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Cold-water or partial-body cryotherapy? Comparison of physiological responses and recovery following muscle damage

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The aim of this study is to compare (a) the physiological responses following cold-water immersion (CWI) and partial-body cryotherapy (PBC) and (b) the effects on recovery following a muscle-damaging protocol (5 × 20 drop jumps). Nineteen healthy males were randomly allocated into either a CWI (10°C for 10 minutes; n = 9) or a PBC (−60°C for 30 seconds, −135°C for 2 minutes; n = 10) group. The physiological variables (thigh muscle oxygen saturation [SmO_2], cutaneous vascular conductance [CVC], mean arterial pressure [MAP], and local skin temperature) were assessed immediately prior and up to 60 minutes post-treatment (10-minutes intervals). The recovery variables (thigh muscle swelling, maximum voluntary contraction [MVC] of the right knee extensors, vertical jump performance [VJP], and delayed onset of muscle soreness [DOMS]) were measured immediately prior and up to 72 hours post-treatment (24-hours intervals). Compared to PBC values, CVC (at 30 minutes), SmO_2 (at 40 minutes), and lower extremity skin temperature (thigh/shin at 60 minutes) were significantly reduced in the CWI group after the treatment (all $P < .05$). Only lower extremity skin temperature was significantly reduced in the PBC group directly post-treatment (all $P < .05$). MAP significantly increased in both groups after the treatments (both $P < .05$). DOMS did not differ between groups. MVC and VJP returned to baseline in both groups after 24 hours ($P > .05$). CWI had a greater impact on the physiological response compared to PBC. However, both treatments resulted in similar recovery profiles during a 72-hours follow-up period.

KEYWORDS
cardiovascular response, cryocabin, muscle damage, muscular recovery

1 | INTRODUCTION

Currently, post-exercise cooling is a widely accepted recovery modality and is believed to improve subjective (eg, ratings of muscle soreness) and objective (eg, measurements of muscle swelling, maximum voluntary contraction [MVC], and functional performance) recovery characteristics.[1,2] Physiological variables, such as muscle oxygen saturation (SmO_2), cutaneous vascular conductance (CVC), mean arterial pressure (MAP), and skin temperature, are used to explain the possible effects of cooling on subjective and objective recovery characteristics.[3-5] Various external post-exercise cooling modalities, including cold-water immersion (CWI), are commonly employed in the fields of sports, medicine, and physiotherapy. During CWI, water temperatures ranging from 5 to 13°C for 10-24 minutes decreased the symptoms of delayed onset of muscle soreness (DOMS) significantly, compared to the control conditions up to 48 hours of recovery.[2,6] Recently, relatively extreme forms

of external cooling, such as whole-body cryotherapy (WBC) and partial-body cryotherapy (PBC), have become commercially available. During WBC, individuals enter two or three closed chambers, where they are exposed to extreme cold air up to 4 minutes, in temperatures ranging from −10°C[7] to −130°C.[8] Similarly, during PBC, individuals, wearing only minimal clothing, are exposed to extreme cold vaporized liquid nitrogen, generating temperatures from −110°C[9] up to −195°C[10] for a very short duration (1-3 minutes). Although the use of these extreme cold treatments is becoming increasingly popular, the optimal cooling protocol required to initiate beneficial physiological responses is currently unknown.[3,4,11] Selfe et al[3] examined the physiological responses following a 1-, 2-, and 3-minute WBC exposure at −135°C and concluded that 2 minutes at −135°C is a safe protocol to induce physiological changes. These results are in line with those of Fonda et al[11] who reported that a PBC exposure of more than 2.5 minutes has no additional beneficial effects on the thermal response and that shorter protocols (1.5-2 minutes) would also have a positive impact on well-being of the users. It has been demonstrated that the thermal response after WBC is similar to the response after PBC.[11,12] Therefore, more research into the use of PBC could be of practical value as cryocabins are portable and less expensive than WBC systems.[12]

The initial reaction of the skin after a cold application is peripheral cutaneous vasoconstriction.[13] This sympathetic reaction facilitates an effect by reducing skin temperature,[14] skin blood flow,[15] and muscle oxygenation.[3] Bleakley et al[16] indicated that although the ability of water (heat-transfer coefficient of 0.58 k) to extract heat from the body is more efficient compared to air (heat-transfer coefficient of 0.024 k), the reduction in skin temperature between cold water (reduction between 6 and 9°C) and cold air (reduction between 3 and 19°C) seemed to vary slightly across the observed studies. Although the physiological and clinical effects of cold water and cold air on biomarkers of inflammatory, antioxidative capacity, and autonomic function during athletic recovery have also been shown to be comparable,[16] in the literature there is still no consensus about the effectiveness of these treatments on subcutaneous tissue temperature reduction (2-3 cm depth).[4,5,16] It has been demonstrated that both CWI and PBC can significantly improve objective recovery variables (jump performance and isometric peak force) and also with PBC, subjective recovery variables (DOMS), compared to passive control interventions after a muscle-damaging protocol.[17,18] Despite the widespread popularity of CWI and PBC in multiple fields, such as post-exercise recovery, to our knowledge only one study has directly compared the treatments as a method of recovery.[19] In this study, the effects of a single CWI (10°C for 10 minutes) on DOMS, isometric strength, jump performance, and creatine kinase were compared to a single PBC (−110°C for 3 minutes) exposure. However, this study failed to include any information on skin blood flow, muscle oxygenation, or tissue temperature, which would have provided further insight into the physiological mechanisms responsible for these findings.

Consequently, there is significant debate regarding the effectiveness of both modalities, and practitioners are unsure whether to employ a traditional CWI or a PBC treatment during athletic recovery. Adequate physiological insights are therefore required to inform the sports medicine community about the differences between these recovery treatments. Therefore, the aim of this study is to examine (a) the physiological effects and (b) the subjective and objective recovery characteristics after exposure to CWI (10°C for 10 minutes) and PBC (−60°C for 30 seconds, −135°C for 2 minutes) following exercise-induced muscle damage. It was hypothesized that, compared to PBC, CWI would have a greater effect on the physiological variables. We also hypothesized that CWI would lead to more favorable recovery characteristics.

