Scand J Med Sci Sports 2014: ••: ••–••
doi: 10.1111/sms.12353

SCANDINAVIAN JOURNAL OF
MEDICINE & SCIENCE
IN SPORTS

One session of partial-body cryotherapy (−110 °C) improves muscle damage recovery

J. B. Ferreira-Junior[1,6], M. Bottaro[1], A. Vieira[1], A. F. Siqueira[1], C. A. Vieira[1], J. L. Q. Durigan[2], E. L. Cadore[1], L. G. M. Coelho[3], H. G. Simões[4], M. G. Bemben[5]

[1]College of Physical Education, University of Brasília, Brasilia, DF, Brazil, [2]Physical Therapy Division, University of Brasília, Brasilia, DF, Brazil, [3]Federal Center for Technological Education of Minas Gerais, Divinopolis, MG, Brazil, [4]Graduate Program on Physical Education, Catholic University of Brasilia, Brasilia, DF, Brazil, [5]Department of Health and Exercise Science, University of Oklahoma, Norman, Oklahoma, USA, [6]Federal Institute of Triangulo Mineiro, Paracatu, MG, Brazil
Corresponding author: João Batista Ferreira-Junior, PhD, Federal Institute of Triangulo Mineiro, Road MG 188, Km 167, 38600-00 Paracatu, MG, Brazil. Tel: 55 3836798200, Fax: 55 3836798200, E-mail: jbfjunior@gmail.com

Accepted for publication 3 October 2014

To evaluate the effects of a single session of partial-body cryotherapy (PBC) on muscle recovery, 26 young men performed a muscle-damaging protocol that consisted of five sets of 20 drop jumps with 2-min rest intervals between sets. After the exercise, the PBC group ($n = 13$) was exposed to 3 min of PBC at −110 °C, and the control group ($n = 13$) was exposed to 3 min at 21 °C. Anterior thigh muscle thickness, isometric peak torque, and muscle soreness of knee extensors were measured pre, post, 24, 48, 72, and 96 h following exercise. Peak torque did not return to baseline in control group ($P < 0.05$), whereas the PBC group recovered peak torques 96 h post exercise ($P > 0.05$). Peak torque was also higher after PBC at 72 and 96 h compared with control group ($P < 0.05$). Muscle thickness increased after 24 h in the control group ($P < 0.05$) and was significantly higher compared with the PBC group at 24 and 96 h ($P < 0.05$). Muscle soreness returned to baseline for the PBC group at 72 h compared with 96 h for controls. These results indicate that PBC after strenuous exercise may enhance recovery from muscle damage.

Muscular performance may be temporarily impaired by high-intensity exercise performed during a training session or competition. The reduction in muscle strength may be transitory, lasting minutes, hours, or several days following training or competition (Barnett, 2006). Longer lasting impairment in muscle strength accompanied by a decrease in range of motion, an increase in muscle proteins in the blood, inflammatory response, muscle thickness, and delayed onset muscle soreness is referred to as exercise-induced muscle damage (Clarkson & Hubal, 2002; Barnett, 2006; Paulsen et al., 2012).

Several modalities of recovery have been used to hasten the recovery period from muscle damage (Barnett, 2006). Among the most common treatment strategies used to restore muscle function are massage (Barnett, 2006; Nelson, 2013), stretching (Barnett, 2006; Herbert et al., 2011), nonsteroidal anti-inflammatory drugs (Schoenfeld, 2012), active recovery (Cheung et al., 2003; Barnett, 2006), compression garments (Hill et al., 2014), contrast water therapy (Bieuzen et al., 2013), and cryotherapy (Bleakley et al., 2012; Leeder et al., 2012), among others (Cheung et al., 2003; Barnett, 2006). A relatively novel modality of cryotherapy is the whole-body cryotherapy (WBC), which refers to a brief exposure (2 to 3 min) to extremely cold air (−110 to −195 °C) in a temperature-controlled chamber or cryocabin (Banfi et al., 2010; Hausswirth et al., 2011; Fonda & Sarabon, 2013). Sessions of partial-body cryotherapy (PBC), in which the head is not exposed to cold, have also been used as a similar modality of WBC (Hausswirth et al., 2013).

