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Citation: Kwiecien, Susan, O'Hara, Denis, Mchugh, Malachy and Howatson, Glyn (2020) Prolonged cooling with phase change material enhances recovery and does not affect the subsequent repeated bout effect following exercise. European Journal of Applied Physiology, 120 (2). pp. 413-423. ISSN 1439-6319

Published by: Springer

URL: https://doi.org/10.1007/s00421-019-04285-5 <https://doi.org/10.1007/s00421-019-04285-5>

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Prolonged cooling with phase change material enhances recovery and does not affect the subsequent repeated bout effect following exercise

Susan Y. Kwiecien[1,2], Denis J. O'Hara[1], Malachy P. McHugh[1,2], Glyn Howatson[2,3]

1: Nicholas Institute of Sports Medicine and Athletic Trauma, Lenox Hill Hospital,

New York, NY, USA.

2: Department of Sport, Exercise & Rehabilitation, Northumbria University,

Newcastle upon Tyne, UK.

3: Water Research Group, North West University, Potchefstroom, South

Africa.

Susan Y. Kwiecien; Nicholas Institute of Sports Medicine and Athletic Trauma; MEETH, 210 East 64 Street, 5th Floor, NISMAT, New York, New York, 10065, USA; 212-434-2752; 212-434-2687; susan@nismat.org

Susan Kwiecien 0000-0002-6683-8127, Malachy McHugh 0000-0002-8371-8151, Glyn Howatson 0000-0001-8494-2043

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Abstract

PURPOSE: The aim of this investigation was two-fold: (1) to examine the effect of prolonged phase change material (PCM) cooling following eccentric exercise of the quadriceps on indices of muscle damage, and (2) to elucidate whether application of PCM cooling blunted the acute adaptive response to eccentric exercise, known as the repeated bout effect (RBE).

METHODS: Twenty-six males (25±6 years) performed an initial bout (B1) of 120 eccentric quadriceps contractions on each leg at 90% of their isometric strength and were then randomized to receive PCM packs frozen at 15°C (treatment) or melted packs (control) worn directly on the skin under shorts for 6 h. The protocol was repeated 14 days later (B2) with all participants receiving the control condition.

RESULTS: PCM cooling provided protection against strength loss in B1 ($P=0.005$) with no difference in strength between treatment groups in B2 ($P=0.172$; bout by treatment by time $P=0.008$). PCM cooling reduced soreness in B1 ($P=0.009$) with no difference between treatment groups in B2 ($P=0.061$). Soreness was overall lower following B2 than B1 ($P<0.001$). CK was elevated in B1 ($P<0.0001$) and reduced in B2 ($P<0.001$) with no difference between treatments. The damage protocol did not elevate hsCRP in B1, with no difference between treatments or between bouts.

CONCLUSIONS: This work provides further evidence that PCM cooling enhances recovery of strength and reduces soreness following eccentric exercise. Importantly, these data show for the first time that prolonged PCM cooling does not compromise the adaptive response associated with the RBE.

Keywords: Cryotherapy, Exercise-induced muscle damage, adaptation, delayed onset muscle soreness

Abbreviations:

CK	creatine kinase
CWI	cold water immersion
EIMD	exercise induced muscle damage
ES	effect sizes
hsCRP	high sensitivity c-reactive protein
MIVC	maximal isometric voluntary contraction
PCM	phase change material
RBE	repeated bout effect

Introduction

A combination of high force and low fibre recruitment during eccentric contractions places a high mechanical stress on the involved muscles that can lead to exercise induced muscle damage (EIMD; Enoka 1996). Muscle damage following unaccustomed eccentric exercise is commonly accompanied by soreness, decrements in performance such as strength loss, a rise in serum creatine kinase (CK) concentration and changes in muscle morphology occurring after the acute bout of exercise (Brown et al. 1997; Ebbeling and Clarkson 1989; Howatson et al. 2007; McHugh et al. 1999; McHugh et al. 2001; Nosaka and Clarkson 1995). Prior unfamiliar eccentric training provides a rapid adaptation referred to as the repeated bout effect (RBE; Clarkson et al. 1987). The RBE results is a protective effect against further muscle damage (Evans et al. 1986; Hyldahl et al. 2017; McHugh 2003) as evident by an attenuation in the signs and symptoms of EIMD following a second bout of exercise (Howatson and van Someren 2008). The exact mechanisms behind the RBE are unknown but are likely a combination of peripheral, non-neural (McHugh 2003), non-contractile adaptation (Pincheira et al. 2018) and connective and cellular processes (Howatson et al. 2007; McHugh et al. 1999).

Non-invasive recovery modalities, such as cryotherapy, are commonly utilized by athletes to mitigate the acute and chronic symptoms of EIMD. Cryotherapy, in the form of cold water immersion (CWI), is one of the most popular recovery modalities utilized by athletes for its ability to diminish perceptions of soreness (Bleakley et al. 2012; Hohenauer et al. 2015; Leeder et al. 2011). It is likely that CWI treatment duration, limited by treatment temperature, is insufficient to markedly influence the physiological mechanisms responsible for enhancing recovery. In an effort to overcome this limitation, recent work has introduced the use of a cooling modality (phase change material; PCM) that can prolong the duration of cryotherapy exposure while allowing the wearer to continue with activities of daily living. Cooling treatment using PCM at 15°C has achieved reductions in the magnitude of intramuscular temperature comparable to 15 minutes of CWI (Kwiecien et al. 2019), but can be tolerated for prolonged durations (3-6 hours). Prolonged PCM cooling has been effectively used for reductions in soreness and strength loss on the days after eccentric quadriceps exercise in recreational athletes (Kwiecien et al. 2018) and after a professional soccer match (Clifford et al. 2018) when applied for 6 and 3 hours, respectively.