2 | MATERIALS AND METHODS

2.1 | Participants

Using data from a study employing a similar methodological design,[17] the sample size was determined using G*Power (version 3.1.9.2; Franz Faul, University Kiel, Germany). The following design specifications were taken into account: $\alpha = 0.05$; power = 0.8; effect size = 0.4; statistical test = repeated measures ANOVA with within-between interaction. The sample size estimated according to these specifications was a minimum of 8 participants per group. Twenty male participants, regularly involved in moderate physical endurance (running, cycling) activity, volunteered for this study. The participants were randomly assigned (by drawing lots from a hat) either to the PBC group or the CWI group. However, one participant withdrew from the CWI group due to illness at the time of testing which was unrelated to this study. The remaining 19 participants (mean ± SD: age = 25.9 ± 4.4 years, height = 177.9 ± 0.09 cm, mass = 78.0 ± 12.0 kg) completed this study, and no adverse events were recorded. Participants were excluded from the study if they were smokers, had any allergy to cold (including Raynaud's disease), cardiovascular disease, cardiac pacemaker, and cardiac arrhythmia, were taking medication, or had any preexisting pain symptoms. All included participants were fully informed about the risks and discomforts related to this study before signing an informed consent form. This study was approved by the local ethical committee of Zurich (PB_2016-01125) in accordance with the Declaration of Helsinki (ICH-GCP), and the study is registered in the clinicaltrials.gov registry (NCT02847663).

2.2 | Experimental design

Using a parallel group design, as recommended by recent reviews in this area,[1,2] this study was carried out over 5

experimental days (08:00-12:00 hours). On Day 1, participants were screened for eligibility and were familiarized with the experimental setup (jump performance on the jump mat and MVC on the ergometer chair). On Day 2 (1 week after Day 1), participants were randomly assigned either to the PBC group or the CWI group (by drawing lots) before the baseline measurements were carried out. Environmental conditions were kept constant throughout the experimental procedure (ambient temperature 22 ± 2°C, relative room humidity 45 ± 5%). A schematic representation of the test protocol is presented in Figure 1.

Following randomization (Day 2), participants' anthropometric characteristics were determined. There were no significant differences for height, mass, body surface area, body fat percentage, body mass index, skinfold thickness (triceps brachii, biceps brachii, subscapular, iliac crest, supraspinal, abdominal, frontal thigh, medial calf and suprapatellar), or somatotype between both groups (all $P > .05$; Table 1).

Directly after all baseline measurements were recorded, the participants performed the muscle-damaging exercise. To evaluate the different physiological responses and the effect on muscle recovery between CWI and PBC, 10 minutes after the muscle-damaging exercise, the CWI group was immersed up to the sternal level in cold water (+10°C) for 10 minutes, while the PBC group entered a cryocabin (−60°C for 30 seconds, −135°C for 2 minutes) for 2.5 minutes. The physiological parameters, such as SmO_2, CVC, MAP, and skin temperature, were measured before the muscle-damaging exercise (pre) and immediately after the cooling treatments up to 60 minutes on Day 2 (at 10-minutes intervals) with the participant in supine position. Additionally, thermal sensation and thermal comfort were recorded. Indirect markers of muscle damage were assessed before the muscle-damaging exercise (pre), 60 minutes (on Day 2), 24 (Day 3), 48 (Day 4), and 72 hours (Day 5) after the cooling treatments, always in the same order: ratings of DOMS, anterior thigh muscle swelling, 2-leg vertical jump performance (VJP), and MVC of the right knee extensors. To minimize the potential effects of circadian rhythm, the participants returned to the laboratory at the same time of the day. The participants were also instructed to refrain from alcohol, supplements, or additional exercise during the experimental period.

2.3 | Exercise-induced muscle-damaging protocol

The muscle-damaging protocol comprised of 5 sets of 20 drop jumps from a 0.6-m box as described previously.[17] Between sets, participants had a 2-minutes break and were allowed to sit down. The encouraged participants were instructed (before the muscle-damaging exercise) and verbally remembered (during the muscle-damaging exercise) to flex their knees at least to 90° after the landing and to maintain their arms akimbo during the entire drop jump.

2.4 | Cryotherapy treatments

Directly after the muscle-damaging protocol, the cryotherapy treatment (PBC or CWI) was administered. During the PBC treatment, participants remained in an upright standing

