

Cold water immersion enhances recovery of submaximal muscle function after resistance exercise

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Submitted 2 May 2014; accepted in final form 6 August 2014

Roberts LA, Nosaka K, Coombes JS, Peake JM. Cold water immersion enhances recovery of submaximal muscle function after resistance exercise. *Am J Physiol Regul Integr Comp Physiol* 307: R998–R1008, 2014. First published August 13, 2014; doi:10.1152/ajpregu.00180.2014.—We investigated the effect of cold water immersion (CWI) on the recovery of muscle function and physiological responses after high-intensity resistance exercise. Using a randomized, cross-over design, 10 physically active men performed high-intensity resistance exercise followed by one of two recovery interventions: 1) 10 min of CWI at 10°C or 2) 10 min of active recovery (low-intensity cycling). After the recovery interventions, maximal muscle function was assessed after 2 and 4 h by measuring jump height and isometric squat strength. Submaximal muscle function was assessed after 6 h by measuring the average load lifted during 6 sets of 10 squats at 80% of 1 repetition maximum. Intramuscular temperature (1 cm) was also recorded, and venous blood samples were analyzed for markers of metabolism, vasoconstriction, and muscle damage. CWI did not enhance recovery of maximal muscle function. However, during the final three sets of the submaximal muscle function test, participants lifted a greater load ($P < 0.05$, Cohen's effect size: 1.3, 38%) after CWI compared with active recovery. During CWI, muscle temperature decreased $\sim 7^\circ\text{C}$ below postexercise values and remained below preexercise values for another 35 min. Venous blood O_2 saturation decreased below preexercise values for 1.5 h after CWI. Serum endothelin-1 concentration did not change after CWI, whereas it decreased after active recovery. Plasma myoglobin concentration was lower, whereas plasma IL-6 concentration was higher after CWI compared with active recovery. These results suggest that CWI after resistance exercise allows athletes to complete more work during subsequent training sessions, which could enhance long-term training adaptations.

cryotherapy; recovery; performance; thermoregulation; muscle damage; blood gases

RECOVERY between exercise training sessions and competitive events is a key determinant of long-term training adaptation and successful performance. Inadequate recovery after exercise may prevent athletes from training at the required intensity or completing the required loads during subsequent training sessions. It may also increase the risk of injury, illness, and overtraining (1). Recognition of the importance of recovery for training adaptation and maintenance of performance has stimulated intense interest in the effectiveness of various strategies to promote faster recovery from intense exercise. Some of these recovery strategies are collectively referred to as “cryotherapy” and include immersion in cold water/ice baths, topical

application of ice, or brief exposure to extreme cold air. Numerous studies have examined the effects of various forms of cryotherapy on muscle function and indirect markers of muscle damage after intense eccentric exercise (6, 8, 9, 16, 18, 20, 37, 39, 40, 42, 45) and plyometric exercise (14, 19, 24). In contrast, fewer studies (10, 12, 13, 23) have assessed the potential benefits of cryotherapy after more traditional resistance exercise, which is more typical of athletic training programs.

The findings from studies on the effects of cryotherapy on recovery from eccentrically biased exercise are not directly applicable to traditional resistance exercise because eccentric exercise causes more severe muscle damage (27). Most of the studies described above assessed the effects of cryotherapy on recovery of muscle function after eccentric exercise by measuring maximal isometric strength from 24 h onward. A few studies have assessed recovery by measuring jump or squat performance (9, 23, 40, 45) or voluntary muscle activation during maximal muscle contractions (39). No research to date has evaluated the effects of cryotherapy on volitional work capacity during subsequent submaximal exercise on the same day. This information is important because recovery from exercise involves both peripheral and central factors (34), and athletes often need to recover quickly between training sessions on the same day.

The mechanisms by which cryotherapies such as cold water immersion can enhance postexercise recovery are not yet well established, particularly in the context of recovery after resistance exercise. Studies (31, 46) on endurance exercise have indicated that cold water immersion reduces blood flow to the muscle and limbs. The vasoactive factors that mediate these changes in blood flow in response to cold water immersion after exercise are unknown. Endothelin (ET)-1 is a potent vasoconstrictor that has been implicated in vascular responses to cold exposure (30, 36) and may therefore regulate the decrease in muscle and limb blood flow that others have reported after cold water immersion (31, 46). Cold water immersion reduces muscle temperature after endurance exercise (15, 31) and intermittent sprint exercise (4, 21), but it is uncertain at present how long this response persists during recovery from exercise. This information is important, because changes in muscle temperature could influence local blood flow and metabolism in muscle. Ihsan et al. (22) reported that cold water immersion after endurance exercise decreases muscle blood flow and metabolic activity. Changes in muscle O_2 saturation during exercise are accompanied by changes in venous blood O_2 saturation (7, 29). However, it is unknown whether changes

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in muscle O_2 saturation after cold water immersion are also associated with changes in venous blood O_2 saturation. This information is important because O_2 availability regulates motor unit activity in skeletal muscle (35), which might have implications for exercise performance.

Cold water immersion has been proposed to benefit recovery from exercise by reducing inflammation (3). However, research into the effects of cryotherapy on systemic markers of inflammation (e.g., cytokines and C-reactive protein) after eccentric exercise (16, 39, 42, 45) and more traditional resistance exercise (10, 12, 13, 23) has produced inconsistent findings. These studies generally collected blood samples immediately, 1 h, or 24 h after exercise and may therefore have overlooked peaks in the inflammatory response that occur between 1 and 24 h after exercise (38). Further research is needed to understand the effects of cold water immersion on inflammation during the first few hours after resistance exercise.

The primary aim of the present study was to compare the effects of cold water immersion versus active recovery on short-term restoration of maximal and submaximal muscle function after resistance exercise. A secondary aim of the present study was to extend existing knowledge of the physiological mechanisms by which cold water immersion could enhance recovery from exercise. To do this, we measured muscle temperature during and after cold water immersion. We also assessed muscle soreness and swelling and collected blood samples at regular intervals in the first few hours after cold water immersion to measure changes in venous blood O_2 saturation, ET-1 as a potential mediator of vasoconstriction, myoglobin as a marker of muscle damage, and lactate/pH and IL-6 as systemic markers of muscle metabolism and inflammation. We hypothesized that compared with active recovery, cold water immersion would 1) enhance recovery of maximal and submaximal muscle function; 2) increase serum ET-1 concentration; and 3) reduce muscle temperature, muscle soreness and swelling, venous blood O_2 saturation, and plasma myoglobin and IL-6 concentrations.

METHODS

Subjects. Ten physically active young men (means \pm SD, age: 21.3 ± 1.6 yr, height: 1.81 ± 0.06 m, and body mass: 84.7 ± 12.4 kg) volunteered to participate in this study. All participants were strength training 2–3 times/wk at the time of the study but were not accustomed to cold water immersion. The experimental procedures and potential risks were explained to the participants before they provided written informed consent. All participants were screened for contraindications to resistance exercise. This study was approved by the Human Research Ethics Committee of the University of Queensland.