In sports medicine, WBC has been used recently as an approach to accelerate recovery from muscle damage, to improve between-training session recovery, and to prevent overtraining syndrome (Banfi et al., 2010; Bleakley et al., 2014). It has been reported that the inflammatory process and muscular enzymes related to muscle damage were reduced after five sessions of WBC (Banfi et al., 2009). Hausswirth et al. (2011) observed that three sessions of WBC hastened muscle damage recovery after downhill running. On the other hand, two sessions of WBC (2 h apart) performed 24 h after muscle-damaging protocol did not improve muscle recovery (Costello et al., 2012a). Additionally, according to Fonda and Sarabon (2013), five WBC exposures accelerated recovery of peak torque, squat jump start power, and decreased muscle soreness. However, biochemical markers, squat, and counter movement jump performance were not affected by WBC exposures (Fonda &

Sarabon, 2013). Therefore, the effects of WBC on muscle damage recovery are equivocal. These conflicting results may be due to methodological differences, such as cross-over vs between-subject design, number of WBC sessions (1 vs 3 vs 5) and the time elapsed between damaging exercise and WBC (immediately post or 24 h following WBC muscle-damaging protocol). There is also ambiguity regarding the optimal treatment protocol in terms of duration, temperature and sex (Fonda et al., 2014; Hammond et al., 2014; Selfe et al., 2014). To the best of our knowledge, there is no study that evaluated the effects of one session of PBC performed immediately after damaging exercise on muscle recovery. Using only one PBC exposure might reduce the cost and time associated with multiple treatments, which has currently been recommended by manufacturers. Thus, this topic requires further investigation.

We hypothesized that the physiological responses to cold exposure from PBC will improve muscle damage recovery. A logic model regarding the physiological, neuromuscular, and perceptual rationale for using PBC has been proposed by Costello et al. (2013). According to this model, PBC causes a vasoconstriction associated with decreased core and muscle temperature (Costello et al., 2012a, b). This vasoconstriction reduces blood vessels permeability to immune cells and decreases the inflammatory process (Hausswirth et al., 2011; Pournot et al., 2011; Ferreira-Junior et al., 2014). This attenuation in the acute inflammatory response could provide a beneficial role by protecting muscle from secondary muscle damage, which would result in a decrease in edema, muscle soreness, and an improvement in muscle strength (Hausswirth et al., 2011; Pournot et al., 2011; Ferreira-Junior et al., 2014). Ferreira-Junior et al. (2014) reported in a mechanistic version that the attenuation in serum sICAM-1 caused by PBC exposure immediately following EIMD may be responsible for the decreased secondary muscle damage. Additionally, hastening muscular recovery is especially important because athletes are usually required to train or compete at maximal intensity almost daily. Thus, the aim of this study was to evaluate the effects of one session of PBC performed immediately after muscle-damaging protocol on muscle recovery in physically active young men.

Methods

Subjects

Sample size for both PBC and control groups were determined by GPower (version 3.1.2; Franz Faul, Universitat Kiel, Germany) according to Beck (2013). The following design specifications were taken into account: $\alpha = 0.05$; $(1-\beta) = 0.8$; effect size $f = 0.25$; test family = F test, and statistical test = analysis of variance (ANOVA) repeated measures, within-between interaction. The sample size estimated according to these specifications was 20 subjects. Twenty-six young male university students (20.2 ± 2.5 years, weight 71.4 ± 9.1 kg and height 174.8 ± 7.3 cm) volunteered to participate. Inclusion criteria included physically active subjects involved in moderate physical activity (jogging, agility, or endurance) for an average of 3 days a week, not performing resistance training or plyometric exercise at least 3 months prior to the study. Subjects were informed of the purpose, procedures, possible discomforts, risks, and benefits of the study prior to signing the written informed consent form. They were considered healthy and fit for physical exercise by answering no to all Physical-Activity Readiness Questionnaire questions (Thomas et al., 1992). Also, based on Pobdielska et al. (2006), this study adopted the following exclusion criteria: untreated arterial hypertension, cardiovascular and respiratory diseases, angina, peripheral artery occlusive disease, venous thrombosis, urinary tract diseases, severe anemia, allergy to cold, tumor diseases, viral and bacterial infections, Raynaud's syndrome, claustrophobia, or convulsions. The present study was approved by the Institutional Ethics Committee of the Catholic University of Brasília (Protocol: 71484/2012).

Experimental design

Subjects were randomly placed, using a random number table, in two groups: PBC and control group (Table 1). Age, weight, height, skin folds (chest, thigh, and abdomen), peak torque, and muscle soreness were not significantly different between groups at baseline ($P > 0.05$); however, anterior thigh muscle thickness was higher in the PBC group when compared with the control group ($P = 0.02$; Table 1). Volunteers visited the laboratory on six occasions. The first visit consisted of a familiarization of experimental procedures and for anthropometric assessment. One week after familiarization, on visit two, volunteers performed a muscle-damaging protocol. In order to test the effects of a single session of PBC performed after damaging exercise on muscle recovery, the PBC group was exposed to 3 min of PBC at −110 °C 10 min after

Table 1. Physical characteristics and baseline peak torque and muscle soreness of the participants of each experimental group