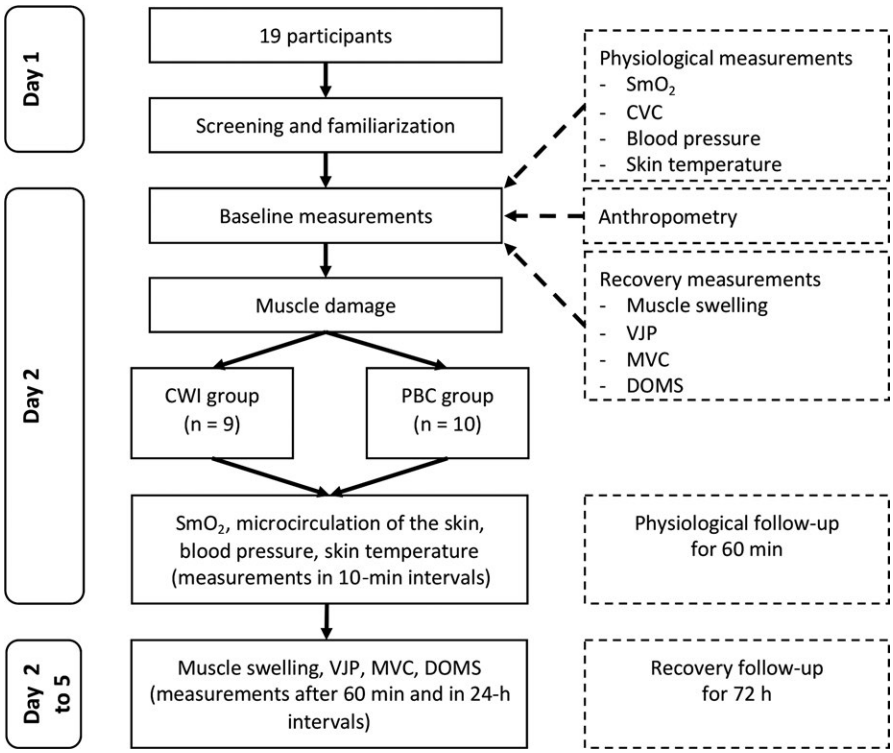

FIGURE 1 Schematic representation of the experimental protocol in function of time. SmO_2, muscle oxygen saturation; CVC, cutaneous vascular conductance; VJP, vertical jump performance; MVC, maximum voluntary contraction; DOMS, delayed onset of muscle soreness

TABLE 1 Anthropometric data of the participants

Parameters	CWI (n = 9)	PBC (n = 10)	*P*-value
Age (y)	26.0 ± 4.3	25.8 ± 4.5	.93
Height (cm)	175.0 ± 0.0	180.5 ± 0.0	.30
Mass (kg)	73.1 ± 8.2	82.3 ± 13.2	.15
BSA (m^2)	1.8 ± 0.1	2.0 ± 0.1	.06
BSA: mass ($m^2\ kg^{-1}$)	0.025 ± 0.001	0.024 ± 0.002	.23
Body fat %	17.2 ± 5.6	20.6 ± 7.5	.24
BMI	24.0 ± 3.2	25.3 ± 3.6	.39
∑ 9 SF (mm)	87.5 ± 38.9	92.0 ± 37.5	.83
Endomorphy	2.8 ± 1.2	2.9 ± 1.3	.96
Mesomorphy	5.0 ± 1.4	4.9 ± 1.5	.96
Ectomorphy	2.2 ± 1.6	2.0 ± 1.6	.83

BSA, body surface area; BMI, body mass index; SF, skinfold.
Values are means ± SD.

position and exposed to vaporized liquid nitrogen (Cryomed s.r.o., Cryosauna Space Cabin, Nové Zámky, Slowakia) for 2.5 minutes. PBC consisted of 30 seconds of pre-cooling at −60°C and then 2-minutes cooling at −135°C as used previously.[11,20] Participants entered the cryocabin in shorts and wearing cold-resistant woollen boots. During the procedure, the participants placed both hands on the upper edge of the cabin and were instructed to slowly move as prescribed in the user's manual.

During CWI, participants sat in a tank of stirred cold water (10 ± 0.5°C) for 10 minutes as recommended[6] and employed previously.[21] Participants wore only shorts and were immersed up to the level of the sternum. The temperature of the water was displayed on digital multimeter (Voltacraft MT52, Wollerau, Switzerland) and kept constant by adding ice. After this treatment, the immersed body parts were towel dried (patted dry) to minimize friction and participants were allowed to change into dry shorts after the immersion.[4,5]

After receiving the respective cryotherapy treatment, the participants' physiological responses were assessed in supine position for the 60-minutes follow-up period in 10-minutes intervals.

2.5 | Muscle oxygen saturation measurements

The SmO_2 was measured with the "Muscle Oxygen Monitor" (MOXY) system (Swinco, Zurich, Switzerland) working with near-infrared spectroscopy (NIRS). The indirect assessment of muscle metabolism using NIRS has been demonstrated to be a valid and reliable assessment tool.[22] The MOXY was taped (Hypafix, BSN, Hamburg, Germany) on the muscle belly of the vastus lateralis of the right quadriceps femoris muscle, as previously described.[3] The device had to be removed during the muscle-damaging exercise and the CWI and PBC treatment. An indicated mark midway between the proximal patella and the inguinal crease allowed for the MOXY to be re-taped in the same spot each time. The values from the MOXY were displayed (Peripedal v.2.4.8., Napoleon, IN, USA), saved on a laptop computer, and are presented normalized to baseline.

2.6 | Skin microcirculation measurements and CVC calculation

The microcirculation of the skin of the left frontal thigh was assessed with a laser speckle contrast imaging (LSCI) device (moorFLPI2; Moor instruments, Millwey, UK). The LSCI device allowed quantification of the average laser speckle imaging perfusion within an arbitrarily set region of interest (ROI) as described elsewhere.[23] The participants were informed to shave their left frontal thigh maximally 24 hours prior to the experiment to minimize biased results of the microcirculation due to the hair-covered skin. To obtain standardized and valid perfusion values, a 21-cm^2 ROI was defined on the left anterior thigh. The area extended from the anterior patellar base in the proximal direction. The ROI was clearly marked (Leukotape classic; BSN, Vibraye, France) to ensure analyses of the microcirculation on the same location during the entire experiment. Due to good temporal and spatial resolutions with a high frame rate, LSCI allowed measurements of acute changes in superficial skin blood flow, measured in arbitrary units (AU), over wide skin areas with very good inter-day reproducibility compared to traditional assessment technologies such as laser Doppler perfusion imaging (LDPI) and laser Doppler flowmetry (LDF).[24,25] CVC ($flux \cdot MAP^{-1}$, $flux \cdot mm\ Hg^{-1}$) is presented normalized to baseline.[4]

2.7 | Blood pressure measurements

Blood pressure was measured using an automated sphygmomanometer monitor (Microlife BP 3BTO-AP; Heerbrugg, Switzerland) from the left brachial artery. MAP (mm Hg) was calculated and is presented normalized to baseline.