Study design. Participants first completed two familiarization sessions to introduce them to the exercise protocols that would be used in the experimental trials followed by baseline testing to measure their strength. Two weeks after the familiarization sessions and baseline testing, participants completed two experimental trials separated by 14 days. They completed self-directed training in the 14 days preceding the baseline testing and completed a training record covering this period. Training was replicated for the period between the first and second experimental trial. The order of the experimental trials was randomized and counterbalanced. These trials involved a single, standardized bout of high-intensity resistance exercise followed by cold water immersion or active recovery. Participants refrained from consuming alcohol, caffeine, and tobacco over the 24 h preceding

both experimental trials. They also refrained from consuming any nutritional supplements between the baseline testing and completion of their second experimental trial. They completed a 24-h nutritional record before the first experimental trial, which was photocopied and returned to them to replicate for the 24 h before the second trial. They refrained from participating in any lower body exercise training for 48 h before each experimental trial.

Preexperimental sessions. Preexperimental sessions were completed 14 days before the first experimental trial and included a repetition maximum (RM) testing session, two familiarization sessions, and a baseline testing session. All preexperimental sessions and experimental trials were performed within the same temperature-controlled laboratory (temperature: $24.3 \pm 0.6^\circ\text{C}$ and humidity $48.6 \pm 1.2\%$). On the first day, RM testing and the first familiarization session were performed. Participants were tested to determine their RM for back squats, front squats, and incline leg presses. These RM data were used to set the workload for resistance exercise in the experimental trials. After 5–10 min of stationary cycling, participants performed a first repetition at an estimated 90% of RM for each exercise. If successful, participants repeated each exercise with gradually increasing resistance until failure or the loss of correct technique (as assessed by L. A. Roberts). A minimum of 3 min of recovery was allowed between attempts. A successful RM was recorded as the greatest mass lifted with correct form through a complete range of movement. Approximately 15–30 min later, participants started the first familiarization session. The familiarization session involved participants practicing the maximal muscle function tests that would be used during the experimental trials. On the second day, participants repeated the familiarization session from the first day before they practiced the submaximal muscle function test. More details of these tests are described below. On the third day, participants were tested to determine their baseline maximal and submaximal muscle function. After these baseline measures were recorded, participants were familiarized with the cold water immersion therapy to be used during the experimental trials.

Experimental trials. For each trial, the participants arrived at the laboratory at 8:30 AM, having eaten breakfast at 7 AM, and consumed 10 ml/kg water over the preceding 2 h. Resting venous blood samples were collected upon arrival. Maximal and submaximal muscle functions were not measured on the morning of each experimental trial due to time constraints and also to avoid any residual fatigue from the testing procedures that could affect performance during the subsequent resistance training session. Instead, maximal and submaximal muscle functions recorded during the third day of the preexperimental sessions were used to compare with changes in maximal and submaximal muscle functions measured during each trial (see details above).

After blood samples had been collected, resting superficial muscle temperature (1 cm deep) was recorded, and segmental limb volumes were calculated (as described below). Participants then completed a high-intensity resistance training session lasting ~ 1 h. This session consisted of the following: 6 sets of front and back squats at loads corresponding to 8, 8, 10, 12, 10, and 10 RM; 3 sets of 12 walking dumbbell lunges with a total mass corresponding to 40% of body mass; and 3 sets of 12 countermovement drop jumps from a height of 50 cm. Recovery time between sets was 90 s, with 120 s between exercises. Strong verbal encouragement was provided where required to maintain repetition tempo, form, and recovery periods. Participants drank water ad libitum during the training session. A venous blood sample was collected, and muscle temperature, upper leg circumferences, muscle soreness, and maximal muscle function were measured as soon as possible after the training session.

In the first 15 min after resistance exercise, participants changed clothes and moved to a different room, where they started a 10-min recovery intervention consisting of cold water immersion or active recovery. After participants completed these recovery interventions, they recovered for a period of 6 h. Maximal muscle function was

assessed again at 2 and 4 h, whereas submaximal muscle function was measured 6 h after completion of the recovery interventions.

During the 6-h recovery period (i.e., after cold water immersion or active recovery), participants remained in the laboratory (23–25°C) and were allowed to walk around at a low intensity. During the first 4 h of recovery, they were given standardized meals that provided 1.2 g·kg⁻¹ carbohydrate and 0.4 g·kg⁻¹ protein.

Cold water immersion and active recovery. For the cold water recovery intervention, participants sat in an inflatable bath (iBody, iCool Australia, Miami, QLD, Australia) containing water at 10 ± 0.3°C. Participants immersed their body up to their clavicle continuously for 10 min in water. We chose 10°C as a suitable water temperature because this has commonly been used in a previous cold water immersion study (2). Furthermore, recent studies have indicated that 10–15 min of immersion in water at 8–10°C is effective for reducing tissue temperature, blood flow, microvascular blood volume, and metabolic activity in skeletal muscle after exercise (4, 21, 22, 31). Water circulation and temperature in the bath were continuously maintained using a circulatory cooling unit (iCool Lite, iCool Australia).

For the active recovery intervention, participants exercised on a cycle ergometer (Wattbike, Wattbike, Nottingham, UK) for 10 min at a low, self-selected intensity. We selected active recovery as a control condition or comparison for cold water immersion instead of passive recovery because, in reality, it is unlikely that athletes would do no activity at all while recovering from prior exercise. Participants cycled a mean ± SD distance of 3.62 ± 0.25 km at an average power output of 45 ± 7 W.

Maximal muscle function tests. Participants completed four tests of maximal muscle function, including a countermovement jump, an unweighted squat jump, a weighted squat jump, and an isometric squat. They performed these tests in random order at 2 and 4 h after the recovery interventions.

Countermovement jump performance was measured while participants adopted a stance shoulder-width apart, holding a wooden bar in the conventional position for a back squat. On instruction, participants maximally flexed their knees before jumping vertically for maximum height. Their hands remained in contact with the wooden bar at all times while the wooden bar remained in contact with the upper back. They performed three jumps, and data from the best jump were analyzed. Participants performed unweighted and weighted squat jumps while holding a wooden bar (unweighted, ~500 g) or a barbell (Australian Barbell) loaded to 30% of RM (weighted, 37 ± 9.8 kg) in the same position. They performed three maximal jumps, and the data from the best jump were recorded. For both jumps, participants lowered their body to 90° knee flexion and paused for 2 s before jumping for maximum height.

Jump performance was measured using a portable force transducer (GymAware, Kinetic Performance Technology). Data were transmitted from the transducer to a hand-held personal digital assistant. For the countermovement jump and unweighted squat, data were calculated for jump height, work, mean velocity, and peak velocity. For the weighted squat, mean and peak power were also calculated. These calculations were done using native GymAware software (Kinetic Performance Technology). The coefficient of variation for these tests was established during the baseline testing procedures. It was 1.9% for countermovement jump height, 2.8% for unweighted squat jump height, and 2.9% for weighted squat jump height.