Physical characteristics	Control group ($n = 13$)	PBC group ($n = 13$)	P-value
Age (years)	20.3 ± 2.2	20.2 ± 2.7	0.88
Weight (kg)	72.1 ± 9.9	70.6 ± 7.8	0.67
Height (cm)	176.0 ± 8.0	173.5 ± 5.9	0.38
$\text{Thigh}_{\text{skin fold}}$ (mm)	15 ± 9	13 ± 7	0.56
$\text{Chest}_{\text{skin fold}}$ (mm)	9 ± 5	8 ± 2	0.63
$\text{Abdomen}_{\text{skin fold}}$ (mm)	19 ± 11	16 ± 6	0.45
Anterior thigh muscle thickness (mm)	37.2 ± 4.6	41.2 ± 3.3*	0.02
Peak torque (N.m)	246.2 ± 61.4	261.8 ± 36.8	0.46
Muscle soreness (mm)	19 ± 14	15 ± 14	0.31

*$P < 0.05$, higher than control group.

PBC, partial-body cryotherapy.

completing the muscle-damaging protocol. The control group was not exposed to PBC 10 min after the damaging exercise. Indirect markers of muscle damage were evaluated before (pre), immediately post, 24, 48, 72, and 96 h following the damaging exercise by measuring always in the same order: anterior thigh muscle thickness, knee extensors isometric peak torque, and knee extensors muscle soreness. To avoid circadian influences, subjects were asked to visit the laboratory always at the same time of day. Volunteers were not allowed to perform any vigorous physical activities or unaccustomed exercise during the experiment period. They were also instructed not to take medications or supplements during the study.

Exercise-induced muscle damage protocol

The muscle-damaging protocol consisted of five sets of 20 drop jumps from a 0.6 m box with 2-min rest intervals between sets. After dropping down from the box and landing on the floor, subjects were instructed to perform a maximally explosive vertical jump upward and then land on the floor. Volunteers were also instructed to flex their knees to at least at 90° (0° full extension) during all landings, and to maintain their hands on their hips during the exercise. Additionally, they received verbal encouragement throughout the exercise. A similar muscle-damaging protocol has been used by other studies (Miyama & Nosaka, 2004; Howatson et al., 2009; Fonda & Sarabon, 2013).

Recovery modalities

During the PBC exposure (Fig. 1), subjects stood in a head out cryochamber based on gaseous nitrogen (Kryos Tecnologia, Brasília, Brazil) at −110 °C for 3 min. The temperature and duration of PBC exposure were based on a study by Costello et al. (2012a, b). Subjects wore bathing suits, gloves, socks, and shoes with thermic protection to protect their extremities. The control group performed a sham treatment control, during which subjects stood in the cryochamber for 3 min at 21 °C. Thigh temperatures (anterior central area) were measured by an infrared thermometer (Fluke, 566, China) before and immediately after PBC exposure and passive recovery. The measurement area was marked with a pen (Pilot 2 mm, Brazil) before each condition.

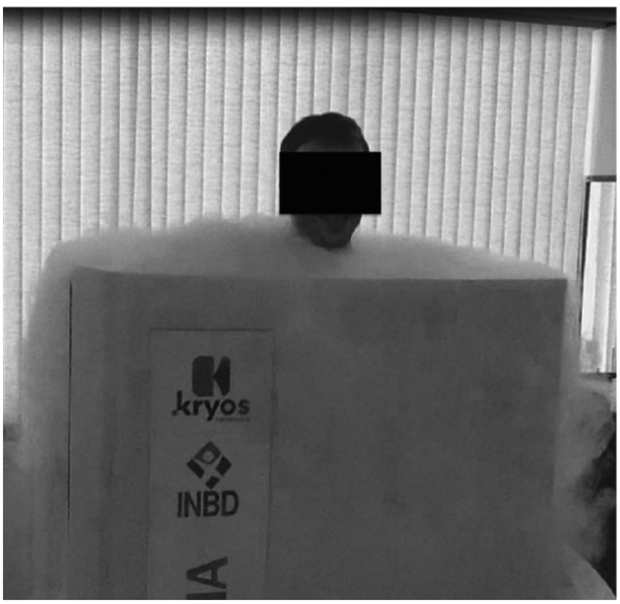

Fig. 1. Subject during head out/partial-body cryotherapy (PBC): 3 min at −110 °C.

Muscle thickness assessment

Muscle thickness of the anterior thigh was measured by ultrasonography using B-Mode ultrasound (Philips-VMI, Ultra Vision Flip, New York, NY, USA, model BF), and a single technician evaluated subjects. A water-soluble transmission gel was applied to the measurement site, and a 7.5-MHz ultrasound probe was placed on the skin, perpendicular to the tissue interface without depressing the skin. Subjects were evaluated in supine position, after resting for 10 min. Muscle thickness of the anterior thigh was measured at 60% of the distance from the greater trochanter to the lateral epicondyle and 3 cm lateral to the midline of the anterior thigh (Chilibeck et al., 2004). Once the technician was satisfied with the quality of the image, it was frozen on the monitor (Bemben, 2002). The images were then digitalized and later analyzed in Image-J software (National Institute of Health, Bethesda, MD, USA, version 1.37). Muscle thickness was defined as the distance from the subcutaneous adipose tissue–muscle interface to the muscle–bone interface (Abe et al., 2000). Additionally, baseline test/retest reliability coefficient (intraclass correlation coefficient; ICC) value for anterior thigh muscle thickness was 0.9.