2.8 | Skin temperature measurements

Skin temperature was measured using iButtons (Maxim Integrated, San Jose, CA, USA) using 5 thermochrons (model: DS1922L). In accordance with ISO 9886, the skin temperature was assessed on 4 standardized regions of the body to obtain the mean skin temperature of the body.[26] An additional thermistor was placed on the right anterior thigh, 2 cm above the MOXY device. All temperature loggers were taped (3M, Tegaderm, Saint Paul Minnesota, USA) to the neck (T_{neck}), the infraspinous fossa ($T_{scapula}$), the right dorsal hand (T_{hand}), the right mid-shin (T_{shin}), and the right frontal

thigh (T_{thigh}) and remained there during the whole experimental procedure. It has been demonstrated that the iButton system is a valid and reliable instrument for measuring skin temperature in humans.[27]

2.9 | Thermal sensation and thermal comfort ratings

Thermal sensation and comfort were assessed according to ISO 10551.[28] For the thermal sensation ratings, the participants were asked "How are you feeling now?" The participants had to rate their thermal sensation according to the following scale: 4 = very hot, 3 = hot, 2 = warm, 1 = slightly warm, 0 = neutral, −1 = slightly cool, −2 = cool, −3 = cold, −4 = very cold. For the thermal comfort ratings, the participants were asked "How do you perceive this?" The scale consisted of: 0 = comfortable, 1 = slightly uncomfortable, 2 = uncomfortable, 3 = very uncomfortable, 4 = extremely uncomfortable.

2.10 | Muscle swelling assessment

Muscle swelling of the right anterior thigh was assessed in B-Mode by ultrasound (MyLabClassC, Esaote, Genoa, Italy) in supine position after the physiological parameters were measured. The area of interest was defined at 60% of the distance between the greater trochanter to the lateral epicondyle and 3 cm lateral to the midline of the anterior thigh.[17] The ultrasound probe was placed on the water-soluble transmission gel without any compression on the skin. After these adjustments, 3 pictures were taken and the mean distance was analyzed (Pixmeo SARL, Osirix V.8.0.2., Bernex, Switzerland) on a laptop computer. Muscle swelling was defined as the distance from the muscle–bone interface to the subcutaneous adipose tissue–muscle interface.[29] Intraclass correlation coefficient for baseline test/retest reliability was 0.70.

2.11 | Vertical jump performance assessment

The VJP was assessed using standardized countermovement jumps as previously described.[30] The jumps were carried out on a jump mat (Just Jump; Probotics Inc., Huntsville, AL, USA). All participants had to perform 3 maximum jumps. The mean value was used to calculate the VJP for each day.[31]

2.12 | Maximum voluntary contraction assessment

The MVC of the right knee extensors was assessed on a custom-made ergometer chair (Cor 1 V.1.0., OT Bioelettronica, Torino, Italy). The measurements were carried out at a knee angle of 120° and a hip angle of 100° as previously described.[32] The participants were strapped into the ergometer chair with a seatbelt (Sparco, Irvine, CA, USA), and the right shin was attached with a strap around the malleoli lateralis and medialis to ensure a MVC. The participants were instructed to maximally extend their knee for the duration of 4 seconds. MVC was measured with a force meter (operating linearly in the range of 0-1000 N) 3 times in a row with a 2-minutes rest between the sets without any encouragement. The mean value of the 3 trials was taken to assess the MVC for each day. The signal was amplified (MISO II, OT Bioelettronica, Torino, Italy), and the maximum voluntary force was displayed on a monitor, not visible for the participants at any time throughout the experiment.

2.13 | Muscle soreness ratings

DOMS of participants' knee extensors was assessed using a visual analog scale (VAS), ranging from the far-left endpoint 0 (no soreness) to the far-right endpoint 10 (severe soreness) over a 10-cm span as previously described.[33] The participants were instructed to perform a squat at a 90° knee angle and maintain it for a duration of 3 seconds and then rate their level of DOMS.

2.14 | Data analysis

Normality was assessed using the Shapiro-Wilk test. Mauchly's test of sphericity was performed to test for homogeneity of differences in variance.

SmO_2, CVC, MAP, muscle swelling, MVC, and VJP were analyzed using normalized values (% mean ± SD). Repeated measures ANOVAs mixed design for treatment (CWI vs PBC) and time (baseline, 0, 10, 20, 30, 40, 50, 60 minutes) were conducted for SmO_2, CVC, MAP, and skin temperature. Repeated measures ANOVAs mixed design for treatment (CWI vs PBC) and time (baseline, 60 minutes, 24, 48, 72 hours) were conducted for MVC and VJP. Bonferroni-corrected post hoc analyses were used where appropriate.

As the anthropometric data were not normally distributed, Mann-Whitney *U* tests were performed to analyze anthropometric differences between the groups (CWI vs PBC). Because of weak power ($1-\beta = 0.17$), the Wilcoxon signed-rank test with Bonferroni correction was used to analyze within-group differences for muscle swelling. Given that DOMS was not normally distributed, the Mann-Whitney *U* (between CWI and PBC) and Friedman (within CWI or PBC) tests were conducted. Wilcoxon signed-rank test was used to analyze within-group differences for thermal comfort ratings (between baseline and immediately post-treatment [0 minute]). The Friedman test was used to analyze differences across time for thermal sensation. In case of significance, Bonferroni-corrected post hoc analyses were performed using Wilcoxon signed-rank tests.

The observed effect size was expressed as partial eta-squared ($\eta^2_{partial}$) values of 0.1-0.29, 0.3-0.49 and >0.5, which were considered small, medium, and large, respectively.[34] All statistical analyses were performed in SPSS (Statistical Package for the Social Sciences), version 23.0 (SPSS Inc, Chicago, IL, USA) with the level of significance set at $P < .05$. For the ease of interpretation, parametric as well as nonparametric data and not normally distributed data (eg, anthropometry) are expressed as means ± SD.

