THE USE OF POST-EXERCISE COOLING AS A RECOVERY STRATEGY: UNRAVELING THE CONTROVERSIES

EDITED BY: Mohammed Ihsan, Robert Allan and Chris R. Abbiss
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Topic Editors:
Mohammed Ihsan, National University of Singapore, Singapore
Robert Allan, University of Central Lancashire, United Kingdom
Chris R. Abbiss, Edith Cowan University, Australia

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EDITORIAL
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Editorial: The Use of Post-exercise Cooling as a Recovery Strategy: Unraveling the Controversies

Mohammed Ihsan[1]*, *Chris R. Abbiss*[2] *and Robert Allan*[3]

[1] Human Potential Translational Research Program, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, Singapore, [2] Centre for Exercise and Sports Science Research, School of Medical and Health Sciences, Edith Cowan University, Perth, WA, Australia, [3] School of Sport and Health Sciences, University of Central Lancashire, Preston, United Kingdom

Keywords: cold water immersion, cryotherapy, phase change material, physiological recovery, muscle strength

Editorial on the Research Topic

The Use of Post-exercise Cooling as a Recovery Strategy: Unraveling the Controversies

INTRODUCTION

Post-exercise cooling is a popular recovery strategy utilized by athletes, and of interest to many research groups. Significant body of research have examined the effects of post-exercise cooling on outcomes such as physical performance, regulation of inflammatory biomarkers, and psychophysical indices related to perceived fatigue, recovery and wellbeing. Despite the research and widespread use by athletes, there is considerable skepticism regarding its efficacy, in-part stemming from mixed findings reported within the literature. Moreover, emerging work demonstrating dampened hypertrophy gains following the regular use of cold-water immersion (CWI) has further questioned the appropriateness of cooling modalities as a recovery technique. Many of these diverse findings may be reconciled through considering factors such as the cooling modality and protocol, nature of exercise stressor, and type and timing of recovery assessment. Regardless, controversy remains with regards to the use of post-exercise cooling, often resulting in mixed and misinformed messages for practitioners in applied settings.

Accounting for and understanding the differences within experimental context is critical for the appropriate use of post-exercise cooling in applied practice. In this Research Topic, we invited scientists to offer commentary and critically examine key areas surrounding the use of recovery cooling strategies. This will aid in developing the available body of literature into appropriate context for practitioners and provide directions for future research. Thirteen articles were accepted for publication (five original research, three reviews, two mini-reviews, two opinion pieces, and one systematic review), written by a total of 53 contributing authors. We have summarized and discussed this collection with regards to improving applied practice, as well as addressing controversies surrounding the use of post-exercise cooling.

COLD WATER IMMERSION AND STRENGTH ADAPTATION

Petersen and Fyfe reviewed the influence of CWI on strength adaptation. The authors concluded that whilst CWI may enhance short-term recovery following resistance exercise, the majority of the evidence aligned toward dampened molecular signatures and physiological adaptations to resistance training.

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Mathieu Nedelec, Institut National du Sport, de l'Expertise et de la Performance (INSEP), France

***Correspondence:**
Mohammed Ihsan
dr.Ihsan.m@gmail.com

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Conversely, Tavares et al. showed no impairments, in fact demonstrated a trend toward improved vertical jump performance following regular use of CWI (i.e., 12 sessions of CWI over 3 weeks) during intensified training. In support, review by Ihsan et al. highlighted other works showing no impairments, instead a trend toward improved strength amongst athletes undertaking post-exercise CWI over 2.5 weeks−8 months. This disconnect between Petersen and Fyfe and others (Tavares et al.; Ihsan et al.) may be reasoned by differences in experimental approach, where laboratory-based studies have typically employed 2–3 training sessions per week, potentially allowing for full recovery between sessions. Conversely, in applied scenarios where recovery between training sessions is limited (i.e., >10 sessions/week), CWI appears to improve subsequent training performances and consequently allow maintenance of a sufficient training stimulus for adaptation. Moreover, beneficial recovery outcomes of CWI may be harnessed by programming this modality following technical or aerobic conditioning, and avoiding it following resistance training sessions (Ihsan et al.).

Recovery periodization thus has been proposed within this Research Topic, where post-exercise cooling is strategically programmed to align with the suitability of the preceding exercise task, the physiological system(s) that requires restoration, and the need for recovery dictated by changes in training demands and athlete wellness (Tavares et al.; Ihsan et al.; Thorpe).

COOLING BETWEEN HIGH-INTENSITY EFFORTS

Egaña et al. investigated the effects of 5–10 min CWI administered between 2 bouts of 4 km cycling time-trials performed in normothermia. The authors reported similar performances compared to passive or active recovery, despite alleviated thermal and perceptual strain following CWI. The mechanisms by which CWI may benefit such performance tasks are not clear, but plausible avenues include haemodynamic changes (influencing venous return), cardiac parasympathetic reactivation and/or reduced perception of effort. Regardless, the use of cooling amidst closely scheduled high-intensity and short duration efforts is at best contentious, especially in the absence of environmental heat stress. This is gathered from long-standing evidence supporting warmer temperatures for improved muscle function, and hyperthermia not being a primary limiting factor during such short task. This perhaps reiterates to first identify the origins of fatigue and the physiological system(s) that need recovery, which then will serve as the driving factor to select appropriate recovery intervention(s) (Thorpe).

POST-EXERCISE COOLING AND PERCEPTUAL RECOVERY

L'Hermette et al. explored how CWI can influence sensorial experiences and accompanying physiological recovery. CWI alleviated muscle soreness, facilitated sympathetic withdrawal and increased parasympathetic activity. Although athletes reported of pain and discomfort upon immersion, these sensations progressively declined over the initial 3 min, and was superseded by improved sense of relaxation, wellbeing, vigor, and vitality. Future work should investigate how such sensorial experiences evolve over intensified training periods. Importantly, it is critical to understand how such changes translate to improved physical function, given that Lindsay and Peake have identified muscle strength and/or power as a direct, training-specific variable encompassing recovery.

INDIVIDUALIZING AND OPTIMIZING COOLING PROTOCOLS

Work within this Research Topic (Freitag et al.) and elsewhere (Stephens et al., 2018) have provided typical muscle and core temperature changes following common CWI protocols. Practitioners can utilize these resources in gathering estimates of body temperature changes, given that regular measurements are impractical in applied settings involving athletes. While such resources are useful, individualizing temperature thresholds associated with specific recovery objectives are difficult to establish, and would require occasional direct measurement of body temperatures to complement/verify estimates derived from such resources. In applied settings, Lindsay and Peake recommend regular assessments of muscle strength and/or power, as they provide specific, interpretable data for practitioners and coaches. Regular performance measures should accompany recovery programs to ascertain the efficacy of a particular cooling modality or protocol for a given training stimulus.

EMERGING MECHANISMS

Key responses to post-exercise cooling include peripheral vasoconstriction, decrease in limb blood flow, and redistribution of blood volume from the periphery to the core. Seeley et al. highlighted that such physiological changes can help attenuate post-exercise orthostatic intolerance, amongst other associated recovery benefits. Further research is warranted to examine how cooling mitigates orthostatic intolerance triggered by exercise heat stress, and translation to physical recovery in athletes and clinical populations.

Chauvineau et al. suggested that including the head (aided by scuba kit) to CWI following evening training can improve subsequent sleep architecture. However, inclusion of head immersion decreased parasympathetic activity and hampered thermal sensation and comfort, although sleep propensity was improved. Future studies should aim to refine cooling methods/protocols to further the development of strategies to enhance sleep and recovery for athletes.

EMERGING COOLING MODALITIES

Other modalities featured include partial- or whole-body cryotherapy (Bouzigon et al.), battery-powered hand cooling devices (Seeley and Sherman) and specialized phase change

material (PCM) (Kwiecien et al.). These modalities purportedly offer advantages over traditional methods (e.g., ice or CWI) to target various recovery objectives. For instance, prolonged (3–6 h) mild cooling stimulus conferred by PCMs are purportedly more effective to mitigate strength loss following muscle damage (Kwiecien et al.). The availability of such modalities undoubtedly allows practitioners to select the most appropriate method to elicit physiological alterations to achieve specific recovery objectives. However, possible disadvantages may include cost, energy supply and logistics when catering to multiple athletes (e.g., team sport). Further research is thus needed to solidify the evidence supporting the use of these modalities, such that associated disadvantages may be deemed secondary.

CONCLUSION AND PERSPECTIVES

This Research Topic has addressed controversies associated with post-exercise cooling, and have highlighted exciting avenues for further research. While this compilation is an informative resource for practitioners and scientists, formulating a recovery cooling program is an endeavor that requires purposeful trial and error, guided by experimental evidence. Among some of the pertinent questions are what physiological system(s) or performance measures are impaired? What is the natural recovery time-course of these measures? What is the nature and timing of the ensuing training activity? Is the utilized recovery assessment reliable, valid and specific? Each of these considerations will then provide the basis for integrating appropriate cooling strategies as part of the athletes' overall recovery program that is individualized and periodized.

AUTHOR CONTRIBUTIONS

All authors listed have made a substantial, direct, and intellectual contribution to the work and approved it for publication.

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MINI REVIEW
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Don't Lose Your Cool With Cryotherapy: The Application of Phase Change Material for Prolonged Cooling in Athletic Recovery and Beyond

Susan Y. Kwiecien[1,2]*, *Malachy P. McHugh*[1,2] *and Glyn Howatson*[2,3]

[1] *Nicholas Institute of Sports Medicine and Athletic Trauma, Lenox Hill Hospital, New York, NY, United States,* [2] *Department of Sport, Exercise and Rehabilitation, Northumbria University, Newcastle upon Tyne, United Kingdom,* [3] *Water Research Group, North West University, Potchefstroom, South Africa*

Strenuous exercise can result in muscle damage in both recreational and elite athletes, and is accompanied by strength loss, and increases in soreness, oxidative stress, and inflammation. If the aforementioned signs and symptoms associated with exercise-induced muscle damage are excessive or unabated, the recovery process becomes prolonged and can result in performance decrements; consequently, there has been a great deal of research focussing on accelerating recovery following exercise. A popular recovery modality is cryotherapy which results in a reduction of tissue temperature by the withdrawal of heat from the body. Cryotherapy is advantageous because of its ability to reduce tissue temperature at the site of muscle damage. However, there are logistical limitations to traditional cryotherapy modalities, such as cold-water immersion or whole-body cryotherapy, because they are limited by the duration for which they can be administered in a single dose. Phase change material (PCM) at a temperature of 15°C can deliver a single dose of cooling for a prolonged duration in a practical, efficacious, and safe way; hence overcoming the limitations of traditional cryotherapy modalities. Recently, 15°C PCM has been locally administered following isolated eccentric exercise, a soccer match, and baseball pitching, for durations of 3–6 h with no adverse effects. These data showed that using 15°C PCM to prolong the duration of cooling successfully reduced strength loss and soreness following exercise. Extending the positive effects associated with cryotherapy by prolonging the duration of cooling can enhance recovery following exercise and give athletes a competitive advantage.

Keywords: muscle damage, injury, cooling, recovery modalities, recovery strategy

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Edited by:
Mohammed Ihsan,
Aspetar Hospital, Qatar

Reviewed by:
Julien Louis,
Liverpool John Moores University, United Kingdom
James Robert Broatch,
Victoria University, Australia

***Correspondence:**
Susan Y. Kwiecien
susan@nismat.org

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RATIONALE

Exercise often involves high intensity physical and physiological stress that ultimately results in structural damage to the skeletal muscle (Armstrong, 1990; Armstrong et al., 1991; Clarkson and Sayers, 1999; Proske and Morgan, 2001; Proske and Allen, 2005). Three pathways exist during exercise that result in the structural damage of the muscle. These include: the increase

in muscle temperature resulting from exercise-induced heat generation (Arbogast and Reid, 2004), the metabolic stress that commonly occurs during exercise of high intensity or prolonged durations (Spiteller, 2006; Clanton, 2007; Supinski and Callahan, 2007; Tee et al., 2007), and/or the direct mechanical stress to the muscle (Staublr, 1989; Friden and Lieber, 1992, 2001; Proske and Morgan, 2001; Lieber, 2018). The initial structural damage occurring within the muscle fiber initiates a positive feedback mechanism during which the aforementioned initial damage response is exacerbated (Kendall and Eston, 2002; Merrick, 2002; Howatson and van Someren, 2008). This phase of muscle damage is referred to as the secondary damage response. Ultimately, secondary muscle damage compounds the symptoms of exercise-induced muscle damage and results in impaired muscle function in the hours and days following exercise (Lapointe et al., 2002). If not managed correctly, these effects can be detrimental to an athlete's recovery and subsequent performance. As a result, accelerating recovery following strenuous exercise has been the focus of much research; particularly when there is inadequate recovery between repeat exercise exposures. In these scenarios, rapid deployment of a recovery strategy is important for athletes to accelerate the return to optimal performance.

Exposure of the damaged muscle to cold (cryotherapy) is believed to retard the secondary injury process (Merrick et al., 1999; Merrick, 2002). Cryotherapy, the reduction of tissue temperature by the withdrawal of heat from the body (Michlovitz, 1990), refers to a range of cooling modalities such as local ice application to the skin (Yackzan et al., 1984; Gulick et al., 1996; Oakley et al., 2013; Nogueira et al., 2019), cold water immersion (CWI) of a large part of the body (Lane and Wenger, 2004; Yeargin et al., 2006; Vaile et al., 2007, 2008, 2010; Halson et al., 2008; Montgomery et al., 2008; Peiffer et al., 2008; Rowsell et al., 2009, 2011; Brophy-Williams et al., 2011; Bleakley et al., 2012; Leeder et al., 2012, 2019; Versey et al., 2013; Webb et al., 2013; Roberts et al., 2015; Garcia et al., 2016; Machado et al., 2016; Wilson et al., 2018), whole body cryotherapy (Banfi et al., 2009, 2010; Costello et al., 2011, 2015; Hausswirth et al., 2011; Pournot et al., 2011; Ziemann et al., 2012; Fonda and Sarabon, 2013; Guilhem et al., 2013; Bleakley et al., 2014; Ferreira-Junior et al., 2015; Vieira et al., 2015; Rose et al., 2017; Broatch et al., 2019; Krueger et al., 2019; WBC) and more recently phase change material (PCM) cooling (Clifford et al., 2018; Kwiecien et al., 2018, 2020a,b; Brownstein et al., 2019; Mullaney et al., 2020), that are employed in various contexts. The most popular cryotherapy modality used following exercise is CWI involving immersion of a large surface area of the body, typically immersion of at least the legs up to at least the umbilicus, in cold water. Most commonly CWI occurs in water temperatures of 15°C or less for a single duration of 15 min or less (Leeder et al., 2012). Evidence supports the use of CWI for accelerating recovery of soreness (Barnett, 2006; Bailey et al., 2007; Montgomery et al., 2008; Ingram et al., 2009; Pournot et al., 2010; Hausswirth and Le Meur, 2011; Pointon et al., 2011; Bleakley et al., 2012; Leeder et al., 2012; Minett et al., 2012; Pointon and Duffield, 2012; Elias et al., 2013; Poppendieck et al., 2013; Hohenauer et al., 2015; Machado et al., 2015; Ihsan et al., 2016; Siqueira et al., 2018). There is also some evidence to support the use of CWI for accelerating recovery of blood markers of muscle damage (Leeder et al., 2012; Hohenauer et al., 2015; Siqueira et al., 2016; Dupuy et al., 2018) and inflammation (Vieira Ramos et al., 2016; Dupuy et al., 2018), as well as functional recovery (Vaile et al., 2008; Leeder et al., 2019) following exercise. However, evidence to support its use for accelerating recovery of strength loss following exercise remains equivocal (Bleakley et al., 2012; Leeder et al., 2012; Poppendieck et al., 2013; Versey et al., 2013; Hohenauer et al., 2015; Machado et al., 2015). Comparably, some studies suggest that WBC might be beneficial in accelerating subjective recovery of soreness (Banfi et al., 2010; Hausswirth et al., 2011; Pournot et al., 2011; Ziemann et al., 2012; Fonda and Sarabon, 2013; Bleakley et al., 2014; Costello et al., 2015; Rose et al., 2017), strength loss (Hausswirth et al., 2011), and might mitigate the signs of functional overreaching (Schaal et al., 2015). However, more recently, there remains little evidence to support improvements in functional recovery (Bleakley et al., 2014; Lombardi et al., 2017; Rose et al., 2017; Broatch et al., 2019; Krueger et al., 2019). On the contrary, local ice application does not improve the symptoms associated with soreness or strength loss (Nogueira et al., 2019). Thus, local ice application is generally not effective in the treatment of structural damage following exercise. Ultimately, the lack of evidence identifying specific guidelines concerning traditional cryotherapy treatment application, temperature, duration, and frequency, as well as the variability in exercise models utilized throughout the literature, likely contribute to the controversy surrounding the efficacy and practicality of cryotherapy for accelerating recovery following exercise. As a result, no consensus exists for optimal cryotherapy treatment criteria and there remains a large gap in the scientific basis for administering cryotherapy for anything other than subjective recovery following exercise.

Recent evidence suggests that the physiological changes that occur following cryotherapy are primarily dependent on the reduction in intramuscular temperature (Wilcock et al., 2006; White and Wells, 2013; Ihsan et al., 2016) and only secondarily reliant on vasoconstriction leading to a decrease in blood flow (Gregson et al., 2011; Ihsan et al., 2013; Mawhinney et al., 2013, 2020) which might decrease muscle metabolism and inflammation, resulting in a reduction in the proliferation of secondary damage (Meeusen and Lievens, 1986; Knight, 1995; Eston and Peters, 1999; Merrick et al., 1999; Merrick and McBrier, 2010). Evidence from animal models suggests that the optimal muscle temperature range for reducing cellular metabolic activity (Osterman et al., 1984; Sapega et al., 1988) and oxygen demand (Fuhrman, 1959; Fuhrman et al., 1961) without causing tissue damage, is 10–15°C (Sapega et al., 1988). However, *in vivo* intramuscular temperatures below 20°C during traditional cryotherapy application in humans have not been reported (Bleakley and Hopkins, 2010; Bleakley et al., 2012). In order to sustain a clinically relevant reduction in intramuscular temperature, the duration of cryotherapy would have to be prolonged (Peiffer et al., 2009). However, treatment duration does not commonly exceed 30 min because extending the duration of cryotherapy is likely to result in increased discomfort particularly at lower temperatures (Bailey et al., 2007; Vaile et al., 2008; Heyman et al., 2009; Versey et al., 2011) or

can be unsafe (Tipton et al., 2017). Rapid reductions in skin temperature, before muscle and core temperatures can catch up, might result in cold related injury to the skin (Gage, 1979; Wilke and Weiner, 2003; Selfe et al., 2007) because the skin is most prone to irreversible damage. To date, maintaining a reduction in muscle temperature without causing cold related injury to the skin could only be achieved by administering traditional cryotherapy modalities (ice, gel packs, CWI, WBC, etc.) in an intermittent fashion (Mac Auley, 2001). However, Cheng et al. (2017) recently reported intramuscular temperature reductions to approximately 15°C following a 120-min localized cooling intervention administered to the upper arm, making the case for a prolonged duration of cryotherapy application in order to achieve clinically relevant reductions in intramuscular temperature. Nevertheless, the protocol utilized by Cheng et al. (2017), 120 min of cooling with ice-chilled water-perfused arm cuffs, is atypical from common practice in the application to athletes in the "real world" (Ihsan et al., 2020).

In humans, the magnitude of change in tissue temperature has been positively correlated with cryotherapy methods that undergo a phase change (Merrick et al., 2003; Dykstra et al., 2009). Specifically, Merrick et al. (2003) demonstrated that modalities such as ice that change phase while they melt (e.g., from solid to a liquid) cause lower skin and intramuscular temperatures than cryotherapy modalities such as gel packs that do not possess these properties (ice bag: 6.5°C, skin temperature; 27.8°C, 1 cm intramuscular temperature; vs. gel pack: 9.9°C, skin temperature; 29.5°C, 1 cm intramuscular temperature; Merrick et al., 2003). A phase change is important because it greatly enhances the ability of a cryotherapy modality to absorb heat (Merrick et al., 2003). The phase change relates to a property called "enthalpy of fusion," which is the quantity of heat required to make the material change phase (Merrick et al., 2003). Enthalpy of fusion greatly enhances the ability of a cold modality to absorb heat by prolonging the latent phase, and thus results in a greater ability to reduce intramuscular temperature. When a substance is changing phases, there is only a change in phase but no change in temperature. This "hidden" energy is defined as the latent phase. On the contrary, sensible heat is heat that can be felt and measured by a thermometer. Neither gel packs nor WBC undergo a phase change, meaning that both modalities only experience sensible heat loss as their temperature equilibrates with the ambient temperature (**Figure 1**). Similarly, CWI does not change phase, but its temperature can be artificially maintained at a constant creating an artificial latent phase period. On the contrary, when ice is heated by exposure to the human body, its temperature increases, and it experiences a change in phase as it melts. While undergoing the phase change, ice experiences latent heat loss during which the temperature remains constant (**Figure 1**). Therefore, modalities experiencing the latent phase have an advantage over modalities only capable of experiencing a sensible heat phase, by providing a greater cooling potential.

Although ice is the most commonly utilized PCM for exercise recovery, its latent phase of 0°C limits it from maintaining a cooling capacity for prolonged periods. Luckily, the latent phase of any PCM can be manipulated. The duration of the latent phase can be prolonged as the temperature of phase change increases above 0°C. The cooling effect of any PCM is dependent on the capacity to absorb heat during periods when external heat load or body heat production exceeds heat loss. Therefore, the duration of the latent phase is variable and dependent on the temperature gradient between the skin and the PCM, the PCM phase transition point, the area covered by PCM and the volume of the PCM (Tiest et al., 2012; Hassabo, 2014). For example, PCMs will melt faster if the skin is warmer, PCM with a phase transition point of 10°C will not hold that temperature as long as PCMs with a set point of 15°C, and a small volume of PCM will melt faster than a larger volume.

The 15°C PCM packs used throughout the course of the subsequently described research studies are filled with a proprietary blend of fully hydrogenated natural fats certified by the U.S. Food and Drug Administration as food-grade chemicals such as palm oil, palm kernel oil, rapeseed oil, coconut oil and soybean oil, mixed with sodium chloride, and encapsulated in flexible plastic (Glacier Tek USDA BioPreferred PureTemp PCM, Plymouth, MN, USA). Similar to ice, the 15°C PCM are firm and look like wax when in their frozen solid state and look like vegetable oil once they reach their melted liquid state (**Figure 2**). A PCM with a latent phase of 15°C is capable of safely prolonging and maintaining the duration of cooling for 3 h (Kwiecien et al., 2019), while avoiding the need for repeat applications. If there is a need to extend the duration of application beyond 3 h, a fresh set of "frozen" PCM packs can be administered for an additional 3 h. Applying 15°C PCM packs directly to the skin fitted under a garment might be less time consuming, logistically simpler to implement than other cryotherapy modalities, and is more practical particularly because they can simultaneously deliver a cooling treatment while the individual continues activities of daily living. Furthermore, 15°C PCM packs can be applied concurrently to multiple athletes, or even entire teams at a time. Recent evidence suggests that prolonging the duration of cooling for three (Clifford et al., 2018; Brownstein et al., 2019; Mullaney et al., 2020) to 6 h (Kwiecien et al., 2018, 2020a) is well tolerated by recreational and professional athletes alike and can accelerate more than just subjective recovery following exercise. Therefore, this narrative review will aim to summarize the current evidence in support of prolonging the duration of cooling by using 15°C PCM, for its capacity to accelerate and aid recovery following exercise. As the literature has already summarized the mechanisms of cryotherapy and its effects on the mechanisms involved with exercise-induced muscle damage (Versey et al., 2013; Hohenauer et al., 2015; Machado et al., 2015; Ihsan et al., 2016; Dupuy et al., 2018; Stephens et al., 2018), an extensive summary is beyond the scope of this review.

EVIDENCE

Much of the previous research utilizing PCM cooling at phase transition points ranging between 10 and 31°C has focused on its temperature-regulating effect (Gao et al., 2010, 2011), and the ability of PCM to elicit thermal comfort from heat strain occurring during strenuous activity (Bennett et al., 1995;

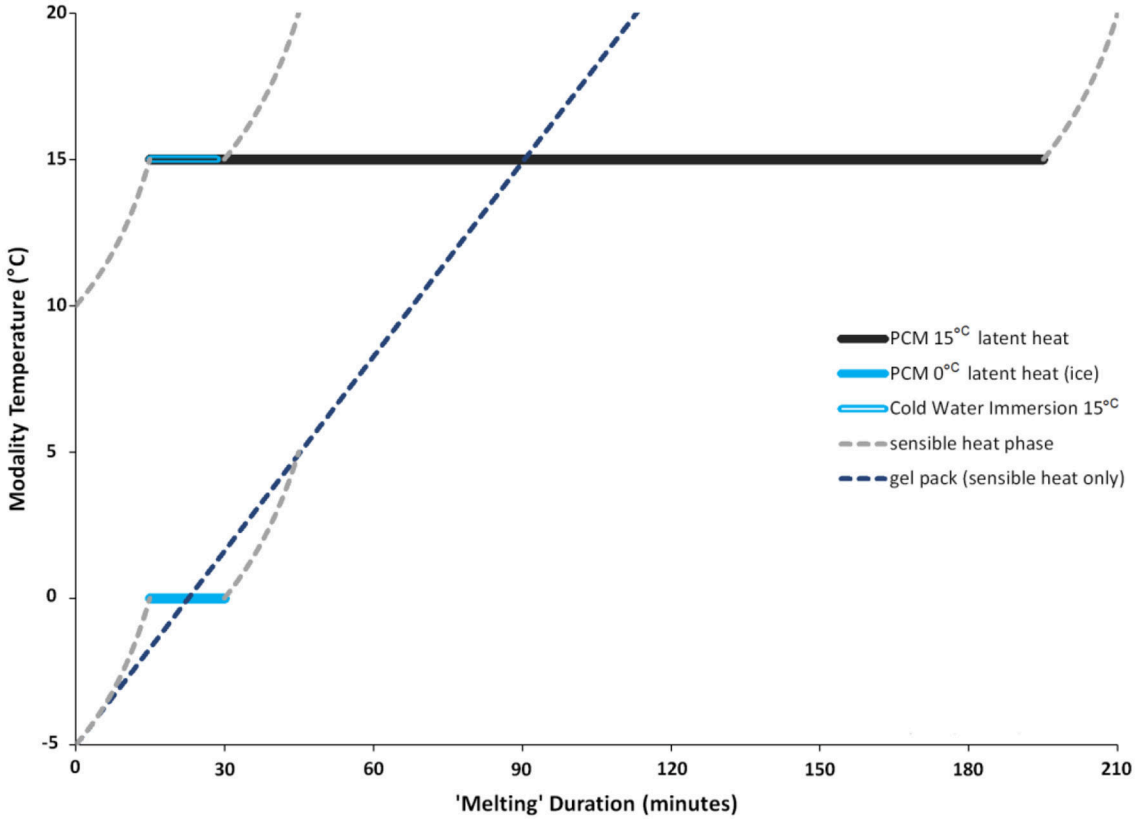

FIGURE 1 | Sample melting pattern of PCM with a phase transition point of 15°C, or 0°C for comparison, and for material with only sensible heat properties (e.g., ice pack). Latent heat period (solid line) demonstrates a change in phase while maintaining a constant temperature. The latent heat phase is longer for 15°C PCM than it is for 0°C PCM (ice melting from solid to liquid). The 15°C PCM is compared with CWI that is kept at a constant temperature of 15°C for a 15-min duration. Once the CWI temperature is no longer held at a constant, the water temperature will begin to equilibrate to room temperature (sensible heat). The 0°C PCM (ice) is compared with a gel pack that is initially frozen at −5°C but warms up to 20°C as it equilibrates with room temperature, so it does not change phase and experiences only sensible heat. Note: PCM melting duration is dependent on the temperature gradient between skin and PCM, the PCM phase transition point, the area covered by PCM and the volume of the PCM.

Zhang, 2003; Chou et al., 2008; Kenny et al., 2011; House et al., 2013), between two bouts of exercise (Duffield and Marino, 2007; Hausswirth et al., 2012), or following exercise (Purvis and Cable, 2000; Tate et al., 2008; Barwood et al., 2009; Brade et al., 2010). In this context, most of the research has applied PCM through pockets in vests worn on the chest in an attempt to reduce elevations in core temperature, for treatment durations longer than CWI. Following strenuous activity, 24°C PCM vests have been shown to be more effective at cooling the skin than 28°C vests, but neither had an effect on core temperature (Gao et al., 2011). A PCM with a melting point <24°C might reduce core temperature more effectively (Gao et al., 2011). In rested individuals, local application of 15°C PCM to the quadriceps for 3 h reduced core temperature by 0.28°C compared with a 0.25°C reduction from 15 min of 15°C CWI (Kwiecien et al., 2019). Although the difference in the reduction in core temperature from both PCM and CWI was negligible, the CWI reduced core temperature faster than the PCM treatment but the reduction in core temperature was maintained for the 3-h duration of 15°C PCM application (Kwiecien et al., 2019). In comparison, following exercise, a pooled data analysis of 13 studies found that CWI reduced core temperature by 0.84°C (Stephens et al., 2018). Following exercise, cryotherapy is expected to induce a greater magnitude in the reduction of core temperature than at rest due to the exercise-induced hyperthermia resulting in a larger thermal gradient. Unfortunately, the effects of 15°C PCM cooling on core and muscle temperature remain to be elucidated following exercise.

In order to induce reductions in local metabolic demand (Ho et al., 1995; Merrick, 2002; Schaser et al., 2006) and the inflammatory response that occurs after the acute structural trauma at the site of injury or muscle damage (Ciolek, 1985; Knight, 1985, 1995; Merrick et al., 1999; Merrick, 2002; Schaser et al., 2006; Swenson et al., 2007), intramuscular temperature must drop to sufficiently low levels (10–15°C; Sapega et al., 1988). Since the secondary injury response extends for several hours post-exercise, a single 15-min CWI treatment may be an inadequate dose to influence recovery. Furthermore, extending the duration of a single CWI treatment would not be well tolerated. As a result, previously the only way to sustain a

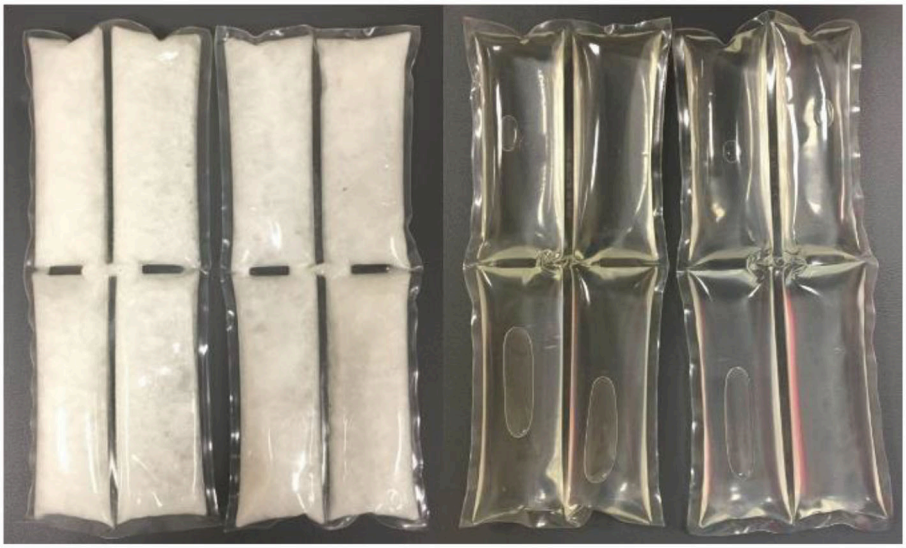

FIGURE 2 | Two Glacier Tek 15°C PCM packs in the frozen state **(left)**, and in their melted state **(right)**.

reduction in intramuscular temperature without causing cold related injury to the skin was through repeat applications of traditional cryotherapy modalities at frequent intervals. However, this practice is not commonplace amongst athletes, as they are unlikely to comply with such a demanding treatment schedule because it would mean that athletes must remain on site for an extended period, and there are major logistical challenges to treating entire teams at one time. As a result, the duration for which muscle temperature is reduced during traditional cryotherapy treatment is likely too short to elicit meaningful reductions in muscle temperature for durations long enough to be clinically relevant (10–15°C; Sapega et al., 1988). Although the intramuscular temperatures reported from 3 h of 15°C PCM cooling are nowhere close to those necessary for reducing local muscle metabolism (26.0 ± 2.2°C at 1 cm and 28.2 ± 2.8°C at 3 cm of the vastus lateralis), the reduction was safely maintained for a prolonged and continuous duration in individuals at rest (Kwiecien et al., 2019). Furthermore, these temperatures were comparable to those occurring from 15 min of 15°C CWI even though two PCM packs (864 cm^2 area; 32.4 × 2 × 13.3 cm) were administered locally and fitted directly on the skin over the quadriceps of each leg, while CWI treatment involved whole body immersion up to the umbilicus (Kwiecien et al., 2019). Therefore, prolonging the treatment duration using 15°C PCM cooling affords athletes the opportunity to safely receive cryotherapy treatment for an extended duration with the ability to leave the training facility and resume their normal routines.

Recent studies examining the effects of prolonged 15°C PCM cooling on indices of recovery in untrained (Kwiecien et al., 2018) and trained (Kwiecien et al., 2020a) individuals found that 6 h of PCM cooling not only accelerated recovery of soreness, but also accelerated recovery of strength loss on the days after isolated eccentric quadriceps exercise (**Table 1**). Importantly, the protection provided by the PCM cooling after an initial bout of eccentric exercise did not interfere with the repeated bout effect, whereby an initial bout confers an adaptive protective effect for a subsequent bout of damaging exercise (Kwiecien et al., 2020a). Similarly, 3 h of 15°C PCM cooling has also been shown to accelerate recovery of quadriceps strength following soccer match play (Clifford et al., 2018; Brownstein et al., 2019; **Table 1**) and shoulder internal rotation strength and grip strength following baseball pitching (Mullaney et al., 2020; **Table 1**). Alleviating strength loss at a rate faster than normal could allow athletes to be better prepared for subsequent performance, giving them a competitive advantage over the opposition. The beneficial effects of PCM cooling on recovery of strength loss following exercise in these five studies are in contrast to the results of research utilizing CWI for recovery, which has generally shown little or no benefit for recovery of strength loss following exercise (Leeder et al., 2012; White and Wells, 2013; Ihsan et al., 2016). Overall, these results suggest that prolonging the duration of cryotherapy might successfully reduce the proliferation of secondary muscle damage and decrease the magnitude of repair necessary to achieve pre-exercise functional integrity, thereby shortening the time required to attain full recovery of muscle strength.

While prolonged cooling using 15°C PCM was shown to be successful in accelerating recovery following eccentric exercise (Kwiecien et al., 2018, 2020a), soccer (Clifford et al., 2018; Brownstein et al., 2019), and baseball pitching (Mullaney et al., 2020), no benefit of accelerated recovery from 15°C PCM cooling was found when administered for 3 h in runners following a marathon (Kwiecien et al., 2020b; **Table 1**). It is possible that the efficacy of prolonged cooling using 15°C PCM is dependent on the mode of exercise stress resulting in muscle damage, i.e., mechanical vs. metabolic stress. Alternatively, the lack of effect on recovery following a marathon could be due to the combination of the long duration of exercise and the delay

TABLE 1 | Summary of evidence available on PCM studies applying 15°C PCM cooling for post-exercise recovery.

Study	Population	Exercise type	Cooling technique	Cooling duration	Variables reported	Main outcomes
Kwiecien et al. (2018)	Recreational athletes	Isolated eccentric exercise of the quadriceps	1) Direct PCM cooling 2) Indirect PCM cooling 3) Control	6 h on the quadriceps	↓ Skin temperature ↓ Strength loss ↓ Soreness	• 6 h of direct local PCM cooling was well tolerated • Recovery of strength loss and soreness was accelerated • Leg receiving indirect cooling was not statistically different from direct cooling indicating a potential systemic effect
Clifford et al. (2018)	Professional athletes	Soccer match	1) Direct PCM cooling 2) Control	3 h on the quadriceps	↓ Strength loss ↓ Soreness ↑ Counter movement jump (results published in McHugh et al., 2019)	• PCM cooling can provide a practical means of delivering prolonged post-exercise cooling to entire teams of athletes • PCM cooling can accelerate recovery in elite athletes
Brownstein et al. (2019)	Semi-professional athletes	Soccer match	1) Direct PCM cooling 2) Control	3 h on the quadriceps	↓ Strength loss ↓ Voluntary activation ✘ Soreness ✘ Counter movement jump ✘ Reactive strength index	• PCM cooling accelerated recovery of central nervous system function but not muscle contractile function • The lack of effect on measures of physical function or perceptual responses might have been due to the relatively small magnitude of change in most of the outcome measures studied, which could be related to the training status of the study participants
Kwiecien et al. (2020a)	Regularly participating in team-sport or other forms of physical exercise but not eccentrically trained	Isolated eccentric exercise of the quadriceps	3) Direct PCM cooling 4) Control	6 h on the quadriceps	↓ Skin temperature ↓ Strength loss ↓ Soreness ✘ Creatine kinase ✘ High sensitivity c-reactive protein	• Recovery of strength loss and soreness was accelerated • No effect on blood markers of muscle damage • Exercise was repeated but repeated bout effect was not hindered by initial cooling (no strength loss or soreness after second exercise bout)
Kwiecien et al. (2020b)	Recreational athletes	Marathon run	1) Direct PCM cooling 2) Control	3 h on the quadriceps	✘ Strength loss ✘ Soreness ✘ Counter movement jump ✘ Creatine kinase ✘ High sensitivity c-reactive protein	• No effect on accelerating recovery of any variable • Might be related to either shorter duration of cooling or exacerbated damage response following marathon • Soreness was inversely correlated with number of prior marathons
Mullaney et al. (2020)	Collegiate athletes	Baseball pitching	1) Direct PCM cooling 2) Control	3 h on the shoulder and elbow	↓ Strength loss ✘ Soreness ✘ Creatine kinase	• PCM cooling can be applied comfortably to the arm and accelerates recovery of muscle function in baseball pitchers

↓ = *decrease*, ↑ = *increase*, ✘ = *no effect.*

in application of the PCM packs to the athletes. Marathon finish times averaged more than 4 h, and it was more than an hour after race completion before the PCM cooling packs were applied to the quadriceps (Kwiecien et al., 2020b). Thus, treatment began more than 5 h after the commencement of the exercise stress. By contrast, in the two soccer studies (Clifford et al., 2018; Brownstein et al., 2019), the PCM cooling packs were applied within 30 min of exercise cessation, which would correspond to approximately 2 h after the commencement of exercise. Although evidence supports the delayed use of CWI (administered 3 h post-exercise) for accelerating recovery of inflammation and improving next day performance (Brophy-Williams et al., 2011), the exercise session performed prior to the cryotherapy treatment lasted a total duration of only 24 min. Therefore, although the delayed cryotherapy modality was not administered immediately upon cessation of exercise, the exercise duration was of significantly shorter duration than a marathon run. Furthermore, the inflammatory response in the control group only increased by 4.1% 24 h following the high intensity interval exercise session performed by the trained athletes in Brophy-Williams et al.'s (2011) study while the marathon run induced the inflammatory response by 8.6% (Kwiecien et al., 2020b), indicating a greater magnitude of exercise stress.

Of the few studies that have shown accelerated recovery of strength with a post-exercise CWI treatment, some involved

exercise in the heat; which results in increased thermal strain and possible central fatigue (Pointon et al., 2011; Minett et al., 2014). In the aforementioned studies, recovery of strength was concomitant with the acute amelioration of voluntary activation and core temperature. It has been suggested that CWI mediated recovery of strength loss following exercise in the heat might not solely reflect recovery from exercise-induced muscle damage but might also include recovery from central fatigue (Ihsan et al., 2016). Therefore, under a large thermal load, CWI might alleviate some of the exercise-induced cerebral perturbations through its ability to ameliorate both hyperthermia and the subsequent central nervous system mediated fatigue (Ihsan et al., 2016), via removal of the heat load alongside preservation of the peripheral physiological state (Minett and Duffield, 2014). However, the peripheral effect from cryotherapy on exercise-induced muscle damage occurring within the skeletal muscle might still be limited by the short cryotherapy duration that occurs during icing, CWI, or WBC. On the contrary, the local effects on skin and muscle temperature occurring during 15°C PCM cooling (Kwiecien et al., 2019) might be insufficient to influence core temperature when the magnitude and duration of thermal load is elevated, such as following a marathon (Deschenes et al., 1998; Mortensen et al., 2008). However, the prolonged duration of cooling possible when using PCM might be advantageous if the goal is to maximize the duration of tolerable decline in peripheral muscle temperature. Recovery with PCM cooling could give athletes an advantage particularly if the duration of cryotherapy application needs to be extended in a safe way.

FUTURE DIRECTIONS

To date, PCM cooling has only been studied in exercise recovery models with benefits shown for reducing strength loss and soreness after isolated eccentric exercise (Kwiecien et al., 2018, 2020a), accelerating recovery in professional (Clifford et al., 2018) and semi-professional soccer players (Brownstein et al., 2019), and improving strength recovery in baseball pitchers (Mullaney et al., 2020). These studies have answered some fundamental questions and provide some good "proof of concept" that continuous and prolonged PCM cooling application can help recovery of muscle function, reduction in soreness, and does not seem to attenuate acute adaptive responses (Kwiecien et al., 2020a). Nevertheless, in the exercise recovery paradigm, many questions remain unanswered, and the application of prolonged PCM cooling for injury management warrants more research.

The pilot study, which established the efficacy of PCM cooling as an alternative cryotherapy intervention for recovery of strength and soreness in the quadriceps on the days following bilateral isolated eccentric exercise, implemented a unilateral treatment design (Kwiecien et al., 2018). Therefore, the study had three treatment groups: direct cooling to one leg, indirect cooling to the leg contralateral to the direct cooling leg, and control (no PCM cooling). The treatment effects for strength and soreness were not different between the direct and indirect cooling conditions. Consequently, the results indicated that a systemic effect might have occurred in the indirect cooling leg, which did not receive PCM cooling, from the leg that did receive direct cooling. These results support the previous evidence that implicates a systemic response from unilateral cryotherapy treatment (Allan et al., 2017). A central effect from local 15°C PCM cooling was since confirmed by the reduction in core temperature and increase in heart rate variability evident during PCM treatment in individuals at rest (Kwiecien et al., 2019). These findings suggested that prolonged PCM cooling delivered a systemic, and not just a local effect; and might explain, in part, the accelerated recovery evident from PCM cooling following eccentric exercise (Kwiecien et al., 2020a), soccer match play (Clifford et al., 2018; Brownstein et al., 2019) and baseball pitching (Mullaney et al., 2020). However, the central effect occurring from PCM cooling remains to be directly elucidated in individuals following exercise.

Since PCM cooling can provide prolonged cooling to soft tissues (Kwiecien et al., 2019), there are obvious clinical applications. PCM cooling has the potential to accelerate recovery following muscle injury but this effect has not been examined. In the first 24–48 h after a muscle strain injury or a muscle contusion there is a proliferation of the tissue disruption around the site of the damaged muscle fibers (Järvinen et al., 2005). Immediate application of ice to a muscle tear or a contusion is standard treatment, with the goal being to reduce the proliferation of tissue degradation at the site of the injury (Järvinen et al., 2005). Evidence from animal models provides a strong rationale for the immediate post-injury application of cryotherapy to reduce the hematoma and inflammatory response, resulting in earlier regeneration of the injured tissue (Merrick et al., 1999; Deal et al., 2002; Schaser et al., 2006, 2007; Bleakley and Hopkins, 2010; Puntel et al., 2011). Further, evidence strongly supports the application of cryotherapy in the immediate stage following injury, as cryotherapy administered within the first 30 min of injury is a good window of opportunity for acute injury management (Merrick and McBrier, 2010). Surprisingly, although the clinical guidelines for post-injury ice application suggest application as soon as possible, several times a day for 15–20 min throughout the 72-h recovery period; (Michlovitz, 1990), there is no good clinical evidence in humans to support immediate icing or the clinically recommended rate of treatment. However, data from animal studies provide a strong rationale for immediate post-injury icing to reduce the hematoma and inflammation, resulting in earlier regeneration (Meeusen and Lievens, 1986; Hurme et al., 1993; Deal et al., 2002; Bleakley et al., 2004; Hubbard and Denegar, 2004).

The efficacy of both post-injury and post-exercise cryotherapy could be improved by delivering prolonged cooling to the damaged tissue within the first 24 h. To maximize efficacy, athletes and practitioners might opt to combine cryotherapy treatments. In practice, athletes could receive a standard 20–30 min ice treatment in the athletic training room or complete a CWI treatment in order to rapidly reduce both their intramuscular and core temperature, and follow this with the immediate application of PCM cooling packs over the muscle groups they wish to keep cool. Collectively, this approach will rapidly cool the tissue using more traditional techniques and would subsequently maintain the reduction of both peripheral

and central temperatures for extended periods by administering 15°C PCM. This could allow the athlete to sustain the treatment effect from the acute cryotherapy for a longer duration in the immediate post-injury or post-exercise period and have a better chance to accelerate the recovery processes. The advantage of this approach is that the athlete can return to normal post-exercise activities (e.g., meal, relaxation, recreational activities) while receiving a cryotherapy dose with no risk of cold injury to the skin or other tissues.

Nevertheless, it would be remiss not to mention the growing trend throughout the literature recommending against icing injuries so as not to delay or impair the regeneration process or the natural healing response (Takagi et al., 2011; White and Wells, 2013). Most recently, the pioneer of the original rest, ice, compression, elevation (RICE) method originally proposed in 1978 (Mirkin and Hoffman, 1978), rescinded his recommendation of the practice because it may delay healing, instead of facilitating it (Mirkin, 2015). Mirkin proposed that the healing process requires an inflammatory response, which is impaired by the application of ice. Specifically, during the inflammatory phase of regeneration, the activity of insulin-like growth factor (IGF-1) is upregulated as it moves into the damaged tissues (Shi and Garry, 2006; Yin et al., 2013). Research in animal models has demonstrated that cryotherapy decreased IGF-1 expression (Takagi et al., 2011; Vieira Ramos et al., 2016), which might be harmful for muscle regeneration. However, this effect has not been demonstrated in humans, and the degree of muscle cooling occurring in animal models is substantially greater than that which occurs in humans (even over the course of 3 h of continuous PCM cooling; Kwiecien et al., 2019). There is no evidence in humans to support the notion that cryotherapy could delay recovery following both injury or exercise, and data from animal models should not be directly compared to what would happen in humans. There is some evidence suggesting that the therapeutic effects attributed to cryotherapy treatment might be due to a placebo effect (Broatch et al., 2014). This limitation is inherent in all cryotherapy-based research, due to the inability to rule out that beneficial results could be due to the participants preconceived belief about how cold exposure might benefit their recovery. However, it is unlikely that the placebo effect would explain in full the beneficial effect from prolonged PCM cooling on recovery of strength loss.

CONCLUSION

The use of PCM cooling as a recovery modality offers a substitute to traditional cryotherapy modalities as it primarily allows for an increase in the duration of the cryotherapy dose in a safe way. The PCM cooling is well tolerated, safe, inexpensive, and delivers a prolonged cooling stimulus while allowing the athlete to continue with activities of daily living. Prolonging the duration of cryotherapy at physiologically comfortable temperatures might be the missing link for accelerating recovery through cryotherapy. Since CWI results in a more rapid initial decline in intramuscular temperature than PCM cooling (Kwiecien et al., 2019), a combination of CWI followed by PCM cooling could provide maximum cooling efficacy (rapid and prolonged). Future research should examine whether this combination further improves the recovery process and what the implications are for longer term athletic development. Future research should also establish evidence in support of prolonged cooling with 15°C PCM across various forms of exercise and whether a difference exists in the optimum duration of application. Gaining a greater understanding of the potential mechanisms by which 15°C PCM works for accelerating recovery will also advance the research, and potentially provide evidence in support of less noxious cooling in being considered a viable option as a treatment for recovery from exercise or soft tissue injury. On a similar note, establishing the potential benefits of prolonged cooling for acute injury management, post-operative inflammation and swelling and chronic disease such as rheumatological conditions, might lead to answers which could translate to the exercise recovery paradigm. Finally, understanding the implications on the adaptive responses to exercise training, particularly given the concerns over CWI and adaptation (for strength) would be of great value (Hyldahl and Peake, 2020).

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The Acute and Longer-Term Effects of Cold Water Immersion in Highly-Trained Volleyball Athletes During an Intense Training Block

Francisco Tavares[1], *Mário Simões*[2,3], *Bruno Matos*[3], *Tiaki Brett Smith*[4] *and Matthew Driller*[5]*

[1] Medical and Performance Department, Sporting Clube de Portugal, Lisbon, Portugal, [2] Research Centre, Polytechnic Institute of Maia, Maia, Portugal, [3] Portuguese Volleyball Federation, Porto, Portugal, [4] School of Health, University of Waikato, Hamilton, New Zealand, [5] Sport and Exercise Science, School of Allied Health, Human Services and Sport, La Trobe University, Melbourne, VIC, Australia

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***Correspondence:**
Matthew Driller
m.driller@latrobe.edu.au

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Background: The use of cold water immersion (CWI) as a recovery strategy following exercise has drawn mixed findings over the last few decades. The purpose of the current study was two-fold; (1) to determine the acute effects of CWI within the training week, and (2) to investigate the longer-term effects of CWI over a 16-day period.

Methods: In a randomized, controlled trial, 13 national-level volleyball athletes were allocated to two groups, an experimental (CWI, $n = 7$) and a control group ($n = 6$) during a 3-week national training camp. The experimental group were exposed to a CWI protocol after the last training session of each day (12 CWI sessions). Measures of lower (countermovement jump and squat jump height) and upper-body (medicine ball throw distance) power were collected pre- and post-training camp. Perceptual and neuromuscular performance measures (countermovement jump) were obtained during the training camp.

Results: No significant differences between groups were observed for any measure ($p > 0.05$), however, *small* effect sizes were observed between experimental and control groups on day two of weeks one and two. Three weeks of training resulted in a significant decrease in countermovement jump height in the control group. A moderate effect size ($d = 0.65$) was found for countermovement jump performance between the experimental and control groups.

Conclusion: Cold water immersion seems to provide little benefit to recovery in the acute setting (within the training week), however, chronically, there was a trend toward a benefit when implementing cold water immersion in well-trained volleyball athletes over 16 days.

Keywords: ice baths, adaptation, recovery, hydrotherapy, jump performance

INTRODUCTION

Muscle damage resulting from exercise has been shown to be related to a decrease in performance (Jamurtas et al., 2005). The most commonly investigated acute outcome of exercise-induced muscle damage (EIMD) is reductions in strength and power (Jamurtas et al., 2005). In volleyball, athletes often generate intensive eccentric and concentric activities such as jumps and locomotive actions (i.e., acceleration and deceleration) (Polglaze and Dawson, 1992). During a volleyball match, an athlete performs up to 145 jumps per game (Polglaze and Dawson, 1992). Landing from a jump typically results in the lower body muscles contracting eccentrically to decelerate the movement of the body in the negative direction (Horita et al., 1999). As a consequence of these activities, the EIMD results in an increase in perceived muscle soreness and decrease in neuromuscular function (Clarkson and Newham, 1995; MacIntyre et al., 1995). The high values of muscle damage and consequent muscle soreness expected from volleyball competition (and training), will likely generate acute fatigue, resulting in temporary performance decreases (Freitas et al., 2017). At an elite level, volleyball athletes train twice a day, for two or more days consecutively (Freitas et al., 2017), and during volleyball tournaments, it is not uncommon for teams to compete on successive days. Given the short time to recover between training or competition days, an accumulated level of fatigue can be expected throughout a training or competition week (Freitas et al., 2017) that may result in under-performance or lead to undesirable fatigue states (Freitas et al., 2014).

Sport scientists and coaches frequently implement recovery strategies in an attempt to speed up the recovery process, in addition to the standard recovery methods (i.e., sleep, rest and nutrition) (Tavares et al., 2017a). Within different recovery methods, the use of cold therapies, in particular cold water immersion (CWI), are the most investigated recovery strategies (Tavares et al., 2017a). The exposure to cold water decreases skin, core and muscle temperatures (White and Wells, 2013). This reduction in temperature leads to vasoconstriction, which in turn decreases the acute inflammation from muscle damage (Wilcock et al., 2006). Moreover, a reduction in nerve conduction properties and a decrease in muscle spasm and pain are also expected due to reduced tissue temperature (Wilcock et al., 2006). Lastly, the hydrostatic pressure-induced changes in blood flow may promote muscle metabolite removal, consequently improving metabolic recovery from intense exercise (Bishop et al., 2008; Ihsan et al., 2016). Given the high values of muscle damage arising from volleyball training, CWI would seem appropriate to be implemented when the recovery time between sessions is reduced (i.e., <24–48 h) (Tavares et al., 2017a). However, inflammation, muscle soreness and transient decreases in performance are considered an important part of the training and adaptation process (Bishop et al., 2008). This has led some researchers to suggest that post-exercise cold water immersion may blunt chronic adaptations by reducing muscle protein synthesis and therefore limiting muscle mass maintenance and growth (Roberts et al., 2015). Although some studies investigating the longer-term effects of CWI demonstrate a decrease in anabolic signaling (Roberts et al., 2015), recent research has demonstrated a positive effect on performance in cyclists chronically exposed to CWI during a 6-weeks training camp (Halson et al., 2014). Therefore, two main theories have been proposed for the use of CWI: (1) it enables athletes to perform subsequent training sessions with a greater overall training load); (2) it decreases adaptations to training due to a reduction in the anabolic pathways (Halson et al., 2014).

To the best of our knowledge, studies investigating the longer-term effects of CWI in a highly-trained athletic population are limited to two; one study performed in endurance-trained cyclists (Halson et al., 2014) and the other in professional rugby union athletes (Tavares et al., 2019a). In the cycling study, highly trained cyclists were exposed to CWI four times a week during a 21-days intensification phase followed by a 11-days taper. The authors found a likely beneficial effect of CWI between baseline and taper in the mean power decrease between two 4-min cycling bouts. A likely beneficial effect was also observed in the 1-s maximum mean sprint power in the CWI group when compared to the control. In the rugby study (Tavares et al., 2019a), 23 elite male rugby union athletes were randomized to either CWI (10 min at 10°C, $n = 10$) or a passive recovery control (CON, $n = 13$) during 3 weeks of high-volume training. Although no significant differences were observed between CWI and CON for any measure, CWI resulted in lower fatigue markers throughout the study as demonstrated by the *moderate* effects on muscle soreness ($d = 0.58$–0.91) and interleukin-6 ($d = -0.83$) and *small* effects ($d = 0.23$–0.38) on countermovement jump in comparison with CON. Therefore, based on these two previous studies and the promising results regarding some of the measures, it is clear that further research on the longer-term effects of CWI in highly-trained athletes is warranted.

Cold water immersion has been associated with enhanced recovery in various sports (Leeder et al., 2012). In volleyball, research investigating the effects of CWI is limited to one study (Freitas et al., 2017). In their study, Freitas et al. (2017) investigated the effect of CWI during one training week in professional volleyball athletes. Large positive effect sizes were observed between groups for the changes in countermovement jump (CMJ) performance, testosterone to cortisol ratio and IGF1, suggesting positive effects of CWI (Freitas et al., 2017). The fact that the study from Freitas et al. (2017) only lasted for 1 week, does not allow an understanding of what would have happened during subsequent training weeks. Indeed, longer term effects of CWI in these settings are yet to be established.

Therefore, the aims of the current study are twofold: to investigate the acute effects of CWI within the training week, and to investigate the longer-term effects of CWI during a 3-weeks period. Given previous literature on the effects of CWI, particularly the study performed in volleyball athletes from Freitas et al. (2017), we hypothesized that (1) CWI will enhance recovery during the training week in comparison to a control group; (2) The levels of accumulated fatigue will lead to an increase in perceptual and physiological markers of fatigue throughout the duration of the 3-week study, which will be attenuated in the CWI group.

MATERIALS AND METHODS

Participants

Fourteen highly-trained volleyball athletes representing the Portugal under-21 national team preparing for qualification to the World Championships, volunteered to participate in the current study (**Table 1**). Perceived muscle soreness, fatigue, total wellness scores, and neuromuscular performance were collected during a national training camp that lasted for 16 days (**Figure 1**). Training load was obtained from every training session. Performance measures were collected on day 1 and day 16. In order to be included in the study, participants were required to complete at least 90% of planned training sessions, and not miss more than two training sessions in a row. From the initial sample size ($n = 14$) one athlete failed to meet the inclusion criteria, resulting in 13 athletes completing the study (**Table 1**). Athletes were matched by positional group (Sheppard et al., 2007) and were randomly divided in one of two groups, a group exposed to 10 min of CWI at the temperature of 10°C with water to the level of the anterior superior iliac spine ($n = 7$; three outside, two middle, one setter, and one libero) and control group ($n = 6$; three outside, one middle, one setter, and one libero). In the experimental group, CWI was performed immediately after the last training session of each training day (totaling 12 CWI sessions). On nine occasions, CWI was performed after the PM training session, and on three occasions, it was following the AM training session. Written informed consent was obtained from each participant, and ethical approval was obtained from the Human Research Ethics Committee of the Institution.

Procedures

On day 1 and day 16 of the study, athletes were tested for lower and upper body power (**Figure 1**). During the first 2 weeks, perceptual and neuromuscular performance measures were obtained on day 1, 2, and 5 of each week, while on the last week data was collected on days 1 and 3 of that week (**Figure 1**). During the experimental period, all training session loads (court and gym-based sessions) from all participants were obtained from individual subjective rating of perceived exertion (RPE). Every athlete attended a meeting with the team nutritionist where they were instructed about meal composition and supplement use. Meal composition and supplement recommendations were equalized for every athlete, but food or diet recall surveys were not conducted. Fat mass was calculated from the three skinfold equation proposed by Evans et al. (2005).

TABLE 1 | Participant characteristics.

	CWI group ($n = 7$)	Control group ($n = 6$)
Age (years)	19.2 ± 0.8	19.0 ± 1.3
Height (cm)	188.1 ± 5.1	187.6 ± 5.6
Body weight (kg)	82.6 ± 8.9	77.4 ± 11.8
Fat mass (%)	10.1 ± 1.9	8.7 ± 0.7
Squat 1-RM (kg)	133.5 ± 20.8	129.2 ± 9.5
Bench Press 1-RM (kg)	72.1 ± 7.1	70.1 ± 10.7

Data shown as Means ± SD.

Training Program

During the first 2 weeks, the athletes performed 12 training sessions per week. For each of the first 2 weeks, moderate to high-load sessions occurred during day 1, day 2, and day 4, while day 3 and day 5 were low- and moderate-load sessions, respectively. During the last week, athletes had a decrease in training load, with a high-load training day on day 1 and low load training day on day 2. The resistance-training program was designed to increase power while maintaining maximum strength (i.e., six exercises of three sets of 6–8 reps with varied loads). There were a total of 29 training sessions (10 resistance training and 19 on-court sessions) throughout the study period.

Jump Performance

Countermovement jump (CMJ) and squat jump (SJ) tests were used to monitor responses to training. Both jump tests were performed on the morning of the first training day and last training day of the study. In addition, CMJ was performed on the morning of days 1, 2, and 5 of week 1 and 2, and days 1 and 3 of week 3. Following a standardized warm-up composed of dynamic stretches and movements (e.g., bodyweight squats, bodyweight CMJ's) athletes performed 3 CMJ with ∼5 s of interval between each jump. In the first and last training days, athletes perform 3 SJ prior to the three CMJ, with 2 min between jumping conditions. All jumps were performed on the top of a 42-cm contact mat (Chronojump-Boscosystem, Barcelona, Spain). The mat was connected to a microcomputer (Chronopic 3, Chronojump-Boscosystem, Barcelona, Spain), which was then connected to a PC via a USB port with analysis conducted by the manufacturers software (Chronojump-Boscosystem Software, Spain). The jumping height was estimated by means of flight time through a standardized kinematic equation $h = t^2 \cdot g/8$, where g is the gravity acceleration (9.81 m/s^2) (Bosco et al., 1983). For the CMJ, each trial started with the athletes standing in the top of the contact mat with their knees fully extended and the hands

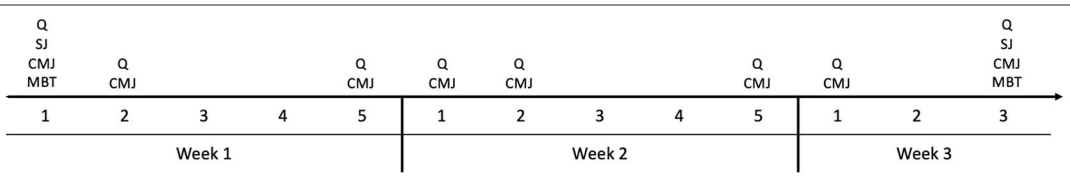


FIGURE 1 | Timeline of study design. Q, questionnaire; SJ, squat jump; CMJ, countermovement jump; MBT, medicine-ball throw.

on hips to eliminate the influence of arm swing (Tavares et al., 2017b). Athletes were then instructed to descend to a self-selected countermovement depth and to jump as high and quickly as possible. For the SJ, athletes were instructed to hold a self-selected position with shoulders aligned with the top, or in back, of knee level, as used during their normal training routines. Athletes were required to hold that position for 3 s before jumping as high as possible on the command "3, 2, 1, go" (Tavares et al., 2017b). All jumps were monitored by an experienced strength and conditioning coach and if any countermovement was observed, the trial was discarded and an additional trial was performed. The best trial for the SJ and CMJ, determined by the jumping height, was retained for later analysis. Jumping height calculated with this system was previously demonstrated to be valid and reliable (Pueo et al., 2016).

Seated Medicine Ball Throw

A seated 3-kg medicine ball throw (MBT) was used to measure upper body power. The athletes seated upright at 90° in a chair to facilitate the optimal trajectory and ensure standardization. Athletes performed a warm-up throw followed by two recorded attempts, with a 1-min rest between each attempt and the best distance was recorded. For each trial, the ready position was assumed with the subject placing the ball against their chest. Instructions were to throw maximally using a concentric only motion. Athletes had to maintain their back in contact with the chair, ensuring their feet remained on the floor. Each attempt was measured using a measuring tape taped to the floor and recorded in meters. A similar protocol has been described previously, and shown good test-retest reliability (ICC $>$ 0.97) (De Groot et al., 2012).

Perceptual Measures

A wellness questionnaire was completed by all participants on the morning of training days 1, 2, and 5 of the first 2 weeks and days 1 and 3 during the last week. The questionnaire was comprised of five questions and was designed to measure the general muscle soreness (1 = very sore, 5 = feeling great), perceived fatigue (1 = always tired, 5 = very fresh), sleep quality (1 = insomnia, 5 = very restful), stress levels (1 = highly stressed, 5 = very relaxed), and mood state (1 = highly annoyed/irritable/down, 5 = very positive mood) of athletes using a 1–5 Likert scale with 0.5-point increments (Tavares et al., 2017b). A total score for each individual was calculated from the average of the five items.

Training Load

The individual RPE for each resistance training and volleyball training session was obtained between 15 and 30 min following the completion of the session (Tavares et al., 2017b). The training load was then calculated as the product of the individual session RPE and the duration of the session using the following formula: Training load (sRPE) = RPE (1–10) × duration of the session (min) (Foster, 1998; Tavares et al., 2017b).

Statistical Analysis

The data collected was analyzed using a XLSTAT 19.02.43965 (AddinSoft, New York, NY, USA). Normality and sphericity assumptions were evaluated with the *Shapiro-Wilk* and *Mauchly's* test, respectively. An ANOVA for repeated measures was used to analyze differences between training days and baseline (day 1). *Post hoc* tests with a *Bonferroni* correction were performed to determine where significant differences were observed. If the repeated measures ANOVA assumptions were not met, or the values were presented on an ordinal scale (wellness questionnaires), a Friedman test was utilized. *Post hoc* analyses were performed using the *Dunn-Bonferroni* test or with the *Bonferroni* procedure according to Conover (2009), if a more conservative approach was deemed necessary. A paired sample *t*-test was used to compare pre- to post-differences in performance markers. From the raw data, changes from baseline for each training day for muscle soreness, perceptual fatigue, total wellness score and CMJ was determined for each athlete and an independent *t*-test or Mann-Whitney test was used to compare between-groups differences for each day. A significance level of $p < 0.05$ was implemented for all statistical tests.

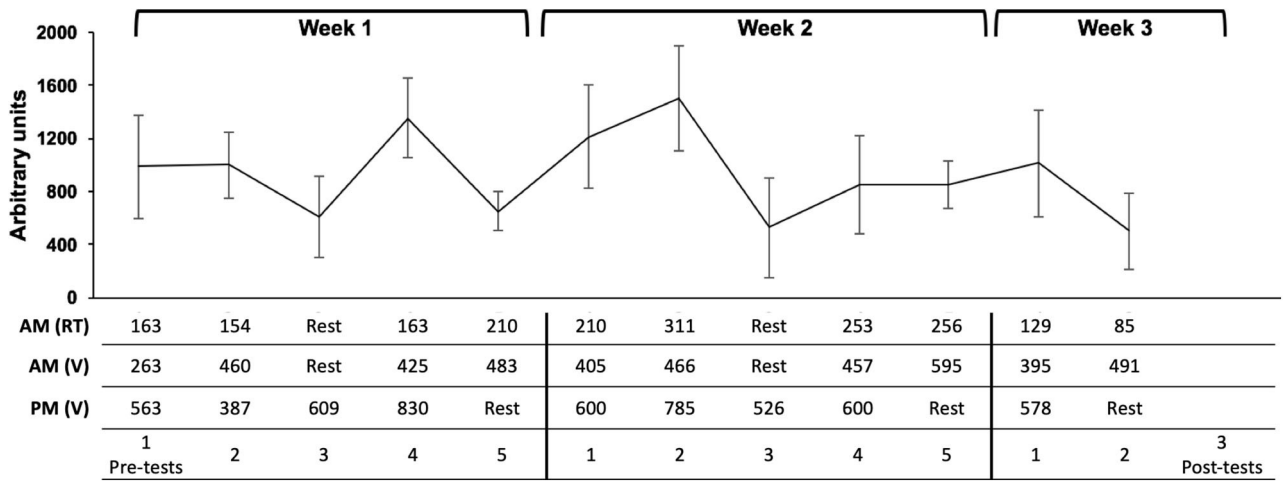
The effect sizes (Cohen's *d*) for pre to post-scores for SJ, CMJ and MBT were calculated to measure the difference in the Δ between CWI and CON using an excel spreadsheet (Hopkins, 2006). The same spreadsheet was used to calculate the interaction of the intervention over time for CMJ, wellness total score, fatigue and soreness (Hopkins, 2006). In addition, within group effect sizes were calculated for each variable of interest. Magnitudes of the standardized effects were interpreted using thresholds of 0.2, 0.6, 1.2, 2 and 4 for *small, moderate, large, very large, and extremely large,* respectively (Hopkins et al., 2009). An effect size of <0.2 was considered *trivial*. Where the 90% confidence limits overlapped *small* (±0.2) positive and negative values, the effect was deemed *unclear*.

RESULTS

No differences were observed between groups for the athletes' characteristics (**Table 1**; $p = 0.051$ to $p = 0.873$) or when individual sRPE for each training day were compared (**Figure 2**; $p = 0.070$ to $p = 0.697$).

Neuromuscular fatigue (from the CMJ) results can be observed in **Figure 3**. A significant decrease from baseline was observed for control group on the second training day of week two ($p = 0.049$). No differences were observed between groups for any time point ($p = 0.237$ to $p = 0.812$), however, *small* effect sizes were observed between CWI and control ($d = 0.52$) on day two of weeks one and two, favoring CWI.

The changes in soreness, fatigue and total wellness scores compared to baseline can be observed in **Figure 4**. Athletes in the control group were significantly more sore in comparison to baseline on days two and five of week one and two ($p = 0.001$ to $p = 0.009$). Athletes in the CWI group perceived a greater muscle soreness on day two of week one and day two and five of week two ($p = 0.005$ to $p = 0.038$) in comparison to baseline. Perceptual fatigue scores were significantly lower in comparison to baseline in the control group on day five of each week and for the CWI group on day five of week one ($p = 0.004$ to $p = 0.041$). The total wellness score was significantly decreased in comparison to baseline on the last training day of week two in both groups ($p = 0.005$ for control and CWI). Although differences were observed within groups, no significant differences were observed



AM (RT)	163	154	Rest	163	210	210	311	Rest	253	256	129	85	
AM (V)	263	460	Rest	425	483	405	466	Rest	457	595	395	491	
PM (V)	563	387	609	830	Rest	600	785	526	600	Rest	578	Rest	
	1 Pre-tests	2	3	4	5	1	2	3	4	5	1	2	3 Post-tests

FIGURE 2 | Figure represents mean ± SD daily sRPE (arbitrary units). The table represents the average of the session sRPE (arbitrary units) for each training session. RT, resistance-training session; V, Volleyball training session.