Isometric squat strength was measured using a modified back squat. Participants adopted a standard squat position on a Smith machine at one-third of maximum depth and pressed upward against a stationary bar (25, 44). Participants positioned themselves on a force platform (Kistler, Ostfildern, Germany) while ground reaction force data were collected at 1,000 Hz using native software (BioWare version 5.1.3). Participants were instructed to press upward against the bar as quickly and as forcefully as possible without any prior flexion of the knees, spine, or hips. They performed three efforts lasting 3 s each, and data

from the best effort were analyzed. Data were filtered with a Butterworth fourth-order digital low-pass filter with a cutoff frequency of 10 Hz before analysis. Isometric squat characteristics comprised peak vertical force and force development. The rate of force development was calculated over 30, 50, 100, and 200 ms. The coefficient of variation for maximum isometric strength was established as 0.9% during the baseline testing procedures.

Submaximal muscle function test. The submaximal muscle function test was similar to that previously described (25). This test was designed to simulate a second training session on the same day (i.e., after the initial resistance training session). It consisted of six sets of back squat repetitions performed at 80% of RM separated by 3 min of passive recovery. Participants attempted to complete 10 repetitions/set. If they were unable to complete 10 repetitions during any set, they stopped lifting and rested for 3 min before beginning the next set. Participants continued this sequence until they had attempted to complete six sets. Repetitions were performed using a squat rack (Force Fitness Equipment, Baltimore, MD), and the number of successful repetitions and mass lifted per set were recorded in addition to the total mass lifted over the six sets. The coefficient of variation for the total mass lifted during this high-intensity resistance exercise test was established as 0.7% during the baseline testing procedures.

Muscle temperature measurement. Muscle temperature was recorded before and after the training session, continuously throughout the recovery interventions, and over the following 2 h. It was measured by inserting an 18-gauge cannula into the vastus lateralis muscle to a depth of 1 cm. Thigh skinfold thickness was measured using Harpenden skinfold calipers (HSK BI, Baty, West Sussex, UK) and divided by two to determine subcutaneous fat thickness (19). A fine wire thermistor (T204E, Physitemp Instruments) was inserted through the cannula to the required depth and removed once temperature had stabilized (~5 s, pre- and postexercise). In a similar manner, a cannula was inserted before the recovery intervention and secured with medical tape and waterproof dressing. Data were logged at 1 Hz using a portable logger (SQ2020, Grant Instruments) and averaged over 1-min intervals for the recovery intervention and 5-min intervals for the first 2 h of recovery.

Blood collection and analysis. Venous blood samples were collected into vacutainers containing serum, EDTA, and lithium heparin. Serum vacutainers were left to clot at room temperature for 30 min before centrifugation. Vacutainers containing EDTA and lithium heparin were immediately put on ice and then centrifuged at 1,000 g at 4°C for 10 min. Plasma and serum were aliquotted and stored at -80°C for later analysis. Samples were collected before and after the initial resistance exercise and 15 min, 30 min, 45 min, 1 h, 1.5 h, 2 h, 4 h, and 6 h after the recovery intervention. A portion (100 µl) of fresh heparinized blood from the samples at all time points except 4 and 6 h after the recovery intervention was pipetted into CG4+ cartridges (Abbott Point of Care). These cartridges were then immediately inserted into a portable point-of-care device (iSTAT, Abbott Point of Care) to measure blood lactate concentration, pH, venous blood O₂ saturation, and venous blood CO₂ saturation.

Plasma myoglobin concentration was measured using an immunoassay (Roche Diagnostics) and an automated clinical analyzer (Cobas E411, Roche Diagnostics). Plasma IL-6 concentration was measured by ELISA (Quantikine HS ELISA, R&D Systems, Minneapolis, MN). Serum ET-1 concentration was also measured by ELISA (Quantikine ELISA, R&D Systems). Myoglobin, IL-6, and ET-1 were analyzed in duplicate, with sample means taken as the result. The intra-assay coefficient of variation was 1.7% for myoglobin, 9.3% for IL-6, and 6.0% for ET-1.

Limb volume assessment. Segmental limb volume for the lower and upper thigh of the right leg was calculated based on three circumferences. Anthropometric tape was used to measure the circumference 1) above the knee, 2) at midthigh, and 3) at the subgluteal fold. Positions were marked with a permanent marker for site identification during

and between trials. Limb volume between *circumferences* 1 and 2 (lower thigh) and between *circumferences* 2 and 3 (upper thigh) were calculated based on the formula proposed by Katch and Katch (15). The coefficient of variation for upper and lower limb volume assessment was 0.5 and 0.6%, respectively.

Muscle soreness perception. Leg muscle soreness was assessed under two conditions: 1) standing with feet shoulder-width apart and 2) squatting to a 90° knee angle, so that the quadriceps muscles were under tension. Perceived soreness was rated on a horizontal visual analog scale from 0 (no soreness) to 100 (maximal soreness).

Statistical analysis. Statistical analysis was conducted using the Statistical Package for Social Sciences program (version 19, IBM, New York, NY). With the exception of IL-6, all data were normally distributed (as confirmed using the Shapiro-Wilks test) and were analyzed using two-factor repeated-measures ANOVA. When significant trial and time \times trial interaction effects were evident ($P < 0.05$), paired *t*-tests were used to compare changes over time and between trials. IL-6 data were analyzed using Friedman's two-way ANOVA by

ranks and a Wilcoxon's signed rank test. The false discovery rate was used for multiple comparisons of time points within and between trials. Absolute values for intramuscular temperature, blood gases, pH, and ET-1 that were recorded preexercise, 15 min, 30 min, and 1 h postexercise were pooled. Cohen's effect size (*d*) was calculated to compare the magnitude of changes over time and differences between the trials and assessed as follows: 0.2 = small effect, 0.5 = moderate effect, and 0.8 = large effect. All data except IL-6 are presented as means \pm SD; IL-6 data are presented as means \pm interquartile range. Significance was set at a level of $P < 0.05$.

