebo group, the group consuming 500 mL of milk and the group consuming 1,000 mL of milk, respectively. Changes in peak torque of the non-dominant leg between baseline and 48 h for the placebo group, and the groups consuming 500 or 1,000 mL of milk were −15 ± 20, −18 ± 22 and −15 ± 18%, respectively. Changes between baseline and 72 h were −13 ± 28, −11 ± 27 and −9 ± 8% for the placebo group, and the groups consuming 500 or 1,000 mL of milk, respectively. All comparisons made between baseline and 48 h, and baseline and 72 h for changes in peak torque of the non-dominant leg were unclear. The *p* value for the interaction effect between time, group and leg was 0.135. A summary of the statistical analysis is shown in Table 3.

### Intramuscular proteins in the serum

A summary of the statistical analysis is shown in Table 4.

#### *Creatine kinase*

Mean baseline CK values were 540.5, 113.4 and 150.4 U $L^{-1}$ for the placebo group, the group consuming 500 mL of milk and the group consuming 1,000 mL of milk, respectively. Baseline CK values for the placebo group are high and this is due to one participant who had a baseline CK value substantially greater than the normal range. However, as results are analysed as the difference between groups in change over time, this participant was not removed from the analysis.

**Table 2** Effect of supplement volume on increases in muscle soreness following muscle damaging exercise

| | Comparsion | Mean effect[a] ± 90% CI[b] | Qualitative inference |
|---|---|---|---|
| Baseline–48 h | | | |
| Passive DOMS | 1,000 mL vs. placebo | 0.7 ± 1.9 | Unclear |
| | 1,000 vs. 500 mL | 1.3 ± 1.3 | Increase possible |
| Active DOMS (DOM) | 1,000 mL vs. placebo | −0.5 ± 1.7 | Unclear |
| | 1,000 vs. 500 mL | 0.4 ± 2.3 | Unclear |
| Active DOMS (NON) | 1,000 mL vs. placebo | −0.3 ± 2.0 | Unclear |
| | 1,000 vs. 500 mL | 0.1 ± 1.9 | Unclear |
| Baseline–72 h | | | |
| Passive DOMS | 1,000 mL vs. placebo | 1.1 ± 2.4 | Unclear |
| | 1,000 vs. 500 mL | 1.1 ± 1.9 | Unclear |
| Active DOMS (DOM) | 1,000 mL vs. placebo | −0.9 ± 2.3 | Unclear |
| | 1,000 vs. 500 mL | 0.6 ± 2.0 | Unclear |
| Active DOMS (NON) | 1,000 vs. placebo | 0.0 ± 2.8 | Unclear |
| | 1,000 vs. 500 mL | 0.4 ± 2.4 | Unclear |

[a] Mean effect refers to the first named group minus second named

[b] ±90% CI: add and subtract this number to the mean effect to obtain the 90% confidence intervals for the true difference. Qualitative inference represents the likelihood that the true value will have the observed magnitude

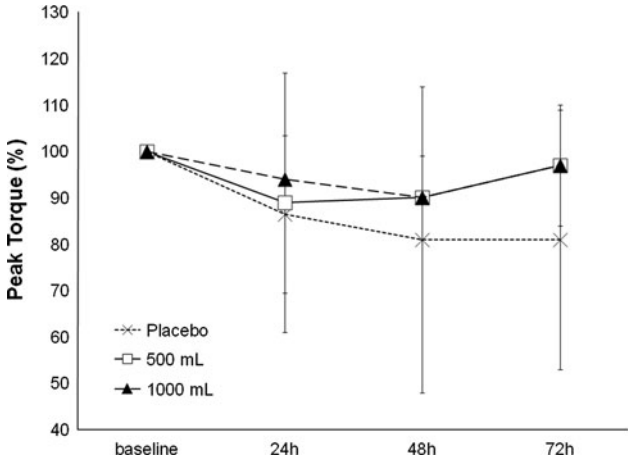


**Fig. 2** Relative peak torque of the dominant leg in response to exercise-induced muscle damage in the placebo ($n = 8$), 500 mL ($n = 7$) and 1,000 mL ($n = 7$) groups. Values presented as mean ± standard deviation

Between baseline and 48 h, changes in CK were 6.7 ×/÷ 13.3, 0.5 ×/÷ 6.6 and 0.1 ×/÷ 9.3 for the placebo group, the group consuming 500 mL of milk and the group consuming 1,000 mL of milk, respectively. There was a very likely benefit of consuming 1,000 mL of milk in comparison to the placebo group for limiting increases in CK, and for the comparison between 500 and 1,000 mL of milk the effect was unclear for changes between baseline and 48 h.