Peak torque assessment

Maximal isometric peak torque of the right knee extensors was measured by the Biodex System 3 Isokinetic Dynamometer (Biodex Medical, Inc., Shirley, New York, USA). Subjects were positioned comfortably on the dynamometer seat with belts fastened across the trunk, pelvis, and thigh to minimize extraneous body movements, which could affect peak torque and power values. The lateral epicondyle of the femur was used to align the knee with the dynamometer's lever arm. With the participants positioned on the seat, the following measures were recorded: seat height, backrest inclination, dynamometer height, and lever arm length in order to standardize the test position for each participant. Gravity correction was obtained at full extension by measuring the torque exerted by the lever arm and the participant's relaxed leg. All isokinetic variables were automatically adjusted for gravity within the Biodex Advantage software. Calibration of the dynamometer was carried out according to manufacturer's specifications.

After having their right leg positioned by the dynamometer at an angle of 60° (0° represented the full extension), subjects were asked to cross their arms across the chest and to maximally contract their right knee extensors for 4 s. They had two attempts to achieve their maximal isometric peak torque. One minute of rest was given between each attempt. Subjects also received verbal encouragement throughout the test and all testing procedures were performed by the same examiner. All procedures were in accordance with Cadore et al. (2012). Moreover, warm-up was not conducted prior to isometric peak torque assessment because in a recent study, it was verified that there was no difference between five types of warm-up protocols and no warm-up protocol on isokinetic performance (Ferreira-Júnior et al., 2013). Baseline test/retest reliability coefficient (ICC) value for knee extensors isometric peak torque was 0.91.

Muscle soreness

Knee extensor muscle soreness was assessed using a 100-mm visual analog scale with "no soreness" (0 mm) and "severe soreness" (100 mm), respectively (Flores et al., 2011). Subjects rated their quadriceps soreness during maximal isometric contractions of the right knee extensors.

Statistical analyses

Data are presented as mean ± standard deviation. The Shapiro–Wilk test was used to check data for normal distribution. Peak torque and muscle thickness were analyzed using percent change from baseline. Considering that the peak torque and muscle thickness data were normally distributed, a two-way [group (PBC and control) × time (before, immediately, 24, 48, 72, and 96 h after damaging exercise)] repeated measures ANOVA was used to analyze peak torque and muscle thickness. In the case of significant differences, a Holm-Sidak post-hoc test was used. The physical characteristics and baseline peak torque values were evaluated by using an independent *t*-test. Given that muscle soreness data did not present a normal distribution, the nonparametric Mann-Whitney (between groups) and Friedman (within group) tests were used to analyze this variable. Significance level was set *a priori* at $P < 0.05$. Additionally, the effect size calculation was used to examine the magnitude of each condition effect. Cohen's ranges of 0.1, 0.25, and 0.4 were used to define small, medium, and large f values, respectively, obtained from the following formula (Cohen, 1988; Beck, 2013):

$$f = \sqrt{\frac{\sum_{j=1}^{k} (\mu_j - \mu)^2}{k\sigma_{\text{error}}^2}} \quad [1]$$

where μ_j is the population mean for an individual group, μ is the overall mean, k is the number of groups, and σ_{error} is the within-group standard deviation.

Results

Knee extensors peak torques are presented in Fig. 2. There was a significant group-by-time interaction for peak torque ($F = 2.3$, $P = 0.049$, power = 0.44, $f = 0.26$). Peak torque dropped immediately after damaging exercise with no difference between groups (32.0 ± 13.3% for control group and 28.6 ± 11.9% for PBC group, $P = 0.46$). The PBC group recovered peak torque 96 h after damaging exercise ($P > 0.05$) while the control group did not recover peak torque throughout the 96 h post-testing period ($P < 0.05$). Peak torque was also higher for the PBC group at 72 (PBC: 238.5 ± 50.4 N.m vs control group: 189.8 ± 52.6 N.m) and 96 h (255.8 ± 41.6 N.m vs 207.3 ± 56.9 N.m) when compared with the control group ($P < 0.05$).

There was a significant group-by-time interaction for anterior thigh muscle thickness ($F = 3.57$, $P = 0.005$, power = 0.78, $f = 0.37$). Muscle thickness increased at 24 h in the control group ($P < 0.001$), while it was not altered in the PBC group throughout the entire 96 h ($P > 0.05$). Moreover, muscle thickness was higher in control group at 24 and 96 h after damaging exercise when compared with the PBC group ($P < 0.05$; Fig. 3). The PBC group recovered from knee extensor muscle soreness at 72 h after damaging exercise ($\chi^2 = 24.53$, $P < 0.001$), while the control group recovered only at 96 h after damaging exercise ($\chi^2 = 29.36$, $P < 0.001$; Fig. 4). There was no difference in muscle soreness between groups ($P > 0.05$).