Cryotherapy interventions that suppress the acute inflammatory response and accelerate recovery might negatively affect damage-repair-adaptation processes of skeletal muscle (Woods et al. 2000), functional performance and proprioceptive acuity (Costello and Donnelly 2011), and might hinder normal adaptive training responses to strenuous exercise (Figueiredo et al. 2016; Fröhlich et al. 2014; Roberts et al. 2015; Yamane et al. 2015; Yamane et al. 2005). Indeed, CWI after routine strength workouts have been shown to attenuate training-induced adaptations during several weeks of training (Yamane et al. 2005). However, it is not known whether cryotherapy interventions inhibit the RBE. If cryotherapy accelerated recovery after an initial bout of exercise, is a RBE still induced in a subsequent bout of exercise? Therefore, the purpose of this study was to determine whether prolonged PCM cooling after an initial bout of eccentric exercise blunts the adaptive response that provides protection against damage following a subsequent repeated bout of eccentric exercise. The hypothesis was twofold: i) that prolonged PCM cooling would accelerate recovery from eccentric exercise compared to a control, and that ii) the accelerated recovery from an initial bout of strenuous quadriceps exercise would reduce the adaptive response to a subsequent bout of strenuous quadriceps exercise.

Methods

Participants

Twenty-six male participants (mean ± SD; age, 25±6 years; height, 179.5±5.6 cm; body mass, 82.9±11.9 kg) volunteered to participate in this study. Prior to participation, volunteers were informed of the procedures and provided written, informed consent. All participants were trained athletes participating in sport e.g. ice hockey, basketball, soccer, Gaelic football, powerlifting. Participants completed the experimental protocol during their offseason. Participants were free from injury within the past 6 months. Participants were instructed to refrain from taking non-steroidal anti-inflammatory drugs, nutritional supplements, pharmacological interventions, therapeutic interventions, and strenuous exercise unrelated to the present study for the duration of the two study periods. The institutional research ethics committee, in line with the Declaration of Helsinki, approved all procedures.

Experimental Design

Participants reported to the laboratory for two periods of four consecutive days, each period separated by two weeks. On day one participants performed bout 1 of eccentric quadriceps exercise on each leg and were randomized to receive either 'treatment' frozen 15°C PCM or 'control' room temperature PCM applied to the quadriceps for 6 hours, starting immediately following the exercise. Two weeks later participants performed bout 2 and repeated an identical bout of eccentric exercise. All participants received room temperature PCM post-exercise following bout 2. Prior to each eccentric exercise bout, and on each of the subsequent days, assessments of soreness, strength (Newton meters; Nm), and blood markers of muscle damage (CK) and inflammation (high sensitivity c-reactive protein; hsCRP) were made (Fig. 1).

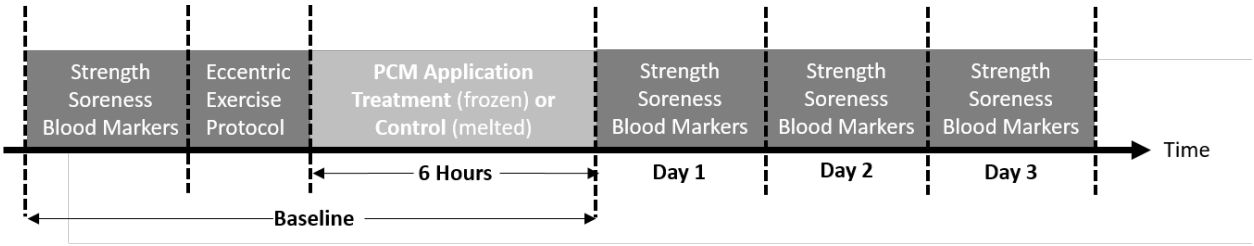


Fig. 1 Experimental Protocol for one bout of exercise. The protocol was repeated for bout two, with the exception that both treatment and control conditions received room temperature PCM following exercise.

Experimental Procedures

Soreness Assessment: Quadriceps soreness was assessed by having participants perform a 2-legged squat to 90° knee flexion and report the discomfort level for each leg using a 0 to 10-point scale (0=no discomfort, 10=too painful to squat to 90°).

Blood Measures: All blood samples were performed prior to any activity being initiated by the participants. The fingertip was cleaned with 95% ethanol before an automatic lancet device was used to puncture the skin to draw capillary blood. The first drop of blood was removed to prevent possible contamination. A 30 μL sample of capillary blood was obtained using a 30 μL pipette (Microsafe Tubule, Safe-Tec Clinical Products, Pennsylvania, USA) for the enzymatic measurement of CK concentration. The sample was then immediately analysed (Reflotron® Plus System, Roche Diagnostics, Basel, Switzerland) using a CK test strip (Reflotron CK, Roche Diagnostics, Mannheim, Germany). A 10 μL sample was obtained in a 10 μL pipette for the immune-chromatographic assay of hsCRP (Nano-Checker 710, Nano-Ditech Corporation, Cranbury, NJ, USA) using a hsCRP test strip (Nano-Check hs-CRP, Nano-Ditech Corporation, Cranbury, NJ, USA) and following the manufacturer's guidelines. The intra-sample coefficient of variation is 3.1% for the CK analyser (Horder et al. 1991), and 15% for hsCRP (Nano-Ditech Corporation, Cranbury, NJ, USA).