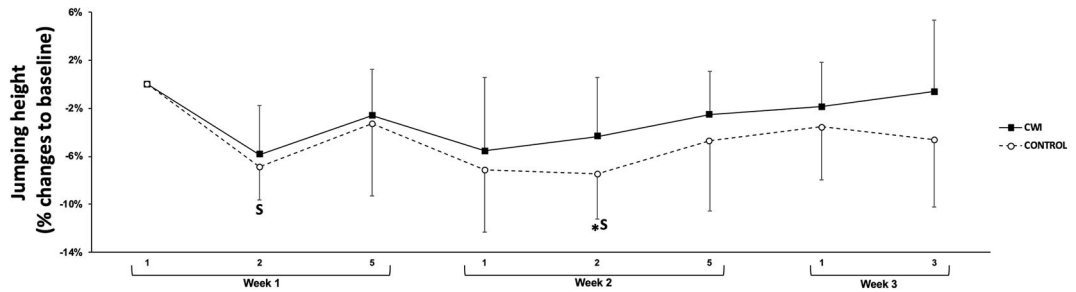


FIGURE 3 | Figure represents countermovement jump height changes to baseline ± SD across time points for CWI and Control groups. *Significant difference ($p < 0.05$) from baseline in the Control group. Effect size (*d*) for between group comparison: S, Small.

between groups for any training day ($p = 0.244$ to $p = 1.000$). In addition, *unclear* effect sizes were observed in the analysis of the interaction between groups and time for all wellness scores.

No significant differences were found for the CWI or control for the SJ (**Table 2**; CWI, $p = 0.309$, control, $p = 0.407$). Nevertheless, both groups decreased significantly in the MBT (**Table 2**; CWI, $p = 0.005$; control, $p = 0.001$). In the CMJ, while the control group decreased significantly ($p = 0.05$), no differences were observed for the CWI group ($p = 0.408$). When the differences of the pre- to post-changes between groups were compared, no significant differences were found for any test ($p = 0.212$ to $p = 0.976$). Nevertheless, *moderate* effect sizes ($d = 0.65$) were found for the CMJ jump between the CWI and control in favor of CWI (**Table 2**).

DISCUSSION

The results from this study are in partial agreement with our original hypothesis which stated that: (1) cold-water immersion would enhance recovery from volleyball training by reducing fatigue levels within the training week; (2) the levels of accumulated fatigue will lead to an increase in perceptual and physiological markers of fatigue, that are attenuated by the use of CWI. The key findings of the study showed that CWI seems to provide little benefit to recovery within the training week. In the longer-term, there was a trend toward a benefit when using CWI in highly-trained volleyball athletes.

In order to understand the acute effects of CWI, results from the first week were analyzed. Both groups significantly increased muscle soreness from day one to day two, however, only muscle soreness in the control group remained elevated on day five (**Figure 4**). Given the associations between increases in muscle soreness and muscle damage and decreases in muscle function, an increase in fatigue (i.e., perceptual fatigue and neuromuscular performance) was expected (Leeder et al., 2012; Pointon and Duffield, 2012). Although there was a decrease in CMJ between days two (CWI: −5.8 ± 4.0%; control: −6.9% ± 2.8%) and five of week one (CWI: −2.6 ± 3.8%; control: −3.2% ± 6.0%) to baseline, these differences did not reach statistical significance (**Figure 3**). Nevertheless, a significant decrease from baseline was observed on day five of week one for perceptual fatigue in both groups (**Figure 4**). This is not surprising as perceptual fatigue is likely to reflect the effect of full body training load rather than only lower body (Tavares et al., 2017b). Although upper body performance was not monitored during the training weeks, the

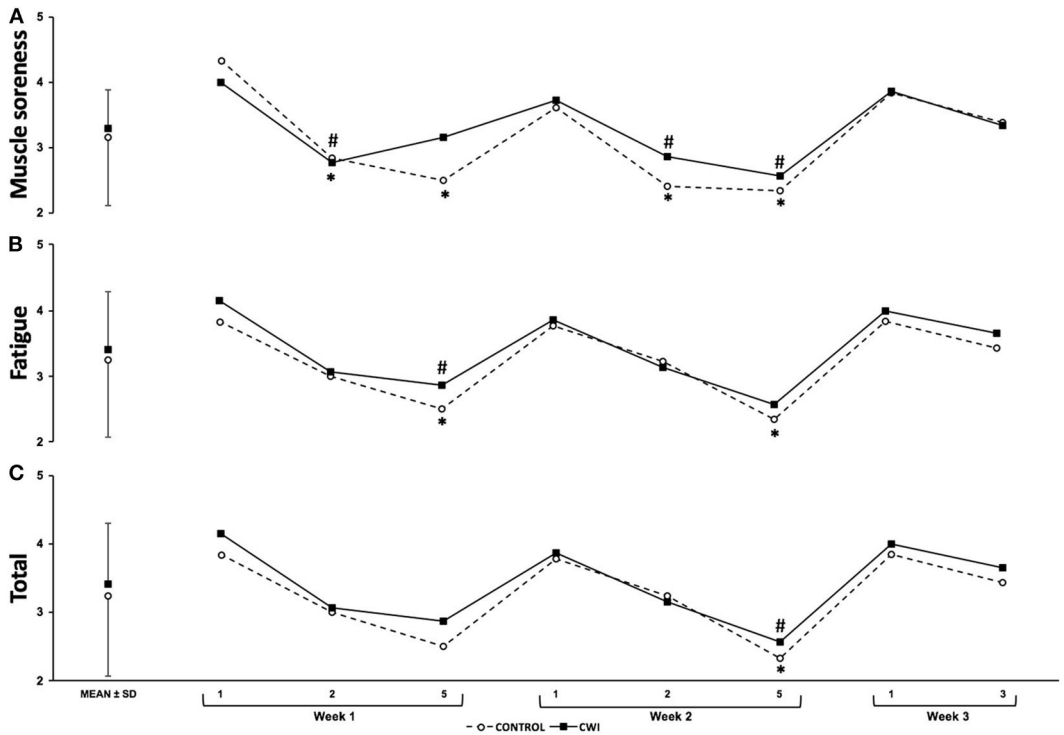


FIGURE 4 | Mean ± SD wellness scores at different time points for CWI and Control groups. **(A)** Soreness; **(B)** Fatigue; **(C)** Total *Significant difference ($p < 0.05$) from baseline in the Control group. #Significant difference ($p < 0.05$) from baseline in the CWI group.

TABLE 2 | Mean ± SD of performance measures in the CWI and Control groups at different time points, and average of individual change (%) between pre- to post-training.

		Pre-training	Post-training	%Δ Post-pre	ΔControl–ΔCWI (Effect Size–*d*)
SJ (cm)	CWI	41.8 ± 2.6	41.0 ± 3.1	−1.6 ± 9.4	−0.9 ± 3.3
	Control	43.0 ± 1.5	43.1 ± 3.6	0.2 ± 5.7	−0.30; *Unclear*
CMJ (cm)	CWI	44.2 ± 1.6	43.9 ± 3.3	−0.6 ± 6.0	2.6 ± 2.7
	Control	49.7 ± 3.4	46.8 ± 4.1*	−5.8 ± 5.6	0.65; *Moderate*
Med ball throw (m)	CWI	6.9 ± 0.3	6.0 ± 0.4*	−13.0 ± 7.3	−0.2 ± 0.5
	Control	6.6 ± 0.6	6.0 ± 0.7*	−10.4 ± 4.1	−0.41; *Unclear*

*Significant difference ($p < 0.05$) from pre-training.

pre- to post-changes observed in medicine ball throw (−12.8 ± 5.8%) support our proposition that perceptual fatigue was significantly higher on day five because of the combination of lower and upper body load.

When the groups were compared, no differences (i.e., individual changes from baseline) in muscle soreness or fatigue were observed between groups on week one (**Figures 3**, **4**). Cold water immersion has been associated with a decrease in muscle soreness and muscle damage markers in various team sports (Tavares et al., 2017a). The high volume of jumps performed in volleyball could potentially result in high values of muscle soreness and fatigue, which may supposedly be attenuated by CWI (Polglaze and Dawson, 1992). In the study from Freitas et al. (Freitas et al., 2017), the authors reported a *small* beneficial effect of CWI on muscle soreness. In addition, *moderate* to *large* effect sizes were observed in biochemical markers measuring endocrine responses, muscle damage and inflammation between groups on the post- to pre-training changes, suggesting a beneficial effect of CWI (Freitas et al., 2017). In agreement with a recent meta-analysis (Leeder et al., 2012), these increases in muscle damage and soreness lead too *small* to *large* effect sizes for CWI enhancing recovery in the Freitas et al. study (Freitas et al., 2017). In the present study, an enhancement in recovery measured by muscle soreness and fatigue (perceptual and physiological) in the group exposed to CWI was expected but not observed. The lack of recorded jumping volumes during training limits our ability to understand if the jumping volume was below the usual expected load, resulting in lower muscle damage and soreness and limiting the effects of CWI. In addition, a low volume, high velocity resistance training programme was predominately implemented during the period of the study. Higher strength training loads are associated with greater muscle disruption (Schoenfeld, 2010)

and consequently, higher levels of muscle soreness (MacIntyre et al., 1995), therefore, the limited volume in comparison to other training phases on this type of training may also have limited increases in muscle soreness. Lastly, the fact that muscle soreness was obtained from a single question may mask instances of specific soreness within a particular muscle (e.g., lower body) (Tavares et al., 2017b).

In order to understand the longer-term effects of CWI, perceptual measures and CMJ's were monitored during the second and third weeks. In addition, pre- to post-changes in performance were compared within and between intervention groups. Similar to week one, no significant differences were observed between groups for any of the perceptual measures or for CMJ performance on any of the training days (**Figures 2**, **3**). Nevertheless, in week two, perceptual fatigue and CMJ performance were significantly decreased only in the control group with no significant changes in the CWI group. Moreover, a *small* effect size was observed when comparing the effect of CWI and control groups for CMJ on day two, in favor of the CWI group. This effect of CWI reducing fatigue is further supported by the significant decrease in the pre- to post-CMJ changes observed in the control group but not in the CWI group. In addition, a *moderate* effect size was observed when pre- to post-changes on CMJ were compared between groups (**Table 2**). The fact that perceptual and neuromuscular fatigue levels were lower in the CWI group, together with the pre- to post-changes in CMJ performance, may demonstrate the efficacy of CWI in reducing the accumulated effects of fatigue. Our results are supported by previous research demonstrating the effects of cold modalities enhancing recovery over prolonged training periods (i.e., 33 days) (Halson et al., 2014). Similarly, the aforementioned study found a beneficial effect of CWI on power output in a highly-trained cyclists (Halson et al., 2014). Research exploring the effects of CWI on performance and perceptual markers of fatigue in team-sport athletes is limited making it difficult to compare our results (Higgins et al., 2012). Although no differences were observed in performance markers between the CWI and control groups obtained from two rugby-specific simulated games separated by 1 week, Higgins et al. (2012) found a trend for beneficial effects of CWI.

As mentioned, previous research has suggested that CWI used in a chronic setting may lead to the blunting of important adaptations, especially in strength and power based sports. For example, Roberts et al. (2015) observed a decrease in the activity of the mammalian target of rapamycin (mTOR) pathway and satellite cells after 10 min of CWI at ∼10°C two times a week after resistance training. However, the characteristics of the subjects (recreationally trained) were different from participants in the current study and training load was fixed (e.g., load lifted) so subjects were not allowed to lift heavier even if they felt more *fresh*. Moreover, the training load used in the Roberts study (2× training sessions per week, compared to 12 sessions a week in the current study) potentially allowed for full recovery between sessions, limiting the rational for the inclusion of cold modalities. The differences in methodologies used (e.g., participants, training frequency, intensity, and duration of CWI) make it difficult to compare between studies. Therefore, we would suggest that further research is needed to provide practical applications to highly-trained team sport athletes, and that understanding the intensity of the training, the density of the week, the athletes' individual goals and the requirements during the season will provide the rational for the implementation of CWI (Tavares et al., 2019b).

Future studies investigating the chronic effect of CWI exposure should monitor training over a longer duration (e.g., >4 weeks) in highly-trained athletes, with high training frequency/load. In addition, given that in volleyball, muscle soreness, damage, and fatigue are likely to be associated with the stretch-shortening cycle activities (e.g., jumps and spikes), these activities should be quantified. Inclusion of tests and questionnaires monitoring upper body neuromuscular performance and soreness may provide important information when exploring the effects of CWI (Tavares et al., 2017b). Finally, while perceptual and mechanical data can provide some information of fatigue and wellness, biochemical measures such as the ones used in the study from Freitas et al. (2017) should be included in future research. This would provide an insight into the mechanisms leading to fatigue and enhance understanding of the effect of CWI in a period where fatigue may be accumulated. We acknowledge that a potential limitation in the current study was the order of tests performed pre and post the study period. The CMJ testing was performed following the SJ testing pre and post study, but not during the training weeks. There is a chance this may have either had a positive effect (e.g., via post-activation potentiation) or a negative effect (e.g., via fatigue) on CMJ performance. However, given the low volume of jumps (3 SJ's) and the time between types of jumps (2 min), we feel it is unlikely to have had a significant impact, and it was also the same for both groups.

Cold water immersion has been shown to enhance recovery (e.g., neuromuscular performance) during periods of accumulated levels of fatigue (Higgins et al., 2012; Halson et al., 2014). In the current study, the effects of CWI were restricted to a *small* effect on CMJ during week 2 and a *moderate* effect on pre to post-CMJ performance. It is however important to mention that the short duration (two and a half weeks) and the fact that training load on week three (i.e., only 2 days) was considerably lower than week one and two, which may have negated a more pronounced effect of CWI on the markers of perceptual and neuromuscular fatigue, soreness and wellness (**Figures 2**–**4**). In conclusion, CWI seems to provide little benefit to recovery within the training week. In the longer term, there was a trend toward a benefit when using CWI in highly-trained volleyball athletes. Further research is needed on the use of CWI in highly-trained athletes, implementing longer time-frames and further mechanistic measures to understand what is happening and the muscular level.

DATA AVAILABILITY STATEMENT

The original contributions generated for the study are included in the article/supplementary material, further inquiries can be directed to the corresponding author.

ETHICS STATEMENT

The studies involving human participants were reviewed and approved by Human Research Ethics Committee, University of Waikato. The patients/participants provided their written informed consent to participate in this study.

AUTHOR CONTRIBUTIONS

FT, MS, and BM contributed to the design, data collection, data analysis, and writing of the final paper. MD and TS contributed to the design, data analysis and writing of the final paper. All authors contributed to the article and approved the submitted version.

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Cold Water Immersion After a Handball Training Session: The Relationship Between Physical Data and Sensorial Experience

Maxime L'Hermette[1], Ingrid Castres[1], Jeremy Coquart[1], Montassar Tabben[2], Nihel Ghoul[1], Bernard Andrieu[3] and Claire Tourny[1]**

[1] University of Rouen UFR STAPS, Cetaps EA, Rouen, France, [2] Aspetar Orthopaedic and Sports Medicine Hospital, Doha, Qatar, [3] University of Paris Descartes, Paris, France

The aim of this study was to examine the relationship between the physiological data from subjects and their reported sensory experiences during two types of recovery methods following a handball training session. Female handball players (average age: 21.4 ± 1.3 years; weight: 59.2 ± 3.3 kg; height: 158 ± 3 cm; body mass index, 23.4 ± 2.0 $kg.m^{-2}$) carried out an athletic training session (rating of perceived exertion RPE: 14.70 ± 0.89) with either a passive recovery (PR) period or cold water immersion (CWI) for 14 min) (cross-over design). Physiological data were collected during the recovery period: CWI had a greater effect than PR on heart rate (HR; bpm), the higher frequencies (HF) of heart rate variability (HRV: 46.44 ± 21.50 vs. 24.12 ± 17.62), delayed onset muscle soreness (DOMS: 1.37 ± 0.51 vs. 2.12 ± 1.25), and various reported emotional sensations. Spectrum HRV analysis showed a significant increase in HF during CWI. Sensorial experiences during the recovery periods were gathered from verbatim reports 24 h later. Players' comments about CWI revealed a congruence between the physiological data and sensorial reports. They used words such as: "*thermal shock*," "*regeneration*," "*resourcefulness*," "*dynamism*," and "*disappearance of pain*" to describe their sensations. In conclusion, this study demonstrated the link between physiological and experiential data during CWI and we propose that action of the parasympathetic system on the autonomic nervous system can, at least in part, explain the observed correlations between the corporeal data measured and the sensorial experiences reported.

Keywords: athletic recovery, handball, physiological data, sensations, cold water immersion, verbatim reports, experiential data

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Juiz de Fora Federal University, Brazil

****Correspondence:***
Maxime L'Hermette
maxime.lhermette@univ-rouen.fr
Claire Tourny
claire.tourny@univ-rouen.fr

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INTRODUCTION

Athletes often experience high levels of fatigue due to high training-loads, as well as frequent competitions (Hausswirth et al., 2011; Versey et al., 2013). Fatigue is multi-dimensional and related to factors such as psychological tension or mild inflammatory disorders (Versey et al., 2013). For the purposes of this study, fatigue is defined as a feeling of exhaustion with a decline in physical performance (Montgomery et al., 2008).

One method to limit the effects of fatigue on performance is cold water immersion (CWI). CWI is an established method of recovery for athletes who carry out intermittent sports (Montgomery et al., 2008; Rowsell et al., 2009; Ascensao et al., 2011).

We wished to investigate the relationship between the corporeal (i.e., physiological data) and sensorial experiences (i.e., described from verbatim descriptions) of handball players during CWI following a training session. The sensorial data was evaluated using a method previously described by Andrieu and Gerardin (2012) called "emersiology" where "emersiology" was defined as "*a reflexive science dealing with the emersion of sentient life from the consciously experienced body*" where "*emersion is the involuntary movement, within our bodies, of connections, humors and images of which only the tip reaches our awareness*" (Andrieu and Gerardin, 2012; Andrieu and Burel, 2014).

In traditional, Western medicine, the corporeal or living body is evaluated by physiological parameters such as delayed onset muscle soreness (DOMS), the subjective intensity of the training session (rating of perceived exertion, RPE), and heart rate variability (HRV) which is the variation in the time interval between heartbeats (Haddad et al., 2013). HRV provides a non-invasive evaluation of autonomic control of the heart (Buchheit et al., 2009) and is sensitive to factors such as fatigue, physiological and psychological stress, and has also been shown to be related to respiratory activity. Respiration affects HRV, producing low frequency (LF) and high frequency (HF), respectively, above and below the breathing frequency (BF) threshold of 0.15 Hz. Fluctuations above 0.15 Hz are associated with vagal activity, whereas fluctuations between 0.04 and 0.15 Hz have been reported to be mediated by both vagal and sympathetic cardiac nerves (Perini and Veicsteinas, 2003; Saboul et al., 2014).

CWI is used to accelerate recovery so that performance can be restored to normal as soon as possible (Montgomery et al., 2008; DeMartini et al., 2011; Bastos et al., 2012; Versey et al., 2013). The effects of CWI are greater than active recovery because of the effects of hydrostatic pressure and the water temperature (Barnett, 2006; Versey et al., 2013). CWI has been shown to increase blood flow and decrease blood lactate concentrations. This reduces fatigue and enables higher training loads to be sustained (Pastene et al., 1996). The CWI protocols described in the literature describe the immersion of either the lower limbs or the whole body, and the temperatures used vary from 10 to 15°C (Montgomery et al., 2008; Buchheit et al., 2009; Ascensao et al., 2011; Bastos et al., 2012; Stanley et al., 2012; Versey et al., 2013). The time of immersion also varies from 3 to 20 min, and involves either one continuous immersion or several immersions, each of 1–5 min with 1–2 min in between (Bailey et al., 2007; Hausswirth et al., 2010; Parouty et al., 2010; Ascensao et al., 2011; Bleakley et al., 2012; Elias et al., 2013).

To our knowledge, none of these previous studies have examined the use of CWI in intermittent sports on both athletes' physical or corporeal performance and on their sensorial experiences. Thus, the purpose of this study was to analyse the rapport between the corporeal and sensorial experienced bodies after CWI. We used passive recovery (PR) data as the control and hypothesized that CWI would: (1) improve corporeal performance recovery data compared to PR after one handball training session as measured by physiological parameters and (2) receive more positive reports from subjects regarding their sensorial experiences as evaluated by the "emersiology" method.

MATERIALS AND METHODS

Subjects

Eight healthy female handball players (age = 21.4 ± 1.3 years; body mass: 59.2 ± 3.3 kg; height: 158 ± 3 cm; body mass index: 23.4 ± 2.0 $kg.m^{-2}$) from the French Handball First Division volunteered to take part in this study. No subjects had medical contraindications to CWI (e.g., Raynaud's syndrome or cold hypersensitivity), and all used an oral contraceptive.

All subjects signed an informed consent form before participation. The protocol was fully approved by the local scientific committee and the study was performed in accordance with the Declaration of Helsinki ethical standards.

Procedure

The study was carried out over 2 days (day 1 = D1; day 2 = D2) 1 week apart. The same protocol was used on both D1 and D2 and it was composed of 5 steps, as shown in **Figure 1**.

Subjects' heart rate was recorded throughout by a zephyr belt and HRV was recorded over 10 min with subjects lying prone on a mat on the floor with their eyes closed. DOMS was evaluated throughout the recovery periods using hooper's scale by moving a cursor along a 7-cm horizontal line and graded according to the following: 1 = no pain, 2 = very slight tiredness, 3 = slight tiredness, 4 = tender but not sore, 5 = slightly sore, 6 = sore, 7 = very sore (Haddad et al., 2013). Corporeal fatigue was evaluated by using a RPE, graded using a borg scale from 6 to 20 (Foster et al., 2001).

The training session began with a 25-min warm-up followed by 4 moderate-to-vigorous interval training exercises (20" /20") with ball-handling or information-decision constraints: each exercise lasted 8 min and was associated with 3 min of recovery (the subjects walked at spontaneous speed around the field). The fifth and final exercise consisted of 12 min of a full handball game without any time-out (the ball was backed into play by the goalkeeper). The training session thus lasted 1 h and 21 min in total.

For the recovery, subjects were randomly divided into 2 groups (group 1 = G1; group 2 = G2). Both groups underwent both types of recovery but in opposite order, i.e., D1/G1 = PR and D1/G2 = CWI and then on D2 /G1 = CWI and D2/G2 = PR.

CWI involved plunging the subject into water up to their waist for 14 min. The water temperature was 12°C and was controlled by a digital thermometer (HI 98509; Checktemp®, Australia) and re-adjusted with ice if necessary. The atmospheric pressure and temperature of the room were measured using a desktop weather station (EW93, Oregon Scientific, USA).

PR involved reclining quietly on a chair for 14 min.

HR and HRV were measured at 1, 3, 5, 9, and 14 min after CWI or PR began. Sensorial data were gathered 24 h later when subjects were interviewed and asked about their perception of the recovery experience.

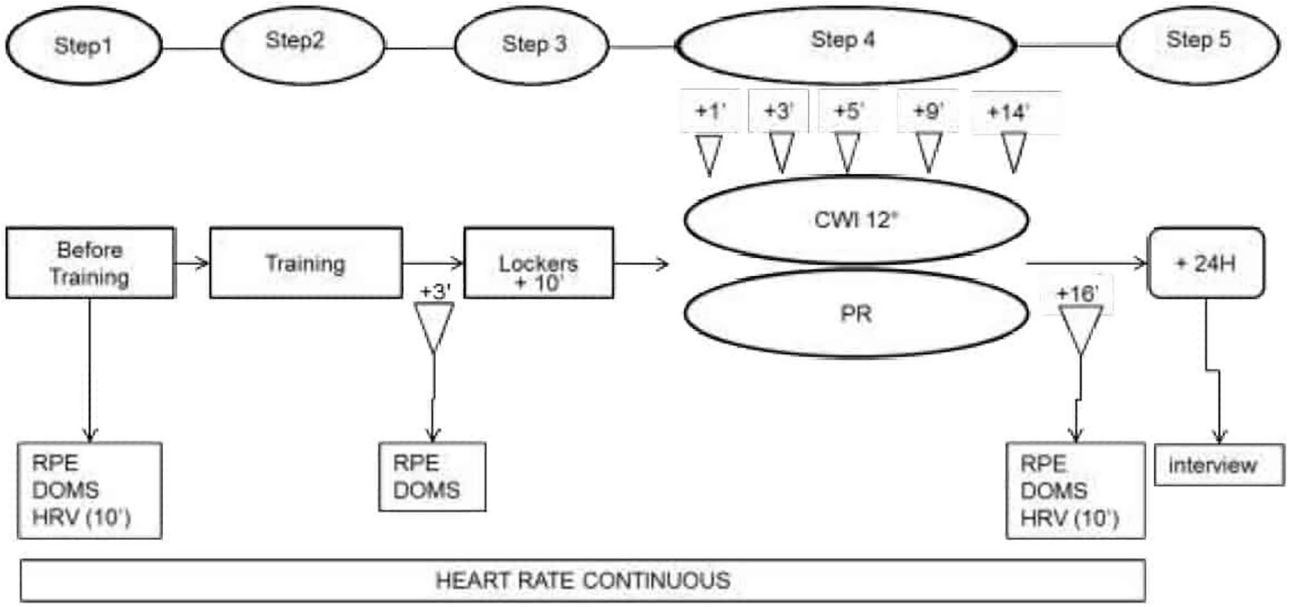

FIGURE 1 | Experimental procedure. RPE, Ratings of Perceived Exertion; DOMS, Delayed Onset Muscle Soreness; HRV, Heart Rate Variability; CWI, Cold Water Immersion; PR, Passive Recovery. The interview was to gather sensorial data.

HR and HRV Measurements

All subjects were equipped with a Zephyr Physiological Status Monitoring Training Ecoteam® chest belt (Zephyr Performance Systems, Annapolis, Maryland, USA) throughout the procedure. The sampling rate was 1,000-Hz. Data were downloaded to the Zephyr software unit which visualized the HR trace and allowed extraction of a cardiac period (R-R interval) file in ".txt" format that was subsequently analyzed by HRV Analysis software (Kubios HRV, Biomedical Signal Analysis Group, Department of Applied Physics, University of Kuopio, Finland).

HRV was estimated by time-domain parameters (the root mean square difference of successive normal R-R intervals (RMSSD), the percentage of pairs of adjacent RR intervals that differed by >50 ms (pNN50) and frequency-domain analysis (HF, LF). For each subject, HRV was calculated from the last 5 min of their 10-min HR data, R-R intervals were extracted and beat-to-beat analysis was carried out. These parameters have classically been used to study autonomic nervous system (ANS) modulation by parasympathetic activity (Buchheit et al., 2009; Stanley et al., 2013). HRV analysis was restricted to indices of parasympathetic reactivation: the RMSSD, the percentage of successive R-R differences >50 ms (pNN50). The presence of high frequency HR has previously been shown to indicate influence of the parasympathetic branch of the ANS, since other work has demonstrated that high frequencies (0.15–0.50 Hz) are caused by vagal nerve activity which no longer occurs following administration of atropine (which blocks the effects of acetylcholine, a neurotransmitter released by the parasympathetic nervous system (PNS).

Improvements in athletic performance have been found to be closely correlated with an increase in spectrum power of the HFs in a lying position (Schmitt et al., 2013). Low frequencies, by contrast, have mostly been shown to reflect sympathetic activity. These two control systems are complimentary (Souza Neto et al., 2003). The LF/HF ratio calculated from these two spectral densities thus represents the vago-sympathetic balance of the ANS and this approach is used to distinguish the dominant spectral band. In athletes this ratio is <1 during warm-up and training conditions and is >1 during periods of high fatigue (Saboul et al., 2013). The ratio is, however, modulated by respiratory frequency, which is why HRV measurements also need to be made during rest (Saboul et al., 2014) and to facilitate this, HRV recordings were carried out in a quiet room in an attempt to minimize or avoid fluctuations in HR.

Statistical Analysis

Normality of the data was confirmed using a Shapiro-Wilks test and a paired *t*-test was carried out for each recovery condition independently for the dependent variables: DOMS and HR (both before and after training) and HR (during recovery at 1, 3, 5, 9, and 14 min). A log transformation was applied to the HRV data from the RR interval to reduce bias from any non-uniform inter-individual value. Additionally, the differences were specified using a magnitude–based Cohen's effect size (ES) presented in **Table 1**. The ES was assessed using the following criteria: <0.2 = trivial, 0.2–0.6 = small, 279 >0.6–1.2 = moderate, >1.2–2.0 = large, and >2.0 very large differences (Hopkins, 2000).

RESULTS

The mean HR (bpm) was measured during both training sessions and no significant differences were found (trivial or small ES). The average HR during training on day 1 was 80.3 ± 17.8% HRmax and was 77.24 ± 17.2% HRmax on day 2. A further

TABLE 1 | Comparison heart rate variabilities (HRV) and delayed onset muscle soreness (DOMS) scores in female handball players following passive recovery (PR) or cold-water immersion (CWI) treatment post-exertion.

		CWI	PR	ES
DOMS	DOMS +16 min	1.37 ± 0.517***	2.12 ± 1.25	0.77
	HF	46.44 ± 21.50***	24.12 ± 17.62	1.13
	LF	53.37 ± 21.61***	75.69 ± 17.65	1.13
HRV	LF/HF	5.76 ± 4.921**	1.812 ± 1.701	1.07
	RMSSD	3.692 ± 0.726	3.396 ± 0.934	0.35
	pNN50	2.523 ± 1.29	2.2510 ± 1.37	0.20

ES, Effect size.
*Levels of statistical significance are indicated where **$p < 0.01$, ***$p < 0.001$.*

TABLE 2 | Fatigue as measured by rating of perceived exertion (RPE) scale of 6 to 20 and heart rate (HR) values (bpm) measured post-training (postT) and post-recovery (postR) of eight female handball players.

Condition	RPE_{postT}	RPE_{postR}	HR_{postT}	HR_{postR}
PR	14.5 ± 3.5 NS	9 ± 3.4 NS	165.29 ± 5.2 NS	88.71 ± 13.1 NS
CWI	13.5 ± 3.0	8.5 ± 2.7	165.71 ± 6.9	85.8 ± 9.0
ES	0.30	0.16	0.06	0.25

NS indicates no significant difference ($p > 0.05$).
PR, passive recovery; CWI, cold water immersion; ES, effect size.

breakdown of HR post-training and post-recovery data are shown with an index of fatigue (RPE scale) in **Table 2**.

The results of the parameters measured during both recovery conditions are shown in **Figure 2** and **Table 1**, including the ES.

There was a significant difference in HR values during the recovery period; HR was significantly higher during CW1 at 1, 3, 5, 9, and 14 min compared with PR.

The HR individual's responses of the subjects to the training session (HRts) and to the CWI are presented in **Figure 3**. This figure highlights that the subjects have all responded to the cold shock in increasing their HR (maximal value is reached at HR1 or HR3).

At the end of the recovery periods, DOMS was significantly lower after CWI than after PR (moderate ES). Spectrum analysis of HRV showed that the number of HFs was significantly increased during CWI than during PR ($p < 0.001$; large ES), a change that was also associated with a decrease in LF ($p < 0.001$; large ES). The LF/HF ratio was thus significantly greater after CWI than after PR (moderate ES). No significant differences were found in variables measured by frequency analysis (RMSSD, pNN50) between CWI and PR (small ES).

DISCUSSION

This study compared the effects of CWI and PR as recovery methods for athletes following a period of intense intermittent exercise (a handball training session and match). No difference was found in HR data at the end of both types of recovery, nor in levels of reported fatigue (as measured by RPE score). The absence of differences in these two variables is important because if either parameter had significantly decreased in the period between training and recovery, it would have been impossible to ascribe any benefits reported to either type of recovery procedure with any certainty.

HRV has been shown to be a sensitive measure of cardiac activity adaptation by the ANS and is evaluated by both frequency and spectrum parameters. We found no difference in the frequency parameters of the HRV as identified by the RMMSD and pNN50 between the two recovery conditions.

With regards to the spectral HRV parameters, however, a significant increase in HF values was found for data from the CWI recovery which was accompanied by a significant decrease in LF values. These changes corroborate the strong action of the PNS on vagal inhibition during the CWI while the changes in the LF data and the LF/HF ratio suggest a concomitant reduction in sympathetic activity (Buchheit et al., 2009). One effect of PNS stimulation is the secretion of growth hormones which are involved in repair and regeneration of muscles (Stanley et al., 2013; Devesa et al., 2016). We believe that the results of this study therefore demonstrate the effectiveness of CWI on this aspect of the recovery process.

Furthermore, studies have also shown that stress and anxiety reduce the activity of the PNS and so increase sympathetic nervous system activity (Dishman et al., 2000; Shaikh al arab et al., 2012), a fact which we believe increased the importance of the increase in the number of HRV LF after CWI (Buchheit et al., 2009). This decrease in LF could also have been related to the reported feelings of peace and well-being that were described by the subjects. We propose that CWI PNS-stimulation could have been involved in the production of these sensorial experiences of new-found energy and renewed vigor.

DOMS scores decreased significantly after CWI in comparison to the scores recorded after PR. This effect, which was followed over a 14-min recovery period, is in accordance with the statement of Dupuy et al. (2018): an explanation of the impact of CWI on DOMS is a reduction in exercice-induced inflammation and muscle damage. The level of immersion and the cold temperature of the water may reduce the formation of oedema and pain sensation (Montgomery et al., 2008; Versey et al., 2013).

HR was significantly higher throughout the 14 min of CWI, compared with 14 min of PR. This may have been due to the thermal shock- or cold shock - following immersion of the lower limbs and pelvis in the cold water, which is characterized by an inspiratory gasp, hyperventilation and increased HR (Dupuy et al., 2018). Moreover, the subjects were not familiarized to CWI which improved the physiological response (Castellani and Tipton, 2015).

The whole body (including the face) submersion in cold water and the cold shock habituation cause bradycardia which reduces the arterial hypertension caused by the sudden flow of blood from the peripheral to the core vasculature in response to the low temperature (Castellani and Tipton, 2015; Park et al., 2018). In our study, only the legs and pelvis were immersed, and so it seems likely that peripheral vasoconstriction only occurred in these lower regions. In addition to hyperventilation, we propose these

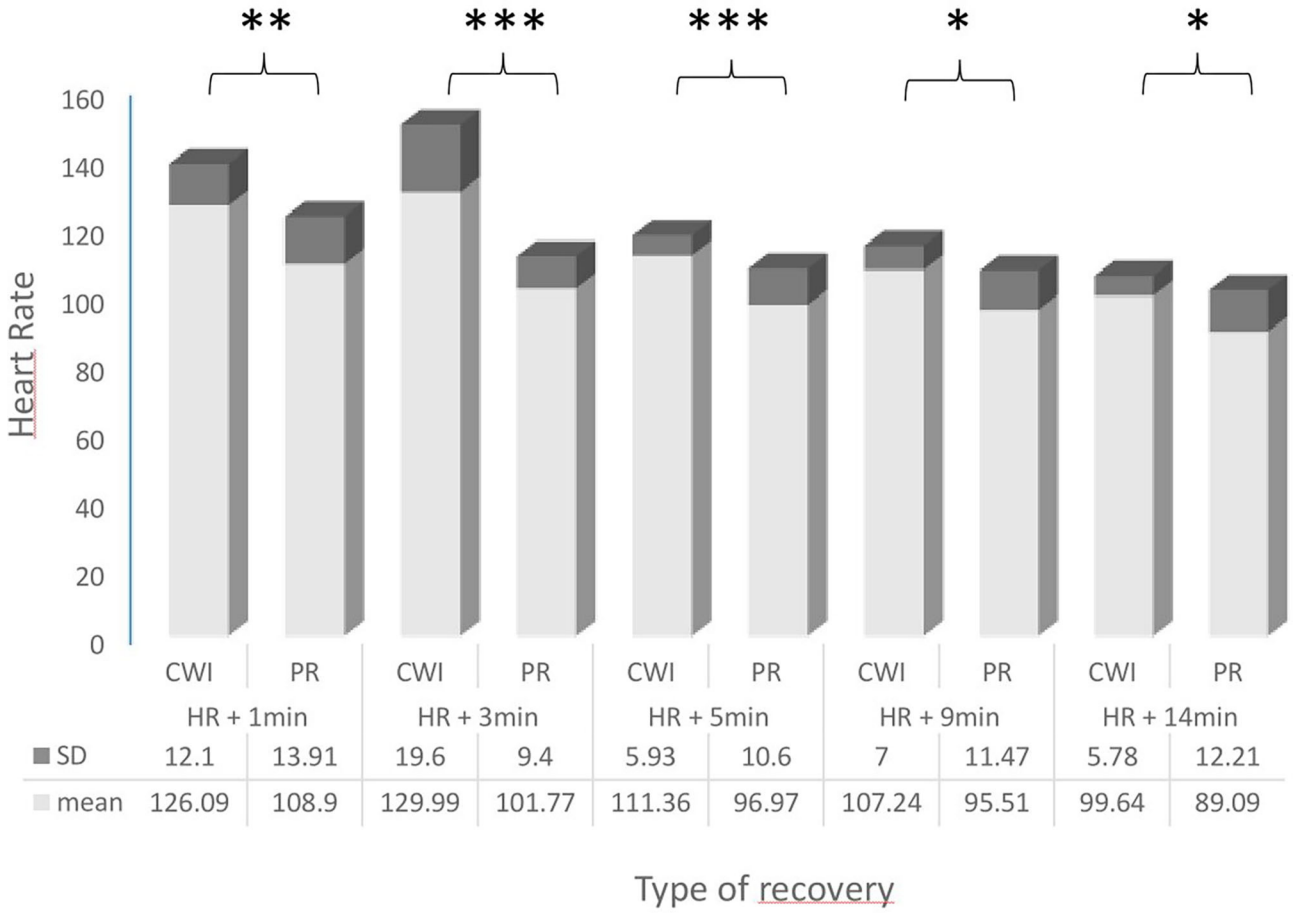

FIGURE 2 | Comparison of heart rates (HR; bpm) in female handball players following passive recovery (PR) or cold-water immersion (CWI) treatment post-exertion. CWI, Cold Water Immersion; PR, Passive Recovery. Levels of statistical significance are indicated where $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

factors caused the observed increase HR rather than the decrease that would have been expected with total body immersion. Indeed, respiration is known to be an important regulator of cardiac rhythm; inhalation temporarily inhibits the influence of the PNS and accelerates cardiac rhythm (Bleakley and Davison, 2010; Stanley et al., 2013).

The subjects also reported a strong emotional experience with CWI. Players described a varied range of different sensations over the 14-min of CWI from the initial discomfort and then on throughout the recovery and acclimatization which represents the common reaction after the cold shock (Castellani and Tipton, 2015; Bouzigon et al., 2018). Subject described sensorial experiences included their initial shock upon first entering the water and then how their first 3 min in the cold was associated with unprecedented and intense sensations that they felt they could not immediately control. The variability between individuals in their thermoregulatory responses during cold exposure should be attributable to anthropometric differences (Bahnert et al., 2013; Castellani and Tipton, 2015). The cold creates a sensation of pain and discomfort which is due at least in part to the muscle tension and dizziness caused by hypotension following peripheral vasoconstriction (Bouzigon et al., 2018). The intensity of these painful sensations reduced over time, producing an analgesic effect and sense of well-being post-immersion (Elias et al., 2013; Park et al., 2018). The reduction in pain was progressive and subjects reported that it took them at least 3 min to acclimatize to the CWI, and around 5 min for their feelings of well-being to set-in: "*from the fifth minute, we no longer felt the cold as strongly as when we first entered the water and we had a good time, we laughed, it was more than relaxation*." One player described how the CWI "*became bearable after 5 minutes, it was less painful, but I was not comfortable, I felt the cold, I got used to it I think. As if it paralysed my toes. I got thermal shock first, then I got used to it*."

Another player described how, during the period of adaptation she "*…was quite cold from the 3rd minute,* [but] *by the 9th minute, I had got used to the water, my muscles relaxed*." Subjects described how they perceived the sensations of being in the CWI as more than just the feeling of their muscles relaxing but that benefits were felt upon leaving the CWI: "*When I started to warm-up, I felt pain in my legs, but it was less intense*."

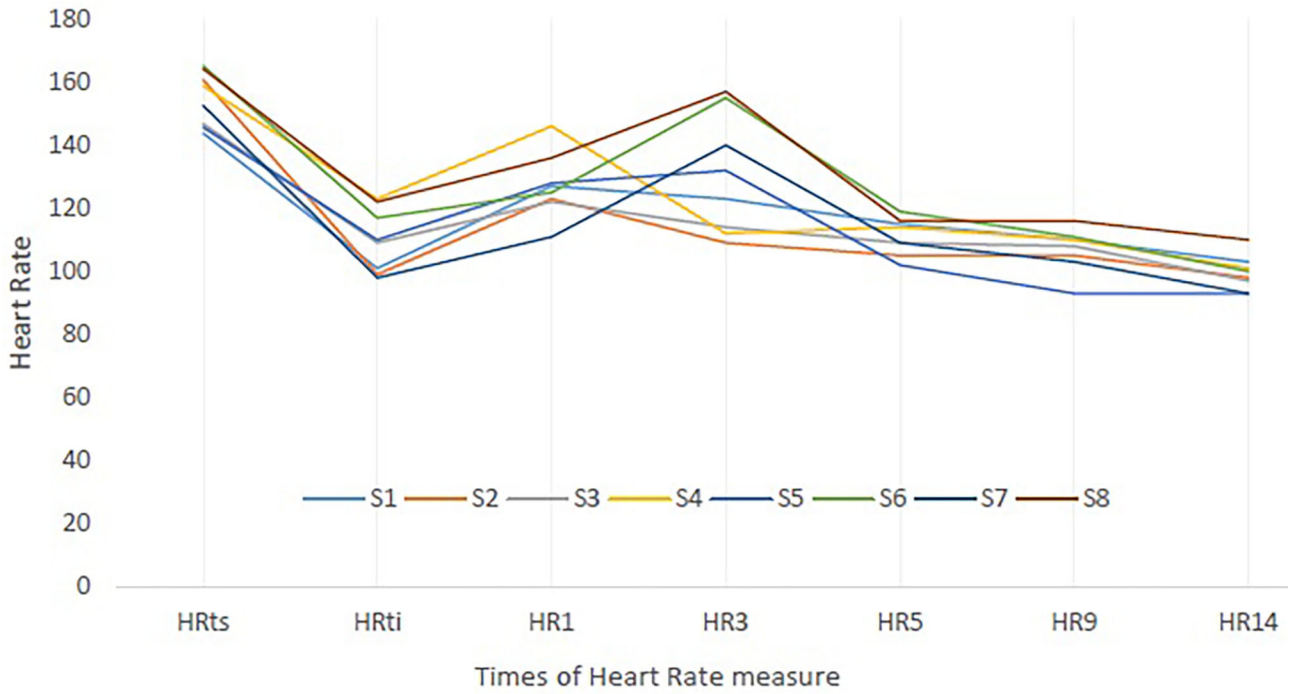

FIGURE 3 | Heart rate (HR) individual's responses to training session (ts) and to cold-water immersion (CWI). S, Subjects; HRts, Mean HR of the training session; HRti, HR at initial time of immersion. HR1, HR3, HR5, HR9, HR14: HR value at 1, 3, 5, 9, and 14 min of CWI.

Subjects reported how the sensation of the CWI generated memories of past sensations, such as swimming in the cold sea which highlights the relation between the sensorial experiences and the perceived sensations (Andrieu and Gerardin, 2012; Andrieu and Burel, 2014). Once acclimatized, subjects reported that the immersion in cold water was pleasurable and more relaxing than simply lying still. The immersion seemed to bring new energy, accompanied by a sensation of body vitality, never experienced before. These statements could be explain by the fact that the subjects are not habituated to the cold shock and have a strong thermoregulation responses to cold, which is attenuated after the first time of CWI (Bailey et al., 2007; Bleakley et al., 2012).

Sensorial experiences described how heat generated by the physical load of the training session contrasted with the cold shock of the CWI. This seemed to have created a sense of euphoria. The keenly felt contrast of temperatures is the sign of internal activity generating heightened perception of the body (Andrieu and Burel, 2014). The desire to return to training or physical effort is facilitated by the sensation of well-being (Montgomery et al., 2008). One player described the tension between the unpleasant sensations that were experienced with the knowledge that the CWI was good for the physical body: "*We were in the cold water, it was not very pleasant but it is good for our bodies*" and how although the awareness of being in cold water did not disappear, "*even if the cold water disturbed me less*."

The awareness of being in cold water did not disappear but instead produced variations in pain that increasingly bearable. Subjects reported a progressive reduction in tension between the physical and sensorial experiences of the cold and we believe it was this reduction which facilitated their acceptance of the CWI as being beneficial for their health.

We acknowledge several limitations associated with the study. Whereas, cold can inhibit the inflammatory process, the overall decrease in DOMS may not be as great after 14 min of CWI. This time could ultimately moderate CWI effectiveness at reducing pain. However, the cold shock reduced soreness more effectively than PR. Due to the nature of water immersion, it was not possible to include several conditions of immersion (durations and temperatures) to determine the optimal protocol specific to each subject. Although this is a key outcome in the recovery of an athlete, further studies should consider the dose–response effect of CWI on other markers of muscle damage, such as performance, in order to identify the best CWI recovery strategy based on different and relevant factors.

PRACTICAL APPLICATIONS

DOMS scores decreased significantly after CWI in comparison to the scores recorded after PR. This effect is important for an intermittent game such as handball, as it is played with short but intense periods of exertion. A sensation of well-being that arose from the reduction in muscle pains was also described by the players 24 h after their training session. The CWI following games may help increase perceptual recovery. Players, coaches and medical staff should be aware that the use of CWI can decrease the post-training physical and psychometric loads.

CONCLUSION

CWI is an efficient mean of triggering immediate, post-exercise parasympathetic activity, and reducing the DOMS. The sensorial experiences described by the subjects demonstrated a positive view of recovery using CWI. The person's sensorial experiences can influence the effectiveness of the CWI recovery as it will indicate whether or not the individual is receptive and able to respond to that potentially uncomfortable of the cold shock.

DATA AVAILABILITY STATEMENT

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

ETHICS STATEMENT

The studies involving human participants were reviewed and approved by the local commitee scientific of university of Rouen. Written informed consent to participate to this study was provided by the participant. The patients/participants provided their written informed consent to participate in this study.

AUTHOR CONTRIBUTIONS

ML'H, JC, IC, NG, MT, BA, and CT as conceived, designed, performed, and analyzed the research. ML'H and CT wrote the manuscript. All authors read, review, and approved the final manuscript.

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Effect of the Depth of Cold Water Immersion on Sleep Architecture and Recovery Among Well-Trained Male Endurance Runners

*Maxime Chauvineau, Florane Pasquier, Vincent Guyot, Anis Aloulou and Mathieu Nedelec**

Laboratory of Sport, Expertise and Performance (EA 7370), French National Institute of Sport (INSEP), Paris, France

Introduction: The aim of the present study was to investigate the effect of the depth of cold water immersion (CWI) (whole-body with head immersed and partial-body CWI) after high-intensity, intermittent running exercise on sleep architecture and recovery kinetics among well-trained runners.

Methods: In a randomized, counterbalanced order, 12 well-trained male endurance runners ($\dot{V}O_2max = 66.0 \pm 3.9$ ml·min^{-1}·kg^{-1}) performed a simulated trail (≈18:00) on a motorized treadmill followed by CWI (13.3 ± 0.2°C) for 10 min: whole-body immersion including the head (WHOLE; $n = 12$), partial-body immersion up to the iliac crest (PARTIAL; $n = 12$), and, finally, an out-of-water control condition (CONT; $n = 10$). Markers of fatigue and muscle damage—maximal voluntary isometric contraction (MVIC), countermovement jump (CMJ), plasma creatine kinase [CK], and subjective ratings—were recorded until 48 h after the simulated trail. After each condition, nocturnal core body temperature (T_{core}) was measured, whereas sleep and heart rate variability were assessed using polysomnography.

Results: There was a lower T_{core} induced by WHOLE than CONT from the end of immersion to 80 min after the start of immersion ($p < 0.05$). Slow-wave sleep (SWS) proportion was higher ($p < 0.05$) during the first 180 min of the night in WHOLE compared with PARTIAL. WHOLE and PARTIAL induced a significant ($p < 0.05$) decrease in arousal for the duration of the night compared with CONT, while only WHOLE decreased limb movements compared with CONT ($p < 0.01$) for the duration of the night. Heart rate variability analysis showed a significant reduction ($p < 0.05$) in RMSSD, low frequency (LF), and high frequency (HF) in WHOLE compared with both PARTIAL and CONT during the first sequence of SWS. No differences between conditions were observed for any markers of fatigue and muscle damage ($p > 0.05$) throughout the 48-h recovery period.

Conclusion: WHOLE reduced arousal and limb movement and enhanced SWS proportion during the first part of the night, which may be particularly useful in the athlete's recovery process after exercise. Future studies are, however, required to assess whether such positive sleep outcomes may result in overall recovery optimization.

Keywords: polysomnography, muscle damage, core body temperature, heart rate variability, slow-wave sleep, performance, limb movements, arousals

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Edited by:
Robert Allan,
University of Central Lancashire,
United Kingdom

Reviewed by:
David Andrew Low,
Liverpool John Moores University,
United Kingdom
Chris Mawhinney,
Mahidol University, Thailand

****Correspondence:***
Mathieu Nedelec
mathieu.nedelec@insep.fr

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INTRODUCTION

Elite sport requires regular competitions and training multiple times a day for consecutive days, which can make athletes' readiness for high performance challenging. An imbalance in training/competition load and recovery increases the risk of overtraining, injury, and underperformance (Kenttä and Hassmén, 1998). Several studies have assessed the interest of different strategies in accelerating the rate of recovery, with sleep and cold water immersion (CWI) demonstrating the highest scientific level of evidence (Halson, 2008; Nédélec et al., 2013).

Cold water immersion is an often-used recovery strategy (Nédélec et al., 2013), which decreases core body temperature (T_{core}) below baseline with a peak difference occurring at 60 min post-immersion (Stephens et al., 2018). This cooling strategy is effective when repairing exercise-induced muscle damage (Ihsan et al., 2016) with larger effect for weight-bearing (running and strength training) compared with non-weight-bearing activities (Halson, 2011), which may be related to CWI-induced muscle cooling and hydrostatic pressure (Wilcock et al., 2006; Leeder et al., 2012). The main benefits of CWI are reductions in delayed onset muscle soreness, edema, and exercise-induced strength loss (Wilcock et al., 2006; Bailey et al., 2007; Leeder et al., 2012). Moreover, subjective measures of fatigue and recovery are improved in the hours and days following CWI (Wilcock et al., 2006; Halson et al., 2008). The effectiveness of CWI may be higher when a whole-body immersion is implemented compared with a partial-body protocol (Wilcock et al., 2006). A meta-analytical review has reported that whole-body immersion is significantly more effective (5.1%, $g = 0.62$) on performance recovery than partial-body immersion, i.e., immersing only the legs or arms (1.1%, $g = 0.10$; Poppendieck et al., 2013). These results can be explained by a higher reduction in T_{core} when the entire body is immersed (Stephens et al., 2017). In addition, the immersion of the head seems especially important to induce a maximal rate of decline in T_{core} (Pretorius et al., 2006). Pretorius et al. (2006) reported that head immersion in cold water (17°C for 30 min) considerably increases core body cooling rate (≈+42%) compared with when the head is not immersed.

Sleep and thermoregulation are closely related (Kräuchi and Deboer, 2010). Previous studies suggested that a maximal rate of decline in T_{core} close to bedtime, which occurs through an increase in cutaneous temperature and heat loss from the periphery, can favor sleep initiation and can enhance sleep propensity (Berger and Phillips, 1995; Deboer, 1998; Kräuchi and Deboer, 2010). Sleep provides a number of important psychological and physiological functions that may be fundamental to the athlete's recovery process (Nédélec et al., 2015; Walsh et al., 2020). It has notably been shown that slow-wave sleep (SWS), a component of non-rapid eye movement (NREM) sleep, is restorative and allows muscle repair and adaptation (Akerstedt and Nilsson, 2003; Dijk, 2009; Halson and Juliff, 2017). Consequently, immersing the whole body—head included—close to bedtime may be a promising CWI strategy to enhance sleep and neuromuscular recovery after a high-intensity exercise.

Some studies have examined the relationship between CWI and sleep (Robey et al., 2013; Lastella et al., 2019). Lastella et al. (2019) reported a shorter sleep onset latency after the use of CWI compared with a placebo condition among elite cyclists during a simulated hill-climbing tour. However, CWI was performed early in the day (13:00–14:00), and sleep was monitored using wristwatch actigraphy, which does not allow sleep architecture assessment. In contrast, Robey et al. (2013) did not report any improvement in sleep architecture after the use of partial-body immersion at ≈20:15 following an intense cycling exercise compared with exercise alone.

To the best of our knowledge, no study has assessed the effect of whole-body CWI (head immersed) on sleep architecture and recovery in a sport setting. The aim of the present study was to investigate the effect of the depth of CWI (whole-body with head immersed vs. partial-body CWI) realized close to bedtime after high-intensity, intermittent running exercise on sleep architecture and recovery kinetics among well-trained runners. We hypothesized that a higher decline in T_{core} induced by whole-body immersion performed post-exercise would enhance SWS proportion and hasten the recovery process compared with a partial-body immersion.

MATERIALS AND METHODS

Participants

Twelve well-trained male runners [mean ± *SD*; age = 28.0 ± 5.8 years; body mass = 65.7 ± 6.6 kg; height = 176.0 ± 8.6 cm; body fat estimated from the method of Durnin and Womersley (1974) = 9.8 ± 3.2%; maximal aerobic speed (MAS) = 18.1 ± 1.0 $km \cdot h^{-1}$; maximal oxygen uptake ($\dot{V}O_2max$) = 66.0 ± 3.9 $ml \cdot min^{-1} \cdot kg^{-1}$] volunteered to participate. Participants were classified at level 4 according to the guidelines of De Pauw et al. (2013) for performance level classification in sport science research. All participants used to train three to six times a week with a required performance level < 38 min per 10 km, $\dot{V}O_2max > 60$ $ml \cdot min^{-1} \cdot kg^{-1}$ and MAS > 17 $km \cdot h^{-1}$. They were not accustomed to CWI and underwent a detailed medical history and examination by a medical doctor. This included an electrocardiogram at rest and an examination to exclude any contraindication to cold water exposure, e.g., cold hypersensitivity (Raynaud's phenomenon). The study was conducted according to the Declaration of Helsinki (1964: revised in 2001), and the protocol was approved by the local ethics committee (East III, France. Ref. 170605). The participants also provided their written informed consent before the initiation of experiments.

Actigraphy data (CamNtech, MotionWare 8) were collected for 3–15 days (BASELINE: 11.0 ± 7.2 days) before the start of the study and during the study to assess the sleep–wake patterns of each participant. They were instructed to sleep in the same home environment throughout the study and to respect the same bedtime and wake-up time (±30 min). No main effect ($p > 0.05$) of time, condition, and interaction between time and condition was noted for any actigraphic values from the two nights before experimentation to the end of each condition. The exclusion criteria checked prior to the start of the study

were as follows: (a) an average sleep duration >9 or <6 h per night from Sunday to Thursday; (b) an average lights-out time earlier than 21:00 from Sunday to Thursday; (c) an average wake-up time later than 09:00 from Monday to Friday (Arnal et al., 2016); (d) a daily consumption of alcoholic beverages and/or more than 300 mg of caffeine per day and/or the use of antidepressant medications; (e) sleep complaints (i.e., Pittsburgh Sleep Quality Index >5; Buysse et al., 1989) and a non-extreme morning or evening chronotype on the Horne and Ostberg questionnaire (i.e., <31 and >69; Horne and Ostberg, 1976); (f) polysomnography-confirmed sleep disorders, such as sleep apnea (apnea–hypopnea index >10) and other sleep disorders (periodic limb movement syndrome, hypersomnia, insomnia, circadian sleep rhythm disorders, or narcolepsy); and (g) shift workers.

Experimental Design

The design consisted of a familiarization session and three experimental conditions—a whole-body CWI including the head (WHOLE), a partial-body CWI up to the iliac crest (PARTIAL), and finally an out-of-water control condition (CONT)—all completed after a standardized simulated trail running (TRAIL). Two participants did not complete the CONT condition. The familiarization session was conducted at least 1 week before the experimental conditions and included the following: (a) a graded-exercise test; (b) a short period (≈5 min) of downhill and climb running on a treadmill, with the purpose of familiarization without inducing muscle damage; (c) familiarization with the protocol of lower limb muscle strength assessment corresponding to a maximal voluntary isometric contraction (MVIC) as well as countermovement jump (CMJ) test; (d) familiarization of WHOLE during ≈6 min; and (e) a night of familiarization with the polysomnography portable device in the same home environment as for the experimental nights to avoid the first night effect (Agnew et al., 1966).

In a randomized, crossover, counterbalanced order, participants performed WHOLE, PARTIAL, and, finally, CONT, all separated by a minimum of 1 week. The one-way ANOVA revealed no main effect of condition ($p > 0.05$) for the ambient temperature in the laboratory (23.5 ± 2.4°C) and relative humidity (44.1 ± 8.0%). Participants were asked to abstain from physical activity the day prior to and for the duration of each experimental condition. Food intake was standardized for all participants 3 days prior to and for the duration of each experimental condition. The meal plan was created by a nutritionist and included a variety of breads, cereals, milk/yogurts, meats, pasta/rice, fruit, and vegetables to ensure the adequate intake of macro- and micronutrients. Participants were hydrated regularly before and after the simulated trail but not during the trail. Additionally, they were not allowed to use any recovery strategy (e.g., compression garments, electrostimulation, massage, and stretching) during the protocol.

In the three experimental conditions, participants arrived at ≈17:00 to start the testing battery (PRE-TRAIL) in the following order: subjective ratings, blood sampling, MVIC, and CMJ (**Figure 1**). They performed the simulated trail at ≈18:00. The test battery was repeated after (POST-TRAIL), and all participants were accompanied to the balneotherapy (approximately 250 m), drinking 500 ml of standardized milk beverage. They changed into their bathing suits and took a cold (≈15°C) shower during ≈2 min for sanitary purposes. They performed WHOLE, PARTIAL, or CONT at ≈19:55 and did not take a shower afterward. They ate a standardized meal at ≈20:15. Finally, the polysomnography equipment was set up (taking approximately 40 min) by a qualified practitioner, and participants went home to sleep in their usual environment. The one-way ANOVA revealed no main effect of condition ($p > 0.05$) for bedtime (23:25 ± 00:38), wake-up time (06:37 ± 00:49), and bedroom ambient temperature (22.1 ± 2.9°C) (iButtons, Embedded Data Systems, Lawrenceburg, Kentucky). Finally, participants returned to the laboratory 24 h (H24) and 48 h (H48) after the simulated trail at ≈17:00 to perform the same test battery.

Graded-Exercise Test

The graded-exercise test was performed until exhaustion to determine MAS and $\dot{V}O_{2peak}$ on a treadmill (Saturn 300/100r, h/p/cosmos Sports & Medical gmbh, Germany). The test started at 12 km·h^{-1} and increased 1 km·h^{-1} every 2 min with a slope at +1%. The last successful 2-min step was assigned to MAS and used to individualize the simulated trail intensity.

Simulated Trail

For each experimental condition, participants completed a simulated trail adapted from Aloulou et al. (2019). The exercise lasted 48 min with five 9-min blocks and was shown to elicit a significant level of fatigue and muscle damage until 48 h after exercise (Aloulou et al., 2019). Heart rate (HR) was measured throughout the exercise. Each block included 4 min of downhill running (−12.5% gradient) followed by 3 min of flat running (0% gradient) and 2 min of uphill running (+10% gradient). During the downhill running, velocity was set to 80% of the MAS achieved during the graded-exercise test. The flat block included three 1-min runs at 100, 60, and 100% of MAS. During the uphill running, velocity was set to 65% of MAS.

Cold Water Immersion Interventions

Following the POST-TRAIL testing battery, each participant completed 10 min of WHOLE, PARTIAL, or CONT. The whole-body immersion included the head and neck in a crouching position. Participants were fitted with a scuba kit and swimming pool glasses. For the partial-body immersion, participants were standing upright and immersed up to the iliac crest. The one-way ANOVA revealed no main effect of condition ($p > 0.05$) for water temperature (13.3 ± 0.2°C), in accordance with recommendations (Machado et al., 2016), and for the time to reach the required immersion level (03:23 ± 01:20 min). For CONT condition, participants sat down for 10 min in a controlled environment (19.2 ± 2.0°C; 47.9 ± 9.6% relative humidity).

Measures

Core Body Temperature

Three hours before the simulated trail, participants ingested a radiotelemetry pill (BodyCap; e-Celsius® Performance) to

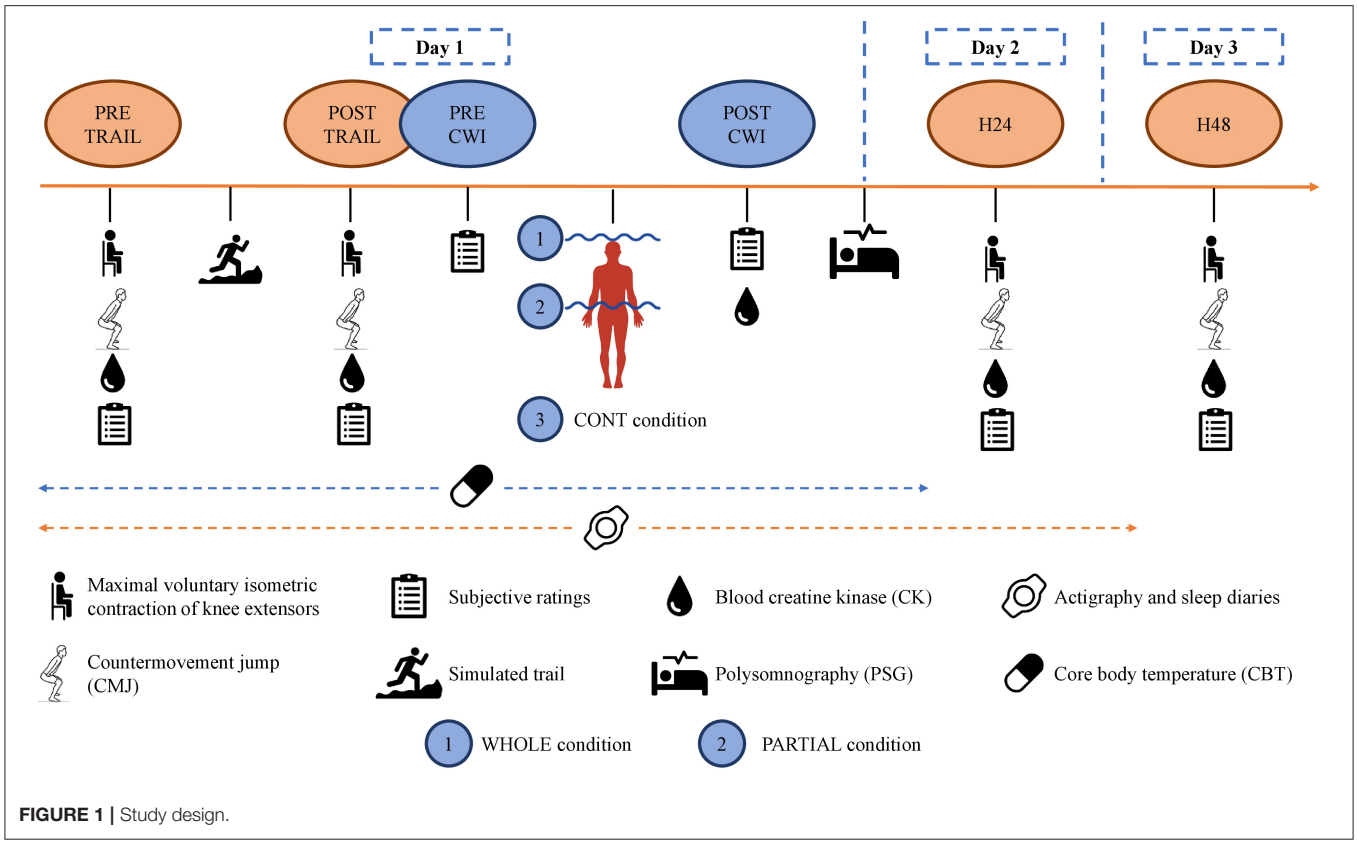

FIGURE 1 | Study design.

continuously record T_{core} with a frequency of 1 value per min. This method has been shown to be reliable and valid (Bongers et al., 2018), with an accuracy of 0.23°C, an intraclass correlation coefficient of 1.00, and a standard error of measurement of 0.03.

Sleep Recording and Analysis

Polysomnography

Polysomnography recordings were obtained the night after each CWI intervention using a portable device (Nox A1; Resmed). Polysomnography was performed following the technical specifications of the American Academy of Sleep Medicine manual for the scoring of sleep and associated events (Berry et al., 2017) including the following: six electroencephalography channels placed according to the international 10–20 electrodes placement system (F3-M2, F4-M1, C3-M2, C4-M1, O1-M2, and O2-M1); left and right electro-oculography; two chin electromyography channels, placed on the mentalis and submentalis; bilateral tibial electromyography; electrocardiography; and rib cage and abdominal wall motion via respiratory impedance. All data were scored using Noxturnal software version 5.1 (Resmed, USA) in 30-s epochs, according to the 2017 American Academy of Sleep Medicine (Berry et al., 2017). Each experimental night was analyzed by one trained specialist (>3,000 analyses in the last 10 years) who was blinded to the condition. Two distinct analyses were performed, one for the whole night and one for the first 180 min after sleep onset (Robey et al., 2013). The following dependent variables were calculated: total sleep time (min), the time spent in any stage of sleep (i.e., N1, N2, SWS, and REM); time in bed (min), the time between lights out getting up; wake after sleep onset (WASO; min), the time spent in bed awake minus sleep onset latency; sleep efficiency (%), total sleep time divided by time in bed × 100; sleep onset latency (min), the time between lights out and the first epoch of any stage of sleep (i.e., N1, N2, SWS, and REM); REM onset latency (min), the time between lights out and the first epoch of any stage of REM sleep stage; wake, N1, N2, SWS, REM, light (N1+N2), and NREM sleep (N1+N2+SWS) were defined as percentage of time spent compared with the time from sleep onset to waking up; arousal, a sudden change in electroencephalographic frequency—alpha, theta, and/or frequency >16 Hz—lasting at least 3 s and with the presence of at least 10 s of sleep before the change; and limb movement, an increase of ≥8 μV in the activity of the tibial electromyography lasting between 0.5 and 10 s. The arousal and limb movement indexes were defined as the number of counts per hour asleep.

The Spiegel Sleep Inventory

The Spiegel Sleep Inventory (SSI) was administered upon waking to assess the perceived sleep quality of each participant in each condition. The SSI is a self-administered questionnaire composed of six questions (score: 1–5) regarding sleep initiation, sleep quality and duration, nocturnal awakenings, dreams, and feeling refreshed in the morning. The global score is the sum of the six

items. There are few data available on the psychometric validity of the SSI; however, it is a very simple and easy-to-use scale that is often used to assess the presence of insomnia (Léger et al., 2006).

Nocturnal Heart Rate

The ECG signal was derived from the right midclavicular and around 6 cm under the left armpit positions. The electrodes were connected to the polysomnographic system (Nox A1; Resmed, USA), and ECG data were continuously recorded at 200 Hz. Data were then converted into a European data format and imported into Kubios heart rate variability (HRV) software (version 3.3.1, 2019; MATLAB) for analysis. Nocturnal HRV and HR indexes were determined using the first 5-min stationary segment (free from arousals) in the first SWS sequence (determined via polysomnographic scoring) that lasted more than 15 min. Slow-wave sleep has been shown to better discriminate the state of sympathovagal balance than waking periods (Brandenberger et al., 2005). Electrocardiogram waveforms were analyzed to obtain temporal and frequency domain components. Time-domain variables, including mean R-R interval (RRi), the standard deviation of normal RRi (SDNN), and the root-mean square difference of successive normal RRi (RMSSD) were assessed. Frequency domains were assessed for the power densities in the low-frequency (LF; 0.04–0.15 Hz) and high-frequency (HF; 0.15–0.50 Hz) bands during each 5-min spectrum. The LF/HF ratios and the normalized LF/(LF+HF) ratios were then calculated.

Markers of Fatigue, Muscle Damage, and Subjective Assessments

Maximal Voluntary Isometric Contraction of Knee Extensors

Maximal voluntary isometric contraction of knee extensors was assessed at PRE-TRAIL, POST-TRAIL, H24, and H48 with a 90° knee angle and 80° hip angle using an isokinetic ergometer (Con-Trex Multi-Joint System) after a 10-min warm-up on a cycle-ergometer at 100 W and 80 rpm. For each maximal contraction, the participants were instructed to extend their knees as fast and as hard as possible for 5 s, and all participants received standardized verbal encouragement from the same experimenter. Three MVICs of the knee extensor muscles were performed, with rest periods of 60 s. The best performance was defined as the highest peak force value of the three trials. The test–retest reliability was assessed by comparing the pre-values of the three experimental conditions. The typical error was 13.3 N·m (95% CI, 9.8 to 21.2 N·m), the intraclass correlation coefficient was 0.90 (95% CI, 0.72 to 0.97), and the coefficient of variation was 5.6%.

Countermovement Jump (CMJ) Performance

Countermovement jump (CMJ) performance was assessed at PRE-TRAIL, POST-TRAIL, H24, and H48 to evaluate neuromuscular fatigue and measured with photoelectric cells (Optojump®, Microgate, Bolzano, Italy) after a free joint warm-up. For each maximal effort, the participants were instructed to jump as high as possible while keeping hands on hips throughout the jump. The range of motion was free, and all participants received standardized verbal encouragement from the same experimenter. Three CMJs were performed, with rest periods of 60 s. The height of each jump was noted, and the best jump was defined as the highest jump of the three trials. The test–retest reliability was assessed by comparing the pre-values of the three experimental conditions. The typical error was 1.6 cm (95% CI, 1.2 to 2.5 cm), the intraclass correlation coefficient was 0.92 (95% CI, 0.79 to 0.98), and the coefficient of variation was 4.5%.