Discussion

The aim of this study was to evaluate the effects of one session of PBC (3 min at −110 °C) 10 min after damaging exercise on muscle recovery in physically active young men. The initial hypothesis was confirmed, as the PBC session resulted in a quicker recovery of muscle strength and relieved pain 72 h after damaging exercise with no alteration in muscle thickness. In contrast, the control group did not recover muscle strength to baseline values, and recovered muscle thickness to baseline values and from pain 48 and 96 h after damaging exercise, respectively. A possible reason for these results may

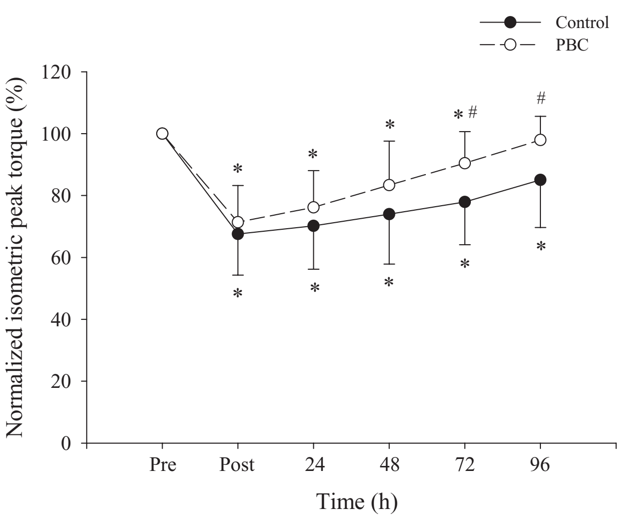

Fig. 2. Mean ± SD percent change from baseline in knee extensors isometric peak torque before (pre), immediately post, and 24–96 h following exercise-induced muscle damage. PBC, partial-body cryotherapy (−110 °C). *$P < 0.05$, lower than pre. #$P < 0.05$, higher than control group.

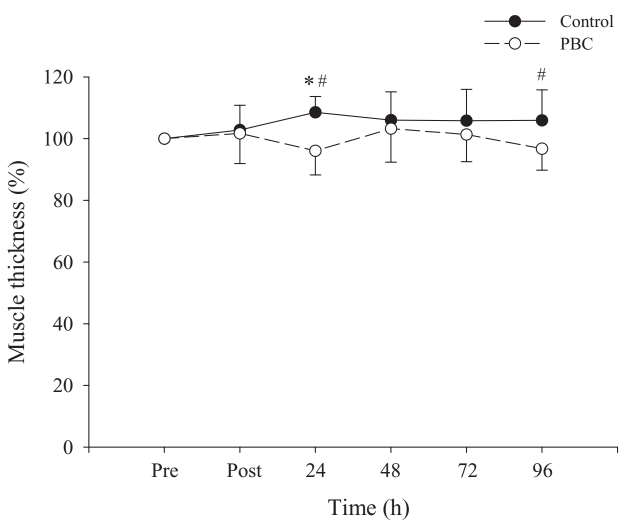

Fig. 3. Mean ± SD percent change from baseline in anterior thigh muscle thickness before (pre), immediately post, and 24–96 h following exercise-induced muscle damage. PBC, partial-body cryotherapy (−110 °C). *$P < 0.05$, higher than pre. #$P < 0.05$, higher than PBC group.

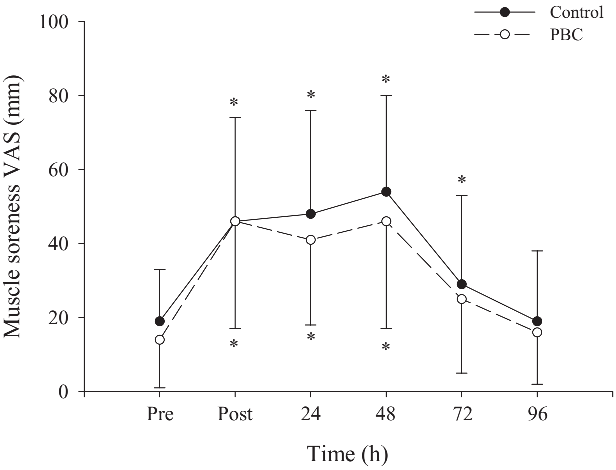

Fig. 4. Mean ± SD of knee extensors muscle soreness visual analog scale (VAS) before (pre), immediately post, and 24–96 h following exercise-induced muscle damage. PBC, partial-body cryotherapy (−110 °C). *$P < 0.05$, higher than pre- within group.

be related to a decrease in core, muscle, and skin temperatures after WBC exposure (Costello et al., 2012a, b). This thermal response may lead to increased vasoconstriction, which can cause a reduction in blood vessel permeability to immune cells and thus decrease the inflammatory process (Hausswirth et al., 2011; Pournot et al., 2011; Ferreira-Junior et al., 2014).