Changes for the placebo group, and the groups consuming 500 mL of milk or 1,000 mL of milk between baseline and 72 h were 12.9 ×/÷ 10.2, 0.2 ×/÷ 9.1 and 3.0 ×/÷ 5.2, respectively. All effects investigated were unclear for changes between baseline and 72 h (Fig. 3). The $p$ value for the interaction effect between time and group was 0.956.

**Table 3** Effect of supplement volume on decreases in muscle function following muscle damaging exercise

| Muscle performance | Comparison | Mean effect[a] ± 90% CI[b] | Qualitative inference |
|---|---|---|---|
| Baseline–48 h | | | |
| Peak torque (DOM) | 1,000 mL vs. placebo | 12 ± 23 | Unclear |
| | 1,000 vs. 500 mL | 0 ± 9 | Unclear |
| Peak torque (NON) | 1,000 mL vs. placebo | 1 ± 18 | Unclear |
| | 1,000 vs. 500 mL | 7 ± 18 | Unclear |
| Baseline–72 h | | | |
| Peak torque (DOM) | 1,000 mL vs. placebo | 23 ± 21 | Likely decrease |
| | 1,000 vs. 500 mL | −2 ± 13 | Unclear |
| Peak torque (NON) | 1,000 mL vs. placebo | 6 ± 19 | Unclear |
| | 1,000 vs. 500 mL | 3 ± 18 | Unclear |

[a] Mean effect refers to the first named group minus second named

[b] ±90% CI: add and subtract this number to the mean effect to obtain the 90% confidence intervals for the true difference. Qualitative inference represents the likelihood that the true value will have the observed magnitude

*Myoglobin*

Mean baseline Mb values were 29.1, 21.8 and 30.2 ng $mL^{-1}$ for the placebo group, the group consuming 500 mL of milk and the group consuming 1,000 mL of milk, respectively. Changes for the placebo group and the groups consuming 500 or 1,000 mL of milk between baseline and 48 h were 7.0 ×/÷ 13.1, 8.1 ×/÷ 6.1 and 1.6 ×/÷ 7.8, respectively. All effects investigated were unclear for changes between baseline and 48 h. Changes in

**Table 4** Effect of supplement volume on increases in intramuscular proteins following muscle damaging exercise

| Intramuscular proteins | Comparison | Mean effect[a] ×/÷ 90% CI[b] | Qualitative inference |
|---|---|---|---|
| Baseline–48 h | | | |
| CK | 1,000 mL vs. placebo | 0.0 ×/÷ 24.8 | Very likely decrease |
| | 1,000 vs. 500 mL | 0.9 ×/÷ 7.9 | Unclear |
| Mb | 1,000 mL vs. placebo | 0.3 ×/÷ 10.9 | Unclear |
| | 1,000 vs. 500 mL | 0.5 ×/÷ 8.4 | Unclear |
| Baseline–72 h | | | |
| CK | 1,000 mL vs. placebo | 0.2 ×/÷ 10.8 | Unclear |
| | 1,000 vs. 500 mL | 3.0 ×/÷ 6.1 | Unclear |
| Mb | 1,000 mL vs. placebo | 1.1 ×/÷ 6.6 | Unclear |
| | 1,000 vs. 500 mL | 17.3 ×/÷ 4.2 | Almost certain decrease |

*CK* creatine kinase, *Mb* myoglobin

[a] Mean effect refers to the first named group minus second named

[b] 90% CI: add and subtract this number to the mean effect to obtain the 90% confidence intervals for the true difference. Qualitative inference represents the likelihood that the true value will have the observed magnitude