3 | RESULTS

3.1 | Physiological parameters

A significant treatment*time interaction ($F_{[7,11]} = 3.33$, $P = .03$, $\eta^2_{partial} = 0.68$) was observed for SmO_2. However, there was no treatment effect ($F_{[1,17]} = 3.05$, $P = .09$, $\eta^2_{partial} = 0.15$). A significantly greater reduction in SmO_2 was observed in CWI compared to PBC between 10 minutes (absolute value, normalized to baseline; PBC: 74.5 ± 6.7%, 98.5 ± 6.1%; CWI: 65.3 ± 7.6%, 88.7 ± 5.8%; $P < .01$) and 40 minutes (PBC: 74.3 ± 10.4%, 97.7 ± 6.1%; CWI: 64.7 ± 8.9, 88.4 ± 11.9%; $P = .04$) after exposure (Figure 2A).

There was a significant time ($F_{[7,11]} = 4.09$, $P < .01$, $\eta^2_{partial} = 0.72$) and treatment effect ($F_{[1,17]} = 12.04.$, $P < .01$, $\eta^2_{partial} = 0.41$) with no interaction ($F_{[7,11]} = 2.18$, $P = .11$, $\eta^2_{partial} = 0.58$) for CVC. Only CWI decreased CVC significantly 10 minutes after the treatment (CWI: 50.9 ± 11.6 AU, 70.5 ± 18.8%; $P = .04$) compared to baseline values (CWI: 76.4 ± 16.8 AU, Figure 2B).

A significant time ($F_{[7,11]} = 15.18$, $P < .01$, $\eta^2_{partial} = 0.90$) and treatment effect ($F_{[1,17]} = 13.83$, $P < .01$, $\eta^2_{partial} = 0.44$) with no interaction effect ($F_{[7,11]} = 1.77$, $P = .19$, $\eta^2_{partial} = 0.53$) was observed for MAP. A significant increase was observed in both groups only directly after (0 minute) the treatments (CWI: 104.6 ± 8.8 mm Hg; 110.7 ± 5.8%; $P = .01$; PBC: 103.8 ± 8.4 mm Hg; 105.7 ± 3.2%; $P < .01$) compared to baseline values (CWI: 96.5 ± 7.0 mm Hg, PBC: 98.3 ± 5.8 mm Hg, Figure 2C).

A significant treatment*time interaction ($F_{[7,11]} = 19.59$, $P < .01$, $\eta^2_{partial} = 0.92$) and treatment effect ($F_{[1,17]} = 3.91$, $P = 0.06$, $\eta^2_{partial} = 0.42$) was observed for mean skin temperature. The mean skin temperature was significantly lower in the PBC group only directly (0 minute) after the treatment compared to the CWI group (PBC: 24.6 ± 0.8°C; CWI: 26.6 ± 0.9°C; $P < .01$ between treatments). However, 10 minutes after the treatment the mean skin temperature was significantly lower in the CWI group compared to the PBC group (PBC: 32.0 ± 0.9°C; CWI: 30.5 ± 0.9°C; $P < .01$) and remained significantly lower up to 60 minutes (PBC: 34.3 ± 0.3°C; CWI: 33.2 ± 0.7°C; $P < .01$; Table 2).

For the thermal sensation ratings, a significant difference was observed for CWI ($\chi^2 = 22.16$, $P = .002$) but not for

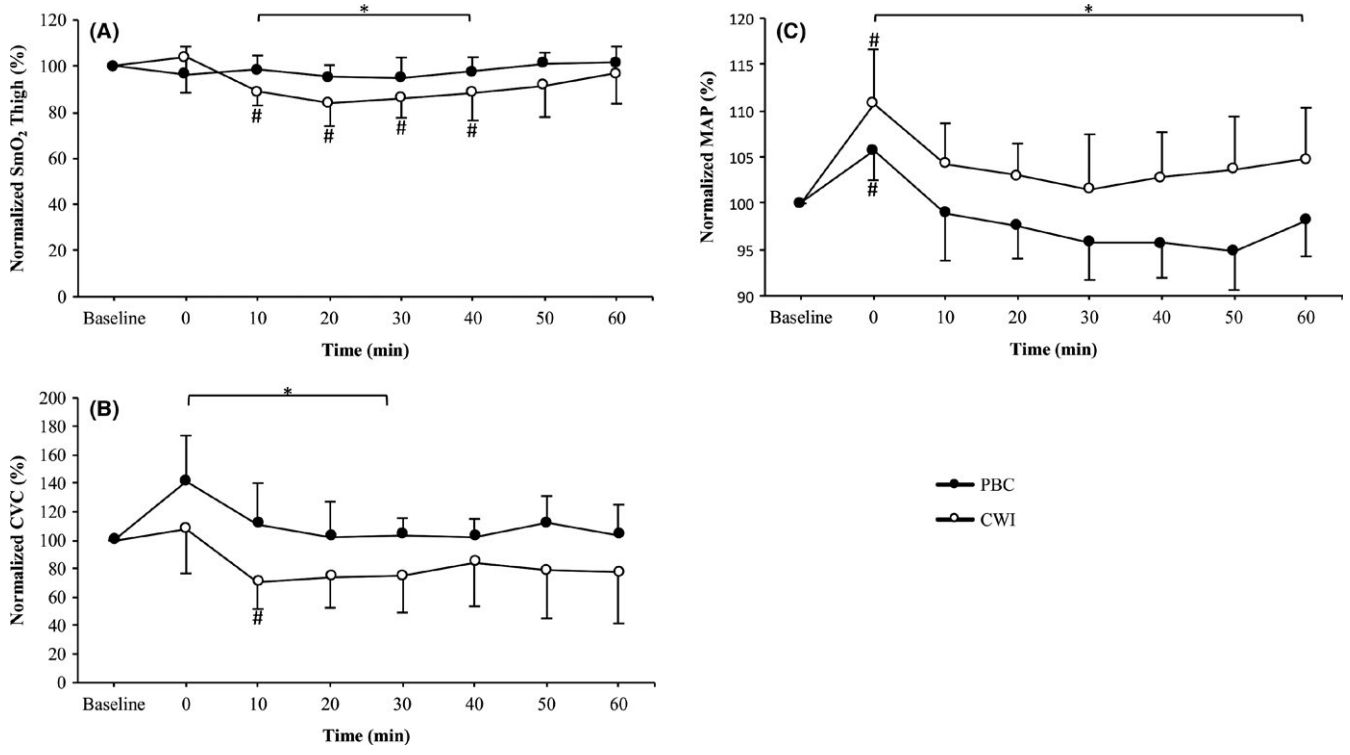

FIGURE 2 Measurements of (A) muscle oxygen saturation of the right vastus lateralis muscle (SmO_2), (B) cutaneous vascular conductance (CVC), (C) mean arterial pressure (MAP), in function of time. Values are normalized to baseline (% mean ± SD) with respect to their initial values. * $P < .05$ between CWI and PBC, # $P < .05$ within-group difference compared to baseline