Blood Creatine Kinase [CK] Concentration

Blood creatine kinase [CK] concentration was assessed at PRE-TRAIL, POST-TRAIL, POST-CWI, H24, and H48 by collecting a 32-µl blood sample from a fingertip capillary puncture. The blood sample was then placed on a measurement strip and analyzed using a Reflotron Plus (Roche Diagnostics). The Reflotron Plus was calibrated according to the manufacturer's recommendations. The test–retest reliability was assessed by comparing the pre-values of the three experimental conditions. The typical error was 165.2 UI/L (95% CI, 125.4 to 262.7 UI/L), the intraclass correlation coefficient was −0.10 (95% CI, −0.44 to 0.39), and the coefficient of variation was 80.4%.

Rating of Perceived Exertion of the Session (RPE- S)

Rating of perceived exertion of the session (RPE-S) was assessed at POST-TRAIL. Participants answered the question "How was your workout?" using a scale from 0 (rest) to 10 (maximal; Foster, 1998).

The Well-Being Hooper Index

The well-being Hooper Index is a rating of general fatigue, stress, sleep perception, and muscle soreness (Hooper and Mackinnon, 1995). Participants were asked to subjectively evaluate at PRE-TRAIL, POST-TRAIL, POST-CWI, H24, and H48 for each condition the four items, using a 1–7 scale, with 1 representing the most positive rating and 7 representing the most negative rating, for each variable.

The Total Quality of Recovery (TQR) Scale

The total quality of recovery (TQR) scale was reported by the participants at POST-CWI, H24, and H48 for each condition. Scores varied from 6, "very, very poor recovery," to 20, "very, very good recovery" (Kenttä and Hassmén, 1998).

Belief in the Anticipated Effectiveness

Belief in the anticipated effectiveness of WHOLE and PARTIAL was assessed at PRE-TRAIL and H48 for these conditions on a scale adapted from Broatch et al. (2014). Participants were instructed to choose on a 5-point Likert scale between two extremes points (1 indicating "strongly agree" and 5 indicating "strongly disagree") by answering the following question: "Whole (or partial-) body cold water immersion allows a better recovery compared with passive recovery (no cold water immersion)?"

Perceived Thermal Comfort

Perceived thermal comfort (on a scale from −2 "very uncomfortable" to +2 "very comfortable") and *sensation* (on a scale from −3 "cold" to +3 "hot"; Zhang and Zhao, 2008) were recorded for each participant at PRE-TRAIL, PRE-CWI, and POST-CWI.

Statistical Analysis

Based on a previous study (Aloulou et al., 2019), we estimated that a sample size of 12 subjects would allow us to detect differences in MVIC after a simulated trail with power (1 – β) set at 0.80, a large effect size (>0.8) and an alpha of 0.05. *Post-hoc* power analysis revealed that this study was adequately powered, with actual power (1 – β) > 0.90 (G*Power program version 3.1.9.7). Statistical analyses were performed with the R program (version 1.4.869). Prior to the analysis, the Shapiro–Wilk test and the Mauchly test were employed to test the normality of the data and sphericity assumption, respectively. The Greenhouse–Geisser correction was conducted when the sphericity was violated to adjust the significance of the *F* ratios. Core body temperature, nocturnal HR, and markers of fatigue and exercise-induced muscle damage in WHOLE, PARTIAL, and CONT conditions were analyzed using a two-way (condition × time) repeated-measures analysis of variance (ANOVA). A log transformation was applied to the non-normalized data (i.e., MVIC, [CK], general fatigue, stress, muscle soreness, TQR, belief in the anticipated effectiveness of intervention, perceived thermal comfort, and sensation) to reduce non-uniformity bias. For T_{core} kinetics, 13-time points were used from the start to 120 min after the start of CWI intervention with 10-min intervals. For nocturnal T_{core}, 13-time points were used from bedtime to 06:00 after bedtime with 30-min intervals. For polysomnography and HRV analysis, one-way ANOVAs with repeated measures were performed. The Friedman non-parametric test was performed for non-normalized data to observe the main effect of the experimental condition. Partial eta squared ($\eta^2 p$) is provided as measures of effect size for the two- and one-way ANOVA for repeated-measures, and Kendall's W value is provided for the Friedman test. When a significant main effect was found, Tukey honestly significant difference (HSD) *post-hoc* test or the non-parametric Conover test was performed. Effect sizes were calculated to interpret the magnitude of the mean difference between conditions with $d < 0.2$, $d = 0.2$–0.5, $d = 0.5$–0.8, and $d > 0.8$ considered trivial, small, moderate, and large, respectively (Cohen, 1988). Correlations between dependent variables were analyzed using the Pearson product-moment correlation coefficient (*r*). Results are expressed as the mean ± standard deviation (*SD*), and the level of significance was set at $p < 0.05$.

RESULTS

The one-way ANOVA revealed no main effect of condition ($p > 0.05$) for the mean HR during the simulated trail and RPE-S. Independent of the experimental condition, mean HR during the simulated trail was 83.5 ± 3.7% of maximal HR, and RPE-S was 7.8 ± 1.8 AU. A main effect of time ($p < 0.001$; $\eta^2_p = 0.92$) for T_{core} throughout the simulated trail was found. Independent of the experimental conditions, T_{core} was 36.99 ± 0.55°C at the beginning of the simulated trail. At the end of the simulated trail, T_{core} significantly increased ($p < 0.001$; $d = 2.75$) with a large effect compared with PRE-TRAIL (38.94 ± 0.53°C).

Effects of Cold Water Immersion Interventions on Thermal Responses, Sleep, and Heart Rate Variability

Core Body Temperature

Significant main effects of condition ($p < 0.001$; $\eta^2_p = 0.68$), time ($p < 0.001$; $\eta^2_p = 0.68$), and the interaction between condition and time ($p < 0.001$; $\eta^2_p = 0.53$) were observed for T_{core} responses (**Figure 2**). *Post-hoc* analysis revealed a significantly lower T_{core} induced by WHOLE than CONT from the end of CWI intervention to 80 min after the start of CWI intervention with large effects ($p < 0.05$; $d = 0.96$–2.45). Compared with PARTIAL, WHOLE induced a significantly lower T_{core} from 20 to 40 min after the start of CWI intervention with large effects ($p < 0.001$; $d = 1.61$–2.04). During the whole night, the two-way ANOVA revealed no significant interaction between condition and time ($p = 0.64$; $\eta^2_p = 0.13$). However, there was a significant main effect of condition ($p < 0.05$) for mean T_{core} (**Table 1**). Mean T_{core} was significantly higher in WHOLE ($p < 0.01$; $d = 0.82$) and PARTIAL ($p < 0.05$; $d = 0.86$) with a large effect compared with CONT condition.

Sensation and Thermal Comfort

There was a significant interaction between condition and time for sensation ($p < 0.01$; $\eta^2_p = 0.42$) and thermal comfort ($p < 0.01$; $\eta^2_p = 0.41$). Immediately after WHOLE, large significantly lower sensation and thermal comfort were observed compared with both CONT and PARTIAL ($p < 0.001$; $d = -2.39$ to -1.37).

Polysomnography Analysis During the Whole Night

There was a main effect between conditions ($p < 0.05$) for N1 sleep, arousals, limb movements, and mean T_{core} (**Table 1**). *Post-hoc* analysis revealed a large significantly lower proportion of N1 sleep in WHOLE than CONT condition ($p < 0.01$; $d = -1.73$). The number of arousals was significantly higher in CONT than both WHOLE ($p < 0.05$; $d = 1.01$) and PARTIAL conditions ($p < 0.05$; $d = 0.74$). A large significant reduction in limb movements was noted in WHOLE compared with CONT condition ($p < 0.01$; $d = -0.98$). There were no main effects of condition on overall night polysomnographic sleep variables (**Table 1**).

Polysomnography Analysis During the First 180 Min After Sleep Onset

A significant main effect of condition ($p < 0.05$) was found for arousals and limb movements (**Table 2**). The number of arousals was significantly higher in CONT than both WHOLE ($p < 0.05$; $d = 0.72$) and PARTIAL conditions ($p < 0.05$; $d = 0.55$). Limb movements were significantly lower in WHOLE than both PARTIAL ($p < 0.01$; $d = -0.62$) and CONT conditions ($p < 0.001$; $d = -0.92$). A trend of a significant main effect of condition was found for SWS ($p = 0.08$) and REM sleep proportions ($p = 0.09$). Slow-wave sleep was moderately higher in WHOLE than PARTIAL ($p < 0.05$; $d = 0.59$). There was no main effect of condition on other polysomnographic sleep variables.

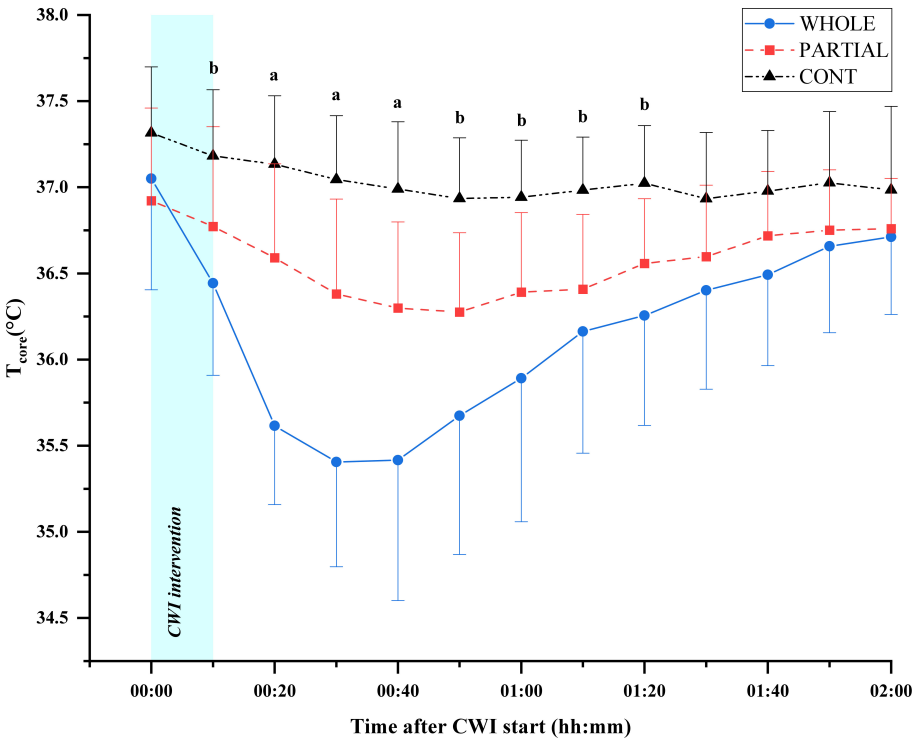

FIGURE 2 | Core body temperature responses (°C) in WHOLE ($n = 12$), PARTIAL ($n = 11$), and CONT ($n = 9$). All data are presented as mean ± *SD*. [a]WHOLE condition different to both PARTIAL and CONT conditions ($p < 0.05$). [b]WHOLE condition different to CONT condition ($p < 0.05$).

TABLE 1 | Polysomnography analysis, mean nocturnal heart rate, and T_{core} during the whole night in WHOLE ($n = 12$), PARTIAL ($n = 12$), and CONT ($n = 10$) conditions.

Condition	WHOLE	PARTIAL	CONT	*p*-Value	η^2_p/W
Total sleep time (min)	398.4 ± 84.8	393.5 ± 57.7	389.6 ± 47.6	0.61	0.05
Time in bed (min)	447.5 ± 65.8	435.0 ± 54.5	441.7 ± 51.4	0.38	0.10
WASO (min)	26.4 ± 19.1	23.6 ± 16.5	37.3 ± 29.4	0.10	0.23
Sleep efficiency (%)	88.4 ± 9.2	90.5 ± 6.3	88.3 ± 6.6	0.50	0.07
Sleep latency (min)	8.7 ± 4.8	10.2 ± 9.8	13.8 ± 12.9	0.45	0.08
REM latency (min)	114.9 ± 44.6	98.2 ± 40.1	105.8 ± 38.8	0.55	0.06
Wake (%)	6.5 ± 5.1	5.6 ± 4.1	8.7 ± 6.5	0.12	0.21
Stage N1 sleep (%)	6.9 ± 2.2[a]	7.8 ± 2.4	8.6 ± 2.2	<0.01	0.41
Stage N2 sleep (%)	45.7 ± 5.9	47.3 ± 7.1	44.6 ± 6.4	0.07	0.25
SWS (%)	24.3 ± 5.9	22.1 ± 5.8	21.8 ± 4.0	0.33	0.12
NREM (%)	77.4 ± 5.5	77.2 ± 5.0	75.0 ± 6.3	0.74	0.03
REM (%)	16.5 ± 3.3	17.2 ± 5.1	16.3 ± 3.0	0.90	0.01
Light sleep (%)	52.7 ± 4.7	55.1 ± 6.9	53.2 ± 5.0	0.50	0.07
Arousals (/h)	9.2 ± 2.5[a]	9.4 ± 2.8[a]	12.4 ± 3.1	<0.01	0.49
Limb movements (/h)	5.2 ± 2.0[a]	8.5 ± 5.4	11.5 ± 6.5	<0.001	0.61
Mean heart rate (bpm)	50.2 ± 7.8	49.0 ± 6.5	49.3 ± 7.6	0.67	0.04
Mean T_{core} (°C)	36.47 ± 0.15[a]	36.43 ± 0.19[a]	36.27 ± 0.26	<0.05	0.43

Data are presented as mean ± SD. WASO, wake after sleep onset; SWS, slow-wave sleep; NREM, non-rapid eye movement; REM, rapid eye movement.
[a]Significantly different from CONT condition ($p < 0.05$).

Subjective Sleep Evaluation

A main effect of condition was found for the "feeling refreshed in the morning" item ($p < 0.05$). Compared with CONT, both WHOLE ($p < 0.05$; $d = 0.82$) and PARTIAL conditions ($p < 0.05$; $d = 0.91$) largely improved the "feeling refreshed in the morning" item. There was no main effect of condition on other SSI items.

TABLE 2 | Polysomnography analysis, mean nocturnal heart rate, and T_{core} during the first 180 min after sleep onset in WHOLE ($n = 12$), PARTIAL ($n = 12$), and CONT ($n = 10$) conditions.

Condition	WHOLE	PARTIAL	CONT	*p*-value	η^2_p/W
Total sleep time (min)	172.2 ± 6.8	173.1 ± 5.3	162.5 ± 22.7	0.50	0.07
WASO (min)	7.7 ± 6.8	6.9 ± 5.2	17.4 ± 22.7	0.58	0.05
Wake (%)	4.3 ± 3.8	3.9 ± 2.9	9.7 ± 12.6	0.58	0.05
Stage N1 sleep (%)	6.9 ± 2.4	7.1 ± 2.5	7.8 ± 2.6	0.70	0.04
Stage N2 sleep (%)	41.9 ± 7.8	42.9 ± 7.8	40.6 ± 7.4	0.24	0.15
SWS (%)	39.7 ± 7.4[b]	34.6 ± 6.6	35.1 ± 10.1	0.08	0.25
NREM (%)	88.6 ± 6.3	84.6 ± 5.6	83.5 ± 13.7	0.41	0.09
REM (%)	7.2 ± 3.8	11.5 ± 5.2	6.8 ± 2.9	0.09	0.23
Light sleep (%)	48.8 ± 7.2	50.1 ± 9.1	48.4 ± 7.4	0.84	0.02
Arousals (/h)	10.6 ± 2.7[a]	10.5 ± 3.3[a]	13.2 ± 3.4	0.05	0.28
Limb movements (/h)	4.4 ± 2.1[a,b]	9.3 ± 9.0	11.4 ± 8.9	<0.01	0.57
Mean heart rate (bpm)	52.3 ± 8.6	50.4 ± 7.4	51.1 ± 8.7	0.53	0.07
Mean T_{core} (°C)	36.51 ± 0.19	36.46 ± 0.25	36.31 ± 0.29	0.07	0.28

Data are presented as mean ± SD. WASO, wake after sleep onset; SWS, slow-wave sleep; NREM, non-rapid eye movement; REM, rapid eye movement.
[a] *Significantly different from CONT condition ($p < 0.05$).*
[b] *Significantly different from PARTIAL condition ($p < 0.05$).*

Nocturnal Heart Rate

There was no significant main effect of condition on the mean HR during the whole night (**Table 1**). HRV analysis showed a significant main effect of condition on RMSSD, LF, and HF ($p < 0.05$; **Table 3**). *Post-hoc* analysis revealed a large significant reduction in RMSSD in WHOLE compared with both PARTIAL ($p < 0.05$; $d = -0.83$) and CONT ($p < 0.01$; $d = -0.82$). A large reduction in LF was noted in WHOLE compared with both PARTIAL ($p < 0.01$; $d = -1.01$) and CONT ($p < 0.05$; $d = -1.01$). After the WHOLE condition, a large significant reduction in HF was observed compared with both PARTIAL ($p < 0.05$; $d = -0.89$) and CONT ($p < 0.01$; $d = -0.93$). No main effect of condition was noted for RRi, HR, the normalized LF/(LF+HF) ratio, and the LF/HF ratio ($p > 0.05$). SDNN in WHOLE was moderately and largely lower than PARTIAL ($p = 0.06$; $d = -0.71$) and CONT ($p < 0.05$; $d = -0.91$), respectively.

Effects of Simulated Trail and Between-Condition Differences on Recovery Kinetics

No main effects of condition and interaction between condition and time ($p > 0.05$) were observed for MVIC, CMJ, [CK], general fatigue, muscle soreness, and stress up to 48 h after simulated trail (**Supplementary Material 1**). There was no significant difference between conditions ($p > 0.05$) at PRE-TRAIL and POST-TRAIL for MVIC, CMJ, [CK], general fatigue, muscle soreness, and stress, suggesting similar fatigue and muscle damage after exercise in WHOLE, PARTIAL, and CONT conditions. However, a main effect of time ($p < 0.05$) was found for MVIC, CMJ, [CK], general fatigue, and muscle soreness. Independent of the experimental conditions, *post-hoc* analysis revealed a significant large and moderate decrease in MVIC at POST-TRAIL ($p < 0.001$; $d = -1.17$) and H24 ($p < 0.01$; $d = -0.56$) compared with PRE-TRAIL. There was a significantly lower CMJ at H24 than PRE-TRAIL with a large effect ($p < 0.001$; $d = 0.93$). An increase in [CK] from POST-TRAIL to H48 ($p < 0.01$; $d = 0.67$–2.16) was found compared with PRE-TRAIL. General fatigue was significantly higher at POST-TRAIL than PRE-TRAIL with a large effect ($p < 0.001$; $d = 1.26$). Muscle soreness was significantly higher than PRE-TRAIL from POST-TRAIL to H48 ($p < 0.01$; $d = 0.64$–1.52). There was a significant main effect of condition ($p < 0.05$) on TQR and belief in the effectiveness of CWI interventions. Irrespective of time assessment, TQR was significantly and moderately higher in both WHOLE ($p < 0.01$; $d = 0.62$) and PARTIAL ($p < 0.01$; $d = 0.62$) compared with CONT. A higher belief in the effectiveness of CWI interventions was found in both WHOLE ($p < 0.001$; $d = 3.33$) and PARTIAL ($p < 0.001$; $d = 3.03$) compared with CONT.

DISCUSSION

The aim of the present study was to investigate the effect of whole- (head immersed) and partial-body CWI after a high-intensity, intermittent running exercise on sleep architecture and recovery kinetics among well-trained athletes. The primary results showed the following: (a) both WHOLE and PARTIAL induced a significant decrease in arousals compared with CONT, while only WHOLE decreased limb movements compared with CONT; (b) WHOLE induced a significant reduction in both sympathetic and parasympathetic modulation compared with both PARTIAL and CONT; and (c) no significant differences were observed in markers of fatigue and exercise-induced muscle damage recovery between conditions.

Irrespective of the experimental conditions, the mean HR during the simulated trail was 83.5 ± 3.7% of maximal HR,

TABLE 3 | Heart rate variability analysis during the first SWS sequence in WHOLE ($n = 12$), PARTIAL ($n = 12$), and CONT ($n = 10$) conditions.

Condition	WHOLE	PARTIAL	CONT	*p*-Value	η^2_p/W
RRi (ms)	1,150.5 ± 212.3	1,219.8 ± 241.5	1,217.9 ± 259.7	0.20	0.17
HR (bpm)	53.7 ± 9.4	50.8 ± 9.0	51.3 ± 10.9	0.41	0.09
SDNN (ms)	43.5 ± 20.5[a]	61.2 ± 36.9	60.7 ± 27.2	0.06	0.28
RMSSD (ms)	46.0 ± 27.6[a,b]	71.1 ± 51.0	70.1 ± 41.5	<0.05	0.39
LF (ms^2)	911.3 ± 628.6[a,b]	2,386.4 ± 2,880.0	1,993.2 ± 1,707.1	<0.01	0.37
HF (ms^2)	751.0 ± 764.7[a,b]	2,055.5 ± 2,538.9	1,748.7 ± 1,766.4	<0.05	0.37
LF/(LF+HF) (AU)	62.6 ± 16.8	59.2 ± 22.0	58.5 ± 17.2	0.61	0.05
LF/HF (AU)	2.6 ± 2.8	2.5 ± 2.8	2.0 ± 1.9	0.15	0.19

Data are presented as mean ± SD.
[a] *Significantly different from CONT condition ($p < 0.05$).*
[b] *Significantly different from PARTIAL condition ($p < 0.05$).*

whereas RPE-S achieved 7.8 ± 1.8 AU. In addition, the simulated trail induced a significant decrease in MVIC (−6.2%) and CMJ (−5.6%) 24 h after the simulated trail, whereas [CK] (+57.5%) and muscle soreness (+1.9 AU) were still significantly higher 48 h after exercise. These results suggest that the simulated trail induced a high level of metabolic and neuromuscular fatigue with the presence of mild muscle damage (Paulsen et al., 2012). To the authors' knowledge, the present study is the first to compare the effects of WHOLE and PARTIAL CWI on sleep architecture and recovery in a sport setting. The present results showed that WHOLE induced a significantly lower T_{core} compared with CONT up to 80 min after the start of immersion. This result is consistent with previous studies (Zhang et al., 2015; Stephens et al., 2017), which highlighted the need to expose an important body surface in cold water to enhance convective heat dissipation and decrease T_{core}. Additionally, the immersion of the head in the present study may have contributed to a larger decline in T_{core} compared with partial-body immersion. Pretorius et al. (2006) showed that immersing the head in cold water (17°C) increases the rate of T_{core} decline (≈42%) compared with keeping the head above the water surface. A redistribution of blood flow to the face and scalp in response to stimulation of thermosensitive and/or trigeminal receptors as well as peripheral vasoconstriction may be potentially involved mechanisms (Pretorius et al., 2006).

We hypothesized that a higher decline in T_{core} induced by WHOLE would enhance SWS proportion during the subsequent night. Our hypothesis was only partially confirmed. To our knowledge, only one study previously used polysomnography to examine the effect of CWI following exercise on sleep quantity and quality among well-trained endurance athletes (Robey et al., 2013). Accordingly, Robey et al. (2013) reported no additional benefits procured by PARTIAL CWI compared with no CWI following an intense cycling exercise on sleep architecture, despite the fact that PARTIAL CWI induced a decrease in T_{core} during 90 min after bedtime. The maximal rate of decline in T_{core} close to bedtime—characterized by the distal-to-proximal skin temperature gradient and heat dissipation—has been associated with an increased sleep propensity (Berger and Phillips, 1995; Kräuchi and Deboer, 2010). In the present study, a significant increase in SWS proportion (+5.1%) during the first 180 min after sleep onset in WHOLE compared with PARTIAL was observed. The first part of the night is characterized by a high proportion of SWS (Akerstedt and Nilsson, 2003), which is involved in important neurophysiological functions such as growth hormone release, immunity, and cleaning of metabolites (Léger et al., 2018). Consequently, this sleep period may play an important part in the athletes' recovery process. The absence of a main effect of condition on overall night SWS proportion may be explained by the high mean SWS proportion during the CONT condition compared with the normal population (21.8 vs. 16%; Ohayon et al., 2004). As a consequence, a ceiling or maximum level of SWS in a single night (Taylor et al., 1997) beyond which even WHOLE could not induce an additional increase in SWS is possible. Finally, WHOLE appears as a promising strategy to increase SWS proportion during the first part of the night, which are crucial in the athlete's recovery process after training or a competition (Halson and Juliff, 2017; Walsh et al., 2020).

In addition to the positive effect of WHOLE on SWS proportion during the first part of the night, WHOLE and PARTIAL decreased arousals during the whole night compared with CONT. An arousal is defined by the National Sleep Foundation as an abrupt change in activity, which may cause a change in sleep stage from a deep stage of NREM sleep to a light stage, or from REM sleep toward wakefulness, with the possibility of awakening as the final outcome (Ohayon et al., 2017); and it is characteristic of sleep fragmentation (Ekstedt et al., 2004). The origin of an arousal is usually cortical, but it can be generated in response to sensory perturbations such as abnormal movement during sleep (Picchietti and Winkelman, 2005; Tuomilehto et al., 2017). This is supported in the present study by a significant correlation between arousals and limb movements ($r = 0.43$; $p < 0.05$; $n = 34$). Previous studies suggested that overreached athletes are more active during sleep compared with a phase of normal or reduced training load (Taylor et al., 1997; Hausswirth et al., 2014). Some authors proposed that higher muscle fatigue and muscle soreness during intensified training periods promote movements during sleep in an attempt to stay comfortable (Taylor et al., 1997; Sargent et al., 2016). Our results showed

that WHOLE induced a large significant reduction in limb movements compared with CONT during the whole night, which may be a relevant recovery strategy to reduce limb movements and favoring sleep continuity. However, WHOLE did not alleviate muscle soreness in the present study, and its responsibility in limb movement reduction cannot be confirmed. Minett et al. (2014) showed a reduction in prefrontal cortex oxygenation following 20 min CWI up to mesosternal (10°C), which may alter the central motor output (Amann and Kayser, 2009). Future studies are required to ascertain the effects of WHOLE on the central nervous system and limb movement reduction during the night.

In the present study, WHOLE induced a significant decrease in RMSSD and HF compared with PARTIAL and CONT during the first SWS sequence of the night. A marked SDNN reduction was found in WHOLE compared with CONT. These results suggest that WHOLE decreased parasympathetic modulation. The present results differ from those reported elsewhere, which notably demonstrated an increase in parasympathetic activity immediately (Buchheit et al., 2009; Stanley et al., 2013) and the morning after (Al Haddad et al., 2012) PARTIAL CWI. The HRV analysis was presently performed during SWS, which offers a self-controlled and quiet moment of HRV observation (Brandenberger et al., 2005), making comparisons with the previously mentioned studies difficult. However, WHOLE probably induced important thermal stress and sympathetic hyperactivity (Datta and Tipton, 2006), which may explain a decrease in parasympathetic modulation during SWS compared with PARTIAL and CONT conditions. Cardiac parasympathetic activity during recovery from exercise may be indicative of an athlete's readiness to perform a high-intensity exercise the following day (Stanley et al., 2013). In the present study, WHOLE-induced parasympathetic activity decrease may be potentially deleterious for elite athletes in the context of consecutive high performance achievement (Hynynen et al., 2006). WHOLE induced a significant decrease in LF compared with PARTIAL and CONT. However, it has been suggested that LF does not reflect sympathetic nerve activity, whereas the measure of baroreflex function using LF is still being debated (Goldstein et al., 2011; Martelli et al., 2014). Additionally, both thermal sensation and comfort after WHOLE were altered compared with PARTIAL and CONT, which may be a potential barrier to implement WHOLE on the field in addition to the logistics constraints inherent to bathing the entire body (Bishop, 2008). Future studies are required to assess cooling strategies inducing a lesser extent of thermal stress while improving sleep architecture, e.g., head-only CWI.

Based on previous findings (Wilcock et al., 2006; Poppendieck et al., 2013; Stephens et al., 2017), we hypothesized that a higher decline in T_{core} induced by CWI interventions may hasten exercise-induced muscle damage recovery. Our hypothesis was rejected since WHOLE and PARTIAL procured no additional benefits on MVIC, CMJ, [CK], muscle soreness, fatigue general, and stress compared with CONT throughout the 48-h recovery period. These results are consistent with previous studies that reported a trivial/small with moderate-to-high heterogeneity effect induced by CWI on strength, jump performance recovery, and muscle soreness (Leeder et al., 2012; Poppendieck et al., 2013). In addition, we noted a more positive belief in the effectiveness of WHOLE and PARTIAL compared with CONT. Broatch et al. (2014) reported that CWI-induced performance recovery may be at least partially related to a placebo effect, highlighting the importance to encourage athletes' belief in order to improve subjective ratings and performance recovery. Future studies are required to assess the effect of daily cooling strategies on the consecutive night's sleep architecture and recovery kinetics.

LIMITATIONS

Due to practical issues, sleep was monitored using polysomnography only during the night after exercising, and markers of fatigue/muscle damage were not collected beyond 48 h after the simulated trail. Future studies are consequently required to assess the effect of WHOLE on the consecutive night's sleep architecture and recovery kinetics beyond 48 h after exercise. Finally, participants performed the out-of-water condition as a control condition. The absence of a thermoneutral water immersion condition (i.e., hydrostatic pressure without cooling) prevents any conclusion on the potential effect of hydrostatic pressure on sleep and recovery kinetics.

CONCLUSION

The present study showed that WHOLE and PARTIAL CWI performed after a high-intensity, intermittent running exercise largely decreased sleep arousals compared with CONT, while WHOLE only decreased limb movements compared with CONT. This study contributes to the development of strategies to enhance sleep and recovery for elite athletes who are exposed to periods of intense training/competition and disturbed sleep (Gupta et al., 2017). Our results suggest that WHOLE may be particularly useful for athletes to reduce limb movements and sleep arousals after exercise. However, WHOLE and PARTIAL did not hasten the recovery process compared with CONT during the 48-h follow-up period, and WHOLE decreased parasympathetic activity. Furthermore, the use of WHOLE by athletes on the field may be hampered by poor sensation and thermal comfort. Future studies should be conducted to explore the potential benefits of solely cooling the head—inducing a lesser extent of thermal stress—to increase sleep propensity during a training and competition period.

DATA AVAILABILITY STATEMENT

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

ETHICS STATEMENT

The studies involving human participants were reviewed and approved by the local ethics committee (East III, France.

Ref. 170605). The patients/participants provided their written informed consent to participate in this study.

AUTHOR CONTRIBUTIONS

MC and MN conceived and designed the research and analyzed and interpreted data. MC conducted experiments, wrote the first draft of the manuscript, and contributed to the writing of the final paper. FP, VG, and AA contributed to the data collection. All authors contributed to the article and approved the submitted version.

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SUPPLEMENTARY MATERIAL

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fspor.2021.659990/full#supplementary-material

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REVIEW
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Post-exercise Cold Water Immersion Effects on Physiological Adaptations to Resistance Training and the Underlying Mechanisms in Skeletal Muscle: A Narrative Review

Aaron C. Petersen[1]* *and Jackson J. Fyfe*[2]

[1] *Institute for Health and Sport, Victoria University, Melbourne, VIC, Australia,* [2] *Deakin University, Institute for Physical Activity and Nutrition (IPAN), School of Exercise and Nutrition Science, Geelong, VIC, Australia*

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***Correspondence:**
Aaron C. Petersen
aaron.petersen@vu.edu.au

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Post-exercise cold-water immersion (CWI) is a popular recovery modality aimed at minimizing fatigue and hastening recovery following exercise. In this regard, CWI has been shown to be beneficial for accelerating post-exercise recovery of various parameters including muscle strength, muscle soreness, inflammation, muscle damage, and perceptions of fatigue. Improved recovery following an exercise session facilitated by CWI is thought to enhance the quality and training load of subsequent training sessions, thereby providing a greater training stimulus for long-term physiological adaptations. However, studies investigating the long-term effects of repeated post-exercise CWI instead suggest CWI may attenuate physiological adaptations to exercise training in a mode-specific manner. Specifically, there is evidence post-exercise CWI can attenuate improvements in physiological adaptations to resistance training, including aspects of maximal strength, power, and skeletal muscle hypertrophy, without negatively influencing endurance training adaptations. Several studies have investigated the effects of CWI on the molecular responses to resistance exercise in an attempt to identify the mechanisms by which CWI attenuates physiological adaptations to resistance training. Although evidence is limited, it appears that CWI attenuates the activation of anabolic signaling pathways and the increase in muscle protein synthesis following acute and chronic resistance exercise, which may mediate the negative effects of CWI on long-term resistance training adaptations. There are, however, a number of methodological factors that must be considered when interpreting evidence for the effects of post-exercise CWI on physiological adaptations to resistance training and the potential underlying mechanisms. This review outlines and critiques the available evidence on the effects of CWI on long-term resistance training adaptations and the underlying molecular mechanisms in skeletal muscle, and suggests potential directions for future research to further elucidate the effects of CWI on resistance training adaptations.

Keywords: cold-water immersion, resistance exercise, exercise performance, skeletal muscle, molecular responses, adaptation

INTRODUCTION

Cold water immersion (CWI) is a popular recovery strategy aimed at enhancing recovery from strenuous exercise. Typical CWI protocols involve the submersion of the limbs and/or torso for ∼5–20 min in water cooled to temperatures of between ∼8–15°C (Versey et al., 2013). Application of CWI usually occurs shortly after exercise cessation and may be performed either continuously [e.g., 1 bout of 15 min at 10°C (Fyfe et al., 2019)] or intermittently [e.g., 3 bouts of 4 min at ∼12°C with 30 s between bouts (Frohlich et al., 2014)].

Application of CWI has been associated with a number of short-term benefits related to post-exercise recovery [as reviewed in Versey et al. (2013)], including a faster recovery of muscle strength (Skurvydas et al., 2006; Bailey et al., 2007; Vaile et al., 2008), muscle soreness (Bailey et al., 2007; Vaile et al., 2008; Ingram et al., 2009; Rowsell et al., 2011; Stanley et al., 2012), perception of fatigue (Parouty et al., 2010; Stacey et al., 2010; Rowsell et al., 2011; Stanley et al., 2012), and markers of inflammation (Montgomery et al., 2008; Peake et al., 2008; Stacey et al., 2010; Pournot et al., 2011) and muscle damage (Eston and Peters, 1999; Skurvydas et al., 2006) after strenuous exercise. However, evidence of the short-term benefits of CWI is equivocal, with some studies finding no influence of CWI on various aspects of post-exercise recovery including muscle strength (Paddon-Jones and Quigley, 1997; Goodall and Howatson, 2008; Howatson et al., 2009; Jakeman et al., 2009; Peiffer et al., 2009), muscle soreness (Paddon-Jones and Quigley, 1997; Sellwood et al., 2007; Howatson et al., 2009; Jakeman et al., 2009), and markers of muscle damage (Eston and Peters, 1999; Bailey et al., 2007; Goodall and Howatson, 2008; Jakeman et al., 2009) and inflammation (Montgomery et al., 2008; Peake et al., 2008). The potential short-term benefits of CWI are nevertheless thought to be primarily mediated by the local vasoconstriction and increased hydrostatic pressure attributed to the cold water temperature and depth associated with CWI, respectively (Wilcock et al., 2006). These factors are thought to exert various physiological effects, including decreased metabolic activity (Ihsan et al., 2013), altered hormonal responses (Earp et al., 2019), infiltration of immune cells (Lee et al., 2005), and reduced limb blood flow (Gregson et al., 2011; Mawhinney et al., 2013, 2017). Ultimately, these purported short-term benefits of CWI are theorized to enhance physiological adaptations to exercise training by improving the quantity and/or quality of subsequent training sessions (Barnett, 2006).

While post-exercise application of CWI can accelerate aspects of post-exercise recovery and enhance subsequent exercise performance, there is accumulating evidence that CWI can influence long-term physiological adaptations to exercise, and in a manner that is exercise mode-specific (Malta et al., 2020). For example, there is accumulating evidence that CWI can attenuate improvements in physiological adaptations to resistance training (including muscle hypertrophy and improvements in strength and power/rate of force development) (Roberts et al., 2015; Fyfe et al., 2019; Poppendieck et al., 2020), whereas CWI associated with endurance training does not appear to influence related adaptations including improvements in cycling time trial performance (either mean power or duration) or maximal aerobic power (Yamane et al., 2006; Halson et al., 2014; Broatch et al., 2017). Mechanistically, the mode-specific influence of CWI on physiological adaptations to exercise training is likely attributed to the short-term physiological effects of CWI on post-exercise molecular-level responses (in skeletal muscle in particular) that mediate physiological adaptations to exercise training.

The following sections will firstly summarize and critique the evidence for the influence of CWI on physiological adaptations to resistance training, including skeletal muscle hypertrophy and improvements in measures of maximal strength, strength endurance, and power/rate of force development, before discussing the potential molecular-level mechanisms in skeletal muscle underlying these effects. Finally, the limitations of current evidence, as well as potential directions for future research, are discussed.

INFLUENCE OF CWI ON PHYSIOLOGICAL ADAPTATIONS TO RESISTANCE TRAINING

Accumulating evidence suggests post-exercise CWI modulates physiological adaptations to exercise training in a mode-specific manner, with a negative influence on aspects of resistance training adaptations, but not on endurance training adaptations (Malta et al., 2020). Modes of exercise can be broadly defined as either endurance/aerobic training, consisting of relatively low-force muscle contractions performed for prolonged durations (such as running/cycling/swimming), or resistance/strength training, characterized by relatively high-force yet brief contractions performed intermittently. The principle of specificity in exercise training dictates that physiological responses, and in turn physiological adaptations, to exercise are highly specific to the mode of exercise performed. Resistance training is the most effective non-pharmacological intervention known to increase skeletal muscle mass and improve both the capacity (strength) and rate (power) of force production by skeletal muscle. For this reason, resistance training (particularly the associated improvements in force production ability) can aid performance enhancement in various athletic disciplines (Suchomel et al., 2016), and also attenuate declines in these parameters occurring across the lifespan that can impair functional ability and increase the risk of both morbidity and mortality (Maestroni et al., 2020).

Given the importance of physiological adaptations to resistance training for optimizing performance and health outcomes, factors that influence the magnitude of these adaptations have critical importance for maximizing the benefits of resistance training. Owing to the popularity of CWI as a post-exercise recovery technique, the potential influence of CWI on physiological adaptations to exercise training, including resistance training, has received increased attention in the literature. The following sections will describe the growing evidence that CWI can influence changes in various physiological adaptations to resistance training, including skeletal muscle hypertrophy, maximal strength, strength endurance, and aspects

of power/rate of force development (RFD). While outside the scope of this narrative review, readers are instead referred elsewhere for discussion of the effects of post-exercise CWI on physiological adaptations to endurance training (Broatch et al., 2018; Malta et al., 2020). A summary of studies investigating the effects of CWI on physiological adaptations to resistance training is provided in **Table 1**.

Skeletal Muscle Hypertrophy

Resistance training is a well-established strategy for increasing skeletal muscle mass—a process known as skeletal muscle hypertrophy (Haun et al., 2019). Before discussing current evidence for the influence of post-exercise CWI application on muscle hypertrophic responses to resistance training, there are several important conceptual and methodological factors related to the assessment of muscle hypertrophy worthy of consideration.

Skeletal muscle hypertrophy is a complex biological construct that may be assessed at different physiological levels (i.e., whole-body, macroscopic, microscopic, and molecular levels), and by using various measurement techniques each differing in aspects of validity, reliability, and specificity (Haun et al., 2019). Whole-body assessments typically measure changes in total or regional lean body/fat-free mass using methods such as Dual X-ray Absorptiometry (DXA), air displacement plethysmography (e.g., BodPod), or bioelectrical impedance analysis/spectroscopy (BIA/BIS). Macroscopic assessments of muscle hypertrophy typically assess changes in whole-muscle/limb size or cross-sectional area (CSA) *via* imaging techniques (such as MRI, CT, or ultrasound) or anthropometric (e.g., limb girth) measurements. Microscopic assessments of muscle hypertrophy assess changes in muscle fiber size and/or muscle fiber type by applying immunohistochemical techniques to skeletal muscle samples obtained *via* muscle biopsy. Less commonly applied in contemporary human exercise studies, molecular-level assessments of muscle hypertrophy involve the quantification of protein sub-fractions (e.g., myofibrillar or sarcoplasmic protein concentrations) within skeletal muscle samples obtained *via* muscle biopsy.

Human studies performed to date have investigated whether CWI influences skeletal muscle hypertrophic responses to resistance training at the whole-body (Fyfe et al., 2019), macroscopic (Ohnishi et al., 2004; Yamane et al., 2006, 2015; Roberts et al., 2015; Poppendieck et al., 2020), and microscopic (Roberts et al., 2015; Fyfe et al., 2019) levels (**Figure 1**). The findings of these studies have been mixed, with some suggesting CWI attenuates resistance training-induced increases in whole-muscle/limb size or cross-sectional area (CSA) (Roberts et al., 2015; Yamane et al., 2015; Poppendieck et al., 2020) and muscle fiber CSA (Roberts et al., 2015; Fyfe et al., 2019), while others have shown no influence of CWI on changes in either muscle/limb size or CSA (Ohnishi et al., 2004; Yamane et al., 2006) or total body or regional lean mass (assessed *via* DXA) (Fyfe et al., 2019) with resistance training.

Three studies (Roberts et al., 2015; Yamane et al., 2015; Poppendieck et al., 2020) have provided evidence for attenuated macroscopic-level (whole-muscle) hypertrophy following resistance training with CWI application. In the only study performed to date using a gold-standard assessment of muscle mass or CSA (MRI) (Roberts et al., 2015), post-exercise application of CWI (10 min at 10.1 ± 0.3°C) attenuated the increase in quadriceps muscle mass (∼+15% for control vs. ∼+2% for CWI) after 12 weeks of resistance training in young resistance-trained men. Two other studies in young, non-resistance trained males (Yamane et al., 2015) or resistance-trained males and females (Poppendieck et al., 2020) found CWI blunted the resistance training-induced increases in both forearm circumference and wrist flexor muscle thickness (Yamane et al., 2015) and in both thigh circumference and quadriceps (vastus medialis) muscle thickness (Poppendieck et al., 2020). The remaining studies that assessed macroscopic-level muscle hypertrophy found no influence of CWI on resistance training-induced changes in total or regional lean body mass (assessed *via* DXA) (Fyfe et al., 2019), wrist flexor muscle thickness (ultrasound) (Yamane et al., 2006), or forearm circumference (assessed anthropometrically) (Ohnishi et al., 2004) in young, non-resistance trained males.

While the majority of studies performed to date have assessed the influence of CWI on macroscopic-level muscle hypertrophy following resistance exercise, two studies (Roberts et al., 2015; Fyfe et al., 2019) have examined microscopic-level hypertrophic responses. Both studies showed that CWI attenuated the resistance training-induced increase in vastus lateralis type II muscle fiber area, with one study (Roberts et al., 2015) also suggesting that combined type I and type II muscle fiber areas (which may have been driven by the change in type II muscle fiber area) were enhanced by resistance training only with an active post-recovery (low-intensity cycling), but not with CWI.

To summarize, there is mixed evidence for the influence of CWI on indices of skeletal muscle hypertrophy, with three of six total studies showing attenuated whole-muscle hypertrophy of either the thigh (Roberts et al., 2015; Poppendieck et al., 2020) or wrist flexor (Yamane et al., 2015) musculature, and both of two available studies (Roberts et al., 2015; Fyfe et al., 2019) showing a negative influence of CWI on muscle fiber (specifically type II) hypertrophy. There is also no evidence that post-exercise CWI has beneficial effects on measures of skeletal muscle hypertrophy.

Maximal Strength

Maximal strength is defined as the capacity of the neuromuscular system to produce force against an external resistance (Suchomel et al., 2016), and may be assessed using multiple methods including dynamic strength [involving concentric and/or eccentric actions, typically assessed as the one-repetition maximum (1-RM) load for a given exercise], isometric strength, or isokinetic strength. Improvements in maximal strength occur due to a combination of neural and morphological adaptations (Folland and Williams, 2007), with the relative contribution of these factors to strength gain with resistance training subject to ongoing debate (Loenneke et al., 2019; Taber et al., 2019). Post-exercise CWI application may theoretically impair strength development with resistance training by interfering with the morphological contributors (e.g., muscle

TABLE 1 | Summary of post-exercise cold-water immersion effects on physiological adaptations to resistance training.

Study	Participants			Study design	Recovery intervention	Resistance training intervention				Main findings
	Sample size	Age	Resistance training status			Intervention length	Exercises trained	Frequency	Volume/ intensity	
Ohnishi et al. (2004)	16 (M)	20.1 ± 2.3 y	Not described	Within-subject/parallel group, repeated measures	CWI: 20 min at 10 ± 1°C CON: Passive sitting for 20 min	6 weeks	Handgrip exercise	3 ×/week	3 × 8-RM	Muscle hypertrophy ↔ Forearm circumference for both control and CWI groups Maximal strength ↔ Isometric (handgrip) strength for both control and CWI groups Strength endurance ↑Number of handgrips (30% 1-RM until volitional fatigue) for both control and CWI groups • No difference between groups
Yamane et al. (2006)	11 (7 M, 4 F)	20.5 ± 0.8 y	Not described	Within-subject, repeated measures	CWI: 20 min at 10 ± 1°C CON: Non-immersion at 25 ± 1°C	4 weeks	Wrist flexion exercise	3 x/week	3 × 8-RM (2 min rest between sets)	Maximal strength ↑ Isometric (handgrip) strength for both control and CWI groups • Greater ↑ for control vs. CWI group Strength endurance ↑ Number of handgrips (30% 1-RM until volitional fatigue) for control group, but ↔ for CWI group
	16 (M)	20.7 ± 2.3 y	Not described	Parallel-group, repeated measures	CWI: 20 min at 10 ± 1°C CON: Non-immersion at 25 ± 1°C	4 weeks	Wrist flexion exercise	3 x/week	3 × 8-RM (2 min rest between sets)	Muscle hypertrophy ↔ Muscle thickness (wrist flexors, ultrasound) for both control and CWI groups Maximal strength ↔ Isometric (handgrip) strength for both control and CWI groups Strength endurance ↑ Number of handgrips (30% 1-RM until volitional fatigue) for both control and CWI groups • No difference between groups
Frohlich et al. (2014)	17 (M)	23.5 ± 2.4 y	At least 6 months of resistance training experience (range 6 months to 5 years).	Within-subject, repeated measures	CWI: 3 * 4 min at 12 ± 1.5°C CON: Non-immersion at 20–23°C	5 weeks	Leg curl	2 x/week	3 × 8–12 repetitions (75–80% 1-RM)	Maximal strength ↑ Dynamic (both 1-RM and 12-RM leg curl) strength for both groups • Greater ↑ in 12-RM for control vs. CWI group • No difference in 1-RM between groups

(Continued)

TABLE 1 | Continued

Study	Participants			Study design	Recovery intervention	Resistance training intervention				Main findings
	Sample size	Age	Resistance training status			Intervention length	Exercises trained	Frequency	Volume/ intensity	
Yamane et al. (2015)	14 (M)	20.2 ± 0.9 y	Recreationally active with no resistance training experience in past year.	Within-subject/parallel group, repeated measures	CWI: 20 min at 10 ± 1°C CON: Non-immersion at room temperature	6 weeks	Wrist flexion exercise	3 x/week	5 × 8 repetitions at 70–80% 1-RM)	Muscle hypertrophy ↑ Muscle thickness (wrist flexors, ultrasound) and forearm circumference for both control and CWI groups • Greater↑ in both measures for control vs. CWI Maximal strength ↑ Maximal isometric (wrist flexor) strength for control group, but ↔ for CWI group Strength endurance ↑ Number of handgrips (35% 1-RM until volitional fatigue) for both control and CWI groups • No difference between groups
Roberts et al. (2015)	21 (M)	21.2 ± 2.2 (CWI group) 21.3 ± 1.9 y (CON group)	At least 12 months experience with resistance training.	Parallel-group, repeated measures	CWI: 10 min at 10.1 ± 0.3°C CON: 10 min active recovery (cycling) at self-selected low intensity (∼60 W)	12 weeks	Leg press Knee extension Knee flexion Walking lunges Plyometric exercises (drop jumps, slow eccentric squat jumps, split lunge jumps, countermovement box jumps)	2 x/week	3–6 × 8–12 RM (1 min rest between sets)	Muscle hypertrophy ↑ Muscle mass (quadriceps, MRI) for both control and CWI groups • Greater ↑ in for control vs. CWI group ↑ Muscle fiber CSA (type II and combined type I + type II) for control group, but ↔ for CWI group Maximal strength ↑ Dynamic 1-RM (leg press and leg extension) strength for both control and CWI groups • ↑ Post-training values for control vs. CWI groups ↑ Isometric (knee extensor, 70°) torque for control group, but ↔ for CWI group • ↑ Post-training values for control vs. CWI groups ↔ Isokinetic (knee extensor, 90°/s) strength for both control and CWI groups

(Continued)

TABLE 1 | Continued

Study	Participants			Study design	Recovery intervention	Resistance training intervention				Main findings
	Sample size	Age	Resistance training status			Intervention length	Exercises trained	Frequency	Volume/ intensity	
										Strength endurance ↑ Isokinetic work (knee extensors, contractions 1–25 of 50, 90°/s) for control group, but ↔ for CWI group ↔ Isokinetic work (knee extensors, contractions 26–50 of 50, 90°/s) for either control or CWI groups Power/RFD ↑ Isometric RFD impulse (knee extensors, 70°) for both control and CWI groups • ↑ Post-training values for control vs. CWI groups
Fyfe et al. (2019)	16 (M)	25.0 ± 4.9 y	Recreationally-active, no resistance training experience in past 6 months	Parallel-group, repeated measures	CWI: 15 min at 10°C CON: Non-immersion at 23°C	7 weeks	Back squat Barbell bench press Lat pulldown Walking lunges Shoulder press Bicep curl Tricep extension Lying leg raise (+ variants for each performed on alternate days)	3 x/week	3 × 12-RM or 20-RM (2 min recovery between sets)	Muscle hypertrophy ↑ Lean mass (total, lower-body and upper-body, DXA) for both control and CWI groups (combined) • No difference between groups ↔ Muscle fiber (type I) CSA for both groups combined • Greater ↑ in muscle fiber (type II) CSA for the control vs. CWI groups Maximal strength ↑ Dynamic 1-RM (leg press and bench press) strength for both control and CWI groups (combined) • No difference between groups Power/RFD ↑ CMJ peak force for control group but ↔ for CWI group • Greater ↑ in for control vs. CWI group ↔ Peak force during squat jump or ballistic push-up for both control and CWI groups (combined)

(Continued)

TABLE 1 | Continued

Study	Participants			Study design	Recovery intervention	Resistance training intervention				Main findings
	Sample size	Age	Resistance training status			Intervention length	Exercises trained	Frequency	Volume/ intensity	
Poppendieck et al. (2020)	11 (9M, 2F)	25.3 ± 3.6 y	At least 6 months of resistance training experience (1–2 sessions per week).	Parallel-group, repeated measures	CWI: 10 min at 14–15°C	8 weeks	Leg press Leg curl Leg extension	3 x/week	3 × 10-RM (3 min recovery between sets)	Muscle hypertrophy ↑ Muscle thickness (vastus medialis, ultrasound) and thigh circumference for the control group, but ↔ for the CWI group • Small (g = 0.27) and large (g = 1.20) effects favoring the control vs. CWI group for leg circumference and muscle thickness, respectively Maximal strength ↔ Dynamic 1-RM (leg press) strength for both control and CWI groups Power/RFD ↔ CMJ height for both control and CWI groups

1-RM, one-repetition maximum; CSA, cross-sectional area; CON, control; CWI, cold water immersion; RFD, rate of force development; DXA, dual x-ray absorptiometry; CMJ, countermovement jump; ↑, statistically significant ($p < 0.05$) increase with training, ↓ statistically significant ($p < 0.05$) decrease with training, ↔ no statistically significant ($p > 0.05$) change with training.

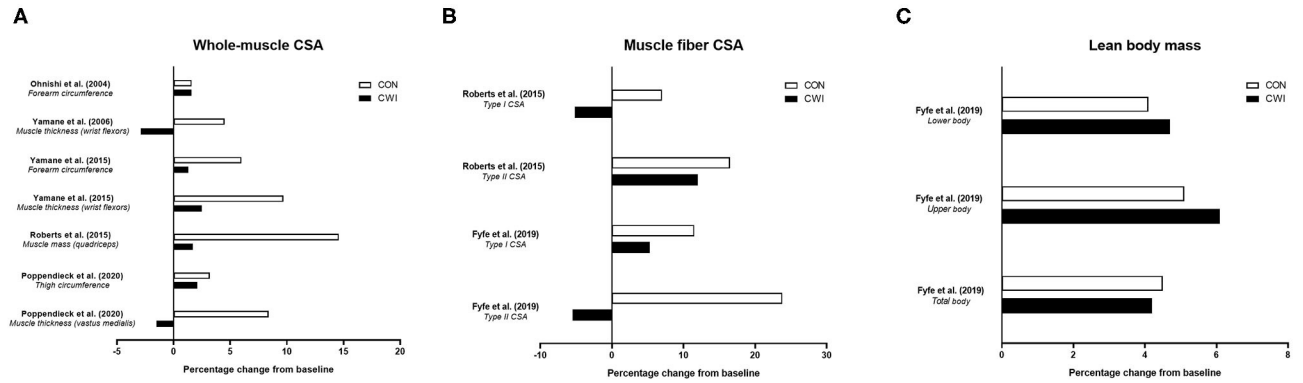

FIGURE 1 | Summary of studies investigating the effects of post-exercise cold water immersion (CWI) on muscle hypertrophic adaptations to resistance training, including effects (shown as mean percentage changes from baseline to post-training) on measures of whole-muscle cross-sectional area (CSA; **A**), muscle fiber CSA **(B)**, and lean body mass **(C)**.

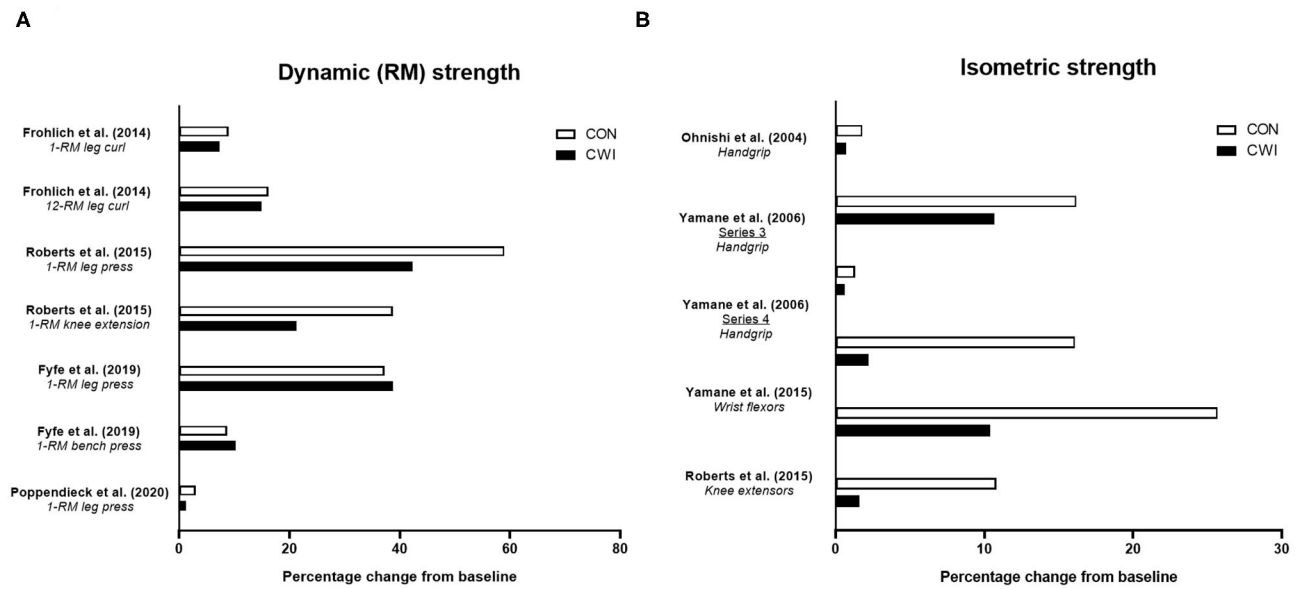

FIGURE 2 | Summary of studies investigating the effects of post-exercise cold water immersion (CWI) on changes in maximal strength with resistance training, including effects (shown as mean percentage changes from baseline to post-training) on dynamic repetition-maximum (RM) strength **(A)** and isometric or isokinetic strength **(B)**. Adapted from Broatch et al. (2018) with permission.

hypertrophy) to improved strength, while the potential effects of CWI on neural adaptations to resistance training remain unclear.

To date, studies have shown mixed findings on the influence of CWI on improvements in various measures of strength with resistance training (**Figure 2**) (Ohnishi et al., 2004; Yamane et al., 2006, 2015; Frohlich et al., 2014; Roberts et al., 2015; Fyfe et al., 2019; Poppendieck et al., 2020). Four studies (Frohlich et al., 2014; Roberts et al., 2015; Fyfe et al., 2019; Poppendieck et al., 2020) have examined the influence of CWI on resistance training-induced changes in dynamic repetition-maximum (RM) strength and five studies (Ohnishi et al., 2004; Yamane et al., 2006, 2015; Frohlich et al., 2014; Roberts et al., 2015) have assessed isometric strength, while only one study (Roberts et al., 2015) has determined changes in isokinetic strength.

In the first study [of only three total studies (Frohlich et al., 2014; Roberts et al., 2015; Poppendieck et al., 2020)] to determine the influence of post-exercise CWI on dynamic RM strength gains with resistance training in participants with resistance training experience, Frohlich et al. (2014) assessed the influence of post-exercise intermittent CWI (3 bouts of 4 min at 12 ± 1.5°C) on dynamic (both 1-RM and 12-RM) leg curl strength gain after 5 weeks of resistance training in young males. The

findings suggested post-exercise CWI application impaired the change in 12-RM strength (∼+16% for control vs. ∼+15% for CWI), while there was a tendency for greater 1-RM strength gain in the control group (∼+9 vs. ∼+7% for the CWI group) that was not statistically significant. In young resistance-trained males and females, Poppendieck et al. (2020) also found no influence of post-exercise CWI on 1-RM (leg press) strength gain, which did not improve in either the control or CWI group (∼+3% vs. ∼+1.5%, respectively) following 8 weeks of resistance training, although there was a small effect for less 1-RM strength gain in the CWI group. The findings of these studies were, however, contrasted by Roberts et al. (2015) who noted that alongside the attenuated muscle hypertrophy responses observed, post-exercise CWI application blunted the improvement in 1-RM leg press strength (∼+59 vs. ∼+42% for control and CWI, respectively), 1-RM knee extension strength (∼+39 vs. ∼+21%, respectively), and isometric knee extensor strength (∼+26 vs. ∼+10%, respectively) after 12 weeks of resistance training in young, resistance-trained males.

Taken together, these studies provide mixed evidence for attenuated dynamic, lower-body RM (1-RM or 12-RM) strength gain following resistance training with CWI application in resistance-trained individuals (Frohlich et al., 2014; Roberts et al., 2015; Poppendieck et al., 2020). More recently, findings from our laboratory in non-resistance-trained males (Fyfe et al., 2019) suggested CWI did not impair dynamic strength development of either the lower- (1-RM leg press) or upper-body (1-RM bench press) after 7 weeks of resistance training. The lack of negative influence of CWI on strength gain occurred despite CWI impairing vastus lateralis type II muscle fiber hypertrophy (but not total or regional lean body mass assessed *via* DXA), highlighting the potential disconnect between changes in measures of strength and muscle hypertrophy with resistance training (Loenneke et al., 2019).

Current evidence (Roberts et al., 2015; Fyfe et al., 2019) therefore provides mixed support for the notion that attenuated strength gain following resistance training with CWI application may be mediated by the negative effects of post-exercise CWI on skeletal muscle hypertrophy. We (Fyfe et al., 2019) theorized the discrepancies in findings on the influence of CWI on dynamic 1-RM strength gain between our study and Roberts et al. (2015) may have related to differences in task complexity of the strength measures chosen and the associated implications for the relative contribution of hypertrophic and non-hypertrophic mechanisms to strength gain. More complex motor tasks likely evoke a greater neural (i.e., non-hypertrophic) contribution to strength gain with resistance training (Rutherford and Jones, 1986), and neural adaptations may be less susceptible to interference from CWI compared with morphological adaptations (e.g., muscle hypertrophy). For this reason, it is possible that strength gain may be attenuated to a greater extent with CWI when assessed during less-complex movements (e.g., or isometric vs. dynamic exercises, or single-joint vs. multi-joint dynamic exercises) that likely involve a greater relative contribution of hypertrophic adaptations to strength gain. Nevertheless, findings on the influence of CWI on isometric strength assessed during less-complex movements has also been mixed. In contrast with the findings of Roberts et al. (2015) who observed a blunted improvement in isometric knee extensor strength following resistance training with CWI, Frohlich et al. (2014) found no influence of CWI on the improvement in isometric knee flexor strength with resistance training. Two other studies from Yamane and colleagues (Yamane et al., 2006, 2015) showed mixed effects of CWI on isometric wrist flexor strength with resistance training, with one study (Yamane et al., 2015) suggesting impaired isometric strength development with CWI, and the other (Yamane et al., 2006) showing no improvement in either group—a conclusion shared with earlier findings by Ohnishi et al. (2004). It is also possible that between-study differences in the resistance training status of the participants studied may explain the discrepant findings regarding the influence of post-exercise CWI on strength gain with resistance training. Since the relative magnitude of strength gain is larger in untrained vs. resistance-trained individuals, and is largely mediated by neural (i.e., non-hypertrophic) adaptations (Del Vecchio et al., 2019a), post-exercise CWI may therefore have less influence on strength gain in untrained populations. Nevertheless, further studies are needed to confirm whether resistance training status indeed influences the effects of post-exercise CWI on strength gain with resistance training.

In summary, only limited evidence exists on the influence of CWI on isokinetic strength development, with one study (Roberts et al., 2015) showing maximal isokinetic knee extension torque was not improved following resistance training combined with either CWI or control. There is mixed evidence on the influence of post-exercise CWI application on improvements in dynamic 1-RM and isometric strength with resistance training, with limited evidence on isokinetic strength gain. Only single studies have shown clear effects for blunted dynamic 1-RM (leg press) (Roberts et al., 2015) or 12-RM (leg curl) (Frohlich et al., 2014) strength gain with CWI, both in resistance-trained participants, and in isometric strength gain of the knee flexors (Roberts et al., 2015) or wrist flexors (Yamane et al., 2015) in those with and without resistance training experience, respectively. Although the findings of the available literature on the influence of CWI on strength development with resistance training are mixed, a recent meta-analysis (Malta et al., 2020) nevertheless concluded that post-exercise CWI attenuated the improvements in both dynamic (1-RM; ES = −0.50) and isometric (ES = −0.65) strength.

Strength Endurance

Strength endurance (also known as local muscular endurance) describes the ability to withstand fatigue during sustained force production, which is underpinned by various physiological factors, including mitochondrial and capillary density, muscle fiber-type proportions, and muscle buffer capacity (Kraemer and Ratamess, 2004). Resistance training, particularly when sets are performed with lighter loads (e.g., ≥12–15-RM) for a prolonged duration and with minimal between-set recovery (e.g., ≤1 min), is a well-established strategy for improving strength endurance. Application of CWI could theoretically influence improvements in strength endurance by modulating changes in the aforementioned factors with resistance training

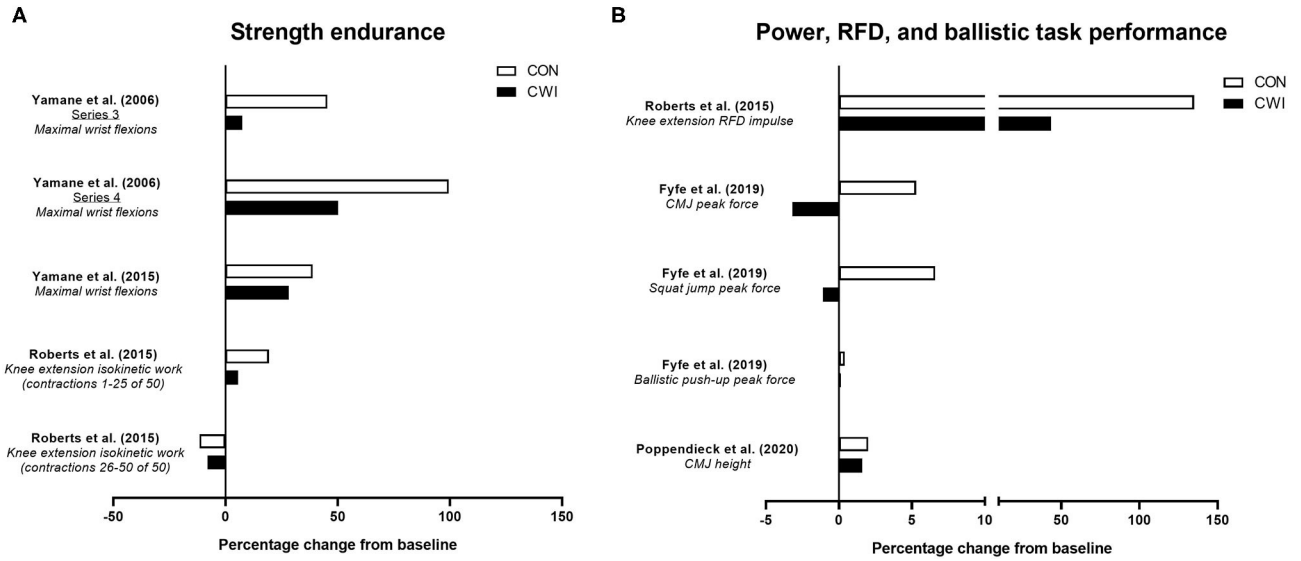

FIGURE 3 | Summary of studies investigating the effects of post-exercise cold water immersion (CWI) on changes in strength endurance **(A)** and measures related to power, rate of force development (RFD) or ballistic task performance **(B)** with resistance training. Effects are shown as mean percentage changes from baseline to post-training.

(as discussed in section Effects of CWI on Molecular Responses to Resistance Training).

Four studies have determined the influence of CWI on improvements in strength endurance of the wrist flexors (Ohnishi et al., 2004; Yamane et al., 2006, 2015) or knee extensors (Roberts et al., 2015) with resistance training (**Figure 3A**). In an initial study, Ohnishi et al. (2004) found strength endurance of the wrist flexors improved after 6 weeks of handgrip resistance training (3 × 8-RM) either with post-exercise CWI (20 min at ∼10°C) of the forearm or with the control (no water immersion) group, but noted the improvement tended to be smaller in the CWI condition. Follow-up studies (Yamane et al., 2006, 2015) using an identical CWI protocol undertaken after similar RT protocols (or passive control) showed mixed effects of CWI on strength endurance. One study (Yamane et al., 2006) showed that in one cohort of participants, CWI blunted the improvement in wrist flexor strength endurance vs. the control group, while in a second cohort of participants, similar improvements in strength endurance occurred in both the CWI and control groups. A more recent study (Yamane et al., 2015) found that improvements in wrist flexor strength endurance occurred following both CWI and control, although there appeared to be less improvement following CWI. Although not a traditional measure of strength endurance *per se*, Roberts et al. (2015) found that isokinetic work completed over the first 25 of 50 total contractions was improved in the control group, but not in the CWI group, suggesting less improvement in aspects of strength endurance with CWI.

The limited available evidence therefore suggests CWI may attenuate improvements in strength endurance with resistance training, albeit when assessed during single-joint movements involving smaller muscle groups (i.e., wrist flexors). The physiological mechanisms for the negative effects of CWI on changes in strength endurance with resistance training remain unclear.

Power, Rate of Force Development, and Ballistic Task Performance

The ability to produce force rapidly (variously described as mechanical power or rate of force development) is recognized as an important component of athletic performance (Cormie et al., 2011) and a key contributor to declines in functional capacity with aging (Foldvari et al., 2000). Neural adaptations are a key determinant of rate of force development (Del Vecchio et al., 2019b), with a smaller relative contribution from muscle morphological factors due to their association with maximal voluntary strength/torque (Maden-Wilkinson et al., 2021).

Resistance training is a well-established strategy for improving various aspects of power development (Cormie et al., 2011), and a limited number of studies have determined whether post-exercise CWI influences power-related adaptation to resistance training (**Figure 3B**). Roberts et al. (2015) were the first to show that power-related adaptations, specifically the improvement in isometric rate of force development of the knee extensors, was blunted (∼+135% for control vs. ∼+44% for CWI) after 12 weeks of resistance training with CWI application. While this suggests CWI can impair resistance training-induced improvements in rate of force development during simple, isometric movements, other studies have determined whether CWI influences performance improvements in rapid, dynamic movements such as the countermovement jump (CMJ) (Fyfe et al., 2019; Poppendieck et al., 2020), which may be more relevant to both athletic performance and activities of daily living. For example, we (Fyfe et al., 2019) showed the improvement in peak force during a CMJ (but not during a squat jump

or ballistic push-up) with resistance training was impaired (∼+5% for control vs. ∼-3% for CWI) with post-exercise CWI, suggesting CWI application may compromise improvements in the ability to produce force during rapid, dynamic movements. The limited evidence on the influence of CWI application on CMJ performance outcomes with resistance training is however equivocal, with Poppendieck et al. (2020) finding no change in CMJ height after 8 weeks of resistance training (with or without CWI), although moderate negative effects of CWI were noted after a 3-week follow-up period.

Taken together, the limited available evidence suggests that improvements in the ability to produce force rapidly during either isometric or dynamic (CMJ) movements with resistance training may be compromised by post-exercise CWI application. Whether these effects are attributed to the influence of CWI on morphological and/or neural adaptations is, however, unclear.

