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ORIGINAL ARTICLE

Cold water immersion recovery following intermittent-sprint exercise in the heat

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Abstract This study examined the effects of cold water immersion (CWI) on recovery of neuromuscular function following simulated team-sport exercise in the heat. Ten male team-sport athletes performed two sessions of a 2 × 30-min intermittent-sprint exercise (ISE) in 32°C and 52% humidity, followed by a 20-min CWI intervention or passive recovery (CONT) in a randomized, crossover design. The ISE involved a 15-m sprint every minute separated by bouts of hard running, jogging and walking. Voluntary and evoked neuromuscular function, ratings of perceived muscle soreness (MS) and blood markers for muscle damage were measured pre- and post-exercise, immediately post-recovery, 2-h and 24-h post-recovery. Measures of core temperature (Tcore), heart rate (HR), capillary blood and perceptions of exertion, thermal strain and thirst were also recorded at the aforementioned time points. Post-exercise maximal voluntary contraction (MVC) and activation (VA) were reduced in both conditions and remained below pre-exercise values for the 24-h recovery ($P < 0.05$). Increased blood markers of muscle damage were observed post-exercise in both conditions and remained elevated for the 24-h recovery period ($P < 0.05$). Comparative to CONT, the post-recovery rate of reduction in Tcore, HR and MS was enhanced with CWI whilst increasing MVC and VA ($P < 0.05$). In contrast, 24-h post-recovery MVC and activation were significantly higher in CONT compared to CWI ($P = 0.05$). Following exercise in the heat, CWI accelerated the reduction in thermal and cardiovascular load, and improved MVC alongside increased central activation immediately and 2-h post-recovery. However, despite improved acute recovery CWI resulted in an attenuated MVC 24-h post-recovery.

Keywords Thermal load · Voluntary activation · Neuromuscular · Exercise performance

Introduction

It is well established that exercise-induced increases in thermal strain result in alterations in central activation (Martin et al. 2004; Nielsen and Nybo 2003) and muscle contractile function (Morrison et al. 2004). Moreover, whole-body exercise performance is known to be reduced in the heat as elevations in thermal strain alter central and peripheral function (Nielsen et al. 2001; Nybo and Nielsen 2001); although athletes may alter pacing according to prevailing conditions (Tucker et al. 2004). Specifically, many team sports are involved in repeated bouts of exercise over consecutive days during training and competition, and when such events are performed in warm environmental conditions, performance decrements may be more pronounced (Vaile et al. 2008a). In an effort to alleviate potential perturbations associated with exercise bouts over multiple days in the heat, post-exercise recovery strategies are often employed. In particular, implementation of cold water immersion (CWI) has become an increasingly popular post-exercise recovery strategy (Barnett 2006). Current evidence highlights varying effectiveness of CWI on both acute and long-term recovery from exercise-induced muscle damage (Vaile et al. 2008b), laboratory cycling protocols (Peiffer et al. 2010b; Vaile

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et al. 2010) and team-sport exercise (Rowsell et al. 2009; 2011). Further, the effect of CWI following team-sport exercise in the heat and the relationship between endogenous thermal load and recovery of skeletal muscle function remains unknown.

Evidence to date suggests CWI decreases body temperature (Peiffer et al. 2010a; Vaile et al. 2010), reduces the inflammatory response (Knight 1989) and minimizes secondary muscle damage responses (Knight 1989; Knight et al. 2000). Despite evidence to suggest that CWI is beneficial in the treatment of acute soft tissue injury (Bleakley et al. 2004), research outlining the efficacy of CWI facilitating recovery of muscle function and exercise performance seems to vary based on the type of exercise mode used (Ingram et al. 2009; Jakeman et al. 2009; Rowsell et al. 2009, 2011). Recently, it has been reported that CWI following exercise in the heat maintained subsequent cycling performance (Vaile et al. 2008a) and resulted in a faster 4-km time trial (Peiffer et al. 2010a) when performed immediately post-CWI. However, despite recovery with CWI reducing muscle and rectal temperature, the rate of recovery in isokinetic strength immediately following a 1-km cycling time trial was not altered with CWI (Peiffer et al. 2010b). Moreover, CWI has been reported to have negative effects on the recovery of neuromuscular function resulting in a 13% decrease in maximal voluntary isometric force up to 90 min following a cycling time trial compared to a passive recovery (Peiffer et al. 2009). Although evidence remains equivocal, investigations reporting augmented recovery of exercise performance with CWI following exercise-induced increases in thermal load have primarily associated improvements to facilitated reductions in body temperature and cardiovascular strain (Vaile et al. 2010; Wilcock et al. 2006; Yeargin et al. 2006).

Although reduced performance during exercise in the heat is associated with alterations in central and peripheral function (Martin et al. 2004; Tucker et al. 2004), only one study has examined the effect of CWI on the recovery of neuromuscular function following exercise in the heat (Peiffer et al. 2009), with no studies to date using team-sport exercise. Therefore, the present study aimed to examine the effects of CWI following simulated team-sport exercise in the heat on the recovery of neuromuscular function, specifically the effects of CWI on central and peripherally mediated mechanisms of skeletal muscle recruitment. With the return of maximal voluntary force and activation observed only when cooling reversed core temperature (Tcore) back to normal values (~37.4°C) following passively induced hyperthermia (Morrison et al. 2004), we hypothesized that CWI following a bout of intermittent-sprint exercise (ISE) in the heat would improve maximal voluntary force due to an enhanced reduction in Tcore and improved recovery of neuromuscular function.

Methods

Participants

Ten male team-sport athletes (rugby league/union) aged (mean ± SD) 19.9 ± 1.1 years, height 179.6 ± 3.8 cm and body mass 78.9 ± 6.3 kg were recruited as participants for this study. At the time of testing, participants completed 3–4 training sessions per week and competed in team-sport competition at least once per week. All participants were informed of the requirements of the study and verbal and written consent was gained prior to the commencement of testing. Human ethics clearance was granted by the Institutional Ethics Committee prior to the completion of any testing procedures. Details that might disclose the identity of the subjects recruited have been omitted.