Strength Testing: Bilateral maximal isometric voluntary contraction (MIVC) of the knee extensors was assessed with participants seated at a trunk angle of ~90° of flexion and was reported as the mean of two efforts at each joint angle of both legs. MIVC was assessed at 50°, 80° and 100° knee flexion on an isokinetic dynamometer (Biodex System 4, Shirley NY). The strength testing was completed on one leg and then immediately repeated on the contralateral leg. The purpose of testing at three angles was to examine any shift in the angle-torque relationship between baseline measures for the initial and repeated bouts. Previously the RBE was associated with a shift in the angle-torque relationship such that there was an increase in torque at long muscle lengths and a decrease in torque at short lengths (Brockett et al. 2001).

Eccentric Exercise: Participants performed 120 isokinetic (Biodex System 4, Shirley, N.Y., USA) eccentric knee extension contractions (10 sets, 12 repetitions, 30-s rest between sets) performed at 1.05 rad s-1 (60° s-1) with the range of motion 40° (0°=full extension) to 100° knee flexion. Target intensity was determined as 90% of MIVC at 80° of knee flexion. The exercise was then immediately repeated on the contralateral leg. Based on previous work (McHugh and Tetro 2003; McHugh and Pasiakos 2004) it was expected that an angular velocity of 1.05 rad s-1 was sufficiently slow enough to allow participants to accurately produce the target torque and would result in significant strength loss and soreness on subsequent days. Both legs performed the eccentric exercise to increase the likelihood of elevating hsCRP (Margaritelis et al. 2015).

Compression Pressure & Skin Temperature: Following each bout of eccentric exercise, and before application of PCM, a skin temperature sensor sticker (VitalSense Dermal Patch, Respironics Inc., Murrysville, PA, USA) was applied directly to the surface of the skin over one thigh at approximately mid-femoral length. Quadriceps skin temperature was recorded continually for the 6 hours of PCM application during both bouts. Following application of PCM, compression of one leg was measured by a pressure measurement manometer (Kikuhime; TT, 160 Medi Trade, Søleddet, Denmark) by placing the bladder of the pressure sensor between the PCM and the anterior surface of the thigh skin (mid-point between the superior aspect of the patella and the inguinal crease). Compression was measured in both seated and standing positions. Measurements were repeated three times with the mean value recorded.

Phase Change Material Application

Immediately after the second leg completed the eccentric exercise, participants were randomly assigned to either receive (i) PCM (Glacier Tek USDA BioPreferred PureTemp PCM, Plymouth, MN, USA) 'frozen' at 15°C, or (ii) the same PCM, but 'melted' and delivered at room temperature. Both interventions were fitted over the quadriceps of both legs inside snug-fitting shorts. Participants were allowed to leave the lab following testing and the initial application of the PCM, but the treatment group were instructed to change the 15°C PCM packs following 3 h to fresh 'frozen' 15°C PCM packs. Both groups removed the shorts and packs after 6 h. On return to the laboratory for bout 2, room temperature PCM packs were worn on both legs for 6 h following the eccentric exercise by both the control and treatment groups.

Statistical analysis

A sample size analyses on strength was performed based on previous PCM literature (Clifford et al. 2018; Kwiecien et al. 2018). It was estimated that a minimum of 13 participants per group were needed to detect a 10% difference in strength with a power of 0. 80 and alpha level of 0.05. Strength was reported as the average of each leg at each of the three test angles (50°, 80°, 100° knee flexion) for assessing strength loss on the days after eccentric exercise since torque differences between angles were not relevant to the effect PCM cooling had on recovery of strength. Strength values are expressed as a percentage change relative to baseline of each respective bout. The effect of PCM cooling across exercise bouts on average strength loss, soreness, CK, and hsCRP on the days after eccentric exercise was assessed using mixed-model analysis of variance (ANOVA): treatment (PCM cooling vs. control) × bout (initial vs. repeated) × time (baseline, Day 1, Day 2, Day 3 after the eccentric exercise). Strength prior to the initial eccentric exercise bout (bout 1) was compared to strength prior to the repeated bout (bout 2) using treatment by bout by angle mixed-model ANOVA. The purpose of these analyses was to see if there was a shift in the angle-torque relationship consistent with the RBE, with a relative increase in torque at long muscle lengths and a decline at short muscle lengths as shown by McHugh and Tetro (2003). The average skin temperature during each bout was compared between treatments using a mixed-model ANOVA.