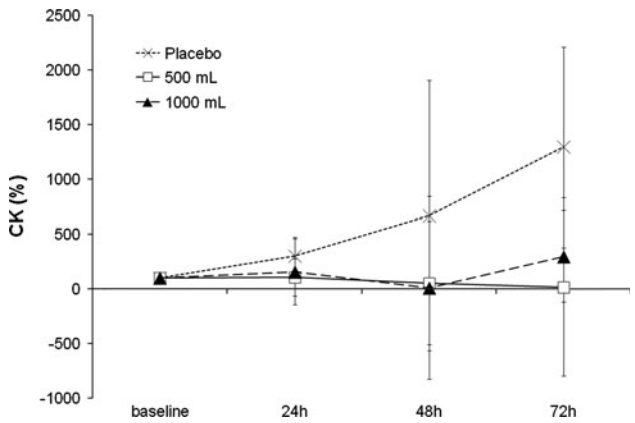


**Fig. 3** Relative creatine kinase (*CK*) in response to exercise-induced muscle damage in the placebo ($n = 6$), 500 mL ($n = 7$) and 1,000 mL ($n = 7$) groups. Values presented as mean ± standard deviation

Mb for the placebo group and the groups consuming 500 or 1,000 mL of milk were 15.5 ×/÷ 8.2, 1.2 ×/÷ 4.6 and 14.7 ×/÷ 3.6 between baseline and 72 h, respectively. Consuming 500 mL of milk was almost certainly beneficial in comparison to consuming 1,000 mL of milk for limiting increases in Mb between baseline and 72 h. The effect was unclear for the comparison between 1,000 mL of milk and the placebo group. The $p$ value for the interaction effect between time and group was 0.859.

### Interleukin-6

IL-6 did not change significantly over time. Baseline values for IL-6 were 1.86, 1.28 and 0.90 pg $mL^{-1}$ for the placebo group, the group consuming 500 mL of milk and the group consuming 1,000 mL of milk, respectively. Between baseline and 48 h, changes in IL-6 for the placebo group and the groups consuming 500 or 1,000 mL of milk were 27.9 ± 134.7, −32.4 ± 595.2 and −44.5 ± 130.4%, respectively. There was a likely benefit of consuming 1,000 mL of milk in comparison to the placebo group for limiting increases in IL-6, with the comparison between 500 and 1,000 mL of milk unclear. Changes in IL-6 between baseline and 72 h were 11.1 ± 413.2, 23.5 ± 449.6 and −9.0 ± 200.7% for the placebo group and the groups consuming 500 or 1,000 mL of milk, respectively. All comparisons made were unclear. The $p$ value for the interaction effect between time and group was 0.634.

## Discussion

The findings of this study demonstrate that decrements in isokinetic muscle performance (dominant leg), and increases in CK can be blunted with the intake of less milk than has been previously shown (Cockburn et al. 2008, 2010). There was no effect of ingesting one litre of milk on passive or active muscle soreness, or Mb. However, changes in IL-6 can be altered with the consumption of one litre of milk immediately following exercise with no differences between milk groups.

The lower volume of milk consumed and, subsequently, the amount of protein and CHO had no lesser impact for attenuating decrements in isokinetic muscle performance and increases in CK than 1,000 mL. It has been proposed that the benefit derived from milk is due to increases in protein synthesis and/or limiting increases in protein degradation. It has been demonstrated that there is a relationship between muscle protein synthesis and amino acid intake after resistance exercise (Borsheim et al. 2002; Miller et al. 2003). Moore et al. (2009) demonstrated that muscle protein synthesis is not further stimulated with intakes of protein above 20 g, and that this may be the upper limit for incorporation of amino acids into protein pools. This study compared 17 g to 34 g protein and found no difference for the attenuation of muscle damage, suggesting that, if the benefits are due to increased protein synthesis, then consuming 34 g does not provide extra amino acids that can be incorporated into new proteins to preserve or synthesise myofibrillar and membrane proteins. The excess protein consumed with 1,000 mL of milk will not be utilised for the synthesis of new proteins, but are more likely to be oxidised (Moore et al. 2009). A change in

CHO intake through reduced supplement volume also occurred. CHO increases insulin that can increase the capacity for muscle protein synthesis, however, sufficient amino acids are required for this to be reflected in elevated synthesis (Biolo et al. 1999; Volek 2004). Therefore, although CHO ingestion was reduced this is likely to have minimal effect on protein metabolism. Furthermore, there is likely to be a ceiling effect to CHO intake whereby consuming more does not provide greater effects. Therefore, there is no additional benefit to attenuating EIMD with the consumption of larger volumes of milk.