Overview

Participants completed an initial session to ensure familiarity with all measures and procedures, followed by two experimental testing sessions. The two testing sessions were identical apart from the recovery intervention implemented and were completed in a randomized, crossover order separated by at least 7 days. Each experimental session consisted of a prolonged high-intensity, ISE protocol (2 × 30 min halves) performed in an enclosed laboratory on a 20-m synthetic running track. The ISE protocol and ensuing recovery conditions were performed in 32.4 ± 1.5°C, 51.1 ± 6.2% relative humidity for CONT and 32.4 ± 1.3°C, 53.8 ± 8.5% relative humidity for CWI ($P = 0.23$–0.90). The ISE was followed by CWI or a passive recovery (CONT), with each testing session performed at the same time of day to minimize diurnal variation. Neuromuscular performance and repeated sprint ability (RSA) were measured pre- and within 5-min post-exercise, post-recovery and again 2- and 24-h post-recovery. Participants were required to present in a rested state and avoid consumption of food or drink (including caffeine) 3 h prior to testing and refrain from alcohol consumption 24 h prior to testing. All food, drink and physical activity in the 24 h prior to the first testing session and during the 24-h recovery period following the exercise protocol were recorded. Participants refrained from any strenuous activity during the 24-h recovery period and activity diaries were monitored throughout. Food, drink and activity prior to and during the first testing session were replicated for all testing sessions. During the ISE protocol, 500 mL of water was provided to

consume ad libitum with complete consumption ensured during each testing session.

Exercise protocol

Upon completion of pre-exercise neuromuscular tests (detailed subsequently), participants completed a warm-up involving running at increasing speeds over a 15-m running track for a period of 3 min, followed by 3 maximal 15-m sprints. The exercise protocol consisted of 2 × 30 min halves, interspersed by 10-min passive recovery. The exercise protocol involved a 15-m maximal sprint, (with a subsequent 5-m deceleration zone before impacting with a large mat) performed every min, followed by sub-maximal exercise of varying intensities in a self-paced, shuttle-run format (hard running, jogging, walking) for the remainder of the minute (Duffield and Marino 2007). Only one exercise mode of hard running, jogging or walking (rotated each minute) was completed each minute before returning to the starting position to complete the ensuing sprint. Every 6th rotation, following the maximal sprint, participants completed eight consecutive double leg bounds, covering as much distance as possible (distance determined for each exercise mode using 1-m markings alongside the 15-m synthetic track. Maximal 15-m sprint performance during the exercise protocol was assessed with infra-red timing gates (Speed-Light, Swift, Australia). As the exercise intensity was set by the participants, distances covered during the exercise protocol were monitored by the investigators and appropriate encouragement was provided to ensure similar workloads were performed during each testing session. Intra-class correlations (r) and coefficients of variation (CV) for distance covered were $r = 0.82$–0.96 and CV = 1.5–3.2%, respectively.

Recovery interventions

Within 10 min of completing the exercise protocol, the recovery intervention was administered in a designated area of the laboratory. CWI consisted of immersion in an ice bath (plunge pool) (8.9 ± 0.9°C) (Banfi et al. 2007) to a level of the iliac crest for 9 min followed by 1 min seated at room air temperature. This procedure was repeated twice for a total duration of 20 min (Peiffer et al. 2009, 2010b). For the passive recovery (CONT), participants remained seated in the laboratory for 20 min.

Procedures

Neuromuscular tests

For the neuromuscular tests, participants were seated on an isokinetic dynamometer (Humac Norm isokinetic dynamometer, Ausmedic, CSMi Medical Solutions, Stoughton, MA, USA) linked to a BNC2100 terminal block connected to a signal acquisition system (PXI1024; National Instruments, Austin, TX, USA). A/D conversion for torque and electromyographic data was performed at 16-bit resolution and synchronously sampled all data at a rate of 1 kHz. Participants were seated upright with a 90° hip angle on the dynamometer chair and securely fastened by adjustable straps tightly across the chest and pelvis with the distal right leg fixed to the dynamometer lever arm. The axis of rotation of the dynamometer was aligned to the lateral epicondyle of the femur indicating the anatomical joint axis of the knee. Torque was measured and recorded instantaneously. Lever arm length, chair length and dynamometer height were recorded during familiarization for accurate re-positioning during subsequent testing sessions.

Muscle activation

Muscle activation was achieved by stimulating the femoral nerve using a felt pad bar cathodal electrode with a tip spacing of 30 mm (Nicolet Biomedical, Madison, WI, USA) positioned at the medio-anterior aspect of the upper thigh, directly below the inguinal fold. The anode was a 90 × 50 mm reusable self-adhesive gel pad electrode (Verity Medical, Ltd., Stockbridge, Hampshire, UK) and positioned on the medio-posterior aspect of the upper thigh, directly below the gluteal fold, opposite the cathode. The current applied to the femoral nerve was delivered by a Digitimer DS7AH stimulator (Digitimer Ltd, Welwyn Garden City, Hertfordshire, UK) using a single square-wave pulse with a width of 200 μs (400 V with a current of 100–450 mA) that was driven by a custom designed instrument using LabView software (version 8.0, LabView; National Instruments). Initially, the current was manually applied in incremental steps until a twitch of moderate amplitude was observed. Following this, the position of the stimulating electrode was adjusted until the site most responsive to the stimulation was located. This location was marked with a permanent pen to ensure identical placement for subsequent testing. The electrode was then securely fastened in position using a Velcro strap with a constant force of 1.5 kg/f applied via an algometer (Pain Test™ FPI Algometer, Wagner Instruments, Greenwich, USA). The stimulus intensity was gradually increased until a plateau in twitch and M-wave amplitude was achieved. The stimulus intensity was then increased by a further 25% to ensure that supramaximal stimulation was applied to the nerve.