Normality of all data sets were examined using the Shapiro–Wilk test and where necessary data were logged transformed) to establish a normal distribution (CK was the only data that had to be log transformed). Additionally, CK as a percent of baseline was analysed using Friedman Tests for effect of time and Wilcoxon Signed Ranks Test for pairwise comparisons. Mauchly's test was used to assess assumptions of sphericity and, where necessary, Greenhouse-Geisser corrections were applied. Where there was a significant treatment or treatment by time interaction effect, differences between treatments at any particular time interval were assessed with independent t-tests using Bonferroni corrections for planned pairwise comparisons. To estimate the magnitude of the treatment effects, Cohen's d effect sizes (ES) were calculated with the magnitude of effects considered either small (0.20–0.49), medium (0.50–0.79), and large (>0.80). Statistical analyses were performed using SPSS v.21 (IBM, Armonk, NY, USA). Data are reported as mean ± SD and an alpha of 0.05 was set a priori.

Results

Strength

There was a significant treatment x bout x time interaction for strength (Fig. 2; P=0.008, ES=0.95). In the initial bout, there was a significant treatment x time interaction (P=0.005, ES=0.96), with greater strength in the PCM cooling vs control group (Day 3 P=0.009, ES=1.16). By contrast, in the repeated bout, there was no difference between treatments (P=0.172, ES=0.54).

There was no change in the baseline angle-torque relationship between the initial and repeated bouts (bout x angle P=0.90, ES=0.13). However, in the PCM cooling group, baseline strength increased across all three angles by 10% from the initial bout to the repeated bout (P=0.001, ES=2.43) but remained unchanged between bouts in the control group (< 1% difference; P=0.809, ES=0.14).

During eccentric exercise, in bout 1, average peak torque (Nm) increased by 9% from the first to the 10th set (P<0.0001, ES=1.15), but this was not different between groups (P=0.319, ES=0.44; 6.5% increase in PCM treatment vs. 11.6% increase in control). In bout 2, eccentric torque was not different from the first to the 10th set (P=0.515, ES=0.38), and there was no difference between groups (P=0.142, ES=0.51). For bout 1, eccentric torque was 100±1.0% of isometric peak torque for PCM treatment and 102±1.0% for control (P=0.740, ES=0.14). For bout 2, eccentric torque was 102±1.2% for PCM treatment and 115±1.3% for control (P=0.032, ES=0.95). Target intensity over the 10 sets was not different from the initial to the repeated bout for PCM treatment (P=0.392, ES=0.51), but increased by 10% in the control group (P=0.011, ES=1.85).

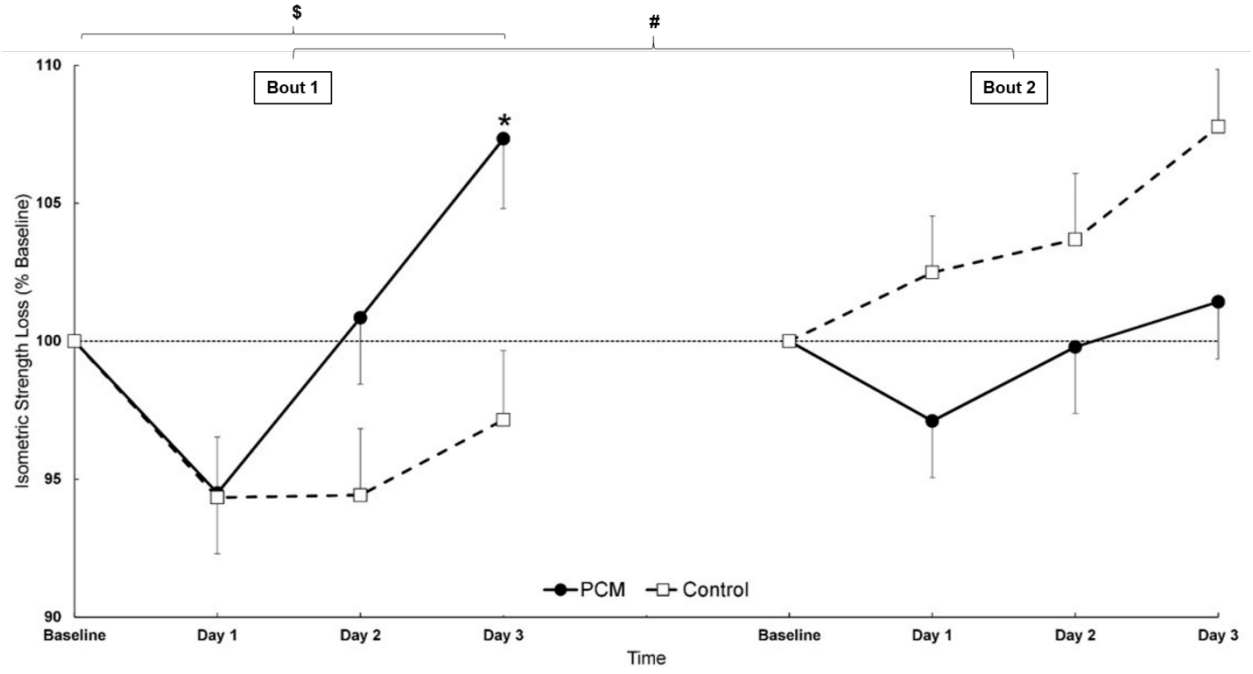


Fig. 2 Percentage change in maximal isometric voluntary contraction for the control and PCM groups before and following bouts 1 and 2. Difference from baseline as calculated using baseline value for each corresponding bout. Consistent with a RBE, force production was greater in the repeated bout than in the initial bout, but this effect was different between treatments (#: bout x treatment x time P=0.008). Dollar symbol indicates significant attenuation of force production over time in the control group but not from PCM treatment in bout 1 (treatment x time P=0.005). This difference was not evident for bout 2 (treatment x time P=0.172). Asterisk denotes greater recovery of strength in the PCM cooling vs control group (P=0.009). Values are mean ± SD