PBC ($\chi^2 = 12.27$, $P = 0.09$). Compared to the PBC group, the participants in the CWI group reported to feel significantly ($P < .05$) colder compared to baseline ratings during the whole experimental procedure. No between or within differences were observed for thermal comfort (all $P > .05$).

3.2 | Recovery parameters

No significant differences between the groups were observed for anterior muscle swelling at baseline (CWI: 3.7 ± 0.6 cm, PBC: 3.9 ± 0.6 cm; $P > .05$). However, compared to baseline muscle, swelling was significantly increased only in the CWI group at 48 hours (CWI: 4.0 ± 0.4 cm, 107.5 ± 7.2%; $P = .01$; see Figure 3A).

VJP revealed a significant treatment*time interaction ($F_{[4,14]} = 6.32$, $P < .01$, $\eta^2_{partial} = 0.64$) but no treatment effect ($F_{[1,17]} = 0.91$, $P = .35$, $\eta^2_{partial} = 0.05$). The CWI group had lower performances at 60 minutes after the treatment (CWI: 40.6 ± 7.8 cm, 86.8 ± 8.0%; $P = .01$) compared to their baseline values (CWI: 46.5 ± 6.5 cm, Figure 3B).

A significant time ($F_{[4,14]} = 16.60$, $P < .01$, $\eta^2_{partial} = 0.82$) but no treatment ($F_{[1,17]} = 16.60$, $P = .32$, $\eta^2_{partial} = 0.05$) or interaction effect ($F_{[4,14]} = 1.92$, $P = 0.16$, $\eta^2_{partial} = 0.35$) was observed for MVC. MVC significantly decreased compared to baseline values (PBC: 729.9 ± 176.2 N, CWI: 633.8 ± 146.9 N) 60 minutes post-treatment in both groups (PBC: 554.7 ± 203.9 N, 72.2 ± 15.2%; $P < .01$; CWI: 475.8 ± 150.5 N, 74.6 ± 10.6%; $P < .01$). However, both groups recovered their MVC after 24 hours (PBC: 671.6 ± 209.1 N, 91.9 ± 16.2%; $P > .05$; CWI: 555.6 ± 164.8 N, 87.5 ± 13.8%; $P > .05$; Figure 3C).

DOMS peaked after 24 hours in the PBC group (PBC: 3.3 ± 3.0 VAS; $P = 0.007$) and after 48 hours in the CWI group (CWI: 3.4 ± 3.0 VAS; $P = .01$) compared to baseline values. Neither the PBC group ($\chi^2 = 25.2$; $P < .01$) nor the CWI group ($\chi^2 = 22.0$; $P < 0.01$) recovered from DOMS 72 hours after the damaging protocol. There was no significant difference for DOMS between the groups ($P > .05$; Figure 3D).

4 | DISCUSSION

The aim of this study is to examine (a) physiological effects and (b) the recovery characteristics after exposure to CWI (10°C for 10 minutes) and a PBC (−60°C for 30 sec, −135°C for 2 minutes) following exercise-induced muscle damage. The primary findings in this study are that (a) the

TABLE 2 Skin temperature measurements in °C

	Baseline	0 min	10 min	20 min	30 min	40 min	50 min	60 min
T_{mean}		*,#,†	*	*	*	*	*	*
CWI	32.9 ± 1.1	26.6 ± 0.9	30.5 ± 0.9	32.1 ± 0.8	32.7 ± 0.8	32.9 ± 0.8	33.1 ± 0.8	33.2 ± 0.7
PBC	32.3 ± 0.6	24.6 ± 0.8	32.0 ± 0.9	33.2 ± 0.7	33.8 ± 0.6	34.1 ± 0.4	34.3 ± 0.4	34.3 ± 0.3
T_{thigh}		*,#,†	*,#	*,#	*,#	*,#	*	*
CWI	32.1 ± 1.4	16.2 ± 1.6	26.5 ± 1.4	29.0 ± 1.4	29.8 ± 1.3	30.2 ± 1.2	30.5 ± 1.1	30.8 ± 1.1
PBC	31.5 ± 0.6	18.2 ± 1.8	30.7 ± 0.4	32.0 ± 0.4	32.4 ± 0.5	32.6 ± 0.6	32.6 ± 0.6	32.7 ± 0.7
T_{shin}		#,†	*,#	*,#	*,#	*,#	*,#	*,#
CWI	32.4 ± 1.3	14.8 ± 1.3	25.5 ± 1.1	27.3 ± 1.1	28.1 ± 1.1	28.6 ± 1.1	28.9 ± 1.2	29.2 ± 1.2
PBC	32.2 ± 0.7	15.3 ± 1.1	30.0 ± 0.9	30.8 ± 1.0	31.3 ± 0.8	31.6 ± 0.7	31.7 ± 0.7	31.7 ± 0.6
T_{neck}		*						
CWI	33.5 ± 0.8	31.7 ± 0.9	32.8 ± 0.9	34.3 ± 1.1	34.9 ± 1.1	35.0 ± 1.2	35.2 ± 1.4	35.3 ± 1.3
PBC	32.9 ± 0.6	29.4 ± 1.5	32.7 ± 1.0	33.9 ± 0.9	34.6 ± 0.8	35.1 ± 0.8	35.2 ± 0.7	35.4 ± 0.7
$T_{scapula}$		*						
CWI	33.6 ± 1.2	31.3 ± 2.6	33.7 ± 0.8	35.2 ± 0.3	36.0 ± 0.3	36.3 ± 0.2	36.5 ± 0.2	36.5 ± 0.2
PBC	32.9 ± 0.7	27.0 ± 1.8	33.7 ± 1.6	35.2 ± 1.0	35.7 ± 0.8	36.1 ± 0.5	36.2 ± 0.4	36.3 ± 0.3
T_{hand}		#			*	*	*	*
CWI	31.4 ± 2.3	29.8 ± 2.8	30.1 ± 3.0	30.7 ± 3.3	30.6 ± 3.1	30.8 ± 2.7	30.6 ± 2.5	30.6 ± 2.5
PBC	30.2 ± 2.0	27.8 ± 1.4	31.3 ± 1.7	32.8 ± 1.2	33.5 ± 1.0	33.5 ± 1.0	33.7 ± 0.9	33.5 ± 1.0