Maximal voluntary isometric contractions (MVC)

A 5-min warm-up at 60 W on a cycle ergometer (Monark 818E, Varberg, Sweden) was initially performed prior to

the measurement of MVC pre-exercise, 2- and 24-h post-recovery. During neuromuscular testing, participants performed 5 × 5-s MVC with 5-s rest between each contraction with the knee flexed at 65° (0° being full extension). Participants were instructed to produce a maximal effort for the entire 5 s at which time they were told to relax until the next MVC. A superimposed electrical twitch was delivered during each MVC when a reduction in peak force was observed. During each contraction, the trigger for stimulation was manually primed within 1–2 s after initiation of each contraction. Once primed, the stimulus was automatically triggered when customized LabView software (version 8.0, LabView; National Instruments) detected a decline in peak force. Manual priming of the trigger was necessary to prevent premature stimulation prior to the attainment of peak force. When primed, the decline in peak force necessary to automatically trigger the stimulus was <1%. Further, within 3 s following each superimposed contraction, a second stimulus was delivered with the muscle at complete rest to determine potentiated twitch properties. Mean voluntary isometric torque was determined by averaging the peak isometric torque produced over the five contractions (mean MVC). Mean torque values were determined during the 25 ms preceding the delivery of the electrical stimulus.

Voluntary activation (VA)

VA was calculated using the twitch interpolation technique (Allen et al. 1995). Peak superimposed torque following the delivery of the stimulus during the MVC's was determined as the peak torque value produced during the 50–150-ms period subsequent to the delivery of the stimulus. Interpolated twitch torque was subsequently determined as peak superimposed torque minus voluntary peak torque and calculated to four decimal places. VA was determined by expressing the interpolated twitch torque (ITT) as a percentage of the peak potentiated evoked twitch torque (Pt) obtained at rest between contractions using the following equation: $\text{VA}\ (\%) = [1 - (\text{ITT}/\text{Pt})] \times 100$. Peak VA was determined from the peak isometric contraction and mean VA was determined from the average of all five superimposed contractions. Both the mean and peak VA of the five contractions were used for subsequent analyses.

Potentiated evoked twitch contractile and M-wave properties

Potentiated twitch and M-wave properties were determined from an electrical stimulus initiated ~3 s following the superimposed contraction on the resting muscle. Torque-time curves from the potentiated evoked twitch contractions were averaged across all trials with mean data used to determine the following characteristics: (1) peak potentiated twitch torque (Pt); (2) the rate of torque development (RTD); (3) time to peak torque (TPt); (4) the rate of relaxation (RR); (5) half-relaxation time (1/2 RT); and (6) contraction duration (CD) (Cannon et al. 2006). Torque onset was determined as the point at which torque increased beyond 2 standard deviations above the mean torque value calculated over a 50-ms period immediately prior to stimulation (Wilder and Cannon 2009). Potentiated M-wave data was averaged across the 5 trials with the mean used to determine: (1) peak to peak amplitude, (2) duration, and (3) latency (Saboisky et al. 2003).

Surface electromyography (EMG)

Surface EMG data were obtained from the vastus lateralis (VL), vastus medialis (VM) and the antagonist biceps femoris (BF). Voluntary EMG data were obtained during the assessment of MVC pre-, post-exercise and all post-recovery assessments. EMG signals were sampled using differential surface electrodes (Bagnoli-16, Delsys Inc, Boston, USA) and positioned on VL, VM, BF according to Cram and Kasman (1998). Low impedance was obtained by shaving, abrading and cleaning the skin prior to positioning of the electrodes at each testing time point. The electrode placement sites were marked with permanent pen to ensure identical placement for subsequent testing sessions. In addition, a reusable self-adhesive electrode was attached to the patella of the opposing limb and acromion process for the arm to ground the signals. EMG signals were pre-amplified and bandpass filtered, with a bandwidth frequency ranging from 20 to 450 Hz (common mode rejection ratio >90 dB; impedance input = 100 MΩ; gain = 1,000).

Voluntary EMG signals were quantified using the root mean square (RMS) amplitude calculated as the average of the 25-ms preceding the superimposed twitch during MVC. The EMG signal was then averaged between vasti muscles to provide a global indication of total KE motor unit activity. For processing, voluntary EMG data were normalized against the peak to peak M-wave amplitude with average RMS data expressed as a percentage of the average VM/VL M-wave amplitude. All data were processed off-line with the determination of mean MVC, VA, potentiated twitch and M-wave properties, and RMS achieved using Matlab software (version R2010a; The MathWorks Inc. Natick, USA). For the isometric contractions, correction for the effect of gravity on the lower leg during the superimposed and potentiated evoked contractions was performed by calculating the average load applied to the force transducer during the 50-ms period immediately prior to force onset. The average load applied to the transducer

during this period was used to offset force data. Once corrected for the effect of gravity, force data were then multiplied by lever arm length and expressed in units of torque (N m^{-1}).

Repeated sprint ability

Participants performed a repeated sprint exercise protocol prior to, post-exercise, post-recovery and 2- and 24-h post-exercise. The RSA protocol consisted of 5 × 15-m maximal sprints performed every 20 s. Maximal 15-m sprint times were assessed with infra-red timing gates (Speed-Light, Swift, Australia) and the percentage decline within each 5 × 15-m bout was calculated [(total time/(fastest time sprint *n*) 100)].

Capillary and venous blood measures

On arrival, a 100 μL sample of capillary blood was obtained for the measurement of Lactate (La^{-}), pH and bicarbonate (HCO_3) with further samples obtained immediately post-exercise and post-recovery intervention. A 5 mL sample of venous blood was obtained from the antecubital vein pre-exercise and at post-recovery intervals (immediately, 2 and 24 h) for the measurement of creatine kinase (CK), C-reactive protein (CRP), aspartate aminotransferase (AST) as markers of muscle damage and cell inflammation. Using an evacuated venipuncture system and serum separator tubes (Monovette, Sarstedt, Numbrecht, Germany), samples were allowed to clot at room temperature prior to centrifugation for 10 min at 4,000 rpm. Serum was then extracted and stored at −20°C until analysis. Before analysis, the serum was allowed to reach room temperature and mixed gently via inversion. CK, CRP and AST were analyzed according to manufacturer's instructions provided in the respective assay kits (Dimension Xpand spectrophotometer, Dade Bearing, USA). Intra-assay Coefficients of Variance were <5% for all venous blood analyses.