Soreness

In bout 1 soreness was lower in the PCM cooling group versus control (Fig. 3; treatment x time P=0.009, ES=0.83). In bout 2 there was no difference between treatment groups (treatment x time P=0.061, ES=0.70). Consistent with a RBE, there was lower soreness in the repeated bout versus the initial bout (P<0.001, ES=1.32) and this effect was not different between treatments (treatment x bout x time P=0.302, ES=0.45). Overall soreness was lower in the PCM cooling group versus control (treatment effect: P=0.044, ES=0.87).

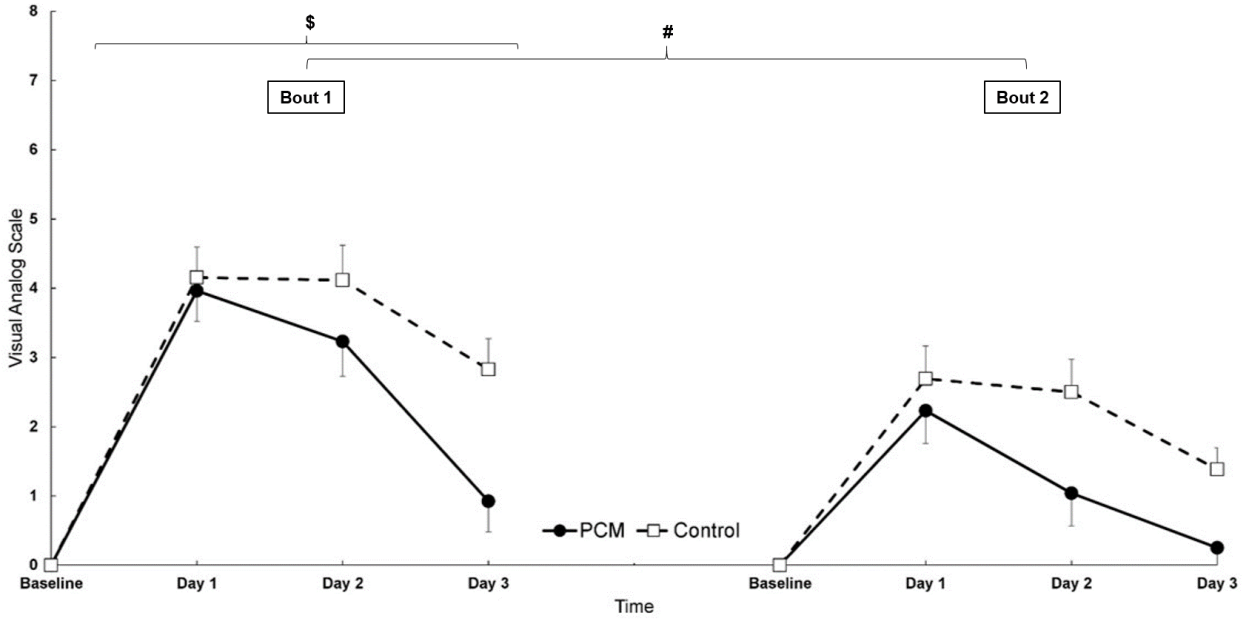


Fig. 3 Muscle soreness for the control and PCM groups before and following bout 1 and bout 2. Soreness was reduced in the repeated bout versus the initial bout (#: P<0.001) and this effect was not different between treatments (bout x treatment x time P=0.302). However, magnitude of soreness was overall lower from PCM than control (treatment effect: P=0.044). Dollar symbol indicates significantly lower soreness over time in the PCM cooling group in bout 1 (treatment x time P=0.009). This difference was not evident for bout 2 (treatment x time P=0.061). Values are mean ± SD

Blood Markers

CK (log transformed) was elevated above baseline on all 3 days after the initial bout (Fig. 4a; time effect P<0.0001, ES=1.88) with no difference between groups (treatment x time P=0.896, ES=0.18). After the repeated bout, CK was only elevated on day 1 (p<0.0001, ES=0.28). CK response was reduced in bout 2 versus bout 1 (bout x time P<0.001, ES=1.32) with no difference between treatment groups (treatment x bout x time P=0.648, ES=0.31).-CK as a percent of baseline was elevated after the initial bout on all 3 days for the PCM and control groups (P<0.05; Fig4b). After the repeated bout, CK (% baseline) was only elevated above baseline on day 1 (P=0.024) for the PCM group and on day 1 (P=0.012) and day 2 (P=0.015) for the control group.

