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Hot- and Cold-Water Immersion Do Not Alter Performance or Perceived Fatigability but Improve Muscle Activation, Cardiac Vagal Modulation, and Cardiorespiratory Recovery After Distinct Running Protocols

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ABSTRACT

This study investigated the effects of cold-water immersion (11°C, $CWI_{11°}$) and hot-water immersion (41°C, $HWI_{41°}$) on components of fatigability after distinct high-intensity endurance running protocols. Participants completed either continuous running at the velocity associated with the respiratory compensation point (vRCP; $CONT_{100\%RCP}$, $n = 12$) or intermittent running at 50% above vRCP ($HIIT_{150\%RCP}$, $n = 10$). Assessments were performed at baseline, immediately after, and at 2, 4, and 24 h postexercise. These included voluntary and evoked knee-extensor contractions, countermovement jumps, mood state, cardiac autonomic modulation, and cardiorespiratory and perceptual responses during submaximal brief runs. Compared to placebo and regardless of preceding running protocol, $CWI_{11°}$ enhanced cardiac vagal modulation at 2 h postexercise (condition × time interaction, $p < 0.05$), whereas $HWI_{41°}$ reduced oxygen consumption rate during submaximal brief runs within 24 h postexercise (condition effect, $p < 0.05$). Furthermore, compared to placebo, $HWI_{41°}$ increased *vastus lateralis* activation during maximal voluntary contractions ($RMS/M\text{-}wave_{amp}$) 2 and 4 h after the $CONT_{100\%RCP}$, while $CWI_{11°}$ increased it at the same time points after the $HIIT_{150\%RCP}$ (condition × time interactions, $p < 0.05$). The efficacy of $CWI_{11°}$ and $HWI_{41°}$ as recovery interventions after running depended on the specific component of fatigability being assessed and the preceding exercise protocol. While $CWI_{11°}$ increased cardiac vagal modulation and $HWI_{41°}$ reduced oxygen consumption rate in post-intervention submaximal brief runs, neither intervention improved knee-extensor voluntary or involuntary peak force, perceptual responses, or mood disturbance. Both $HWI_{41°}$ and $CWI_{11°}$ enhanced muscle activation during maximal voluntary contractions; however, this benefit was observed with $HWI_{41°}$ after continuous endurance running and with $CWI_{11°}$ following high-intensity intermittent running.

1 | Introduction

High-intensity and endurance running, key components of training in many individual and team sports [1, 2], impose significant metabolic and mechanical stresses that induce fatigability [3, 4]. Fatigability is a transient state characterized by a decline in force-generating capacity (i.e., assumed as performance fatigability), sensations of tiredness and soreness (i.e., assumed as perceived fatigability), and homeostatic perturbations, including cardiac autonomic nervous system imbalance [3]. Depending on

its severity, fatigability may persist from minutes to days, potentially compromising performance and increasing the risk of injury [5]. Cold-water immersion (CWI) and hot-water immersion (HWI) have been proposed to mitigate fatigability and promote sustained performance and well-being in training and competition [6–9]. However, given the well-established physiological effects of CWI and HWI [6, 8–11], these interventions are unlikely to have a uniform influence across all running scenarios and components of fatigability [12, 13].

For example, running at intensities far exceeding the maximum metabolic steady state (MMSS; high-intensity exercises) can exacerbate exercise-induced microtrauma to muscles and connective tissues [14]. These events may, in turn, intensify contractile function impairments [15], muscle soreness [15], and cardiac autonomic nervous system imbalance [16] following exercise. Given these components of fatigability, it can be argued that CWI may be a more effective intervention than HWI in mitigating fatigability [12, 13]. CWI primarily decreases muscle temperature and blood flow, which, combined with hydrostatic pressure, initiates secondary events [17]. These secondary events include the redistribution of blood flow from the periphery to the core and the activation of the transient receptor potential cation channel M8 (TRPM8) [17]. Collectively, these processes may attenuate short-term inflammation [18], reduce impairments in contractile function [6, 19], attenuate muscle soreness [10], and enhance cardiac vagal modulation [20]. Moreover, because exercises above MMSS accelerate core temperature increases [21], CWI is unlikely to trigger disturbances related to undue heat strain [22], which can occur when combining high-intensity exercises with HWI [23]. However, decreased muscle temperature may eventually impair the restoration of voluntary peak force and power hours after exercise cessation [24]. Therefore, while CWI can be beneficial for some components of fatigability (e.g., cardiac vagal modulation), it may have a concurrent detrimental effect on other components (e.g., rapid force-production capacity) [8, 24].

Conversely to high-intensity exercises, exercising at intensities near the MMSS induces less severe metabolic disturbances within the exercised muscles [14, 25], along with attenuated postexercise impairments in both contractile function [15] and cardiac autonomic nervous balance [16]. HWI is known to increase muscle temperature [26, 27]. This thermal effect, combined with secondary mechanisms such as the inhibition of transient receptor potential vanilloid channels (e.g., TRPV 1 and TRPV4) [28] and improved sarcoplasmic reticulum Ca^{2+} release and myofibrillar Ca^{2+} sensitivity [24], may offer greater benefits than CWI for force-generating capacity [8, 29], performance maintenance [30], and muscle soreness [8, 28]. Therefore, using HWI following exercise at intensities close to the MMSS may be a more effective, low-risk intervention for mitigating fatigability than CWI [13, 23]. Nonetheless, HWI can also reduce cardiac vagal modulation [22, 31], potentially exacerbating homeostatic disturbances postexercise [23] while concurrently benefiting other components of fatigability (e.g., voluntary peak force) [8].

Although the literature extensively explores the protocols and mechanisms of CWI and HWI efficacy [6, 8–11], less attention has been given to understanding which exercise scenarios and components of fatigability may benefit most from these interventions [12, 13]. To our knowledge, a comprehensive investigation into how the efficacy of CWI and HWI in mitigating fatigability varies across different types of exercise and specific components of fatigability remains lacking. This knowledge could help tailor these methods based on the specific exercise protocol and fatigability components targeted for improvement. Thus, this study aimed to investigate the effects of CWI and HWI on fatigability after two distinct running protocols: (i) continuous running at a velocity associated with the respiratory compensation point (RCP; $CONT_{100\%RCP}$), and (ii) intermittent running at 50% above RCP-associated velocity ($HIIT_{150\%RCP}$). A comprehensive assessment of fatigability was performed, including measures of global, central, and peripheral markers of neuromuscular function, countermovement jumps performance, mood state, and heart rate variability (HRV). We also monitored cardiorespiratory and perceptual responses during submaximal brief runs, as this provides an ecological assessment of systemic disturbance caused by fatigability [32]. We hypothesized that CWI would be more effective in mitigating fatigability after $HIIT_{150\%RCP}$, while HWI would be more effective after $CONT_{100\%RCP}$. We further hypothesized that CWI would particularly benefit cardiac vagal modulation and perceived fatigability markers, whereas HWI would enhance fatigability components associated with force-generating capacity.

2 | Methods

A crossover design was employed to investigate the effects of CWI and HWI. To ensure participant adherence, given the demanding procedures involved in the research (e.g., multiple peripheral electrical stimulation sessions and eight laboratory visits lasting 6 h each), the investigation was separated into two distinct crossover studies. Study 1 evaluated the effects of CWI and HWI on fatigability after $CONT_{100\%RCP}$, while Study 2 examined their effects on the same outcomes following $HIIT_{150\%RCP}$. All reported results and conclusions are derived from analyses performed within each study; no direct statistical comparisons were made between the two studies.

2.1 | Participants and Ethics Approval

Twelve males gave written consent to participate in Study 1 (mean ± SD; age: 26 ± 7 years; stature: 177 ± 7 cm; body mass: 74.2 ± 6.3 kg; running frequency in the last 6 months: 3 ± 1 days/week; running volume in the last 6 months: 21.8 ± 8.2 km/week; maximal oxygen uptake [$\dot{V}O_{2max}$]: 48.5 ± 3.4 mL/kg/min; and velocity associated with the RCP: 11.4 ± 1.2 km/h), and 10 males to participate in Study 2 (mean ± SD; age: 24 ± 5 years; stature: 175 ± 4 cm; body mass: 74.6 ± 6.5 kg; running frequency in the last 6 months: 3 ± 1 days/week; running volume in the last 6 months: 24.2 ± 8.6 km/week; $\dot{V}O_{2max}$: 49.5 ± 4.5 mL/kg/min; and velocity associated with the RCP: 11.1 ± 0.9 km/h). The participants in both studies fall into Tier 1 (recreationally/trained) according to the framework set out by McKay et al. [33]. A 30%–50% attrition rate was adopted to account for the high dropout rate observed in previous studies by our research group using similar procedures. Participants were included if they were not undergoing medical or nutritional supplementation treatments in the 6 months preceding the study, and if they had no known neurological, cardiovascular, or musculoskeletal disorders that

could potentially affect the outcomes evaluated in this investigation. The studies were approved by the São Paulo State University Research Committee (Number 50075421.9.0000.5398), and all procedures were conducted in accordance with the Declaration of Helsinki.