Nude mass, heart rate and core temperature

Nude mass was recorded on arrival and immediately after the exercise protocol on a set of calibrated scales (HW 150 K, A and D, Tokyo, Japan). Heart rate (HR) was measured with a heart rate monitor and wrist watch receiver (F1, Polar Electro-Oy, Finland). Core temperature (Tcore) was measured with a telemetric capsule (Vital Sense, Mini Mitter, Oregon, USA) ingested 4 h prior to each testing session to ensure it had passed into the gastrointestinal tract. Tcore was assessed with a hand-held monitor that telemetrically received measures from the ingested capsule (VitalSense, Mini Mitter, Oregon, USA). HR and Tcore were measured prior to the initial warm-up, every 5 min during the exercise protocol, immediately post-recovery and 2-h post-recovery.

Perceptual measures

Perceptual measures of rating of perceived exertion (RPE) were determined using the Borg 6–20 Scale (Morrison et al. 2004). Muscle soreness (MS), thirst and thermal strain were all determined using 10-point Likert scales (MS: 0 = no pain and 10 = very very sore; thirst: 0 = not thirsty and 10 = extremely thirsty; thermal strain: 0 = unbearably cold and 10 = unbearably hot). RPE, thirst and thermal strain were determined pre- and post-exercise and every 5 min during the exercise protocol, whilst MS was determined pre- and post-exercise and throughout the recovery period (post-recovery, 2 and 24 h).

Statistical analysis

Data recorded from neuromuscular, physiological and perceptual measures are reported as mean ± SD. A repeated measures analysis of variance (ANOVA) (condition × time) was used to determine significant difference between conditions and over time for each recovery intervention. Significant difference ($P < 0.05$) between time points was determined using planned within-subject contrasts. Mauchly's test of sphericity was performed to test for the homogeneity of variance (Portney and Watkins 2009) with Greenhouse-Geisser correction applied if sphericity was significant ($P < 0.05$). All data collected were analyzed using SPSS™ version 16.0 (Statistical Package for the Social Sciences, Chicago, IL, USA).

Results

Distance covered and sprint time

There were no significant differences between conditions for the total distance covered during the exercise protocol ($P = 0.80$; 4,207 ± 550 m CONT vs. 4,170 ± 342 m CWI). Total distance covered for hard running was 1,843 ± 224 m CONT and 1,842 ± 180 m CWI ($P = 0.10$). Distance covered for jogging was 1,449 ± 223 m CONT and 1,436 ± 133 m CWI ($P = 0.78$); whilst 915 ± 122 m CONT and 892 ± 88 m CWI were covered during walking ($P = 0.54$). There were no significant differences between conditions for double leg bound distance at any time point (134.8 ± 10.1 m CONT vs. 134.5 ± 9.2 m CWI; $P =$ 0.30–0.80). Total time for maximal 15 m sprints during the exercise protocol was 176.1 ± 8.2 s CONT and 175.2 ± 3.2 s CWI ($P = 0.10$–0.90).

Repeated sprint ability

Mean sprint time and percentage decline in 5 × 15-m maximal sprints were significantly increased post-exercise and remained above pre-exercise values up to 2-h post-recovery in both conditions ($P < 0.05$; Table 1). No significant differences were evident between recovery conditions for repeated 5 × 15-m sprints during any time point ($P > 0.05$).

Maximal voluntary contractions and activation

Post-exercise mean MVC and VA were significantly reduced compared to pre-exercise values in both conditions ($P = 0.001$–0.01; Fig. 1). Mean VA did not return to baseline values until 2-h post-recovery, with MVC remaining below pre-exercise values for the 24-h recovery period ($P = 0.01$; Fig. 1) in both conditions. Compared to CONT, mean MVC and VA were significantly higher post-recovery following CWI ($P = 0.01$). However, 24-h post-recovery mean MVC was greater in the CONT condition compared to CWI ($P = 0.04$).

Potentiated twitch and M-wave properties

The exercise protocol resulted in significantly reduced Pt, RR and TPt ($P = 0.002$–0.05; Table 2). Pt and RR remained below pre-exercise values post-recovery and 2-h post-recovery in both conditions ($P = 0.001$–0.01). Despite the post-exercise reduction in Pt and TPt, no significant differences were evident between the recovery conditions at any time point ($P = 0.06$–0.99; Table 2). Compared to CONT, CWI resulted in a significantly reduced RR and CD post-recovery ($P = 0.01$), which remained evident 24-h post-recovery for RR only ($P = 0.01$). Exercise did not significantly alter the duration and latency of the M-wave ($P = 0.09$–0.90; Table 3). Compared to CONT, post-recovery M-wave duration was significantly increased following CWI ($P = 0.02$; Table 3). No significant differences were evident between respective recovery conditions at any time point for M-wave latency ($P = 0.10$–0.90; Table 3).

Voluntary EMG

Voluntary EMG (RMS) expressed as a percentage of the M-wave amplitude was not significantly different between conditions at any time point ($P = 0.10$–0.80). Post-exercise mean RMS of VM/VL was significantly reduced compared to pre-exercise values in both conditions ($P = 0.02$–0.05; Fig. 2). Compared to CONT, mean RMS of VM/VL was significantly increased post-recovery in CWI ($P = 0.05$; Fig. 2). However, 24-h post-recovery, mean RMS values of VM/VL were significantly increased

Table 1 Mean and percentage decline ± SD maximal 5 × 15-m repeated sprint time for cold water immersion (CWI) and passive recovery (CONT)

	Pre	Post	Post-rec	2-h Post	24-h Post
Mean sprint time					
CONT	2.64 ± 0.07	2.92 ± 0.13	2.80 ± 0.14	2.70 ± 0.08	2.62 ± 0.08
CWI	2.61 ± 0.06	3.02 ± 0.20	2.81 ± 0.11	2.71 ± 0.09	2.64 ± 0.08
% Decline					
CONT	2.33 ± 1.02	8.43 ± 4.04	4.55 ± 2.96	2.50 ± 1.42	1.93 ± 1.05
CWI	2.24 ± 0.67	8.29 ± 4.04	3.59 ± 1.97	1.94 ± 1.30	1.71 ± 0.50