RESULTS

Maximal muscle function. Performance in all jumps decreased after the resistance exercise ($P < 0.05$) but then progressively increased from 2 to 4 h after resistance exercise in both cold water immersion and active recovery trials (Table 1). Countermovement and unweighted squat jump

Table 1. Changes in countermovement jump, unweighted squat jump, and weighted squat jump performance before, immediately after, and 2 and 4 h postexercise for CWI and ACT conditions

	Time			
	Before exercise	Immediately after exercise	2 h postexercise	4 h postexercise
<i>Countermovement jump</i>				
Jump height, cm				
CWI	33.7 \pm 6.4	26.7 \pm 8.2*	29.6 \pm 7.1*	30.3 \pm 6.5*
ACT		27.6 \pm 6.0*	30.6 \pm 6.9	30.6 \pm 6.3
Peak velocity, m/s				
CWI	3.4 \pm 0.5	3.1 \pm 0.5*	3.2 \pm 0.5	3.3 \pm 0.6
ACT		3.1 \pm 0.5*	3.3 \pm 0.3	3.3 \pm 0.4
Mean velocity, m/s				
CWI	2.0 \pm 0.4	1.7 \pm 0.2*	1.8 \pm 0.2	1.8 \pm 0.2
ACT		1.7 \pm 0.1*	1.8 \pm 0.2	1.8 \pm 0.2
Work, kJ				
CWI	39.6 \pm 8.7	32.1 \pm 8.9*	35.0 \pm 8.5	37.0 \pm 9.6
ACT		32.8 \pm 6.9*	36.5 \pm 7.6	35.3 \pm 8.5
<i>Unweighted squat jump</i>				
Jump height, cm				
CWI	31.3 \pm 6.7	24.9 \pm 7.4*	27.0 \pm 6.1*†	27.6 \pm 6.5*
ACT		23.7 \pm 4.7*	28.3 \pm 6.1*†	27.6 \pm 6.6*
Peak velocity, m/s				
CWI	3.1 \pm 0.4	2.8 \pm 0.3*	3.0 \pm 0.3	3.0 \pm 0.4
ACT		2.9 \pm 0.3*	3.0 \pm 0.3	3.1 \pm 0.4
Mean velocity, m/s				
CWI	1.6 \pm 0.2	1.5 \pm 0.2*	1.6 \pm 0.1	1.6 \pm 0.2
ACT		1.5 \pm 0.1*	1.6 \pm 0.1	1.6 \pm 0.2
Work, kJ				
CWI	35.7 \pm 8.5	28.9 \pm 8.0*	34.5 \pm 10.9	33.7 \pm 7.1
ACT		30.1 \pm 5.3*	34.3 \pm 8.3	34.6 \pm 6.4
<i>Weighted squat jump</i>				
Jump height, cm				
CWI	16.3 \pm 3.4	12.5 \pm 3.1*	13.7 \pm 3.1*	14.8 \pm 3.9
ACT		13.4 \pm 3.6*	14.6 \pm 3.5	14.6 \pm 3.4
Peak velocity, m/s				
CWI	2.4 \pm 0.2	2.1 \pm 0.2*	2.2 \pm 0.2*	2.2 \pm 0.2*
ACT		2.1 \pm 0.3*	2.1 \pm 0.2*	2.2 \pm 0.2*
Mean velocity, m/s				
CWI	1.2 \pm 0.1	1.0 \pm 0.1*	1.1 \pm 0.1*	1.1 \pm 0.1*
ACT		1.1 \pm 0.1*	1.1 \pm 0.1*	1.1 \pm 0.1*
Peak power, kW				
CWI	1.3 \pm 0.4	1.2 \pm 0.3*	1.2 \pm 0.3*	1.2 \pm 0.3*
ACT		1.1 \pm 0.4*	1.1 \pm 0.3*	1.2 \pm 0.3*
Mean power, W				
CWI	503 \pm 188	436 \pm 137*	464 \pm 156*	463 \pm 139*
ACT		443 \pm 117*	454 \pm 131*	453 \pm 136*

Data are means \pm SD. CWI, cold water immersion trial; ACT, active recovery trial. Baseline strength and power data were recorded during the third day of preexperimental sessions. See *Experimental trials* in METHODS for details. *Significant difference from preexercise ($P < 0.05$); †significant difference between trials ($P < 0.05$).

Table 2. Changes in peak force and RFD during isometric squats before, immediately after, and 2 and 4 h postexercise for CWI and ACT conditions

	Time			
	Before exercise	Immediately after exercise	2 h postexercise	4 h postexercise
Peak force, kN				
CWI	2.3 ± 0.6	2.1 ± 0.4*	2.2 ± 0.7	2.2 ± 0.6
ACT		2.1 ± 0.4*	2.3 ± 0.5	2.8 ± 0.68
RFD 0–30 ms, N/s				
CWI	972 ± 135	887 ± 151*	954 ± 243*	958 ± 199
ACT		881 ± 128*	1,026 ± 204*	1,007 ± 202
RFD of 0–50 ms, N/s				
CWI	1,015 ± 128	938 ± 147	992 ± 246	996 ± 212
ACT		1,043 ± 165	1,062 ± 211	1,037 ± 194
RFD 0–100 ms, N/s				
CWI	1,137 ± 151	1,088 ± 195	1,127 ± 259	1,074 ± 244
ACT		1,066 ± 207	1,156 ± 220	1,117 ± 180
RFD 0–200 ms, N/s				
CWI	1,349 ± 216	1,285 ± 279	1,378 ± 288	1,263 ± 346
ACT		1,218 ± 270	1,364 ± 296	1,209 ± 418

Data are means ± SD. RFD, rate of force development. Baseline strength and power data were recorded during the third familiarization session. See *Experimental trials* in METHODS for details. *Significant difference from preexercise ($P < 0.05$).

height remained below preexercise values ($P < 0.05$) at 2 and 4 h (approximately -3.5 cm or -11% for both countermovement and unweighted squat jump height). Performance in all jumps was generally similar after cold water immersion and active recovery. Similar trends existed for changes in peak velocity, mean velocity, work, peak power, and mean power during the recovery period for all jumps. Peak isometric strength followed a similar trend, decreasing postexercise ($P < 0.05$) and recovering toward preexercise values by 4 h (Table 2). There were no significant differences in the rate of force development between the trials at any time point (Table 2).

Submaximal muscle function. Submaximal muscle function was assessed at 6 h after the recovery interventions by measuring the average and total load that participants lifted during 6 sets of 10 squats at 80% of one RM. There were no significant differences in average or total load between cold water immersion and active recovery trials during the first three sets of the submaximal function test ($P > 0.05$; Fig. 1). However, the average load ($P = 0.025$, d : $+1.3$, $+38\%$) and total load ($P = 0.021$, d : $+0.7$, $+16\%$) that participants were able to lift during the final three sets was significantly greater after cold water immersion compared with active recovery. The average load lifted during the final three sets was not significantly different after cold water immersion compared with the average load recorded during the baseline testing ($P > 0.05$).