2.2 | Experimental Design

The sample size of this investigation was calculated based on the meta-analysis by Choo et al. [34]. Specifically, Choo et al. [34] reported an increase in strength-related outcomes after endurance exercise when CWI was used compared to passive rest (Hedges' $g = 0.45$). This Hedges' g value was converted to a partial eta squared (η_p^2) of 0.048, using a previous suggested equation [35]. Based on this η_p^2 and an alpha of 0.05, a minimum of twelve participants for each study was required to achieve a power of 0.80 in f tests, according to the G*power 3.1 software [36]. The input parameters for the software were *ANOVA: repeated measures, within factors*; *Effect size f: 0.225* (as determined by the software using the cited η_p^2 value); *Number of groups: 4; Number of measurements: 5; Correlation among repeated measures: 0.8* (a ballpark value for neuromuscular function outcomes according to data from our research group [37]); *nonsphericity correction: 1; Options: as in Gpower 3.0*.

The experimental design is presented in Figure 1. The studies were very similar, differing only in the running trials: Study 1, $CONT_{100\%RCP}$; Study 2, $HIIT_{150\%RCP}$. Both studies consisted of 9 visits to the laboratory performed within 5–6 weeks. The first visits were used to evaluate anthropometric characteristics, assess cardiorespiratory fitness through a graded exercise test (GXT), and familiarize the participants with the experimental procedures. On visits 2, 4, 6, and 8, participants performed $CONT_{100\%RCP}$ or $HIIT_{150\%RCP}$, depending on the study. After the exercise was completed, participants underwent either HWI, CWI, a placebo intervention, or passive rest (control condition) in a randomized order. On visits 3, 5, 7, and 9, participants visited

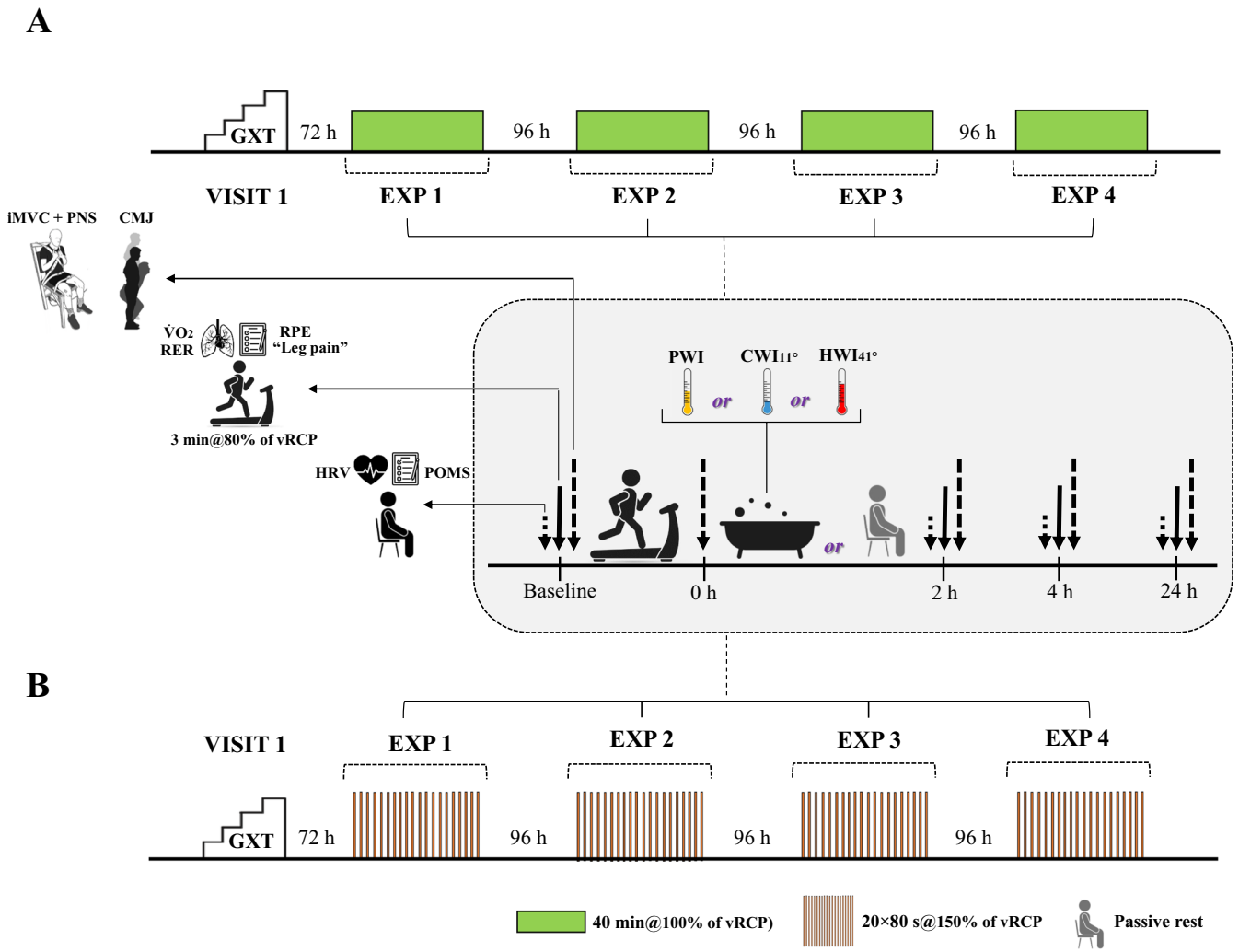

FIGURE 1 | Schematic representation of the investigation design. CMJ, countermovement jump; $CWI_{11°}$, immersion in a water at 11°C for 15 min; EXP, experimental trials consisted of two visits to the laboratory, spaced 24 h apart; GXT, graded exercise test to assess cardiorespiratory function; HRV, heart rate variability; $HWI_{41°}$, immersion in a water at 41°C for 45 min; iMVC, maximal voluntary isometric contraction of knee-extensor (dominant lower limb, self-reported); passive rest, 15 min sitting in a chair; PNS, peripheral electrical stimulation; POMS, profile of mood state questionnaire; PWI, immersion in colored thermoneutral water (placebo) for 10 min; RER, respiratory exchange ratio; RPE, rate of perceived effort; $\dot{V}O_2$, rate of oxygen consumption; vRCP: Velocity associated with the respiratory compensation point.

the laboratory for 24 h follow-up evaluations. Neuromuscular assessments comprising isometric maximal voluntary contraction (iMVC) of knee-extensor, peripheral nerve electrical stimulation (PNS), and countermovement jump (CMJ) performance analysis were performed at baseline, immediately after exercise, 2, 4, and 24 h postexercise. HRV and mood state were assessed at baseline, 2, 4, and 24 h postexercise. In addition, brief submaximal runs with monitoring of cardiorespiratory and perceptual responses were performed at baseline, 2, 4, and 24 h postexercise. All running efforts (i.e., GXT, $CONT_{100\%RCP}$, $HIIT_{150\%RCP}$, and submaximal runs) were performed on a motorized treadmill (ATL, Inbramed, Brazil). Prior to all visits, participants were instructed to refrain from consuming food and caffeinated beverages for at least 2 and 8 h, respectively. To ensure a similar dietary pattern across trials, participants completed a detailed 24 h dietary recall immediately before and after the first experimental trial (Visit 2). They were then required to replicate this exact dietary pattern for all subsequent visits. Additionally, participants were required to abstain from vigorous physical activity for 24 h prior to all visits. The day before each laboratory visit, participants received a text message reminder regarding adherence to these instructions, which all participants confirmed they followed. Furthermore, Visits 2, 4, 6, and 8 (and consequently visits 3, 5, 7, and 9) were conducted at the same time of the day (±1 h) for each participant in an environmentally controlled room (temperature: 20°C–22°C; humidity 40%–60%). These room temperature and humidity ranges were chosen in accordance with prior guidelines for fitness testing [38], and because such conditions are considered a "thermoneutral" zone that does not induce changes in metabolic heat production or evaporative heat loss [39].

2.2.1 | Exercise Protocols

2.2.1.1 | Graded Exercise Test. The GXT was performed to determine the $\dot{V}O_{2max}$, RCP, and the velocity associated with the RCP. The GXT started at 8 km/h, and the velocity was increased by 1.5 km/h every 2 min until task failure. The GXT protocol, previously used by our research group [40, 41], was developed according to prior recommendations [42, 43] to assess maximal and submaximal physiological and work rate parameters during running. Five minutes after the end of the GXT, participants performed a constant-load exercise until task failure at a velocity 5% greater than the highest velocity achieved during GXT. This effort was performed to add confidence in the establishment of a $\dot{V}O_2$ response more likely to reflect the true $\dot{V}O_{2max}$ [44]. In both cases, task failure was determined when participants disengaged from the effort despite receiving strong verbal encouragement.

2.2.1.2 | $CONT_{100\%RCP}$ and $HIIT_{150\%RCP}$. The $CONT_{100\%RCP}$ consisted of 40 min continuous run effort at the velocity associated with the RCP, while the $HIIT_{150\%RCP}$ consisted of 20 × 80 s running bouts at 50% above RCP-associated velocity, interspaced by 160 s of passive rest. The duration of the bouts in $HIIT_{150\%RCP}$ was chosen aiming to match the estimated distance covered by the participants between $CONT_{100\%RCP}$ and $HIIT_{150\%RCP}$ (i.e., distance in km = velocity in km/h × exercise time in hours). Blood samples from the earlobe (25 μL) were collected 3 and 5 min after the end of all $CONT_{100\%RCP}$ and $HIIT_{150\%RCP}$. These samples were used to evaluate the peak blood lactate concentration ($[La]^-$) after exercise using a biochemical analyzer YSI 2900 (Yellow Spring Instruments, Ohio, USA).