EFFECTS OF CWI ON MOLECULAR RESPONSES TO RESISTANCE TRAINING

Several studies have investigated the effects of CWI on the molecular responses to resistance training to try to identify the mechanisms by which CWI attenuates phenotypic adaptations to resistance training. A summary of studies investigating the potential molecular mechanisms that may contribute to the effects of CWI on adaptations to resistance training in human skeletal muscle is provided in **Table 2**. An integrated summary of these molecular mechanisms demonstrating their interactions and potential links to performance outcomes is shown in **Figure 4**. The following section of the review discusses the effects of CWI on each of the mechanisms identified in **Figure 4**.

Anabolic Responses

Protein Synthesis

As discussed previously (section Skeletal Muscle Hypertrophy), CWI can attenuate measures of muscle hypertrophy in response to resistance training (Roberts et al., 2015; Yamane et al., 2015; Fyfe et al., 2019; Poppendieck et al., 2020). Muscle hypertrophy in response to resistance training is driven primarily through transient increases in muscle protein synthesis (Biolo et al., 1997) and decreases in protein breakdown (Phillips et al., 1997), which suggests CWI may alter rates of protein synthesis and/or breakdown in response to resistance exercise.

Only one study to date has investigated the effects of CWI on muscle protein synthesis (Fuchs et al., 2020), demonstrating that 20 min of CWI (single-leg immersion in 8°C) attenuated myofibrillar protein synthesis rate by ∼20% in the 5 h recovery period following an acute resistance exercise bout compared to the contralateral leg, which was immersed in 30°C water. These authors also investigated the chronic effects of post-exercise CWI on myofibrillar protein synthesis by applying D_2O tracer methodology during a 2-week resistance training program consisting of seven lower-body training sessions. Single-leg immersion for 20 min in 8°C water after each training session reduced daily myofibrillar protein synthesis by 12% compared to the contralateral leg (Fuchs et al., 2020). Based on the limited available evidence, it therefore appears that CWI impairs the synthesis of muscle proteins in response to acute and chronic resistance exercise. Presumably, reduced muscle protein synthesis is a major contributor to the potential impairments in resistance training-induced muscle growth with CWI application; however, since changes in muscle size were not measured concurrently with muscle protein synthesis rates (Fuchs et al., 2020), this cannot be directly inferred. Future studies, which include concurrent measurement of protein synthesis and muscle size following resistance training with CWI application are needed to resolve this.

Anabolic Signaling

Transient increases in muscle protein synthesis in response to resistance exercise are primarily regulated by the mechanistic target of rapamycin complex 1 (mTORC1) signaling pathway, which controls protein translation by the ribosome (Bodine et al., 2001; Drummond et al., 2009; Goodman et al., 2011). Any effects of CWI on resistance exercise-induced muscle protein synthesis or hypertrophy may therefore be due to altered mTORC1 signaling. Three studies have investigated CWI effects on mTORC1 signaling, with conflicting findings. Roberts et al. (2015) reported that CWI blunted the post-exercise phosphorylation of p70S6K$^{Thr421/Ser424}$ and there was a non-significant trend toward reduced phosphorylation of p70S6K^{Thr389}, however there was no effect on several other markers of mTORC1 signaling, including phosphorylation of 4E-BP1 (as assessed by the mobility shift appearance of the γ isoform), rpS6$^{Ser240/244}$, or rpS6$^{Ser235/236}$. In contrast, we observed that CWI attenuated phosphorylation of rpS6$^{Ser235/236}$ and 4E-BP1$^{Thr36/47}$, but had no effect on p70S6K^{Thr389} phosphorylation (Fyfe et al., 2019). Despite impairing myofibrillar protein synthesis, CWI had no effect on phosphorylation of mTOR^{Ser2448}, p70S6K$^{Thr421/Ser424}$, rpS6$^{Ser235/236}$, rpS6$^{Ser240/244}$, or 4E-BP1$^{Thr36/47}$ (Fuchs et al., 2020). Paradoxically, phosphorylation of p70S6K^{Thr389} was elevated in the CWI leg compared to the control leg immediately after immersion, suggesting elevated protein synthesis, however this effect had dissipated by 2 and 5 h after immersion (Fuchs et al., 2020). Overall, although the evidence is sparse and inconsistent, there may be some effects of CWI on resistance exercise-induced mTORC1 signaling. The absence of more robust effects suggests that other mechanisms are also likely to contribute to the CWI-induced inhibition of muscle protein synthesis and hypertrophy in response to resistance exercise.

Ribosome Biogenesis

Rates of protein translation, and thus protein synthesis, during periods of chronic resistance training depend not only on activation of translation by existing ribosomes but also on the capacity for protein translation, which is dependent on ribosomal content. As such, ribosomal biogenesis is likely to be important for muscle hypertrophy, as indicated during muscle overload in rodents (Goodman et al., 2011). Evidence suggests it may also be involved in regulating protein synthesis and muscle hypertrophy in response to resistance exercise in humans (Figueiredo et al., 2015; Fyfe et al., 2018). To date, only one

TABLE 2 | Summary of post-exercise cold-water immersion effects on molecular responses to resistance exercise in human skeletal muscle.

Study	Participants			Study design	Recovery intervention	Resistance training intervention				Muscle sampling times	Outcome measures (effects of CWI compared to control)
	Sample size (sex)	Age	Resistance training status			Intervention length	Exercises trained	Frequency	Volume/ intensity		
Roberts et al. (2015)	21 (M)	21.2 ± 2.2 (CWI group) 21.3 ± 1.9 y (CON group)	At least 12 months experience with resistance training.	Parallel-group, repeated measures	CWI: 10 min at 10.1 ± 0.3°C CON: 10 min active recovery (cycling) at self-selected low intensity (~60 W)	12 weeks	Lower-body resistance exercises and plyometrics	2 x/week	3–6 × 8-12 RM (1 min rest between sets)	4–5 days pre-training 6–7 days post-training	↓ type II fiber CSA ↓ myonuclei per fiber
	9 (M)	22.1 ± 2.2	At least 12 months experience with resistance training.	Within-subject, crossover, repeated measures	CWI: 10 min at 10.1 ± 0.3°C CON: 10 min active recovery (cycling) at self-selected low intensity (~60 W)	N/A	Lower-body resistance exercises	Single exercise session	3–6 × 8–12 RM (1 min rest between sets)	Pre-exercise 2, 24, 48 h post-exercise	↓p70S6K protein at 48 h ↓ p-p70S6K$^{Thr421/Ser424}$ at 2 and 24 h ↔ p-p70S6K^{Thr389} ↔ p-4E-BP1 ↓ rpS6 protein at 24 and 48 h ↔ p-rpS6$^{Ser240/244}$ ↔ p-rpS6$^{Ser235/236}$ ↓ PAX7^{+} satellite cells at 24 and 48 h ↓ NCAM^{+} satellite cells at 24 h ↔ ERK1/2 protein ↔ p-ERK1$^{Thr202/Tyr204}$ ↔ p-ERK2$^{Thr185/Tyr187}$ ↔ p-ERK1/2
Figueiredo et al. (2016)	9 (M)	22.1 ± 2.2	At least 12 months experience with resistance training.	Within-subject, crossover, repeated measures	CWI: 10 min at 10.1 ± 0.3°C CON: 10 min active recovery (cycling) at self-selected low intensity (~60 W)	N/A	Lower-body resistance exercises	Single exercise session	3–6 × 8–12 RM (1 min rest between sets)	Pre-exercise 2, 24, 48 h post-exercise	↓ p-p38$^{Thr180/Tyr182}$ at 2 h (tendency $p = 0.068$) ↓ p-MNK1^{Thr197} at 2 h ↓ p-eIF4E^{Ser209} at 2h ↔ eIF4E protein ↓ Cyclin D1 protein at 2, 24, and 48 h ↔ *Cyclin D1* mRNA ↓ p-Akt^{Thr308} at 48 h ↓ p-PRAS40^{Thr246} at 48 h ↓ rDNA transcription signaling (overall effect from several markers) ↓ pre rRNA expression (overall effect from several markers) ↓ rDNA transcription mRNA (overall effect from several markers)

(Continued)

TABLE 2 | Continued

Study	Participants			Study design	Recovery intervention	Resistance training intervention				Muscle sampling times	Outcome measures (effects of CWI compared to control)
	Sample size (sex)	Age	Resistance training status			Intervention length	Exercises trained	Frequency	Volume/ intensity		
Peake et al. (2017)	9 (M)	22.1 ± 2.2	At least 12 months experience with resistance training.	Within-subject, crossover, repeated measures	CWI: 10 min at 10.1 ± 0.3°C CON: 10 min active recovery (cycling) at self-selected low intensity (~60 W)	N/A	Lower-body resistance exercises	Single exercise session	3–6 × 8–12 RM (1 min rest between sets)	Pre-exercise 2, 24, 48 h post-exercise	↔ neutrophil ($CD66b^+$) infiltration ↔ macrophage ($CD68^+$) infiltration ↔ HSP70 cytosolic content ↔ HSP70 cytoskeletal content ↔ αB-crystallin cytosolic content ↔ αB-crystallin cytoskeletal content ↔ αB-crystallin positive fibers ↔ macrophage (*MAC1, CD163*) mRNA ↔ cytokine and chemokine (*IL1β, TNF-α, IL6, CCL2, CCL4, CXCL2, IL8, LIF*) mRNA ↔ *HSP70* mRNA
D'Souza et al. (2018)	21 (M)	21.2 ± 2.2 (CWI group) 21.3 ± 1.9 y (CON group)	At least 12 months experience with resistance training.	Parallel-group, repeated measures	CWI: 10 min at 10.1 ± 0.3°C CON: 10 min active recovery (cycling) at self-selected low intensity (~60 W)	12 weeks	Lower-body resistance exercises and plyometrics	2 x/week	3–6 × 8-12 RM (1 min rest between sets)	4–5 days pre-training 6–7 days post-training	↔ Fiber type % (type I, type IIa, type IIx and IIa/IIx) ↔ MyHCI, MyHCIIa protein ↓ Δ*MYH7* mRNA (type I gene) ↑ Δ*MYH2* (type IIa), Δ*MYH1* (type IIx) mRNA ↓ ΔmiR-208b, ΔmiR-499a ↑ ΔSox-6 ↑ capillaries per total fibers (tendency $p = 0.051$) ↑ capillaries around type II fibers ↔ capillaries around type I fibers ↑ ΔVEGF protein ↔ ΔSPRED-1 protein ↑ Δ*VEGF1*, Δ*SPRED-1* mRNA ↓ ΔmiR-15a, ΔmiR-16, ΔmiR-126

(Continued)

TABLE 2 | Continued

Study	Participants			Study design	Recovery intervention	Resistance training intervention				Muscle sampling times	Outcome measures (effects of CWI compared to control)
	Sample size (sex)	Age	Resistance training status			Intervention length	Exercises trained	Frequency	Volume/ intensity		
Fyfe et al. (2019)	16 (M)	20.9 ± 3.4 (CWI group) 25.0 ± 4.9 y (CON group)	Recreationally-active, no resistance training experience in past 6 months	Parallel-group, repeated measures	CWI: 15 min at 10°C CON: Non-immersion at 23°C	7 weeks	Whole-body resistance exercises	3 x/week	3 × 12–RM or 20-RM (2 min recovery between sets)	Pre-training (prior to first training session) Post-training (prior to last training session)	↔ type I CSA ↓ type II CSA ↔ p70S6K protein ↔ rpS6 protein ↔ 4E-BP1 protein ↑ FOXO1 protein ↔ FOXO3a protein ↔ MuRF-1 protein ↓ HSP27 protein ↓HSP72 protein ↔ αB-crystallin protein
						N/A	Whole-body resistance exercises	Single session	3 × 12–RM or 20-RM (2 min recovery between sets)	Pre-exercise 1, 48 h post-exercise (performed during first (PRE) and last (POST) training sessions	↔ p-p70S6K^{Thr389}c ↓ p-rps6$^{Ser235/236}$ at POST 1 h and POST 48 h ↓ p-4E-BP1$^{Thr36/47}$ at PRE 1 h ↓ p-FOXO1^{Ser256} at POST 1 h and POST 48 h ↔ p-FOXO3a^{Ser253} ↓ p-HSP27^{Ser15} at PRE 1 h ↔ p-HSP27^{Ser82} ↔ p- αB-crystallin ^{Ser59}
Peake et al. (2020)	9 (M)	22.1 ± 2.2	At least 12 months experience with resistance training.	Within-subject, crossover, repeated measures	CWI: 10 min at 10.1 ± 0.3°C CON: 10 min active recovery (cycling) at self-selected low intensity (∼60 W)	N/A	Lower-body resistance exercises	Single exercise session	3–6 × 8–12 RM (1 min rest between sets)	Pre-exercise 2, 24, 48 h post-exercise	↔ FOXO3a cytosolic expression ↔ FOXO3a nuclear expression ↔ Tenascin C protein ↔ Δ*IGF-1 Ec*, Δ*IGF-1 Ea*, Δ*IGF-1 receptor* mRNA ↔ Δ*Myogenin* mRNA ↔ Δ*Gadd45a*, Δ*Gadd45b* mRNA ↔ Δ*MuRF-1*, Δ*Atrogin-1* mRNA ↔ Δ*Myostatin* mRNA ↔ Δ*collagen type 1 alpha chain 1*, Δ*collagen type III alpha chain 1*, Δ*laminin subunit beta 1*, Δ*TIMP 1* mRNA

(Continued)

TABLE 2 | Continued

Study	Participants			Study design	Recovery intervention	Resistance training intervention				Muscle sampling times	Outcome measures (effects of CWI compared to control)
	Sample size (sex)	Age	Resistance training status			Intervention length	Exercises trained	Frequency	Volume/ intensity		
Fuchs et al. (2020)	12 (M)	21 ± 2	Recreationally active but not participating in structured resistance exercise	Within-subject, repeated measures	CWI (single leg): 20 min at 8°C CON (contralateral leg): 20 min at 30°C	2 weeks	Leg press, knee extension	3 x/week	4 × 10 RM (80% 1–RM)	2 h post-immersion following the first and last training sessions	↓daily myofibrillar protein FSR
						N/A	Leg press, knee extension	Single exercise session	4 × 10 RM (80% 1-RM)	0, 2, and 5 h post-immersion	↓L-[1-^{13}C]-phenylalanine incorporation into Myofibrillar protein at 5 h ↓Myofibrillar protein FSR at 5 h ↔ mTOR^{Ser2448} ↔ p70S6K$^{Thr421/Ser424}$ ↔ rpS6$^{Ser240/244}$ ↔ rpS6$^{Ser235/236}$ ↔ 4E-BP1$^{Thr37/46}$ ↑p70S6K^{Thr389} at 0 but not 2 or 5 h ↔ *FOXO1, MuRF1, atrogin-1* mRNA ↔ *mTOR, p70S6K* mRNA ↔ *GLUT4* mRNA ↔ *IL-6* mRNA ↑*TNFα* mRNA at 0 h ↓SNAT2 protein at 0h ↓CD98 protein at 2 and 5 h

Δ, change from pre- to post-training; 1-RM, one repetition maximum; 4E-BP1, eukaryotic translation initiation factor 4E-binding protein 1; Akt, protein kinase B; CCL2, Monocyte chemotactic protein 1; CCL4, Macrophage inflammatory protein 1β; CD163, cluster of differentiation 163; CON, control group or condition; CSA, cross-sectional area; CWI, cold-water immersion; CXCL2, Macrophage inflammatory protein 2α; eIF4E, eukaryotic translation initiation factor 4E; FOXO, forkhead box O; FSR, fractional synthesis rate; Gadd45, growth arrest and DNA damage-inducible protein 45; HSP, heat-shock protein; IGF-1, insulin-like growth factor 1; IL, interleukin; LIF, Leukemia inhibitory factor; MAC1, macrophage-1 antigen; miR, microRNA; MNK1, mitogen-activated protein kinase–interacting serine/threonine-protein kinase 1; MuRF-1, muscle-specific ring finger 1; MyHC, myosin heavy chain; NCAM, neural cell adhesion molecule; PAX7, paired box 7; p38, mitogen-activated protein kinase; PRAS40, protein-rich AKT1 substrate 1; p70S6K, ribosomal protein S6 kinase beta-1; rDNA, ribosomal deoxyribonucleic; rpS6, ribosomal protein S6; rRNA, ribosomal ribonucleic acid; SOX6, SRY-box 6; SPRED, sprouty-related EVH1 domain-containing protein; TIMP, tissue inhibitor of metallopeptidase; TNF-α, tumor necrosis factor alpha; VEGF, vascular endothelial growth factor; ↑, significantly greater than CON ($p < 0.05$); ↓, significantly less than CON ($p < 0.05$); ↔, not significantly different than CON ($p > 0.05$).

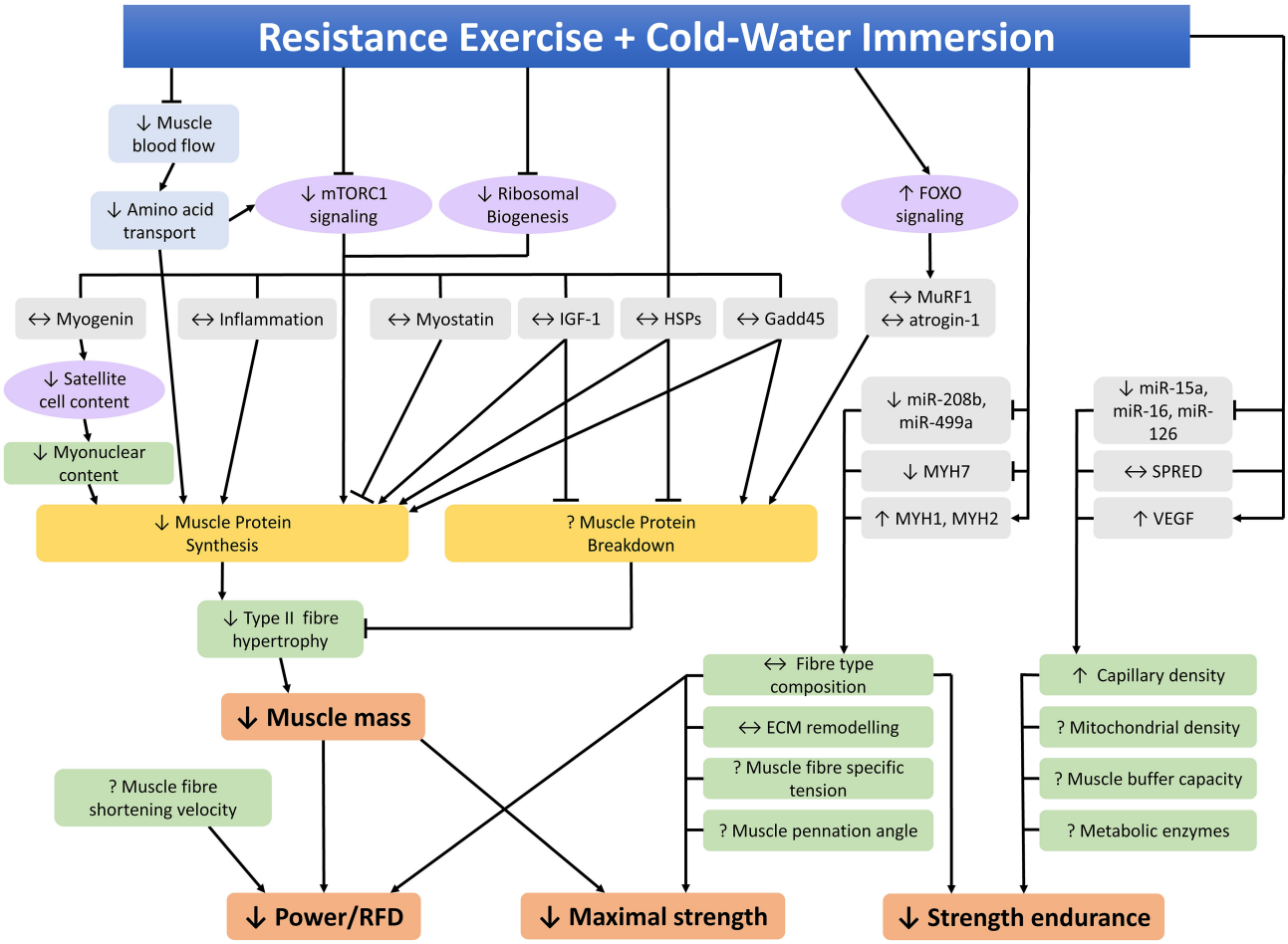

FIGURE 4 | Molecular mechanisms within skeletal muscle that may contribute to the effects of post-exercise cold-water immersion on adaptations to resistance training. ↑, ↓, ↔ indicates increased, decreased, or unchanged response compared to the control condition, ? indicates that the effects of cold-water immersion on this variable have not been investigated, *RFD* rate of force development.

study has investigated the effects of CWI on ribosomal biogenesis responses to resistance exercise. In that study, CWI following an acute resistance exercise bout attenuated signal transduction pathways and transcription of key genes involved in ribosome biogenesis, however it had no effect on the content of mature ribosomal RNA (rRNA) components, such as 28S, 18S, and 5.8S rRNA (Figueiredo et al., 2016). Nonetheless, the impairment of ribosomal biogenesis signaling and transcription suggests that increases in ribosomal content may be attenuated by CWI during chronic resistance training, however whether this occurs has yet to be investigated.

Satellite Cells

Satellite cells are involved in muscle regeneration following injury, however there is debate whether they are involved in resistance exercise-induced muscle hypertrophy. For example, depletion of satellite cells had no effect on muscle growth during short-term muscle overload in mice (McCarthy et al., 2011), but did reduce hypertrophy during more prolonged overload (Fry et al., 2014). In human models of resistance exercise, muscle growth was greatest in participants with the highest pre-training satellite cell population (Petrella et al., 2008) and muscle hypertrophy was accompanied by increased satellite cell content (Snijders et al., 2016). Thus, there is growing evidence that satellite cells do play a role in resistance-exercise-induced muscle hypertrophy in humans. Although evidence is limited, CWI appears to inhibit the satellite cell response to resistance exercise. The upregulation of paired box protein (Pax7) positive satellite cells, a marker of satellite cell abundance, following a single resistance exercise bout was completely blocked by CWI (Roberts et al., 2015). In the same study, the post-exercise increase in neural cell adhesion molecule (NCAM) positive satellite cells appeared to be delayed by CWI (Roberts et al., 2015). These responses are consistent with the observed satellite cell response to chronic resistance training, as the increase in type II muscle fiber myonuclear content was blocked by CWI following 12 weeks of lower body resistance training (Roberts et al., 2015). However, the mRNA expression of myogenin, which promotes

differentiation of satellite cells into myonuclei (Asfour et al., 2018), was not altered by CWI following acute resistance exercise (Peake et al., 2020). These observations suggest the reduction in myonuclear content caused by CWI is due to impaired satellite cell proliferation and not differentiation.

Insulin-Like Growth Factor-1

Insulin-like growth factor-1 (IGF-1) is expressed systemically and locally within skeletal muscle. It is involved in promoting muscle hypertrophy, regeneration, and satellite cell proliferation and differentiation as well as inhibiting muscle protein degradation (Yoshida and Delafontaine, 2020). The mRNA expression of the IGF-1 receptor and the IGF-1 isoforms IGF-1Ea and IGF-1Ec in skeletal muscle were not different between the CWI and control groups following a resistance exercise bout (Peake et al., 2020), suggesting that CWI does not impair muscle hypertrophy *via* this pathway.

Mechanisms Contributing to Impaired Anabolic Responses

Reduced skeletal muscle blood flow and nutrient delivery to the muscle may contribute to the impaired anabolic response caused by CWI during recovery from resistance exercise. Several studies have shown that CWI reduces skeletal muscle blood flow (Gregson et al., 2011; Mawhinney et al., 2013) and muscle blood flow is positively related to increased rates of muscle protein synthesis (Fujita et al., 2006; Timmerman et al., 2010). The gene expression of some markers of amino acid transport was reduced in skeletal muscle by CWI following resistance exercise (Fuchs et al., 2020), which is consistent with reduced blood flow and would further reduce the availability of amino acids for muscle protein synthesis.

Another mechanism by which CWI may attenuate post-exercise anabolism is *via* its effects on inflammation. The inflammatory response is important for muscle repair following injury (Grisbrook et al., 2013; Urso, 2013) and appears to be at least partially involved in the post-exercise increase of muscle protein synthesis (Trappe et al., 2002). Although cold exposure is commonly cited as reducing post-exercise inflammation, much of the evidence to support this comes from animal models of muscle injury or human eccentric exercise models, which are not representative of typical resistance exercise due to the much greater muscle damage they induce. Indeed, studies investigating the inflammatory or immune cell response to resistance exercise show either no effect (Gonzalez et al., 2014a,b; Fragala et al., 2015; Jajtner et al., 2015; Yamane et al., 2015; Peake et al., 2017; Fuchs et al., 2020) or a potentiated response (Roberts et al., 2014; Jajtner et al., 2015; Fuchs et al., 2020) due to CWI, with only one study reporting a decreased response (Earp et al., 2019). Most studies have investigated the effects of CWI on systemic inflammation. Five studies showed unchanged levels of inflammation (Gonzalez et al., 2014a,b; Fragala et al., 2015; Jajtner et al., 2015; Yamane et al., 2015), two showed increased levels (Roberts et al., 2014; Jajtner et al., 2015) and one study observed decreased inflammation (Earp et al., 2019). Only two studies have investigated CWI effects on intramuscular inflammation. Similar to systemic inflammation, markers of intramuscular inflammation were either not affected (Peake et al., 2017; Fuchs et al., 2020) or were increased by CWI (Fuchs et al., 2020), thus indicating no clear difference between the effects of CWI on systemic and intramuscular inflammation. The majority of evidence therefore indicates that CWI effects on inflammation are unlikely to contribute to the attenuated anabolic response to resistance exercise.

Catabolic Responses

Protein Breakdown

In addition to muscle protein synthesis, rates of muscle protein breakdown could influence net protein balance and therefore changes in muscle mass over time. To date, the effects of CWI on rates of muscle protein breakdown following resistance exercise have not been investigated, therefore it is currently unknown whether elevated muscle protein breakdown contributes to the impaired muscle hypertrophy observed in some studies following repeated post-exercise CWI. Although the effects of CWI on rates of muscle protein breakdown following resistance training have not been directly measured, some studies have investigated the molecular mechanisms that regulate muscle protein breakdown.

Ubiquitin Proteasome Pathway

Skeletal muscle protein breakdown is primarily controlled by the ubiquitin proteasome pathway (Goll et al., 2008). Key components of the ubiquitin proteasome pathway include the Forkhead Box O (FOXO) family of transcription factors, which are responsible for the regulation of numerous atrophy-related genes including the E3 ubiquitin ligases atrogin-1 and MuRF-1 (Milan et al., 2015). Atrogin-1 and MuRF-1 bind ubiquitin molecules to specific substrates, which includes myofibrillar proteins, thus targeting the ubiquitinated substrate for degradation by the 26S proteasome (Bodine and Baehr, 2014). CWI had no effect on the gene expression of several markers of the ubiquitin proteasome pathway, including FOXO1, MuRF-1, and atrogin-1 following an acute bout of resistance exercise (Fuchs et al., 2020; Peake et al., 2020). At the protein level, CWI after a single resistance exercise session had no effect on levels of FOXO3a within the cytosol or nucleus (Peake et al., 2020) or on phosphorylation of FOXO1^{Ser256} or FOXO3a^{Ser253} (Fyfe et al., 2019). The long-term effects of repeated CWI following a period of resistance training on markers of the ubiquitin proteasome pathway have only been investigated in one study. We observed that repeated CWI during 7-weeks of resistance training increased protein content of FOXO1 but had no effect on FOXO3a or MuRF-1 (Fyfe et al., 2019). Phosphorylation of FOXO1^{Ser256} at 1 and 48 h after the first resistance training session was not different between the control and the CWI groups. However, phosphorylation of FOXO1^{Ser256} after the last session of a 7-week resistance training program increased to a greater extent in the control compared to the CWI group (Fyfe et al., 2019). This indicates that repeated post-exercise CWI exposures may influence the molecular response to an acute post-exercise CWI exposure, which is consistent with the altered acute molecular responses observed before and after a period of exercise training (Wilkinson et al., 2008; Vissing et al., 2013). Since phosphorylation of FOXO1 at Ser256 reduces

its DNA binding activity (Wang et al., 2016), the reduced phosphorylation caused by chronic CWI may promote higher rates of protein breakdown, while post-exercise phosphorylation of FOXO3a^{Ser253} was not altered by chronic CWI (Fyfe et al., 2019).

Myostatin

Myostatin is a negative regulator of muscle growth, which is typically downregulated for 24–48 h following resistance exercise (Hulmi et al., 2007; Louis et al., 2007). CWI may prolong the downregulation of myostatin mRNA following a resistance exercise bout, as it appeared to be reduced at 24 and 48 h post-exercise in the CWI group, whereas it was not different from pre-exercise in the control group (Peake et al., 2020). However, due to large variability within the results, there was no statistically significant difference from pre-exercise in either group, nor were there significant differences between groups.

Growth Arrest and DNA Damage-Inducible 45

Growth arrest and DNA damage-inducible 45 protein (Gadd45) is upregulated in response to anabolic stimuli, such as synergist ablation-induced overload (Carson et al., 2002) and resistance exercise (Peake et al., 2020) and also in response to catabolic stimuli, such as fasting and skeletal muscle denervation or immobilization (Ebert et al., 2012; Bongers et al., 2013), suggesting that it plays a role in muscle protein turnover or remodeling. The effects of CWI on Gadd45 have only been investigated in one study, whereby a single bout of resistance exercise increased the mRNA expression of Gadd45a and Gadd45b, however this response was not altered by CWI (Peake et al., 2020).

In summary, the available evidence indicates that CWI attenuates muscle hypertrophy in response to resistance exercise *via* a reduction in muscle protein synthesis, which appears to be driven by multiple factors, including blunted mTORC1 signaling, ribosomal biogenesis, myonuclear content, and muscle amino acid transport. Some, albeit very limited, evidence suggests that increased protein breakdown may also contribute to the reduced muscle hypertrophy caused by CWI, although this may only occur following repeated CWI exposures. Additional studies, which concurrently measure muscle mass and muscle protein synthesis or protein breakdown following resistance training with post-exercise CWI are required to determine whether CWI impairs muscle growth *via* altered muscle protein synthesis and/or muscle protein breakdown.

Skeletal Muscle Remodeling

Muscular adaptations that contribute to increased strength following a period of resistance training involve not only muscle hypertrophy, but also skeletal muscle remodeling, which includes increased muscle fiber specific tension (Pansarasa et al., 2009), altered muscle fiber pennation angle (Folland and Williams, 2007), preferential hypertrophy of type II muscle fibers (Folland and Williams, 2007), and enhanced lateral transfer of force between the sarcomere and extracellular matrix (Erskine et al., 2011). Resistance training-induced increases in power/RFD may also involve altered fiber type, increased muscle fiber shortening velocity and other morphological factors (Pansarasa et al., 2009; Schiaffino and Reggiani, 2011; Maden-Wilkinson et al., 2021), whereas increased strength endurance may result from fiber type shifts, increased mitochondrial density, enhanced capillary density, improved muscle buffer capacity, and altered metabolic enzymes.

To date, the effects of CWI on only a few of the above-mentioned mechanisms has been investigated.

Extracellular Matrix Remodeling

The ECM is a scaffold of collagens and proteins that has multiple roles within skeletal muscle, one of which is the lateral transfer of force from the sarcomeres to the muscle connective tissue (Csapo et al., 2020). Increased lateral force transfer has been proposed as a mechanism of increased muscle specific tension following resistance training (Erskine et al., 2011). An acute bout of resistance exercise upregulates the mRNA expression of several ECM-related genes, including collagen type I alpha chain 1, collagen type III alpha chain 1, laminin, and tissue inhibitor of metallopeptidase 1, however the expression levels were not altered by CWI (Peake et al., 2020). Tenascin-C is upregulated in response to muscle contractions and is thought to be involved in ECM remodeling (Mackey and Kjaer, 2017). The protein content of tenascin-c was upregulated 24 h after a single resistance exercise session, however this was not altered by CWI. This suggests that CWI does not alter ECM remodeling in response to resistance exercise, however future studies investigating chronic effects of CWI on ECM proteins are warranted to confirm this.

Muscle Fiber Type Composition

In addition to muscle hypertrophy, a shifting of muscle fiber type composition is another classic adaptation to resistance training (Staron et al., 1990). Muscle fiber type shifts with exercise training typically manifest as conversions between fast-twitch type IIx and type IIa fibers (Staron et al., 1990, 1994), with conflicting observations of switching between type I and type II fiber types (Adams et al., 1993; Paddon-Jones et al., 2001). These muscle fiber type shifts are thought to promote a shift toward a more fatigue-resistant skeletal muscle phenotype.

Since muscle fiber type is a key determinant of its contractile properties (Schiaffino and Reggiani, 2011), potential shifts in muscle fiber type composition with CWI may influence changes in performance outcomes with resistance training, such as improvements in strength, power/rate of force development, and strength endurance.

There is indirect evidence suggesting cold exposure may promote a shift toward a faster muscle phenotype, with divers exposed to prolonged habitual CWI showing higher proportions of type IIx muscle fibers vs. physically-active controls (Bae et al., 2003), while type I to type II fiber type shifts were observed in rat soleus muscle following CWI (Walters and Constable, 1993; Lee et al., 2004).

Using data from a previous investigation (Roberts et al., 2015), only a single study (D'Souza et al., 2018) has determined whether post-exercise CWI influences muscle fiber type composition shifts with resistance training. The findings suggested that CWI did not alter the shifts in muscle fiber type composition measured

via histological staining for myosin heavy chains (reduced percentage of type IIx fibers and increased percentage of type IIa fibers) seen with the control condition (active recovery). These findings were confirmed by Western blot analysis of MyHCI and MyHCIIa protein content, which did not differ between CWI and control. Interestingly, additional analyses of MYH7 (type I gene), MYH1 (type IIx gene), and MYH2 (type IIa gene) mRNA expression, as well as expression of two microRNAs thought to be involved in regulation of muscle fiber type (miR-208b and miR499a) were consistent with a CWI-induced type II muscle fiber shift (D'Souza et al., 2018). This raises the possibility that CWI effects on muscle fiber type may occur following a longer period.

Fiber Type-Specific Hypertrophy

In addition to altered muscle fiber type composition, fiber type-specific changes in muscle fiber size also occur following resistance training. Type II fibers appear to hypertrophy to a greater extent than type I fibers following resistance training (Thorstensson et al., 1976; Dons et al., 1979; Houston et al., 1983; Fyfe et al., 2019). There is also some evidence that type II muscle fibers have a higher specific tension and shortening velocity than type I fibers (D'Antona et al., 2006; Pansarasa et al., 2009). These two factors combined may therefore contribute to increased strength and power/RFD after a period of resistance training. No studies have yet investigated the effects of CWI on muscle fiber specific tension or shortening velocity, so it is unknown whether CWI-induced impairments in these variables may contribute to the reduced strength and power caused by CWI. As discussed in section Skeletal Muscle Hypertrophy, CWI attenuated the resistance training-induced increase in type II muscle fiber area (Roberts et al., 2015; Fyfe et al., 2019). This suggests that the smaller resistance training-induced gains in strength caused by CWI may be partially attributable to impaired growth of type II fibers. However, although changes in type I fiber cross-sectional area were not reported, the increase in type I and II fiber combined area was attenuated by CWI (Roberts et al., 2015). Thus, it is possible that CWI also blunts type I fiber hypertrophy. This may not have been detected in our study (Fyfe et al., 2019) as the 7-week training intervention was possibly too short to induce a substantial increase in type I fiber area.

Cell Stress Response

The heat shock family of proteins are well-known for their roles in protection from cellular stress (Lindquist, 1986). Heat shock proteins are important for cellular homeostasis and protein preservation and degradation (Noble et al., 2008) and play key roles in several processes involved in adaptation to exercise. For example, HSP72 regulates processes involved in protein synthesis and degradation (Ku et al., 1995; Zhou and Thompson, 1997; Beere et al., 2000; Senf et al., 2008; Dokladny et al., 2015). The small HSPs, HSP27 and αB-crystallin, inhibit protein degradation pathways (Dodd et al., 2009; Vasconsuelo et al., 2010; Adhikari et al., 2011) and bind to cytoskeletal and myofibrillar proteins following muscle-damaging exercise, where they are thought to stabilize disrupted elements and assist in regeneration and remodeling (Koh and Escobedo, 2004; Paulsen et al., 2007). CWI following a single bout of resistance exercise had no effect on HSP72 mRNA expression or levels of HSP72 protein within cytosolic or cytoskeletal fractions (Peake et al., 2017), however HSP72 protein content was decreased by repeated CWI following 7 weeks of resistance training, whereas the control group was unchanged (Fyfe et al., 2019). CWI had no effect on the cytosolic or cytoskeletal content of αB-crystallin, the number of αB-crystallin positive fibers, or phosphorylation of αB-crystallin^{Ser59} following a resistance exercise session (Peake et al., 2017; Fyfe et al., 2019). Nor did CWI alter the increase in αB-crystallin protein content following resistance training (Fyfe et al., 2019). CWI amplified the increase in HSP27^{Ser15} phosphorylation following the first but not the last training session of a 7-week resistance training program, however it had no effects on the resistance exercise-induced phosphorylation of HSP27^{Ser82} (Fyfe et al., 2019). Following 7 weeks of resistance training HSP27 protein content increased to a greater extent in the control group compared to the CWI group. These combined results indicate that some components of the cellular stress response following resistance exercise are altered by CWI, which may contribute to the impaired adaptive responses observed.

Angiogenesis

Angiogenesis, which describes the formation of new blood vessels, has been shown to occur in response to resistance training (Cocks et al., 2014; Verdijk et al., 2016; D'Souza et al., 2018; Holloway et al., 2018), however whether this is a proactive adaptation that facilitates muscle hypertrophy or a reactive adaptation in response to exercise-induced metabolic stressors and/or muscle fiber hypertrophy-driven changes in perfusion is unclear (Holloway et al., 2018). Following 12 weeks of resistance training, the number of capillaries per muscle fiber, the capillary-to-fiber perimeter exchange index, and capillary density increased in the CWI group but not in the control group (D'Souza et al., 2018). The authors also investigated some of the molecular mechanisms regulating angiogenesis and found that CWI increased the mRNA expression of the pro-angiogenic factor VEGF1 and the anti-angiogenic factor SPRED-1 compared to control (D'Souza et al., 2018). At the protein level, VEGF1 but not SPRED-1 content was higher in the CWI compared to the control group (D'Souza et al., 2018). The expression of several microRNAs that inhibit VEGF1 (miR-15a and miR-16) and SPRED-1 (miR-126) was also investigated, with each of these being upregulated in control and downregulated in CWI (D'Souza et al., 2018). Although this is the only study to investigate the effects of CWI on angiogenic responses to resistance training, the results indicate that CWI induces a predominantly pro-angiogenic environment. Since muscle fiber hypertrophy appears to be blunted by CWI, this suggests that the pro-angiogenic environment may occur in response to possible CWI-induced alterations in metabolic stressors or reduced muscle perfusion. Longer duration resistance training studies may be needed to determine whether the favorable effects of CWI on angiogenic signaling eventually translate to increased capillarization which may then enable muscle fiber hypertrophy.

LIMITATIONS AND FUTURE DIRECTIONS

While there appears to be little evidence for beneficial effects of CWI on physiological adaptation and molecular responses to resistance training, there are a number of limitations and additional considerations when interpreting the available evidence.

Effectiveness of the Resistance Training Intervention

To determine whether CWI application influences physiological adaptations to resistance training, it is necessary to compare changes in training outcomes with post-exercise CWI to a control condition. The comparison in training-induced responses in the CWI and control conditions are therefore critical for drawing conclusions on whether CWI influences responses to resistance training alone. There are a number of examples in the literature whereby changes in outcome measures, including dynamic 1-RM strength (Poppendieck et al., 2020), isometric strength (Ohnishi et al., 2004; Yamane et al., 2006), isokinetic strength (Roberts et al., 2015), CMJ height (Poppendieck et al., 2020), or peak force during a squat jump or ballistic push-up (Fyfe et al., 2019), were not evident with resistance training when combined with neither post-exercise CWI or a control condition. It must be considered, however, that it may not be possible to demonstrate whether CWI had an influence on physiological adaptations if the resistance training intervention was itself (i.e., the control condition) not sufficient to elicit substantial changes in these outcomes. It is therefore likely that the effectiveness of a given resistance training intervention for eliciting physiological adaptations can influence interpretation of whether post-exercise CWI indeed modulates these responses.

Since the effectiveness of any exercise training intervention for eliciting physiological adaptations is dependent on a multitude of factors, including the specifics of the training intervention itself and characteristics of the participant cohort (e.g., age, sex, genetics, training status, nutritional status, among others), these factors must be considered when interpreting evidence for the influence of CWI on physiological adaptations to exercise training.

Training Status of Participants

The majority of studies investigating whether CWI influences physiological adaptations to resistance training have been conducted in participants with limited or no resistance training experience. Indeed, only three studies (Frohlich et al., 2014; Roberts et al., 2015; Poppendieck et al., 2020) have been undertaken in participants with some degree of resistance training experience. The resistance training experience of participants likely has implications for both the ability of relatively short-term resistance training interventions to induce substantial changes in outcome measures (and in turn detect any potential influence of CWI on these outcomes), as well as the applicability of study findings to athletic populations.

The principle of diminishing returns suggests the magnitude of physiological adaptations to exercise training are reduced in trained compared with untrained individuals. It is therefore possible that detecting any potential effect of CWI on resistance training adaptations is more challenging in trained vs. untrained individuals. For example, one (Poppendieck et al., 2020) of the three studies (Frohlich et al., 2014; Roberts et al., 2015; Poppendieck et al., 2020) performed in resistance-trained individuals found dynamic 1-RM (leg press) strength did not improve with resistance training (either with CWI application or control), which precludes the possibility of detecting any potential influence of post-exercise CWI. Nevertheless, studies in trained individuals are essential to ensure the relevance of study findings to athletic populations, while longer-term training interventions may be necessary to induce the substantial changes in outcome measures required to understand the potential effects of CWI on training responses in these populations. Given the time course of resistance training adaptations, whereby neural adaptations are considered to largely mediate short-term improvements in muscle force characteristics (Del Vecchio et al., 2019a), with morphological adaptations traditionally considered to occur at a slower rate (Folland and Williams, 2007), the length of the resistance training interventions used by studies may also influence conclusions on the effects of CWI on muscle hypertrophy and aspects of strength or power development. Since the potential negative influence of post-exercise CWI on changes in aspects of muscle function (i.e., strength and power/RFD) with resistance training are thought to be mediated *via* the effects of CWI on muscle hypertrophy, rather than neural adaptations *per se*, it is possible that short-term resistance training interventions, particularly in non-resistance trained individuals, may therefore be less likely to find negative effects of CWI on these responses. To date, studies have employed resistance training interventions of between 4 and 12 weeks in duration (Ohnishi et al., 2004; Yamane et al., 2006, 2015; Frohlich et al., 2014; Roberts et al., 2015; Fyfe et al., 2019; Poppendieck et al., 2020). It is likely that longer interventions may be required, particularly in resistance-trained populations, to detect substantial changes in aspects of strength and/or power/RFD and particularly muscle hypertrophy, to determine whether CWI influences these adaptations relative to resistance training alone.

Measures Used to Assess Muscle Hypertrophy Outcomes

Even if a given resistance training intervention is sufficient to elicit physiological adaptations including skeletal muscle hypertrophy and improvements in strength and/or power-related measures, the tools used to assess these responses can also influence the ability of a given study to detect these changes. The assessment of skeletal muscle hypertrophy is particularly challenging, due not only to conceptual issues when defining muscle hypertrophy as a biological construct, but also because of the multitude of tools available to assess indices of muscle hypertrophy at multiple physiological levels (Haun et al., 2019). While imaging techniques including MRI and CT are considered gold-standard for assessing changes in whole-muscle size/CSA, only a single study (Roberts et al., 2015) has used these methodologies to assess the influence of CWI on hypertrophic responses to resistance training.

It is worth noting that one (Fyfe et al., 2019) of the two studies (Roberts et al., 2015; Fyfe et al., 2019) that have assessed hypertrophic responses at both macroscopic and microscopic levels following resistance training with or without CWI showed mixed findings depending on the particular measure of hypertrophy used. Specifically, CWI application impaired the resistance training-induced increase in type II muscle fiber CSA of the vastus lateralis (∼+12% for control vs. ∼+5% for CWI) but did not affect improvements in lean mass assessed *via* DXA (Fyfe et al., 2019). It should be considered, however, that while DXA-derived assessments of lean mass are highly correlated (r = 0.85–0.94) with gold-standard assessments of whole-muscle size (e.g., MRI and CT) when measured at a single time point (Levine et al., 2000; Maden-Wilkinson et al., 2013), DXA may be less sensitive (r = 0.33 vs. CT) for detecting changes in indices of skeletal muscle hypertrophy following a period of resistance training (Delmonico et al., 2008). It is therefore unclear whether similar discrepancies between muscle hypertrophy outcomes assessed *via* muscle biopsy (muscle fiber CSA) and DXA (lean body mass) may have occurred if another gold-standard measurement of macroscopic-level muscle hypertrophy (e.g., MRI, CT, or ultrasound) was undertaken. Nevertheless, such discrepancies in muscle hypertrophy outcomes assessed at different physiological levels (e.g., macroscopic vs. microscopic) is consistent with other findings in response to resistance training alone (Narici et al., 1996; Aagaard et al., 2001; Esmarck et al., 2001). For example, several studies have shown greater resistance training-induced changes in vastus lateralis muscle CSA when assessed at the muscle fiber vs. whole-muscle levels (Narici et al., 1996; Aagaard et al., 2001; Esmarck et al., 2001). These observations further highlight the complexities of assessing hypertrophic responses in human skeletal muscle, and in turn, interpretation of the potential effects of CWI on resistance training-induced muscle growth.

Additional studies assessing muscle hypertrophy following resistance training with or without CWI at multiple physiological levels (e.g., whole-body, macroscopic, and microscopic) concurrently, and particularly using gold-standard measures (such as MRI or CT) are required to improve current understanding of the influence of CWI on muscle hypertrophy with resistance training.

Measures Used to Assess Strength Outcomes

As when assessing the influence of CWI on skeletal muscle hypertrophic responses to resistance training, there are various considerations when interpreting changes in maximal strength with resistance training, and therefore the potential influence of CWI on these responses. Because strength is a highly task-specific phenomenon (Morrissey et al., 1995), the magnitude of strength gain following a resistance training intervention is influenced by various factors, including the complexity of the movement patterns performed within the training intervention, and the degree of similarity (in terms of movement patterns, range of motion, lifting velocity, and intensity/loads used) between the resistance training exercises and the methods used to assess changes in strength (Buckner et al., 2017). For these reasons, the potential influence of the type of resistance training performed on the magnitude of strength gain (which is also influenced by participant characteristics), as well as the specificity of methods used to assess changes in strength, should be considered when interpreting the effects of CWI (vs. a control condition) on strength development with resistance training.

The specificity of strength gains with resistance training has important implications for the measures used to detect changes in strength with resistance training. In some cases, it is possible the measure of strength chosen could influence (e.g., underestimate) changes in strength with resistance training, and in turn, compromise the ability to detect potential effects of CWI on resistance training-induced strength gain. In some studies investigating the effects of CWI on resistance training adaptations, the measure of strength used was somewhat inconsistent with the modality of resistance training employed. For example, the studies by Yamane et al. (2006, 2015) and Ohnishi et al. (2004) employed dynamic resistance training of the wrist flexors, but assessed changes in isometric handgrip strength as the sole strength outcome. It is therefore possible that strength gains may have been greater in magnitude if a dynamic strength measure was instead assessed, which may have increased the possibility of detecting any impairments to strength gain that may have been caused by post-exercise CWI.

Future studies should therefore include multiple measures of strength (e.g., dynamic RM, isometric, and/or isokinetic strength) (Buckner et al., 2017) assessed during tasks that replicate those in the resistance training intervention to best capture the potential influence of post-exercise CWI application on strength development with resistance training.

Resistance Training Interventions Used

There are a number of limitations regarding the resistance training interventions used in existing studies, which likely influence their applicability to real-world scenarios. For example, some studies have used resistance training interventions that incorporate single exercises that target only smaller muscle groups during single-joint movements (Ohnishi et al., 2004; Yamane et al., 2006, 2015; Frohlich et al., 2014), with only three studies (Roberts et al., 2015; Fyfe et al., 2019; Poppendieck et al., 2020) incorporating exercises targeting larger muscle groups during multiple dynamic, multi-joint movements commonly found in resistance training interventions in athletic settings. Additionally, only a single study has employed a whole-body resistance training intervention (Fyfe et al., 2019), which may be necessary to determine whether CWI exerts local or systemic effects on physiological adaptations to resistance training (e.g., whether lower-body CWI influences upper-body muscle hypertrophy or strength/power), and to mimic common resistance training practices.

A further limitation regarding the relevance for elite athletes of studies performed to date is the relatively low training volumes and frequencies employed in those studies. Athletes typically train at least five times per week and often at considerably higher frequencies (Smith, 2003). At higher training frequencies, optimizing recovery between sessions is likely

of greater importance to enhance the quality of subsequent training sessions, and presumably increase the stimulus for physiological adaptation. Up to now, CWI studies have used training frequencies of only two or three sessions per week, with at least 1 day of recovery between sessions. These relatively low training frequencies may allow adequate recovery between training sessions and therefore reduce any potential recovery-enhancing benefits of CWI that may be more important with higher training frequencies.

Potential Sex-Specific Effects

Very few studies performed to date have involved female participants. The exceptions to this include Poppendieck et al. (2020), in which two of the 11 participants were females and Yamane et al. (2006), in which four of 27 participants were females. In both cases, male and female data were not presented or analyzed seperately, precluding any insight into whether sex-specific effects of CWI on resistance training outcomes occurred. Females typically have greater subcutaneous fat thickness and a higher surface area to body mass ratio than males (Kruschitz et al., 2013), which alters the level of cooling (Petrofsky and Laymon, 2009; Castellani and Young, 2016) and may therefore influence the effects of post-exercise CWI. Given the potential for sex-specific effects of CWI on adaptations to resistance training and the implications of this for a large proportion of athletes, further research in this area is warranted.

Potential Acclimation Effects

Repeated or prolonged cold exposure results in acclimation, which alters the thermoregulatory and metabolic responses to cold (Castellani and Young, 2016). As a result, the effects of CWI may be altered following prolonged use. In support of this hypothesis, we observed different responses for phosphorylation of HSP27^{Ser15}, FOXO1^{Ser256}, and rpS6$^{Ser235/236}$ in the CWI group following the last session of a 7-week resistance training program compared to the first session (Fyfe et al., 2019). However, whether acclimation to CWI changes its effects on resistance training-induced performance adaptations has not been investigated. Given the complexity of the various adaptations that occur with cold acclimation it is difficult to predict whether the effects of CWI would be attenuated or exacerbated with repeated CWI exposure.

CONCLUSIONS

Post-exercise CWI is a widely-used recovery modality among athletes. Nonetheless, there are relatively few studies investigating the effects of repeated CWI on adaptations to exercise, especially resistance exercise. Although post-exercise CWI may enhance short-term recovery following resistance exercise, current evidence suggests CWI has either nil or detrimental effects on physiological adaptations to resistance training, including muscle hypertrophy and measures of maximal strength, strength endurance, and power/RFD, as well as the molecular responses that underpin adaptation to resistance training in skeletal muscle. Importantly, no studies have shown benefits of CWI on resistance training adaptations. As such, there is currently no evidence to support the use of post-exercise CWI during periods of resistance training. It is important to note, however, that given the lack of available evidence and its associated limitations, there may be many potential circumstances whereby CWI application following resistance training could be beneficial, such as in females, in chronically-trained and elite athletes, during periods of high-frequency training, or in cold-acclimated individuals. Further research is required to determine the effects of post-exercise CWI on physiological adaptations resistance training in these circumstances, and to address the additional methodological limitations of previous studies.

AUTHOR CONTRIBUTIONS

AP and JF contributed to the writing and editing of the article and approved the submitted version.

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An Ice Vest, but Not Single-Hand Cooling, Is Effective at Reducing Thermo-Physiological Strain During Exercise Recovery in the Heat

Afton D. Seeley[1,2,3]* *and Ross A. Sherman*[4]

[1] Department of Human Performance and Health Education, Western Michigan University, Kalamazoo, MI, United States, [2] Thermal and Mountain Medicine Division, US Army Research Institute of Environmental Medicine, Natick, MA, United States, [3] Oak Ridge Institute of Science and Education, Belcamp, MD, United States, [4] Department of Movement Science, Grand Valley State University, Allendale, MI, United States

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United Kingdom

***Correspondence:**
Afton D. Seeley
afton.d.seeley.ctr@mail.mil

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Sports limit the length of breaks between halves or periods, placing substantial time constraints on cooling effectiveness. This study investigated the effect of active cooling during both time-limited and prolonged post-exercise recovery in the heat. Ten recreationally-active adults (VO_{2peak} 43.6 ± 7.5 ml·kg^{-1} · min^{-1}) were exposed to thermally-challenging conditions (36°C air temperature, 45% RH) while passively seated for 30 min, cycling for 60 min at 51% VO_{2peak}, and during a seated recovery for 60 min that was broken into two epochs: first 15 min (REC_{0-15}) and total 60 min (REC_{0-60}). Three different cooling techniques were implemented during independent recovery trials: (a) negative-pressure single hand-cooling (~17°C); (b) ice vest; and (c) non-cooling control. Change in rectal temperature (T_{re}), mean skin temperature ($\overline{T}_{sk}$), heart rate (HR), and thermal sensation (TS), as well as mean body temperature ($\overline{T}_{b}$), and heat storage (S) were calculated for exercise, REC_{0-15} and REC_{0-60}. During REC_{0-15}, HR was lowered more with the ice vest (−9 [−15 to −3] bts·min^{-1}, $p = 0.002$) and single hand-cooling (−7 [−13 to −1] bts·min^{-1}, $p = 0.021$) compared to a non-cooling control. The ice vest caused a greater change in $\overline{T}_{sk}$ compared to no cooling (−1.07 [−2.00 to −0.13]°C, $p = 0.021$) and single-hand cooling (−1.07 [−2.01 to −0.14]°C, $p = 0.020$), as well as a greater change in S compared to no cooling (−84 [−132 to −37] W, $p < 0.0001$) and single-hand cooling (−74 [−125 to −24] W, $p = 0.002$). Across REC_{0-60}, changes in $\overline{T}_{b}$ (−0.38 [−0.69 to −0.07]°C, $p = 0.012$) and $\overline{T}_{sk}$ (−1.62 [−2.56 to −0.68]°C, $p < 0.0001$) were greater with ice vest compared to no cooling. Furthermore, changes in in $\overline{T}_{b}$ (−0.39 [−0.70 to −0.08]°C, $p = 0.010$) and $\overline{T}_{sk}$ (−1.68 [−2.61 to −0.74]°C, $p < 0.0001$) were greater with the ice vest compared to single-hand cooling. Using an ice vest during time-limited and prolonged recovery in the heat aided in a more effective reduction in thermo-physiological strain compared to both passive cooling as well as a single-hand cooling device.

Keywords: exercise in heat, thermoregulation, post-exercise recovery, skin temperature, heat storage, core temperature, sport

INTRODUCTION

Both professional and recreational sporting events frequently take place in thermally-stressful environments, including the familiar summer Olympic Games (Barwood et al., 2009). Unabated heat gain during exercise performed in hot environments, the product of both elevated muscular work and a reduced skin temperature to ambient temperature (T_{sk}-T_a), and therefore core temperature to skin temperature (T_c-T_{sk}), thermal exchange gradient, is capable of eliciting hyperthermic core body temperatures (T_c) >39°C (Wendt et al., 2007). Heated exercise-induced core and skin temperatures elevations contribute to the deterioration of aerobic exercise capacity, although the exact cardiovascular mechanisms by which this decrement occurs appears to be a function of both exercise intensity and duration (Nybo et al., 2011). At maximal exercise intensities, augmentation of skin blood flow to facilitate heat loss impairs cardiac filling, reducing central venous pressure, and maximal cardiac output, and therefore competitively impairs arterial oxygen delivery to exercising skeletal muscle. At prolonged submaximal intensities when muscle blood flow and oxygen consumption are often not significantly changed relative to a more temperate environment, high skin temperatures likely influence the perception of fatigue via alterations in afferent feedback (Nybo et al., 2011). Continually increasing core and skin temperatures, especially during prolonged exercise, additionally pose an enhanced likelihood of heat illness. Heat stroke, the most serious heat related syndrome, is designated by a severely elevated core temperature and failure of an individual's sweating mechanisms (Coris et al., 2004).

Athletes that participate in outdoor sporting events of a prolonged heated nature, such as ultramarathons, and those with a limited 10–15 min half-time, such as soccer and rugby, may be especially susceptible to the deleterious influence of hyperthermia. Strategic pre-exercise behaviors including heat acclimation (Lorenzo et al., 2010) and intra-exercise hydration strategies (Montain and Coyle, 1992; Travers et al., 2020) have proven helpful to reduce the physiological stress and performance decrement induced by hyperthermia. Furthermore, athletes engaging in prolonged or limited breaking sport have the potential to both offset aerobic performance decrement as well as the risk of heat illness by implementing efficient cooling strategies available during sporting breaks. While application of cooling devices in a continually heated environment may influence overall cooling effectiveness, sport, and occupational requirements often impede the removal of a hyperthermic individual from a heated environment. Furthermore, the implementation of techniques such as whole-body cold water immersion, deemed to be most efficient for thermoregulatory recovery (Casa et al., 2015; Zhang et al., 2015), is practically difficult in prophylactic cooling scenarios. Additionally, neck cooling, although effective in reducing the perception of heat strain (Sunderland et al., 2015), does not appear to meaningfully assist in thermo-physiological rebound from hyperthermia (Tyler and Sunderland, 2011; Sunderland et al., 2015). Therefore, using more practical and physiology-influencing cooling techniques during heated endurance exercise recovery, if deemed time-efficient in their cooling, may help to offset decrement of discontinuous aerobic exercise performance and the accumulated risk of severe heat illness.

Numerous commercially available small and portable cooling devices have been proposed to reduce cardiovascular strain, skin temperature, and core temperature following exercise-induced hyperthermia.

A phase-changing "ice" vest provides a widened T_c-T_{sk} gradient, by reducing skin temperature, which favors heat loss from the blood perfusing the skin. The cooled blood then circulates back to the core, effectively contributing to the maintenance or development of a negative heat balance (House et al., 2013). The donning of an ice vest in occupational situations beneath clothing has effectively demonstrated reductions in heat strain (House, 1996; Cadarette et al., 2002; House et al., 2003; Amorim et al., 2010). The use of ice vests by hyperthermic athletes post-exercise, though, has proven less successful in reducing core temperature, with studies citing a loss in evaporative cooling with the torso encompassing vests (House et al., 2013).

Additional post-exercise cooling efforts have monopolized on hand cooling technologies some of which utilize a rigid chamber with a flexible airtight vacuum-seal about the wrist. Hand cooling is thought to optimize the loss of body heat through the arteriovenous anastomoses (AVAs) present in the palm, effectively dissipating heat at elevated core and skin temperatures (Bergersen, 1993). Battery powered cooling devices create a thermal gradient with the palm while the negative pressure in the vacuum chamber draws a large volume of blood into the AVAs to speed heat exchange and prevent cold-reactive vasoconstriction (Zhang et al., 2009). Research has focused largely on the use of this relatively light and portable hand cooling device during exercise (Grahn et al., 2005, 2012; Hsu et al., 2005), with only a small and inconclusive body of support for single-hand use in recovery (Zhang et al., 2009; Kuennen et al., 2010). One of the most recent investigations, using a commercially available hand cooling device (Adams et al., 2016) following heated treadmill exercise, suggests that dual hand cooling may reduce T_{re} more than passive cooling alone, bolstering similar conclusions drawn from dual hand immersion in a simpler cold water bath (Barwood et al., 2009). Yet still, a variety of literature suggests a lack of advantageous core temperature reduction with administration of a single hand cooling device during exercise recovery (Balldin et al., 2007; Walker et al., 2009; Amorim et al., 2010).

Existing literature suggests augmented cooling by single-hand cooling when heavy heat retardant clothing is worn/retained following heated exercise and into heated recovery (Kuennen et al., 2010) and/or when hand cooling is imposed simultaneously with removal from the heated environment (Zhang et al., 2009). Furthermore, dual hand cooling, although much more logistically burdensome to dexterity, appears efficacious to efficiently reduce core temperature following heated exercise, likely due to the heightened cooling exposed skin surface area (Barwood et al., 2009; Adams et al., 2016). Still, little literature exists regarding the influence of a negative pressure hand cooling device on a sport-mimicking recovery environment where minimal clothing (shorts and t-shirt) is worn, individuals are not removed from

the heated environment following exercise, and only single-hand cooling is utilized to retain dexterity.

The primary aim of this study is to investigate the effectiveness of active vs. passive cooling during time-limited and prolonged recovery in sport-mimicking conditions (minimal clothing, continued exposure to heat, dexterity maintenance) following submaximal exercise in the heat. A secondary aim of this study is to examine thermo-physiological and perceptual differences during recovery in the heat between negative pressure single-hand cooling and an ice vest. We hypothesized that active post-exercise cooling would significantly improve thermo-physiological function and perceived thermal sensation (TS) in both time-limited and prolonged recovery. Additionally, it was proposed that an ice vest would be a more effective cooling strategy than single-hand cooling.

MATERIALS AND METHODS

Participants and Procedural Controls

Ten recreationally active participants (six males, four females, age 25 ± 3 years, body mass 75.5 ± 12.5 kg, height 173 ± 9 cm, VO_{2peak} 43.6 ± 7.5 ml·kg^{-1} · min^{-1}) participated in the study. All participants met the following inclusionary criteria: non-smoker; healthy, free of disease, and free of medication use which may affect the cardiovascular or metabolic responses during exercise; free of any orthopedic injuries or conditions that would make exercise difficult; classified as "Low Risk" by the American College of Sport Medicine (Pescatello et al., 2014); and not obese (body mass index <30 kg·m^{-2}). Participants gave written, informed consent prior to participation in the study, which had been approved by the Human Subjects Institutional Review Board (Project Approval Number: 13-12-10) at Western Michigan University.

Participants were asked to refrain from ingesting any caffeine and engaging in exercise the day of the visits to the laboratory. Eumenorrhoeic female participants were asked to self-report the date of last menses to restrict collection of thermoregulatory data only within the early follicular phase (cycle days 3–6). Those that reported using a biphasic oral contraceptive ($n = 1$) were tested within the first 3–6 days of active pills following the withdrawal week, in an attempt to minimize the day-to-day variability in thermoregulatory variables, especially rectal temperature (Lei et al., 2019). Notably, pre-exercise rectal temperature did not significantly differ between conditions (control: 37.13 ± 0.30, ice vest: 37.23 ± 0.28, single-hand cooling: 37.17 ± 0.30, $p = 0.734$). Each participant was asked to wear a t-shirt, shorts, socks, and athletic shoes each time they visited the laboratory. All trials for a given participant were conducted during the morning (± 1 h) to avoid diurnal variation in core temperature (Morris et al., 2009). The research was conducted outside of the summer months (September–April) in order to minimize any seasonal heat acclimation. Furthermore, all heated trials were separated by at least 1 week to minimize the likelihood of induced heat acclimation.

Research Design

The study was conducted utilizing a randomized counterbalanced cross-over design with three recovery conditions: (1) negative pressure single-hand cooling, (2) ice vest, and (3) a non-cooling control. Participants visited the laboratory on four separate occasions. The first visit consisted of a graded exercise test and the following three visits consisted of exercise bouts in the heat followed by one of the three recovery cooling conditions.

Graded Exercise Test

Upon arrival to the laboratory, height and body mass were measured using standardized techniques and a wall-mounted stadiometer and digital scale, respectively. Each participant completed a graded exercise test on an electromagnetically-braked cycle ergometer (Corival, Lode B.V., Groningen, Netherlands) to determine peak oxygen consumption (VO_{2peak}) as a measure of cardiorespiratory fitness. The VO_{2peak} value obtained allowed determination of the appropriate exercise intensity (∼50% VO_{2peak}) for all experimental trials. Each participant was fitted for seat height on the cycle ergometer, with the participant's knee at 10–15° of flexion at the pedal's lowest point. Additionally, each participant was fitted with a nose clip and a mouthpiece for the collection of 15-s averaged expired respiratory gases using a metabolic cart (TrueOne 2400, ParvoMedics, Sandy, UT), and a heart rate (HR) monitor (Polar USA, Lake Success, Long Island, NY). The assessment consisted of a graded protocol that began with two- min of cycling at 40 W for female and 60 W for male participants. The cycling intensity was increased every minute thereafter by 20 W until volitional fatigue. Volitional fatigue was determined as the point during exercise when each participant felt like they could exercise no longer or could no longer maintain a pedaling frequency of at least 50 rpm. Each participant was asked to assess their ratings of perceived exertion (RPE) using a standard 6–20 scale (Borg and Linderholm, 1967) during the last 30 s of each stage. Once the exercise test protocol was terminated, each participant was provided water *ad libitum* and continued to cycle at a low intensity for 5–10 min whilst being monitored for normal, post-exercise cardiovascular recovery.

Experimental Trials

Upon arrival at the laboratory, participants' nude body mass was measured. Thirty minutes prior to entering the heated (36°C, 45% RH) environmental chamber (Thermotron, Holland, MI) a bolus of plain water equivalent to 5 ml·kg^{-1} body mass was administered in an attempt to standardize pre-exercise hydration status. A flexible probe (Physitemp Instruments Inc., Clifton, NJ) was inserted 13 cm past the anal sphincter for the measurement of rectal temperature (T_{re}). Thermocouples (Physitemp Instruments Inc., Clifton, NJ) were also attached to the surface of the skin at four sites on the right side of the body (chest, triceps, quadriceps, calf) using waterproof tape (Hy-tape, Hytape International Inc., Patterson, NY) for the measurement of skin temperature (T_{sk}). The four sites contributed to the calculation of mean skin temperature ($\overline{T}_{sk}$; Ramanathan, 1964):

$$\overline{T}_{sk} = 0.3(T_{chest} + T_{arm}) + 0.2(T_{thigh} + T_{calf})$$

Both T_{re} and $\overline{T}_{sk}$ were then used to calculate mean body temperature ($\overline{T}_b$; Colin and Houdas, 1965):

$$\overline{T}_b = (0.8 \cdot T_{re}) + (0.2 \cdot \overline{T}_{sk})$$

Stored heat (S) was calculated for the exercise bout and recovery using the following equation:

$$S = \Delta\overline{T}_b \cdot 3.48 \cdot \ mass/t$$

where: $\Delta\overline{T}_b$ = change in mean body temperature, the average specific heat of body tissues was assumed as 3.48 kJ·kg^{-1} · °C^{-1}, mass = pre-test mass of the participant (kg), t = time (s).

Rectal and skin thermocouples were connected to a data acquisition system (Thermes USB, Physitemp Instruments Inc., Clifton, NJ) that was interfaced to a PC computer. Lastly, a HR monitor was fitted.

Each trial consisted of the following sequence performed entirely within the environmental chamber at 36°C, 45% RH:

- 30 min of seated rest
- 60 min of cycling at ∼50% VO_{2peak} or until T_{re} ≥39.5°C
- 60 min of seated rest with one of the cooling conditions.

Time to complete this trial sequence within the environmental chamber across all conditions was 154.12 ± 1.26 min. Each participant's target exercise VO_2 and starting power output was determined utilizing the established power output to VO_2 relationship determined using the VO_{2peak} assessment. During each participant's first trial, expired respiratory gases were collected during the first 10 min of exercise. If the 15-s averaged VO_2 from 5:15 to 7:00 after the start of exercise was not 50 ± 5% VO_{2peak}, the intensity was adjusted in 5 W increments until target VO_2 was reached. Exercise intensity was monitored for an additional 2 min upon readjustment to ensure VO_2 stability. The progression of exercise intensity for each subsequent trial followed an identical scheme: (1) 2-min warm-up at half of the 50% VO_{2peak} power output, (2) at 2 min, the intensity was increased to the 50% VO_{2peak} eliciting power output, and if needed (3) power output was adjusted from 7 to 10 min of exercise.

Both T_{re} and T_{sk} were continuously monitored throughout all phases of each trial. T_{re} and T_{sk} were recorded during the last 5 min of passive rest, and every 5 min during exercise and recovery. Participants were also asked to assess their TS using a standard 0–8 scale (Gagge et al., 1967) during the last 5 min of passive rest, and every 5 min during exercise and recovery. Participants were prevented from ingesting any fluids throughout the full duration of each trial (start of passive rest through to the end of recovery). Nude body mass was again measured immediately after the end of recovery in each trial to allow for the determination of fluid loss via change in nude body mass.

Recovery Phase

The 60 min post-exercise seated rest portion of the study (REC_{0-60}), where each of the three cooling interventions was applied while within 36°C, 45% RH conditions based on the randomized order, was split into two epochs; the first time-limited, 15 min recovery period (REC_{0-15}) and the prolonged 60 min recovery period (REC_{0-60}) to allow for short- and long-term responses to be monitored and assessed. The control condition consisted of passive seated recovery following exercise cessation and had participants sit quietly with minimal movement in a backed chair with their feet planted on the floor.

Hand Cooling

The hand cooling device (CoreControl, AVAcore Technologies, Ann Arbor, MI) was administered with participants seated in a backed chair, and fixed to the dominant hand and forearm. Participants were instructed to place their hand over the small soft disc at the bottom of the device to ensure standardized exposure. Using a water-dwelling thermometer, the temperature of the continuously perfusing water was maintained at ∼17°C with the addition of more ice to the slurry mix when necessary. The device also used a low pressure (∼15 mmHg) vacuum around the forearm to facilitate blood transport through the AVAs and prevent acute vasoconstriction. This low pressure vacuum was maintained for the duration of the exposure.