Muscle temperature. Muscle temperature increased consistently in all participants after the resistance exercise ($P < 0.05$). In contrast, the changes in muscle temperature during and after cold water immersion and active recovery were more variable. Individual responses are shown in Fig. 2, A and B. During cold water immersion, muscle temperature decreased by up to 12°C below postexercise values in some participants, whereas in others it only decreased by 1 – 2°C . During active recovery, muscle temperature increased by 1 – 2°C above postexercise values in some participants. In other participants, muscle temperature was 3 – 4°C below postexercise values at the start of active recovery and then only increased by $\sim 1^\circ\text{C}$. After active recovery, for some inexplicable reason, muscle

temperature rose unusually high (i.e., 41°C) in some participants. These participants were therefore excluded from further analysis of muscle temperature. Group data for five participants in the cold water immersion and active recovery trials are shown in Fig. 2C. In these participants, muscle temperature did not change significantly after cold water immersion, whereas it increased significantly during active recovery ($P < 0.05$) and remained elevated for 2 h. Muscle temperature was significantly higher ($P < 0.05$) between the 10th and 70th minute of the recovery period after active recovery compared with cold water immersion.

Blood gases, pH, and lactate. Blood lactate concentration was higher than preexercise values after the resistance exercise ($P < 0.05$). It returned to preexercise values within 15 min after active recovery, whereas it remained significantly higher than preexercise values for 2 h after cold water immersion ($P < 0.05$; Fig. 3A). Blood pH was lower than preexercise

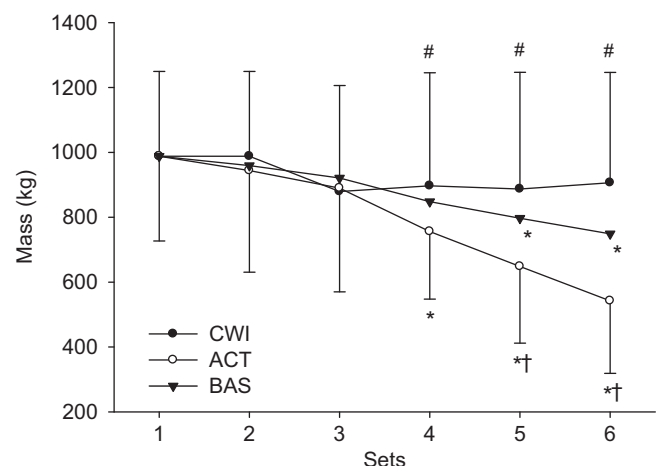


Fig. 1. Mean load lifted per set (means ± SD) over 6 sets for cold water immersion (CWI) and active recovery (ACT) conditions as well as the baseline trial conducted during the familiarization session (BAS). *Significant difference from set 1 ($P < 0.05$); #significant difference between CWI and ACT ($P < 0.05$); †significant difference between ACT and BAS trials ($P < 0.05$).

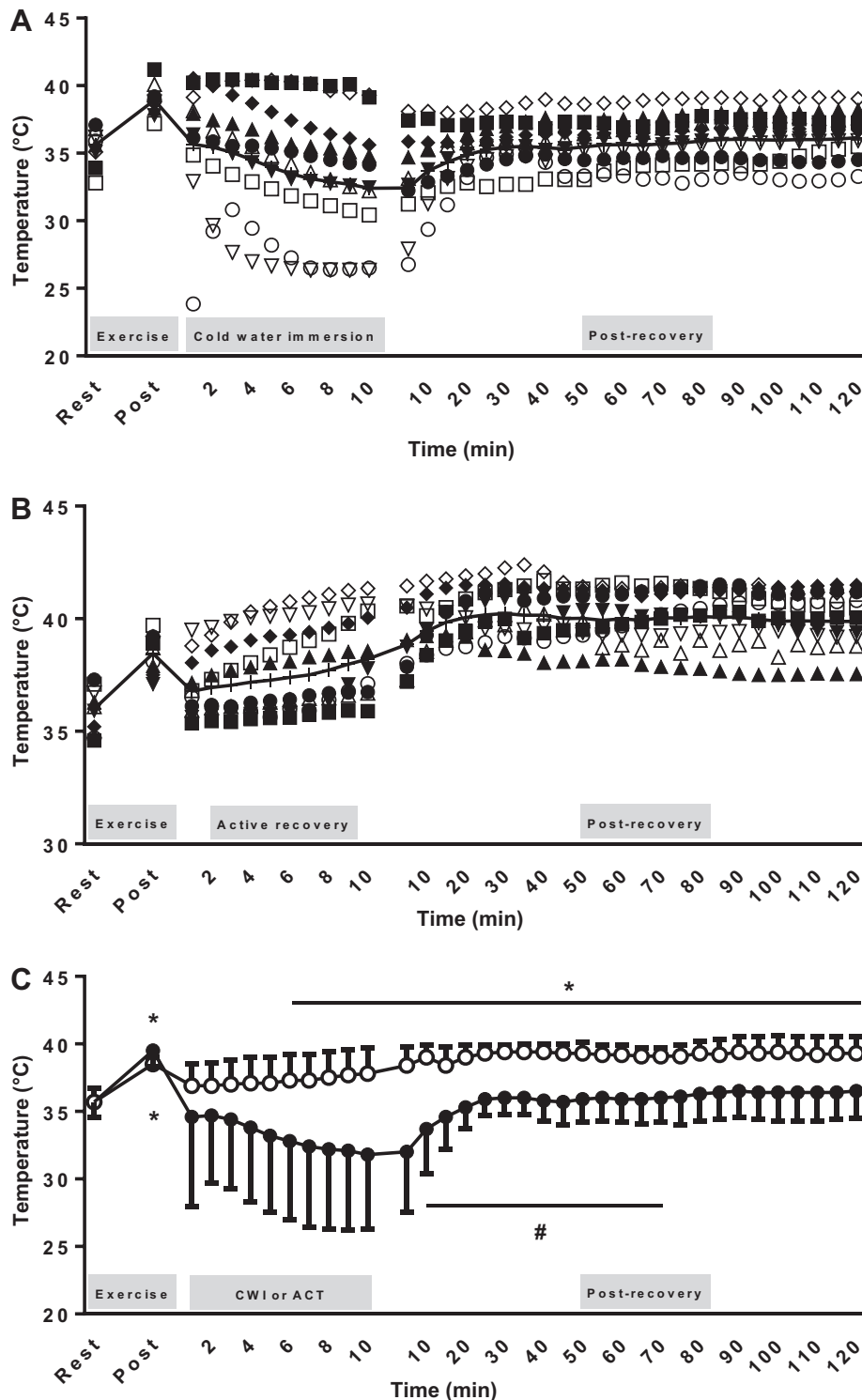


Fig. 2. Individual data for muscle temperature during the CWI trial (A) and ACT trial (B). Plus symbols and solid line represent the mean. C: mean \pm SD group data for $n = 5$ (see RESULTS for explanation). Post, immediately after exercise. *Significant difference from baseline ($P < 0.05$); #significant difference between trials ($P < 0.05$).

values after the resistance exercise ($P < 0.05$; Fig. 3B). It then increased above preexercise values after active recovery but not after cold water immersion. Between 15 min and 2 h after cold water immersion, it was lower compared with active recovery. Venous blood CO_2 saturation followed a similar trend to blood pH (Fig. 3D). Venous blood O_2 saturation did not change after the resistance exercise. However, it decreased below preexercise values after cold water immersion and

remained significantly lower compared with the active recovery trial for the next 2 h (Fig. 3C).