In contrast to our previous studies (Cockburn et al. 2008, 2010), there was no benefit of one litre of milk on limiting increases in active muscle soreness (DOMS) or Mb. The increase in active DOMS for the placebo group after 48 and 72 h in our previous study (Cockburn et al. 2010) was greater than those observed in the current study. As changes with consumption of one litre of milk consumed immediately following muscle damaging exercise were similar in both studies, this may have masked any effect of milk supplementation on this variable. It is unknown why the placebo group demonstrated less muscle damage, based on measures of muscle soreness, and this requires further investigation. Regarding effects on Mb, 500 mL of milk was almost certainly beneficial for limiting increases in Mb in comparison to 1,000 mL of milk after 72 h. Again it is difficult to know why this occurred since if a smaller volume of milk consumption limits increases in Mb then consuming more of the supplement should produce similar benefits. One reason may be related to the analysis of Mb. Mb should be used with caution since assays to determine concentrations cannot distinguish between Mb released from the heart and skeletal muscle (Sorichter et al. 1999). Hence caution should be exercised when interpreting Mb.

The results demonstrated a benefit of 1,000 mL milk in comparison to the placebo for limiting reductions in isokinetic muscle performance of the dominant leg only. Based on the placebo group data the dominant leg showed greater reductions in isokinetic muscle performance after both 48 and 72 h possibly implying greater muscle damage. As the non-dominant leg exhibited less damage, the effects were less clear. Further research is required to understand the underlying mechanisms of this finding.

To investigate possible inflammatory-related mechanisms, IL-6 was assessed. This is because inflammatory cytokines have been implicated in many of the pathways responsible for myofibrillar and membrane damage and thus changes in indirect markers. There was a likely benefit of a litre of milk in comparison to the placebo group after 48 h on IL-6 with no differences between the milk groups. There were also no differences in changes of peak torque between both milk groups. One study has shown that muscle proteolysis is limited with branched chain amino acid supplementation, but this response is independent of the IL-6 response (Rohde et al. 1997). More recently, it was shown that CHO supplementation for 48 h following high force eccentric exercise did not attenuate the response of IL-6 (Miles et al. 2007). However, there was no impact of CHO on other indirect markers of EIMD (Miles et al. 2007). Therefore, the relevance of this finding requires further investigation. The mechanisms underlying the benefits of acute milk supplementation require further investigation as the results presented here are inconclusive. Future studies should concentrate on measuring a wider array of cytokines specifically those that are pro-inflammatory to assess if they are altered by milk consumption. TNF-$\alpha$ and IL-1$\beta$ are the most pro-inflammatory combination of cytokines (Pyne 1994), both with potential for limiting protein synthesis (Frost et al. 1997) and increasing degradation (Andreu and Schwartz 1995). Furthermore, the current study measured IL-6 systemically and not locally, therefore, future studies should possibly use microdialysis techniques or muscle biopsies.

A limitation of this study was that only one placebo, which was 1,000 mL of water, was utilised. The study would have been strengthened by the inclusion of another placebo of 500 mL of water. This would have allowed direct comparisons of 500 mL of milk with 500 mL of water. As this fourth treatment was not included only two main comparisons (volume effect and constituent effect) could be made which has limited the study.

## Conclusion

In conclusion, decrements in isokinetic muscle performance (dominant leg) and increases in CK following muscle damaging exercise can be minimised with the immediate consumption of 500 mL of milk. The attenuation of EIMD, specifically isokinetic muscle performance, with protein–CHO is not novel; however, this study provides athletes with important information regarding the volume to be consumed. This is important as consuming 500 mL rather than 1,000 mL following exercise may be easier for athletes to implement as fewer calories will be consumed and it may lead to less stomach fullness and discomfort. Consuming a supplement with more than 20 g provides no further benefit on reducing the impact of muscle damaging exercise. An insight into the role of IL-6 in the context of EIMD with milk has been provided but a wider array of cytokines should be examined. This is a preliminary research and further investigation is warranted investigating the potential role of the inflammatory process and protein metabolism following EIMD and milk ingestion.

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