Regarding the effects of WBC on inflammatory process, it has been reported that five sessions of WBC (30 s at 60 °C and 2 min at −110 °C) in athletes decreased blood concentrations of muscular enzymes (creatine kinase and lactate dehydrogenase) and pro-inflammatory response (prostaglandin E2, adhesion molecule sICAM-I, interleukin IL-2 and IL-8; Banfi et al., 2009). Additionally, anti-inflammatory cytokine IL-10 was increased (Banfi et al., 2009). Moreover, Pournot et al. (2011) evaluated the effect of three sessions of WBC (3 min at −110 °C) after damaging exercise on the acute inflammatory response of well-trained runners. They observed an increase in IL-1ra and a decrease in IL-1β and C-reactive protein. Thus, according to the authors, WBC exposure decreased the inflammatory response via vasoconstriction at the muscular level caused by drop in muscle temperature. This hypothesis is supported by Costello et al. (2012a, b), who found a decrease of 1.6 ± 0.6 °C in the vastus lateralis temperature after WBC session. It was also reported that rectal temperature decreased 0.3 ± 0.2 °C 60 min after WBC session (Costello et al., 2012a, b). Although muscle temperature was not measured, skin thigh temperature in the present study dropped from 33.0 ± 0.9 °C to 15.7 ± 3.9 °C immediately after WBC exposure.

The results reported in the present study are similar to others that evaluated the effect of WBC on exercise-induced muscle damage recovery (Hausswirth et al., 2011; Fonda & Sarabon, 2013). A previous study verified that three sessions of WBC (3 min at −110 °C) after muscle-damaging protocol in well-trained runners improved muscle strength and perceived sensation, and also decreased muscle pain (Hausswirth et al., 2011). Additionally, five WBC exposures (3 min at −140 to −190 °C) accelerated recovery of peak torque, squat jump start power, and decreased muscle soreness in a different investigation (Fonda & Sarabon, 2013). Besides the number of WBC exposures, the current study differs from those cited above (Hausswirth et al., 2011; Fonda & Sarabon, 2013) in the experimental design used. The present study used a between-subject design, whereas the others (Hausswirth et al., 2011; Fonda & Sarabon, 2013) used a crossover design. According to Fonda and Sarabon (2013) and Ferreira-Junior et al. (2014), the major limitation of a crossover design to evaluate the effects of exercise-induced muscle damage is that it can be influenced by the repeated bout effect (Clarkson & Hubal, 2002; McHugh, 2003). Additionally, a between-subject design has been considered the gold standard when evaluating healthcare interventions (Schulz et al., 2010).

On the other hand, using a between-subject design, Costello et al. (2012a, b) reported that two sessions of WBC (20 s at −60 °C and 3 min at −110 °C) in healthy subjects did not hasten muscle strength nor decrease muscle soreness. The main difference between our study and Costello et al.'s (2012a, b) study was the timing of the WBC session. In the current study, the subjects were exposed 10 min after damaging exercise, while the sessions of WBC was applied 24 h after damaging exercise in the other study (Costello et al., 2012a, b). Immediately after damaging exercise, neutrophils and lymphocytes are mobilized to the injured tissue, and pro-inflammatory cytokines are produced in muscle by lymphocytes and monocytes (Clarkson & Hubal, 2002; Peake et al., 2005; Paulsen et al., 2012). Together, these substances cause an intramuscular degradation, which amplify the initial muscle damage (Clarkson & Hubal, 2002; Peake et al., 2005; Paulsen et al., 2012). Thus, it would make sense to suggest that WBC applied 24 h after damaging exercise did not avoid or decrease the secondary muscle damage caused by the acute inflammatory process.

As expected, the muscle-damaging protocol used in the present study caused significant muscle damage, observed through a reduction of 32.0 ± 13.3% in peak torque, an increase in muscle thickness of 8.5 ± 5.1%, and moderate muscle soreness in the control group. This muscle damage corroborates the findings from other studies that used drop jump exercise as a muscle-damaging protocol (Miyama & Nosaka, 2004; Howatson et al., 2009).