*$P < .05$ between CWI and PBC.

#$P < .05$ lower than baseline for CWI.

†$P < .05$ lower than baseline for PBC.

T_{mean}, mean skin temperature.

Values are means ± SD.

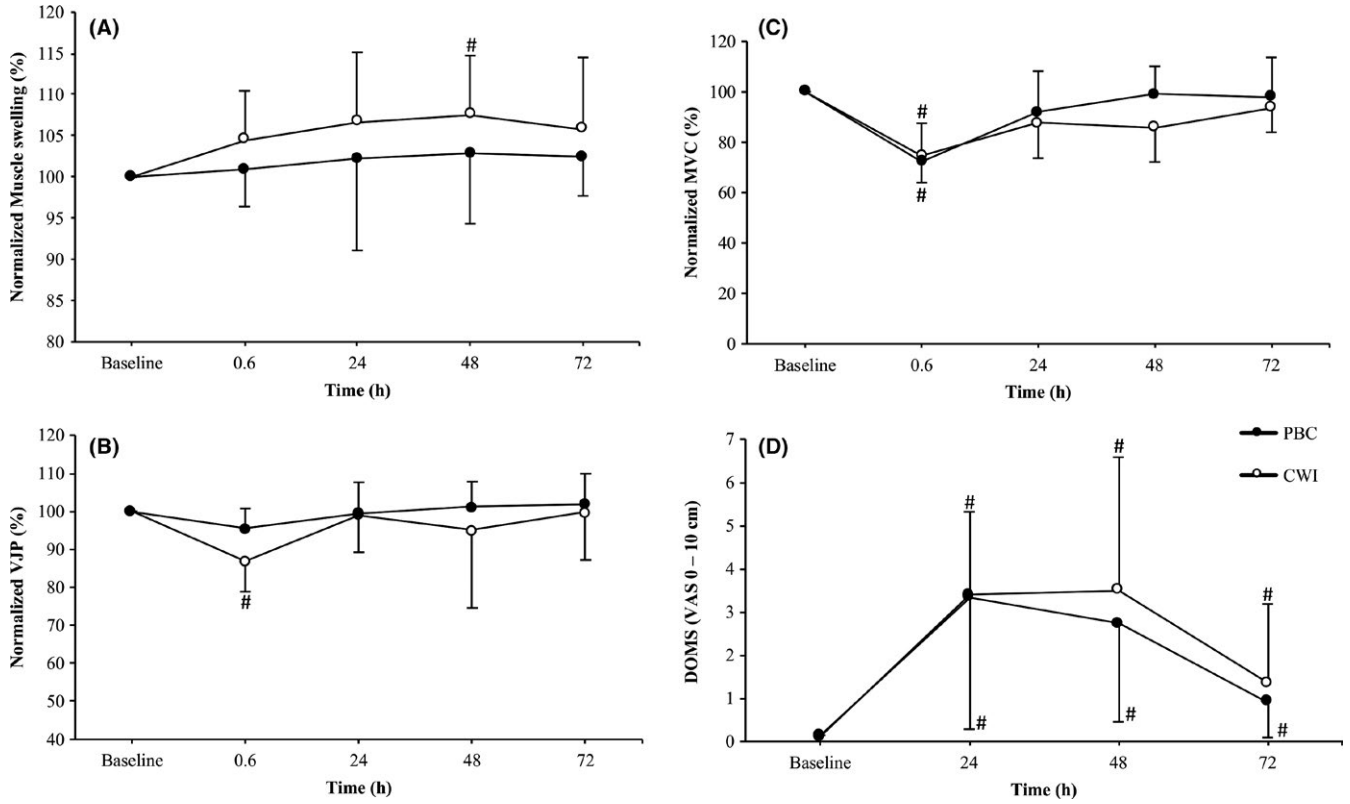

FIGURE 3 Measurements of (A) muscle swelling of the right anterior thigh, (B) vertical jump performance (VJP), (C) maximum voluntary contraction (MVC) of the right knee extensors, (D) delayed onset of muscle soreness (DOMS), in function of time. Values are normalized to baseline (a,b,c; % mean ± SD) with respect to their initial values. # $P < .05$ within-group difference compared to baseline

physiological impact of CWI was significantly greater than PBC and (b) no differences in objective and subjective recovery were observed between CWI and PBC up to 72 hours post-exercise.