2.2.1.3 | Submaximal Brief Runs. To better evaluate systemic disturbances caused by fatigability, we assessed cardiorespiratory and perceptual responses during submaximal brief runs, alongside surrogate markers of fatigability (e.g., maximal force-generating capacity, CMJ performance, perceptual measures) [32]. The runs lasted 3 min and were performed at 80% of the velocity associated with the RCP. This intensity and duration were selected to induce *meaningful* stress in the body's systems while minimizing the risk of exacerbating fatigability [32].

2.2.2 | Recovery Interventions

Immediately after the postexercise assessments (nearly 20 min after exercise cessation), participants performed the assigned recovery interventions. The control condition (CON) consisted of passive rest, during which participants seated in a comfortable chair for 10 min. For the HWI, CWI, and placebo interventions, participants were seated with legs fully extended in an inflatable bath (120 cm × 55 cm × 50 cm, VG plus, Brazil) with the water level set to the xyphoid process. Water temperature was assessed every 5 min using a digital thermometer. For the HWI intervention ($HWI_{41°}$), participants were immersed in water at 41.2°C ± 0.6°C for 45 min. This protocol was selected for its demonstrated efficacy in elevating core and muscle temperature, as well as improving contractile function [11, 26], processes considered beneficial for mitigating fatigability [8, 29]. For the CWI intervention ($CWI_{11°}$), participants were immersed in water at 11.4°C ± 0.5°C for 15 min. This duration and temperature have been shown to attenuate the postexercise decline in force-generating capacity [45], reduce muscle soreness [10], and enhance cardiac vagal modulation [20]. Notably, although $HWI_{41°}$ and $CWI_{11°}$ differed in duration, this parameter was selected to deliver an effective "exogenous therapeutic dose" of each recovery intervention based on existing literature [8, 18, 26, 27, 45, 46]. For the placebo intervention (PWI), participants were immersed for 10 min in colored water at a neutral temperature of 26.1°C ± 0.8°C. A pH-balanced water coloring agent (Pinta água, Color Brinque, Brazil) was added to the water immediately before immersion, a technique based on a prior study [47]. Participants were informed that the substance was being tested in the laboratory and that the manufacturers claimed it could "accelerate recovery after exercise." They were also told that it was not possible to provide details about the substance's mechanisms of action or the manufacturer until the study was published. High-resolution thermal images of the anterior thigh were captured using a FLIR C3-X camera (FLIR Systems, USA; infrared resolution: 128 × 96 pixels, emissivity: 0.95, accuracy: ±3%). Images were obtained immediately before and after the recovery interventions. The camera was activated 10 min prior to data collection and positioned perpendicular to the thigh at 1 m from the participant. Before post-intervention measurements, the skin was gently dried with a towel. During image acquisition, participants stood wearing only their underwear. All images were analyzed using FLIR Tools+ software (FLIR Tools+, FLIR Systems, USA) by the same researcher

[48]. After all recovery interventions, participants waited for postexercise assessments in a quiet, temperature-controlled room (20°C–22°C; 40%–60% humidity) inside the laboratory. During this period, participants were permitted to engage in personal activities (e.g., using smartphones, laptops, or books for work/study) and use the restroom, but were prohibited from engaging in physical activity, napping, or showering. To qualitatively assess the feelings/beliefs regarding PWI, $HWI_{41°}$, and $CWI_{11°}$, the participants were surveyed after completing all visits. They were asked two questions: (i) "Which method do you believe was most helpful for improving postexercise recovery?"; and (ii) "Which method do you believe was least helpful for improving postexercise recovery?"

2.3 | Data Collection and Analysis

2.3.1 | Respiratory Data

Breath-by-breath pulmonary gas exchange and ventilation ($\dot{V}_E$) were continuously measured during GXT and submaximal brief runs using a metabolic cart (Quark, CPET, COSMED, Italy). Breath-by-breath data were examined to exclude errant breaths, and those values lying more than ±3 SD from the local mean were removed [49]. The breath-by-breath data were subsequently interpolated every 1 s [49] using the software OriginPro 8.0 (Origin Lab Corporation, United States). The $\dot{V}O_{2max}$ was computed as the highest 20 s rolling average during the GXT. When the highest $\dot{V}O_2$ average from the GXT was similar to the highest $\dot{V}O_2$ average recorded during the constant-load exercise (difference lower than ±2.1 mL/kg/min), it was considered to be a $\dot{V}O_2$ response that more likely reflects the true $\dot{V}O_{2max}$ [49]. All participants of both studies reached a $\dot{V}O_2$ response that more likely reflects the true $\dot{V}O_{2max}$. For the estimation of RCP, raw profiles of $\dot{V}_E$, carbon dioxide output ($\dot{V}CO_2$), their combination (i.e., $\dot{V}_E/\dot{V}CO_2$), and end-tidal pressure of CO_2 ($PetCO_2$) were plotted against time. RCP was identified as the point at which $PetCO_2$ began a precipitous fall after a period of isocapnia concomitantly with a second and first breakpoint in the $\dot{V}_E$ and $\dot{V}_E/\dot{V}CO_2$ relationships, respectively [49]. The velocity associated with the RCP was identified by projecting of RCP (i.e., the time at which it occurs) onto the GXT stages. During the brief submaximal runs, the $\dot{V}O_2$ and respiratory exchange ratio (RER) were computed from 20 s rolling averages. The highest $\dot{V}O_2$ and RER averages were recorded and further expressed as a percentage change from baseline.

2.3.2 | Neuromuscular Function

2.3.2.1 | Knee-Extensor Function Outcomes. Participants sat in a custom chair maintaining 90° flexion at the hips and knees. They performed 5 s iMVCs of the dominant knee-extensor, with PNS applied during force production and at rest. The force signal was acquired using a load cell (MK Controle, Brazil) attached to the ankle by a metal rod immediately above the region of the medial malleolus. The force signal was acquired at a sampling rate of 2000 Hz using a PowerLab system (16/35, ADInstruments, New Zealand). The surface electromyography activity (EMG) signal from the *vastus lateralis* was recorded during effort and at rest. The EMG was recorded through a pair of self-adhesive Ag/AgCl surface electrodes (Meditrace 100, USA) placed on the 1/3 distal of the muscle. The reference electrode was placed on the patella. Before electrode placement, the skin was shaved, lightly abraded, and cleaned with rubbing alcohol. The EMG signal was acquired at a sampling rate of 2000 Hz using an amplifier FE136 (ADInstruments, New Zealand) coupled with a PowerLab system (16/35, ADInstruments, New Zealand). Both force and EMG signals were registered by LabChart 8 software (ADInstruments, New Zealand).

Femoral nerve stimulations (pulse duration: 1 ms; square wave) were delivered by a high-voltage electrical stimulator (Bioestimulador V2 400 V peak-to-peak; Insight, São Paulo, Brazil) on the femoral triangle (cathode) and at the femoral greater trochanter level (anode). To assess voluntary activation (VA_{PNS}), doublet high frequency stimulations (100 Hz) were delivered at the visual peak force of the iMVC effort (~2–3 s after iMVC started) as well as at 3 s after iMVC. Contractile properties were evaluated using single and doublet low frequency (10 Hz) stimulations delivered to the relaxed muscle at 7 and 10 s after iMVC, respectively. Stimulus intensity was individually determined in each visit by applying twitches to relaxed muscle, starting at 30 mA, and increasing in 30 mA increments until force plateau (267 ± 57 mA across Study 1 and 281 ± 74 mA across Study 2). The final PNS intensity was set 30% above this threshold to ensure full activation of the muscle. In Study 1, postexercise assessments were conducted 66 ± 5 s after exercise cessation, whereas in Study 2, assessments occurred 69 ± 7 s afterwards.

The highest voluntary force recorded within a 100 ms period during the force plateau of the iMVC was defined as the peak voluntary force ($iMVC_{Force}$) and used as a global marker of neuromuscular function. The superimposed force of the doublet evoked during the iMVC, and the force evoked by the potentiated doublet at rest after iMVC ($Db100_{Force}$) were used to measure the VA_{PNS} using the following equation: VA_{PNS} (%) = [1 − (superimposed force) × (force level at stimulation/iMVCForce)/($Db100_{Force}$)] × 100 [50]. The potentiated peak force evoked by the single stimulus (Qtw_{pot}) was used as a marker of contractile function. The ratio between the force evoked by the doublet low frequency stimulus ($Db10_{Force}$) and $Db100_{Force}$ (i.e., $Db10_{Force}/Db100_{Force}$ ratio) was calculated and used as a marker of low frequency fatigue. For EMG analysis, the signal was band-pass filtered (20–500 Hz). The amplitude of EMG response evoked by the single stimulus (M-wave amplitude) was recorded as an indicator of the sarcolemma's capacity to propagate action potentials. The root mean square of the *vastus lateralis* EMG signal (RMS_{VL}) was determined over a 0.5 s period around the $iMVC_{Force}$. The ratio between RMS_{VL} and M-wave amplitude (i.e., $RMS/M\text{-}wave_{amp}$) was calculated to estimate the activation of *vastus lateralis* (i.e., propagation of electrical signals from the central nervous system to the muscle) [51]. All analysis procedures were performed using the LabChart 8 software. All data were expressed as a percentage change from baseline.