Methodological considerations

A major limitation of the present study was that core and quadriceps muscle temperature, muscular enzymes, and biochemical inflammatory markers were not measured. Future studies are necessary in order to understand the WBC effects on muscle inflammatory process caused by damaging exercise. The current study evaluated only physically active young men. WBC might be more accessible to athletes' population and muscle damage can also be less profound in this population (Barnett, 2006). In addition, anthropometric characteristics and sex seem to affect magnitude of skin cooling following WBC exposure (Hammond et al., 2014). Thus, further studies on these topics are necessary in order to verify if WBC can improve muscle recovery after high intense training or competition in other populations, such as athletes and women. Moreover, taking into account that there is ambiguity regarding the optimal treatment protocol in terms of number of sessions, duration, and temperature (Fonda et al., 2014; Selfe et al., 2014), future studies should evaluate the effects of these issues on exercise-induced muscle damage recovery.

Perspectives

The results of the present study showed that a single session of PBC (3 min at −110 °C) 10 min after muscle-damaging protocol enhanced muscle recovery in physically active young men. From a practical standpoint, PBC might be applied after an intense training session in order to improve muscle recovery. However, a question that should be investigated in future studies is if the same effect would be observed in athletes who used to experience less profound muscle damaging but more regularly use WBC. Further studies also need to evaluate biochemical inflammatory markers, tissue blood flow, and tissue temperature in order to understand the mechanistic effects of PBC. In addition, findings reported in this study can only be applied when PBC is administered 10 min after exercise-induced muscle damage.

Key words: Recovery modality, peak torque, muscle thickness, muscle soreness.

Acknowledgement

The study was partially funded by CAPES-Brazil.

References

Abe T, DeHoyos DV, Pollock ML, Garzarella L. Time course for strength and muscle thickness changes following upper and lower body resistance training in men and women. Eur J Appl Physiol 2000: 81: 174–180.

Banfi G, Lombardi G, Colombini A, Melegati G. Whole-body cryotherapy in athletes. Sports Med 2010: 40: 509–517.

Banfi G, Melegati G, Barassi A, Dogliotti G, d'Eril GM, Dugue B, Corsi MM. Effects of whole-body cryotherapy on serum mediators of inflammation and serum muscle enzymes in athletes. J Therm Biol 2009: 34: 55–59.

Barnett A. Using recovery modalities between training sessions in elite athletes – Does it help? Sports Med 2006: 36: 781–796.

Beck TW. The importance of a priori sample size estimation in strength and conditioning research. J Strength Cond Res 2013: 27: 2323–2337.

Bemben MG. Use of diagnostic ultrasound for assessing muscle size. J Strength Cond Res 2002: 16: 103–108.

Bieuzen F, Bleakley CM, Costello JT. Contrast water therapy and exercise induced muscle damage: a systematic review and meta-analysis. PLoS ONE 2013: 8: e62356.

Bleakley C, McDonough S, Gardner E, Baxter GD, Hopkins JT, Davison GW. Cold-water immersion (cryotherapy) for preventing and treating muscle soreness after exercise. Cochrane Database Syst Rev 2012: (2): CD008262.

Bleakley CM, Bieuzen F, Davison GW, Costello JT. Whole-body cryotherapy: empirical evidence and theoretical perspectives. Open Access J Sports Med 2014: 5: 25–36.

Cadore EL, Izquierdo M, Conceicao M, Radaelli R, Pinto RS, Baroni BM, Vaz MA, Alberton CL, Pinto SS, Cunha G, Bottaro M, Kruel LF. Echo intensity is associated with skeletal muscle power and cardiovascular performance in elderly men. Exp Gerontol 2012: 47: 473–478.

Cheung K, Hume PA, Maxwell L. Delayed onset muscle soreness – treatment strategies and performance factors. Sports Med 2003: 33: 145–164.

Chilibeck PD, Stride D, Farthing JP, Burke DG. Effect of creatine ingestion after exercise on muscle thickness in males and females. Med Sci Sports Exerc 2004: 36: 1781–1788.

Clarkson PM, Hubal MJ. Exercise-induced muscle damage in humans. Am J Phys Med Rehabil 2002: 81: S52–S69.

Cohen J. Statistical power analysis for the behavioral sciences. 2nd edn. Hillsdale, NJ: Lawrence Erlbaum Associates, 1988: 567.

Costello JT, Algar LA, Donnelly AE. Effects of whole-body cryotherapy (−110 degrees C) on proprioception and indices of muscle damage. Scand J Med Sci Sports 2012a: 22: 190–198.

Costello JT, Baker PRA, Minett GM, Bieuzen F, Stewart IB, Bleakley C. Whole-body cryotherapy (extreme cold air exposure) for preventing and treating muscle soreness after exercise in adults. Cochrane Database Syst Rev 2013: (10): CD010789.

Costello JT, Culligan K, Selfe J, Donnelly AE. Muscle, skin and core temperature after −110 degrees C cold air and 8 degrees C water treatment. PLoS ONE 2012b: 7 (11): e48190.