Muscle damage, swelling, and soreness. Plasma myoglobin concentration increased above preexercise values from 2 to 4 h after both cold water immersion and active recovery trials ($P < 0.05$; Fig. 4A). It was significantly lower after cold water immersion compared with active recovery at each time point ($P < 0.05$, d : -1.0 to -1.4 , -51% to -139%).

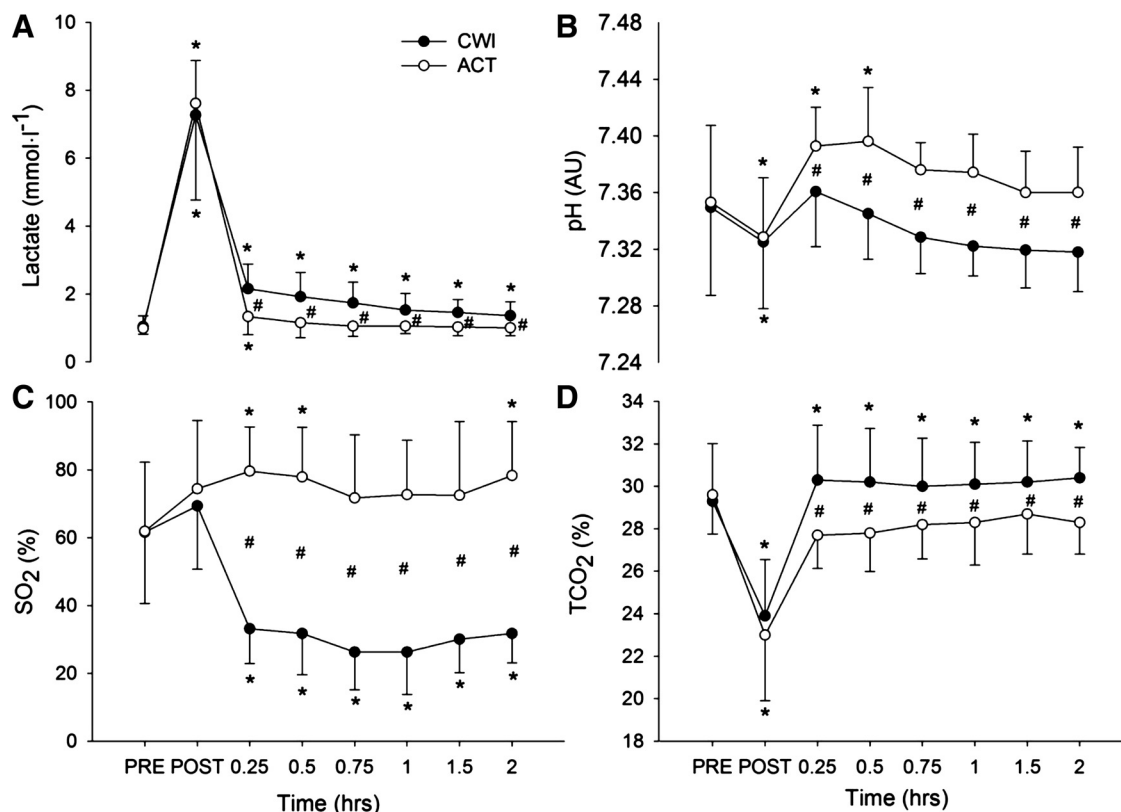


Fig. 3. Changes in blood lactate concentrations (A), blood pH (B), blood O₂ saturation (SO₂; C), and blood CO₂ saturation (TCO₂; D) before (Pre), immediately after (Post), and 0.25–6 h after exercise for CWI and ACT conditions. AU, arbitrary units. Data are expressed as means \pm SD. *Significant difference from preexercise ($P < 0.05$); #significant difference between trials ($P < 0.05$).

Lower and upper thigh volume changed over time ($P < 0.001$; Table 3). Both lower and upper thigh volume returned to preexercise values within 1 h after cold water immersion ($P > 0.05$) and remained stable over the remaining 5 h. In contrast, lower thigh volume remained above preexercise values after active recovery ($P < 0.05$). Upper thigh volume displayed a similar trend after active recovery. Lower thigh volume ($P < 0.05$, $d: -0.4, -2.2\%$, -0.1 cm) and upper thigh volume ($P < 0.05$, $d: -0.2, -2.7, -0.1$ cm) were significantly lower from 2 to 6 h after cold water immersion compared with active recovery.

Muscle soreness while standing upright and squatting at 90°C changed significantly over time ($P < 0.001$), increasing preexercise to postexercise, before gradually decreasing toward preexercise values over 6 h. Soreness while squatting was significantly lower after cold water immersion after 5 h ($P = 0.036$, $d: -0.6, -31\%$) and 6 h ($P = 0.011$, $d: -0.6, -37\%$).

ET-1 and IL-6. Serum ET-1 concentration decreased after the active recovery trial ($p < 0.05$) but did not change after the cold water immersion trial. It was below preexercise values at 0.25 h and 0.5 h after active recovery ($P < 0.05$). There were no differences in ET-1 between trials at any time point ($P > 0.05$; Fig. 4B).

Plasma IL-6 concentration was significantly higher than preexercise values from 0.25 to 2 h after both cold water immersion and active recovery trials ($P < 0.05$; Fig. 4C). It was significantly higher at 1.5 h ($P = 0.028$, $d: +1.5, +122\%$) and 2 h ($P = 0.038$, $d: +1.4, +98\%$) after cold water immersion compared with active recovery.

DISCUSSION

In the present study, we investigated how cold water immersion influences the recovery of maximal and submaximal muscle function after high-intensity resistance exercise. Contrary to our hypothesis, compared with active recovery, cold water immersion did not alter recovery of maximal strength or countermovement jump performance. However, it did enhance recovery of submaximal muscle function during a high-intensity resistance exercise test. Cold water immersion also substantially reduced muscle temperature, muscle soreness and swelling, venous O₂ saturation, and plasma myoglobin concentration compared with active recovery. Surprisingly, cold water immersion did not alter serum ET-1 concentration, whereas it induced a greater increase in plasma IL-6 concentration compared with active recovery. These findings add to the existing knowledge of the performance benefits and physiological effects of cold water immersion after exercise.