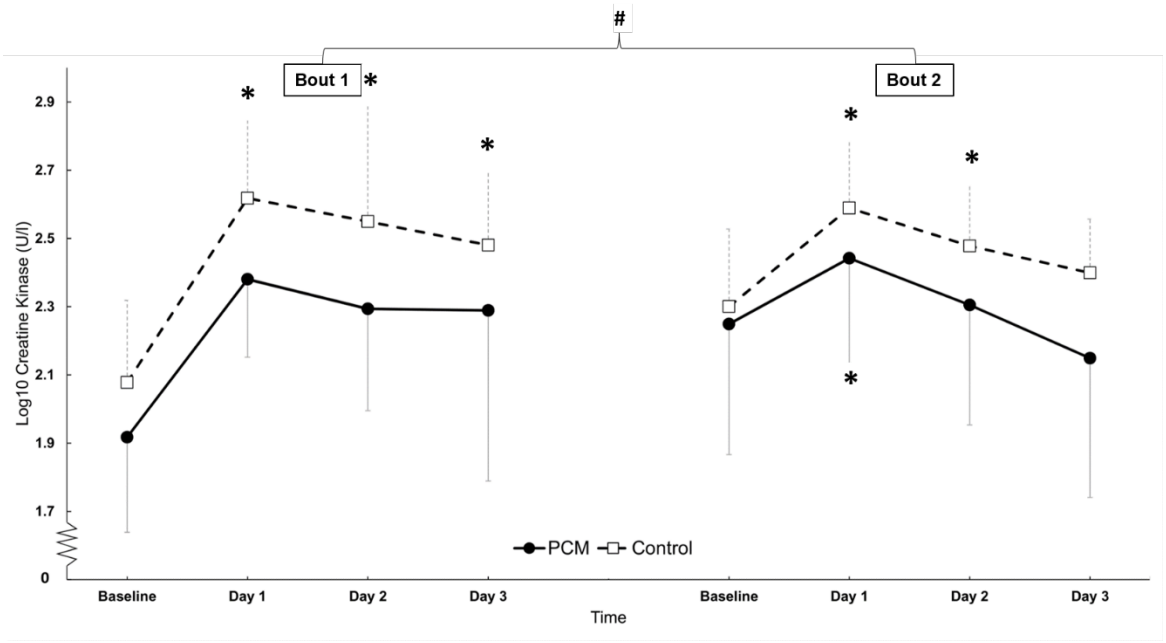
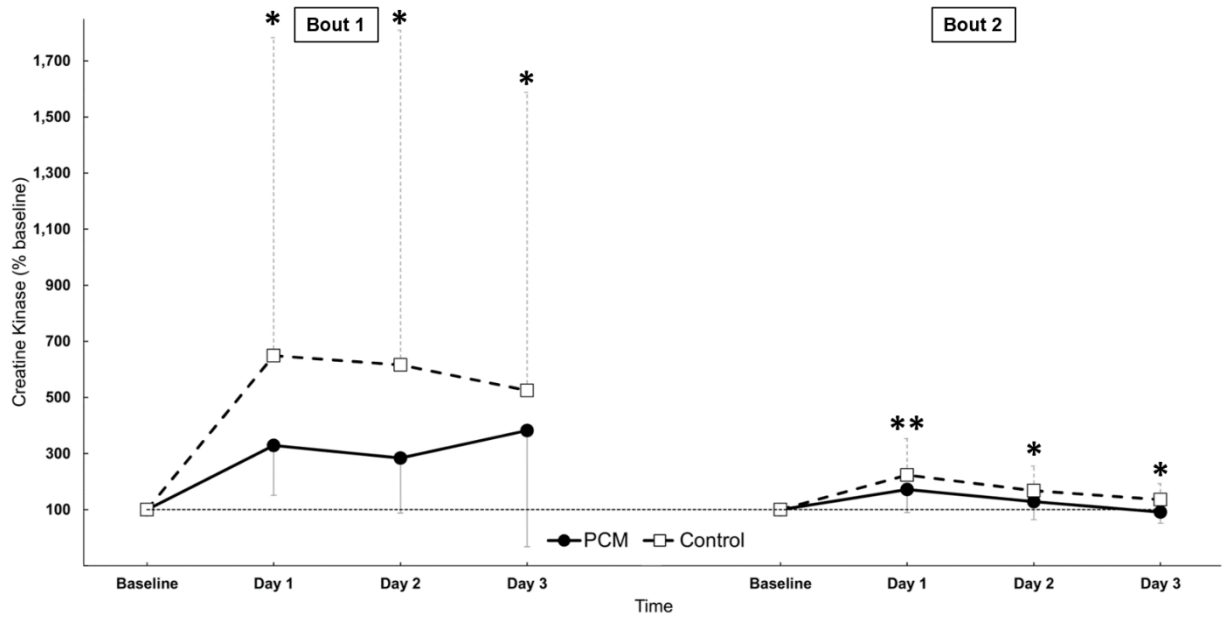


Fig. 4a) Plasma creatine kinase activity (log transformed) before and following bout 1 and bout 2. After the initial bout of exercise, CK was elevated above baseline on all 3 days (*: P<0.0001) but only on day 1 in the PCM treatment group and on day 1 and 2 after the repeated bout (*: P<0.0001), with no difference between groups. CK response was reduced in bout 2 versus bout 1 (#P<0.001) with no difference between groups. Values are mean ± SD.