Ice Vest

The ice vest (Kool Max Poncho Vest, Polar Products, Stow, Ohio) was adjustable to fit all torso sizes, and was equipped with frozen ice packs (Kool Max Ice Packs, Polar Products, Stow, Ohio) fixed in 10 individual pockets dispersed equally on the front and the back of the torso. Upon initial application, the temperature of the packs was 0°C with no attempt to control heat gain over the duration of application. Participants were seated in a backed chair with their feet planted on the floor.

Statistical Analysis

A one-way analysis of variance (ANOVA) was used to assess exercise differences in VO_2, pre-post trial nude body mass, as well as change in $\overline{T}_b$, S, T_{re}, $\overline{T}_{sk}$, HR, and TS across the three experimental exercise trials. Normality of dependent variables was assessed using a Shapiro-Wilk test and sphericity for each main and interaction effect using Mauchly's sphericity test. Delta data for $\overline{T}_b$, S, T_{re}, $\overline{T}_{sk}$, HR, and TS from recovery baseline values were analyzed using linear mixed modeling with restricted maximum likelihood and Satterthwaite small-sample correction of degrees of freedom. The model had fixed factors of condition (control, ice vest, single-hand cooling), time (REC_{0-15}, REC_{0-60}), and condition*time with a covariate (value at start of recovery) equal across conditions. The model included a random intercept by participant to account for the hierarchical data structure (repeated measures within participants). A secondary linear mixed model with baseline recovery covariate was conducted to further investigate REC_{0-15} components (REC_{0-5}, REC_{5-10}, REC_{10-15}) across conditions. For all statistical procedures, when appropriate, *post-hoc* pairwise comparisons were conducted using a Sidak correction to reduce Type I error. Statistical significance was set at $p < 0.05$ for all analyses, and data was analyzed using IBM SPSS version 26.0 (IBM Corporation, Chicago, IL). All absolute data are presented as mean ± standard deviation and all delta data are

presented as mean difference with 95% confidence intervals where appropriate.

RESULTS

Exercise in the Heat

The VO_2 averaged across all exercise bouts was 1.65 ± 0.42 L·min^{-1} (51 ± 2% VO_{2peak}), which was elicited by 92 ± 35 W. No differences in VO_2 existed between experimental trials ($p = 0.483$). Thermo-physiological responses were not different across the three exercise bouts in the heat, with similar heat storage (control: 89 ± 35 W, ice vest: 91 ± 32 W, single hand-cooling: 82 ± 26 W; $p = 0.297$) and end-exercise T_{re} (control: 38.28 ± 0.31°C, ice vest: 38.37 ± 0.24°C, single hand-cooling: 38.22 ± 0.40°C; $p = 0.250$) across the three trials.

Post-exercise Recovery in the Heat

Thermoregulation

Stored heat (S) reduction during REC_{0-15} was different between cooling conditions, with the application of an ice vest eliciting a significantly greater reduction of stored heat across time-limited recovery (REC_{0-15}) compared to a non-cooling control (−84 [−132 to −37] W, $p < 0.0001$) and single-hand cooling (−74 [−125 to −24] W, $p = 0.002$). A deeper look into the components of time-limited recovery indicates that heat loss was increased across the first 5 min of ice vest application when compared to both control (−44 [−73 to −15] W, $p = 0.001$) and single-hand cooling (−31 [−61 to −0.3] W, $p = 0.047$). Specifically for the ice vest recovery condition, the rate of heat loss was greater during the time-limited recovery than during prolonged recovery (−69 [−107 to −31] W, $p = 0.001$). Mean delta and individual data for S can be found in **Table 1** and **Figure 1**, respectively.

No differences between conditions existed with regards to T_{re} reduction across time-limited or prolonged recovery ($p = 0.990$). Across all conditions, T_{re} recovery was greater during REC_{0-60} compared to REC_{0-15} (control: −0.45 [−0.67 to −0.24]°C, $p < 0.0001$; ice vest: −0.47 [−0.69 to −0.25]°C, $p < 0.0001$; single-hand cooling: −0.45 [−0.67 to −0.23]°C, $p < 0.0001$), which was expected given the difference in total recovery length. However, T_{re} recovery during REC_{0-15} accounted for 36% of the total T_{re} reduction during the full 60 min recovery, while it only accounted for 32% of T_{re} recovery for the control and single-hand cooling conditions. Interestingly, T_{re} recovery with the ice vest condition was significantly larger across both 5–10 min (−0.09 [−0.17 to −0.002]°C, $p = 0.042$) and 10–15 min (−0.10 [−0.18 to −0.02]°C, $p = 0.009$) of recovery compared to the first 0–5 min of recovery, indicating an initial delay in the ability of an ice vest to reduce T_{re}. Mean delta and individual data for T_{re} is visualized in **Table 1** and **Figure 2**, respectively.

During time-limited recovery, the ice vest was more effective at reducing $\overline{T}_{sk}$ compared to both control (−1.07 [−2.00 to −0.13]°C, $p = 0.021$) and single-hand cooling (−1.07 [−2.01 to −0.14]°C, $p = 0.020$). More specifically, the ice vest heightened recovery of $\overline{T}_{sk}$ within the first 5 min of recovery compared to both control (−0.74 [−1.12 to −0.36]°C, $p < 0.0001$) and single-hand cooling (−0.69 [−1.07 to −0.30]°C, $p < 0.0001$). The first 5 min of recovery appear to be especially important for the $\overline{T}_{sk}$ reducing effects of the ice vest, as $\overline{T}_{sk}$ was reduced more in the first 5 min as compared to 5–10 (−0.59 [−0.97 to −0.21]°C, $p = 0.001$) or 10–15 (−0.61 [−0.99 to −0.23]°C, $p = 0.001$) minutes. $\overline{T}_{sk}$ was also more effectively reduced with ice vest application when considering prolonged exercise recovery (REC_{0-60}) compared to both control (−1.62 [−2.56 to −0.68]°C, $p < 0.0001$) and single-hand cooling (−1.68 [−2.61 to −0.74]°C, $p < 0.0001$). Although, the ice vest was efficient in reducing $\overline{T}_{sk}$ within the first 15 min of recovery, considerable further skin cooling was apparent with extending the length of vest cooling exposure (REC_{0-60}). For the ice vest condition, prolonged recovery elicited greater reductions in $\overline{T}_{sk}$ compared to time-limited recovery (−1.29 [−2.05 to −0.53]°C, $p = 0.001$). However, $\overline{T}_{sk}$ recovery during REC_{0-15} accounted for 56% of the total $\overline{T}_{sk}$ recovery during the full 60 min recovery for the ice vest, while it only accounted for 46% of $\overline{T}_{sk}$ recovery for control and 47% for single-hand cooling. Mean delta and individual data for $\overline{T}_{sk}$ can be found in **Table 1** and **Figure 3**, respectively.

Considering the entire recovery period (REC_{0-60}), an ice vest was able to more effectively recover $\overline{T}_b$ compared to both control (−0.38 [−0.69 to −0.07]°C, $p = 0.012$) and single-hand cooling (−0.39 [−0.70 to −0.08]°C, $p = 0.010$), although this could not confidently be extended to the time-limited recovery window (REC_{0-15}). Considering the components of the time-limited recovery, an ice vest was also able to more effectively reduce $\overline{T}_b$ compared to control specifically during the first 5 min of recovery (−0.13 [−0.23 to −0.03]°C, $p = 0.008$), likely the result of $\overline{T}_{sk}$ reductions. Across all conditions, $\overline{T}_b$ was recovered to a greater extent with prolonged recovery compared to time-limited recovery (control: −0.51 [−0.76 to −0.26]°C, $p < 0.0001$; ice vest: −0.63 [−0.89 to −0.38]°C, $p < 0.0001$; single-hand cooling: −0.50 [−0.75 to −0.24]°C, $p < 0.0001$). However, $\overline{T}_b$ recovery during REC_{0-15} accounted for 46% of the total $\overline{T}_b$ recovery during REC_{0-60} for the ice vest, while it only accounted for 36% of $\overline{T}_b$ recovery for control and 37% for single-hand cooling. Mean delta and individual data for $\overline{T}_b$ can be found in **Table 1** and **Figure 4**, respectively.

Single-hand cooling did not display any significant differences compared to a non-cooling control or ice vest with regards to change in S, $\overline{T}_b$, T_{re}, or $\overline{T}_{sk}$ at REC_{0-15} or REC_{0-60} of post exercise recovery.

Heart Rate

Across all conditions, HR recovery was greatest during the first 5 min of recovery compared to the subsequent 10 min but was overall greater during REC_{0-60} vs. REC_{0-15} as a function of time (see **Tables 1**, **2**). During time-limited recovery (REC_{0-15}) both the ice vest (−9 [−15 to −3] bts·min^{-1}, $p = 0.002$) and single-hand cooling (−7 [−13 to −1] bts·min^{-1}, $p = 0.021$) were able to recover HR more effectively compared to control however, only the ice vest was able to recover HR more effectively compared to control (−7 [−13 to −1] bts·min^{-1}, $p = 0.029$) during the prolonged recovery window (REC_{0-60}). Mean delta and individual data for HR is visualized in **Table 1** and **Figure 5**, respectively.

TABLE 1 | Delta mean [95% confidence interval] rectal temperature (T_{re}), skin temperature ($\overline{T}_{sk}$), body temperature ($\overline{T}_{b}$), stored heat (S), heart rate (HR), and thermal sensation (TS) during prolonged recovery (REC_{0-60}), and time-limited recovery (REC_{0-15}) with 5 min block component analysis (REC_{0-5}, REC_{5-10}, REC_{10-15}) when using a non-cooled control, ice vest, and single hand-cooling during heated exercise recovery.

	Recovery Time (min)	Control	Ice Vest	Single-Hand Cooling	Condition	Time	Interaction
T_{re} (°C)	0–5	−0.05 [−0.10 to 0.003]	−0.03 [−0.08 to 0.03]	−0.07 [−0.12 to −0.02]	*p = 0.619*	***p = 0.037***	*p = 0.252*
	5–10	−0.07 [−0.13 to −0.02]	−0.11 [−0.16 to −0.06][a]	−0.06 [−0.11 to −0.01]			
	10–15	−0.09 [−0.14 to −0.04]	−0.13 [−0.18 to −0.08][a]	−0.08 [−0.13 to −0.03]			
	0–15	−0.21 [−0.39 to −0.03][b]	−0.26 [−0.44 to −0.09][b]	−0.21 [−0.39 to −0.04][b]	*p = 0.673*	***p < 0.0001***	*p = 0.990*
	0–60	−0.66 [−0.84 to −0.49]	−0.73 [−0.91 to −0.56]	−0.66 [−0.84 to −0.49]			
$\overline{T}_{sk}$ (°C)	0–5	−0.22 [−0.44 to −0.001]	−0.96 [−1.18 to −0.74][*†]	−0.27 [−0.49 to −0.05]	***p < 0.0001***	***p = 0.010***	***p = 0.039***
	5–10	−0.20 [−0.42 to 0.02]	−0.37 [−0.59 to −0.15][a]	−0.14 [−0.36 to 0.08]			
	10–15	−0.19 [−0.41 to 0.03]	−0.35 [−0.57 to −0.13][a]	−0.19 [−0.41 to 0.03]			
	0–15	−0.61 [−1.17 to −0.04]	−1.67 [−2.24 to −1.10][*†b]	−0.60 [−1.17 to −0.03]	***p < 0.0001***	***p < 0.0001***	*p = 0.463*
	0–60	−1.34 [−1.91 to −0.77]	−2.96 [−3.53 to −2.39][*†]	−1.28 [−1.85 to −0.71]			
$\overline{T}_{b}$ (°C)	0–5	−0.08 [−0.14 to −0.02]	−0.21 [−0.27 to −0.15][*]	−0.11 [−0.17 to −0.05]	***p = 0.001***	*p = 0.611*	*p = 0.755*
	5–10	−0.10 [−0.16 to −0.04]	−0.16 [−0.22 to −0.10]	−0.08 [−0.14 to −0.02]			
	10–15	−0.11 [−0.17 to −0.05]	−0.17 [−0.23 to −0.11]	−0.10 [−0.16 to −0.04]			
	0–15	−0.29 [−0.50 to −0.08][b]	−0.54 [−0.75 to −0.34][b]	−0.29 [−0.50 to −0.08][b]	***p = 0.001***	***p < 0.0001***	*p = 0.697*
	0–60	−0.80 [−1.01 to −0.59]	1.18 [−1.39 to −0.97][*†]	−0.79 [−1.00 to −0.58]			
S (W)	0–5	−19 [−36 to −1]	−63 [−80 to −45][*†]	−32 [−51 to −13]	***p < 0.0001***	*p = 0.452*	*p = 0.569*
	5–10	−24 [−41 to −6]	−44 [−61 to −27]	−20 [−39 to −0.4]			
	10–15	−30 [−48 to −12]	−49 [−66 to −32]	−29 [−48 to −9]			
	0–15	−72 [−100 to −44]	−156 [−184 to −129][*†b]	−82 [−112 to −51]	***p < 0.0001***	***p = 0.005***	*p = 0.097*
	0–60	−58 [−86 to −31]	−87 [−115 to −60]	−62 [−93 to −32]			
HR (bts·min^{-1})	0–5	−47 [−52 to −42]	−56 [−61 to −51]	−54 [−59 to −49]	*p = 0.339*	***p < 0.0001***	*p = 0.213*
	5–10	−7 [−12 to −2][a]	−4 [−9 to 1][a]	−4 [−9 to 2][a]			
	10–15	−3 [−8 to 2][a]	−6 [−11 to −1][a]	−6 [−12 to −1][a]			
	0–15	−57 [−65 to −50][b]	−67 [−74 to −60][*b]	−64 [−72 to −57][*b]	***p < 0.0001***	***p < 0.0001***	*p = 0.473*
	0–60	−69 [−76 to −62]	−76 [−83 to −69][*]	−72 [−79 to −65]			
TS	0–5	−0.9 [−1.1 to −0.6]	−1.0 [−1.3 to −0.8]	−1.0 [−1.2 to −0.8]	*p = 0.426*	***p < 0.0001***	*p = 0.993*
	5–10	−0.3 [−0.6 to −0.1][a]	−0.5 [−0.7 to −0.2][a]	−0.5 [−0.7 to −0.2][a]			
	0–15	−0.2 [−0.5 to −0.03][a]	−0.3 [−0.5 to −0.01][a]	−0.3 [−0.5 to −0.05][a]			
	10–15	−1.4 [−1.8 to −1.0][b]	−1.8 [−2.2 to −1.4][b]	−1.8 [−2.2 to −1.4][b]	*p = 0.186*	***p < 0.0001***	*p = 0.906*
	0–60	−2.3 [−2.7 to −1.9]	−2.6 [−3.0 to −2.2]	−2.5 [−2.9 to −2.1]			

Mixed Linear Model Covariates: T_{re}: 38.29°C; $\overline{T}_{sk}$: 37.32°C; $\overline{T}_{b}$: 38.09°C; HR: 159 (bts·min^{-1}); TS: 6.8; S: 87 W.

[a] *Significantly ($p < 0.05$) different than 0–5 min.*

[b] *Significantly ($p < 0.05$) different than 0–60 min.*

[*] *Significantly different than control ($p < 0.05$).*

[†] *Significantly different than single-hand cooling ($p < 0.05$).*

Bold values represent significant ($p < 0.05$) main and/or interaction effects.

Perceptual Index

Across all conditions, reductions in TS were greatest during the first 5 min of recovery compared to the subsequent 10 min but were overall greater during REC_{0-60} vs. REC_{0-15} as a function of time. No differences between conditions existed with regards to TS reduction across time-limited or prolonged recovery ($p = 0.906$). Mean delta data for TS is presented in **Table 1**.

Body Fluid Balance

Pre-post fluid loss, as indicated by change in nude body mass, was similar across the three

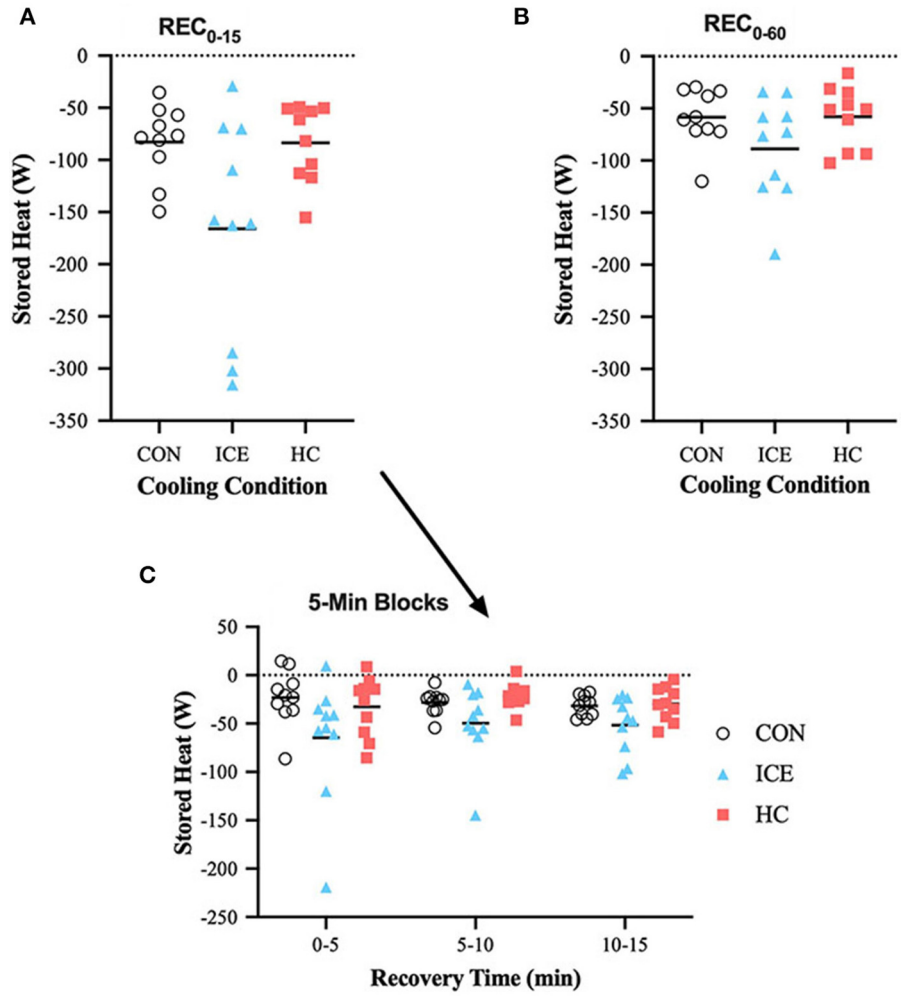

FIGURE 1 | Mean (bar) and individual delta stored heat (*S*) during REC_{0-15} **(A),** REC_{0-60} **(B),** and 5-min blocks during REC_{0-15} **(C)** when using ice vest (ICE), single-hand cooling (HC), and non-cooled control (CON) during heated exercise recovery.

trials (single-hand cooling: 1.19 ± 0.56%, ice vest: 1.27 ± 0.47%, non-cooling control: 1.40 ± 0.68%; $p = 0.490$).

DISCUSSION

The primary aim of this study was to investigate the effectiveness of time-limited and long-term active cooling compared to passive recovery following prolonged submaximal exercise in the heat with sport-mimicking recovery conditions. Neither the ice vest nor single-hand cooling were able to improve T_{re} or TS recovery at any time post-exercise compared to a non-cooling control. However, during time-limited recovery an ice vest induced greater recovery of $\overline{T}_{sk}$ and improved heat loss compared to a non-cooling control, both of which were apparent within the first 5 min of recovery. Reductions in $\overline{T}_{sk}$ were also greater with an ice vest compared to a non-cooling control when considering the prolonged 60 min recovery (REC_{0-60}), translating into greater reductions in calculated mean body temperature. Single-hand cooling was unable to augment recovery of $\overline{T}_b$, S, or $\overline{T}_{sk}$ compared to a non-cooling control with any investigated recovery length. Yet, both the ice vest and single-hand cooling enhanced HR recovery compared to a non-cooling control early during exercise recovery (REC_{0-15}), although this remained distinguishable into prolonged recovery (REC_{0-60}) only for the ice vest (see **Table 2**).

A secondary aim of this study was to examine thermo-physiological and perceptual differences during heated recovery between negative pressure hand cooling and the application of an ice vest. An ice vest was no more effective at reducing T_{re}, TS, or HR compared to single-hand cooling. That said, compared to single-hand cooling, an ice vest more effectively reduced $\overline{T}_{sk}$ during both time-limited (REC_{0-15}) and prolonged recovery (REC_{0-60}). The time-limited recovery reduction in $\overline{T}_{sk}$ elicited by an ice vest concurrently enhanced heat loss compared to single-hand cooling (see **Table 2**).

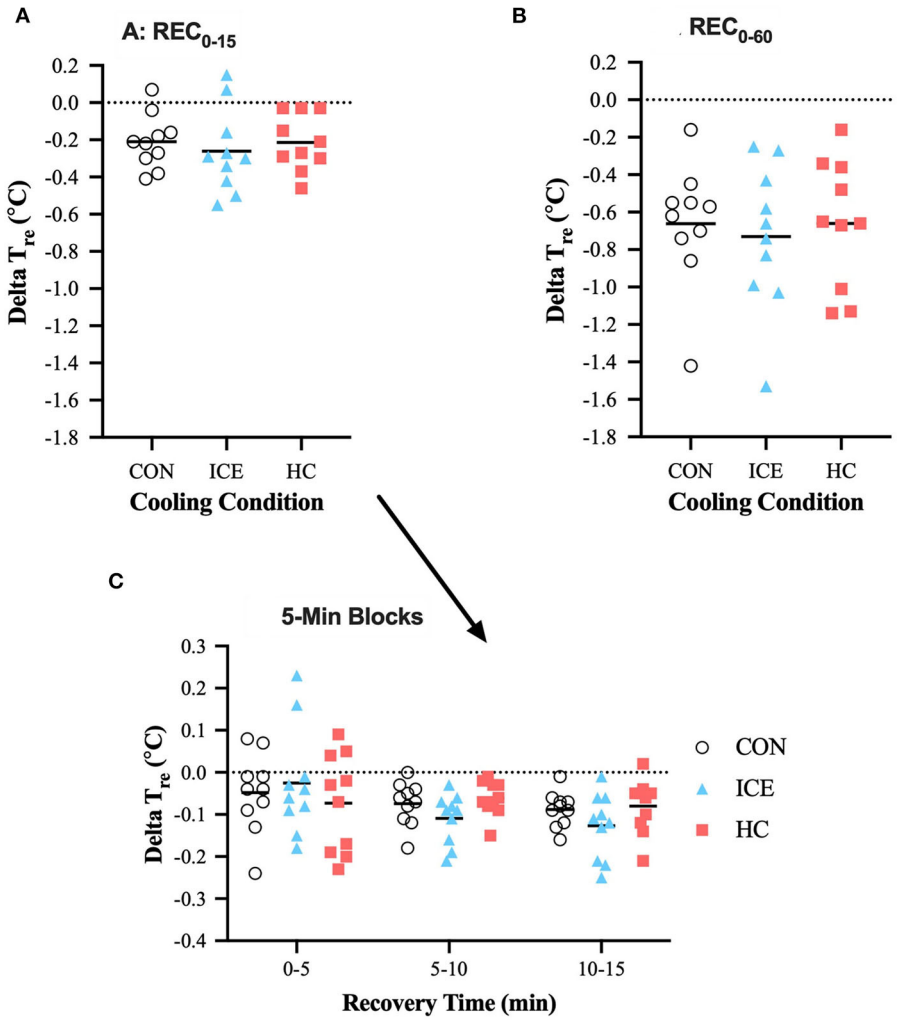

FIGURE 2 | Mean (bar) and individual delta rectal temperature (T_{re}) during REC_{0-15} **(A)**, REC_{0-60} **(B),** and 5-min blocks during REC_{0-15} **(C)** when using ice vest (ICE), single-hand cooling (HC), and non-cooled control (CON) during heated exercise recovery.

Exercise Recovery: Ice Vest Influence on Thermoregulatory Recovery

Wearing an ice vest during time-limited heated exercise recovery enhanced heat loss and $\overline{T}_{sk}$ reductions compared to a non-cooling control The application of 0°C ice packs to the temperature elevated skin provided a widened heat loss gradient that facilitated greater heat loss from the blood perfusing the skin. Due to the 30% contribution of the chest thermocouple to the $\overline{T}_{sk}$ calculation (Ramanathan, 1964), the enhanced reduction of $\overline{T}_{sk}$ may be a more accurate representation of the change in chest skin temperature than a change in whole body skin temperature. Despite this influence, a lowered T_{sk} at the chest likely still has an impact on thermoregulatory function via peripheral thermoregulatory mechanisms (Huizenga et al., 2004).

Interestingly, the improvements in $\overline{T}_{sk}$ reduction by the phase-changing ice vest were not able to translate to a significantly greater recovery of T_{re} compared to a non-cooling control during heated exercise recovery. Similar investigations support this notion that phase-changing cool inserts may be effective at cooling the skin but tend to have a substantially smaller effect on measured deep body temperature (Duffield and Marino, 2007; Barwood et al., 2009). One possible explanation lies in the large surface area covered by the vest (∼26% of total body surface area) which may impede immediate post-exercise evaporative cooling that would otherwise contribute to reductions in deeper body temperature (Barwood et al., 2009). As subjects were cooled by the ice vest, so too the ice vest was warmed by the subject and ambient environment, further reducing heat loss gradients and therefore cooling magnitude over time. Cooling investigations using temperature-maintained liquid perfused vests suggest they may be more viable to manipulate deeper body temperature (Balldin et al., 2007; Amorim et al., 2010), however it may be argued that maintenance of water temperature or other liquid-perfused vests may be logistically difficult, especially when attempting to cool in thermally-challenging environmental conditions. Despite the

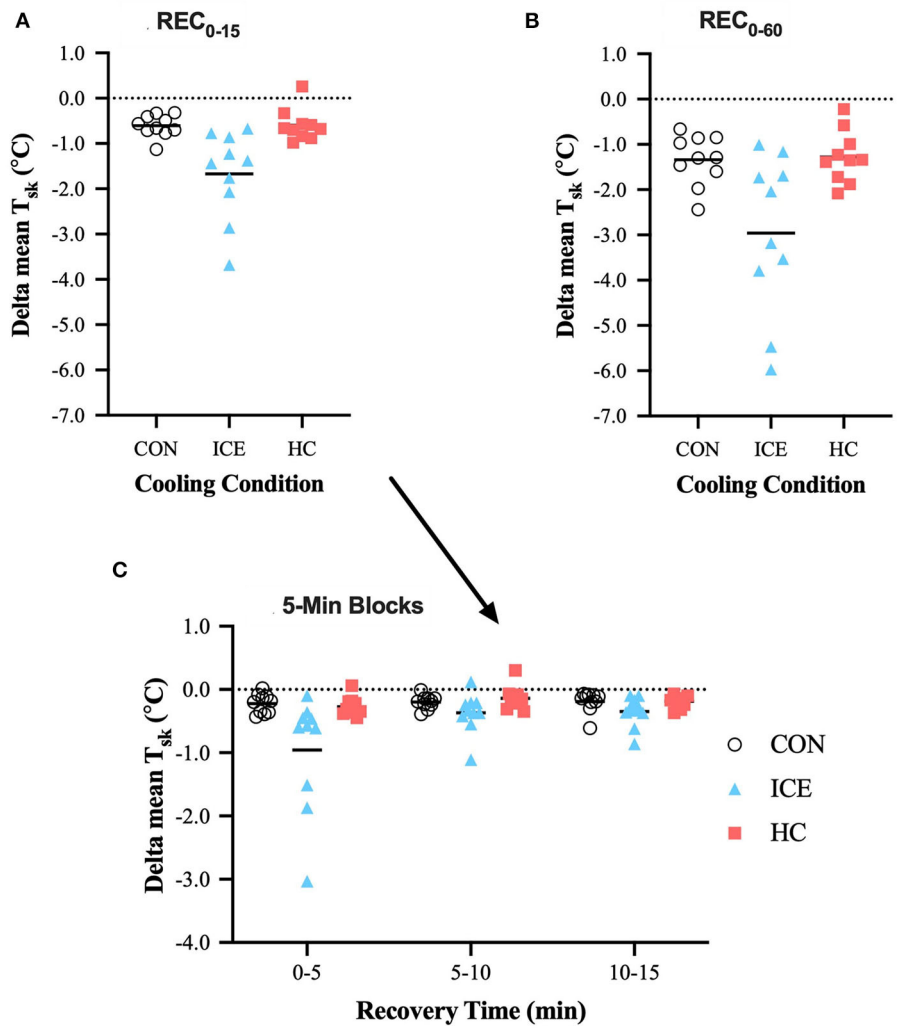

FIGURE 3 | Mean (bar) and individual delta mean skin temperature (T_{sk}) during REC_{0-15} **(A)**, REC_{0-60} **(B)**, and 5-min blocks during REC_{0-15} **(C)** when using ice vest (ICE), single-hand cooling (HC), and non-cooled control (CON) during heated exercise recovery.

core temperature independent perceptual link between TS and localized $\overline{T}_{sk}$ (Schlader et al., 2011), TS was also not significantly altered by the ice vest. Personal subjective bias resulting from differences in heat tolerance and psychological bias may lead to overestimation of thermal relief upon cessation of exercise.

Wearing an ice vest reduced HR beyond that of a non-cooling control during both time-limited and prolonged heated recovery. A reduction in peripheral vasodilation, due to ice vest application, likely results in an increase in central venous pressure via a shift of cutaneous blood into the thoracic vasculature. This shift simultaneously stimulates high arterial pressure and low cardiopulmonary pressure baroreflexes, effectively eliciting an increase in cardiac vagal tone (Pump et al., 2001) and resultant expedited decrease in HR beyond that demonstrated by passive exercise recovery. This hypothesis along with the much smaller T_{re} recovery within the first 5 min of recovery for the ice vest further supports the notion that reactive peripheral vasoconstriction, depending on the magnitude as reflective of cold severity, may to some extent reduce the immediate cooling capability of the ice vest. However, it may be argued that the phase-changing nature of the ice vest reduces the impact of this initial vasoconstrictive clamping as the ice packs melt. Similarly to the ice vest, single-hand cooling was able to reduce HR beyond that of the non-cooling control after 15 min of exposure. This is to some extent surprising, but may indicate that even single hand cooling is sufficient to initiate reflexive baroreceptor controlled increases in cardiac vagal tone.

An ice vest was superior to single-hand cooling in reducing $\overline{T}_{sk}$ during REC_{0-5}, REC_{0-15}, and REC_{0-60} and heat storage during time-limited recovery. This may be explained by the ice vest's greater surface area (∼26 vs. 1% of total body surface area), upon which the widened cooling gradient is applied. Further evaluation of the rate of T_{re} recovery for all conditions, especially during the first 5 min of recovery, indicates that single-hand cooling may be the quickest, of the strategies tested, to initially reduce T_{re} (single-hand cooling: $-0.015°C \cdot min^{-1}$, ice vest: $-0.007°C \cdot min^{-1}$, non-cooling control: $-0.010°C \cdot min^{-1}$). The rate of cooling by single-hand cooling in the present study

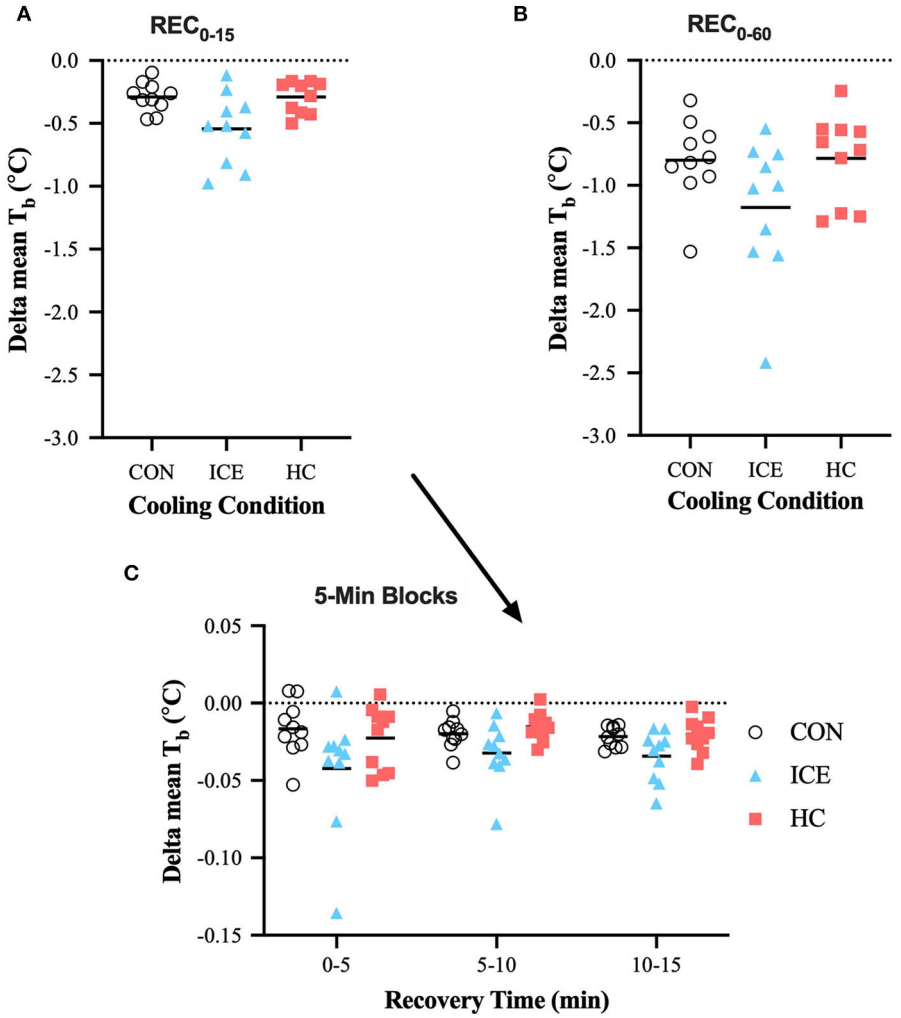

FIGURE 4 | Mean (bar) and individual delta mean body temperature (T_b) during REC_{0-15} **(A)**, REC_{0-60} **(B),** and 5-min blocks during REC_{0-15} **(C)** when using ice vest (ICE), single-hand cooling (HC), and non-cooled control (CON) during heated exercise recovery.

is similar to that reported in previous literature investigating cooling of one hand (-0.017 °C·min^{-1}) (Grahn et al., 2009). The comparatively small rate of T_{re} change with the ice vest supports the likelihood of reactive cutaneous vasoconstriction and/or a sudden reduction in evaporative cooling due to the surface area covered by vest application. Two subjects (**Figure 2**) demonstrated continued rise in T_{re} despite 5 min of vest application indicating that although a quick change in skin temperature did tend to occur within the first 5 min (**Figure 3**) it likely contributed to some degree of vasoconstriction and/or lack of evaporative cooling capability. While an ice vest was slower to recover T_{re} immediately upon application compared to single-hand cooling and a non-cooling control, the ice vest demonstrated superior cooling rates at both 5–10 (single-hand cooling: -0.012°C·min^{-1}, ice vest: -0.022°C·min^{-1}, non-cooling control: -0.015°C·min^{-1}) and 10–15 min (single-hand cooling: -0.017°C·min^{-1}, ice vest: -0.025°C·min^{-1}, non-cooling control: -0.018°C·min^{-1}). Combined with the larger influence of an ice vest on thermo-physiological responses during heated exercise recovery, these rates indicate that, of the methods tested, an ice vest may be the most applicable to achieve effective cooling during time-limited recovery.

Exercise Recovery: Single-Hand Cooling Influence on Thermoregulatory Recovery

Single-hand cooling using a negative pressure device was unable to enhance heated exercise thermoregulatory recovery beyond that of the non-cooling control regardless of application length (15 vs. 60 min). A similar investigation capable of minimally increasing T_{re} to 37.72°C also indicated a single-hand cooling device was no more effective at decreasing T_{re} following heated (35°C, 85% RH) exercise than control or vest conditions (Balldin et al., 2007). This minor thermal gain likely stimulated a smaller volume of blood to the skin providing a very small heat dissipation gradient, thereby minimizing the influence of the hand-cooling device. Still, even with much greater

TABLE 2 | Visual summary of thermo-physiological and thermal perceptual differences between cooling conditions across immediate (REC_{0-5}), time-limited (REC_{0-15}), and prolonged recovery (REC_{0-60}).

	Ice Vest > Control			Single-Hand Cooling > Control			Ice Vest > Single-Hand Cooling		
	0–5	0–15	0–60	0–5	0–15	0–60	0–5	0–15	0–60
T_{re} (°C)									
$\overline{T}_{sk}$ (°C)									
$\overline{T}_{b}$ (°C)									
S (W)									
HR (bts·min^{-1})									
TS									

Gray boxes indicate significant difference ($p < 0.05$) for the column described difference in recovery magnitude; White boxes indicate a lack of significant difference ($p > 0.05$) for the column described difference in recovery magnitude; 0–5, 0–5 min of recovery; 0–15, 0–15 min of recovery; 0–60, 0–60 min of recovery.

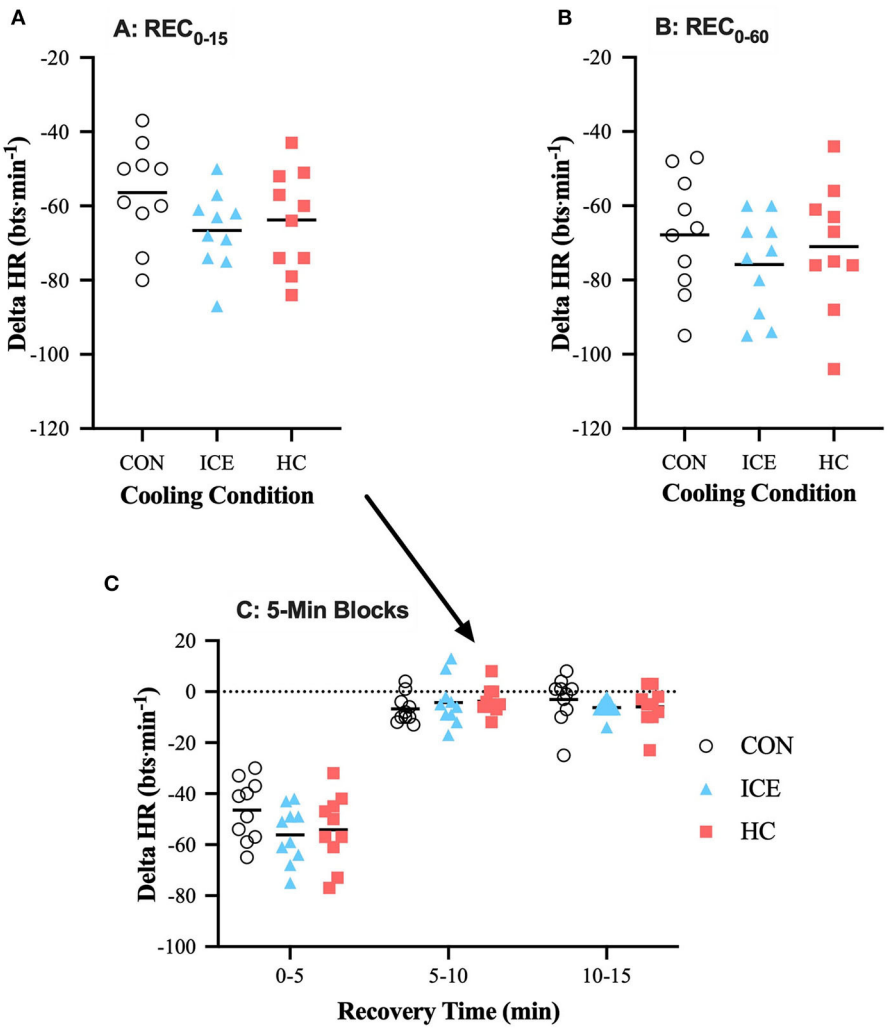

FIGURE 5 | Mean (bar) and individual delta heart rate (HR) during REC_{0-15} **(A)**, REC_{0-60} **(B)**, and 5-min blocks during REC_{0-15} **(C)** when using ice vest (ICE), single-hand cooling (HC), and non-cooled control (CON) during heated exercise recovery.

thermoregulatory stress, the result of both a higher ambient temperature (42°C) and heat loss retardant clothing, negative pressure single hand cooling was no more effective at deep body temperature recovery following exercise (T_{re} 38.5°C) than a non-cooling control (Amorim et al., 2010). The significantly lower body surface area cooled with single-hand cooling vs. a torso-encompassing vest may help to explain single-hand cooling's thermoregulatory shortcomings as both the present study and Amorim et al. indicate vest application, a phase-changing ice and cold water perfused vest, respectively, as a superior cooling technique. The unique presence of a continued environmental heat stress during recovery with the cooling device may therefore reduce the overall likelihood of single-hand cooling to enhance core temperature recovery.

Core temperature at cessation of exercise, and therefore start of exercise recovery, likely also plays a key role in the efficacy of single-hand cooling as a thermoregulation enhancing device. As both core and skin temperatures tend to rise commensurately (Gleeson, 1998), a greater achieved core temperature is likely reflective of a greater skin temperature, effectively providing a widened heat loss gradient when a cooling device is applied that favors improved heat loss. Firefighters recovering from heavy physical work in 36°C, 44% RH performed to a T_{re} of 39°C cooled 144% more with single-hand cooling for 40 min compared to passive cooling (Zhang et al., 2009). Furthermore, individuals heated using 39.3°C 38% RH ambient conditions paired with vigorous walking to a T_{re} of 39.44°C experienced enhanced T_{re} recovery following a 20 min application of an ∼17°C cooling device on each hand (Adams et al., 2016). Barwood et al. (2009) additionally investigated a two-hand cold water immersion exercise recovery strategy that induced heat loss of ∼162 W after 10–15 min and was effective at augmenting reduction in T_{re} compared to a non-cooling control condition (heat loss ∼99 W). Heat loss induced via this dual hand cold water immersion is similar to that induced by the ice vest in the current investigation over a similar 15 min time period, ∼166 W, and yet the ice vest was unable to elicit enhanced T_{re} recovery. As heat storage is determined utilizing a change in $\overline{T}_b$, it appears that dual hand cooling and ice vest application may modulate calculated body temperature divergently within the first 15 min of cooling, with dual hand cooling having greater influence on T_{re} hypothesized to result from significant cooling of the blood, without significant change in $\overline{T}_{sk}$ and vice versa for ice vest application.

Single-hand cooling in our investigation induced heat loss of ∼84 W, roughly half that demonstrated by dual-handed immersion at the same ∼17°C exposure temperature (Barwood et al., 2009), over the first 15 min of recovery, indicating the importance of maximizing the amount of surface area cooled to thermoregulatory recovery. Additionally as hand water immersion sans the use of a negative pressure vacuum seal was successful in augmenting T_{re} recovery, the necessity or usefulness of this feature to offset otherwise inhibiting vasoconstriction is thought questionable. Overall, literature suggests the influence of cooling with or without a vacuum on heat loss appears small (∼12 W) (Kuennen et al., 2010). Ultimately, the cooling effectiveness of cold water hand immersion is determined by the maintenance of peripheral blood flow as well as the heat loss gradient magnitude between the skin and the immersion water. Water of ∼15°C is sufficient to induce peripheral vasoconstriction in individuals with maintained deep body temperatures (Tipton et al., 1993), rendering the selected hand cooling temperature of the current study near optimal. Collectively, literature that has been successful at modulating T_{re} during recovery in the heat beyond that possible by passive cooling demonstrates that hand cooling may be capable of greater cooling compared to a non-cooling control. It does appear that a few conditions may be necessary to facilitate: (1) T_{re} of ∼39°C that facilitates a larger heat loss gradient and reduces the influence of peripheral vasoconstriction; and (2) the use of dual hand vs. single hand cooling to maximize the amount of total body surface area cooled.

Methodological limitations to measurement of thermoregulatory variables must also be considered. Post-exercise T_{re} values indicate a similar level of thermal stress between our three separate trial conditions. Similar literature places this exercise thermal stress in a "moderately high" category. Other investigations, utilizing T_{re}, have successfully produced a larger thermal gain, achieving temperatures as high as 39–39.44°C (Zhang et al., 2009; Adams et al., 2016). These temperatures were set as exercise end-points rather than a product of a given duration of exercise time and were accomplished with a combination of both environmental and clothing manipulation. We chose instead to impose a time relative end-point to exercise (60 min) for two reasons: (1) The subject population recruited consisted primarily of natives to a variable climate region with little exposure to the experience of performing exercise in considerable heat. For this reason, two preliminary subjects exhibited significant difficulty in completing the exercise task to a core temperature >38.5°C; (2) In a real-world scenario, the likelihood of athletes or occupational workers performing the same duration of physical work is much greater than achieving an identical exercise core temperature. Numerous investigators have provided data to suggest the inability of T_{re} to respond as readily to rapid changes in core temperature compared to esophageal temperature (Lee et al., 2000; Easton et al., 2007). While subjects wore heavy heat retardant uniforms after exercising in the heat to an esophageal temperature of 38.8°C, a single-hand cooling device elicited significantly lower esophageal and skin temperatures from 15 to 50 min of exercise recovery (Kuennen et al., 2010). Esophageal temperatures may offer improved sensitivity and responsiveness over rectal temperatures, especially as rate of cooling is prioritized. Esophageal temperature, though, does pose significant practicality concerns as many subjects struggle to place and tolerate the temperature probe. Due to logistical difficulties with efficiently replicating wind velocity, it was omitted from the current design. Wind velocity should, however, be considered as a variable that may reduce or otherwise alter the efficiency of the investigated cooling devices.

CONCLUSIONS

Wearing an ice vest but not single hand-cooling, using a cold water low pressure vacuum device, was effective at reducing thermo-physiological strain during both time-limited

and prolonged heated exercise recovery. When a prolonged heated recovery time is accessible, an ice vest is superior to both passive and single-hand cooling to reduce mean body and skin temperatures. An ice vest also appears efficacious in reducing mean skin temperature after as little as 15 min of application, making its use specifically advantageous for time-limited sport or occupational recovery. Although the lower mean skin temperatures after 15 and 60 min were not able to elicit a significantly lower T_{re} or a reduced thermal strain, this peripheral activity may still impact overall thermoregulatory function. With moderately, rather than severely, elevated core temperatures, time-limited sport thermoregulatory recovery in the heat is likely enhanced with the use of techniques that encompass a larger degree of body surface area, like an ice vest. This may be particularly meaningful during sporting half-time to proactively offset progressive increases in thermal stress during a second-half physical effort. Further research is necessary to pinpoint the exact environmental conditions and level of hyperthermia for which single-hand cooling or phase-changing ice vest application may be most beneficial.

DATA AVAILABILITY STATEMENT

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

ETHICS STATEMENT

The studies involving human participants were reviewed and approved by Human Subjects Institutional Review Board Western Michigan University. The patients/participants provided their written informed consent to participate in this study.

AUTHOR CONTRIBUTIONS

AS performed the experiments and collected all participant data. AS and RS conceived and designed the experiments, analyzed the data, summarized the results, wrote and revised the manuscript. All authors contributed to the article and approved the submitted version.

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Intramuscular Temperature Changes in the Quadriceps Femoris Muscle After Post-Exercise Cold-Water Immersion (10°C for 10 min): A Systematic Review With Meta-Analysis

Livia Freitag[1], *Ron Clijsen*[1,2,3,4], *Carlina Deflorin*[1], *Wolfgang Taube*[5], *Jan Taeymans*[3,4] *and Erich Hohenauer*[1,2,4,5,6]*

[1] Rehabilitation Research Laboratory 2rLab, Rehabilitation and Exercise Science Group, Department of Business Economics, Health and Social Care, University of Applied Sciences and Arts of Southern Switzerland, Landquart, Switzerland, [2] International University of Applied Sciences THIM, Landquart, Switzerland, [3] Department of Health, Bern University of Applied Sciences, Berne, Switzerland, [4] Department of Movement and Sport Sciences, Vrije Universiteit Brussel, Brussels, Belgium, [5] Department of Neurosciences and Movement Sciences, University of Fribourg, Fribourg, Switzerland, [6] School of Sport, Health and Exercise Science, University of Portsmouth, Portsmouth, United Kingdom

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Mohammed Ihsan, National University of Singapore, Singapore

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Hui Cheng Choo, Singapore Sports Institute (SSI), Singapore
Jonathan Peake, Queensland University of Technology, Australia
Angus Lindsay, Deakin University, Australia

***Correspondence:**
Erich Hohenauer
erich.hohenauer@supsi.ch

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Post-exercise cold-water immersion (CWI) is a widely accepted recovery strategy for maintaining physical performance output. However, existing review articles about the effects of CWI commonly pool data from very heterogenous study designs and thus, do rarely differentiate between different muscles, different CWI-protocols (duration, temperature, etc.), different forms of activating the muscles before CWI, and different thickness of the subcutaneous adipose tissue. This systematic review therefore aimed to investigate the effects of one particular post-exercise CWI protocol (10°C for 10 min) on intramuscular temperature changes in the quadriceps femoris muscle while accounting for skinfold thickness. An electronic search was conducted on PubMed, LIVIVO, Cochrane Library, and PEDro databases. Pooled data on intramuscular temperature changes were plotted with respect to intramuscular depth to visualize the influence of skinfold thickness. Spearman's rho (r_S) was used to assess a possible linear association between skinfold thickness and intramuscular temperature changes. A meta-analysis was performed to investigate the effect of CWI on pre-post intramuscular temperature for each measurement depth. A total of six articles met the inclusion criteria. Maximum intramuscular temperature reduction was 6.40°C with skinfold thickness of 6.50 mm at a depth of 1 cm, 4.50°C with skinfold thickness of 11.00 mm at a depth of 2 cm, and only 1.61°C with skinfold thickness of 10.79 mm at a depth of 3 cm. However, no significant correlations between skinfold thickness and intramuscular temperature reductions were observed at a depth of 1 cm ($r_S = 0.0$), at 2 cm ($r_S = -0.8$) and at 3 cm ($r_S = -0.5$; all $p > 0.05$). The CWI protocol resulted in significant temperature reductions in the muscle tissue layers at 1 cm ($d = -1.92$ [95% CI: −3.01 to −0.83] and 2 cm ($d = -1.63$ [95% CI: −2.20 to −1.06]) but not at 3 cm ($p < 0.05$). Skinfold thickness and thus, subcutaneous adipose tissue, seems to influence temperature reductions in the muscle

tissue only to a small degree. These findings might be useful for practitioners as they demonstrate different intramuscular temperature reductions after a specific post-exercise CWI protocol (10°C for 10 min) in the quadriceps femoris muscle.

Keywords: cold-water immersion, adipose tissue, intramuscular temperature, exercise, metabolism

INTRODUCTION

Cold-water immersion (CWI) is one of the most common modalities for athletic muscle recovery (Bleakley et al., 2012). Post-exercise CWI is reported to exert a positive effect on neuromuscular performance and subjective recovery (Higgins et al., 2017). Exercising and assessment of leg muscles are commonly conducted in the area of post-exercise cooling studies, which clearly demonstrate a high relevance of optimal recovery strategies, especially for the knee extensor muscles (Bleakley et al., 2012; Costello et al., 2015). High-intensity or unaccustomed exercise can induce delayed onset of muscle soreness (DOMS), which has been investigated by several research groups over the last decade (Adamczyk et al., 2016; Fonseca et al., 2016; Hohenauer et al., 2018; Siqueira et al., 2018; de Freitas et al., 2019). CWI is reported to attenuate DOMS to a significant extent, reducing the symptoms of DOMS (up to 96 h) compared to passive control interventions (Hohenauer et al., 2015). In addition to subjective recovery variables, objective outcomes also indicate positive effects of CWI such as reduced inflammation (Leeder et al., 2012).

Reduction of intramuscular temperature and its interaction with metabolism has attracted significant research interest. It has been shown that CWI (8°C, 10 min) induces significant decreases in intramuscular temperature (Gregson et al., 2013). Lower intramuscular temperatures are speculated to affect enzymatic activity and rates of intramuscular glycogen synthesis but are also associated with attenuated training adaptions following strength training (Roberts et al., 2015a; Mawhinney and Allan, 2018). However, in cryotherapy research, the most relevant and divisive question pertains to the optimal cooling modality, temperature, and duration to elicit the required physiological response (Costello et al., 2012a). For example, to induce a significant analgesic effect, skin temperature needs to be <13°C for stimulation of neuronal changes, as consistently demonstrated by different research groups (e.g., Bleakley and Hopkins, 2010; Costello et al., 2012a). Currently, one of the most often applied post-exercise CWI protocols comprises a water temperature of around 10°C for a duration of around 10 min (Hohenauer et al., 2015; Vromans et al., 2019). However, the magnitude of heat extraction has been shown to be affected by the subcutaneous adipose tissue thickness, as well as environmental, hormonal, temporal, and nutritional factors and varies between different body parts (Jutte et al., 2001; Costello et al., 2012b; Garami and Székely, 2014; Adams et al., 2015; Romanovsky, 2018; Baker et al., 2020). Despite the increasing number of published articles on CWI and its effectiveness in athletes' recovery, limited studies have focused on the impact of post-exercise CWI on one specific muscle (group) and taking the magnitude of the subcutaneous adipose tissue into account. Previous conducted reviews included various articles with different CWI-protocols (temperature and duration), exercise protocols before CWI, investigated muscles and varying subcutaneous adipose tissue profiles of the participants (Leeder et al., 2012; Hohenauer et al., 2015). The current review article aimed to investigate the effects of one particular post-exercise CWI (i.e., 10 ± 2°C for 10 ± 2 min) on intramuscular temperature changes on one selected muscle (i.e., quadriceps femoris muscle) by taking the skinfold thickness into account.

The collective results may help to improve the estimation of the degree of intramuscular temperature reductions induced by post-exercise CWI on this specific muscle group in various depths. The results of the current study might help health practictioners, coaches, and athletes to estimate the effects of this popular post-exercise CWI protocol on intramuscular temperature changes with respect to the subcutaneous adipose tissue.

TABLE 1 | Keywords and Boolean logic combinations.

Databases	Keywords and Boolean logic combinations
PEDro	cold water immersion and temperature
PubMed, LIVIVO cochrane library	(cold water immersion OR CWI OR cold water immersion therapy OR CWIT OR ice water immersion OR ice baths OR cold water therapy OR cryotherapy OR ice application OR cooling water OR cold treatment) AND [(intramuscle temperature) OR (intramuscular temperature) OR (skeletal temperature) OR (muscle temperature)]
Google scholar	"cold water immersion" AND "intramuscular temperature" AND adipose thickness

METHODS

Literature Search Strategies and Data Sources

A computerized literature search of online databases was undertaken by one researcher (LF) up to June 2020. The databases searched included PubMed with MeSH Terms (Medical Subject Headings), LIVIVO, Cochrane Library, PEDro, and Google Scholar. The literature search was performed following an *a priori* search strategy using the keywords and combinations presented in **Table 1**.

Selection Criteria

Selection criteria were as follows: (1) a defined CWI treatment (10 ± 2°C for 10 ± 2 min) with a minimum immersion depth of the legs (i.e., up to the iliac crest), (2) assessment of intramuscular temperature in the quadriceps femoris muscle before exercise and after the post-exercise CWI intervention at a depth of 1 cm and/or 2 cm and/or 3 cm, (3) exercise protocol of any type before CWI, (4) all participants were healthy, (5) only experimental studies were included, (6) no sex-defined inclusion or exclusion criteria, (7) English and German language restrictions, and (8) the studies measured skinfold thickness of the exercised muscle.

Studies were excluded in case they (1) used cooling techniques other than CWI, (2) combined CWI with any other intervention post-exercise, (3) did not involve human participants, (4) did not use the pre-defined CWI protocol. **Figure 1** depicts the systematic search strategy and selection process.

Data Extraction

General data on the CWI interventions (protocol: °C and min), environmental conditions (°C, % relative humidity), exercise protocol, muscle temperature before and after CWI (including intramuscular temperature at a specific depth), and skinfold thickness (mm) of individual studies were extracted independently by two researchers (LF, EH). In cases where data were missing for the calculations, the authors were contacted by email and, where possible, data were directly extracted from the graphs.

Risk of Bias

Systematic errors were assessed using the Cochrane's risk of bias tool for both randomized and non-randomized studies (Sterne et al., 2016; Higgins et al., 2019). This strategy has the advantage that risk of bias for different components (domains) is determined separately. Thus, two researchers (LF, EH) independently scored each trial for risk of bias. In case of disagreement, a third researcher (CD) rated the questionable item and agreement was sought by consensus.

Specific domains (random sequence generation, allocation concealment, blinding participants, blinding personnel, blinding outcome assessors, incomplete outcome data, selective reporting, and other bias) were graded for each study. Three options were available for evaluation: low (+) or high (–) risk of bias or "unclear" (?) rating in cases where insufficient information or deficient evidence for bias evaluation was given.

For each non-randomized study, seven domains were rated, including bias due to confounding, selection of participants into the study (pre-intervention), bias in classification of interventions (at intervention) and due to deviations from intended interventions, missing data, measurement of outcomes, and selection of the reported results (post-intervention). Grading options included low risk (+ +), moderate risk (+), serious risk (–), critical risk of bias (– –) and no information (?).

Data Analysis

Studies were included, if they met all inclusion criteria. Conformity was established in cases where the water temperature and immersion duration did not differ from 10°C and 10 min by more than 2°C and 2 min, respectively, to minimize the impact of protocol variations on intramuscular temperature changes. Means and standard deviations (mean value ± SD) are presented in this study where appropriate. To determine the relationship between subcutaneous adipose tissue and reductions in intramuscular temperature at a specific depth, graphs were plotted with skinfold thickness on the X-axis and changes in intramuscular temperature on the Y-axis. The outermost points were marked and connected to show the extent of the possible effects per depth. The absolute baseline and post-CWI values were applied to estimate mean temperature changes within the quadriceps femoris muscle at a specific muscle depth. Spearman's rho (r_s) was used to evaluate possible correlations between skinfold thickness and intramuscular temperature change with the level of significance set to $p < 0.05$. The analyses were performed in SPSS (Statistical Package for the Social Sciences), version 26.0 (SPSS Inc, Chicago, IL, USA). Because control groups were very heterogeneous (e.g., "active recovery," "passive rest," "whole body cryotherapy") it was decided to meta-analyse the pre-post intamuscular temperature data of the intervention arms of the different studies only. This decision enabled the inclusion of the single-group Rech (2013) study. Meta-analyses with random-effects model were used to examine the overall weighted mean effect size of post-exercise CWI on intramuscular temperature for each measurement depth (1, 2, 3 cm). Inverse-variance method was used to calculate the weighting factors. Meta-Analyses of the pre-post intramuscular temperature data were calculated assuming a correlation coefficient of 0.7 (Borenstein et al., 2009). To assess the robustness of the overall weighted estimate, sensitivity analyses with correlation coefficients of 0.5 and 0.9, respectively, were conducted (Borenstein et al., 2009). The Cochran Q statistic and its corresponding p-value, as well as I^2 were calculated to assess across studies' heterogeneity and its degree, respectively. Higgins suggested benchmarking values for the interpretation of I^2 as followed: I^2 around 25% (low heterogeneity), I^2 around 50% (moderate heterogeneity) and I^2 around 75%, or more (high heterogeneity) (Higgins and Thompson, 2002). The Comprehensive Meta-Analysis 2 software (CMA- Version 2 Professional, Biostat Inc., Englewood, USA) was used for the calculations of the weighted overall effect size, the corresponding 95% confidence intervals (95% CI), the sensitivity analyses and to establish the forest plots.

RESULTS

Risk of Bias rating

The Cochrane's risk of bias tool for randomized studies was used in five studies (Mawhinney et al., 2013, 2017a,b; Roberts et al., 2014, 2015b). Risk of bias analysis demonstrated high risk of performance bias and unclear risk of detection bias. Low risk of selection, attrition, reporting, and other bias was obtained for all five studies. Comprehensive details of risk of bias for collective and individual studies are presented in **Figures 2**, **3**.

One study was rated with the ROBINS-I tool for assessing risk of bias in non-randomized studies of interventions (Rech, 2013). In this case, low risk of bias was observed for selection

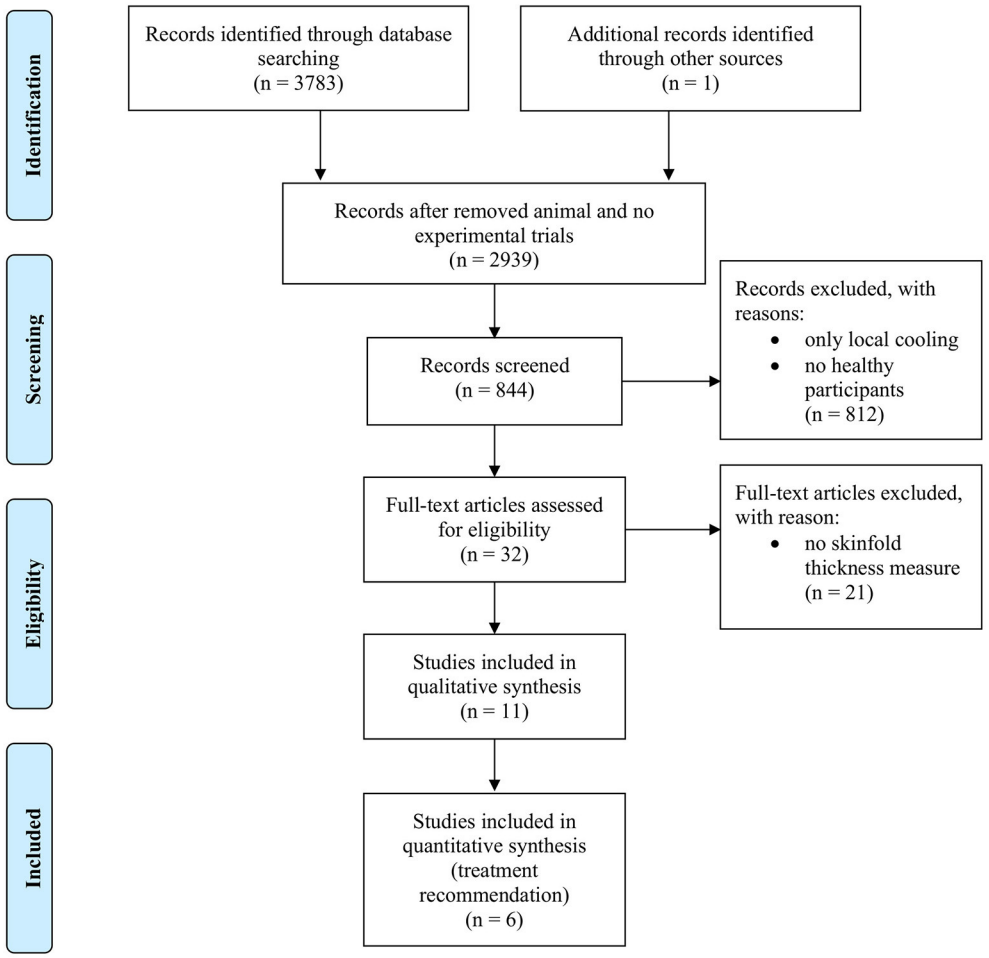

FIGURE 1 | Flowchart describing the systematic search strategy and selection process.

of results and missing data. Moderate risk of bias was reported for confounding selection of participants, classification of intervention, and measurement of outcomes, and serious risk of bias for deviation from intended interventions.

Included Studies

A total of $n = 11$ articles were taken into consideration for the analysis (Gregson et al., 2013; Mawhinney et al., 2013, 2017a,b; Rech, 2013; Broatch et al., 2014; Roberts et al., 2014, 2015b; Solianik et al., 2015; Joo et al., 2016; Choo et al., 2018). However, after screening for the quantitative analysis, only $n = 6$ studies met all of our inclusion criteria and were used for further evaluation (Mawhinney et al., 2013, 2017a,b; Rech, 2013; Roberts et al., 2014, 2015b). The total study population included $n = 70$ healthy volunteers. The majority of selected studies was performed on recreationally or physically active male volunteers. In one study, including $n = 16$ volunteers, sex distribution and fitness status was not specified (Rech, 2013). The mean sample size was $n = 11.66$ (range: 10–16 volunteers) and mean age ($\pm$ SD) of the total study population was 23.5 $\pm$ 3.25 years. The characteristics of the included studies are summarized in **Table 2**.

The remaining five studies were excluded from the quantitative analysis as the CWI protocol did not meet conformity (water temperature at 10 $\pm$ 2°C and treatment duration 10 $\pm$ 2 min) or data reporting was incomplete. Three studies used CWI protocols ranging between 8 and 14°C for 5 min (Choo et al., 2018) and 10°C for 15 min (Broatch et al., 2014) or 14°C for 20 min (Solianik et al., 2015). Furthermore, we were unable to extract all necessary data from two studies that employed a CWI protocol using cold water at 8°C for the duration of 10 min and 2 × 5 min (Gregson et al., 2013; Joo et al., 2016).

The reasons for exclusion were, that variations of the CWI protocols (temperature and duration) would have led to potential under- or overestimations of the impact of the CWI protocol on muscle cooling, whilst incomplete data reporting of the skinfold thickness would have made it not possible to evaluate the influence of the subcutaneous adipose thickness on the intramuscular cooling rate.

Environmental Conditions

Environmental temperature and relative humidity were between 22 and 24.4°C (mean 23.54°C) and between 40 and 48.6% (mean 44.0%) in the included studies. One study did not provide information on the environmental conditions (Rech, 2013).

Exercise Program

Participants had to perform a submaximal cycling endurance protocol in three studies (Mawhinney et al., 2013, 2017b; Rech, 2013) and maximum or high-intensity exercises (Roberts et al., 2014, 2015b; Mawhinney et al., 2017a) prior to the CWI intervention. Two studies used a cycling protocol at 70% VO_{2max} until the core temperature reached 38°C (Mawhinney et al., 2013, 2017b). In one study, participants were required to cycle for 30 min and heart rates between 130 and 150 bpm were recorded (Rech, 2013). Maximum or high-intensity exercises were carried out as follows: 4 × 10 maximal squat exercises with a 2 min break between sets (Mawhinney et al., 2017a), 10 × 20 maximal isokinetic knee extension exercises with a 2 min rest (Roberts et al., 2015b) and high-intensity resistance training lasting for around 1 h (Roberts et al., 2014).

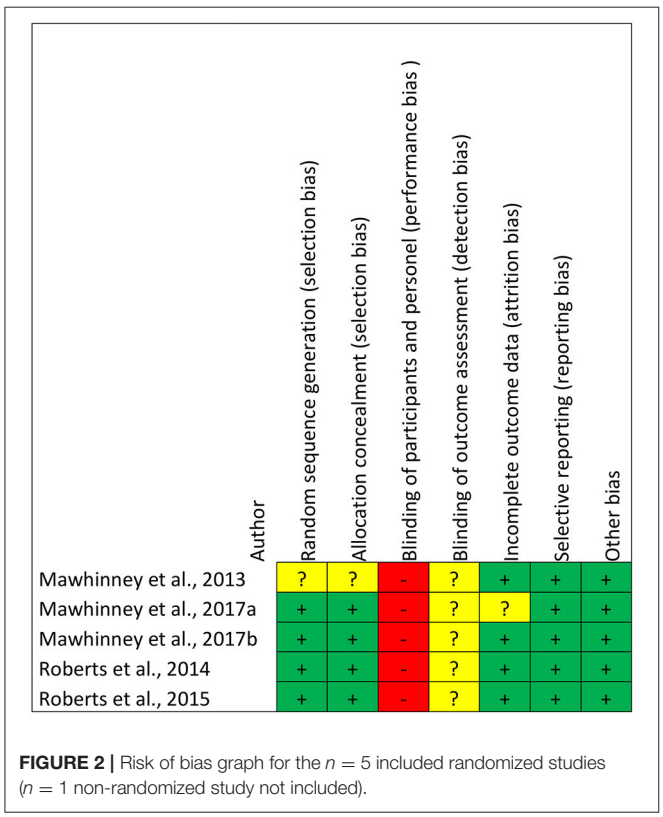

FIGURE 2 | Risk of bias graph for the $n = 5$ included randomized studies ($n = 1$ non-randomized study not included).

Skinfold Thickness

The mean skinfold thickness for all six studies was 10.29 ± 3.86 mm, ranging between 6.40 and 15.65 mm. One study used an ultrasound device to determine the subcutaneous adipose tissue above the assessed muscle (Rech, 2013). The remaining five studies used a Harpenden skinfold caliper and divided the result by two to determine the subcutaneous adipose tissue thickness (Mawhinney et al., 2013, 2017a,b; Roberts et al., 2014, 2015b). Skinfold thickness of the thigh was measured 5 cm proximal from the patella (Roberts et al., 2015b), 15 cm proximal from the superior margin of the patella (Mawhinney et al., 2013, 2017a), mid-way between the inguinal crease and the patella (Rech, 2013; Roberts et al., 2014) and was not described in one study (Mawhinney et al., 2017a).

Intramuscular Temperature Measurement

Five studies investigated the intramuscular temperature of the vastus lateralis of the quadriceps femoris muscle (Mawhinney et al., 2013, 2017a,b; Roberts et al., 2014, 2015b) while one evaluated the temperature in the rectus femoris (Rech, 2013).