Contrary to our hypothesis, cold water immersion did not enhance recovery of maximal muscle function, as measured by jump performance and isometric strength. Pointon et al. (39) also reported no significant effects of applying ice packs on recovery of maximal isometric torque 2 h after eccentric exercise. In contrast, Vaile et al. (45) observed that cold water immersion enhanced recovery of peak force during isometric squats and peak power during jump squats 24 h after eccentric exercise. Fonda and Sarabon (9) also noted that 3 min of exposure to extremely cold air (-140 to -190°C) assisted recovery of muscle power during jump squats and counter-

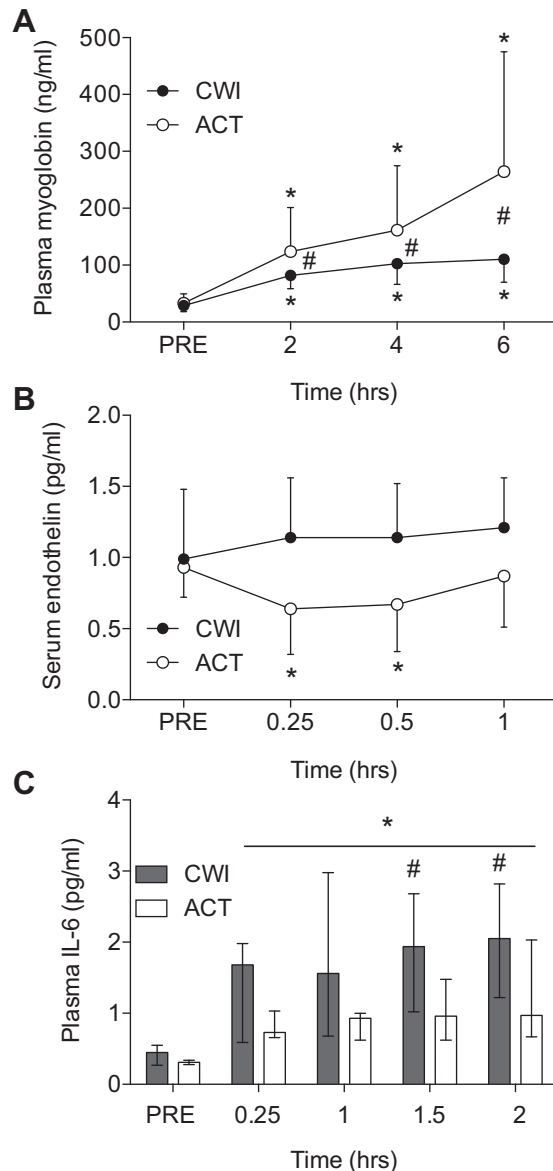


Fig. 4. Changes in plasma myoglobin concentration (A), serum endothelin-1 concentration (B), and plasma IL-6 concentration (C) before (Pre), immediately after (Post), and 0.25–6 h after exercise for CWI and ACT conditions. Data are expressed as means \pm SD except for IL-6, where data are medians \pm interquartile range. *Significant difference from preexercise ($P < 0.05$); #significant difference between trials ($P < 0.05$).

movement jumps 1 h after eccentric exercise. All other studies have failed to demonstrate any benefit of cryotherapy on recovery of maximal strength 24 h or more after eccentric exercise (6, 8, 16, 18, 20, 37, 40) and plyometrics (14, 19, 24). The results of the present study are not directly comparable with these other studies because the exercise was not exclusively eccentric in nature and participants were already familiar with resistance exercise. Consequently, the muscle damage after exercise was probably less severe in this study compared with the studies described above. Nevertheless, our findings suggest that cryotherapy generally does not influence any metabolic or neuromuscular factors that reduce maximal strength and/or power after exercise [e.g., impaired Ca^{2+} release from the sarcoplasmic reticulum (17)].

In support of our hypothesis, cold water immersion enhanced recovery of submaximal muscle function. Participants in the present study were able to lift a greater average and total load during the final three sets of the submaximal muscle function test after cold water immersion compared with active recovery. The obvious implication of this finding is that cold water immersion may assist athletes who sometimes need to train (or compete) twice within the same day. In contrast with our findings, two other studies (13, 23) discovered that cold water immersion did not enhance the total number of squats that participants could perform or the average power during each squat 24 and 48 h after resistance exercise. There are two obvious differences that may account for these conflicting findings. First, in these other studies (13, 23), participants only performed 4 sets of up to 10 squat exercises, whereas participants in our study performed 6 sets of up to 10 squat exercises. Second, in these other studies (13, 23), submaximal muscle function was tested at 24 and 48 h after exercise, whereas we tested submaximal muscle function 6 h after exercise. Therefore, the benefits of cold water immersion may depend on how and/or when submaximal muscle function is tested.

Cold water immersion elicited various physiological responses, some of which could explain the improvement in submaximal muscle function. We measured muscle temperature continuously during cold water immersion and for a further 2 h. Intramuscular temperature increased by $\sim 3\text{--}4^\circ\text{C}$ after resistance exercise. After cold water immersion, it was $\sim 4.0^\circ\text{C}$ below preexercise values and $\sim 7^\circ\text{C}$ below postexercise values. Because of substantial interindividual variations, these changes were not statistically significant. Muscle temperature then returned to preexercise values at 20–25 min after cold water immersion. The first 30 min after tissue injury is recognized as a potential window of opportunity to treat muscle injuries (32). A decrease in muscle temperature during this period can reduce secondary tissue damage (33). We did not assess secondary tissue damage directly. However, we found that cold water immersion significantly reduced plasma myoglobin concentration after exercise. This finding provides tentative evidence that cold water immersion may have minimized secondary tissue damage. This result contrasts with most other research indicating no significant effect (9, 13, 14, 16, 18, 19, 23, 24, 39, 40, 45) or an increase (12, 42) in plasma myoglobin concentration or creatine kinase activity in response to cryotherapy after eccentric or resistance exercise. Differences in the extent of muscle damage between resistance exercise and eccentric exercise, the timing of blood collection, or the timing of cryotherapy treatments after exercise could partially account for this disparity. Cold water immersion also reduced muscle soreness and swelling, which may have alleviated feelings of discomfort, thereby allowing participants to perform better during the last three sets of the submaximal exercise test.

Cold water immersion may also benefit recovery from exercise by inducing vasoconstriction and restricting the infiltration of inflammatory cells into muscle (28). We measured the serum concentration of ET-1 to determine whether this potent vasoconstrictor might explain previous observations that cold water immersion reduces blood flow to the limbs (46) and in skeletal muscle (22, 31). Contrary to our hypothesis, serum ET-1 concentration did not increase significantly after cold water immersion. This result was somewhat surprising, con-

Table 3. Changes in lower and upper thigh volume as well as muscle soreness while standing and squatting before, immediately after, and 0.5–6 h postexercise for CWI and active recovery conditions