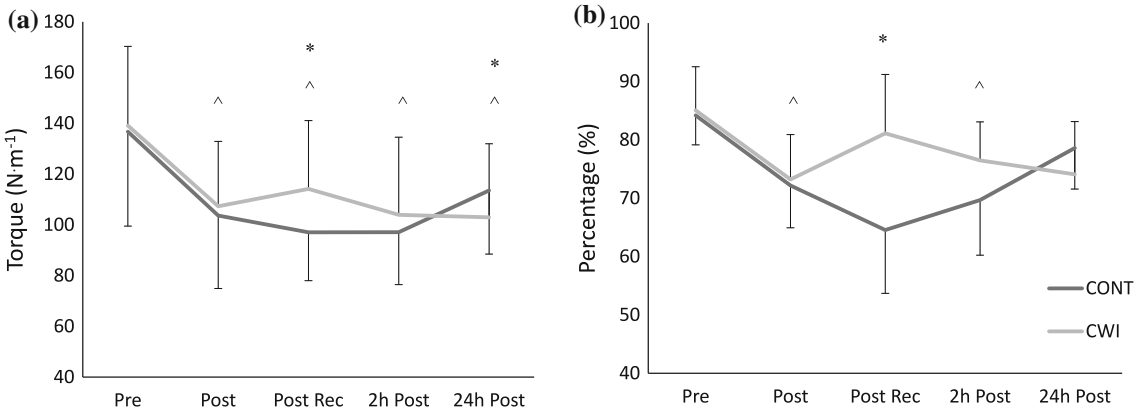

Fig. 1 Mean ± SD **a** mean voluntary torque (*MVC*) and, **b** activation (*VA*) for cold water immersion (*CWI*) and passive recovery (*CONT*). ^Significant time effect from pre-exercise values ($P < 0.05$). *Significant difference between conditions ($P < 0.05$)

Table 2 Mean ± SD potentiated twitch properties for cold water immersion (CWI) and passive recovery (CONT)

	Pre	Post	Post-rec	2-h Post	24-h Post
Pt (Nm)					
CONT	43.1 ± 7.1	33.9 ± 11.8^	30.7 ± 9.2^	34.9 ± 10.1^	36.1 ± 10.7
CWI	41.9 ± 10.3	37.5 ± 7.1^	35.6 ± 8.4^	36.0 ± 7.5^	40.5 ± 8.8
TPt (ms)					
CONT	95.3 ± 9.6	87.8 ± 14.7^	84.2 ± 11.2	86.1 ± 6.0	88.2 ± 18.0
CWI	99.8 ± 14.8	86.8 ± 14.4^	103.3 ± 17.1	92.1 ± 17.4	98.9 ± 16.4
½ RT (ms)					
CONT	81.6 ± 11.2	73.5 ± 9.7	86.2 ± 8.9	92.8 ± 11.5	91.8 ± 5.1
CWI	81.6 ± 12.9	72.6 ± 13.1	84.0 ± 12.3	90.0 ± 13.8	81.3 ± 14.6
RTD (Nm s^{-1})					
CONT	454.4 ± 64.6	402.8 ± 156.4	362.3 ± 142.9	411.1 ± 120.2	417.5 ± 139.0
CWI	437.7 ± 65.5	438.5 ± 80.1	346.9 ± 68.8	398.8 ± 88.2	417.4 ± 98.4
RR (Nm s^{-1})					
CONT	−273.6 ± 71.7	−249.6 ± 61.9^	−176.5 ± 54.6*^	−194.9 ± 65.9^	−200.1 ± 61.9*
CWI	−270.3 ± 91.3	−271.2 ± 77.8^	−217.6 ± 49.1^	−209.4 ± 69.3^	−263.0 ± 85.4
CD (ms)					
CONT	176.8 ± 9.9	162.5 ± 13.4	168.9 ± 10.5*	178.9 ± 15.3	179.0 ± 17.8
CWI	181.4 ± 16.1	163.4 ± 13.0	187.2 ± 14.5	182.1 ± 20.0	180.2 ± 10.3
Latency (ms)					
CONT	22.4 ± 6.5	20.5 ± 7.9	24.9 ± 6.2	21.6 ± 8.2	25.7 ± 9.0
CWI	21.2 ± 6.0	21.4 ± 8.1	25.4 ± 8.3	24.1 ± 9.3	23.5 ± 4.5

Pt peak twitch, *TPt* time to peak twitch, *½ RT* half-relaxation time, *RTD* rate of torque development, *RR* rate of relaxation and *CD* contraction duration

* Significant difference between conditions ($P < 0.05$)

^ Significant difference within conditions from pre-exercise values ($P < 0.05$)

in CONT compared to CWI ($P = 0.05$; Fig. 2). Absolute RMS of BF was not altered by the exercise protocol and no significant differences were evident between conditions at any time point ($P > 0.05$; Fig. 2).

Nude body mass, heart rate and core temperature

Nude body mass was significantly reduced post-exercise ($P = 0.01$) in CONT (1.60 ± 0.50 kg) and CWI (1.48 ± 0.41 kg), with no significant differences evident between the conditions ($P = 0.50$). Tcore and HR significantly increased post-exercise compared to pre-exercise values in both conditions ($P = 0.01$). Following CWI, absolute Tcore values were not significantly different compared to CONT ($P = 0.50$; Fig. 3); however, the relative rate of change in Tcore was significantly faster post-recovery and 2-h post-recovery compared to CONT ($P = 0.05$ and 0.04, respectively; Fig. 3). In addition, CWI resulted in a significantly reduced HR post-recovery compared to a CONT ($P = 0.02$; 90 ± 10 vs. 101 ± 14 beats min^{-1}, respectively).