2.3.2.2 | Countermovement Jump Performance. Participants performed 5 CMJs interspaced by 20 s rest on a force plate (Cefise, Nova Odessa, Brazil). Force plate signal (1000 Hz) was acquired using the PowerLab system (16/35, ADInstruments, New Zealand) and recorded with LabChart 8 software (AD

Instruments, New Zealand). The force plate was calibrated prior to each experimental visit using six known weights to establish a linear relationship between force (N) and voltage output (mV). Calibration was accepted if the regression coefficient (r) was ≥0.99. For offline analysis, the data were initially filtered with a fourth-order Butterworth filter (low-pass cutoff frequency 25 Hz) using LabChart 8 software (AD Instruments, New Zealand). A custom-made Matlab algorithm (MathWorks, Natick, USA), provided in the Appendix S1, was then used to calculate the CMJ height, concentric peak force, and power using the equations described by Linthorne et al. [52]. The average obtained from the five CMJ trials was calculated. All data were expressed as a percentage change from baseline.

2.3.3 | Heart Rate Variability

Measurements of HRV were taken at rest while participants sat alone in a dimly lit, quiet room for 6 min, breathing spontaneously. The HRV signal was acquired using a Bio-amplifier FE136 (ADInstruments, New Zealand) and a PowerLab system (16/35, ADInstruments, New Zealand) via self-adhesive Ag-AgCl surface electrodes (Meditrace 100, Covidien, USA) in a modified lead-II arrangement [53]. The signal was recorded using LabChart 8 software (AD Instruments, New Zealand), which was also used for the offline calculation of the root mean square of successive R–R interval differences (RMSSd) from the final 5 min of the signal. The RMSSd was expressed as a percentage change from baseline.

2.3.4 | Perceptual Responses

The mood of the participants was assessed using the Profile of Mood State Questionnaire (POMS) [54]. The Total Mood Disturbance (TMD) score, a summary of positive mood derived by adding subscales and subtracting vigor, was calculated. Higher scores on the TMD indicate a greater mood disturbance [54]. TMD was expressed as a percentage change from baseline. During brief submaximal runs, rating of perceived exertion (RPE) and rating of pain in the lower limbs were recorded immediately after cessation of effort. RPE was assessed by 6–20 top-to-bottom Borg's scale [55], while exercising leg pain was assessed by 0–10 top-to-bottom Borg's scale [56]. RPE and leg pain were expressed as *raw* values. All perceptual responses were evaluated in a quiet, climate-controlled room (20°C–22°C; 40%–60% humidity), with only the participant and the investigator present.

2.4 | Statistical Analysis

The SPSS software package, version 25.0 (IBM Corp., Armonk, NY) was used for all statistical analyses. Statistical significance was set at α of 0.05. All data are presented as mean ± SD within the text and figures. Normality of the data was verified at each time point in each condition using the Shapiro–Wilk test. A one-way repeated measures ANOVA was used to compare the peak blood $[La]^-$ between trials in each study (i.e., CONT, PWI, $CWI_{11°}$, and $HWI_{41°}$). A two-way repeated measures ANOVA (condition and time) was used to assess the effect of recovery interventions over time in each study. Assumptions of sphericity were explored and controlled for all variables with the Greenhouse–Geisser adjustment, where necessary. If significant main effects or interactions (time × condition) were observed, a Bonferroni post hoc pairwise comparison was performed. The η_p^2 with 95% confidence interval (95% CI) was calculated to estimate effect sizes, with values of 0.01, 0.06, and 0.14 representing small, moderate, and large effects, respectively. The complete set of descriptive statistics for all data is available in the Appendix S1.

3 | Results

3.1 | Study 1

The $CONT_{100\%RCP}$ was performed at 11.4 ± 1.2 km/h (73.5% ± 5.0% of $v\dot{V}O_{2max}$) and had a volume of 7.5 ± 0.8 km. No significant differences were observed in peak blood $[La]^-$ across conditions ($p = 0.62$, $\eta_p^2 = 0.02$). The skin temperature of the anterior area of the thigh increased after CON and $HWI_{41°}$; and decreased after PWI and $CWI_{11°}$ (condition × time interaction: $p < 0.01$; $\eta_p^2 = 0.94$ [95% CI: 0.86–0.96]; post hoc: $p < 0.05$). These results are summarized in Table 1. Of the 12 participants, 9 considered $CWI_{11°}$ as the "most helpful" recovery intervention, while 2 chose PWI and 1 chose $HWI_{41°}$. For the "least helpful" recovery intervention, 8 participants selected $HWI_{41°}$ and 4 chose PWI.

3.1.1 | Effects of $CWI_{11°}$ and $HWI_{41°}$ on Neuromuscular Function

Compared to baseline, $_iMVC_{Force}$ and VA decreased immediately after exercise and remained reduced for at least 24 h (Figure 2A,B—time effects: $p \leq 0.01$; $\eta_p^2 \geq 0.49$; post hoc: $p < 0.05$). Qtw_{pot} and $Db_{10Force}/Db_{100Force}$ presented lower values compared to

TABLE 1 | Peak of blood lactate concentration after continuous running at the velocity associated with the respiratory compensation point and skin temperature of the anterior area of the thigh before and after recovery interventions.

	CON	PWI	$CWI_{11°}$	$HWI_{41°}$
$[La]^-_{Peak}$ (mmol/L)	2.8 ± 1.4 (1.95 to 3.70)	2.8 ± 2.1 (1.66 to 4.07)	2.9 ± 2.3 (1.52 to 4.34)	2.9 ± 2.1 (1.82 to 4.12)
Skin temperature (°C)	*Before*: 30.8 ± 1.4	*Before*: 30.5 ± 1.1	*Before*: 30.3 ± 1.9	*Before*: 29.4 ± 1.1
	After: 32.6 ± 1.4*	*After*: 27.2 ± 3.7*	*After*: 17.3 ± 1.6*	*After*: 35.5 ± 1.6*

Note: Data are presented as mean ± standard deviation (95% confidence interval). $[La]^-_{Peak}$: peak of blood lactate concentration after exercise. *$p < 0.05$, different from before (condition × time interaction effect in a two-way repeated measure ANOVA, post hoc $p < 0.05$).
Abbreviations: CON, passive rest (control); $CWI_{11°}$, immersion in water at 11°C for 15 min; $HWI_{41°}$, immersion in water at 41°C for 45 min; PWI, immersion in colored water at neutral temperature (placebo).