Ferreira-Junior JB, Bottaro M, Loenneke JP, Vieira A, Vieira CA, Bemben MG. Could whole-body cryotherapy (below −100 °C) improve muscle recovery from muscle damage? Front Physiol 2014: 5 (247): 1–4.

Ferreira-Júnior JB, Vieira CA, Soares SRS, Magalhães IEJ, Rocha-Júnior VA, Vieira A, Bottaro M. Effects of different isokinetic knee extension warm-up protocols on muscle performance. J Sports Med Phys Fitness 2013: 53 (Suppl. 1 to No. 3): 25–29.

Flores DF, Gentil P, Brown LE, Pinto RS, Carregaro RL, Bottaro M. Dissociated time course of recovery between genders after resistance exercise. J Strength Cond Res 2011: 25: 3039–3044.

Fonda B, De Nardi M, Sarabon N. Effects of whole-body cryotherapy duration on thermal and cardio-vascular response. J Therm Biol 2014: 42: 52–55.
Fonda B, Sarabon N. Effects of whole-body cryotherapy on recovery after hamstring damaging exercise: a crossover study. Scand J Med Sci Sports 2013: 23: E270–E278.
Hammond LE, Cuttell S, Nunley P, Meyler J. Anthropometric characteristics and sex influence magnitude of skin cooling following exposure to whole body cryotherapy. Biomed Res Int 2014: 2014: 1–7.
Hausswirth C, Louis J, Bieuzen F, Pournot H, Fournier J, Filliard JR, Brisswalter J. Effects of whole-body cryotherapy vs far-infrared vs passive modalities on recovery from exercise-induced muscle damage in highly-trained runners. PLoS ONE 2011: 6 (12): e27749.
Hausswirth C, Schaal K, Le Meur Y, Bieuzen F, Filliard JR, Volondat M, Louis J. Parasympathetic activity and blood catecholamine responses following a single partial-body cryostimulation and a whole-body cryostimulation. PLoS ONE 2013: 8 (8): e72658.
Herbert RD, de Noronha M, Kamper SJ. Stretching to prevent or reduce muscle soreness after exercise. Cochrane Database Syst Rev 2011: (7): CD004577.
Hill J, Howatson G, van Someren K, Leeder J, Pedlar C. Compression garments and recovery from exercise-induced muscle damage: a meta-analysis. Br J Sports Med 2014: 48 (18): 1340–1346.
Howatson G, Goodall S, van Someren KA. The influence of cold water immersions on adaptation following a single bout of damaging exercise. Eur J Appl Physiol 2009: 105: 615–621.
Leeder J, Gissane C, van Someren K, Gregson W, Howatson G. Cold water immersion and recovery from strenuous exercise: a meta-analysis. Br J Sports Med 2012: 46: 233–240.
McHugh MP. Recent advances in the understanding of the repeated bout effect: the protective effect against muscle damage from a single bout of eccentric exercise. Scand J Med Sci Sports 2003: 13: 88–97.
Miyama M, Nosaka K. Influence of surface on muscle damage and soreness induced by consecutive drop jumps. J Strength Cond Res 2004: 18: 206–211.
Nelson N. Delayed onset muscle soreness: is massage effective? J Bodyw Mov Ther 2013: 17: 475–482.
Paulsen G, Mikkelsen UR, Raastad T, Peake JM. Leucocytes, cytokines and satellite cells: what role do they play in muscle damage and regeneration following eccentric exercise? Exerc Immunol Rev 2012: 18: 42–97.
Peake J, Nosaka K, Suzuki K. Characterization of inflammatory responses to eccentric exercise in humans. Exerc Immunol Rev 2005: 11: 64–85.
Pobdielska H, Strek K, Bialy D. Whole body cryotherapy. Wroclaw: Kriotechnika Medyczna, 2006: 110.
Pournot H, Bieuzen F, Louis J, Fillard JR, Barbiche E, Hausswirth C. Time-course of changes in inflammatory response after whole-body cryotherapy multi exposures following severe exercise. PLoS ONE 2011: 6: e22748.
Schoenfeld BJ. The use of nonsteroidal anti-inflammatory drugs for exercise-induced muscle damage: implications for skeletal muscle development. Sports Med 2012: 42: 1017–1028.
Schulz KF, Altman DG, Moher D, CONSORT Group. CONSORT 2010 statement: updated guidelines for reporting parallel group randomised trials. BMJ 2010: 340: c332.
Selfe J, Alexander J, Costello JT, May K, Garratt N, Atkins S, Dillon S, Hurst H, Davison M, Przybyla D, Coley A, Bitcon M, Littler G, Richards J. The effect of three different (−135 degrees C) whole body cryotherapy exposure durations on elite rugby league players. PLoS ONE 2014: 9: e86420.
Thomas S, Reading J, Shephard RJ. Revision of the Physical-Activity Readiness Questionnaire (Par-Q). Can J Sport Sci 1992: 17: 338–345.