Intramuscular temperature was assessed using an implantable probe (Roberts et al., 2015b), fine wire thermistor or needle thermistor (Mawhinney et al., 2013, 2017a,b; Roberts et al., 2014) or a thermocouple probe (Rech, 2013). Two studies assessed intramuscular temperature at a frequency of 1 Hz (Roberts

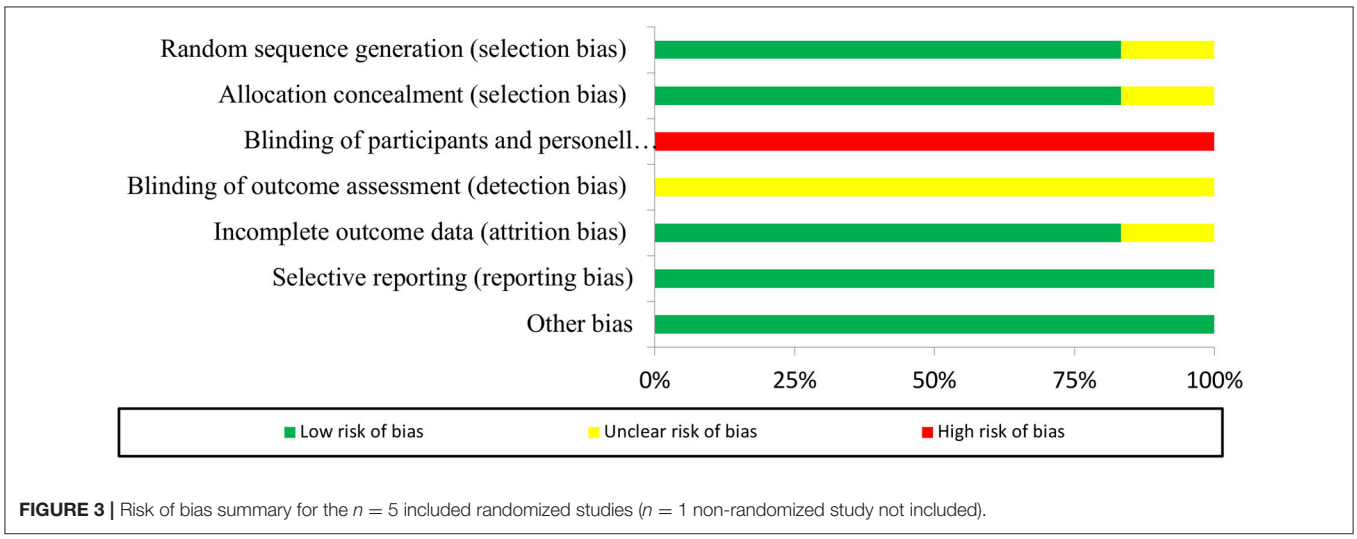

FIGURE 3 | Risk of bias summary for the $n = 5$ included randomized studies ($n = 1$ non-randomized study not included).

TABLE 2 | Summary of included studies.

References	Sample size (*n*), sex, and age	EC	CWI protocol: water temperature (°C) and duration (min)	Exercise protocol	Intramuscular measurement	SFT in mm (mean ± SD)	Intramuscular temperature in °C baseline (mean ± SD)	Intramuscular temperature in °C post-treatment (mean ± SD)
Mawhinney et al. (2013)	*n* = 12 male 25.5 ± 4.7 yr	22–24°C	8°C for 10 min (up to the iliac crest)	Cycling at 70% VO_{2max} until Tcore reaches 38°C	MQF VL, 3.0 cm (plus 1/2 of SF), 2.0 and 1.0 cm depth	11.42 ± 2.65	3.0 cm = 36.27 ± 0.37 2.0 cm = 35.85 ± 0.43 1.0 cm = 34.98 ± 0.66	3.0 cm = 36.58 ± 0.64 2.0 cm = 34.49 ± 1.09 1.0 cm = 31.10 ± 1.68
Mawhinney et al. (2017a)	*n* = 12 male 26 ± 6 yr	22–24°C	8°C for 10 min. (up to the iliac crest)	4 × 10 maximum squat exercise with 2 min. rest	MQF VL, 3.0 cm (plus 1/2 of SF), 2.0 and 1.0 cm depth	10.79 ± 2.73	3.0 cm = 36.19 ± 0.32, 2.0 cm = 35.69 ± 0.45, 1.0 cm = 34.92 ± 0.59	3.0 cm = 34.58 ± 0.9 2.0 cm = 32.04 ± 1.38 1.0 cm= 29.48 ± 1.47
Mawhinney et al. (2017b)	*n* = 10 male 22.3 ± 3.4 yr	22–24°C ∼40% rh	8°C for 10 min. (up to iliac crest)	Cycling at 70% VO_{2max} until Tcore reaches 38°C	MQF VL, 3.0 cm (plus 1/2 of SF), 2.0 and 1.0 cm depth	15.65 ± 7.58	3.0 cm = 36.14 ± 0.66 2.0 cm = 35.53 ± 0.81 1.0 cm = 34.80 ± 0.90	3.0 cm = 35.57 ± 0.49 2.0 cm = 33.09 ± 1.36 1.0 cm = 30.94 ± 1.51
Rech (2013)	*n* = 16 subjects 24.3 ± 1.84 yr	unknown	10°C (up to iliac crest) until Tm decreased 7°C below pre-exercise (= cooling rate 0.27 ± 0.18°C/min) or 30 min	Cycling for 30 min. (HR130-150 bts/min.)	MQF rectus femoris, 2.0 cm sub-adipose	11.0 ± 3.7	35.3 ± 1.2	30.8 ± 3.7
Roberts et al. (2014)	*n* = 10 male 21.3 ± 1.6 yr	24.3 ± 0.6°C 48.6 ± 1.2% rh	10.0 ± 0.3°C for 10 min. (up to the clavicle)	High intensity resistance training for 1 h: 6 sets of 8, 8, 10, 12, 10, and 10 front and back squats, 3 × 12 walking dumbbell lunges, 3 × 12 countermovement drop jumps	MQF VL, 1.0 cm depth	6.4 ± 3.1	35.2 ± 0.5	32.99 ± 4.64
Roberts et al. (2015a)	*n* = 10 male 21.4 ± 2.0 yr	24.4 ± 0.2°C 43.5 ± 1.6% rh	10.0 ± 0.2°C for 10 min. (up to umbilicus)	Maximal unilateral isokinetic knee extensor exercise 10 × 20 reps with 2 min. rest	MQF VL, 1.15 cm depth	6.5 ± 3.4	34.5 ± 0.9	28.1 ± 6.1

EC, Environmental conditionsl; HR, heart rate; MQF, M. quadriceps femoris; rh, relative humidity; SF, skinfold; SFT, skinfold thickness; Tcore, core temperature; Tm, Temperature; VL, vastus lateralis; yr, year.

et al., 2014, 2015b). In one investigation, data acquisition was conducted at ∼0.03339 Hz (Rech, 2013) while the exact measurement frequency was unclear in the remaining three studies (Mawhinney et al., 2013, 2017a,b).

Differences in Intramuscular Temperature

The intramuscular temperature changes are presented in **Figure 4**.

Intramuscular Temperature Differences at a Muscle Depth of 1 cm

Five studies, with a total sample size of $n = 54$ participants, investigated the effects of CWI on intramuscular temperature at a depth of 1 cm in the vastus lateralis of the quadriceps femoris muscle (Mawhinney et al., 2013, 2017a,b; Roberts et al., 2014, 2015b). The mean water temperature was 8.8 ± 1.0°C for a mean duration of 10 ± 0 min. We observed an intramuscular temperature decrease of 4.36 ± 1.61°C (range: −2.21°C to −6.40°C) at a mean skinfold thickness of 10.15 ± 3.86 mm (range: 6.40–15.65 mm). No correlation ($p = 1.0$) between skinfold thickness and intramuscular temperature reduction was observed ($r_s = 0.0$). The use of CWI decreased intramuscular temperature significantly (**Figure 5**) between baseline and post-exercise CWI measurements (standardized differences in means (d) = −1.92 [95% CI: −3.01 to −0.83]), based on this limited set of published studies. High and statistically significant heterogeneity was observed (Q = 41.86, df (Q): 4, $p = 0.001$; I^2: 90.4%). After conducting sensitivity analyses using correlation coefficients of 0.5 and 0.9, the results remained statistically significant and in favor of for reduced intramuscular temperature after CWI $d = -2.12$ [95% CI: −3.32 to −0.92] and $d = -1.37$ [95% CI: −2.20 to −0.55], respectively.

Intramuscular Temperature Differences at a Muscle Depth of 2 cm

Four studies, with a total sample size of $n = 50$ participants, evaluated the effects of CWI on intramuscular temperature at a depth of 2 cm in the vastus lateralis of the quadriceps femoris muscle (Mawhinney et al., 2013, 2017a,b) and rectus femoris (Rech, 2013). The mean water temperature was 8.5 ± 1.0°C for a mean duration of 10.0 ± 0.0 min. The intramuscular temperature decrease was 2.98 ± 1.37°C (range: −1.36 to −4.50°C) at a mean skinfold thickness of 12.21 ± 2.30 mm (range: 10.79–15.65 mm). No correlation ($p = 0.2$) between skinfold thickness and intramuscular temperature reduction was observed ($r_s = -0.8$). Based on this limited set of published studies, post-exercise CWI reduced intramuscular temperature significantly, which can be seen in **Figure 6** ($d = -1.63$ [95% CI: −2.20 to −1.06]). Moderate and statistical significant heterogeneity was observed [$Q = 8.66$, df (Q): 3, $p = 0.034$, I^2: 65.3%]. After the sensitivity analysis with a correlation coefficient of 0.5 and 0.9 results remainded statistically significant in favor for decreased muscle temperature after CWI with $d = -1.79$ [95% CI: −2.41 to −1.17] and $d = -1.16$ [95% CI: −1.60 to −0.73], respectively.

Intramuscular Temperature Differences at a Muscle Depth of 3 cm

Three studies, with a total sample size of $n = 34$ participants, investigated the effects of CWI on intramuscular temperature at a depth of 3 cm in the vastus lateralis of the quadriceps femoris muscle (Mawhinney et al., 2013, 2017a,b). The mean water temperature was 8.0 ± 0.0°C for a mean duration of 10.0 ± 0.0 min. The mean intramuscular temperature decrease was 0.62 ± 0.96°C (range: −1.61 to + 0.31°C) at a skinfold thickness of 12.62 ± 2.64 mm (range: 10.79–15.65 mm). No correlation ($p = 0.6$) between skinfold thickness and intramuscular temperature reduction was observed ($r_s = -0.5$). Based on this limited set of published studies, post-exercise CWI reduced intramuscular temperature albeit not statistically significant (**Figure 7**) with $d = -0.70$ [95% CI: −2.04 to 0.63]. High and statistically significant heterogeneity was observed ($Q = 32.54$, df (Q): 2, $p < 0.001$, I^2: 93.9%). After the sensitivity analysis with a correlation coefficient of 0.5 and 0.9 results remainded in favor for decreased muscle temperature after CWI albeit statistically not significant with $d = -0.77$ [95% CI: −2.25 to 0.7] and $d = -0.52$ [95% CI: −1.50 to 0.46], respectively.

DISCUSSION

The main aim of this systematic review is to provide an overview on intramuscular temperature changes at different depths in the quadriceps femoris muscle for a well-established post-exercise CWI protocol (10°C for 10 min), taking subcutaneous adipose tissue into account. Our results demonstrate that CWI reduces intramuscular temperature after exercise significantly in the upper muscle layers by around 4°C at 1 cm ($p = 0.001$) and by 3°C at 2 cm ($p < 0.001$) but not at a depth of 3 cm (1°C, $p = 0.304$). Reduction in muscle tissue temperature was not significantly correlated to subcutaneous thickness measurements. Furthermore, potential interindividual changes in muscle tissue temperature at all measurement depths (i.e., 1, 2, and 3 cm) can be seen in **Figure 4** and are also reflected in the **Figures 5**–**7**. In general, we saw most pronounced temperature drops after CWI at superficial muscle depth (1 cm) that were progressively alleviated in deeper layers (2 and 3 cm). At 3 cm, muscle temperature was often close to baseline values. In one study, the temperature was even above the baseline value after exercises (Mawhinney et al., 2013). This result is not surprising taking into account that heat loss differs between superficial and deep tissues due to different temperature gradients. Skin temperature can be easily and rapidly decreased due to direct contact between the tissue and the cooling modality. The relatively large temperature differences between skin tissue and cooling modality can lead to a considerable drop in skin temperature. In muscle tissue, heat loss takes place indirectly via conduction to overlying cooler tissues. The temperature gradient allows transfer of heat from deep to superficial tissues, but the magnitude of heat loss is obviously smaller in tissues at greater depth (Merrick et al., 2003). Consequently, with greater heat produced during exercise in deep muscle tissues, more cooling time is required to decrease the intramuscular temperature below baseline values. Indeed,

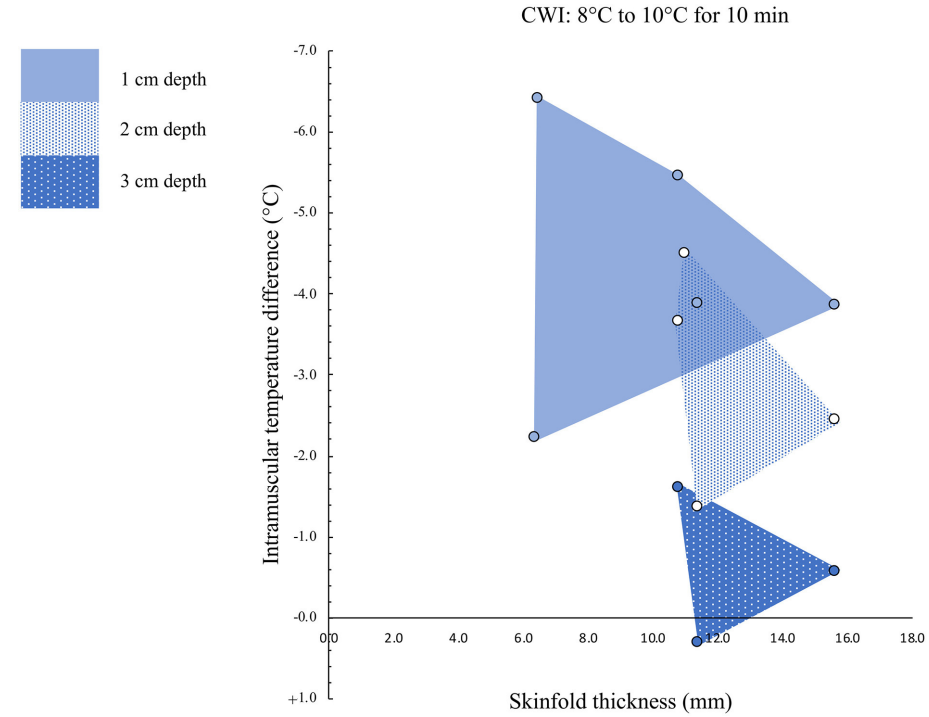

FIGURE 4 | Intramuscular temperature changes in relation to the skinfold thickness. Muscle temperature was measured at depths of 1, 2, and 3 cm.

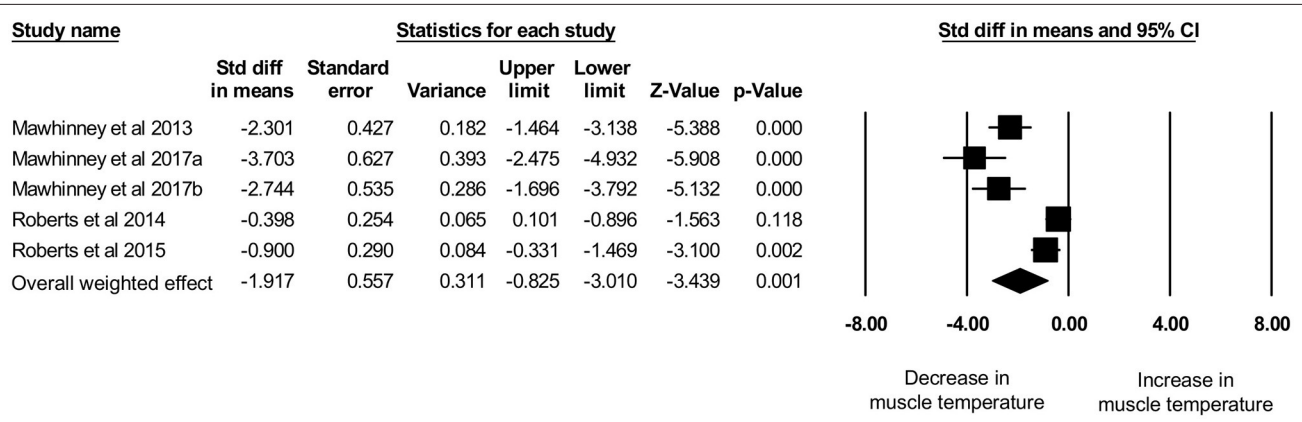

FIGURE 5 | Forest plot of the meta-analysis illustrating the comparison between baseline and post-exercise CWI temperature values at a muscle depth of 1 cm.

the cooling time might have been too short in the study of Mawhinney et al. (2013), where the intramuscular temperature was even after the CWI treatment 0.31°C higher compared to baseline at a depth of 3 cm. In the remaining $n = 2$ studies, the cooling time was sufficient to decrease the muscle tissue temperature at a depth of 3 cm by 1.61 and 0.56°C (Mawhinney et al., 2017a,b). However, it has to be mentioned that the post-CWI data showing only a 0.57°C reduction was collected 10 min after the cold treatment (Mawhinney et al., 2017b). Although the afterdrop has to be taken into account, it is possible that maximal intramuscular temperature reduction had already occurred at an earlier time-point in this study, which would result in underestimation of the magnitude of muscle temperature decrease in this case.

The subcutaneous fat layer, which varied in our included studies from 6.40 mm (Roberts et al., 2014) to 15.65 mm (Mawhinney et al., 2017b) has been assumed to play a large role to limit muscle temperature reductions (Jutte et al., 2001; Otte et al., 2002). Although skinfold thickness of the included recreationally active males in the studies was measured, different body composition (ecto-, meso und endomorphy) might be an additional confounding factor (Stephens et al., 2018). Compared

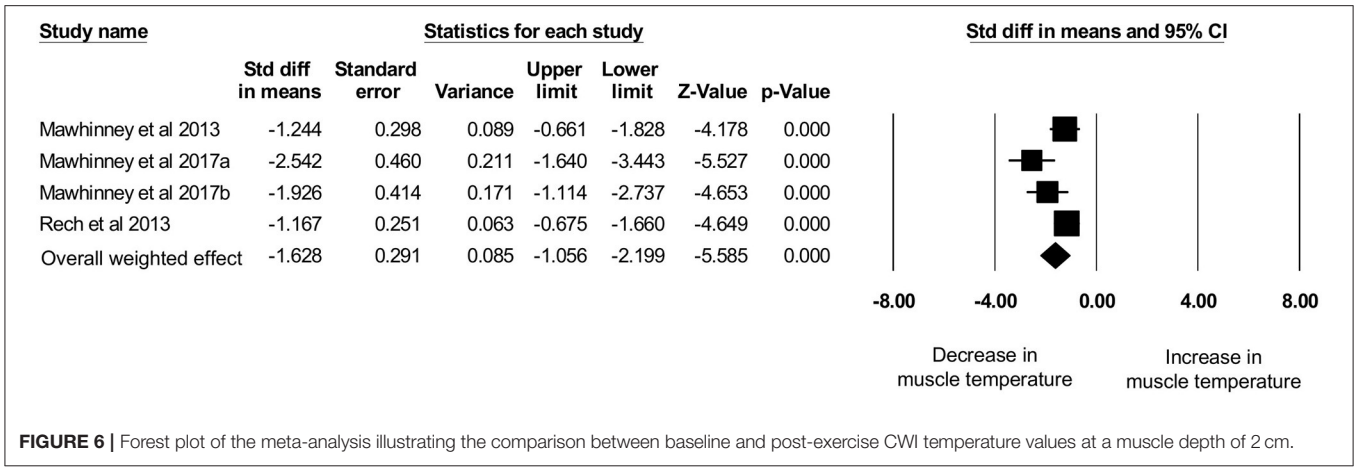

FIGURE 6 | Forest plot of the meta-analysis illustrating the comparison between baseline and post-exercise CWI temperature values at a muscle depth of 2 cm.

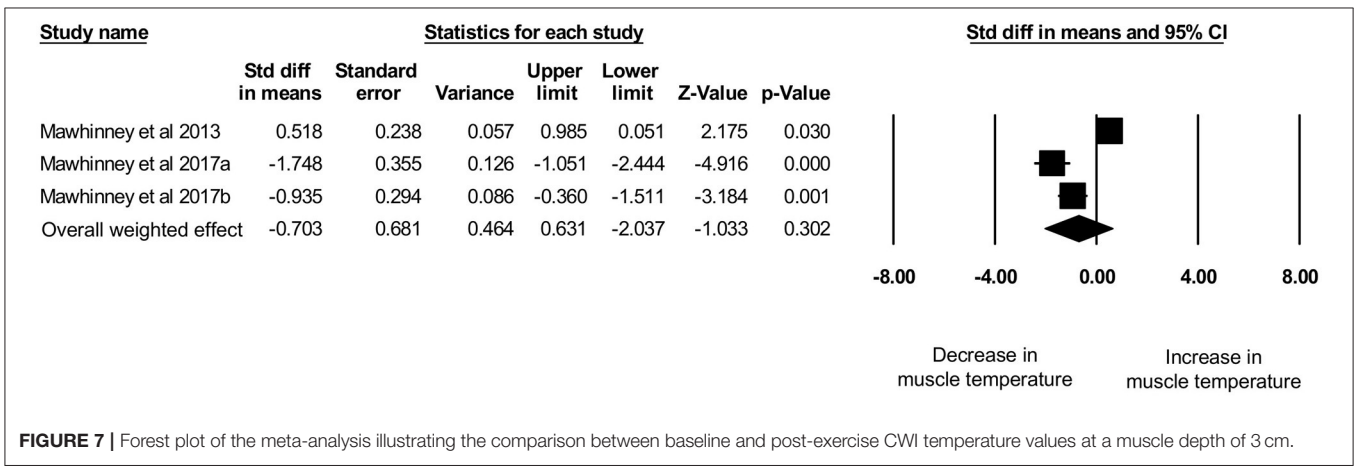

FIGURE 7 | Forest plot of the meta-analysis illustrating the comparison between baseline and post-exercise CWI temperature values at a muscle depth of 3 cm.

to muscle tissue, adipose tissue has low thermal conductivity (0.23 vs. 0.46 k) and thus, serves as a physiological heat insulator (El-Brawany et al., 2009). From this perspective, an inverse relationship between subcutaneous adipose tissue thickness and intramuscular temperature decrease was expected. However, our results suggest that lower skinfold thickness values do not guarantee higher reductions in intramuscular temperature after cooling, indicating that intraindividual factors such as microvascular blood flow or intramuscular perfusion must be additionally considered (Mawhinney et al., 2013). Indeed, it has been shown that variations in skin microvasculature and muscle perfusion are known to be normally present (Mayrovitz et al., 1997; Mawhinney et al., 2020). Additionally, it has been demonstrated, that in cold adapted humans, cold exposure is subjectively considered less stressful and that the physiological responses to cold are attenuated (Rintamaki, 2007). Consequently, the included studies in the current review were carried out in different countries and the environmental conditions (season when the experiments were carried out) might have had a significant impact on the physiological responses to the CWI. Also the type of exercise and muscle contraction, potentially influence the magnitude of heat extraction from muscle tissue. In our review, three of the six included studies focused on performance of a high-intensity exercise task (Roberts et al., 2014, 2015b; Mawhinney et al., 2017a) and submaximal cycle ergometer tests prior to CWI (Mawhinney et al., 2013, 2017a; Rech, 2013). Different mechanical load/resistance programs might have caused active muscle to produce more or less energy in the form of work and heat, derived from chemical reactions (Yamada, 2017). Further research and analysis with individual customized exercise protocols is necessary to evaluate the potential relationship between the type of exercise task and intramuscular temperature development.

Our results revealed that a CWI protocol (10 ± 2°C, 10 ± 2 min) maximally lowered the intramuscular temperature of the quadriceps femoris muscle to 6.40°C at a depth of around 1 cm, 3.65°C at around 2 cm, and 1.61°C at around 3 cm. From a rehabilitation perspective, the impacts of even these maximum temperature reductions may be too small to decrease cellular metabolism to a clinically significant extent for protection of damaged muscle tissue from secondary ischemic and enzymatic injuries (Merrick, 2002; Bleakley and Hopkins, 2010). Research on animal models has demonstrated that cellular metabolism is optimally reduced when tissue temperatures between 5 and 15°C are reached (Osterman et al., 1984; Merrick, 2002). In our review, the minimum intramuscular

temperatures post-exercise CWI were around 28°C at a depth of 1 cm, 30°C at 2 cm and 32°C at 3 cm (Rech, 2013; Roberts et al., 2015b; Mawhinney et al., 2017a). Although the ability of water to extract heat from the body is extremely high (24 times higher than air), other materials should be considered when the primary aim is heat extraction from muscle tissue. For example, ice possesses a four times higher heat transfer coefficient than water, which is related to phase changes during the melting process (Bleakley et al., 2014). However, the potential positive effects of cold water on cellular metabolism reductions and further of enhanced muscle recovery after exercise (e.g., DOMS) may not be primarily attributed to its intramuscular temperature reduction effect (Wilcock et al., 2006). Indeed, the impact of the cold water on muscle strength was shown to be no greater than thermoneutral placebo water immersion (34.7°C) after a high-intensity interval training session (Broatch et al., 2014). Hydrostatic pressure leading to intracellular-intravascular fluid shifts, reduction of edema and increased cardiac output may also play an important role in enhancing muscle recovery. Immersion in cool and thermoneutral water could provide comparable recovery results unless soft tissue injuries have occurred, in which case cooled tissue may provide greater benefits (Sellwood et al., 2007; Mutlu and Yilmaz, 2020).

Another variable that may have a positive effect on post-exercise recovery is immersion depth of the body, although this variable was not investigated in the current study. The hydrostatic pressure, which varies with immersion depth, causes displacement of body fluids from the extremities to the central cavity. As a result of exercise or muscle damage, oxygen delivery by these fluids is reduced to localized cells, leading to increased cellular damage or death (Friden and Lieber, 2001). Post-exercise water immersion of the body (not exclusively cold water) may reduce the occurrence of potential cell-damaging edema and inflammation by increasing the pressure gradient between the interstitial and intravascular space and promoting re-absorption of interstitial fluid, similar to compression stockings (Partsch et al., 2004). A combination of cold water and high hydrostatic pressure (e.g., through immersing the body up to the clavicle) could act synergistically, as decreased muscle temperature may reduce edema formation through suppression of muscle perfusion and fluid diffusion into the interstitial space (Yanagisawa and Fukubayashi, 2010; Mawhinney et al., 2017b; Tipton et al., 2017). Lower intramuscular temperatures can also lead to reduced inflammatory markers like creatine-kinase after exercise induced muscle damage and therefore might attribute to muscle function recovery (Eston and Peters, 1999). Additionally, after exercise induced muscle damage, maximum voluntary isometric contraction has been shown to recover faster after CWI compared to a control group (Machado et al., 2017). However, as seen in our results, it is questionable if significant temperature reductions can be achieved in deep muscle tissue with this protocol and as a result, if inflammatory responses can be reduced in these deep tissue layers. However, we have to consider, that also non-significant differences might make the difference between winning and loosing in a high athletic population.

Although one of the most popular post-exercise CWI protocol was used for evaluation in this study, only a small number of studies could be identified for the current analysis. Taking this into consideration, the analyses and results which are based on a limited set of published studies, should be interpreted with caution as publication bias can't be excluded. The results are also limited to this investigated CWI protocol and can't be transferred to other (post-exercise) CWI protocols. However, this also highlights the need for further (post-exercise) CWI studies in this field with specific inclusion criteria to further assess the impact of this recovery strategy. Further studies using standardized exercise protocols, identifying further key variables and taking the above mentioned key variables into account are warranted, to evaluate the tissue cooling magnitude of specific CWI protocols on different muscle groups in a well-defined population.

CONCLUSION

In conclusion, the collective findings of this review indicate that post-exercise CWI (10 ± 2°C for 10 ± 2 min) decreases intramuscular temperature in the quadriceps femoris muscle significantly in the upper muscle layers (1, 2 cm). However, the intramuscular temperature reductions in the quadriceps femoris muscle showed a wide variation and the subcutaneous adipose tissue did not significantly correlate to the temperature reduction. Beside skinfold thickness, additional key variables like the intensity and length of the exercise protocols, intramuscular perfusion and the investigated population itself might have a significant influence on the magnitude of intramuscular heat extraction during post-exercise CWI.

DATA AVAILABILITY STATEMENT

The original contributions presented in the study are included in the article/**Supplementary Material**, further inquiries can be directed to the corresponding author/s.

AUTHOR CONTRIBUTIONS

EH, LF, and RC: conceived and designed the study. LF, EH, JT, RC, and CD: analyzed the data. CD, EH, LF, RC, WT, and JT: wrote the paper. All authors: contributed to the article and approved the submitted version.

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SUPPLEMENTARY MATERIAL

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fspor.2021.660092/full#supplementary-material

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REVIEW
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Post-exercise Body Cooling: Skin Blood Flow, Venous Pooling, and Orthostatic Intolerance

Afton D. Seeley[1,2]*, *Gabrielle E. W. Giersch*[1,2] *and Nisha Charkoudian*[1]

[1] *Thermal and Mountain Medicine Division, US Army Research Institute of Environmental Medicine, Natick, MA, United States,* [2] *Oak Ridge Institute of Science and Education, Belcamp, MD, United States*

Athletes and certain occupations (e.g., military, firefighters) must navigate unique heat challenges as they perform physical tasks during prolonged heat stress, at times while wearing protective clothing that hinders heat dissipation. Such environments and activities elicit physiological adjustments that prioritize thermoregulatory skin perfusion at the expense of arterial blood pressure and may result in decreases in cerebral blood flow. High levels of skin blood flow combined with an upright body position augment venous pooling and transcapillary fluid shifts in the lower extremities. Combined with sweat-driven reductions in plasma volume, these cardiovascular alterations result in levels of cardiac output that do not meet requirements for brain blood flow, which can lead to orthostatic intolerance and occasionally syncope. Skin surface cooling countermeasures appear to be a promising means of improving orthostatic tolerance via autonomic mechanisms. Increases in transduction of sympathetic activity into vascular resistance, and an increased baroreflex set-point have been shown to be induced by surface cooling implemented after passive heating and other arterial pressure challenges. Considering the further contribution of exercise thermogenesis to orthostatic intolerance risk, our goal in this review is to provide an overview of post-exercise cooling strategies as they are capable of improving autonomic control of the circulation to optimize orthostatic tolerance. We aim to synthesize both basic and applied physiology knowledge available regarding real-world application of cooling strategies to reduce the likelihood of experiencing symptomatic orthostatic intolerance after exercise in the heat.

Keywords: cold water immersion, vasoconstriction, mean arterial pressure, autonomic, heat

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***Correspondence:**
Afton D. Seeley
afton.d.seeley.ctr@mail.mil

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INTRODUCTION

Occupations such as firefighting and the military often require work levels, clothing and/or ambient temperature exposures that are well-beyond thermoneutral "comfort" levels that most humans would consciously choose. Nonetheless, human physiological thermoregulation is remarkably capable of regulating elevations of core body temperature (T_c) in the face of major challenges to this system. Furthermore, autonomic regulation of blood flow, sweating and other responses during increases in internal temperature are coordinated with other essential processes to maintain normal physiological function even in environmental extremes. The goal of the present paper is to discuss the regulation of body temperature and blood pressure in a specific setting: post-exercise hyperthermia and its relationship with decreases in orthostatic tolerance (OI). We will then discuss

the mechanisms by which post-exercise body cooling may be an effective countermeasure to both protect against heat illness and counteract any tendency for orthostatic intolerance that may occur in the post-exercise state.

PART I. THERMOREGULATION IN THE HEAT

Human physiological thermoregulation is controlled by reflex neural mechanisms, which are complemented by local vascular mechanisms and behavioral responses to changes in internal and/or ambient temperature. In the present discussion, we focus primarily on reflex physiological mechanisms, supplemented by information from other areas as appropriate.

The primary central controller of thermoregulation in humans and other mammals is the preoptic area of the anterior hypothalamus (PO/AH). This region contains temperature sensitive neurons that respond with changes in firing rate to their own (local brain) temperature as well as to inputs they receive from peripheral thermoreceptors (Boulant, 2006). Elevation of body temperatures are sensed by warm-sensitive neurons in the PO/AH, which are activated and elicit reflexive increases in heat dissipation mechanisms. In humans, these are primarily sweating and cutaneous vasodilation. Increases in sympathetic cholinergic activity to eccrine sweat glands results in the production and release of sweat. The evaporation of sweat from the skin absorbs heat, thus lowering skin temperature and increasing the effective thermal gradient for heat transfer from the core to the periphery, and then to the environment.

The skin circulation works in concert with sweating to increase dissipation of heat from the body during increases in body temperature. Skin blood flow in humans is controlled by two branches of the sympathetic nervous system. Sympathetic noradrenergic vasoconstrictor nerves exhibit tonic activity at rest in thermoneutral environments, whereas the sympathetic active vasodilator system is only activated during increases in internal body temperature. During heat exposure, the initial thermoregulatory response in the skin is to withdraw the activity of the vasoconstrictor system. If body temperature continues to increase, the cutaneous vasodilator system is activated (Charkoudian, 2010; Johnson et al., 2011). This latter system is responsible for 80–90% of the large increases in skin blood flow that occur with severe heat stress, that can increase to as much as 60% of cardiac output (Rowell, 1983). Importantly for the present discussion, the skin circulation is very compliant, and contains venus plexuses which augment the amount of volume in the skin when blood flow increases (Rowell, 1983). This is helpful for the purposes of heat exchange and thermoregulation but can result in a decrease in venous return and insufficient cardiac filling – particularly if a person is standing still in a hot environment after exercise (i.e., muscle pump activity has stopped).

PART II. BLOOD PRESSURE REGULATION

The autonomic nervous system in humans has a central role in the regulation of arterial pressure. The sympathetic nervous system controls heart rate, cardiac contractility and peripheral vascular resistance via cardiac and vascular innervation, respectively. Post-ganglionic sympathetic nerves innervating the heart release primarily norepinephrine, which interacts with beta-adrenergic receptors at the pacemaker cells (sinoatrial (SA) and atrioventricular (AV) nodes) and across the myocardium to increase heart rate and contractility.

In terms of human cardiovascular function, the parasympathetic nervous system is primarily limited to vagal control of heart rate. The vagus nerve releases acetylcholine at the SA and AV nodes, decreasing heart rate via a decrease in the slope of the pacemaker potential in these cells. With the onset of exercise and with assumption of upright posture, the first mechanism to increase heart rate is a withdrawal of parasympathetic activity, followed by an increase in sympathetic activity.

The arterial baroreflex is the major autonomic reflex controlling blood pressure in humans. This reflex responds primarily to changes in blood pressure sensed by changes in activity of baroreceptors located in the carotid sinus and aortic arch (Raven et al., 2006; Charkoudian and Wallin, 2014). A decrease in blood pressure elicits reflex increases in sympathetic activity and decreases in parasympathetic activity with the goal of increasing blood pressure back to baseline. An increase in blood pressure elicits the opposite reflex responses in the baroreflex. This reflex is considered to "buffer" large swings in blood pressure, which might otherwise be dangerous to the health of the individual. If we consider the "blood pressure equivalent" of Ohm's Law,

Mean Arterial Pressure (MAP):

$$= \textit{Cardiac Output (CO)} \times \textit{Total Peripheral Resistance (TPR)}$$
$$= [\textit{Heart Rate (HR)} \times \textit{Stroke Volume (SV)}] \times \textit{Total Peripheral Resistance (TPR)}$$

We note that sympathetic and parasympathetic neural mechanisms are able to control all three of the major variables that contribute to the maintenance of a normal arterial pressure.

Measurement of Autonomic Control of Cardiovascular Function in Humans

Direct measurement of autonomic function in humans has proven challenging and thus has driven somewhat of a reliance on directly or indirectly measured cardiovascular components (CO, HR, SV, TPR) to evaluate change in mean arterial pressure. In this section, we will briefly review some of the most common autonomic measurement techniques; the interested reader is referred to several comprehensive reviews on this topic (Charkoudian and Wallin, 2014; Hart et al., 2017; Shoemaker et al., 2018; Holwerda et al., 2020).

The gold standard for measurement of sympathetic nerve activity in humans is the technique of microneurography, developed in the late 1960s by Karl-Erik Hagbarth and colleagues at the University of Uppsala (Vallbo et al., 2004). This approach involves the use of a tungsten microelectrode, which is placed across the skin at the area of interest (usually the peroneal, median or radial nerve) and is manipulated with

small movements to be close enough to the nerve of interest to record the activity of that nerve. The most common recordings of human sympathetic activity are multi-unit recordings, in which several action potentials are recorded simultaneously, allowing the investigator to observe "bursts" of activity. Each burst represents a group of action potentials associated with norepinephrine release and downstream vasoconstriction (Charkoudian and Wallin, 2014; Hart et al., 2017). The most common measurements using microneurography are of sympathetic activity to the muscle vasculature (MSNA) and sympathetic activity to the skin (SSNA). One of the limitations of microneurography is that it can only measure activity of nerves that are accessible by percutaneous placement of electrodes, and cannot be used (in humans at least) for measurement of cardiac, renal or other regional activity of the sympathetic nervous system.

The most common way to get an index of the activity of the sympathetic nervous system in humans is with a simple measure of plasma norepinephrine (NE). Under many (but not all) conditions, including rest, plasma norepinephrine is strongly correlated with directly measured activity of the sympathetic nervous system (see next). However, the concentration of norepinephrine in the plasma at a given time is the net result of release (spillover), reuptake and metabolism – so that changes in any of these could result in changes in the plasma [NE], without changes in actual sympathetic noradrenergic activity. Thus, while this approach is helpful for assessing sympathetic activity, plasma NE data should be interpreted in the context of its limitations.

A method that has received increasing attention in recent decades is the approach of using frequency (spectral) analysis of cardiovascular variables (usually heart rate variability [HRV] or blood pressure) to give insight into the activity of sympathetic or parasympathetic nerves controlling those variables (Malliani and Montano, 2002). The major attraction of this approach is that it can be completely non-invasive and relatively simple to do (many systems offer automated HRV analyses of as little as 5 min of a 3-lead electrocardiogram). The basic idea behind frequency analyses is that the parasympathetic/vagal control of heart rate can change its activity very quickly. The vagus nerve transmits signals rapidly because it is large and myelinated, and the kinetics of acetylcholine at the heart are also rapid because of the presence of acetylcholinesterase at the synaptic junction (Draghici and Taylor, 2016). The sympathetic nerves, on the other hand, are small and unmyelinated and therefore transmit impulses relatively more slowly. This is the basis for the idea that "low frequency" power of frequency analyses is associated with sympathetic activity, whereas "high frequency" power is associated with the parasympathetic system (Draghici and Taylor, 2016). Various additional permutations of these calculations (e.g., low frequency/high frequency (LF/HF) ratio, alpha index, etc.) have been put forth over time (Malliani and Montano, 2002). Unfortunately, frequency analysis has many limitations and should not be used as a replacement for more direct measurement (such as those described above). For example, the high frequency component of HRV is not consistently associated with the tachycardia associated with direct pharmacological manipulation of the vagal system using atropine (Picard et al., 2009). If these analyses are used, they should be interpreted in light of their limitations and when possible as adjuncts to other approaches (Diaz and Taylor, 2006).

PART III. POST-EXERCISE REGULATION OF BLOOD PRESSURE AND BODY TEMPERATURE

The mechanisms that control thermoregulation and blood pressure are markedly challenged during exercise, particularly during exercise in the heat. Exercise increases the metabolic need for oxygen delivery at the skeletal muscle (Rowell, 1974), which is achieved via complementary mechanisms. During exercise, there is a decrease in sympathetic nerve activity of the vasoconstrictor organs (Chen and Bonham, 2010), allowing greater circulation to the working skeletal muscle to meet increased metabolic demands. These central mechanisms are aided by local vasodilator mechanisms including an increase in nitric oxide synthase activity (McNamara et al., 2014). Since dynamic exercise generates heat, it contributes to elevations in body temperature and therefore stimulates cutaneous vasodilation to a degree reflective of both elevations in skin and internal temperatures (Johnson, 2010).

Post-exercise, there is also a shift to a lower baroreflex setpoint (Halliwill et al., 2000), prompting greater venous pooling around the skeletal muscle (Halliwill 2013, Chen and Bonham, 2010) which can persist for several hours. Although this may be helpful to some aspects of post-exercise recovery, this persistent vasodilation in the periphery (which can contribute to post-exercise hypotension), tends to exacerbate orthostatic intolerance. The persistent vasodilation leads to more blood pooling in the extremities, decreasing venous return. This is particularly true immediately post-exercise when muscular contractions cease to serve as a skeletal muscle pump assisting in venous return (Rowell, 1974). Both high-intensity and endurance exercise can produce this effect of blood pooling in the skeletal muscle exacerbating orthostatic intolerances (Bjurstedt et al., 1983; Halliwill, 2001; Halliwill et al., 2013; Luttrell and Halliwill, 2015; Mundel et al., 2015). While blood pressure is markedly reduced immediately post-exercise, this hypotensive response is prolonged and in some cases has been observed to last up to 12 h (Claydon et al., 2006). Prolonged post-exercise hypotension is thought to aid in exercise recovery and adaptation. Specifically, post-exercise vasodilation, caused primarily by histamine receptor activation (Halliwill et al., 2013), may help to enhance plasma volume recovery by increasing albumin in the dilated vessels (Halliwill, 2001), allow for rapid storage of glycogen, and enhance muscle capillary density in endurance trained athletes (Halliwill et al., 2013).

Exercise Heat Stress and Contributing Mechanisms

The mechanisms governing blood pressure and body temperature regulation are further challenged when ambient heat is added to the exercise challenge (Johnson, 2010). During exercise in the heat, cardiac output, at a point determined by both

exercise intensity and degree of thermal stress, cannot increase sufficiently to fuel both the exercising skeletal muscle, and the skin to allow heat dissipation, thus, there is a competition for blood flow between these two circulations (Johnson, 2010). These cumulative demands can exacerbate post-exercise orthostatic intolerance as they contribute to a greater venous pooling in cutaneous and skeletal muscle compartments resulting from reductions in vascular resistance (Schlader et al., 2016b) effectively decreasing venous return and cerebral blood flow. For example, both elevated core and skin temperatures have been observed to reduce tolerance to lower body negative pressure (LBNP) (Pearson et al., 2017). Importantly, heat stress also leads to significant reductions in body mass reflective of sweat production and evaporation meant to dissipate heat. Sweat water loss is, at least partially, drawn from blood plasma (González-Alonso et al., 2008) further exacerbating competition for a diminished blood volume, lending to an augmented risk of orthostatic intolerance both during, and post-exercise (González-Alonso et al., 2008). Overall, when combined with heat stress, body water loss has been shown to have an additive effect on orthostatic intolerance and its symptoms (Schlader et al., 2015).

Variability in Orthostatic Tolerance

There is extensive inter-individual variability when it comes to orthostatic intolerance, which is related to factors such as age, sex, fitness status, hydration status, and certain medications. For example, while older individuals experience orthostatic intolerance and post-exercise syncope, the mechanisms governing post-exercise circulation are different (Murrell et al., 2009). Specifically, with stroke volume reduction post-exercise, younger athletes maintained total peripheral resistance, where older athletes experienced decreased TPR suggesting a decrease of sympathetic tone in both the arterial and venous vessels with age (Murrell et al., 2009). Women also appear more susceptible to orthostatic intolerance (Ganzeboom et al., 2003; Joyner et al., 2016). This might be explained by reduced cardiac filling and subsequent stroke volume in women (Fu et al., 2004), decreased mean sympathetic nerve activity and diastolic arterial pressure coherence (Yang et al., 2012), or decreased sympathetic nerve activity with respect to vasoconstriction (Joyner et al., 2016). Additionally, fitness status impacts the mechanisms associated with post-exercise hypotension and orthostatic intolerance with aerobically fit and sedentary men experiencing similar effects of hypotension post-exercise, but via distinct mechanisms (Senitko et al., 2002).

PART IV. POST-EXERCISE COLD COUNTERMEASURES TO MINIMIZE ORTHOSTATIC INTOLERANCE

Cardiovascular Responses to Cold Exposure

Cardiovascular responses to ambient cold at rest provide a foundational glimpse into how cold exposure might assist in efforts to improve orthostatic tolerance following exercise in the heat. Both local and whole-body responses to cooling contribute to increases in arterial pressure, primarily via their effect to increase peripheral vasoconstriction (Korhonen, 2006). Heart rate contributions to a cold-induced pressor response vary, with severe local and whole body (Korhonen, 2006) cold capable of inducing tachycardia, while mild to moderate whole body exposure induces bradycardia, likely via a baroreflex response caused by vasoconstriction (Yamazaki et al., 2000). In response to moderate skin surface cooling, sensitivity of heart rate control appears to be mediated by the arterial baroreflex rather than the carotid baroreflex, suggesting a central convergence and interaction between arterial baroreceptor and skin cold receptor afferents, predominantly in the aortic baroreflex pathway.

Adjustments in stroke volume may also contribute to the cold-induced pressor response. For example, 30 min of seated cool air (14.4°C) exposure, with minimal influence on T_c ($\leq$0.6°C), contributed to an intravascular fluid shift, decreasing plasma volume by 205 mL. A shift in net filtration of plasma from the blood into the interstitium is postulated to result from an increase in capillary hydrostatic pressure as a result of increased cutaneous venomotor tone (Harrison, 1985). Heightened venous return due to peripheral vasoconstriction stimulates increased atrial stretching and therefore stimulates release of plasma atrial natriuretic peptide (ANP) (Stocks et al., 2004). Circulating ANP results in enhanced sodium and water excretion and is therefore likely to be one of the mediators of cold-induced diuresis. This diuresis reduces plasma volume in response to cold stress, with cold air capable of reducing plasma volume by 7–15% (Bass and Henschel, 1956; Young et al., 1986) and cold water immersion by 15–20% (Young et al., 1986; Deuster et al., 1989). Despite reductions in plasma volume, stroke volume tends to increase in response to cold (Raven et al., 1970; Wagner and Horvath, 1985) due to a redistribution of blood from the periphery to the thoracic circulation. Blood redistribution occurs largely in response to changes in skin temperature, with maximum cutaneous vasoconstriction elicited by skin temperatures below 31°C, and is facilitated by an increase in sympathetic release of norepinephrine capable of interacting with cutaneous alpha-adrenergic receptors (Castellani and Young, 2016). The degree to which stroke volume increases appears to be linked intimately to the severity of cold, with lower ambient temperatures associated with greater increases in stroke volume (Wagner and Horvath, 1985).

Whole-body cold water immersion (CWI) has gained popularity as a post-exercise recovery technique due to its efficacy in recovering thermoregulatory variables including T_c and heart rate (Young et al., 1986) compared to air. CWI is a unique stimulus as it elicits physiological responses to both cold and hydrostatic pressure. Some reports have sought to differentiate between hydrostatic pressure alone (head-out thermoneutral water immersion) vs. hydrostatic pressure plus cold exposure (head-out cold water immersion). While plasma NE, systolic blood pressure, diastolic blood pressure, and TPR decreased with neutral water immersion, an increase in these variables were seen with CWI when compared to cold air. An increase in cardiac parasympathetic activity, marked by a decrease in heart rate, was elicited in both immersion conditions compared to air, with

larger response induced by CWI (Mourot et al., 2008). Therefore, it appears that despite the presence of hydrostatic pressure in both neutral and CWI, a complex modulation of autonomic response ensues with cold water.

Influences of Cold Exposure on Orthostatic Responses

Orthostatic tolerance is typically evaluated in a controlled laboratory setting using a head-up tilt test or lower body negative pressure (Yamazaki et al., 2000; Wilson et al., 2002, 2007; Durand et al., 2004; Cui et al., 2005; Johnson et al., 2017). In normothermic environments, acute moderate orthostatic stress decreases venous return and central venous pressure. Normal baroreflex responses, outlined above, result in reflex increases in heart rate and vascular sympathetic nerve activity, increasing peripheral vasoconstriction and preventing drop in arterial pressure. When cardiovascular adjustments are complicated by the concurrent presence of hyperthermia, the fall in central venous pressure and stroke volume is greater and accompanied by a blunted increase in total peripheral resistance. This TPR attenuation may be attributed to the continued prioritization of thermoregulatory convective skin perfusion thus contributing to a reduction in arterial blood pressure (Rowell, 1993; Yamazaki and Sone, 2000).

Wilson et al. (2002) examined the effects of combining whole-body heating using a water-perfused suit (46°C) combined with 10-min 60° head-up tilt to elicit orthostatic stress. The presence of concurrent heat and head-up tilt resulted in reductions in MAP as well as cerebral blood flow velocity that were attenuated, alongside an increase in total peripheral resistance, by the imposition of 15°C skin cooling, without an appreciable change in T_c. The increase in MAP induced by skin cooling appeared to be the result of both a decrease in heart rate alongside a more influential increase in TPR. Skin surface cooling reduced mean skin temperature during normothermic tilt to ∼28.3°C (∼Δ 6°C) and was able to similarly reduce mean skin temperature during heated tilt to ∼29.6°C (∼Δ 8.5°C). Furthermore, a cool water perfused suit, applied during 5 min of active 70° head-up tilt, similarly decreased skin temperature to 28°C. Compared to post-tilt normothermia, cooling induced a tilt response marked by greater mean arterial pressure largely attributable to an exaggerated increase in total peripheral resistance (Yamazaki et al., 2000).

A series of investigations further considered 16°C skin surface cooling as a countermeasure for orthostatic intolerance induced using progressive lower body negative pressure (LBNP). Durand et al. (2004) began LBNP at −30 mmHg for 3 min and progressively reduced LBNP until the occurrence of pre-syncopal symptoms while subjects were exposed to a cold water perfused suit. Compared to normothermia, skin surface cooling enhanced a standardized cumulative stress index (mmHg/min) by 33% indicating enhanced orthostatic tolerance. At most levels of LBNP, blood pressure during cooling was greater than during normothermia and during the early stages of LBNP, cooling attenuated a reduction in cerebral blood flow velocity. Furthermore, concentrations of plasma NE increased with skin surface cooling indicating an improvement of orthostatic tolerance modulated by an increase in sympathetic activity. Skin surface cooling before and during 5-min progressive LBNP stages (−10, −15, −20, −40 mmHg) solidified the capability of cooling to augment central blood volume and consequently central venous pressure. At low enough LBNPs (−20 and −40 mmHg), the increase in central venous pressure was reflected as an elevated stroke volume believed to contribute to the enhanced MAP induced by skin surface cooling (Cui et al., 2005). Further increasing the duration of LBNP to ∼15 min at −15 and −30 mmHg confirmed a 24% increase in central venous pressure accompanied by a 17% increase in pulmonary capillary wedge pressure during 16°C skin surface cooling (Wilson et al., 2007).

The results of these studies clearly established the efficacy of whole-body skin surface cooling as a countermeasure for orthostatic intolerance. However, the feasibility of implementation of water perfused suits in real-world scenarios of orthostatic stress, which are often more reactive than preventative, is low. Johnson et al. (2017) considered the "reactive" use of 0°C water face cooling during −30 mmHg LBNP stress to offset central hypovolemia. Cooling was applied to the forehead, eyes, and cheeks using a plastic bag of ice water and was maintained during 15 min of LBNP in an effort to stimulate the trigeminal nerve and consequently increase blood pressure (Schlader et al., 2016a). Face cooling effectively increased MAP via increases in cardiac output and forearm vascular resistance.

Real-World Application of Post-exercise Cooling

Skin surface cooling can clearly increase blood pressure; however, orthostatic stress resulting from exercise, heat, and/or a combination of both introduces additional circulatory stress, potentially complicating the effectiveness of cooling efforts. The majority of the aforementioned studies use skin surface cooling, in the absence of hyperthermia, to augment total peripheral resistance, enhance central venous return and thereby increase blood pressure. The degree to which these adjustments can be made when thermoregulatory demand remains high after the cessation of exercise is often overlooked. Logic may dictate that effectiveness in offsetting post-exercise blood pressure reduction may necessitate cooler water, greater body surface area exposure to cold, or alternative cooling media. A complicating factor in this context is the so-called "sympatholytic" effect of exercise and whole-body heat stress: vascular responses to sympathetic stimulation are blunted when compared with resting conditions (Tschakovsky et al., 2002; Wilson et al., 2002). Thus, even a strong stimulator of noradrenergic vasoconstrictor nerve activity may not elicit the degree of increased peripheral vascular resistance needed to maintain or improve arterial pressure.

Early work by Franklin et al. (1993) suggests that recovery from exercise in warm conditions (31.1°C, 53% RH), albeit only post and not during exercise, contributes to elevation of T_c and mean skin temperature up to 60 min after exercise cessation alongside a meaningful decrease in MAP compared to baseline (76.5 ± 2.0 vs. 81.2 ± 2.4 mmHg). In contrast, when subjects

are exposed to a neutral (21.4°C, 52% RH) or cool (17°C, 58% RH) post-exercise condition, both T_c and mean arterial pressure tend to return to baseline levels after 60 min. The likelihood of hypotension after exercise appears to be removed with cooler recovery conditions as a function of a quickened T_c recovery facilitated by a significant reduction in mean skin temperature. Furthermore, this study supports the notion that thermoregulatory mechanisms do play a significant role in the persistence of peripheral vasodilation post-exercise lending to the development of lowered blood pressure.

Cutaneous and Limb Blood Flow

As mentioned previously, persistent vasodilation post-exercise combined with the loss of the skeletal muscle pump, leads to blood pooling in the extremities, decreasing venous return and consequently arterial pressure (Rowell, 1974). For example, vastus lateralis perfusion continues to elevate above exercising levels after cessation of 40 min of treadmill running in ∼24°C (Ihsan et al., 2013). This post-exercise blood flow distribution may contribute to orthostatic hypotension, expected to be further exacerbated by the presence of skin thermoregulatory perfusion. Furthermore, exposing an exercised leg to 15 min of 10°C CWI reduced vastus lateralis total hemoglobin levels, suggesting that CWI may be capable of attenuating post-exercise microvascular perfusion (Ihsan et al., 2013). While a majority of the literature commenting on changes in post-exercise perfusion focus on cold water immersion, earlier reports utilized the simple application of an ice bag and yet still demonstrated attenuation of acute post-exercise perfusion elevation and edema compared to a non-cooled control limb (Yanagisawa et al., 2004). Similarly, whole-body CWI is capable of reducing post-exercise femoral vein diameter (Peiffer et al., 2009) and conductance (Mawhinney et al., 2013, 2017), arm blood flow (Vaile et al., 2011) as well as cutaneous perfusion (Mawhinney et al., 2013, 2017), although the extent of these reductions hold a non-linear relationship with CWI temperature.

Overall, elevated skeletal muscle temperature and skin perfusion following exercise in the heat contribute to a reduction in central venous pressure and a failure of TPR to increase appropriately with upright posture, leading to orthostatic intolerance. Cooling countermeasures appear to reduce both cutaneous and muscle blood flow to elicit a redistribution from the periphery to the thoracic vasculature at least when exercise is performed in thermoneutral conditions. Limited research has indicated that a reduction in large skeletal muscle microvascular perfusion following heated exercise is possible, although it appears to be smaller in magnitude than those changes seen following exercise performed in neutral ambient conditions. To improve our understanding of the influence of cooling countermeasures to prevent cardiovascular adjustments causing orthostatic intolerance, investigations examining the extent muscle and cutaneous vascular responsiveness may be blunted in response to varied cold stimuli following exercise performed in the heat are both warranted and necessary. Furthermore, it should be acknowledged that redistribution of cutaneous blood flow centrally could influence the degree of heat dissipation from the skin in a post-exercise setting. However, with a significantly widened thermal gradient elicited by skin surface cooling combined with a large preexisting degree of cutaneous vasodilation due to increased body temperatures, meaningful reductions in heat dissipation from the skin are likely minimal.

Cerebral Blood Flow and Oxygenation

Very few studies have specifically evaluated post-exercise cerebral blood flow modulation resultant from post-exercise cooling strategies. Post-exercise cooling may offset reductions in central venous pressure that would otherwise contribute to reductions in cerebral blood flow, reducing the risk of orthostatic intolerance. Skin surface cooling using a 15°C water-perfused suit immediately before head-up tilt induced orthostatic stress was successful in preventing the fall in cerebral blood flow velocity by increasing mean arterial pressure (Wilson et al., 2002). In contrast, other literature indicates that CWI may further reduce a pre-frontal lobe NIRS-measured index of cerebral blood volume and oxygenation following heated high-intensity exercise (Minett et al., 2014). Because reduced cerebral blood flow velocity is strongly linked to orthostatic intolerance (Novak, 2016) and methodological considerations limit the interpretation of specific regional blood volume quantifications, it is likely that post-exercise cooling efforts are capable of augmenting cerebral perfusion and consequently reducing the likelihood of orthostatic intolerance. Still, further investigation of skin surface cooling vs. cold water immersion to prevent reductions in cerebral blood flow velocity specifically following heated exercise is warranted.

Autonomic Cardiac Control: CWI and Heart Rate Variability

The ability of water immersion to increase central venous pressure via a shift of peripheral blood into the thoracic vasculature simultaneously stimulates high arterial pressure and low cardiopulmonary pressure baroreflexes (Pump et al., 2001), which can then elicit an increase in cardiac parasympathetic (vagal) tone. Since it is difficult/impossible to directly measure cardiac autonomic activity, heart rate variability (HRV) has served as a surrogate measure to evaluate post-exercise parasympathetic activity related to water immersion, and as an index of cardiovascular and hemodynamic recovery. Water temperature appears to play a key role in the effectiveness of water immersion to influence parasympathetic reactivation. Several reports implicate cold water immersion post-exercise as a greater modulator of cardiac parasympathetic reactivation compared to neutral or warm water immersions, both when exercise is performed in thermoneutral (Al Haddad et al., 2010; Stanley et al., 2012; de Oliveira Ottone et al., 2014) as well as a heated environment (Buchheit et al., 2009; Choo et al., 2018). Further reduction of water temperature beyond 14°C does not appear to elicit a greater benefit in terms of cardiovascular recovery (Choo et al., 2018). Importantly, the limitations of HRV are discussed earlier in this review and as such future use of HRV to assess post-exercise cooling responses are best used and interpreted in conjunction with more directly mechanistic measurements.

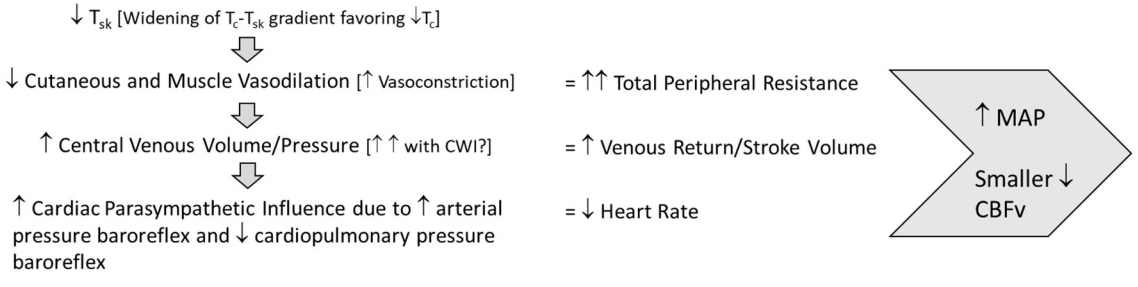

FIGURE 1 | Post-exercise cooling cardiovascular adjustments to maintain orthostatic tolerance. T_{sk}, skin temperature; T_c, core temperature; MAP, mean arterial pressure; CBF_v, cerebral blood flow velocity.

CONCLUSIONS AND FUTURE DIRECTIONS

Successful orthostatic tolerance requires appropriate baroreflex responses to upright posture. During and after exercise in the heat, the ability of the baroreflex to cause vasoconstriction necessary to defend mean arterial pressure is limited by cutaneous vasodilation, elevated tissue temperature and peripheral venous pooling. Post-exercise cooling, especially cold water immersion, appears to augment both mean arterial pressure and cerebral vascular perfusion to minimize or prevent orthostatic intolerance after exercise in the heat (**Figure 1**). Still, the uniform skin temperatures created by the use of a water-perfused suit in many of the research investigations discussed within this review limit real-world applicability. Therefore, more research is necessary to further understand and optimize real-world approaches to post-exercise cooling to definitively improve orthostatic tolerance and minimize injury. Optimal timing of cooling strategies (before, during, or after exercise heat stress) to effectively offset the development of OI should also be investigated, as proactive strategies may be safer and more logistically feasible than reactive strategies. Lastly, continued evaluation of post-exercise cooling techniques specifically with women is necessary to determine the influence of estradiol and its fluctuations specifically on the cardiovascular adjustments that control skin perfusion.

AUTHOR CONTRIBUTIONS

All authors designed and outlined the work, performed literature reviews and interpreted findings, and drafted and revised the manuscript. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

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Muscle Strength and Power: Primary Outcome Measures to Assess Cold Water Immersion Efficacy After Exercise With a Strong Strength or Power Component

Angus Lindsay[1]* *and Jonathan M. Peake*[2,3]

[1] *Institute for Physical Activity and Nutrition, School of Exercise and Nutrition Sciences, Deakin University, Geelong, VIC, Australia,* [2] *School of Biomedical Sciences, Queensland University of Technology, Brisbane, QLD, Australia,* [3] *Sport Performance Knowledge and Innovation Excellence, Queensland Academy of Sport, Brisbane, QLD, Australia*

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***Correspondence:**
Angus Lindsay
a.lindsay@deakin.edu.au

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INTRODUCTION

Recovery from exercise-induced muscle damage, fatigue or stress is critical for restoration of exercise performance. In most exercise activities or sports, performance is regulated through the physiological capacity of muscle. Therefore, athletes, coaches, and scientists have continued to explore post-exercise recovery modalities that focus on expediting muscle functional recovery. Several post-exercise recovery strategies have been developed, tested, and used in amateur and professional athletes to expedite muscle functional recovery. A cost-effective and well-researched practice to achieve this goal is cold water immersion (CWI). CWI requires submersion of a limb or the whole body in cold water of a specified temperature (usually $<15^{\circ}$C) for a specified duration immediately post-exercise, or over several succeeding days. However, the equivocal findings on the efficacy of CWI and the extensive number of outcome variables has made it challenging to interpret and correctly implement this intervention. In this short opinion piece, we briefly review CWI research and the challenges that practitioners and athletes face when deciding whether to use CWI as a post-exercise recovery intervention. We then discuss why muscle strength and/or power should be considered the primary outcome variable in CWI research with a strong strength and power component, and why excitation–contraction coupling and/or rate of force development assessment is necessary to evaluate strength/power-specific changes. Finally, we present systematic evidence that there is a dearth of strength measurements in CWI research, which could be limiting our understanding of this post-exercise recovery strategy.

A BRIEF HISTORY OF CWI RESEARCH

Some of the first evidence that a single CWI application might be beneficial for recovery from muscle damage or injury was provided by Hayden (Hayden, 1964) and Hocutt et al. (1982). Both studies showed that CWI expedited return-to-duty in soldiers after injury, or return to full activity after ankle sprain, respectively. In contrast, Matsen et al. and Marek et al. showed that application of cold water to an injury significantly increased oedema (Matsen et al., 1975; Marek et al., 1979). Subsequent research about the effects of CWI has also produced some contrasting findings. For example, CWI immediately following blunt trauma to skeletal muscle of rats significantly reduced oedema formation (Dolan et al., 1997). By contrast, CWI following eccentric contractions did not affect muscle soreness or strength in humans (Eston and Peters, 1999). More recently, Naderi et al.

showed that CWI did not attenuate a loss in muscle strength following a single bout of strength training (Naderi et al., 2021), whereas Kodejška et al. (2018) demonstrated that CWI increased the force time integral in handgrip performance compared with passive recovery in rock climbers. Differences in CWI study outcomes could be associated with different modes of exercise, methods or timing of applying CWI and approaches to assessing muscle damage.

Contradictory findings related to repeated CWI applications have also been reported and confirmed in a recent systematic review and meta-analysis (Malta et al., 2021). Fu et al. (1997) showed that when CWI was regularly applied to rats after exercise training, it caused advanced ultrastructural damage to myofibrils. Several human studies have also shown negative adaptive effects of repeated CWI applications after resistance training (Fröhlich et al., 2014; Roberts et al., 2015; Yamane et al., 2015; Fyfe et al., 2019; Poppendieck et al., 2020). However, Lindsay et al. (2016) showed that repeated CWI applied to mixed martial artists during a training camp attenuated the inflammatory response, but did not affect measures of performance. Repeated CWI applied after high intensity interval training (HIIT) or a combination of HIIT with low-moderate intensity aerobic exercise also does not influence indices of performance or muscle cellular signaling (Halson et al., 2014; Aguiar et al., 2016; Christiansen et al., 2018). Although these studies represent only a small proportion of published CWI research, they do demonstrate the complexity in understanding the value of this recovery intervention.

INTERPRETING CWI RESEARCH

Regardless of the inconsistencies in CWI study outcomes, anecdotal evidence suggests that professional athletes from various sports use CWI as a post-exercise recovery strategy. The reasons behind this persistent practice are uncertain, but may reflect a disconnect between the scientific findings of the studies, and how coaches and athletes interpret these findings. The practitioners' guide to determining if, and when, to implement CWI is confounded by the vast performance, biochemical, and qualitative analyses that have been used to evaluate its efficacy. Other than the variability in CWI protocols (which can range from 4 to 15°C, 5 to 30 min durations, 1 to 10 applications, immediate to delayed submersion) and level of exercise intensity, participant sex and training status, the outcome variables of interest provide an added level of complexity in CWI study comparisons. First, a practitioner or self-coached athlete without in-depth scientific knowledge of biological processes, may not be able to interpret correctly the results of CWI studies that focus on indices of inflammation, gene expression or rates of protein synthesis. Second, drawing comparisons between performance and biochemical or molecular variables could be challenging for a non-scientist. Physiological performance analysis offers a direct and interpretable option for practitioners that is training-specific. We therefore propose that consistently measuring maximal muscular strength and/or power [product of load lifted and angular displacement (distance load moved) divided by time spent moving the load (Sapega and Drillings, 1983; Winter et al., 2016; Horta-Gim et al., 2021)] will provide the exercise community with a more appropriate understanding of whether CWI enhances recovery from exercise-induced damage or fatigue, and improves the performance and work capacity of athletes. From our perspective as exercise physiologists, maintenance of muscular strength and power, irrespective of any changes in muscle ultrastructural integrity, will likely benefit overall physical performance.

MUSCLE STRENGTH/POWER AND MECHANISMS OF STRENGTH/POWER LOSS

Muscle strength and power outcomes is a multi-faceted coordination of electrical and chemical events, together with interactions between structural components of muscle tissue. Strength and power are measured using the 1-repetiton maximum (actual or estimated), or with force transducers or plates associated with lab-based dynamometers that measure absolute torque production and rate of force development. Loss of muscle strength associated with eccentric contractions (which lengthen the muscle during simultaneous force production) can be primarily attributed to excitation–contraction uncoupling, and to a lesser extent, loss of contractile protein and structural damage (Warren et al., 2001, 2002). Therefore, post-exercise recovery interventions should target the processes of excitation–contraction coupling to accelerate recovery from eccentric contraction-biased exercise. The triad of skeletal muscle is the site of excitation–contraction uncoupling following eccentric contraction-induced strength loss. More specifically, it is the voltage-sensitive dihydropyridine receptors (DHPR) located in the T-tubules and the ryanodine receptor (RyR) calcium release channel of the sarcoplasmic reticulum (Ingalls et al., 1998; Warren et al., 2001, 2002; Corona et al., 2010; Baumann et al., 2014). The sensitivity of both the DHPR and RyR are not affected by eccentric contraction-induced strength loss (Ingalls et al., 2004a). However, the expression of proteins that associate with the DHPR and RyR to modulate cross-talk and calcium release is significantly decreased (Corona et al., 2010; Baumann et al., 2014). Thus, assessing the effectiveness of CWI for restoring muscle strength could include molecular measurement of the DHPR, RyR, junctophilin, FKPB12, calmodulin, or calsequestrin (proteins associated with the triad of muscle fibers and known to interact with channels and receptors regulating skeletal muscle calcium kinetics). Because cold acclimation can influence calcium handling/kinetics of skeletal muscle and improve indices of muscle performance (Bruton et al., 2010), additional calcium measurements following CWI could supplement analyses of excitation–contraction coupling. However, we do acknowledge that such measurements of proteins following CWI would require time course evaluation, multiple muscle biopsies that would complicate human studies with respect to recruitment and full participation, and confound interpretations of findings by non-scientists.

Researchers can indirectly assess excitation–contraction uncoupling *in vivo* by comparing the low-frequency to high-frequency torque loss prior to and following CWI. The greater reduction in low-frequency torque compared with high-frequency torque indicates excitation–contraction uncoupling (Edwards et al., 1977; Jones et al., 1982; Ingalls et al., 2004b). Cheng et al. (2017) showed that in isolated single muscle fibers of mice, cold application following fatiguing contractions dampened submaximal force without altering maximal force during recovery. In fact, the ratio of submaximal to maximal force was lowest with the coldest temperature, suggesting greater excitation–contraction uncoupling with colder applications. Additionally, we acknowledge that force-generating capacity during rapid, dynamic movements is also relevant to athletic performance and may represent a more sensitive measure to detect changes in neuromuscular function. Rate of force development can generally be determined by measuring the change in peak force divided by a change in time (maximal rate of contraction to accommodate for inter-individual variability in peak force development time) using lab-based force transducers and associated software. Central nervous system (CNS) fatigue likely also influences recovery of muscle strength and/or power following a single or repeated applications of strenuous exercise (Peiffer et al., 2009). Therefore, measuring CNS fatigue would also improve the assessment of muscle function recovery. Non-invasive CNS assessments could use an interpolated twitch during a maximal voluntary contraction (Allen et al., 1995) but would require the use of stimulation units. Collectively, more research of this nature will help to improve understanding of how CWI influences muscle function.