Table 3 Mean ± SD potentiated M-wave properties for the mean of vastus lateralis and vastus medialis for cold water immersion (CWI) and passive recovery (CONT)

	Pre	Post	Post-rec	2-h Post	24-h Post
Latency (ms)					
CONT	10.9 ± 1.9	10.2 ± 1.8	11.3 ± 1.7	9.6 ± 1.9	11.1 ± 1.7
CWI	11.1 ± 4.2	11.2 ± 1.8	12.2 ± 3.0	11.7 ± 2.5	11.0 ± 2.6
Duration (ms)					
CONT	4.9 ± 1.9	4.2 ± 0.9	3.6 ± 1.1*	4.6 ± 1.4	4.4 ± 1.5
CWI	5.2 ± 2.0	5.4 ± 2.4	4.9 ± 1.2	4.8 ± 2.1	4.5 ± 1.2
Amplitude (mV)					
CONT	1.2 ± 0.2	1.3 ± 1.1	0.9 ± 0.2	1.0 ± 0.4	1.0 ± 0.3
CWI	1.0 ± 0.3	1.0 ± 0.4	1.2 ± 0.4	1.0 ± 0.3	1.0 ± 0.3

* Significant difference between conditions ($P < 0.05$)

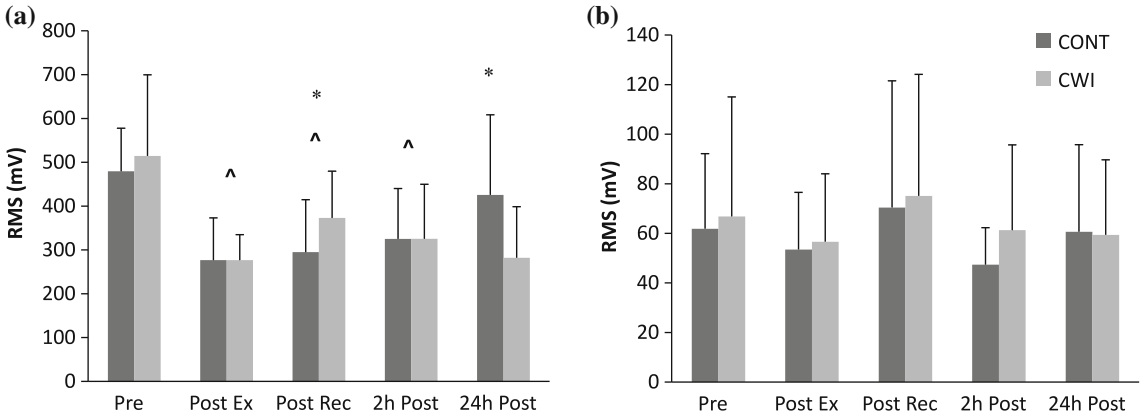

Fig. 2 Mean ± SD of root mean square (*RMS*) for **a** average vastus medialis and vastus lateralis and, **b** biceps femoris for cold water immersion (*CWI*) and passive recovery (*CONT*). *Significant difference between conditions ($P < 0.05$). ^Significant time effect from pre-exercise values ($P < 0.05$)

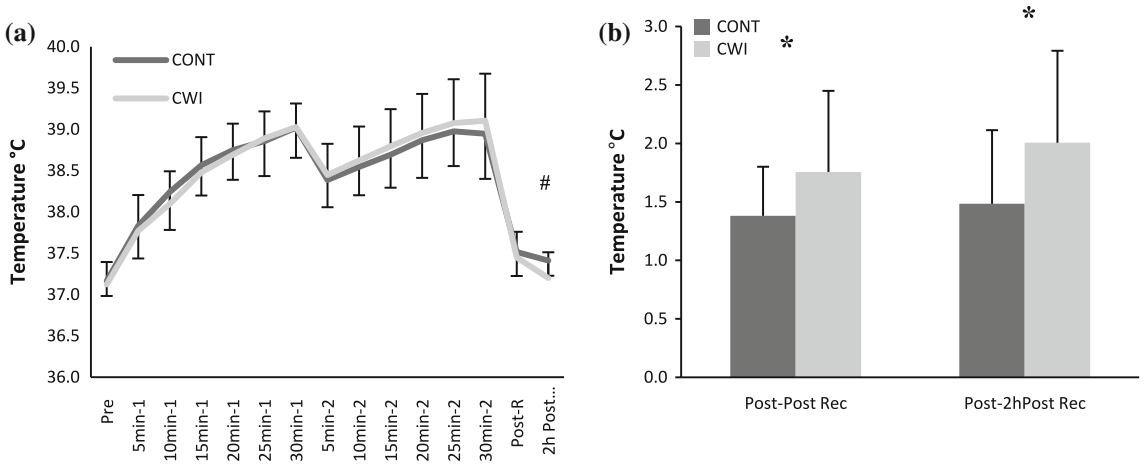

Fig. 3 Mean ± SD **a** Core temperature during the exercise protocol and post-recovery and, **b** rate of change in core temperature from post-exercise to post-recovery, and post-exercise to 2-h post-recovery for cold water immersion (*CWI*) and passive recovery (*CONT*)

Venous and capillary blood variables

Significant post-exercise increases in CK, AST and La^- were evident, with CK values remaining elevated during the 24-h recovery period ($P = 0.01$–0.03) as presented in Table 4. Despite exercise-induced elevations in CK, AST and La^-, no differences were evident between recovery conditions at any time point ($P = 0.10$–0.80; Table 4). CRP was not significantly altered by the exercise protocol and no significant differences were observed between recovery conditions at any time point ($P = 0.10$–0.80; Table 4). Further, no significant differences were evident between the conditions for the % change in CK, AST and CRP at any time point ($P = 0.14$–0.90; Table 4). Post-exercise pH and HCO_3^- decreased significantly from pre-exercise values in both conditions ($P = 0.01$–0.05; Table 4); however, no significant differences were evident between conditions at any time point ($P = 0.10$–0.80).