4b) Plasma CK (% baseline) before and following bout 1 and bout 2. After the initial bout of exercise, CK was elevated above baseline on all 3 days (*: P<0.05). After the repeated bout, CK (% baseline) was only elevated above baseline on day 1 (*: P=0.024) for the PCM group and on day 1 (*: P=0.012) and day 2 (*: P=0.015) for the control group. Values are mean ± SD

Overall, hsCRP was slightly raised one day after exercise regardless of bout (time effect: P=0.022, ES=0.75; D1 P=0.009, ES=0.90), with no difference between bouts (P=0.358, ES=0.43) and no difference between treatments (treatment x time P=0.714, ES=0.28; treatment x bout x time P=0.563, ES=0.34). In bout 1, hsCRP was 0.66±0.32 µg/ml in the PCM cooling group and 0.76±0.60 µg/ml in control at baseline. On day 1, these values were 0.80±0.38 µg/ml and 0.87±0.63 µg/ml, for PCM treatment and control respectively. In bout 2, hsCRP values were 0.73±0.42 µg/ml and 0.86±0.68 µg/ml at baseline and 0.80±0.32 µg/ml and 1.24±1.13 µg/ml on day 1, respectively.

Skin Temperature and Compression

There was a treatment effect for skin temperature (P<0.001, ES=5.44). In the first bout, PCM treatment resulted in an average skin temperature of 23.9±0.7°C for the 6-hour treatment duration compared with 33.9±0.9°C during the control condition. Skin temperature did not differ between treatments in the second bout (P=0.264, ES=0.52). Compression pressure was not different between groups or across bouts in both seated (treatment x bout P=0.621, ES=0.22) or standing (treatment x bout P=0.209, ES=0.57) positions. Average compression pressure was 14.5±1.7 mm Hg while seated and 8.7±1.4 mm Hg while standing, which is negligible compared with the pressure needed to influence recovery through compression garments (14.8 ± 2.2 mm Hg while standing; Hill et al. 2017).

Discussion

This study investigated whether protection provided by PCM cooling after an initial bout of eccentric exercise compromised the RBE. In the initial bout, PCM cooling accelerated recovery of strength and soreness versus the control group with no difference in CK elevation and minimal hsCRP elevation in either group. Contrary to our hypothesis, there was no difference in strength, soreness, CK or hsCRP between groups after the repeated bout, indicating that the protection provided by PCM cooling in the initial bout was not compromised when cooling was not provided after the repeated bout. This is the first investigation to examine the effect of prolonged cooling from PCM on the RBE, and the first investigation to demonstrate that PCM did not compromise the adaptive response associated with the RBE in bout 2. These findings add to the literature that demonstrates PCM to be an effective strategy when recovering from EIMD (Clifford et al. 2018; Kwiecien et al. 2018).

In bout 1, the exercise was successful in inducing muscle damage, which was evident from the strength loss one day following the exercise in the control group (Fig. 2). Following exercise in bout 1, PCM cooling augmented quadriceps muscle strength compared to the control condition by day 3. These data are similar to previous findings using a similar muscle damaging protocol, but in untrained individuals, which also showed strength recovered by day 2 following PCM treatment (Kwiecien et al. 2018). Athletes have adopted CWI as a popular recovery modality aimed at limiting muscle damage and accelerating recovery (Versey et al. 2013). In a majority of the literature single post-exercise CWI treatments have been most commonly used, with some studies using CWI at intervals of 24 hours. Overall, the literature has failed to exhibit a favourable effect of CWI on recovery of muscle strength using these immersion protocols (Leeder et al. 2011). When multiple immersions were administered within the first 24 hours following lower body exercise, CWI was effective in accelerating recovery of muscle strength compared to control (Skurvydas et al. 2006). Repeat immersions within the first 24 hours are logical considering that exacerbation of muscle injury in the post-exercise period occur in several stages (Paulsen et al. 2010). Parts of which do not initiate until 2-6 hours following exercise (Armstrong et al. 1991). Unfortunately, multiple immersion sessions within this window may be inconvenient for athletes. Especially considering that access to CWI equipment over multiple sessions are difficult, and could mean that athletes need to remain at the training facility for long periods of time after competition. PCM presents an attractive alternative method of prolonging the duration of cryotherapy application, with little logistical challenges.

In addition to accelerating recovery of strength, PCM cooling also significantly reduced soreness compared to the control condition in bout 1. This is consistent with our prior work using PCM cooling and involving isokinetic eccentric quadriceps exercise in recreationally trained individuals (Kwiecien et al. 2018), and in soccer players (Clifford et al. 2018). Importantly, in bout 2, a trend was evident for reduced soreness from PCM cooling (P=0.061). Reduced soreness is also the most consistent effect of CWI (Bleakley et al. 2012; Hohenauer et al. 2015; Leeder et al. 2011; Machado et al. 2016). However, the overall effect of CWI on soreness might be dependent on exercise mode, as well as the degree of muscle damage, or the training status of the individual. Leeder et al (2011) demonstrated that CWI alleviated soreness up to 96 hours following high intensity exercise, but only up to 48 hours following eccentric exercise. CWI seems to be more effective in ameliorating effects of EIMD induced by whole

body prolonged endurance/intermittent based exercise modalities (Ihsan et al. 2016). The findings of the present study imply that prolonged cooling not only accelerated recovery of soreness following an initial bout of exercise, this effect did not inhibit the adaptive RBE response.