Regarding the effects of CWI on SmO_2, our study is in line with previously published studies which have demonstrated that CWI (12-15°C for 10 minutes) decreases SmO_2.[35,36] In our study, a >10% decrease in SmO_2 was observed post-CWI (Figure 2A). However, PBC did not alter SmO_2. Costello et al[5] investigated muscle temperature in the vastus lateralis following CWI and WBC. The authors demonstrated that both interventions were successful in reducing muscle temperature up to 60 minutes post-treatment. More recently, Mawhinney et al[4] reported that muscle temperatures were significantly reduced only after CWI up to 40 minutes post-treatment compared with WBC. These results suggest that there is still no consensus regarding the effectiveness of WBC on deeper tissues. In contrast to the present experiment, these studies used a WBC treatment and not a PBC treatment. Different physiological responses between WBC and PBC might be explained by the fact that hands and the head (face) are not exposed during PBC. These body parts are known to have the highest density of adrenergic fibers, and thus, stimulating these sympathetic adrenergic fibers during a WBC treatment could explain the different physiological responses compared with a PBC treatment.[11] Similar to SmO_2, CVC was only reduced after the CWI in the current study. These results are in line with Mawhinney et al[4], where CWI (8°C for 10 minutes) reduced femoral artery blood flow and cutaneous blood flow more than WBC.

The short PBC exposure might have induced an elevation of the microcirculation of the skin directly after the treatment (41.3 ± 32.3% change increase from baseline). These results are in line with a published study that examined skin blood flow after ten WBC sessions using LDF.[37] This increase in microcirculation may have occurred due to the sudden cooling of the body, stimulating sympathetic vasoconstrictors, leading to a strong reactive hyperemia after the exposure.[38] The images of the LSCI device revealed that a strong reactive response was not observed immediately after the CWI treatment, indicating that the short and extreme cold exposure to the vaporized nitrogen itself may have triggered these primary neuronal-driven control mechanisms of skin blood flow.[13] However, it has to be taken into account that the participants moved during the entire PBC treatment and this may also elevate the skin microcirculation as previously observed after CWI treatments.[39] Further, this was the first study which employed LSCI to examine the effects of both CWI and PBC on a larger skin surface area, and this may also explain the findings. It is well established that fast changes in skin blood

flow can be measured accurately using full-field techniques, such as LSCI, compared to single-point measurement techniques such as LDF. This might be due to lower inter-site variability when using larger ROIs.[25]

The significant effect of CWI on both the CVC and the local skin temperature of the lower extremity can be explained by the way heat is extracted from the participants' body. Heat extraction of the human body is much more efficient with water compared to air (heat-transfer coefficient is 24.2 times higher in water compared to air).[16] Therefore, it is not surprising that the physiological impact after CWI was significantly greater than PBC. Although the participants' neck and scapula were inside the cabin, the temperatures did not change. The smallest reduction in skin temperature was observed at the surface of the right dorsal hand in the CWI and the PBC group, where the skin temperatures' decrease was $2 \pm 2°C$ in both groups. These results are in line with Savic et al[12] who concluded that the upper part of a cryocabin is warmer than the lower part and that the actual temperature in the cabin is substantially different from those reported by the manufacturer.

To our knowledge, only one study has directly compared CWI and PBC as a method of recovery following exercise-induced muscle damage.[19] MVC provides the best non-invasive measure of muscle damage as it provides the primary means for determining muscle function in human studies.[40] Although CWI was more effective in reducing the physiological parameters (SmO_2, CVC, and local skin temperature), muscle swelling approached statistical significance for increased values after 24 hours ($P = .06$) and reached significance after 48 hours ($P = .01$), while these values did not change in the PBC group (Figure 3A).

MVC significantly decreased compared to baseline in both groups only at 60 minutes ($P < .01$) after the treatment. The values for MVC showed a trend to decrease between 24 and 48 hours in the CWI group ($P = .2$, $P = .1$) but not in the PBC group ($P = 1.0$, $P = 1.0$; Figure 3C). However, the conservative Bonferroni corrections might be one reason for non-significant post hoc findings. Similar to MVC, VJP was only decreased in the CWI group after 60 minutes with values returning to baseline after 24 hours. Interestingly, both groups rated to have significant anterior muscle soreness throughout the entire experiment despite differences in muscle swelling.

This is the first experiment that has assessed both the physiological responses and recovery parameters after a muscle-damaging protocol followed by a CWI and a PBC treatment. Only one study has provided comparable data. Abaidia et al[19] indicated that PBC failed to accelerate countermovement jump performance compared with CWI during a 72-hours follow-up period. These results differ with the present findings of our study. However, both the muscle-damaging protocol (dynamometer vs. drop jumps) and the study design (crossover vs. parallel group) were different from this study, which make comparisons to the current data difficult. Other studies have demonstrated that both WBC and PBC can be effective in accelerating recovery from exercise compared to passive control interventions.[7,17]

A limitation of this study is that no control group was implemented. A passive control group would have provided additional insights into the efficiency of CWI and PBC compared to the control intervention. As participants moved during the PBC, it is likely that these movements impacted both SmO_2 and skin microcirculation. However, in the user's manual of the cryocabin, it is indicated that movement should be undertaken to help prevent cold injuries. Furthermore, although repositioning of the equipment was standardized following the CWI and the PBC treatment, we concede that this may have confounded the results.

In conclusion, the current study demonstrated that CWI decreases muscle oxygen saturation, CVC, mean skin temperature, and local skin temperature after a muscle-damaging exercise. However, although CWI had a greater physiological impact, no differences in objective and subjective markers of recovery were observed following CWI and PBC.

5 | PERSPECTIVES

Existing literature comparing PBC and CWI is limited.[1] The current findings will be of interest to sport science practitioners and medical personnel who are considering using either PBC or CWI interventions to improve short-term recovery after exhaustive exercise. Our study is the first one that directly compared the physiological responses and muscle recovery effects between CWI and PBC, demonstrating that the physiological impact of CWI is significantly greater than PBC. However, objective recovery and subjective recovery were not different following the 2 treatments. This study might help to further our knowledge of the physiological effects between CWI and PBC. These findings might also help inform practitioners' decisions when they have to choose between a CWI and a PBC treatment for their athletes. However, it is important that further studies include a control group which can be used to compare the effectiveness of both interventions to passive recovery.

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