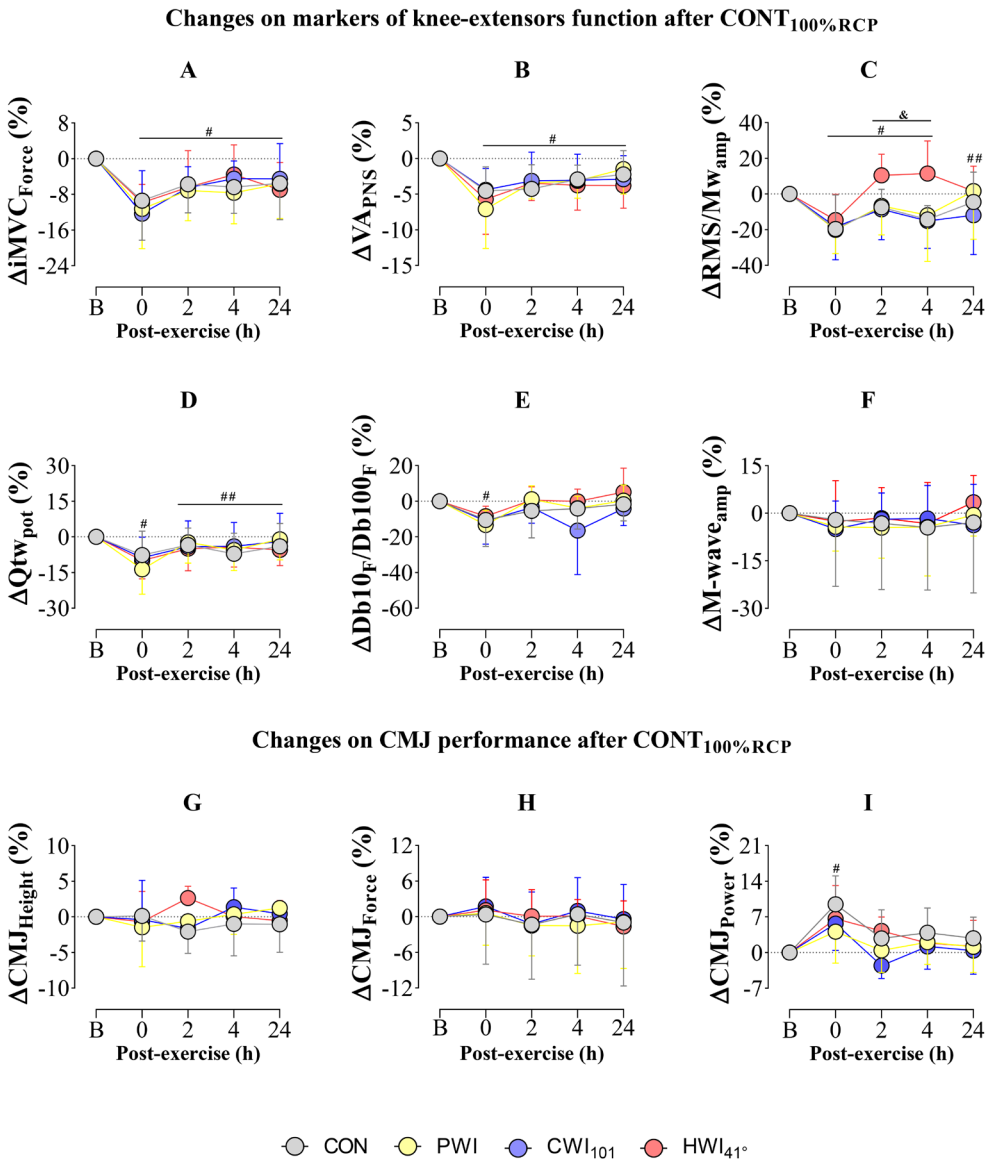

FIGURE 2 | Percentage change from baseline on knee-extensor function outcomes and countermovement jump performance. $CONT_{100\%RCP}$, 40 min continuous running at velocity associated with the respiratory compensation point. B, baseline assessments; CMJ_{Height}, CMJ_{Force}, and CMJ_{Power}, average of the height, force and power recorded in 5 CMJs; CON, passive rest (15 min sitting in a chair); $CWI_{11°}$, immersion in water at 11°C for 15 min; $Db10_{Force}/Db100_{Force}$, ratio between the force evoked by a supramaximal high frequency (100 Hz) and low frequency (10 Hz) doublet stimulus at relaxed knee-extensor; $HWI_{41°}$, immersion in water at 41°C for 45 min; $iMVC_{Force}$, highest voluntary force recorded during maximal voluntary isometric contractions of knee-extensor; PWI, immersion in colored thermoneutral water (placebo) for 10 min; Qtw_{pot}, force evoked by a potentiated single stimulus at relaxed knee-extensor; $RMS/M\text{-}wave_{amp}$, ratio between the root mean square of electromyography signal from *vastus lateralis* during iMVC (0.5 s around $iMVC_{Force}$) and peak-to-peak M-wave amplitude; VA_{PNS}, voluntary activation measured through twitch interpolation technique. The statistical reports come from a two-way repeated measures ANOVA. #, different from baseline (main effect of time, $p < 0.05$, post hoc $p < 0.05$); ##, different from 0 h (main effect of time, $p < 0.05$, post hoc $p < 0.05$); &, $HWI_{41°}$ different from CON, PWI and $CWI_{11°}$ (condition × time interaction, $p < 0.05$, post hoc $p < 0.05$). $N = 12$.

baseline only immediately postexercise, while $RMS_{VL}/M\text{-}wave_{amp}$ remained reduced for up to 4 h postexercise (Figure 2C–E—time effects: $p \leq 0.02$; $\eta_p^2 \geq 0.37$; post hoc: $p < 0.05$). The $RMS/M\text{-}wave_{amp}$ was higher 2 and 4 h after exercise when used $HWI_{41°}$ compared to CON, PWI and $CWI_{11°}$ (condition × time interaction: $p < 0.01$; $\eta_p^2 = 0.18$ [95% CI: 0.02 to 0.23]; post hoc: $p < 0.01$). Power of CMJ increased immediately postexercise (Figure 2I—time effect: $p < 0.01$; $\eta_p^2 = 0.59$; post hoc: $p < 0.05$). No other time, condition, or interaction effects were observed for the remaining knee-extensor's function or CMJ performance indicators ($p > 0.07$; $\eta_p^2 < 0.18$).

3.1.2 | Effects of $CWI_{11°}$ and $HWI_{41°}$ on Cardiac Vagal Modulation, Mood State, and Responses During Submaximal Runs

The cardiac vagal modulation (i.e., RMSSd) decreased only immediately postexercise (Figure 3A—time effect: $p < 0.01$; $\eta_p^2 = 0.75$; post hoc: $p < 0.05$). It was higher 2 h postexercise when used $CWI_{11°}$ compared to CON, PWI and $HWI_{41°}$ (condition × time interaction: $p < 0.01$; $\eta_p^2 = 0.34$ [95% CI: 0.16–0.40]; post hoc: $p = 0.01$). $POMS_{TMD}$ remained decreased for 4 h after

exercise (Figure 3B—time effect: $p < 0.01$; $\eta_p^2 = 0.38$; post hoc: $p < 0.05$). There was no condition ($p = 0.06$; $\eta_p^2 = 0.17$) or interaction effects ($p = 0.08$; $\eta_p^2 = 0.12$) for $POMS_{TMD}$.

The $\dot{V}O_2$ was higher for at least 4 h compared to baseline values (Figure 3C) while RER remained lower for at least 24 h postexercise (Figure 3D—time effects: $p < 0.01$; $\eta_p^2 \geq 0.58$; post hoc: $p = 0.01$). A condition effect ($p = 0.02$; $\eta_p^2 = 0.26$ [95% CI: 0.01–0.43]; post hoc: $p < 0.01$) evidenced that increases in $\dot{V}O_2$ after exercise were lower when used $HWI_{41°}$ compared to CON, PWI, and $CWI_{11°}$. No condition effect was observed for RER ($p = 0.37$; $\eta_p^2 = 0.09$), nor were there interaction effects

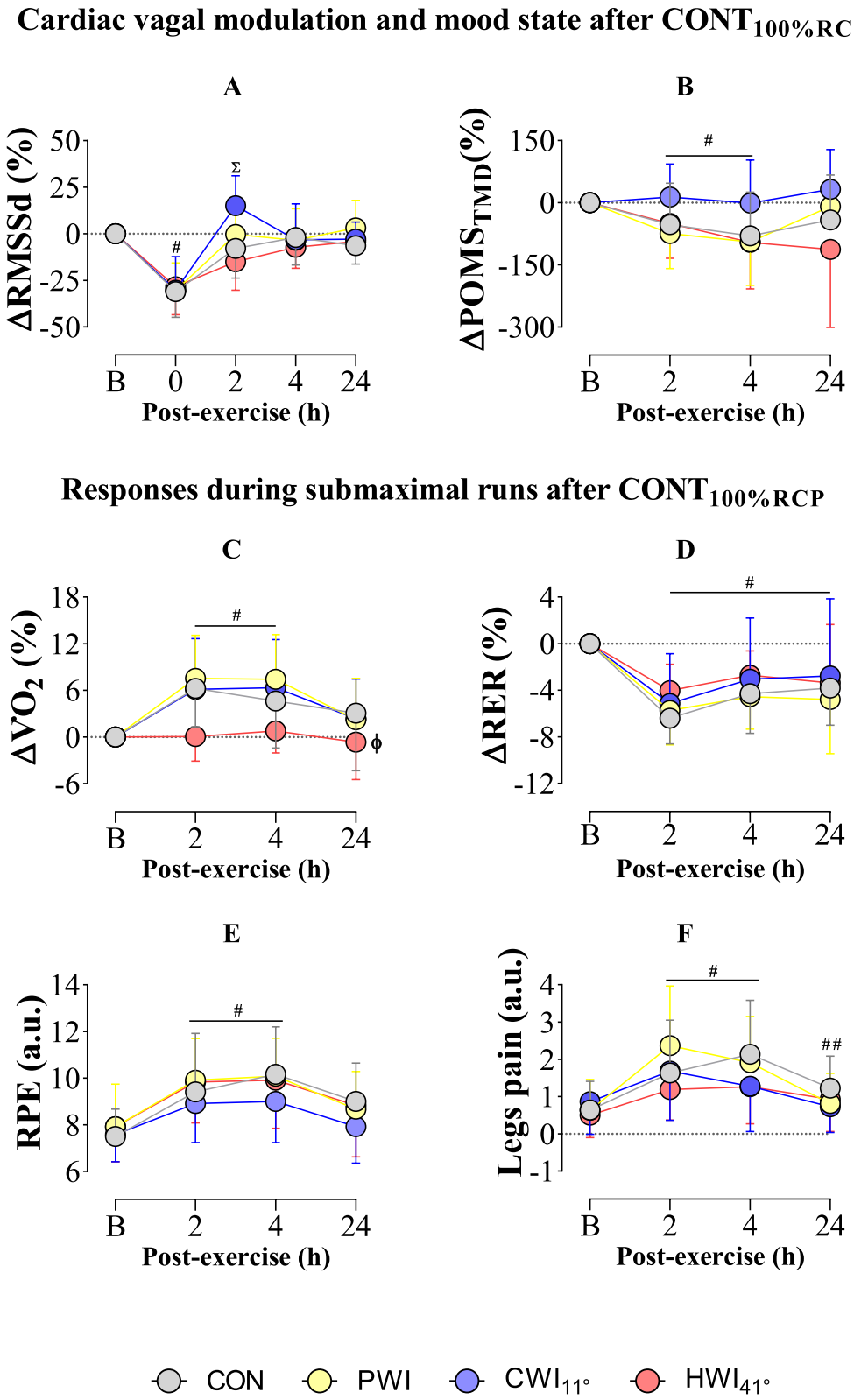

FIGURE 3 | Legend on next page.

FIGURE 3 | Cardiac vagal modulation, mood state, cardiorespiratory and perceptual responses during submaximal runs (3 min @ 80% of velocity associated with the respiratory compensation point). B, baseline assessments; $CONT_{100\%RCP}$, 40 min continuous running at velocity associated with the respiratory compensation point; CON, passive rest (control condition; 15 min sitting in a chair); $CWI_{11°}$, immersion in water at 11°C for 15 min; $HWI_{41°}$, immersion in water at 41°C for 45 min; $POMS_{TMD}$, total mood disturbance calculated through the profile of mood state questionnaire; PWI, immersion in colored thermoneutral water (placebo) for 10 min; RER, respiratory exchange ratio; RMSSd, root mean square of successive R-R interval differences; RPE, rate of perceived effort; $\dot{V}O_2$, rate of oxygen consumption. The statistical reports come from a two-way repeated measures ANOVA. #, different from baseline (main effect of time, $p < 0.05$, post hoc $p < 0.05$); ##, different from 0 h (main effect of time, $p < 0.05$, post hoc $p < 0.05$); Σ, $CWI_{11°}$ different from CON, PWI and $HWI_{41°}$ (condition × time interaction, $p < 0.05$); Φ, $HWI_{41°}$ different from CON, PWI and $CWI_{11°}$ (main effect of condition, $p < 0.05$, post hoc $p < 0.05$). $N = 12$.