Loss of muscle strength and/or power can also be caused by fatiguing contractions (i.e., short-term strength and/or power loss caused primarily by energy depletion, and/or short-term "reversible" decrements in excitation contraction coupling) or blunt force trauma—the latter of which causes damage to structural and force-generating proteins of the muscle. Therefore, restoration of muscle strength and/or power by CWI would ideally need to affect several components of excitation contraction-coupling, synthesis of essential proteins, and restoration of the muscle architecture. CWI is thought to expedite recovery from exercise by lowering skin, intramuscular and body temperature, cardiovascular strain, blood flow and increasing metabolism, blood pressure and heart rate (Bleakley and Davison, 2010b; Ihsan et al., 2016). Although CWI does not influence glycogen resynthesis rates after exhaustive exercise in humans (Gregson et al., 2013), other cryotherapy applications can reduce inflammatory cell infiltration after soft tissue injuries in animal studies (Bleakley and Davison, 2010a) and CWI can lower inflammatory biomarkers after contact sport (Lindsay et al., 2017) and resistance exercise in humans (Missau et al., 2018). However, there are equivocal findings that CWI does not affect muscle-specific or circulating inflammatory biomarkers after resistance exercise (Peake et al., 2017a), repeated sprints (White et al., 2014) or volleyball training (De Freitas et al., 2019) in humans. This variation may be attributed to the level of muscle damage imposed by the initial exercise. The first wave of responders to sites of muscle damage (strength and/or power loss) includes granulocytes, and mononucleated cells such as macrophages, eosinophils and monocytes. Considering that cold-stress limits mononuclear cell activity (Lindsay et al., 2016; Reynés et al., 2019), and inflammation is integral to muscle repair and regeneration (Peake et al., 2017b), it follows that CWI may in fact delay the sequence of events involved in muscle repair (Tidball, 2011) and the recovery of muscle strength and/or power. Additionally, CWI may slow recovery from structural protein damage, because protein synthesis, ribosomal biogenesis and anabolic signaling are temperature-dependent (Roberts et al., 2015; Figueiredo et al., 2016; Fuchs et al., 2020). Overall, the mechanisms by which CWI may affect recovery of muscle strength and/or power have not definitively been determined.

Ensuring muscle strength and power measurements are considered as a primary outcome measure for CWI studies investigating forms of exercise in which recovery of strength/power is important (independent of inflammatory status or the ultrastructural integrity of the muscle) is critical. This is because even muscle that is severely structurally compromised, with a steady state of inflammation and heightened sensitivity to exercise-induced loss of sarcolemmal excitability, can produce strength and power. For example, skeletal muscle from dystrophin-deficient mice, a model of Duchenne muscular dystrophy, undergoes continuous cycles of degeneration and regeneration, inflammation, exercise-induced loss of sarcolemmal excitability and replacement of muscle with adipose and fibrotic tissue (Tanabe et al., 1986; Baumann et al., 2020). Functional analyses indicate that absolute strength and rate of force development during a twitch and tetanic contraction of these dystrophin-deficient muscles in mice is not different to healthy skeletal muscles (Lindsay et al., 2019). However, although inflammatory status and skeletal muscle integrity might not affect muscle strength and/or power in a diseased state, it may predispose muscle of healthy individuals to greater levels of exercise-induced stress that could, in turn, lead to poorer long-term performance or extended recovery periods.

BRIEF SYSTEMATIC REVIEW—CWI AND MUSCLE STRENGTH

Despite the variation in outcome variables and advancements in muscle strength and power assessment technologies for CWI research in humans, relatively few studies have included the measurement of muscle strength and power as a measure of the effectiveness of CWI. A literature search in PubMed identified a total of 427 peer-reviewed studies on "cold water immersion" AND "muscle" (01/12/2020). Of these 427 studies, 31 (7%) measured muscle strength prior to and following exercise and CWI. Of the 31 studies that measured strength prior to and following an intervention, 14 studies showed positive effects for CWI over a passive or active recovery modality on strength and/or power variables, six studies showed that CWI was detrimental to muscle strength and/or power, and 11 studies showed no effect. Twenty-one of the 31 studies completed only a single application of CWI, whereas 10 studies completed two or more applications of CWI. Overall, our literature search of CWI

and muscle strength measurements provides conflicting evidence that CWI has beneficial effects for muscle strength variables.

CONCLUSION

The efficacy of CWI has been tested and studied for decades, with large variation in outcomes. Although outcome measures remain relatively constant, the difficulty in assessing CWI as a strategy for post-exercise recovery is associated with the variability in the intervention itself. While investigating CWI protocol variables does provide additional information, it somewhat contributes to the level of confusion accompanying this modality for amateur and professional athletes. Therefore, we re-iterate that independent of the CWI protocol used in a study setting, that measures of absolute or relative muscle strength and/or power should be the primary measurement. This approach will at least offer scientists, athletes and coaches a comparison among CWI studies in the outcome variable that is relatively easy to interpret, and matters most to athletic performance.

AUTHOR CONTRIBUTIONS

AL conceived and wrote the opinion. JP conceived and critically reviewed the opinion. All authors contributed to the article and approved the submitted version.

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MINI REVIEW
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Adaptations to Post-exercise Cold Water Immersion: Friend, Foe, or Futile?

Mohammed Ihsan[1,2]*, *Chris R. Abbiss*[3] *and Robert Allan*[4]

[1] Human Potential Translational Research Programme, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, Singapore, [2] Research and Scientific Support, Aspetar Orthopaedic and Sports Medicine Hospital, Doha, Qatar, [3] Centre for Exercise and Sports Science Research, School of Medical and Health Sciences, Edith Cowan University, Perth, WA, Australia, [4] School of Sport and Health Sciences, University of Central Lancashire, Preston, United Kingdom

In the last decade, cold water immersion (CWI) has emerged as one of the most popular post-exercise recovery strategies utilized amongst athletes during training and competition. Following earlier research on the effects of CWI on the recovery of exercise performance and associated mechanisms, the recent focus has been on how CWI might influence adaptations to exercise. This line of enquiry stems from classical work demonstrating improved endurance and mitochondrial development in rodents exposed to repeated cold exposures. Moreover, there was strong rationale that CWI might enhance adaptations to exercise, given the discovery, and central role of peroxisome proliferator-activated receptor gamma coactivator-1α (PGC-1α) in both cold- and exercise-induced oxidative adaptations. Research on adaptations to post-exercise CWI have generally indicated a mode-dependant effect, where resistance training adaptations were diminished, whilst aerobic exercise performance seems unaffected but demonstrates premise for enhancement. However, the general suitability of CWI as a recovery modality has been the focus of considerable debate, primarily given the dampening effect on hypertrophy gains. In this mini-review, we highlight the key mechanisms surrounding CWI and endurance exercise adaptations, reiterating the potential for CWI to enhance endurance performance, with support from classical and contemporary works. This review also discusses the implications and insights (with regards to endurance and strength adaptations) gathered from recent studies examining the longer-term effects of CWI on training performance and recovery. Lastly, a periodized approach to recovery is proposed, where the use of CWI may be incorporated during competition or intensified training, whilst strategically avoiding periods following training focused on improving muscle strength or hypertrophy.

Keywords: recovery, cryotherapy, hypertrophy, mitochondrial biogenesis, muscle adaptations

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Edited by:
Oliver Faude,
University of Basel, Switzerland

Reviewed by:
James Robert Broatch,
Victoria University, Australia
Erich Hohenauer,
THIM University of Applied Sciences for Physiotherapy, Switzerland

***Correspondence:**
Mohammed Ihsan
Ihsan.m@nus.edu.sg

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INTRODUCTION

Cold water immersion (CWI) is a strategy aimed at enhancing recovery from strenuous exercise, typically involving the submersion up to the waist or mid-torso for ∼5–20 min in temperatures between ∼8 and 15°C (Versey et al., 2013; Ihsan et al., 2016; Machado et al., 2016). Following contemporary work by Vaile et al. (2008a,b,c); Vaile et al. (2011) and Peiffer et al. (2010a,b) in

late-2000s investigating the effect of CWI on the recovery of physical performance, research in this area has extended to investigate a plethora of recovery outcomes including thermoregulatory response (Stephens et al., 2018), hemodynamics (Mawhinney et al., 2013; Choo et al., 2016), hormonal balance (Halson et al., 2008), skeletal muscle damage (Goodall and Howatson, 2008), and autonomic nervous function (Bastos et al., 2012; Choo et al., 2018). Complementing this growth in research, CWI has become one of the most popular post-exercise recovery strategies utilized amongst athletes during training and competition (Périard et al., 2016; Crowther et al., 2017; Murray et al., 2018; Cross et al., 2019).

Meta-analyses and experimental research in general show that CWI can beneficially influence the recovery of physical performance (Montgomery et al., 2008; Bleakley and Davison, 2010; Rowsell et al., 2011; Poppendieck et al., 2013; Tabben et al., 2018). Nevertheless, many consider the efficacy of CWI to be equivocal. The inconsistent findings of CWI hence must be acknowledged, and this is likely driven by factors such as the nature of exercise modality preceding CWI, nature of recovery variables assessed, timing between recovery assessment and completion of CWI, and the CWI protocol itself. While such complexities need to be resolved to appropriately compare study findings and interpret with context, recent discussions have revolved extensively around how the regular use of CWI for recovery might in parallel influence adaptations to exercise (Broatch et al., 2018; Malta et al., 2021). Yet, the first study to address this extends considerably back to 2006 (Yamane et al., 2006). Following a hiatus, a series of studies examining the influence of CWI on adaptation to endurance exercise emerged from independent laboratories (Ihsan et al., 2014b, 2015, 2020b; Aguiar et al., 2016; Joo et al., 2016; Allan et al., 2017, 2019, 2020; Broatch et al., 2017), which was followed up by others examining the influence of CWI on resistance training adaptations (Frohlich et al., 2014; Roberts et al., 2015; Figueiredo et al., 2016; D'Souza et al., 2018; Fyfe et al., 2019; Fuchs et al., 2020; Peake et al., 2020; Poppendieck et al., 2020). A recent meta-analytical review showed that CWI effects on exercise adaptations are mode-dependant, where resistance training adaptations were diminished, whilst aerobic exercise performance seemed unaffected (Malta et al., 2021). Alongside these publications, detailed narrative reviews on the adaptative response following regular CWI have been published within this research topic series (Petersen and Fyfe, 2021), and elsewhere (Broatch et al., 2018). Additionally, editorials, point-counterpoints, and opinion pieces (Allan and Mawhinney, 2017; McPhee and Lightfoot, 2017; Méline et al., 2017; Peake, 2017, 2020; White and Caterini, 2017; Cheng, 2018; Ihsan et al., 2020a) have been published discussing the suitability of CWI as a post-exercise recovery tool given its dampening effect on hypertrophy gains. However, potential adaptive benefits that can be harnessed from CWI following endurance exercise, or from a recovery objective are often overlooked.

In this mini-review, we have adopted an introspective approach in discussing the molecular mechanisms and rationale surrounding CWI and endurance exercise adaptations. Moreover, we review and discuss key studies which provide information on the applied scenarios where CWI can be utilized to promote physical recovery and adaptation whilst avoiding potential negative effects on hypertrophy and strength gains.

COLD EXPOSURE AND ENDURANCE EXERCISE ADAPTATION

Cold stimulus is a physiological stressor capable of triggering primary signals and downstream cascades implicated in exercise-induced improvements in muscle oxidative function (Ihsan et al., 2014a). Evidence for cold-induced changes in a/the mammalian oxidative profile can be derived from as early as 1960, where Hannon (1960) demonstrated robust increases in the enzyme activities of several electron transport chain components of rat gastrocnemius muscle following 3–4 weeks of cold exposure. Subsequent studies extended these initial findings by demonstrating comparable increases in muscle oxidative enzymes between cold acclimation and exercise (Hamilton and Ferguson, 1972; Harri and Valtola, 1975). More recent studies have since shown improved fatigue resistance and exercise capacity following cold adaptation, in line with molecular signatures implicating increased mitochondrial content (Schaeffer et al., 2003; Bruton et al., 2010). In summary, research within rodent models highlight cold exposure as a viable modality to enhance muscle oxidative adaptations and endurance.

Seminal work by Spielgeman's group in the late 90's generated major breakthroughs in the mechanisms underpinning mitochondrial biogenesis (Puigserver et al., 1998; Wu et al., 1999), which incidentally further supported the use of cold therapeutic strategies such as CWI. Their work investigating the mechanisms of adaptive thermogenesis led to the discovery of the transcriptional coactivator peroxisome proliferator-activated receptor gamma coactivator-1α (PGC-1α), which was found to robustly increase in response to cold exposure (3 and 12 h exposure @ 4°C) in mice brown fat and skeletal muscle, concomitant with an increase in numerous mitochondrial markers (Puigserver et al., 1998; Wu et al., 1999). Subsequent work highlighted a regulatory role for PGC-1α in mitochondrial biogenesis (Lira et al., 2010), and other oxidative adaptations such as angiogenesis through the vascular endothelial growth factor (VEGF) (Chinsomboon et al., 2009), muscle fiber type transformation (Lin et al., 2002), glucose (Wende et al., 2005), and fat metabolism (Vega et al., 2000) in cell culture and murine models. Following evidence within human exercise models demonstrating a regulatory role for PGC-1α in skeletal muscle aerobic adaptations (Pilegaard et al., 2003; Russell et al., 2003; Perry et al., 2010), there was understandably speculation that CWI might additively enhance adaptations to exercise through common mechanisms involving PGC-1α (Ihsan et al., 2014a).

ENDURANCE EXERCISE ADAPTATION TO POST-EXERCISE CWI: DISCREPANCY BETWEEN MOLECULAR SIGNALING AND EXERCISE PERFORMANCE

Whilst CWI was not conceived as a strategy specifically meant to supplement exercise adaptations, there was substantial interest

in examining how recovery-based CWI might influence skeletal muscle adaptations to endurance exercise (Ihsan et al., 2014b, 2015, 2020b; Aguiar et al., 2016; Joo et al., 2016; Allan et al., 2017, 2019, 2020; Broatch et al., 2017). This line of enquiry is likely motivated by work in cell cultures and rodents demonstrating robust increases in mitochondrial markers following exercise and cold exposure with common mechanisms involving PGC-1α. Moreover, recent work in humans extends further support, where markers of mitochondrial development have been shown to be enhanced in the skeletal muscle after acute aerobic exercise in the cold compared with room temperature (Shute et al., 2018), or when post-exercise recovery was undertaken in a cold environment (Slivka et al., 2013). In agreement, CWI (10–15 min @ 8–10°C) administered independently, or following an acute bout of endurance exercise was shown to increase the mRNA of PGC-1α and VEGF (Ihsan et al., 2014b; Joo et al., 2016). Additionally, regular CWI (15 min @ 10°C) application during 4 weeks of endurance training (30 s to 8 min interval bouts @ 80–110% of peak power/velocity) has been shown to increase a variety of mitochondrial markers (mRNA and protein abundance) and upstream regulatory kinases (Ihsan et al., 2015; Aguiar et al., 2016). However, an increase in PGC-1α protein content was not consistently observed in these studies (Ihsan et al., 2015; Aguiar et al., 2016) with only Ihsan et al. (2015) reporting an CWI-mediated increase following training. Regardless, complimenting the aforementioned studies showing an increase in VEGF mRNA (Ihsan et al., 2014b; Joo et al., 2016), regular CWI (10–15 min @ 10°C) incorporated during exercise training lasting 4–12 weeks has been shown to enhance skeletal muscle microvascular function (Ihsan et al., 2020b) and increase skeletal muscle capillarity (D'Souza et al., 2018). Conversely, Broatch et al. (2017) found no effect of CWI (15 min @ 10°C) on molecular markers indicative of mitochondrial development (i.e., mRNA responses, phosphorylation status, and protein abundance) following a single sprint interval training session or following 6 weeks of sprint interval training. Factors such as subcutaneous fat, muscle mass, body surface area, and acclimation status may influence the adaptations that are driven by the magnitude of tissue temperature change, and hence partly account for such disparity in findings. Alternatively, high-intensity exercise/training as undertaken by Broatch et al. (2017) can robustly increase mitochondrial markers (Granata et al., 2016) creating a ceiling effect, resulting in diminished potential for CWI augment further adaptations. Collectively, these findings indicate that the effects of post-exercise CWI may be less pronounced following high-intensity exercises, but is able to influence molecular and structural adaptations befitting muscle oxidative function following lower intensity endurance exercise.

Although such molecular responses would expectedly improve endurance performance in the longer term, Yamane et al. (2006) reported attenuated improvements in maximal oxygen uptake and cycling time to exhaustion following regular CWI (2 × 20 min at 5°C) during 4 weeks of endurance training. The authors suggested that the decrease in muscle temperature and metabolism following cooling might have suppressed regenerative mechanisms mediated through inflammatory and heat shock proteins (HSP). This mechanism is unlikely prevalent during recovery-based CWI protocols resulting in mild to moderate decreases in tissue temperature. Indeed, typical post-exercise CWI involves 10–15°C immersion for 10–15 min, and such protocols have been shown to not influence skeletal muscle inflammatory response, HSP expression, or trafficking (Aguiar et al., 2016; Peake et al., 2017). Interestingly, HSF-1, a transcription factor for multiple HSPs, has been shown to be upregulated following regular CWI administered throughout 4 weeks of endurance training (Aguiar et al., 2016). As such, the findings demonstrated by Yamane et al. (2006) likely involve other mechanisms such as increased muscle proteolysis, increased oxidative stress or lowered tissue metabolism and consequently remodeling (Fu et al., 1997; Manfredi et al., 2013; Broatch et al., 2018) that are associated with aggressive cooling and extreme decreases in tissue temperature.

In contrast to Yamane et al. (2006) initial report, studies investigating the longer-term effects of recovery-based CWI do not support concerns of this modality being detrimental to endurance training adaptations. For instance, regular CWI administered during 4–6 weeks of sprint- or aerobic-interval training similarly improved maximal oxygen uptake, peak aerobic power, and time-trial performance compared to control conditions (Aguiar et al., 2016; Broatch et al., 2017). Likewise, CWI administered to competitive cyclists undergoing 3-4 weeks of intensified training reported similar improvements in most cycling performance parameters, although some parameters were reported to improve to a greater extent following CWI (Halson et al., 2014).

While these findings refute suggestions that CWI might counteract endurance adaptations, it nevertheless questions whether post-exercise CWI is an effective strategy to promote muscle adaptations resulting in improved exercise performance. Indeed, changes in acute signaling response (Ihsan et al., 2014b; Joo et al., 2016; Broatch et al., 2017), training-induced protein accretion (Ihsan et al., 2015; Aguiar et al., 2016), and vascular adaptations (D'Souza et al., 2018; Ihsan et al., 2020b) do not seem to translate into improved exercise performance following regular CWI. Some have reasoned that endurance performances are largely governed by central factors (e.g., cardiovascular, hematological adaptations), and changes in muscle aerobic function following CWI may only marginally contribute (Malta et al., 2021). Alternatively, frequent CWI might have de-sensitized transcriptional responses. For instance, the magnitude of PGC-1α mRNA increases have been shown to progressively diminish in response to repeated exercise stimulus (Perry et al., 2010). Similarly, PGC-1α mRNA has been shown to robustly increase following exercise in a cold environment, but demonstrated a blunted PGC-1α mRNA response to an identical stimulus following 3 weeks of endurance training in the cold (Shute et al., 2020). However, it remains to be ascertained if this attenuated response is due to habituation to cold, exercise or a combination of both stimuli. Regardless, it must be re-iterated that CWI does not appear to impair aerobic training adaptations, and can

be confidently incorporated as a recovery modality following endurance training sessions.

EFFECT OF CWI ON ENDURANCE AND RESISTANCE EXERCISE ADAPTATIONS: DIVERGENT EFFECTS OR COORDINATED REGULATION?

While some studies have shown that CWI can enhance physical recovery following resistance exercise (Vaile et al., 2008c; Roberts et al., 2014), practitioners should avoid scheduling this modality at least during the immediate recovery period. Indeed, regular CWI has been shown to attenuate the magnitude of anabolic signaling (Roberts et al., 2015) and protein synthesis (Fuchs et al., 2020), leading to reduced magnitude of strength and muscle mass gain following resistance training (Frohlich et al., 2014; Roberts et al., 2015; Fyfe et al., 2019; Poppendieck et al., 2020). Readers are directed to excellent reviews elsewhere (Broatch et al., 2018; Malta et al., 2021) and within this research topic (Petersen and Fyfe, 2021) elaborating on the mechanisms surrounding CWI and resistance training.

Complimenting these mechanisms, we suggest that the attenuated increase in muscle mass observed following CWI and resistance training may be part of a macro-level mechanism protecting the oxidative profile of the muscle. This is supported by D'Souza et al. (2018) demonstrating increased muscle capillarity following 12 weeks of resistance training with regular CWI application with concomitant decreases in muscle mass reported in other companion papers (Roberts et al., 2015; Peake et al., 2017). Reductions in muscle blood flow and metabolism during CWI may reduce O_2 supply and utilization (Ihsan et al., 2013; Mawhinney et al., 2013, 2020; Choo et al., 2016), triggering compensatory adaptation involving decreased muscle mass and microvascular expansion to maintain perfusion capacity. Further support for such a phenotypic response can be derived from rodent and human models of cold-acclimation. Cross-sectional areas of gastrocnemius and soleus muscle fibers were found to be 15–21% smaller in cold-acclimated rats, with concomitant increases in capillarity (Suzuki et al., 1997). Similarly, Bae et al. (2003) showed that cold-acclimated breath-hold divers possessed higher skeletal muscle capillary density, a lower oxygen diffusion distance and a smaller muscle fiber CSA, whist no such adaptations were evident in breath-hold divers who dived at moderate water temperatures (29–30°C) (Park et al., 2005).

While we rationalize that the dampened increase in muscle mass observed following CWI is a compensatory mechanism improving oxidative function, further research is needed to understand how this might influence athletic function and performance. For instance, it is currently unknown if CWI influences the regulation of muscle mass following aerobic exercise, and whether this hypothetical trade-off involving the attenuated increase in muscle mass and strength might be beneficial to endurance performance. On the other hand, co-assessment of muscle aerobic function within these resistance training studies (Frohlich et al., 2014; Roberts et al., 2015; Fyfe et al., 2019; Poppendieck et al., 2020) would have furthered our understanding of the functional consequence of the dampened increase in mass coupled with increased capillarity.

CWI AND RESISTANCE TRAINING: INSIGHTS FROM APPLIED RESEARCH

Athletes embark on a variety of training sessions such as cardiovascular conditioning, strength/resistance training, technical, and tactical work. In sports science practice, CWI is likely to be incorporated at various instances to promote recovery, particularly when recovery time between sessions is limited. Caution should be warranted against the regular use of CWI particularly following resistance exercise sessions.

Recent work (**Table 1**) examining the longer-term effects of CWI on training performance and recovery amongst professional and semi-professional athletes provides invaluable insights regarding CWI programming and recovery-adaptation interaction throughout training/competition phases (Lindsay et al., 2016; Tavares et al., 2019, 2020; Seco-Calvo et al., 2020). These studies collectively demonstrate no impairments in strength gains despite administering frequent post-exercise CWI over 2.5 weeks to 8 months. In contrast to the current literature (Frohlich et al., 2014; Roberts et al., 2015; Fyfe et al., 2019; Poppendieck et al., 2020), most of these studies (**Table 1**) report a trend for improved strength gains over the training/study period. Such divergent findings are hard to reconcile. One possibility, as Broatch et al. (2018) highlighted is that laboratory-based experimental studies are designed with 2–3 sessions per week permitting adequate recovery between sessions, and by extension not capitalizing on the recovery effects of CWI.

Training frequency reported within these applied studies surmounts to at least 10 sessions per week (**Table 1**). Perhaps, in scenarios where recovery between training sessions may be limited, CWI can improve training performances and consequently the stimulus for adaptation. In support, post-exercise CWI has been shown to enhance the ability to perform more volitional work during subsequent squat exercise (Roberts et al., 2014), or better maintain day-to-day exercise performance during intensified periods of endurance training (Vaile et al., 2008b; Stanley et al., 2013). Given that adaptations to exercise stimulus are volume and intensity dependant, it seems reasonable to consider that the recovery benefits of CWI (and resultant increase in training quality) might outweigh its dampening effects on hypertrophy response. Conversely, it can be argued that anabolic adaptations are better enhanced if CWI is avoided, albeit this might deter the quality of subsequent training sessions. It is currently unknown which approach would better influence athletes' recovery-adaptation interaction. Longer term applied studies similar to those highlighted in this review (**Table 1**) will significantly contribute to our understanding in this area.

Another key feature of these studies (**Table 1**), and perhaps within sport science practice is that CWI is often not administered immediately following a resistance training session, but instead following technical/tactical or conditioning sessions. These findings show promise that beneficial recovery outcomes can be harnessed whilst avoiding negative effects of CWI on

TABLE 1 | Summary of studies examining the longer-term effect of CWI on the recovery of exercise performance.

Study	Level/Sport	Training Phase/Duration	Training description	CWI frequency and protocol	CWI Timing	Main outcomes
Lindsay et al., 2016	Semi-professional MMA athletes	6-week pre-competition training camp	Strength and conditioning (60–90 min, 3x/week), MMA, wrestling, jiu-jitsu, and boxing (90–120 min, 7x/week)	3x/week whole body CWI @ 10°C for 15 min	Performed following last session of the day which consisted of MMA or wresting training	Similar improvements in SBJ, pull-ups, and press-ups in CON vs. CWI group
Tavares et al., 2019	Elite Rugby Union	3 weeks during pre-season period	Strength sessions (4x/week), technical/tactical sessions (7x/week), speed, and conditioning (5x/week)	4x/week whole body CWI @ 10°C for 10 min	Performed following afternoon sessions which consisted of technical/tactical or conditioning	Better maintenance in CMJ performance in CWI group
Seco-Calvo et al., 2020	Professional Basketball players from the Spanish Premier League	Competitive season (8 months)	Gym sessions (4x/week), conditioning (3x/week), speed, and reaction (2x/week)	4x/week whole body CWI @ 10°C for 5 × 2 min	Performed following the speed and conditioning or following match-play	Better maintenance of shoulder strength
Tavares et al., 2020	U21 Portuguese national players	2.5-week pre-competition training camp	10 resistance training and 19 on-court sessions over 2.5 weeks	CWI @ 10°C for 10 min after last training session	Performed following on-court volleyball sessions	Better maintenance in CMJ performance in CWI group

MMA, mixed martial arts; SBJ, standing broad jump; CMJ, counter-movement jump.

strength gains by simply avoiding the use of this recovery modality in the proximity of resistance training sessions. Regardless, we acknowledge that these studies were not specifically designed to address this notion. Moreover, the majority of these studies were relatively short-term (2.5–6 weeks). Specific, longer-term studies are therefore required to address the effect of CWI timing on strength adaptation.

SUMMARY AND PERSPECTIVES

Cold water immersion is widely utilized by athletes during training and competition. Given that both a cold stimulus and exercise are independent stressors capable of enhancing muscle oxidative function, there remains substantial interest in examining how this modality might influence adaptations to exercise. Although post-exercise CWI up-regulates mitochondrial-related signaling, longer-term changes in protein content and result in vascular adaptations, these changes do not seem to translate to improved endurance performance. As such, further research is required to elucidate how endurance performance can be improved through its positive molecular signaling outcomes for CWI to be incorporated to enhance exercise-induced oxidative adaptations. It must be re-iterated that CWI does not impair aerobic training adaptations, and can be incorporated as a recovery modality following endurance training if needed. In contrast, regular CWI recovery incorporated into a resistance training program will dampen strength adaptations, and therefore the use of this modality following resistance exercise sessions should be discouraged. However, there is emerging data showing no impairments in strength gains in athletes incorporating regular use of CWI during intensified training periods; this either indicates that the recovery benefits conferred by CWI may outweigh its dampening effects on hypertrophy response, or the negative effects of CWI on strength may be circumvented by programing CWI following technical or aerobic conditioning sessions. In this regard, "recovery periodization" may be an important approach, where the use of CWI may be incorporated during competition or intensified training, whilst strategically avoided following training focused on improving muscle strength or hypertrophy.

AUTHOR CONTRIBUTIONS

All authors listed have made a substantial, direct and intellectual contribution to the work, and approved it for publication.

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frontiers in Sports and Active Living

OPINION
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Post-exercise Recovery: Cooling and Heating, a Periodized Approach

Robin T. Thorpe [1,2]*

[1] Football Exchange, Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Liverpool, United Kingdom, [2] College of Health Solutions, Arizona State University, Phoenix, AZ, United States

Keywords: recovery, fatigue, exercise, injury, cooling, heating, muscle damage (DOMS), periodization

RISING IMPORTANCE OF RECOVERY

Recovery is regarded as a multifaceted (e.g., physiological, psychological) restorative process relative to time and modulated by external load, individual response to stress, and often dictated by external athletic competition and demand (Kellmann et al., 2018). The increasing physical demands of athletic competition, particularly, team sports (Barnes et al., 2014), involving high fixture frequency, has further exacerbated the physical and mental load placed on athletes (Ekstrand et al., 2018). Athletes are now routinely exposed to longitudinal demands with, in some cases, only 48 h of recovery time between competitions. Fatigue may be defined as "an inability to complete a task that was once achievable within a recent time frame" (Pyne and Martin, 2011; Halson, 2014) and derived from central and/or peripheral origins. Recovery time between successive competitions may be insufficient to allow athletes to fully regenerate leading to fatigue, which may increase the risk of under-performance, non-functional overreaching, injury, and illness (Dupont et al., 2010; Bengtsson et al., 2013). Demands are further increased in athletes competing in continental leagues, play-off phases, international tournaments, and are further aggravated in circumstances such as the English Premier League that does not include a winter break (Ekstrand et al., 2018) or in recent times the effect of the COVID-19 pandemic (Seshadri et al., 2021). Increased athlete training and competition availability as a result of a reduction in injuries, substantially improves the likelihood of success of an individual or team (Hägglund et al., 2013). Changes in injury occurrence also have a significant impact, particularly, financial implications (team underachievement and player salaries) of sporting organizations due to injury-related decrements in performance (Eliakim et al., 2020). Growing demands and the rising importance of improving recovery have also prompted athletes to inclusively invest in further bespoke personal support in an attempt to accelerate recovery.

EVIDENCE AND PRACTICE: A CONFUSING LANDSCAPE

A certain degree of fatigue, resulting in functional overreaching, is required to mediate adaptations to training, which drive performance enhancement (Noakes, 2000). However, excessive fatigue through insufficient recovery may increase susceptibility to non-functional over-reaching, injury, and illness of the players (Nimmo and Ekblom, 2007). Fatigue can be compensated with recovery strategies which serve to restore homeostasis on a physiological and psychological level (Kellmann, 2002). Researchers and practitioners alike have investigated the efficacy of commonly utilized interventions to combat the deleterious effects of athletic training and competition (Barnett, 2006; Howatson and van Someren, 2008; Nédélec et al., 2012; Dupuy et al., 2018). A recent investigation (Altarriba-Bartes et al., 2020) reviewing commonly used recovery strategies in professional soccer found that all teams were utilizing at least one recovery strategy following games; however, the range of interventions used was substantially different between teams with water immersion (cold

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Edited by:
Robert Allan,
University of Central Lancashire,
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Reviewed by:
James Malone,
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United Kingdom
Chris Mawhinney,
Mahidol University, Thailand
Susan Y. Kwiecien,
Nicholas Institute of Sports Medicine and Athletic Trauma (NISMAT),
United States

***Correspondence:**
Robin T. Thorpe
robin.thorpe@live.co.uk

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and hot), massage, and foam rolling representing 74, 70, and 57% respectively (Altarriba-Bartes et al., 2020).

It is imperative that the origin of fatigue is understood in order to most effectively return the human body to homeostasis following exercise. Furthermore, an understanding of the origin of fatigue may help with tailoring an appropriate recovery strategy to enhance the accelerated return to homeostasis. Recovery time from training-induced stress may differ within and between the different organismic systems of the human body (Kellmann et al., 2018). The increased focus on athlete recovery within professional sport has naturally been followed by many scientific investigations attempting to understand the efficacy of a range of commonly performed strategies (Howatson and van Someren, 2008; Leeder et al., 2012). However, few studies have been able to demonstrate the efficacy of strategies improving recovery in athletes following training or competition (Bieuzen et al., 2013; Hill et al., 2014; Dupuy et al., 2018; Davis et al., 2020).

Much of the positive evidence for recovery strategies lies with an enhanced perceptual outcome of recovery, often attributed to an athlete's belief in the modality or the placebo effect (Broatch et al., 2014; Wilson et al., 2018). Indeed, evidence exists whereby recovery strategies have not improved fatigue levels further than that of the placebo effect (Cook and Beaven, 2013; Broatch et al., 2014; Malta et al., 2019). Research has traditionally focused on administering one recovery intervention at a time, whereas in the applied setting athletes are more likely to administer multiple interventions in varying sequences due to the many strategies available, of which many lack efficacy (Costello et al., 2016; Davis et al., 2020; Skorski et al., 2020). Although extensive, the existing literature base investigating recovery strategy efficacy still lacks clarity and directional influence for practitioners and athletes alike. Much of the data involves study designs investigating changes in physical performance and perceptual or muscle damage markers following an exhaustive protocol or athletic competition (Leeder et al., 2012; Davis et al., 2020). These methodological variances alongside less realistic laboratory protocols detached from contextual performance and investigation of only the acute recovery response (0–72 h), including sub-elite subject cohorts may be some of the reasons why inconclusive data exists (i.e., sole strategies performed across entire recovery continuum), indirectly, creating confusion for practical application. Movement toward a more periodized research design approach has occurred where multiple strategies have been assessed in an attempt to improve recovery (Martínez-Guardado et al., 2020; Pooley et al., 2020). Reasons for applying multiple modalities may arise from the fact that athletes are now exposed to a variety of strategies and professional philosophy, proposed to enhance recovery, rather than a physiology-based rationale. Athletes performing multiple strategies rather than a singular modality may be a step forward; however, a more critical, evidence-based reasoning for the application of periodizing varying strategies is required. A better understanding of the exact physiological systems and mechanisms of fatigue may provide a clearer landscape into unraveling recovery from exercise, performance, and injury.

MATCHING THE STRESS AND INTERVENTION: MONITORING-BASED PRACTICE

Physical demands of both individual and team sports involve varying contributions of metabolic and mechanical stress to tissue. Mechanical stress deriving primarily from eccentric contractions results in a temporary reduction in muscle function, an increase in intracellular proteins in the blood, an increase in perceptual muscle soreness, and evidence of swelling (Howatson and van Someren, 2008). Thereafter, secondary damage is linked to the subsequent inflammatory response and macrophage and neutrophil infiltration which, further, in isolation compromise the mechanically stressed area (Merrick, 2002). Metabolic factors such as reductions in adenosine triphosphate (ATP), creatine phosphate, glycogen (Krustrup et al., 2006), and pH (Brophy et al., 2009) may also induce fatigue following exercise. Biochemical changes in electrolytes and calcium may also have negative effects alongside hypoxia at the muscle cell level contributing to metabolic fatigue. Mechanical stress and/or metabolic fatigue may also contribute to neuromuscular cost *via* altered muscle potassium and pH levels (Tee et al., 2007) and excitation contraction coupling, respectively (Jones, 1996). Environmental factors and exercise-induced heat generation (Arbogast and Reid, 2004), which increases the concentration of nicotinamide adenine dinucleotide phosphate oxidase within the muscle fiber resulting in an increase in the production of reactive oxygen species from the mitochondria and from the infiltrating inflammatory cells (Powers and Jackson, 2008) further exacerbating potential mechanical damage. The variance in physiological origin associated with exercise and competition infers that it is illogical that a single recovery strategy and/or a generic "one size fits all" approach would accelerate each of the systems discussed (Minett and Costello, 2015). Evidence exists where a singular temperature-based strategy applied locally to the quadriceps over the entire recovery continuum failed to further accelerate recovery beyond the acute period (0-72 h), *albeit*, following severe marathon running–derived mechanical and metabolic stress (Kwiecien et al., 2020a,b). Moreover, Petersen and Fyfe (2021) suggested, from a chronic perspective, long-term application of a singular intervention may have disadvantages relating to adaptation (Petersen and Fyfe, 2021). It appears that a binary perspective to recovery has arisen within the literature, which in turn may have influenced the applied setting. Alternatively, a framework where strategies are periodized to match the individual symptoms, organismic fatigued system, external load or the response to stress may be a more preferred approach (Thorpe et al., 2017; Kellmann et al., 2018). Indeed, monitoring of recovery or the response to load may provide insights into the exact physiological stress an athlete is currently experiencing. A recent review stated that the quantification of physiological stress *via* athlete response outcome measures, athlete self-report, heart rate-derived autonomic nervous system, neuromuscular functional jump/eccentric/concentric/isometric protocols, biochemical/immunological/endocrine, and joint range of motion could improve practical prescription of

modalities in enhancing recovery (Thorpe et al., 2017). For example, assessing changes in perceived muscle soreness or the autonomic nervous system *via* heart rate-derived metrics (heart rate variability and/or heart rate recovery) may establish whether or not an athlete is experiencing symptoms associated with mechanical damage (Dupuy et al., 2018), thus a gateway to understanding and quantifying which strategies may be most appropriate for improving this fatigued system. Attention ought to be prioritized to framework strategies that match the associated physiological stress along the recovery continuum in a systematic manner.

TEMPERATURE-DERIVED APPROACH: PERIODIZING COOLING AND HEATING

Beyond sleep, nutrition, and hydration, recent work has focused on the application of various temperature-based modalities in an attempt to accelerate recovery (McGorm et al., 2018; Kwiecien and McHugh, 2021). Indeed, among the vast array of recovery strategies commonly used by athletes, temperature-based modalities have shown the most promise, although, still the data are inconclusive (Jakeman et al., 2009; Stanley et al., 2012; Broatch et al., 2014). One of the most common recovery strategies used is cryotherapy, or the application of cooling (Altarriba-Bartes et al., 2020). Cooling has been performed for decades in relation to injury, and intuitively, transferred to recovery from exercise in more recent times. Topical cooling, cold water immersion, whole body cryotherapy, and more recently phase change material are most commonly used in both the clinical and professional sports settings (Kwiecien and McHugh, 2021). The mechanistic response between these modalities has been shown to differ and in some circumstances provides a completely different physiological effect (Mawhinney et al., 2017; Kwiecien and McHugh, 2021). The ultimate objective for cooling is to reduce deep muscle temperature, in an attempt to favorably reduce blood flow and metabolism at the affected muscle site, in an effort to diminish the secondary damage phase (Merrick, 2002). A recent review suggested that repeat application or elongating cooling time would lead to the most advantageous results in reducing deep muscle temperature, in turn, the proliferation of the secondary damage phase (Kwiecien and McHugh, 2021). Importantly, local changes in muscle temperature (cooling or heating) may influence enzymatic activity and effect rates of intramuscular glycogen resynthesis (Cheng et al., 2017). Indeed, research has also investigated the effects of heating regarding performance, adaptation, and to a lesser extent recovery (McGorm et al., 2018). Heat therapy including hot water immersion has not been widely investigated in terms of athletic recovery, although, anecdotally performed frequently in athletes across many sports (Altarriba-Bartes et al., 2020). Data exists supporting heating for stimulating local blood supply and metabolism in tissues, and emerging evidence indicate that heat activates more specific molecular events, including changes in gene expression, anti-inflammatory and antioxidant effects, glycogen resynthesis, mitochondrial biogenesis, heat shock protein expression, and cellular healing (Hoekstra et al., 2008; McGorm et al., 2018; Nadarajah et al., 2018). Data from animal and human studies have shown metabolic-based recovery to be accelerated following heat application which in turn modified the release of tetanic $[Ca^{2+}]$ and glycogen resynthesis rates compared to cooling (Cheng et al., 2017). Considering the existing evidence of the possible recovery kinetics to both cooling and heating, it appears that increasing or decreasing tissue temperature may provide advantageous responses at varying points on the recovery continuum, which are associated to mechanical damage and metabolic fatigue.

A PRACTICAL GUIDE

An array of different strategies are used by athletes in an attempt to alleviate the deleterious symptoms associated with exercise and competition (Nédélec et al., 2013; Altarriba-Bartes et al., 2020). However, there is a lack of consensus in how to design and prescribe strategies in improving the multifactorial systems of recovery. It appears that the first and most critical physiological event to attempt to mediate is the secondary damage phase shortly following mechanical damage. The latest evidence suggests that prolonged cooling is the most suitable intervention (Kwiecien and McHugh, 2021). Cooling *via* water immersion (in some cases multiple exposures) or local phase change material has been shown to have the most effective results in reducing tissue temperature (Mawhinney et al., 2017; Kwiecien et al., 2020b). Hereafter, and to promote removal and enhanced transportation of metabolic byproducts, and possible modulation of cellular healing, hemodynamics and substrate resynthesis (McGorm et al., 2018) heating is preferred *via* sauna microwave diathermy, water-perfused garments, hot water immersion, or steam/heat sheets (Hyldahl and Peake, 2020). This proposed framework (**Figure 1**) may be individualized based on the proportionate expense of mechanical and metabolic fatigue and whereby increasing or decreasing tissue temperature beyond purely an individualized approach, and when response to load/fatigue monitoring is limited (Thorpe et al., 2017), periodizing strategies to not only consolidate recovery across a training period but also in an attempt to enhance adaptation is proposed. Indeed, sequencing cooling strategies following endurance dominant stress or heating strategies following strength-derived stress may induce advantageous gene expression-related adaptations (Allan et al., 2017; Cheng et al., 2017; Hyldahl and Peake, 2020). The role of cooling and heating modalities should be chosen in reflection of external physical demand and matched accordingly to negate any contraindicative effect to adaptation interactions (Peake et al., 2020).

There is a clear physical and mental stress induced by exercise, competition, and acute injury. A unique physiological and immunological cascade then ensues. Identifying the different and proportionate mechanistic alterations is paramount in order to mitigate against further reduced performance, injury, and illness risk. Prioritizing sleep, rest, nutrition, hydration, and joint range of motion during this phase is fundamental, thereafter,

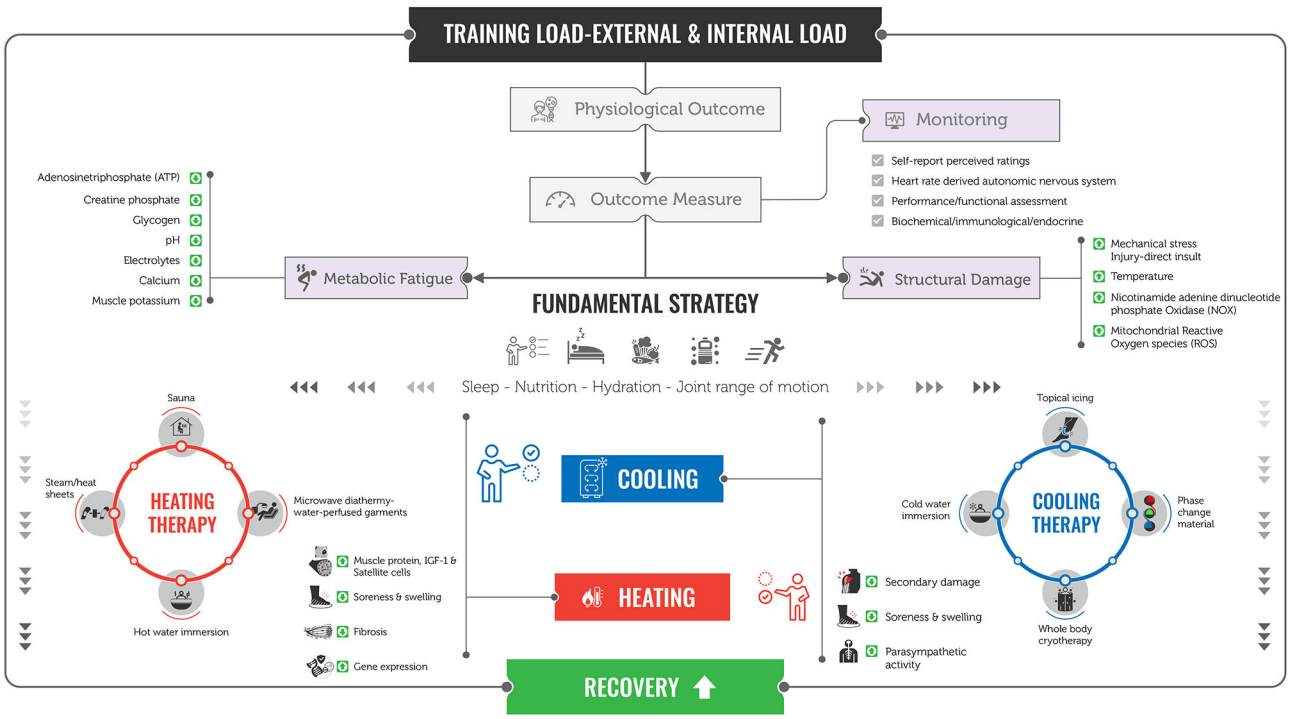

FIGURE 1 | Practical framework to enhance recovery in athletes. Following external and internal load–stress response outcome measures, including, self-report [soreness (DOMS), perceived fatigue, sleep quality]; heart rate-derived autonomic nervous system [heart rate variability (HRV), heart rate recovery (HRR), submaximal heart rate (HRex)]; performance/neuromuscular functional assessment (force–time jump, eccentric, concentric, isometric protocols); and biochemical/immunological/endocrine (creatine kinase, IL-6, C-reactive protein) may be used to distinguish limiting components of physiological fatigue systems {structural damage [mechanical stress, direct insult, *increases in* temperature and nicotinamide adenine dinucleotide phosphate oxidase (NOX), and mitochondrial reactive oxygen species (ROS)] and metabolic fatigue [*fluctuations in* adenosine triphosphate (ATP), creatine phosphate, glycogen, pH, electrolytes, calcium, and muscle potassium]} following exercise and injury. Following an initial fundamental strategy (*optimal* sleep, nutrition, hydration, and joint range of movement) the use of cooling (topical icing/phase change material, cold water immersion, whole body cryotherapy) and heating (sauna, microwave diathermy water-perfused garments, hot water immersion or steam/heat sheets) strategies may be used systematically and independently to match the stress to assist in alleviating structural damage (secondary damage, soreness, swelling, and parasympathetic reactivation) and/or metabolic fatigue (muscle protein, IGF-1, satellite cells, soreness, swelling, fibrosis, and gene expression), respectively.

recovery interventions should be considered that alleviate the particular physiological stress incurred at any given time point on the recovery continuum (Kellmann et al., 2018). Reducing tissue temperature *via* cooling has shown to mediate secondary damage derived from mechanical stress (Merrick, 2002), whereas heating has been shown to enhance tissue temperature, blood flow, and metabolism alleviating metabolic-associated fatigue (McGorm et al., 2018). Identifying origins of fatigue *via* the use of practical monitoring processes is recommended for individualization of recovery strategy prescription (Thorpe et al., 2017). In the absence of fatigue monitoring, a generic approach in which reducing secondary damage *via* cooling as the initial strategy followed by heating once the inflammatory cascade diminishes is recommended because of the timeline and functional detrimental properties of this process. The utilization of cooling and heating to navigate and facilitate the associated perturbations may be considered appropriate to accelerate recovery *via* the different physiological demands in athletes. A periodized, systematic recovery process matching appropriate thermoregulatory strategies to associated physiological systems should be considered as a framework to enhance recovery in athletes.

AUTHOR CONTRIBUTIONS

The author confirms being the sole contributor of this work and has approved it for publication.

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Post-exercise Cold Water Immersion Does Not Improve Subsequent 4-km Cycling Time-Trial Compared With Passive and Active Recovery in Normothermia

Mikel Egaña[1], Lynn Allen[2], Kate Gleeson[1], Norita Gildea[1] and Stuart Warmington[3]*

[1] Department of Physiology, School of Medicine, Trinity College Dublin, The University of Dublin, Dublin, Ireland, [2] Faculty of Science & Health, Athlone Institute of Technology, Athlone, Ireland, [3] Institute for Physical Activity and Nutrition, School of Exercise and Nutrition, Deakin University, Geelong, VIC, Australia

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***Correspondence:**
Mikel Egaña
megana@tcd.ie

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Background: We investigated whether a brief cold water immersion between two cycling time trials (TT) improves the performance of the latter compared with passive and active recovery in normothermic conditions (∼20°C).

Methods: In *Experiment 1* 10 active participants (4 women) completed two 4-km TT (Ex1 and Ex2, each preceded by a 12 min moderate-intensity warm-up) separated by a 15 min recovery period consisting of: (a) passive rest (PAS) or (b) 5 min cold water immersion at 8°C (CWI-5). In *Experiment 2,* 13 different active males completed the same Ex1 and Ex2 bouts separated by a 15 min recovery consisting of: (a) PAS, (b) 10 min cold water immersion at 8°C (CWI-10) or (c) 15 min of moderate-intensity active recovery (ACT).

Results: In both experiments, the time to complete the 4-km TT-s was not different ($P > 0.05$, ES = 0.1) among the trials neither in Ex1 (*Experiment 1*: PAS: 414 ± 39 s; CWI-5: 410 ± 39 s; *Experiment 2*: PAS: 402 ± 41 s; CWI-10: 404 ± 43 s; ACT: 407 ± 41 s) nor Ex2 (*Experiment 1*: PAS: 432 ± 43 s; CWI-5: 428 ± 47 s; *Experiment 2*: PAS: 418 ± 52 s; CWI-10: 416 ± 57 s; ACT: 421 ± 50 s). In addition, in all conditions, the time to complete the time trials was longer ($P < 0.05$, ES = 0.4) in Ex2 than Ex1. Core temperature was lower ($P < 0.05$) during the majority of Ex2 after CW-5 compared with passive rest (*Experiment 1*) and after CWI-10 compared with PAS and ACT (*Experiment 2*). Perceived exertion was also lower ($P < 0.05$) at mid-point of Ex2 after CWI-5 compared with PAS (*Experiment 1*) as well as overall lower during the CWI-10 compared with PAS and ACT conditions (*Experiment 2*).

Conclusion: A post-exercise 5–10 min cold water immersion does not influence subsequent 4-km TT performance in normothermia, despite evoking reductions in thermal strain.

Keywords: recovery, time trial, hydrotherapy, cycling, core temperature

INTRODUCTION

In a variety of sporting events such as track cycling or athletic events, participants are often required to compete more than once on the same day during competitions. For instance, in track cycling, where events range from a 200 m flying sprint (lasting ∼10s) to the 50 km points race (lasting ∼1 h), cyclists often compete more than once per day, such as in the Omnium, a multi-race event, typically comprising of four different races held on the same day, having at times <1 h between events. These events are performed at a very high intensity and result in fatigue. Therefore, an optimal recovery strategy may play a significant role to ameliorate the decline in performance during these subsequent events. Cold water immersion (CWI) is one such strategy increasingly being employed by athletes, having been shown to be efficacious when applied between two equal bouts of high-intensity continuous or intermittent endurance exercise to elicit a superior performance in the second bout, both in normothermic (Crampton et al., 2013; Dunne et al., 2013; Mccarthy et al., 2016; Stephens et al., 2018; Egaña et al., 2019) and hyperthermic conditions (Yeargin et al., 2006; Peiffer et al., 2010). The beneficial effect of CWI appears to outperform other frequently used recovery strategies, such as contrast water therapy, active recovery or thermoneutral water immersion (Vaile et al., 2008; Crampton et al., 2013). Although the mechanisms governing the effects of CWI on subsequent performance remain to be elucidated, potential mediators include increased heat storage capacity (Kay et al., 1999; Marsh and Sleivert, 1999), increased venous return in response to the cold stimulus or hydrostatic pressure of the immersion (Wilcock et al., 2006), reactivation of cardiac parasympathetic activity (Stanley et al., 2012) and/or reduced perception of effort (Mccarthy et al., 2016).

However, the "same-day" performance effects subsequent to post-exercise CWI are still inconclusive due to methodological variations. For instance, all-out sprint cycling performance has been shown to be reduced after short-term, post-exercise CWI (Schniepp et al., 2002; Crowe et al., 2007; Crampton et al., 2014) likely owing to compromised contractile capabilities of cooled muscles (Bergh and Ekblom, 1979; Bigland-Ritchie et al., 1992; Crampton et al., 2011). By contrast, when incorporating upper-body arm-cranking exercise during lower-body CWI, all-out sprint cycling capacity in a subsequent bout is improved when compared against a lower-body CWI alone (Crampton et al., 2014). This was because core temperature (T_{core}) was maintained during the active CWI recovery, likely enhancing the neurophysiological mechanisms that drive muscle activation compared with passive CWI. Although, therein un-immersed active recovery preserved sprint performance whereas both passive and active CWI recoveries did not (owing to CWI-induced muscle cooling and the subsequent afterdrop response). On the other hand, some studies followed CWI with extended recovery periods exceeding 1–2 h prior to the subsequent exercise bout (Versey et al., 2011; Stanley et al., 2012). Despite the applied nature of these experimental protocols (i.e., by way of simulating training and competing twice in 1 day, such as in track cycling), inter-individual influences other than water immersion (i.e., warm-down, stretching or passive rest) may affect the subsequent performance.

When the effect of post-exercise CWI has been explored on subsequent high-intensity endurance exercise performance completed immediately after the recovery protocol under normothermic conditions (∼19–20°C), and thus, when recovery is not complicated by other influences, the time to failure during high-intensity constant-load exhaustive efforts is enhanced both, during cycling (Crampton et al., 2013) as well as running (Dunne et al., 2013). Furthermore, CWI evokes benefits during subsequent intermittent exhaustive high-intensity exercise (Mccarthy et al., 2016) as well as intermittent sprint protocols that mimic the typical sprint characteristics and metabolic demands of many team sport games (Egaña et al., 2019). Under hot ambient conditions (35°C), it was shown that a 5 min CWI employed after a 4-km cycling time trial (TT) significantly reduced the completion time of a subsequent 4-km TT (both TT-s preceded by a 25 min moderate-intensity exercise bout) compared with a control (passive rest) condition (Peiffer et al., 2010), while Yeargin et al. (2006) demonstrated that a post-exercise 12-min CWI improved the subsequent 2 mile running time trial.

To the best of our knowledge the effect of CWI on subsequent time trial performance has not been explored under normothermic ambient conditions when the exercise is performed immediately after the immersion. Accordingly, the aim of the present study was to compare the effects of post-exercise brief cold water immersions (5- and 10-min durations) with both passive rest and active recovery interventions on subsequent 4-km TT performance that was preceded by a 12 min moderate-intensity exercise bout. In attempting to explore the mechanistic basis of any CWI-induced effects on subsequent performance (i.e., 4-km completion time), T_{core}, heart rate (HR) and ratings of perceived exertion were assessed. It was hypothesized that compared with passive and active recovery control conditions, CWI interventions would improve subsequent 4-km TT performance.

MATERIALS AND METHODS

Participants

Two experiments were performed. *Experiment 1* tested the effect of post-exercise 5 min CWI on a subsequent 4-km cycling TT compared with passive recovery, whereas *Experiment 2* tested the effect of post-exercise 10 min CWI on a subsequent 4-km cycling TT compared with passive as well as active recovery.

Ten (4 women) active participants (mean ± SD; age: 21 ± 1 year; height: 178 ± 8 cm; body mass: 71 ± 11 kg, peak oxygen uptake ($\dot{V}O_{2peak}$): 48.1 ± 7.8 ml·kg^{-1}·min^{-1}, peak power (PO_{peak}): 282 ± 86 W) who were accustomed to recreational cycling took part in *Experiment 1.* They visited the Human Performance Laboratory in the Department of Physiology of the Institution, on 3 days separated by at least 72 h. Thirteen different young men (mean ± SD; age: 29 ± 7 year; height: 181 ± 7 cm; body mass: 80 ± 12 kg, $\dot{V}O_{2peak}$: 51.2 ± 15.6 ml·kg^{-1}·min^{-1}, PO_{peak}: 348 ± 53 W) also accustomed to recreational cycling participated in *Experiment 2,* whereby they visited the same

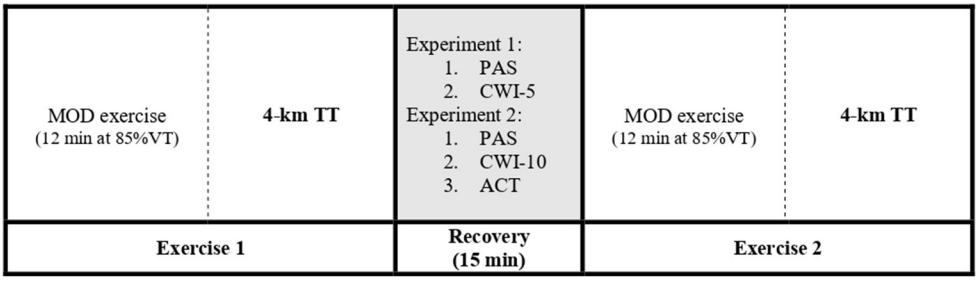

FIGURE 1 | Timeline of experimental protocol. In *Experiment 1* the recovery treatments included passive recovery (PAS) and 5 min cold water immersion (CWI-5); in *Experiment 2*, passive recovery (PAS), 10 min cold water immersion (CWI-10), and active recovery (ACT). VT, first ventilatory threshold; MOD, moderate-intensity cycling exercise; TT, time trial.

laboratory on four separate occasions (at least 72 h apart). All participants were free from any medical conditions, (assessed by medical questionnaire and physical examination) and were non-smokers. All participants provided written informed consent prior to participation. The study was approved by the Faculty of Health Science Research Ethics Committee of the Institution and carried out in accordance with the Declaration of Helsinki.

Experimental Protocol

Overview

An overview of the experimental protocol is shown in **Figure 1**. In both experimental conditions, *Experiment 1* and *2*, participants performed a preliminary incremental cycling test and familiarization to the CWI and 4-km TT test (visit 1). Thereafter, in *Experiment 1* participants were required to carry out 2 separate randomized trials (visits 2–3) separated by a minimum of 2 days, and in *Experiment 2*, three separate randomized trials separated by a minimum of 2 days (visits 2–4). Each experimental trial required the participants to complete an exercise bout (Ex1: 12 min of moderate-intensity cycling + a 4-km TT) followed by a randomized 15 min recovery period and a subsequent second identical exercise bout (Ex2).

All testing sessions within each experimental protocol were completed over a course of 7 weeks, with *Experiment 2* being carried out several weeks after *Experiment 1* was completed. Participants were asked to ensure that weekly training regimens were similar and maintained throughout this time frame. During visit 1 to the laboratory, all participants completed a 24 h food and fluid recall diary, which they were asked to replicate as closely as possible in the 24 h prior to subsequent experimental sessions. In addition, they were instructed to consume a meal consisting of approximately 200 g of carbohydrate 3 h prior to all experimental sessions. Adequate hydration status, i.e., within the accepted euhydration ranges 1,000 and 1,020, was ensured at the start of each visit by measuring urine specific gravity using an optical refractometer (Bellingham & Stanley, Hants, UK). In an effort to limit diurnal fluctuations in T_{core}, fatigue and overall exercise capacity, all experimental sessions were held at the same time of day. Participants were asked to refrain from exercise training for at least 12 h and to avoid alcohol and caffeine consumption for 24 h prior to each visit. The experimental exercise sessions were performed in the upright position using a cycle ergometer (*Experiment 1*: Wattbike, Nottinghamshire, UK; *Experiment 2*: Excalibur Sport, Lode, Groningen, The Netherlands). In *Experiment 1* the gearing was self-selected by the participants on the Wattbike during the practice trial and then replicated during each TT. In *Experiment 2* the Lode ergometer was set to linear mode so that with increasing pedaling rate the work rate increased. During all experimental sessions the ambient temperature of the laboratory wherein both the exercise and recovery protocols were carried out was held constant (20 ± 1°C). In addition, during the exercise protocols, participants were cooled with a 300-mm diameter fan (Micromark, UK) placed 1 m in front of them that produced an air flow equivalent to 3 $km.h^{-1}$.

Graded Incremental Test and Familiarization

All participants performed a graded incremental test to failure to determine $\dot{V}O_{2peak}$ and the first ventilatory threshold (VT) on an upright cycling ergometer (for both experiments: Excalibur Sport, Lode, Groningen, The Netherlands). After a 3 min period of rest in a seated position, the test commenced with participants cycling at 30W for 1 min, with incremental increases of 20W (women) or 30W (men) every min until task failure. The VT was determined using the V-slope method (Beaver et al., 1986). After the graded incremental test, participants were familiarized with the constant-load bout and 4-km TT cycling protocol and cold water immersions.

Experimental Trials

Exercise Bouts (Ex1 and Ex2)

Each of the experimental sessions was comprised of two identical exercise protocols (Ex1 and Ex2) separated by a 15 min recovery interval. The exercise protocol consisted of a 3 min "baseline" cycling period at 10W followed by a 12 min constant load cycle at 85% of each participants VT, a 2 min seated rest period, and finally a 4-km cycling TT. A 4-km TT test was chosen because it has been shown to be a reproducible test (Stone et al., 2011). Participants were instructed to complete the 4-km cycling TT in as fast a time as possible whilst receiving visual feedback for distance completed, but all were blinded to their exercise times. The 12 min moderate-intensity constant-load bouts, which were

initiated from a 3 min baseline at 10W, were carried out to warm up the active musculature.

Recovery Interventions

All recovery interventions were performed in a balanced randomized order throughout the study. On each testing day of *Experiment 1,* one of the following recovery interventions were performed: (a) passive un-immersed rest in a seated position (PAS) and (b) 5 min CWI at 8°C (CWI-5). On testing days of *Experiment 2* the following recovery interventions were performed: (a) PAS, (b) 10 min CWI at 8°C (CWI-10) and (c) active recovery (ACT) comprising cycling at 40% VO_{2peak}.

Between min 5 to 10 of the CWI-5 trial, participants were immersed in a custom-built bath (Sturdy Products, Co. Wicklow, Ireland) positioned next to the cycling ergometer. Five min periods of transition from the ergometer to the bath, as well as from the bath to the ergometer were allowed. These transition times however were shortened in CWI-10 testing trials to 2.5 min to allow for the longer immersion time where participants were immersed between 2.5 and 12.5 min. During the transitioning period to the bath, participants removed their cycling shoes, training top, shorts and socks and changed into swimwear. During the second transition, participants were provided with towels to dry themselves prior to redressing for Ex2. Any surplus time during the transition periods was spent passively resting in a seated position on a stool. During the recovery treatments, participants remained upright in a seated position, with their backs placed firmly against the posterior wall of the bath and feet against the anterior wall with their knees flexed (∼90°) to ensure full leg immersion. During the passive condition, participants sat in this same position, albeit in an empty bath. During the periods of immersion, the water remained relatively stagnant, and at approximately sternal level for each participant to evoke a significant muscle (and core) cooling effect, without drastically influencing core temperature, as can occur with deeper (i.e., neck level) immersion (unpublished observations). The 8°C water temperature utilized herein was selected given its frequent application in water immersion protocols for recovery post-exercise (Wilcock et al., 2006). Furthermore in similar normothermic conditions, in comparison to passive rest, cold water immersion at 8°C has been shown to better enhance subsequent sustained running performance than with cold water immersion at 15°C (Dunne et al., 2013). The 5 min CWI was chosen because it has previously been shown to evoke significant benefits in hyperthermic conditions during subsequent 4 km TT, as well as in normothermic conditions during brief (6–8 min) intermittent exhaustive high-intensity exercise protocols (Mccarthy et al., 2016) and prolonged (40 min) intermittent sprint protocols designed to mimic activity patterns of team-sports (Egaña et al., 2019). The 10 min duration was chosen to explore whether it would induce a larger reduction in thermal and cardiovascular strain (compared with 5 min CWI) and subsequent superior 4 km TT performance. The water temperature was monitored with a 6000 series bench thermometer (TM Electronics Ltd., West Sussex, UK) with a type T thermocouple, and ice was added to decrease the temperature when needed. In order to limit potential bias in treatment response, participants were neither informed of expected outcomes of each recovery intervention, nor had information in relation to the belief effect of each intervention collected before the study.

Measurements

Core Temperature

During each testing session T_{core} (gastrointestinal temperature) was recorded continuously using ingestible body temperature sensors swallowed with tepid water approximately 6 h prior to testing, and a hand held data receiver (CorTemp, HQ, Florida, USA). This method provides a valid index of core temperature in comparison with esophageal temperature, the best available index for core temperature in exercise studies (Byrne and Lim, 2007).

Pulmonary Gas Exchange, Heart Rate, and Ratings of Perceived Exertion

During the incremental cycling tests, participants wore a facemask to continuously collect expired air using an online metabolic system (Cosmed Quark CPET, Rome, Italy), and HR was recorded on a second-by-second basis (S610i, Polar Electro Oy, Finland). Prior to, and at the end of each 12 min constant-load bout and 4-km TT exercise protocol, ratings of perceived exertion were documented in relation the Borg scale (6–20) (Borg, 1990).

Statistical Analysis

Data are presented as mean ± SD. Two-way repeated measures ANOVA (trial by time) was used to analyse the 4-km TT times to failure, HR, T_{core} and ratings of perceived exertion responses within each experiment. Differences were detected using Holm-Sidak *post-hoc* tests. Sphericity was assessed and adjusted where the assumption of sphericity could not be assumed ($\varepsilon > 0.75$ = Huynh–Feldt; $\varepsilon < 0.75$ = Greenhouse–Geisser). Significance was set at $P \leq 0.05$. Effect sizes (ES) were also calculated using Cohen's *d* to compare the magnitude of the difference in time to completion between the trials (Cohen, 1988). Thresholds for effect sizes were set as the following: <0.19, trivial; 0.20–0.49, small; 0.5–0.79, moderate; >0.8, large; with an effect size of 0.2 being considered as the smallest worthwhile positive effect. Effect size was computed as d = [(mean Ex1 – mean Ex2) / pooled standard deviation]. Within-participant consistency of time trial performance during Ex1 was established using the coefficient of variation (CV) derived from the log-transformed data using the Excel spreadsheet of Hopkins (Hopkins, 1997). A power analysis indicated that nine participants per group were required to detect a ∼ 4% improvement in 4 km TT with a power of 0.80 and alpha of 0.05 for an ANOVA calculation design based on 3 trials. This was estimated using means and standard deviations from previously published similar studies (Peiffer et al., 2010; Tomazini et al., 2020).

RESULTS

4-km Cycling Time Trial Performance

In Experiment 1, the time to complete the 4-km TT in both trials was not different ($P > 0.05$, trivial ES) in both, Ex1 (PAS: 414 ±

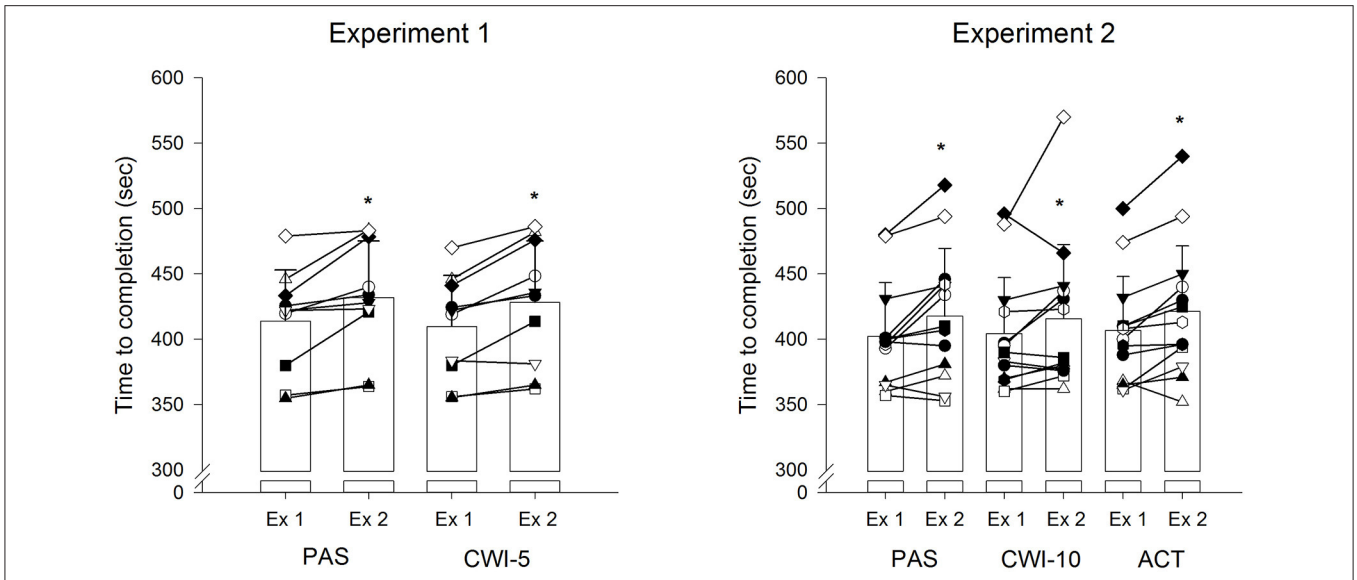

FIGURE 2 | Individual and mean (±SD) cycling completion times for the 4-km time trials (TT) during Exercise 1 (Ex 1, pre-recovery) and Exercise 2 (Ex 2, post recovery) for the experimental conditions. PAS, passive recovery; CWI-5, 5 min cold water immersion; CWI-10, 10 min cold water immersion; ACT, active recovery. *Significantly different from Ex1 ($P < 0.05$).