This was the first study to measure CK following prolonged PCM cooling. There was a clear elevation in CK in bout 1 with no effect of PCM cooling and a clear RBE that was not different between treatments (Fig. 4). Numerous studies have previously demonstrated an RBE for CK (Howatson et al. 2007; Nosaka et al. 2001). The lack of effect from PCM cooling on CK elevation in bout 1 is consistent with CWI literature showing no effect of CWI on CK efflux on the days after exercise (Anderson et al. 2018; Bailey et al. 2007; Bleakley et al. 2010; Fonseca et al. 2016; Halson et al. 2008; Howatson et al. 2008; Leeder et al. 2015; Pointon et al. 2012; Rowsell et al. 2009). This was also the first study to measure CRP following PCM cooling. Unfortunately, the exercise stress was insufficient to elevate CRP. Based on previous work, it was anticipated that performing bilateral eccentric quadriceps exercise at a high intensity would sufficiently elevate CRP (Margaritelis et al. 2015; Michailidis et al. 2013; Paulsen et al. 2005). However, compared to the present study which involved 120 eccentric contractions of each leg in the exercise protocol, the aforementioned studies performed 300 eccentric contractions (Michailidis et al. 2013; Paulsen et al. 2005) or 70 eccentric contractions repeated over multiple days (Margaritelis et al. 2015) at various ranges of motion. The exercise protocol in the present study was perhaps insufficient in inducing myofibrillar disruption that would result in inflammation. Other studies have shown no elevation in CRP from similar eccentric protocols (Bowtell et al. 2011). Studies using CRP to quantify the inflammatory response following CWI have also reported no effect (Banfi et al. 2007; Halson et al. 2008; Ingram et al. 2009).

This study is not without limitations. Although the participants performed the eccentric exercise at 101% of their MIVC at 80° of knee flexion, the muscle damage response in bout 1 was smaller than anticipated. The lower limbs are less susceptible to EIMD than the limbs of the upper extremity (Saka et al. 2009) because the lower limbs are regularly exposed to prior bouts of eccentric exercise during day-to-day locomotion. The literature primarily utilizes an upper extremity model to study EIMD (Clarkson and Tremblay, 1988; Newham et al. 1988; Nosaka et al. 1991; Nosaka and Newton 2002). The upper limb muscle groups are less accustomed to eccentric loading and for this reason are more susceptible to damage, but lack specificity when making inference to many sports and exercise (Howatson and van Someren 2008). In the present study participants performed eccentric contractions from 40-100° knee flexion to maximize the damage response. Marked strength loss and soreness were previously demonstrated performing the same intensity isokinetic eccentric exercise from 70-110° knee flexion (McHugh and Pasiakos 2004). By asking participants to reach a target torque of 90% MIVC during the eccentric exercise protocol in the present study, it transpired that participants exceeded the target torque and reached 100% of their MIVC, or greater. In bout 2, although the participants in the control condition exceeded 100% of their MIVC torque, performing the eccentric exercise at a greater intensity did not confound the results, as there was clearly a RBE for all variables on the days following exercise. Further, participants hit the target torque at the lower spectrum of the knee flexion range of motion and therefore did not do much eccentric work at the longer muscle lengths. In retrospect, range of motion of 70-110° would have necessitated peak torque occurring at greater knee flexion causing more damage. Compounding this problem, participants were relatively well trained in the quadriceps (college ice hockey, and other sports requiring strong quadriceps). While these participants would have been susceptible to damage at long muscle lengths, they were likely protected against damage with the eccentric peak torque occurring at a shorter muscle length than intended.

Current evidence indicates that CWI is not universally beneficial for adaptation to exercise. In particular, regular repeat immersions during strength training attenuate muscle adaptations, and improvements in both maximal strength (Roberts et al. 2015; Yamane et al. 2015) and muscle mass (Fyfe et al. 2019; Roberts et al. 2015; Yamane et al. 2015). Some of the proposed mechanisms behind these effects are an attenuation in the molecular mechanisms regulating resistance training adaptations (Broatch et al. 2018; Earp et al. 2019; Figueiredo et al. 2016; Lindsay et al. 2015; Lindsay et al. 2016; Roberts et al. 2015). In sports with multiple games is short periods, such as tournament play or fixture congestion, facilitating recovery is a priority. In the regular season of many sports, the goal of in-season management of the athlete is to facilitate recovery and avoid catabolism as opposed to inducing anabolic training adaptations. Interventions such as prolonged PCM cooling successfully accelerate recovery of not just soreness, but also strength, and may be useful during periods of training when the exercise stress is higher than normal or when there is inadequate time for recovery.

The only study examining the effects of CWI following damaging exercise on the RBE to date failed to show any favourable effect of CWI on variables of EIMD following the initial bout of exercise (Howatson et al. 2008). Thus,

both the CWI and control groups exhibited a RBE that was not compromised by the CWI. In contrast, following the initial bout of exercise in the present study, recovery of strength and soreness were accelerated from prolonged PCM cooling. Importantly, the adaptive response was not compromised by the prolonged cooling as evident by the RBE apparent in both the PCM and control conditions following the second bout of exercise. Thus, the present study supports the use of prolonged PCM cooling for its ability to mitigate decrements of strength and soreness following exercise while not interfering with the adaptive RBE response. The present study adds to evidence indicating that prolonged PCM cooling following exercise may enhance both short and longer term recovery. The precise mechanisms for accelerated recovery from prolonged PCM cooling remain to be elucidated.

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