TABLE 2 | Peak of blood lactate concentration after continuous running at velocity associated to respiratory compensation point and skin temperature of anterior area of thigh before and after recovery interventions.

	CON	PWI	$CWI_{11°}$	$HWI_{41°}$
$[La]^-_{Peak}$ (mmol/L)	5.2 ± 0.9 (4.54 to 5.86)	5.6 ± 1.8 (4.39 to 6.80)	5.4 ± 1.4 (4.51 to 6.29)	5.1 ± 0.9 (4.56 to 5.67)
Skin temperature (°C)	*Before*: 30.0 ± 2.2	*Before*: 28.9 ± 2.4	*Before*: 29.3 ± 1.8	*Before*: 28.7 ± 2.3
	After: 31.8 ± 1.5*	*After*: 26.4 ± 2.3*	*After*: 17.3 ± 1.6*	*After*: 36.2 ± 1.5*

Note: Data are presented as mean ± standard deviation (95% confidence interval). $[La]^-_{Peak}$: peak of blood lactate concentration after exercise. *$p < 0.05$, different from before (condition × time interaction effect in a two-way repeated measure ANOVA, post hoc $p < 0.05$).
Abbreviations: CON, passive rest (control); $CWI_{11°}$, immersion in water at 11°C for 15 min; $HWI_{41°}$, immersion in water at 41°C for 45 min; PWI, immersion in colored water at neutral temperature (placebo).

for RER ($p = 0.86$; $\eta^2_p = 0.04$) or $\dot{V}O_2$ ($p = 0.07$; $\eta^2_p = 0.14$). The RPE was higher over 4 h after exercise (Figure 3E), while leg pain remained increased for 4 h postexercise (Figure 3F—time effects: $p < 0.01$; $\eta^2_p \geq 0.53$; post hoc: $p = 0.01$). No condition or interaction effects were observed for RPE or leg pain ($p > 0.06$; $\eta^2_p < 0.18$).

3.2 | Study 2

The $HIIT_{150\%RCP}$ was performed at 16.7 ± 1.3 km/h (105.5 ± 5.7% of $v\dot{V}O_{2max}$) and had a volume of 7.4 ± 0.6 km. No significant differences were observed in peak blood $[La]^-$ across conditions ($p = 0.72$; $\eta^2_p = 0.01$). The skin temperature of the anterior area of the thigh increased after CON and $HWI_{41°}$; and decreased after PWI and $CWI_{11°}$ (condition × time interaction: $p < 0.01$; $\eta^2_p = 0.88$ [95% CI: 0.81–0.93]; post hoc: $p < 0.05$). These results are summarized in Table 2. Of the 10 participants, 7 selected $CWI_{11°}$ as the "most helpful" recovery intervention, while 2 chose PWI and 1 chose $HWI_{41°}$. For the "least helpful" recovery intervention, $HWI_{41°}$ was selected by 5 participants, PWI by 4, and $CWI_{11°}$ by 1.

3.2.1 | Effects of $CWI_{11°}$ and $HWI_{41°}$ on Neuromuscular Function

Compared to baseline, $_iMVC_{Force}$ and VA reduced immediately after exercise and remained decreased for at least 24 h (Figure 4A,B—time effects: $p \leq 0.01$; $\eta^2_p \geq 0.51$; post hoc: $p < 0.05$). The RMS_{VL}/M-$wave_{amp}$ and Qtw_{pot} were reduced immediately after exercise compared to baseline and remained decreased for 2 h (Figure 4C,D—time effect: $p < 0.01$; $\eta^2_p = 0.46$; post hoc: $p < 0.05$). $Db_{10Force}/Db_{100Force}$ presented lower values compared to baseline only immediately postexercise (Figure 4E—time effects: $p \leq 0.02$; $\eta^2_p \geq 0.28$; post hoc: $p < 0.05$). The M-wave amplitude decreased specifically 2 and 4 h postexercise compared to baseline values (Figure 4F—time effect: $p < 0.01$; post hoc: $p < 0.05$). The RMS_{VL}/M-$wave_{amp}$ was higher 2 and 4 h after exercise when used $CWI_{11°}$ compared to CON, PWI, and $HWI_{41°}$ (condition × time interaction: $p < 0.01$; $\eta^2_p = 0.26$ [95% CI: 0.05–0.30]; post hoc: $p < 0.05$). No other condition or interaction effects were observed for the knee-extensor's function outcomes ($p > 0.07$; $\eta^2_p < 0.19$). No time, condition, or interaction effects were observed for height, concentric peak force, or power of CMJ (Figure 4G–I—$p > 0.06$; $\eta^2_p < 0.21$).

3.2.2 | Effects of $CWI_{11°}$ and $HWI_{41°}$ on Cardiac Vagal Modulation, Mood State, and Responses During Submaximal Runs

The cardiac vagal modulation (i.e., RMSSd) remained decreased for 4 h after exercise (Figure 5A—time effect: $p < 0.01$; $\eta^2_p = 0.64$; post hoc: $p < 0.05$), and it was higher 2 h postexercise when used $CWI_{11°}$ compared to CON, PWI, and $HWI_{41°}$ (time × condition interaction: $p < 0.01$; $\eta^2_p = 0.25$ [95% CI: 0.05–0.31]; post hoc: $p = 0.01$). $POMS_{TMD}$ decreased specifically 4 h after exercise (Figure 5B—time effect: $p < 0.01$; $\eta^2_p = 0.39$; post hoc: $p < 0.05$). No condition ($p = 0.39$; $\eta^2_p = 0.10$) or interaction effects ($p = 0.74$; $\eta^2_p = 0.07$) were observed for $POMS_{TMD}$.

The VO_2 increased over 4 h after exercise while RER remained lower for at least 24 h postexercise (Figure 5C,D—time effects: $p < 0.01$; $\eta^2_p \geq 0.67$; post hoc: $p = 0.01$). A condition effect ($p = 0.02$; $\eta^2_p = 0.44$ [95% CI: 0.10–0.60]; post hoc: $p < 0.01$) evidenced that the increases in $\dot{V}O_2$ after exercise were lower when used $HWI_{41°}$ compared to CON, PWI and $CWI_{11°}$. No condition effect was observed for RER ($p = 0.32$; $\eta^2_p = 0.12$); and there were no interaction effects for $\dot{V}O_2$ ($p = 0.06$; $\eta^2_p = 0.17$) or RER ($p = 0.07$; $\eta^2_p = 0.17$). The RPE was higher for 4 h after exercise, while leg