	Time						
	Before exercise	Immediately after exercise	0.5 h postexercise	1 h postexercise	2 h postexercise	4 h postexercise	6 h postexercise
Lower thigh volume, liters							
CWI	3.1 ± 0.2	3.2 ± 0.3*	3.1 ± 0.3*	3.1 ± 0.3*	3.0 ± 0.2*†	3.0 ± 0.2*†	3.0 ± 0.3*†
ACT	3.1 ± 0.2	3.2 ± 0.2*	3.1 ± 0.2*	3.1 ± 0.2*	3.1 ± 0.2	3.1 ± 0.2	3.1 ± 0.2
Upper thigh volume, liters							
CWI	2.8 ± 0.5	2.9 ± 0.4*	2.9 ± 0.4*†	2.9 ± 0.5*	2.8 ± 0.4*†	2.8 ± 0.5*†	2.8 ± 0.4*†
ACT	2.8 ± 0.5	2.9 ± 0.5*	3.0 ± 0.5*	2.9 ± 0.5	2.9 ± 0.5	2.9 ± 0.5	2.9 ± 0.5*
Standing soreness, mm							
CWI	9.6 ± 8.6	73.5 ± 17.6*			26.1 ± 28.1*	25.0 ± 26.7*	23.5 ± 30.0*
ACT	10.0 ± 10.2*	68.1 ± 17.0*			30.8 ± 22.9*	30.8 ± 21.3*	30.4 ± 20.5*
Squatting soreness, mm							
CWI	3.8 ± 5.0	44.4 ± 20.4*			13.7 ± 16.0*	13.9 ± 16.5*†	12.2 ± 17.5*†
ACT	4.0 ± 6.5*	41.8 ± 19.9*			20.3 ± 13.2*	18.7 ± 11.4*	19.4 ± 14.1*

Data are means ± SD. *Significant difference from preexercise ($P < 0.05$); †significant difference between trials ($P < 0.05$).

sidering that other research has demonstrated that serum ET-1 concentration increases significantly immediately after immersion of only the hand in cold water ($\leq 13^{\circ}\text{C}$) (30, 36). Several factors could account for why serum ET-1 concentration did not increase in response to cold water immersion in the present study. Circulating ET-1 may have increased rapidly during cold water immersion and then returned to baseline levels < 15 min after cold water immersion when we collected a blood sample (36). Cold water immersion may also have increased the secretion of ET-1 in muscle tissue independently of any significant change in circulating ET-1 concentration (43). Alternatively, prior resistance exercise itself likely caused vasodilation in skeletal muscle (31), which may have attenuated ET-1 secretion into the circulation after cold water immersion. The decrease in serum ET-1 concentration that occurred after active recovery was probably due to the sustained vasodilation of skeletal muscle in response to the low-intensity cycling.

Several studies have investigated whether cryotherapy aids recovery by reducing inflammation. Tseng et al. (42) observed that topical ice treatment did not influence plasma cytokine concentrations 1 h after eccentric exercise, whereas it reduced plasma IL-6 and TNF- α concentrations 24 h postexercise. Most other studies have reported no effects of cold water immersion (13, 45), application of ice packs (39), or air-pulsed cryotherapy (-30°C) (16) on systemic inflammatory mediators after resistance exercise or eccentric exercise. In contrast with these findings, we observed that plasma IL-6 concentration was higher after cold water immersion compared with active recovery. IL-6 has traditionally been used as a marker of inflammation after exercise, yet it is not consistently associated with greater muscle damage after exercise (38). It is therefore difficult to suggest with confidence that cold water immersion enhanced systemic inflammation after exercise in the present study. Instead, the higher plasma IL-6 concentration may reflect sustained release of IL-6 from skeletal muscle in response to glycogenolysis (26), which increases in response to cold water immersion (41). The rise in plasma IL-6 concentration after cold water immersion in the present study was unexpected; we are therefore uncertain if or how this response might influence recovery from exercise.

The decrease in microvascular perfusion in skeletal muscle after cold water immersion is accompanied by a decline in local metabolic activity (22). To determine if changes in

muscle tissue oxygenation after cold water immersion alter systemic O_2 supply/demand, we measured changes in venous blood O_2 saturation. We observed that cold water immersion substantially reduced venous blood O_2 saturation to between 25% and 30%, whereas it raised venous blood CO_2 saturation. These effects persisted throughout the initial 2 h of recovery from exercise. The decrease in venous blood O_2 saturation that occurred after cold water immersion was similar in magnitude to the decrease that occurs at the onset of exercise (7). This decline in venous blood O_2 saturation therefore likely represents a genuine physiological response to cold water immersion. For example, it might reflect an increase in O_2 extraction in skeletal muscle after cold water immersion. Vascular occlusion reduces venous blood O_2 saturation (49) and increases motor unit activity and the discharge rate of high-threshold units in skeletal muscle (35). Furthermore, during muscle contractions with vascular occlusion, the integrated electromyogram correlates with muscle tissue oxygenation ($r = 0.562$, $P < 0.05$) (47). Thus, it is possible that by reducing O_2 availability, cold water immersion stimulated the recruitment of type II motor units, which could also possibly account for the improvement in performance during the last three sets of the submaximal exercise test.

Perspectives and Significance.

In designing the present study, we aimed to address some of the knowledge gaps and limitations of previous research in the broader field of cryotherapy. We did this through the following means: 1) using traditional resistance exercise as an exercise protocol to simulate common training practices of athletes, 2) measuring both maximal and submaximal muscle function during the early recovery period, 3) using active recovery as a control treatment, and 4) using a randomized cross-over design to minimize any potential series order effects and interindividual variations. We acknowledge some limitations to the present study. First, we only recorded muscle temperature superficially within skeletal muscle (i.e., 1 cm). The reasons for the substantial individual variation in changes in muscle temperature during and after recovery interventions are not immediately obvious. The intensity of active recovery and adipose tissue thickness at the site that we measured muscle temperature varied between individuals. However, muscle temperature

did not correlate with either exercise intensity or adipose tissue thickness. Second, we did not assess muscle blood flow or O₂ saturation within the muscle. Finally, we did not collect muscle tissue to examine in greater detail the local mechanism(s) by which cold water immersion may have enhanced recovery from exercise. Despite these limitations, our findings are strengthened by the nature of the research design. Our finding that cold water immersion allowed participants to perform more volitional work hints at some central benefits of cold water immersion. Whether cold water immersion provides more than a simple “placebo” effect remains a contentious issue (4, 5). “Central” perceptions of better recovery may play a more dominant role than “peripheral” physiological factors in the capacity for athletes to recover from exercise. If cold water immersion does allow athletes to undertake greater workloads during subsequent training sessions, then this may lead to better training adaptations. Alternatively, cold water immersion could also reduce training adaptations (11, 48) by attenuating some of the key biochemical and molecular processes that underpin local adaptations in skeletal muscle, including protein synthesis, mitochondrial biogenesis, and angiogenesis. Future studies in the field of cold water immersion should focus on addressing the central versus peripheral effects and acute versus chronic effects of cold water immersion.

ACKNOWLEDGMENTS

The authors thank the participants for time and efforts during this study as well as Vince Kelly and Gary Wilson for technical assistance during the study.

GRANTS

This work was supported by research grants from Sports Medicine Australia and the Centre of Excellence for Applied Sport Science Research at the Queensland Academy of Sport. L. Roberts is supported by an International Postgraduate Research Scholarship at The University of Queensland.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

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