39 s; CWI-5: 410 ± 39 s) and Ex2 (PAS: 432 ± 43 s; CWI-5: 428 ± 47 s). In both conditions, the time to complete the time trial was longer ($P < 0.05$, small ES) in Ex2 than Ex1 (**Figure 2**).

Similarly, in *Experiment 2,* the time to complete the 4-km TT in the 3 trials was not different ($P > 0.05$, trivial ES) in both, Ex1 (PAS: 402 ± 41 s; CWI-10: 404 ± 43 s; ACT: 407 ± 41 s) and Ex2 (PAS: 418 ± 52 s; CWI-10: 416 ± 57 s; ACT: 421 ± 50 s). In all conditions, the time to complete the time trials was longer ($P < 0.05$, small ES) in Ex2 than Ex1 (**Figure 2**).

Core Temperature

T_{core} responses across all conditions over time are presented in **Figure 3A** (*Experiment 1*) and **Figure 3D** (*Experiment 2*). During all time points of Ex,1 T_{core} values were similar between conditions in both experiments. T_{core} progressively increased until the end of Ex1 by a magnitude that was not different among the conditions (*Experiment 1*: PAS: 38.2 ± 0.4°C; CWI-5: 38.1 ± 0.3°C; *Experiment 2*: PAS: 38.1 ± 0.4°C; CWI-10: 38.1 ± 0.4°C; ACT: 38.1 ± 0.4°C). T_{core} then decreased during the 15 min recovery phase with no significant differences among conditions. At the onset of the constant-load bout immediately after the recovery period and until the beginning of the 4-km time trial, CWI-5 evoked significantly lower ($P = 0.02$) T_{core} responses compared with the passive rest trial (trial × time interaction, $P = 0.03$) in *Experiment 1* (**Figure 3A**). In *Experiment 2*, during the entire period of Ex2, CWI-10 evoked significantly lower ($P < 0.001$) T_{core} responses compared with the passive and active recovery trials (trial × time interaction, $P < 0.01$) (**Figure 3D**).

Heart Rate

There was no significant difference in HR between the experimental conditions within both experiments during Ex1 (*Experiment 1*: **Figure 3B**; *Experiment 2*: **Figure 3E**). In *Experiment 1*, there was a tendency for HR to be lower during CWI-5 than PAS during Ex2 (trial × time interaction, $P = 0.09$), however, it did not reach statistical significance (**Figure 3B**). In *Experiment 2*, there was a trial × time interaction ($P < 0.01$) where ACT recovery evoked a larger ($P < 0.01$) HR compared with PAS and CWI-10 conditions during the recovery period and onset of the subsequent constant-load bout; and at mid-point of the constant-load bout HR was also larger ($P = 0.01$) during ACT compared with CWI-10 as well as PAS compared with CWI-10 conditions. HR responses were similar among conditions during the 4-km time trials during Ex2 (**Figure 3E**).

Rating of Perceived Exertion

In *Experiment 1,* ratings of perceived exertion were significantly lower ($P < 0.001$) at the end of the moderate-intensity cycling bout of Ex2 during CWI-5 compared with PAS rest (trial × time interaction, $P = 0.001$) (**Figure 3C**). In *Experiment 2,* CWI-10 evoked lower ratings of perceived exertion than PAS and ACT conditions (main effect, trial; $P = 0.001$), but there was no group × time interaction ($P = 0.3$) (**Figure 3F**).

DISCUSSION

The main finding of the present study, in contrast with our principal hypothesis, was that the cold water immersion interventions did not improve the completion time during the subsequent 4-km cycling TT compared with PAS or ACT recovery interventions. Both CWI treatments elicited reductions in T_{core} (and most likely muscle temperature) responses during the second exercise bout, however, herein, these adaptations did not evoke performance benefits.

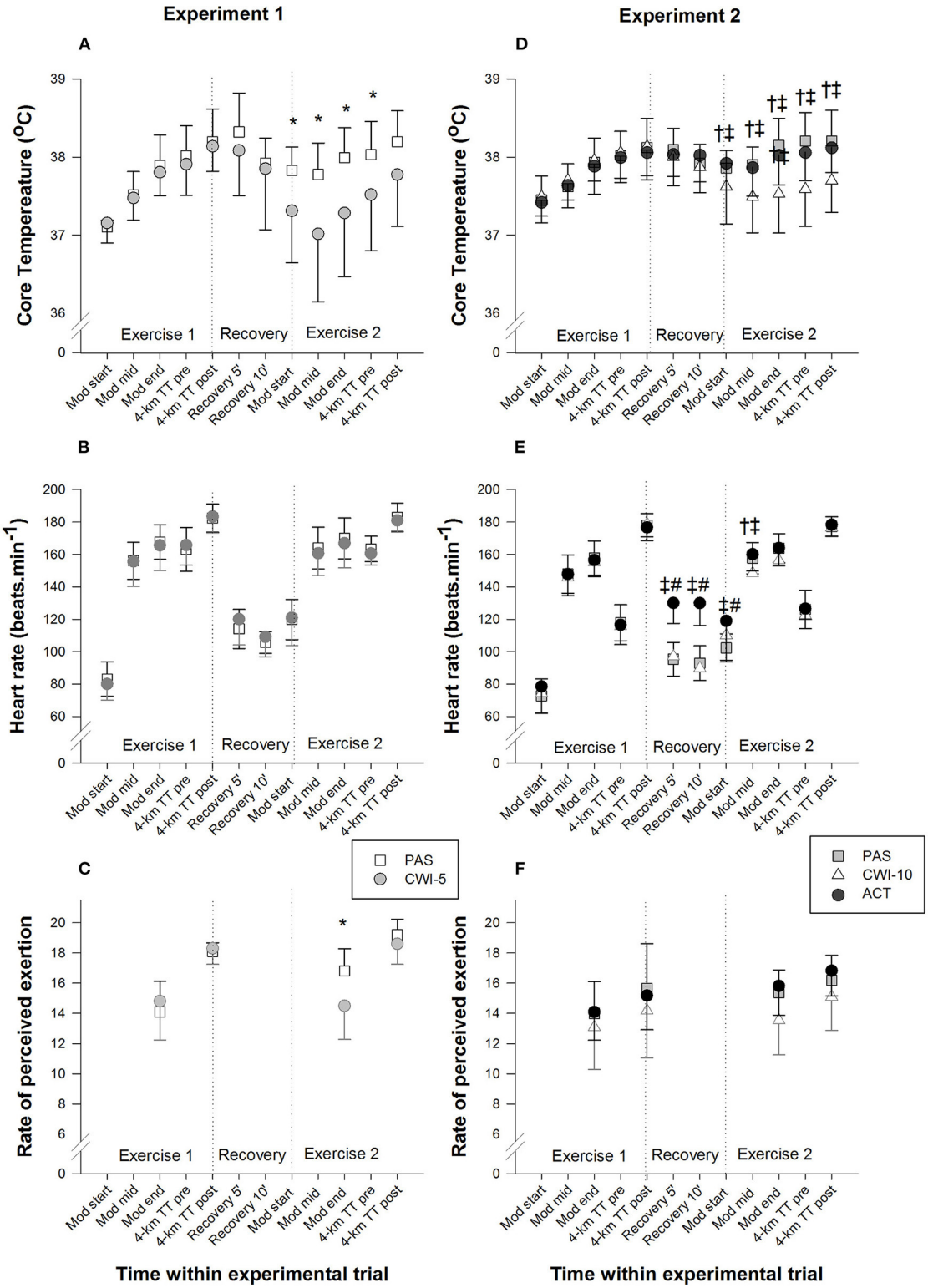

FIGURE 3 | Mean (±SD) core temperature (**A**, *Experiment 1*; **D**, *Experiment 2*), heart rate (**B**, *Experiment 1*; **E**, *Experiment 2*) and ratings of perceived exertion (**C**, *Experiment 1*, **F**, *Experiment 2*) responses at different time points during the experimental conditions. PAS, passive recovery; CWI-5, 5 min cold water immersion; CWI-10, 10 min cold water immersion; ACT, active recovery; Mod, 12-min moderate-intensity constant-load exercise; mid, mid time point; TT, time trial. *CWI-5 significantly different from PAS ($P < 0.05$). †CWI-10 significantly different from PAS ($P < 0.05$). ‡CWI-10 significantly different from ACT ($P < 0.05$). #Active recovery significantly different from PAS ($P < 0.05$).

Our findings are in contrast to similar previous studies that reported benefits in times to exhaustion during both, constant-load (Crampton et al., 2013; Dunne et al., 2013), and intermittent high-intensity exercise in normothermia (Mccarthy et al., 2016), as well as in time trial performance in hyperthermic conditions (Yeargin et al., 2006; Peiffer et al., 2010) when exercise was performed immediately following a short CWI when compared with passive and/or active recovery. In agreement with the present findings, Stephens et al. (2018) showed no difference in post-CWI 4-min time trial performance compared with a passive control condition, however, the time trial was carried out more than 40 min after the recovery interventions.

Herein, a significant afterdrop (hypothermic undershoot) effect was elicited (∼0.3°C), immediately after each water immersion intervention, likely attributed to a rapid dissipation of blood from the cooled peripheral tissues to the core (Arborelius et al., 1972; Mittleman and Mekjavic, 1988; Bristow et al., 1994). These afterdrop effects are consistent with previous studies that explored the effects of CWI employed between two equal high-intensity exercise protocols carried out immediately before and after the CWI recovery period under normothermia where the T_{core} achieved at the end of the pre-recovery exercise bout (Ex1) was similar (∼38–38.5°C) (Crampton et al., 2013; Dunne et al., 2013; Mccarthy et al., 2016; Egaña et al., 2019). Interestingly, during the 15 min recovery period, CWI-5 induced a larger absolute drop in T_{core} (∼0.8°C) compared with CWI-10 (∼0.5°C) which is consistent with findings reported by Mccarthy et al., 2016 under similar conditions. This is likely because some participants showed an exaggerated drop in T_{core} during the CWI-5 conditions (and hence, the variation for this variable for this condition was larger than for the other conditions, see **Figure 3A**), whereas some participants during the CWI-10 trial reported shivering which could have induced increases in metabolic heat production and T_{core}. Consequently, CWI-10 (as was the case for CWI-5) did not induce any ergogenic effects compared with PAS and ACT interventions (trivial effect sizes in all conditions), suggesting that the duration of the CWI, within a 15-min recovery phase, does not affect the completion time of a subsequent 4 km time trial in normothermia.

That the observed drop in T_{core} herein was accompanied by reductions, albeit non-significant, in HR during the 12-min constant-load bouts, suggests CWI reduced thermal and cardiovascular strain (Parkin et al., 1999; Marino, 2002). Nevertheless, it is noteworthy that this prolonged reduction in T_{core}, negatively impacts the contractile apparatus of cooled muscles (Bergh and Ekblom, 1979; Bigland-Ritchie et al., 1992) particularly during maximal efforts. Specifically, individuals performing all-out cycling bouts (i.e., 30 s "Wingate" sprints) immediately after CWI, demonstrate significant reductions in performance when compared with passive or active recovery conditions (Schniepp et al., 2002; Crampton et al., 2014). Indeed, when an intermittent sprint protocol that replicates the typical sprint characteristics of many team sport games (20–30 brief, 6–8 s, all-out sprints separated by 14–120 s active recovery periods within a 40 min "half-time") was carried out immediately after CWI, the total work done and power output during the early sprints was reduced, while later sprints showed higher work and power output, leading to an overall significant increase in the second-half sprint performance (Egaña et al., 2019). As such, it is likely that once muscles are adequately warmed up, the increased heat storage capacity facilitated by the lower T_{core} response contributed to the improvements observed in the subsequent sprint performance (Lee and Haymes, 1995; Booth et al., 1997; Kay et al., 1999). In line with this notion, participants performing 3 × 4 min high-intensity efforts after CWI showed a likely higher anaerobic contribution (at least partly due to a reduced perfusion in cooled muscles) during the first bout (compared with passive rest), but the anaerobic contribution was less likely to be different between CWI and PAS conditions in the three remaining bouts (Stanley et al., 2014).

For that reason, in the present study participants performed a 12-min moderate-intensity warm-up prior to carrying out the 4-km TT bouts. Importantly, Mccarthy et al. (2016) used the same CWI recovery as well as post-recovery warm-up protocol and observed that the time to failure during an intermittent high-intensity cycling protocol lasting ∼6–8 min (i.e., similar duration as the times to complete the 4-km TT in the current study) was significantly improved compared with passive rest. In addition, using longer (i.e., 15–30 min) CWI periods and a similar post-recovery warm-up, the time to failure during a subsequent constant-load exercise was also significantly prolonged during both, running (exercise bouts lasting ∼20–27 min) (Dunne et al., 2013) and cycling (exercise bouts lasting ∼10–20 min) (Crampton et al., 2013) compared with passive rest. This would suggest that CWI is more efficient at enhancing exhaustive intermittent and/or constant-load efforts compared with time trial efforts, at least in normothermic conditions. It is worth noting, however, that Peiffer et al. (2010) reported CWI-induced benefits in subsequent 4-km TT-s in hyperthermia, where participants completed a longer (25 min) moderate-intensity exercise bout prior to the 4-km TT-s. Thus, we cannot exclude the possibility that a longer warm-up period is needed to adequately warm up the active muscles and maximize subsequent 4 km TT performance.

In the present study active recovery did not induce any improvements in cycling TT performance compared with PAS and CWI recoveries. While active recovery is one of the most frequently employed recovery interventions in sports, given, among other factors, its ability to accelerate lactate clearance (Bangsbo et al., 1994); its effectiveness regarding subsequent exercise performance is inconclusive. For instance, the effectiveness of active recovery and CW1 appear to be on a par in maintaining the endurance performance of eccentric exercises, well-known to induce a substantial degree of muscle damage and high lactate levels, such as "lead" style climbing (Heyman et al., 2009). However, active recovery appears less effective than CWI in maintaining time to exhaustion during concentric non-damaging submaximal exercise akin to that employed herein (Crampton et al., 2013).

LIMITATIONS

A number of limitations of the present study must be acknowledged. First, our study participants cycled recreationally, and thus, day to day variation may be greater than in trained cyclists. However, all participants performed at least one

familiarization trial which helped reduce any potential learning effect. Indeed, the overall coefficient of variation across the Ex1 time trials herein was low (∼3.2%) yet still higher than that reported for trained cyclists (∼1.9%) (Stone et al., 2011). Nevertheless, the involvement of recreational active participants in the present study, limits the applicability of the results to athletic settings. Second, we acknowledge that it would have been preferable if the same participants completed all recovery interventions herein. For practical reasons this was not feasible. *Experiment 1* was designed to mimic the study protocol (i.e., comparing CWI-5 vs. PAS conditions) used by Peiffer et al. (2010) under hot ambient conditions (35°C), albeit in normothermia. However, upon observation that a 5 min CWI stimulus was not sufficient to induce benefits in subsequent TT performance, several weeks later in *Experiment 2* we explored whether a longer 10 min CWI would be more effective (than CWI-5) on subsequent TT in different participants, while also including a commonly used active recovery intervention. Third, the two experiments herein used different cycling ergometers, while it would have been preferable if they used the same one. This was not feasible also for practical reasons (i.e., the Wattbike was not available to use in *Experiment 2*). Despite this, both the Lode (Driller, 2012) and Wattbike (Bellinger and Minahan, 2014) cycling ergometers have been shown to be reliable for TT performance, so, it is unlikely that this would have affected the present study outcomes. Finally, we acknowledge that additional physiological parameters such as muscle temperature, muscle perfusion, blood lactate or $\dot{V}O_2$ kinetics measures would have been helpful to further explain the present results and demonstrate whether the warm-up period prior to the second TT was of sufficient duration to warm up the active muscles.

CONCLUSION

In conclusion, the present study showed that in comparison to passive and active recovery strategies, a brief (5–10 min) post-exercise cold water immersion at 8°C does not improve the performance during a subsequent 4-km cycling time trial. Our findings do not support the use of cold water immersion in between two time trial efforts lasting approximately 7 min when preceded by a 12 min moderate-intensity warm-up in normothermic ambient conditions. Further studies should assess whether CWI affects subsequent sustained high-intensity efforts when these efforts are preceded by longer warm-up periods to ensure active muscles are suitably warmed up.

DATA AVAILABILITY STATEMENT

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

ETHICS STATEMENT

The studies involving human participants were reviewed and approved by Faculty of Health Science Research Ethics Committee, Trinity College Dublin. The patients/participants provided their written informed consent to participate in this study.

AUTHOR CONTRIBUTIONS

ME, SW, LA, and KG conceived and designed the research and analyzed data. LA and KG conducted experiments and collected all participant data. ME drafted the paper. ME, SW, and NG interpreted data and contributed to the writing of the final paper. All authors contributed to the article and approved the submitted version.

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REVIEW
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Cryostimulation for Post-exercise Recovery in Athletes: A Consensus and Position Paper

Romain Bouzigon[1,2,3]*, *Olivier Dupuy*[4,5], *Ivo Tiemessen*[6,7], *Massimo De Nardi*[8,9], *Jean-Pierre Bernard*[10], *Thibaud Mihailovic*[1,2], *Dimitri Theurot*[4], *Elzbieta Dorota Miller*[11], *Giovanni Lombardi*[12,13] and *Benoit Michel Dugué*[4]

[1] Université de Franche-Comté, UFR STAPS Besançon, Laboratoire C3S (EA4660), Axe Sport Performance, Besançon, France, [2] Society Inside the Athletes 3.0, Sport Performance Optimization Complex (COPS25), Besançon, France, [3] Society Aurore Concept, Noisiel, France, [4] Université de Poitiers, Laboratoire MOVE (EA 6314), Faculté des Sciences du Sport, Poitiers, France, [5] Ecole de Kinésiologie et des Sciences de l'Actvivité Physique (EKSAP), Faculté de Medecine, Université de Montreal, Montreal, QC, Canada, [6] ProCcare BVBA, Antwerp, Belgium, [7] Mobilito Sport, Amsterdam, Netherlands, [8] Krioplanet Ltd, Treviglio, Italy, [9] Department of Experimental Medicine, Università Degli Studi di Genova, Genoa, Italy, [10] Air Liquide Group International Expert in Cryogenic Applications Cryolor, Ennery, France, [11] Department of Neurological Rehabilitation, Medical University of Lodz, Lodz, Poland, [12] Laboratory of Experimental Biochemistry and Molecular Biology, IRCCS Istituto Ortopedico Galeazzi, Milan, Italy, [13] Department of Athletics, Strength and Conditioning, Poznań University of Physical Education, Poznań, Poland

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Edited by:
Robert Allan,
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Erich Hohenauer,
THIM University of Applied Sciences for Physiotherapy, Switzerland
Jill Alexander,
University of Central Lancashire,
United Kingdom

***Correspondence:**
Romain Bouzigon
romain.bouzigon@gmail.com

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Recovery after exercise is a crucial key in preventing muscle injuries and in speeding up the processes to return to homeostasis level. There are several ways of developing a recovery strategy with the use of different kinds of traditional and up-to-date techniques. The use of cold has traditionally been used after physical exercise for recovery purposes. In recent years, the use of whole-body cryotherapy/cryostimulation (WBC; an extreme cold stimulation lasting 1–4 min and given in a cold room at a temperature comprised from −60 to −195°C) has been tremendously increased for such purposes. However, there are controversies about the benefits that the use of this technique may provide. Therefore, the main objectives of this paper are to describe what is whole body cryotherapy/cryostimulation, review and debate the benefits that its use may provide, present practical considerations and applications, and emphasize the need of customization depending on the context, the purpose, and the subject's characteristics. This review is written by international experts from the working group on WBC from the International Institute of Refrigeration.

Keywords: cryotherapy, cryostimulation, recovery, sport recovery, cold therapy, whole-body cryotherapy, whole-body cryostimulation

INTRODUCTION

Concerning sport competition and training, a well-organized training program combined with appropriate recovery strategies are keys for success. Different kinds of strategies for enhancing recovery and recovery capacities have been developed and quite recently, we presented a series of meta-analysis dealing with the use of different recovery techniques after physical exercise (training and/or competition) to reduce markers of muscle damage, soreness, fatigue, and inflammation. In addition, we compared the effects of one strategy versus another (Dupuy et al., 2018). Among the reviewed recovery techniques, whole-body cryotherapy or whole-body cryostimulation (WBC) was shown to positively impact the recovery.

Cold exposure (locally or whole-body) has been used for a very long time in the context of sports and medicine to relieve pain and inflammatory symptoms through cold-induced analgesia. Nowadays, WBC is defined as extreme cold therapy or stimulation which is applied by placing a subject in a cold room, for 1–3 or 4 min, where the air temperature can reach extremely low values (felt temperature ranging from −60 to −195°C). Individuals are exposed with minimal clothing with protections on the feet, hands, and ears. A small surgical mask is applied to protect the airways. An alternative is the use of partial-body cryotherapy/cryostimulation (PBC) where the subject's body is exposed in a cabin but the head is not exposed (Bouzigon et al., 2016).

The main effect of WBC is analgesia, related to the impact of very-low temperatures on the nervous system disabling the functional connections with the sensory receptors and proprioceptors conduction in sensory fibers. This process is responsible for the reduction of increased muscle tension. The use of WBC/PBC as cold therapy reduces post-traumatic edema, inflammation (facilitates the exchange of inflammatory products, carbon dioxide, accelerates metabolism, provides nutritional substrates, molecular oxygen, etc.), and facilitates post-exercise recovery by reducing the sensations of delayed onset muscle soreness (DOMS) and exercise-induced muscle damage (EIMD; Dupuy et al., 2018).

This paper aimed to provide general information on WBC and PBC when it is used after physical exercise. The technologies that are regularly used are described and a specific focus is made on the potential recovery benefits that athletes may benefit from following exercise and physical activity.

CRYOSTIMULATION DEFINITIONS (PROTOCOL AND TECHNOLOGIES)

Cryostimulation is a relatively new technique consisting on exposing a part of or the whole-body to extreme cold for a short period of time. Based on the positive effects of cold immersion used since the antiquity, cryostimulation is an innovative technology that is developing significantly (Bouzigon et al., 2016). The two types of technologies are available in the market, WBC and PBC. The WBC chambers, named cryochambers, are composed of 1–3 rooms in which the individual stays between 1 and 4 min and where the whole-body is exposed. The two main cold production methods that can be used are: mechanical refrigeration based on vapor compression cycles with refrigerants and/or cryogenic fluids, mainly liquid nitrogen, as it is a widely used and common fluid in many industries (**Figure 1**). There are two types of WBC chambers: static cold chambers and forced convection chambers based on wind chill. Cryochambers are more complex devices, but they avoid anoxia issues by using indirect injections in the chambers and the whole body is in a cold environment for the treatment. PBC tanks, named cryosaunas, are adapted for single usage for an individual, where cryogenic fluid is injected and vaporized around the body. However, the head must stay above the gaseous environment to preserve breathing (Dugué et al., 2020).

The cryostimulation devices provide the cold temperatures. The manufacturers state that temperatures range from −110 to −195°C in cryosaunas, from −60 to −160°C in static cold WBC technologies and from −40 to −60°C in forced convection WBC technologies (Bouzigon et al., 2016, 2017). Temperatures recorded inside the different devices during an exposure actually range from −10 to −42°C in cryosaunas and −34°C in a forced convection WBC chamber (Savic et al., 2013; Bouzigon et al., 2019). The temperatures in a static cold WBC chamber during the exposure are not shown in the literature.

Mean skin temperature variation after a 3-min exposure is ∼-8°C in a cryosauna (mean skin temperature after exposure between 22 to 24°C); ∼-11°C in a static cold WBC chamber (mean skin temperature after exposure between 18 to 20°C); and ∼-14°C in a forced convection WBC chamber (mean skin temperature after exposure between 16 to 18°C; Bouzigon et al., 2016, 2019). The exposure time ranges from 1 to 4 min (Bouzigon et al., 2016). The exposure duration depends on the protocol used but could be the same between the different devices. The 1-min exposure protocol is commonly used for the first exposure in a non-initiated individual.

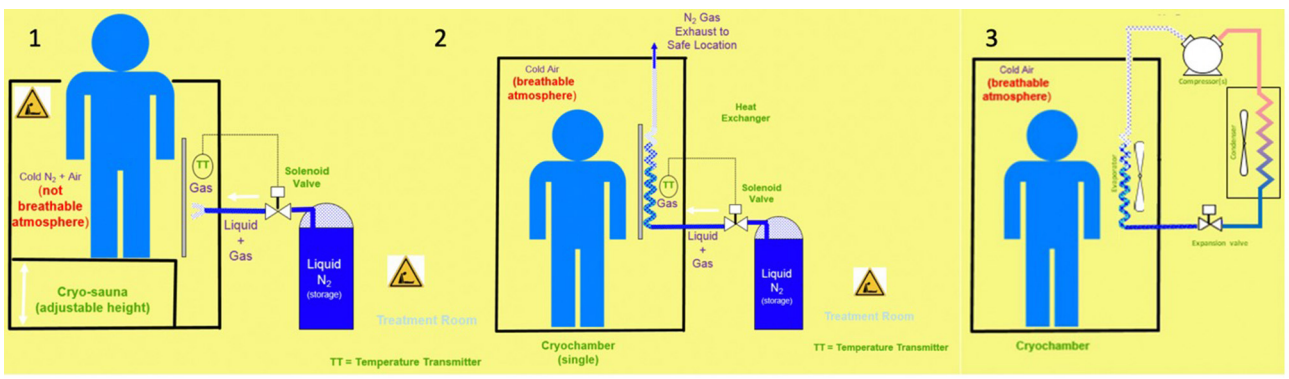

FIGURE 1 | 1: Cryosauna: Principle with liquid N2/direct injection/PBC/individual. 2: Cryochamber: Principle with liquid N2/indirect injection/WBC/individual or collective. 3: Cryochamber: Principle with mechanical refrigeration (single stage)/WBC/collective.

GENERAL PHYSIOLOGICAL REACTIONS AFTER WBC/PBC

Humans have a constant internal core temperature of ∼37°C. To maintain such internal temperature, the body needs to thermoregulate (Moellering and Smith, 2012). Thermoregulation is a complex physiological process, which is not entirely understood. A great part of the total body energy is produced as heat to maintain the internal temperature in our organism (Stocks et al., 2004). During cold stress, changes in the endocrine, circulatory, neuromuscular, and immunological systems occurred (Kellogg, 2006). The key point in the thermoregulation is cutaneous circulation. The dermis is characterized by the presence of a higher number of cold receptors (10-fold higher) in comparison to heat receptors.

The current view is the following after a WBC/PBC exposure: vasoconstriction that is induced after cold exposure may play a role in decreasing the amount of blood in and around the muscles and in some organs (Charkoudian, 2003), in decreasing the cell permeability and leaking, in diminishing the fluid diffusion in interstitial space that can occur after physical exercise to reduce edema development, and in reducing inflammation (Banfi et al., 2009b). In the context of recovery after physical exercise, a WBC/PBC exposure induces a lower muscle temperature that may decrease muscle enzyme activities, metabolism, inflammation, and secondary degradation after hypoxia (lowering ischemia/reperfusion problems) that may help recovery (Bouzigon et al., 2016). The effects of the WBC/PBC on physical recovery are discussed in a paragraph below (Section WBC—PBC Can Diminish Inflammation and Induce Analgesic Effects). During vasoconstriction, there is a strong activation of the sympathetic system accompanied with the release of noradrenalin. Noradrenalin is known to have an impact on pain, and this may explain how WBC can contribute in relieving the pain symptoms. Other compounds, such as endorphins may have an impact. Besides such mechanisms, cryogenic temperature reduces the conduction velocity of sensory nerve fibers and impulsion in the slow conducting C fibers disabling the sensory receptors as well as their connections with the proprioceptors. In addition, it seems that there is a decrease in the production of pro-inflammatory and oxidative substances whereas the anti-inflammatory and anti-oxidative compounds are produced in larger quantities (Banfi et al., 2010; Lombardi et al., 2017).

After exposure, cold-induced vasodilation occurs, enabling a four-fold higher blood flow than normal. Such a situation is observed about 4 min after the contact with cryogenic temperatures. Moreover, this robust blood circulation change can last a few hours following exercise, resulting in the elimination of metabolic products. It is estimated that the return to basal skin temperatures is likely after 14 min. At distance from the cold stimulation, an increase in the parasympathetic cardiac control may also happen (Louis et al., 2020) even during the night (Douzi et al., 2018). Such changes related to the central nervous system may have impacts on the psyche where lower fatigue sensation and mood improvement have been noted with possible positive impacts on clinical depression syndromes, and improvement in sleep quality (Rymaszewska et al., 2008). However, there are a range of individual responses to cold due to inter-individual differences, such as body size, fitness level, amount of subcutaneous fat, and gender (Miller et al., 2012).

WBC—PBC Can Diminish Inflammation and Induce Analgesic Effects

The main aim of cryostimulation is represented by the anti-inflammatory and analgesic effects. These effects, although proven and reported by several research, are highly variable and there is still no consensus on the reliability of the outcomes (Bouzigon et al., 2016; Lombardi et al., 2017). This is clearly evidenced by the fact that participants who underwent cycles of cryostimulation almost invariably reported an improved sense of well-being feeling, accompanied with the improvement of inflammatory symptoms and pain (when present) (Lombardi et al., 2017). Several studies have tried to provide mechanistic explanations for this phenomenon by measuring clinically relevant changes in certain blood biomarkers of inflammation [e.g., interleukin (IL)-6, IL-1, IL-1 receptor antagonist (IL-1ra), IL-10, IL-4, tumor necrosis factor (TNF)-α, and C-reactive protein (CRP)] (Lombardi et al., 2017).

There are two published randomized controlled trials evaluating the effects of WBC on post-exercise recovery in physically active subjects. In a cross-over study by Hausswirth et al. (2011), well-trained runners who underwent three separate simulated races to induce muscle damage, followed by the three sessions (0, 24, and 48 h) of three different recovery strategies (passive, far-infrared therapy, and WBC). WBC was performed in a three-room device at −10, −60, and −110°C, respectively; the subjects entered the warmer rooms and remained in the therapy room for 3 min. Despite the similar kinetics of the muscle damage marker creatine phosphokinase (CK), a better and faster improvement of perceived pain, tiredness, and well-being following WBC compared with the other recovery strategies were reported (Hausswirth et al., 2011). In parallel, Selfe et al. (2014) failed to record any change in IL-6, regardless of the duration of the WBC exposure (1, 2, or 3 min, at −135°C). In this study, each WBC exposure consisted of 30 s precooling at −60°C followed by a randomized exposure for either 1, 2, or 3 min at −135°C.

However, several published reports demonstrated the anti-inflammatory effects of WBC. Tennis players exposed to WBC (20–30 s at −60°C and 3 min at −120°C) twice a day over 5 consecutive days during a training camp showed decreased circulating TNFα and increased circulating IL-6 in the treated athletes more than in the untreated athletes (60 and 30%, respectively). The blood samplings were performed 3 days before and 2 days after the camp (Ziemann et al., 2012). In 18 professional male volleyball players, WBC treatment (10–20 s at −60°C, 100–110 s at −120°C) performed before a session of submaximal exercise prevented exercise-induced rise of IL-1β and IL-6 that, in the absence of any treatment, attested about +60%. The setting expected two blood sampling, before and after the physical effort (Mila-Kierzenkowska et al., 2013). In healthy young men, IL-6 increased after a single session of WBC (as recorded after 30 min and 24 h) although less consistently, after 10 days of the treatment (Lubkowska et al., 2010). The WBC was

performed by exposing subjects for 30 s at −60°C and 2.5 min at −130°C; baseline blood samples were taken in the morning before the first WBC session while the post-treatment sampling was performed in morning after the last WBC session. In a recent meta-analysis, Dupuy et al. (2018) reported that acute cryostimulation increased plasmatic IL-6.

The fact that a change in IL-6, either toward increase or decrease, is considered beneficial or not depends on the coexistence of both pro- and anti-inflammatory roles of this cytokine depending on the site and kinetics of secretion (i.e., liver-derived, chronically slightly elevated IL-6 acts in a pro-inflammatory fashion; skeletal muscle-derived, and temporarily limited peaked IL-6 acts in an anti-inflammatory fashion; Lombardi et al., 2016). Furthermore, in a group of volleyball players who underwent a 2-week training program, daily WBC led to an improvement in the growth factor profile [brain-derived neurotrophic factor (BDNF) and insulin-like growth factor 1 (IGF-1)] that may sustain muscle regeneration and limit the deterioration of physical performances (Jaworska et al., 2018). However, in elite athletes subjected to randomized WBC (3 min, −120°C, two times a day for 7 days) or no treatment, despite the significant reduction of reactive oxygen and nitrogen species (H_2O_2 and NO) and the concentrations of the pro-inflammatory mediators IL-1β and CRP, cryostimulation caused the decrease in IGF-1, BDNF, hepatocyte growth factor (HGF), platelet-derived growth factor (PDGFBB), and vascular endothelial growth factor (VEGF; Zembron-Lacny et al., 2020). These findings further suggest that WBC may act as an exercise-mimetic treatment, possibly associated to the cold-induced muscle contraction, stimulating the expression of those myokines whose release are typical following an acute bout of physical activity.

From a cellular point of view, the leukocytes were mostly unaffected by the WBC treatment in a group of professional rugby players (Lombardi et al., 2013) and in professional kayakers (Sutkowy et al., 2014), who underwent two-daily sessions of cryostimulation, during a training camp. On the contrary, Szygula and colleagues (Szygula et al., 2014) recorded an increase in leukocyte count during the first 20 sessions of WBC in students of a military academy and a return to baseline at the 30th session. Similarly, Ziemann et al. (2012) found an increase in whole leukocyte counts but not in the neutrophil and lymphocytes subpopulations in the tennis players. Noteworthy, when recorded, the changes in leukocyte count remained always within the physiological range. Consequently, mobilization, rather than generation, may explain this phenomenon although the biological significance remains unknown. The platelets, however remained mostly unaffected by the WBC treatment (Lombardi et al., 2013; Szygula et al., 2014; Ziemann et al., 2014), contrarily to what was observed in other situations of cold exposure as described in the case of winter swimming (Lombardi et al., 2011). These variable results may be due to the differences in the applied protocols as well as in the differences in the study cohorts that sum to high inter-individual variability often observed in the terms of response to cryostimulation.

Important variables, although hardly definable to the field, that can be the cause for the limited "biochemical evidence" in support of cryostimulation effectiveness, may be represented by the frequency and length of the treatment, the timing of application within the timing practice (e.g., training sessions), as well as subjective features that may alter the responsivity. For this reason, Lubkowska et al. (2011) demonstrated that the number of sessions importantly impacted the kinetics of secretion of biochemical markers of inflammation. Indeed, in 45 healthy men, five consecutive sessions of WBC increased IL-10 by 30% while 10 sessions determined a 17% decrease of IL-1α, 10% increase of IL-6, and 14% increase of IL-10. In both cases, the changes were no longer detectable after 2 weeks. On the contrary, 20 sessions of WBC had a similar impact on the cytokine profile, as for 10 sessions, but those effects were maintained 2 weeks after the end of the treatment. Other inflammatory mediators, such as IL-1β, TNFα, and IL-12 were unaffected by the treatment.

WBC Can Diminish DOMS

The study of the preventive effects of WBC on DOMS was mostly demonstrated in the settings where specific exercise/training programs were applied. Compared with passive recovery, following four sessions of WBC (three-room device at −10, −60, and −110°C, with permanence in the therapy room for 3 min), the CRP increase was attenuated, and the anti-inflammatory IL-1 receptor antagonist (IL-1ra) was strongly induced in the runners who underwent a 45-min trail run specifically designed to induce the muscle damage. However, the exercise-associated changes in the TNF, IL-6, and IL-10 were unaffected. In addition, WBC led to a greater increase in the neutrophil count, compared with passive recovery, that may have accounted for a greater stimulus for angiogenesis that resulted in increased muscle perfusion and, hence, better recovery and lesser soreness (Pournot et al., 2011).

In physically active college-aged men who underwent an eccentric workout designed to induce DOMS, two-daily sessions of WBC over 5 days blunted the response of IL-1β and IL-6 while stimulating the secretion of IL-10 (Ziemann et al., 2014). Noteworthy, WBC reduced the physiological burden of an eccentric cycling bout and the rise of myoglobin and IL-15 concentrations, thus indicating the potential modulatory effects of WBC also on muscle strength (Jaworska et al., 2020). In a recent meta-analysis, Dupuy et al. (2018) reported that an acute cryostimulation exposure is a convenient tool to reduce DOMS. More recently, Poignard et al. (2020) found that cooling strategies, such as cryotherapy, is an effective strategy to reduce DOMS in professional tennis players. In this article, in comparison with cold-water immersion (CWI), WBC induced a greater feeling of recovery in this population. In line with this, Wilson et al. (2019) found that the WBC is a more effective strategy to recover from resistance training in comparison with CWI. However, the comparison of WBC with CWI in the management of DOMS still needs to be confirmed since several articles found no difference between WBC and CWI (Abaidia et al., 2016).

TABLE 1 | The studies investigating the acute effects of cryostimulation on the physical recovery.

References	Outcomes /subjects	WBC or PBC treatment protocol	Cryostimulation effects compare to control condition ("+": positive effect; "-": negative effect; "=": no change)
Bouzigon et al. (2020)	Isometric muscle recovery Motocross riders 18 males	WBC (Aurore Concept, France) 1 exposure After training 30 s at −25°C and 3 min at −70°C with wind chill	+ Isometric strength - CMJ performance = Reaction time = Handgrip strength + Perceptive recovery
De Nardi et al. (2020)	The range of motion 11 young adult females	PBC (Criomed, Ukraine) 1 exposure 2 min 30 between −130 and −170°C	+ Range of motion
De Nardi et al. (2017)	Isometric strength Healthy adults 100 females 100 males	PBC (Criomed, Ukraine) 1 exposure 2 min 30 s between −130 and −160°C	+ Handgrip strength
De Nardi et al. (2015)	The range of motion Healthy adults 60 females 60 males	PBC (Criomed, Ukraine) 1 exposure 2 min 30 s between −130 and −140°C	+ Range of motion
Ferreira-Junior et al. (2014)	Neuromuscular performance Recreationally resistance-trained participants 13 males	PBC (Cryoness, Poland) 1 exposure 3 min at −110°C	= Peak torque = Average power = Total work = Muscle activity
Fonda and Sarabon (2013)	Muscle damage Healthy men 11 males	PBC Criomed 6 exposures 1 per day/6 days 3 min −140 to −195°C	+ Pain + knee flexion rate of torque development + Squat jump start power + Maximal torque production = CK concentration in blood + Heart rate variability (HRV) indices
Hausswirth et al. (2011)	Markers of muscle damage Well-trained runners 9 males	WBC (Zimmer Elektromedizin, Germany) 3 exposures at 1, 24, and 48 h post exercise −10°C, −60°C and 3 min at −110°C then 10 min seated comfortably in a temperate room (24°C)	Effect post 1 h/post 24 h/post 48 h +/+/+ MVC =/=/= CK concentration in blood +/+/+ perceived pain +/+/+ perceiced tiredness =/+/+ Well-being
Hohenauer et al. (2020)	Recovery after muscle damage Recreationally trained participants 28 females	PBC (Criomed, Ukraine) 1 exposure After training 30 s at −60°C and 2 min at −135°	- Muscular oxygenation = Arterial pressure + DOMS = Muscle swelling = MVIC = CMJ performance
Kruger et al. (2015)	Acute recovery Endurance athletes 11 males	WBC (Zimmer, Germany) 1 exposure After training −10°C, −60°C and 3 min at −110°C.	During subsequent exercise: + Maximal endurance performance + Cardio-respiratory parameters (VO_2, HR) + Rating of perceived exertion + Muscular oxygenation
Piras et al. (2019)	Recovery during concurrent training Rugby players 9 males	PBC (Criomed, Ukraine) 1 exposure Between strength and endurance training 3 min at −160°C	During recovery: + HRV indices (SDNN, RMSSD, HF) + Baroreflex sensitivity During endurance training: + HR + VO_2 + Minute ventilation + Energy cost = Respiratory exchange ratio = Blood lactate concentration = Net energy expenditure derived from aerobic energy sources
Russell et al. (2016)	Physiological performance and perceptual responses English Premier League academy soccer players 14 males	WBC (Juka, Poland) 1 exposure After training 30 s at −60°C and 2 min at −135°C	= CMJ performance = Blood lactate concentration = CK concentration in blood + Salivary testosterone concentration = Salivary cortisol concentration

(Continued)

TABLE 1 | Continued

References	Outcomes /subjects	WBC or PBC treatment protocol	Cryostimulation effects compare to control condition ("+": positive effect; "-": negative effect; "=": no change)
			= Perceived soreness = Perceived recovery
Vieira et al. (2015)	Vertical jump recovery Resistance-trained participants 12 males	PBC (Cryoness, Poland) 1 exposure After training 3 min at −110°C	= Muscle power
Wilson et al. (2019)	Recovery after resistance training Resistance-trained participants 24 males	WBC (Juka Cryotherapy Chamber) 2 exposures After training 3 min at −85°C, 15 min warming period and 4 min at −85°C	Effects post 24 h / post 48 h / post 72 h: = / - / = DALDA score + / = / = Perceived soreness - / = / = Peak torque = / = / = MVIC = / - / - Reactive strength - / = / - CMJ performance = / + / = Isometric peak force - / + / = RFD = / - / - CK concentration in blood = IL-6 concentration in blood = / - / - CRP concentration in blood = / - / = TNF-α concentration in blood
Wilson et al. (2017)	Recovery following a marathon Endurance athletes 31 males	WBC (Mecotec, Germany) 2 exposures After marathon 3 min at – 85°C, 15 min warming period and 4 min at−85°C	Post 24 h / post 48 h effects: = / = DALDA score = / + Perceived soreness - / - Muscle function (peak torque, reactive strength index) - / = CK concentration in blood - / - IL-6 concentration in blood - / + CRP concentration in blood = / = TNF-α concentration in blood

WBC, whole-body cryotherapy; PBC, partial-body cryotherapy; DALDA, The Daily Analysis of Life Demands for Athletes; DOMS, delayed onset of muscle soreness; CMJ, countermovement jump; MVIC, maximal voluntary isometric contraction; RFD, rate of force development; HR, heart rate; HRV, heart rate variability; RMSSD, root mean square standard deviation; SDNN, standard deviation of the NN (R–R) intervals; HF, high-frequency power; LF, Low-frequency power; VO_2, Oxygen consumption; VO_2 max, Maximal oxygen consumption; CK, creatine kinase; IL-6, interleukin 6; IL-15, interleukin 15; CRP, C reactive protein; TNF- α, tumor necrosis factor-α; BDNF, brain-derived neurotrophic factor; IGF1, insulin-like growth factor.

WBC—PBC and Performance

All the following articles used in this Section are presented in **Tables 1**, **2** of the Section Cryostimulation Protocols.

In the past decades, cryostimulation was often used by athletes, trainers, and sport physicians to promote enhanced athletic performance and recovery (Banfi et al., 2010; Lombardi et al., 2017). However, in high-performance sport, the question remains if cryostimulation can or should be used before an event to enhance the performance of an individual and the physical well-being or only to improve the recovery parameters in the athletes.

Current evidence supporting its utilization is limited and contradictory: the testimony of athletes is generally positive but it could be influenced by a possible placebo effect. From what can be deduct from testimonies of coaches and athletes, it seems that exposure to cryostimulation prior than ∼3 h to competition could have a performance enhancement effect (Partridge et al., 2019). The use of cryostimulation before training or competition may have a beneficial effect by a multi-factorial hypothesis, such as the positive effects of hormonal changes (Hornery et al., 2005; Rose et al., 2017), with an increase of circulating cortisol and testosterone, and by a peripheral vasoconstriction, which consequently leads to high muscle oxygenation afterward (Hornery et al., 2005), and by a psychological well-being. The scientific community will have to study the possible effects of cryostimulation on sport performance, since further studies are required to provide recommendations for the coaches and athletes based on evidence. In fact, only few studies examined this topic.

Studying physical performance, Ziemann et al. (2012) found improvements in tennis drills execution after a cycle of 10 WBC exposures (3 min; −120°C) carried out for 5 consecutive days (two times a day) in 12 high-ranking professional tennis players, demonstrated by a 7% improvement in stroke effectiveness, measured through shot accuracy and speed. Schaal et al. (2014) found, in 10 elite synchronized swimmers, a reduction of swimming speed in a 400 m trial between before and after an overreaching period in the experimental group after a cycle of 10 sessions of WBC exposures (3 min; −110°C) compared with the control group. Further studies, maybe without methodological differences in terms of exposure temperature, duration, and sessions dose, are required to confirm the cryostimulation effectiveness in sport specific performances, such as tennis stroke accuracy or swimming speed.

Le Meur et al. (2017) have studied the effect of 1 week WBC exposures (3 min; −110°C) in 16 functionally overreached triathletes. The results showed that completing multi exposures to WBC induced a larger performance supercompensation after a simulated 1-week taper in the functionally overreached endurance athletes. These preliminary findings showed that

TABLE 2 | The studies investigating the chronic effects of cryostimulation on the physical recovery.

References	Outcomes /subjects	WBC or PBC treatment protocol	Cryostimulation effects compare to control condition ("+": positive effect; "-": negative effect; "=": no change)
Broatch et al. (2019)	Physiological and performance adaptations Recreational athletes (triathlon or cycling) 22 males	WBC (Zimmer, Germany) 12 exposures After training −10°C, −60°C and 3 min at – 110°C	= Maximal aerobic power = VO_2 max = Time to exhaustion = Performance in the time trial = Blood markers (adrenaline, noradrenaline, cortisol) = Sleep quality (time in bed, sleep duration, sleep latency sleep efficiency …)
Jaworska et al. (2021)	Growth factors concentrations, amino acids profile and motor abilities in professional judokas	WBC (Zimmer, Germany) 10 exposures – One a day 2 h after training 30 s at −60°C and 3 min at −110°C	+ Circulating levels of two growth factors (BDNF and IGF-1) + Amino acid profile + Specific judo abilities + Muscle function
Jaworska et al. (2020)	Resistance training supported by cryostimulation Untrained students 17 females 13 males	WBC (Unknown model) 12 exposures during 4 weeks of resistance training 30 s at – 60°C and 3 min at −110°C	+ Isokinetic muscle strength + Pedal force + Myoglobin concentration in blood + Blood marker concentration (myostatin, IL-15) + Muscle pain
Jaworska et al. (2018)	Specific training supported by cryostimulation University volleyball players 10 females 10 males	WBC (Unknown model) 10 exposures during 2 weeks of specific volleyball training 30 s at −60°C and 3 min at −110°C	+ Limitation of physical performance decrease + Concentration of growth factors (BDNF, IGF1) in blood
Klimek et al. (2010)	Aerobic capacity and maximal anaerobic power Students of physical education 15 females 15 males	WBC (Unknown model) 10 exposures −60°C and 3 min at −110°C	= Aerobic capacity + Maximal anaerobic power
Le Meur et al. (2017)	Maximal incremental running test 16 triathletes in functional overreaching	WBC Zimmer 7 exposures on 7 days during 1 week of tapering	+ Performance in maximal incremental test = Change in perceived fatigue with CONT
Lubkowska and Szygula (2010)	Aerobic capacity 25 healthy males	WBC (Unknown model) 15 exposures 30 s at −60°C and 3 min at −130°C	= VO_2max - Red Blood Cell concentration in blood - Hemoglobin concentration

WBC, whole-body cryotherapy; PBC, partial-body cryotherapy; DALDA, The Daily Analysis of Life Demands for Athletes; DOMS, delayed onset of muscle soreness; CMJ, countermovement jump; MVIC, maximal voluntary isometric contraction; RFD, rate of force development; HR, heart rate; HRV, heart rate variability; RMSSD, root mean square standard deviation; SDNN, standard deviation of the NN (R–R) intervals; HF, high-frequency power; LF, Low-frequency power; VO_2, Oxygen consumption; VO_2 max, Maximal oxygen consumption; CK, creatine kinase; IL-6, interleukin 6; IL-15, interleukin 15; CRP, C reactive protein; TNF- α, tumor necrosis factor-α; BDNF, brain-derived neurotrophic factor; IGF1, insulin-like growth factor.

multiple exposures to WBC after a period of heavy training may mitigate the signs of accumulated fatigue during the intensified training blocks.

Aerobic and Anaerobic Performances

In the scientific literature, only few studies evaluated the influence of cryostimulation on the aerobic adaptation parameters. Klimek et al. (2010) enrolled 30 subjects (15 men and 15 women) to assess the influence of WBC on aerobic and anaerobic capacities. The participants underwent two ergocycle trials before and after a cycle of 10 consecutive WBC sessions. The authors calculated baseline aerobic capacity by means of a progressive cycle ergometer test and the anaerobic power by performing a 20-s Wingate test. After finishing the 10 WBC sessions, there were no changes in the aerobic capacity for both genders. Only men showed an increase in the maximal anaerobic power and capacity after 10 sessions in a cryogenic chamber. The authors concluded that in sports disciplines with a predominance of anaerobic metabolism, it could be advisable to introduce WBC treatment in the training periodization, at least in a male population. However, the lack of a control group could be a limit of the aforementioned study.

The same group of authors evaluated (Klimek et al., 2011) the effects of a single WBC treatment on the dynamics and the level of maximal aerobic power, enrolling 30 subjects (15 women and 15 men) and applying a Wingate test after each WBC session (six consecutive treatments at −130°C) in the 15, 30, 45, 60, 75, and 90th min after leaving the cryo-chamber. They found that a single WBC treatment may have a minor influence on the short-term anaerobic performance without significant changes in both genders but leads to the improvement of velocity during the start as expressed by a shortened time to obtain the maximal anaerobic power.

An interesting study (Kruger et al., 2015) showed that WBC improves acute recovery during high-intensity intermittent exercise in thermoneutral conditions. In a randomized crossover trial, 11 endurance-trained male athletes performed two ramp-test protocols to individual exhaustion within an hour, interspersed with a high intensity running protocol, consisting of 5 × 5 min at 90% of maximum velocity with 4 min of active recovery between the intervals. During the recovery period, which lasted 1 h, the subjects were randomly assigned to WBC (3 min, −110°C) or control condition (3 min slow walk). The authors found that the difference in the time from the beginning of the ramp tests to the individual exhaustion of the participants was significantly lower during WBC intervention than in the control group. In addition, the rating of perceived exertion (RPE) values was lower at submaximal intensities, as well as for oxygenation of the vastus lateralis muscle, heart rate, and peak oxygen uptake (VO_{2peak}) values.

On the other hand, Broatch et al. (2019) involved 22 well-trained men in a two-group parallel research where the participants performed 4 weeks of cycling high-intensity interval training (HIT), with each training session followed by a 3 min of WBC exposure (−110°C) or to passive control. The authors measured basal adrenal hormones changes, sleep patterns, and performance tests, such as a graded exercise test, a time to-exhaustion test, a 20-km time trial, and a 120-min submaximal test. The statistical analysis did not show significant effects of WBC on the performance parameters, suggesting that a regular post-exercise cryostimulation is not an effective strategy to increase the training-induced aerobic adaptations to 4 weeks of HIT. Considering this information, further studies are required to elucidate the influence of cryostimulation on aerobic and anaerobic mechanisms.

Strength

Despite the increasing popularity of cryostimulation in sport medicine, very few studies have investigated the acute or long-term effects of very-low temperatures on the muscle strength performance, while, on the contrary, there are significantly more studies on the influence of cryostimulation as a recovery technique after strength exercises (Rose et al., 2017).

In a very recent article, Jaworska et al. (2020) found an improvement in the average power and isokinetic extension muscle strength when combining cryostimulation with resistance training. Twenty-five volunteers completed a 4-week protocol which included 12 sessions of resistance training lasting ∼50 min, focused on the lower limbs, each session followed either by WBC exposure (3 min, −120°C) or passive recovery. They observed that training combined with WBC induced a significant increase of maximal average power in the knee isokinetic extension strength test, while in the control group, the level of the aforementioned test remained unchanged. These changes were accompanied by a drop in the myostatin and IL-15 concentration in the experimental group. In the current study, the authors did not record changes in muscle mass, therefore, they stated that the increase of isokinetic muscle strength could be connected to a better motor unit recruitment.

In another study, a single session of PBC (3 min, −110°C) was used to evaluate the effects of cryostimulation on the elbow's flexor neuromuscular performance by Ferreira-Junior et al. (2014). They enrolled 13 subjects and exposed them to two different experimental conditions separated by 72 h: a single session of PBC and a control condition (3 min, 21°C). The protocol consisted of a maximal isokinetic elbow flexion test repeated 30 min before and 10 min after each condition. The authors did not find significant differences in peak torque, average power, total work, nor muscle activity between the conditions, suggesting that cryostimulation could be utilized before training or rehabilitation without compromising the neuromuscular performance of the elbow flexors.

In addition, Westerlund et al. (2009) studied the effects of single and repeated WBC sessions (2 min, −110°C) on neuromuscular performances in healthy subjects. The authors enrolled 14 participants and administered the WBC sessions three times a week for 3 months. The neuromuscular performance tests included a drop-jump exercise and a maximal voluntary contraction force of the wrist flexors which were performed before and after the WBC at the beginning (a single WBC session) and at the end of the 3-month study period (repeated WBC sessions). After a single very-low temperature exposure, the flight time decreased significantly, however after repeated WBC, only a similar tendency was found. This adaptation was accompanied by a decreased co-contraction of lower leg muscles during the drop-jump. The maximal force level did not change significantly, either after a single or after repeated WBC sessions. The authors stated that, concerning dynamic exercises, neuromuscular functioning may be able to adapt to repeated WBC exposures.

Concerning isometric strength, Costello et al. (2012) enrolled 36 volunteers in a cross-over study, which included a WBC session (3 min, −110°C) and control condition (3 min, 15°C). All subjects were exposed to both treatments after a lapse of 2 h. They were asked to complete maximal voluntary isometric contraction (MVIC) of the right knee extensors. The baseline tests were completed immediately before the WBC session and post-tests and followed immediately after each exposure and again 15 min later for both of the temperature conditions. MVIC was recorded in a sitting position: the participants were asked to maximally contract their right leg for 3 s. They completed the protocol three times and the maximum value of MVIC was recorded. The authors did not find significant differences in MVIC between the groups following treatment suggesting that the WBC treatment did not affect MVIC of the knee extensors.

De Nardi et al. (2017) reported an improvement in the concentric hand-grip strength after PBC. Enrolling a consistent number of participants (200 healthy adults divided both in a PBC (150 s, −130/−160°C) and in the control group (150 s, 22°C), the authors administered a baseline handgrip strength test before each condition. Immediately after the exposure in the cabin, both groups performed a subsequent handgrip test. Data showed that both the groups exhibited an increase in their handgrip strength values, especially the PBC group. The authors concluded that

PBC could also be performed before a training session or a sports event.

The results mentioned above show that WBC and PBC exposures are not deleterious in strength performance. WBC exposures performed after resistance training could even have positive effects on the strength development through a better motor unit recruitment.

Flexibility

A variety of studies (Ma et al., 2013; Giemza et al., 2015; Romanowski and Straburzynska-Lupa, 2020) have found favorable outcomes in the improvement of range of motion (ROM) and flexibility after one or multiple cryo-exposures. Ma et al. (2013) studied the effects of a cycle of 24 WBC sessions (3 min, −110°C) on the active ROM of flexion, abduction, internal, and external rotation of the shoulder in subjects suffering from adhesive capsulitis of the shoulder. The authors divided 30 patients into two groups: the WBC group received physical therapy modalities, passive joint mobilization of the shoulder, and cryostimulation, whereas the control group received only physical therapy modalities and passive join mobilization of the shoulder. Each group showed a significant improvement in the ROM measures from baseline to the end of the study, with the WBC group showing a significant difference for active ROM of flexion, abduction, internal rotation, and external rotation compared with the controls.

On the other hand, Romanowski and Straburzynska-Lupa (2020) evaluated the beneficial supplement to exercise therapy of cryostimulation on functional parameters, such as spine mobility and chest expandability in patients with ankylosing spondylitis. Curiously, they divided 92 patients in the three groups, evaluating the effects of two different WBC modalities (−60 and −110°C) combined with an exercise therapy and respectively with exercise therapy alone. After an 8-day period, they demonstrated that the −110°C WBC group manifested a significant improvement in spine mobility and chest expandability.

Giemza et al. (2015) evaluated the effects of frequent WBC (3 min, −120°) treatments on back pain therapy in elderly men, focusing their attention on the lumbo-thoracic spine mobility, measuring active flexion and extension, rotation to the right and the left, and lateral flexion to the right and the left. The patients who exercised and underwent WBC five times a week showed a significant increase in the range of the lumbar spine mobility.

De Nardi et al. (2015) studied the acute effects of a single session of PBC (150 s, −130/−160°C) on sit-and-reach amplitude in a population of both genders, enrolling 60 men and 60 women. Male and female were in turn randomly divided into two groups: the PBC group and the control group (150 s, 21°C) and after an initial sit-and-reach test, they were exposed to the experimental condition. Immediately after the experimental session, both groups performed another sit-and-reach test. The results showed that the PBC group improved their sit-and-reach amplitude to a greater extent than the control group, leading to the conclusion that the ROM is increased immediately after a single session of cryostimulation.

In a very recent paper, the same group of authors (De Nardi et al., 2020), studied the impact of different sessions of PBC (150 s, −130/−160°C) on trunk- and lower limbs flexibility in a sample of young women. To evaluate the flexibility responses to the interventions, they proposed three tests: standing hamstring, stretch test, sit-and-reach test, and active knee extension test. For this study, 11 healthy women were enrolled and were randomly subjected to the four conditions (hamstring stretching in an upright position; hamstring stretching in an upright position in conjunction with whole-body vibration; PBC; and rest, alias sitting position). All of the aforementioned dependent variables were measured before and immediately after each experimental condition. Concerning active knee extension, the outcomes revealed a significant improvement in ROM after PBC in comparison to the control.

Even if each aforementioned study on very-low temperatures exposures lead to a positive effect on the flexibility and ROM, we agree with Bleakley and Costello (2012), highlighting that further studies are needed to determine the *in vivo* effect of WBC/PBC on ROM, flexibility and tissue stiffness, splitting, among the other issues, between exposure to wet and dry cold, which is the typical condition of WBC/PBC.

WBC—PBC and Sleep

Sleep is probably one of the most important phases in the recovery of an athlete. Unfortunately, many elite athletes suffer from sleep problems and the accumulated risk of sleep debt can lead to the risk of injury and increase the prevalence of overtraining (Lastella et al., 2018; Sargent et al., 2021). It has been proposed that cryostimulation may lead to improved sleep. Currently, there are still few studies on the subject, however the existing data is encouraging. Indeed, Bouzigon et al. (2014) report that exposure to cryostimulation improves the perception of sleep in high-level basketball players. This finding was also reported by Schaal et al. who showed that repeated cryostimulation exposure during a phase of intense training improves the perception of sleep in a group of female swimmers (Schaal et al., 2014). In addition to these perceptive data, Douzi et al. reported that exposure to cryostimulation (immediately after a training session) is associated with a reduction in the number of movements during the night (measured through actigraphy), which could mean a better quality of sleep (Douzi et al., 2018, 2019). All these results are encouraging but need to be confirmed.

CRYOSTIMULATION: FOE OR FRIEND

The use of cold application in the context of sports recovery has received special attention and several studies have drawn attention to the fact that exposure to cold can be harmful to the training adaptations and therefore lead to negative consequences on physical performance. These studies were mainly carried out with cold baths and examined whether the repeated use of cooling applications after endurance training and resistance training influenced physical performance and molecular and

morphological adaptations of skeletal muscle. For further details, we encourage reading the excellent review by Hyldahl and Peake (2020). Currently, the use of repeated cold applications appears to affect the strength and/or aerobic performance underlying that the physiological adaptations may occur in different ways. Indeed, Malta et al. (2021) very recently reported that exposure to repeated cold could lead to negative consequences on strength performance but not on aerobic performance. A review by Ihsan et al. (2021) showed that a "recovery periodization" using cold therapies may be of importance to improve the performances of athletes. Nevertheless, the authors advised to avoid CWI following a training focused on improving the muscle strength or hypertrophy.

The available data on the potential consequences of repeated exposure to cold on the physiological adaptations to aerobic training appear to show no negative effects on aerobic performance. Only one study to date has shown that CWI in six sedentary subjects reduced adaptations to aerobic training. The cold exposure in this study consisted of 20 min at 5°C, four times per week. This protocol seems to be in contrast with the recommendations of Machado et al. (2016) who proposed immersion in cold water for 10–15 min at 10–15°C. Later, the studies have shown non-damaging effects of cold exposure on aerobic performance (Halson et al., 2014; Broatch et al., 2019). The studies have sometimes reported that the cold can lead to better muscle perfusions and even mitochondrial biogenesis (Hyldahl and Peake, 2020). However, these results need to be confirmed. Regarding cryostimulation, only Broatch et al. (2019) reported no effect of cryotherapy on the increase in VO_2max. To date, the exposure to cold does not seem to be contraindicated for aerobic performance and seems to be suitable in a case of important fatigue.

Concerning the impact of cold exposure on the physiological adaptations of muscular training, Yamane et al. (2006) provided the first evidence that repeated immersion in cold water after resistance training attenuated grip strength gains while not influencing changes in muscle endurance. Since this study, several studies have confirmed the risk of using cold exposure after strength training (Hyldahl and Peake, 2020). Malta et al. (2021) in their recent meta-analysis confirmed this finding. This decrease in muscle performance may be associated with a negative effect of cold on muscle hypertrophy and protein synthesis. However, as Hyldahl and Peake (2020) suggested, these findings generally indicate that regular cooling attenuates chronic gains in strength following traditional resistance training, whereas it has no influence on short-term adaptation after muscle-damaging exercise. It has even been shown that during weeks with an intense training load, the cold immersion can reduce the risks of maladaptation to training (Tavares et al., 2019). These cold baths might even be recommended in the phases of intense training where there is not enough time to recover. It is therefore important to periodize the use of cold in recovery according to the objectives. For example, during periods of significant muscular work aimed at hypertrophy, cold is not recommended. However, during periods of overload training, during periods of tapper or competition, it may be recommended. However, one needs to keep in mind that all these studies were conducted using the cold baths.

Nevertheless, a very recent study (Jaworska et al., 2021) showed that the use of WBC daily after exercise training in 13 Judokas of the National Judo Team of Poland during a 10-day traineeship did not alter muscle performance, sustained muscle function, and slightly improved specific judo abilities. The authors explained the differences with the results shown with the cold bath by the duration between the end of the exercises and the exposures (2 h for the cryo vs. immediately after the cold bath). Another very recent study demonstrated that the combination of 12 resistance training sessions with WBC induced a positive and likely significant improvement of isokinetic muscle strength. The authors showed that the resistance training combined with cold exposure modified the muscle strength through the modulation of myostatin and IL-15 concentrations (Jaworska et al., 2020).

Some investigations are required to increase the knowledge concerning the effect of cryostimulation exposure on muscular function and recovery. It is not relevant to draw general conclusions such as "it works" or "it does not work." The cryostimulation can be efficient in some aspects and not in others.

INDICATIONS—CONTRAINDICATIONS OF WBC—PBC

Cryostimulation application must adhere to the precise guidelines and indications. There are some contraindications which can limit its field of applications (Lombardi et al., 2017; as shown in the **Appendix A**). However, it should be underlined that the indications and contraindications come from the empirical application of the treatment and theoretical precautions (e.g., the potential deleterious effects of cold in cardiovascular diseases), rather than from the established guidelines that are still lacking, as well as approval from the national and supranational regulatory agencies. Some safety concerns exist, as partially revised by Costello et al. (2015). Indeed, while WBC is generally performed in cryochambers labeled as medical devices, the safety concerns are limited and mainly account for the patient-related reasons that escaped the anamnesis of physicians, PBC is mainly performed in non-medical centers with non-medical devices and, hence, safety rules are less stringent. Further, the direct exposure to liquid nitrogen vapors is related to the potential risk of asphyxia. The cold burns and cold urticaria represent the most common adverse events. However, some rare cases with significant health problems have been reported during and after the exposure (Camara-Lemarroy et al., 2017; Carrard et al., 2017; Greenwald et al., 2018).

In general, cryostimulation must be performed in controlled conditions in the presence of specifically formed personnel. WBC is considered to be a safe procedure and no deleterious

effects have been reported neither for the lungs (Smolander et al., 2006), regardless of the inspiration of cold air, nor for the heart (Banfi et al., 2009a). Precaution might be taken regarding the treatment of subjects affected by cardiovascular diseases since the previous studies reported a slight, although clinically irrelevant increase in systolic blood pressure (Lubkowska and Szygula, 2010). PBC needs more attention and control because the individuals are in direct contact with nitrogen. Nitrogen should not be inspired, and the direct nitrogen jet increases the risk of frostbite. Consequently, cryostimulation is not indicated for the patients with unstable angina pectoris, cardiac failure in III and IV stages according to the New York Heart Association (NYHA), the United States (Lombardi et al., 2017).

CRYOSTIMULATION PROTOCOLS: PROCEDURE SUGGESTIONS AND RECOMMENDATIONS

In the case of a new subject wishing to use cryostimulation exposure, an intake consultation is highly recommended. An intake consultation is the first interaction and consists of an intake interview and intake session. An intake interview should be designed to address the purpose and needs of the users, exclude the absolute- and relative contraindications (**Appendix A**), determine the profiles of the users, and assess the preferences of users. An intake session, a first mild-dosage session, provides the operator information on how the user responds to the cryostimulation and helps to get acquainted. At the end of an intake session, the treatment settings and cycle should be recommended.

To ensure effective, repeatable, and standardized effects, the application of cryostimulation entails a fixed procedure tailored to the needs and personal profile of an individual. As there is no one-size-fits-all solution, and there are many "moving parts" in this decision-making, it is crucial to gather feedback, observe progress, and adjust throughout the treatment cycle.

Recommendations:

1. Although cryostimulation is a safe treatment with no reported adverse side effects in proper usage, it may have health risks in certain medical conditions. Therefore, it is imperative to exclude the absolute and relative contraindications before each session, as the health condition of users is a safety and liability issue.
2. The cryo-operator should explain the methodology and safety precautions to each person before each session, as the person changes into the proper safety attire consisting of headwear, footwear, a mask, and gloves.
3. The type, severity, and stage of the symptoms should determine the cold dose; more acute, severe symptoms require more significant skin temperature drops and/or more regular exposures. Too little cryo exposure will not produce the desired results, while too much may become harmful.
4. Cryostimulation should not be a one-size-fits-all approach. A one-size-fits-all approach will be less safe, is considered one of the reasons for the lack of consistency and significant benefits in the research, and creates a considerable variation in how cryostimulation is perceived. Multiple studies illustrate significant differences in the outcomes based on the personal profile differences, such as body composition, gender, training status, age, skin type, and responsiveness (Cholewka et al., 2012; Hammond et al., 2014; Cuttell et al., 2017; Polidori et al., 2020; Kujawski et al., 2021). For instance, women cool down quicker than men and therefore require a milder cold dose. Different approaches are recently validated (Polidori et al., 2016; Broede et al., 2017) using the thermophysiological models that take these personal differences into account and standardize the effects.
5. A fixed number of sessions within a constrained time frame to obtain optimal benefits of optimal individuals is recommended (as shown in Section Cryostimulation Protocols). For instance, after intense activity, the cycle for performance recovery purposes should contain 1–5 sessions, with at least two sessions within 24 h. On the other hand, the cycle for treating moderate, rheumatoid arthritis complaints requires 20 consecutive sessions, one session per day for 4 weeks.
6. Habituation throughout a cycle is evident and should be acknowledged (Louis et al., 2020). In case of occurrence, the thermal shock will be less amplified (Yurkevicius et al., 2021). Consider increasing the treatment dose over time to prevent this from happening.
7. Real-time monitoring of the physiological and thermal responses combined with personal feedback will further optimize the complete WBC approach. For example, one could assess the thermal profile of individuals by taking pre- and post-blood pressure (i.e., the parasympathetic response), pre-and post-skin temperature measurements, and considering the reactions and susceptibility of individuals, such as shivering, cold pain, body language during the exposure, and the thermal comfort of the clients.
8. The treatment cycle should be fitted within the existing regime of the indication, whole-body cryo should not be considered as an act on its own.

CONCLUSION

This position/consensus work should be seen as a companion paper of Bouzigon et al. (2016), Lombardi et al. (2017), Dupuy et al. (2018), and the information note published by the International Institute of Refrigeration (Dugué et al., 2020), but here focused on the potential use of WBC and PBC for recovery purposes after exercising in the athletes. The review globally confirms the interest in the use of the cryostimulation in sport field. However, as expressed in our recently published information note, more information and data should be available to fully justify and understand the benefits of cryostimulation. The diversity of the cold apparatus that is used as well as the protocol discrepancies and the very different circumstances in which data have been collected retain to provide good robust evidence of the benefits. There are

several issues, such as the dose effect/treatments to optimize the protocols (depending on the exposure, the desired effect, and the characteristics of the subject) as well as safety issues that should be tackle.

In Pubmed, the number of published works concerning WBC is over 700 and the number related to the sub-topic of recovery of athletes is <60. These numbers are globally quite low but interestingly, in the latter case, more than one-third of the articles have been published in the past 3 years reflecting a growing understanding of the importance of WBC in the recovery process of the athletes.

AUTHOR CONTRIBUTIONS

RB, OD, and BD contributed to manuscript revision. RB, OD, BD, IT, MD, J-PB, TM, DT, EM, and GL wrote sections of the manuscript. All authors contributed to manuscript revision, read, and approved the submitted version.

SUPPLEMENTARY MATERIAL

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fspor.2021.688828/full#supplementary-material

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