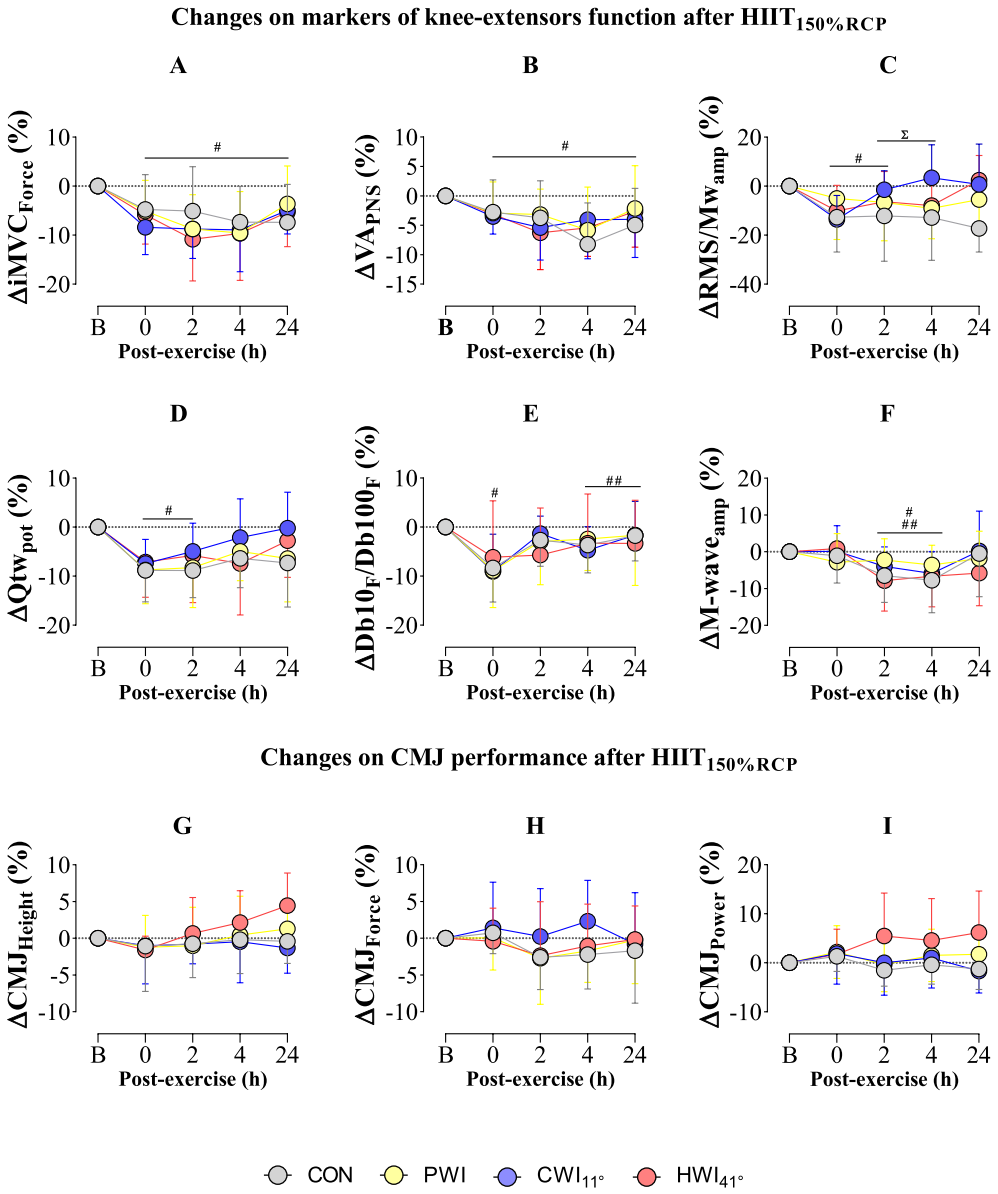

FIGURE 4 | Percentage change from baseline on knee-extensor function outcomes and countermovement jump performance. $HIIT_{150\%RCP}$, 20×80 s running bouts at 50% above the velocity associated with the respiratory compensation point. B, baseline assessments; CMJ_{Height}, CMJ_{Force}, and CMJ_{Power}, average of the height, force and power recorded in 5 CMJs; CON, passive rest (control condition; 15 min sitting in a chair); $CWI_{11°}$, immersion in water at 11°C for 15 min; $Db10_{Force}/Db100_{Force}$, ratio between the force evoked by a supramaximal high frequency (100 Hz) and low frequency (10 Hz) doublet stimulus at relaxed knee-extensor; $HWI_{41°}$, immersion in water at 41°C for 45 min; $iMVC_{Force}$, highest voluntary force recorded during maximal voluntary isometric contractions of knee-extensor; PWI, immersion in colored thermoneutral water (placebo) for 10 min; Qtw_{pot}, force evoked by a potentiated single stimulus at relaxed knee-extensor; $RMS/M\text{-}wave_{amp}$, ratio between the root mean square of electromyography signal from *vastus lateralis* during iMVC (0.5 s around $iMVC_{Force}$) and peak-to-peak M-wave amplitude ($M\text{-}wave_{amp}$); VA_{PNS}, voluntary activation measured through twitch interpolation technique. The statistical reports come from a two-way repeated measures ANOVA. #, different from baseline (main effect of time, $p < 0.05$, post hoc $p < 0.05$); ##, different from 0 h (main effect of time, $p < 0.05$, post hoc $p < 0.05$); Σ, $CWI_{11°}$ different from CON, PWI and $HWI_{41°}$ (condition × time interaction, $p < 0.05$, post hoc $p < 0.05$). $N = 10$.

pain remained increased for 24 h postexercise (Figure 5E,F—time effects: $p < 0.01$; $\eta_p^2 \geq 0.68$; post hoc: $p = 0.01$). No condition ($p > 0.07$; $\eta_p^2 < 0.22$) or interaction effects ($p > 0.39$; $\eta_p^2 < 0.10$) were observed for RPE or leg pain.

4 | Discussion

Neither $CWI_{11°}$ nor $HWI_{41°}$ mitigated exercise-induced impairments in voluntary and involuntary isometric peak force of knee-extensor, mood disturbance, or perceptual responses during submaximal runs. These outcomes were consistent across both running protocols investigated. However, $CWI_{11°}$ and $HWI_{41°}$ enhanced *vastus lateralis* activation during iMVCs (i.e., $RMS/M\text{-}wave_{amp}$) 2 and 4 h postexercise, though the effect depended on the running protocol: $CWI_{11°}$ showed improvement after $HIIT_{150\%RCP}$, while $HWI_{41°}$ was effective after $CONT_{100\%RCP}$. Additionally, irrespective of running protocol, $CWI_{11°}$ increased cardiac vagal modulation (i.e., RMSSd) 2 h postexercise, and $HWI_{41°}$ reduced cardiorespiratory demand

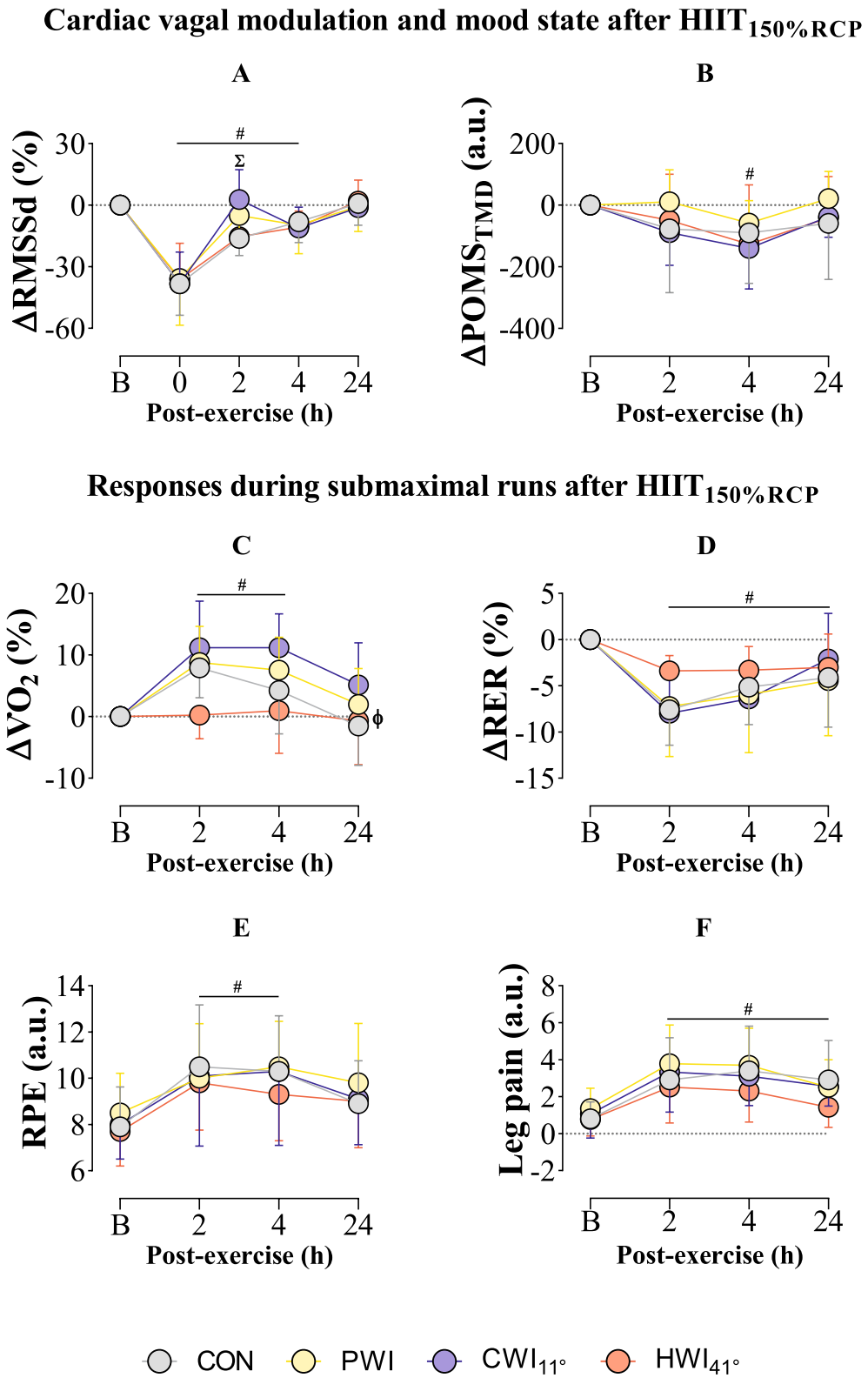

FIGURE 5 | Cardiac vagal modulation, mood state, cardiorespiratory and perceptual responses during submaximal runs (3 min @ 80% of velocity associated with the respiratory compensation point). $HIIT_{150\%RCP}$, 20×80 s running bouts at 50% above the velocity associated with the respiratory compensation point. B, baseline assessment; CON, passive rest (control condition; 15 min sitting in a chair); $CWI_{11°}$, immersion in a water at 11°C for 15 min; $HWI_{41°}$, immersion in a water at 41°C for 45 min; $POMS_{TMD}$, total mood disturbance calculated through the profile of mood state questionnaire; PWI, immersion in colored thermoneutral water (placebo) for 10 min; RER, respiratory exchange ratio; RMSSd, root mean square of successive R-R interval differences; RPE, rate of perceived effort; $\dot{V}O_2$, rate of oxygen consumption. The statistical reports come from a two-way repeated measures ANOVA. #, different from baseline (main effect of time, $p < 0.05$, post hoc $p < 0.05$); Σ, $CWI_{11°}$ different from CON, PWI and $HWI_{41°}$ (condition × time interaction, $p < 0.05$, post hoc $p < 0.05$); Φ, $HWI_{41°}$ different from CON, PWI and $CWI_{11°}$ (main effect of condition, $p < 0.05$, post hoc $p < 0.05$). $N = 10$.