Perceptual measures

Ratings of MS were significantly increased post-exercise and remained elevated above pre-exercise values during the 24-h recovery period ($P = 0.01$). Compared to CONT, perceptions of MS were significantly reduced immediately post-recovery in CWI ($P = 0.02$; 4.5 ± 0.9 CWI vs. 5.7 ± 0.9 CONT). Ratings of thermal strain, perceived exertion and thirst were significantly increased during the exercise protocol ($P = 0.01$). Compared to CONT, thermal strain was significantly lower immediately following CWI ($P = 0.01$; 3.5 ± 1.2 CWI vs. 6.2 ± 0.9 CONT); whilst, no significant differences were

Table 4 Mean ± SD capillary and venous blood variables for cold water immersion (CWI) and passive recovery (CONT)

	Pre	Post-ex	Post-rec	2-h Post	24-h Post
La^- (mmol l^{-1})					
CONT	1.18 ± 0.15	4.62 ± 1.17^	1.43 ± 0.47		
CWI	1.14 ± 0.14	4.73 ± 1.50^	1.80 ± 0.60		
pH (mmol l^{-1})					
CONT	7.36 ± 0.03	7.37 ± 0.02^	7.39 ± 0.02^		
CWI	7.37 ± 0.02	7.37 ± 0.05^	7.40 ± 0.03^		
HCO_3 (mmol l^{-1})					
CONT	22.29 ± 0.69	19.70 ± 1.44^	22.42 ± 0.79*		
CWI	22.19 ± 0.65	19.59 ± 1.82^	21.87 ± 0.54		
CK (IU l^{-1})					
CONT	308.1 ± 189.7	418.8 ± 205.5^	394.5 ± 209.1^	419.3 ± 228.3^	551.7 ± 359.5^
CWI	359.1 ± 157.9	477.1 ± 199.9^	489.0 ± 208.2^	544.5 ± 252.3^	708.9 ± 362.7^
CK (% change)					
CONT		146.7 ± 30.8	135.2 ± 20.0	143.8 ± 26.0	194.7 ± 106.0
CWI		135.6 ± 13.5	141.5 ± 28.1	160.5 ± 50.5	219.7 ± 118.5
AST (IU l^{-1})					
CONT	25.30 ± 3.86	32.75 ± 3.56^	33.25 ± 3.51	31.88 ± 3.79	32.50 ± 4.13
CWI	33.13 ± 5.43	39.00 ± 7.67^	40.00 ± 5.42	37.13 ± 6.16	39.13 ± 5.42
AST (% change)					
CONT		110.7 ± 25.5	111.5 ± 27.5	106.6 ± 23.5	106.9 ± 33.6
CWI		117.2 ± 18.5	135.8 ± 32.5	126.5 ± 41.1	128.5 ± 32.2
CRP (IU l^{-1})					
CONT	1.6 ± 0.9	1.8 ± 1.3	1.7 ± 1.2	1.7 ± 1.1	2.1 ± 1.3
CWI	1.9 ± 1.2	2.0 ± 1.2	1.9 ± 1.3	2.0 ± 1.0	2.5 ± 1.3
CRP (% change)					
CONT		100.6 ± 24.0	98.7 ± 24.9	104.0 ± 19.7	133.6 ± 45.2
CWI		106.7 ± 9.1	100.7 ± 28.6	116.8 ± 31.4	161.0 ± 83.1

La^- Lactate, pH, *HCO_3* bicarbonate, *CK* creatine kinase, *AST* aspartate aminotransferase and *CRP* c-reactive protein

* Significant difference between conditions ($P < 0.05$)

^ Significant difference from pre-exercise values ($P < 0.05$)

evident between recovery conditions at any time point for thirst and RPE ($P > 0.05$).

Discussion

This investigation aimed to examine the efficacy of CWI on the recovery of performance and physiological function following simulated team-sport exercise in the heat and during the ensuing 24-h recovery period. Following exercise-induced reductions in muscle contractile force generation, CWI significantly improved acute recovery of MVC and VA. Furthermore, a faster reduction in post-recovery Tcore and HR, together with reduced perceptions of muscle soreness and thermal strain were evident following CWI. The reduction in post-recovery Tcore and HR with CWI likely contributed to the enhanced acute recovery of MVC, and the novel finding of increased central activation (VA) and motor unit recruitment (RMS) observed in the present study. Despite initial improvements in MVC and VA following CWI, an additional novel finding of this investigation was the suppression of voluntary force and RMS 24-h post-recovery following CWI compared to passive recovery. That is, MVC was ~85% of pre-exercise values in CONT compared to ~74% in CWI; thus resulting in an 11% impaired recovery of voluntary force 24-h post-recovery. However, despite altered muscle function, no changes in RSA were evident following cooling; highlighting the divergence in isolated joint versus whole-body exercise performance. Accordingly, based on the results of the present study, implementation of CWI on the recovery of neuromuscular and contractile function following team-sport exercise in the heat may be time and mode dependent providing immediate beneficial effects to recovery of isometric MVC; however, counter-productive to long-term recovery of single-joint isometric voluntary force production.

Simulated team-sport exercise in the heat resulted in prolonged reduction in MVC and VA which is commonly observed following exercise-induced elevations in thermal stress (Martin et al. 2004; Nybo and Nielsen 2001; Thomas et al. 2006). Impaired performance with increased thermal load has previously been associated with a reduction in centrally mediated recruitment and activation (Martin et al. 2004; Nybo and Nielsen 2001; Thomas et al. 2006). The exercise protocol in the current study also resulted in reduced peak twitch contractile force, evident up to 2-h post-recovery. Therefore, reductions in post-exercise MVC likely resulted from a combination of reduced neural activation of skeletal musculature (Nybo and Nielsen 2001) and suppression of peripheral contractile ability (Hargreaves 2004).

In accordance with previous research (Peiffer et al. 2010a; Vaile et al. 2008a, 2010), implementation of CWI recovery resulted in a faster rate of reduction in Tcore and HR, with subsequent improvements in performance. Indeed, Vaile et al. (2010) and Peiffer et al. (2010a) recently demonstrated a more rapid reduction in Tcore and HR with post-exercise CWI, and subsequent improved repeated cycling performance in the heat. Peiffer et al. (2010a) observed smaller reductions in power output and thus a faster completion of a subsequent 4-km cycling time trial when performed immediately following CWI compared to a seated recovery (1.5 ± 0.2% CWI vs. 14 ± 1.0% control). The authors postulated that the mechanisms for improved exercise performance were related to CWI reducing post-exercise thermal and cardiovascular strain (Yeargin et al. 2006). Whilst the faster reduction in Tcore is a likely explanation for the observed performance enhancement, a further explanation may include the role of enhanced centrally mediated skeletal muscle recruitment. Accordingly, a novel finding of the present investigation was the ameliorated recovery of central activation; together with increased RMS following CWI. Thus, acute improvements in post-recovery MVC are likely a result of centrally mediated mechanisms increasing skeletal muscle activation and recruitment based on CWI producing a faster reduction in internal thermal load. Therefore, CWI interventions to hasten recovery of increased thermal load may also act to negate the reported centrally mediated suppression of VA (Morrison et al. 2004); hence allowing greater skeletal muscle recruitment and improved acute MVC performance following post-exercise CWI.

Further to improvements in central activation, CWI recovery resulted in altered peripheral contractile properties. Slower CD, RR and TPt of the potentiated twitch and duration of the M-wave were evident immediately following CWI compared to CONT. To control for the effects of temperature on the evoked signal, M-wave amplitude was normalized to the voluntary EMG signal. Upon normalization, no differences between conditions were evident. Unfortunately, a limitation of the present study was that the temperature of the muscle during evoked twitches was not measured. Despite this, it is well known that reduced muscle temperature with the application of cold significantly alters muscle contractile properties and slows nerve conduction velocity (Eston and Peters 1999). Implementation of a re-warm up in the present study performed prior to 2- and 24-h post-recovery measurements attempted to provide a similar level of potentiation in the evoked signal prior to MVC measures. With no differences evident between the conditions for twitch and M-wave duration at 2- and 24-h post-recovery, the re-warm up was sufficient to counter potential negative effects of cold temperatures on the evoked signal. Regardless of the potential for such changes in evoked twitch contractile properties and whilst the effect of temperature remained evident (post-recovery), the immediate recovery of voluntary force (MVC) following CWI was enhanced compared to a passive recovery. Although CWI slowed the response to the evoked signal, increased VA and RMS highlights the influence of central activation in enhancing voluntary force production. Thus, results of the present study indicate that ameliorated recovery of acute MVC is likely due to other factors including reductions in whole-body endogenous thermal and cardiovascular strain (Yeargin et al. 2006), and improved perceptions of thermal recovery and MS.

The ISE protocol in the current study resulted in significant reductions in muscle contractile properties (Pt and M-wave amplitude) and prolonged elevations in CK and AST. Recent studies examining the influence of CWI on markers of muscle damage after a simulated cycling time trial in the heat (Halson et al. 2008) and a 90-min rugby training session (Banfi et al. 2007) have reported no effect of CWI on the appearance of CK and CRP. However, these studies measured the appearance of CK and CRP immediately post-recovery (within 40 min of exercise cessation) and, therefore, the peak expression of such markers is unlikely to have been evident, which may explain the lack of difference between conditions. Regardless, in accordance with previous research, the results of the present investigation also demonstrated that CWI was ineffective in reducing the immediate and prolonged (24 h) appearance of blood markers of muscle damage (Bailey et al. 2007; Halson et al. 2008; Rowsell et al. 2009). Further, a common finding in the literature is the improved perception of MS for up to 48 h following the post-exercise use of CWI recovery interventions (Ascensão et al. 2011; Parouty et al. 2010). For example, Parouty et al. (2010) recently reported an immediate increase in the perception of recovery when CWI was performed between 2 × 100-m swimming sprints. Further, both acute (30 min) and prolonged (24 h) reductions in leg soreness were observed

when CWI immediately followed a one-off soccer match (Ascensão et al. 2011). Similarly, the results of the present study demonstrated an immediate reduction in MS following CWI. Whether the improved perception of recovery from CWI contributes to the observed acute improvements in MVC and VA is unknown, and further research may be required to fully elucidate the role of perceptual recovery.

Despite acute improvements in MVC and VA, suppression of force production and global KE RMS was evident 24-h post-recovery following CWI compared to CONT. The decrement in exercise performance 24-h post-recovery contrasts with previous investigations examining CWI following ISE (Bailey et al. 2007; Ingram et al. 2009). Possible explanations for differences between the present study and previous investigations may be due to the incorporation of double leg bounds, resulting in greater exercise-induced muscle damage (EIMD) compared to intermittent running and cycling exercise in previous studies (Bailey et al. 2007; Ingram et al. 2009). Indeed, previous investigations reporting no benefit of CWI on recovery of muscle function and strength loss often elicit EIMD via single-joint modalities, frequently causing trauma to muscle contractile properties (Eston and Peters 1999; Howatson et al. 2005; Jakeman et al. 2009). Thus, the implementation of cold therapy post-damaging exercise may not be beneficial to the repair of contractile trauma (Yamane et al. 2006). Although the application of cold has been shown to reduce acute inflammation following musculoskeletal injury (Knight 1989), it has recently been suggested that repressing acute inflammation negatively affects repair and regenerative processes of skeletal muscle and may be detrimental to prolonged muscle performance (Barnett 2006; Yamane et al. 2006). As such, although CWI was effective in reducing Tcore, HR and thermal strain, resulting in enhanced short-term recovery of voluntary force, CWI was ineffective in maintaining long-term recovery of isometric muscle function compared to CONT. Despite CWI-induced decrements in 24-h post-recovery single-joint MVC, RSA did not differ. Accordingly, from an applied perspective, while CWI may result in the reduction of single-joint isometric MVC, the ability to produce repeated maximal effort sprints was not compromised.

In conclusion, CWI recovery following simulated team-sport exercise in the heat enhanced the rate of reduction in Tcore, HR and thermal strain, resulting in improved acute recovery of MVC. With an increase in VA and RMS observed post-CWI, it is likely that reductions in thermal and cardiovascular strain improved centrally mediated mechanisms increasing skeletal muscle recruitment contributing to ameliorated short-term recovery of MVC. Despite acute improvements to the recovery of MVC, CWI resulted in a decrement in voluntary force production and global KE RMS 24-h post-recovery. The precise mechanisms responsible for observed decrements in force production 24-h post-recovery with CWI are unknown and, therefore, further research is required to fully elucidate the long-term effects of CWI on muscle repair and adaptation processes necessary for improved isometric muscle function. However, regardless of the recovery of isolated skeletal muscle contractile function, no differences in RSA were evident. Accordingly, practitioners should be aware of the mode and duration specific responses to CWI as a recovery intervention following team-sport exercise in the heat.

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