(i.e., $\dot{V}O_2$) during submaximal runs performed within 24 h after exercise. These findings demonstrate, for the first time, that the efficacy of $CWI_{11°}$ and $HWI_{41°}$ in mitigating fatigability after running depends on the preceding exercise protocols and the specific components of fatigability being assessed.

4.1 | Effects of $HWI_{41°}$ and $CWI_{11°}$ on *Vastus Lateralis* Activation During MVCs

Our findings suggest that $HWI_{41°}$ and $CWI_{11°}$ likely triggered a compensatory central nervous system response, increasing activation (RMS_{VL}/Mw_{amp}) to knee-extensor (herein represented by *vastus lateralis*) in an attempt to maintain high force output during the iMVCs postexercise [57]. The running protocol-dependent effects of $HWI_{41°}$ and $CWI_{11°}$ likely stem from differences in the mechanisms underpinning muscle activation reduction following the $CONT_{100\%RCP}$ and $HIIT_{150\%RCP}$ protocols [12].

Previous studies have shown that continued repeated stretch-shortening cycles, such as those in $CONT_{100\%RCP}$, decrease Ia afferent activity, which reduces excitatory input from Ia afferents onto alpha motoneurons [58]. This reduced facilitation has been linked to decreased voluntary peak force and impaired muscle activation [58] and seems to contribute to the reduced muscle activation after running with low-levels of metabolic perturbation [59, 60]. Immersion in water at 40°C–42°C for 45–60 min has been reported to increase muscle temperature by approximately 2°C [26, 27]. The $HWI_{41°}$ used here (41°C for 45 min) would therefore be expected to produce a similar increase. Indeed, the increase of ~6°C in the thigh's skin temperature after $HWI_{41°}$ in Study 1 suggests the temperature of the lower limb was elevated, despite the unknown magnitude of the muscle temperature change. We propose that this likely increase in muscle temperature, aside from other potential HWI-induced effects such as relaxation and stress reduction [23], may have restored Ia afferent fibers reflex facilitation [61]. Thus, by likely mitigating the impairment in Ia afferent fibers reflex facilitation after $CONT_{100\%RCP}$, $HWI_{41°}$ enhanced the sending of electrical signals from the central nervous system to *vastus lateralis* during iMVCs. These findings align with prior research that demonstrates HWI improves the late phase (100–200 ms) rate of force development during concentric contractions [8]. Since muscle activation capacity plays a critical role in the late phase rate of force development [62], HWI's effectiveness in enhancing postexercise explosive strength may stem from its ability to improve muscle activation beyond other mechanisms [23, 63]. Such an assumption, however, requires further investigation.

In contrast to $CONT_{100\%RCP}$, $HIIT_{150\%RCP}$ consisted of high-intensity bouts (~16.7 km/h) interspersed by 160 s rest. Notably, a 90 s rest period appears sufficient to restore Ia afferent facilitation capacity when this reflex is compromised [64]. Therefore, given the inter-bout rest periods in $HIIT_{150\%RCP}$, we argue that Ia afferent facilitation was less compromised during this exercise. Conversely, because $HIIT_{150\%RCP}$ was performed at a high-velocity, it likely resulted in microtrauma to muscle and connective tissues [14]. Although we were unable to obtain more direct measures of muscle microtrauma, the observed reductions in M-wave amplitude 2 and 4 h after $HIIT_{150\%RCP}$ suggest that sarcolemma integrity may have been compromised [65]. Consequently, inhibitory feedback from group III/IV muscle afferents to the central nervous system, which is triggered by events related to muscle microtrauma and inflammation, likely underpins the impaired muscle activation following $HIIT_{150\%RCP}$ [66]. Immersion in water at 8°C–15°C for 10–30 min has been reported to reduce muscle temperature by approximately 7°C [18, 46], which has been linked to the attenuation of muscle microtrauma and inflammation hours postexercise [18]. Thus, by attenuating muscle microtrauma and inflammation, $CWI_{11°}$ (11°C for 15 min) may likely mitigate the negative influence of these events on central nervous system function via group III/IV muscle afferents [66], thereby enhancing muscle activation capacity after $HIIT_{150\%RCP}$. Unfortunately, we were unable to directly assess this potential mechanism of $CWI_{11°}$ in the current study. However, CWI has previously been reported to improve markers of knee-extensor muscle activation after exercise scenarios strongly associated with muscle microtrauma and inflammation, such as soccer and rugby [6, 19]. Our findings suggest that such a beneficial effect also occurs when CWI is used following high-intensity intermittent running. Notably, the benefit of $CWI_{11°}$ on muscle activation was evidenced in comparison to PWI. Although we cannot directly infer changes in muscle temperature, $CWI_{11°}$ decreased the thigh's skin temperature by ~12°C, whereas PWI resulted in only a ~3°C decrease. This suggests a greater cooling effect of the lower limbs after $CWI_{11°}$. Therefore, the specific muscular thermal effect related to CWI protocols such as $CWI_{11°}$ [18, 46], combined with hydrostatic pressure and secondary effects [17], likely serves as the primary mechanism underpinning the efficacy of this intervention on muscle activation. However, additional mechanisms, such as cold-induced change in brain functioning [67] and even a placebo effect [47] cannot be ruled out. Further investigations are needed to better understand the mechanisms underpinning the effectiveness of CWI in enhancing muscle activation after high-intensity, weight-bearing exercises.

4.2 | $CWI_{11°}$ Effects on Cardiac Vagal Modulation at Rest After $CONT_{100\%RCP}$ and $HIIT_{150\%RCP}$

$CWI_{11°}$ increased cardiac vagal modulation (i.e., RMSSd) following both $CONT_{100\%RCP}$ and $HIIT_{150\%RCP}$. According to proposed mechanisms, CWI protocols (immersion in water at 10°C–15°C for 5–10 min) induce peripheral vasoconstriction, which, combined with the hydrostatic pressure effects, increases central blood volume and cardiac preload, thereby enhancing stroke volume and cardiac output [20]. Interestingly, this mechanism seems to require the combined stimulus of hydrostatic pressure and cold-water, as PWI (i.e., immersion in thermoneutral water) did not affect cardiac vagal modulation. These hemodynamic changes activate cardiopulmonary baroreflexes, inhibiting sympathetic activity and increasing cardiac vagal modulation [20]. These mechanisms likely explain the observed effect of $CWI_{11°}$ on cardiac vagal modulation, although cold-induced increase in endorphin release [68] and changes in brain functioning [67] might also play a role in this scenario. Notably, this effect was observed within 2 h after exercise (approximately 90 min posttreatment). Given that the impact of CWI on cardiac vagal modulation appears to last for a few hours after exposure [20] its benefits for

postexercise cardiac autonomic recovery may be limited to a short time frame (~2 h).

4.3 | $HWI_{41°}$ Effects on Cardiorespiratory Demand During Submaximal Runs After $CONT_{100\%RCP}$ and $HIIT_{150\%RCP}$

$HWI_{41°}$ attenuated the cardiorespiratory demand (i.e., decreased $\dot{V}O_2$) during submaximal runs after both $CONT_{100\%RCP}$ and $HIIT_{150\%RCP}$. Koga et al. [69] reported reduced $\dot{V}O_2$ during heavy-domain cycling with simultaneous lower limb passive heating, which increased muscle temperature by ~1.5°C. Our findings expand on this by demonstrating that $HWI_{41°}$, which, based on previous studies [26, 27], is likely to increase muscle temperature to a similar extent, can reduce cardiorespiratory demand during submaximal exercises for hours after the intervention. The most plausible mechanism for the $\dot{V}O_2$ reduction observed in the current study is the well-established increase in muscle contractile function following elevated muscle temperature [11, 26]. This improvement is typically attributed to temperature-regulated enhancements in calcium influx and calcium sensitivity in muscle cells [11, 26]. This increased muscle efficiency consequently reduces the oxygen cost of muscle contraction [69]. Other potential mechanisms for attenuating the cardiorespiratory demand may include temperature-facilitated oxygen unloading from hemoglobin in muscle capillaries [70] and an increase in nitric oxide bioavailability [71]; however, these remain speculative and require further investigation.

4.4 | Further Considerations

While VA measured by the twitch interpolation technique is a key metric, RMS_{VL}/Mw_{amp} is also used to assess changes in motor drive output [51]. Although caution is warranted when interpreting bipolar EMG signals due to technical concerns [72], previous research has demonstrated that RMS_{VL}/Mw_{amp} is more sensitive than VA in detecting changes in motor drive output following activities involving the stretch-shortening cycle [60, 73, 74]. Therefore, even with a mismatch between VA and RMS_{VL}/Mw_{amp} in the current study, our findings provide trustworthy evidence regarding the effect of $CWI_{11°}$ and $HWI_{41°}$ on muscle activation. Notably, the benefits of $CWI_{11°}$ on muscle activation capacity were evident after high-intensity running, which likely induced greater muscle microtrauma [14] and body temperature increases [21] compared to endurance running of a similar distance ($CONT_{100\%RCP}$: 7.5 km and $HIIT_{150\%RCP}$: 7.4 km). Therefore, $CWI_{11°}$ might also elicit a beneficial effect after long-distance endurance running, which can induce significant muscle microtrauma [75] and increase body temperature [76]. Neither $CWI_{11°}$ nor $HWI_{41°}$ improved markers of voluntary or involuntary maximal neuromuscular function (e.g., $_{i}MVC_{Force}$, CMJ performance metrics, Qtw_{pot}, $Db10_{Force}/Db100_{Force}$ ratio). These results were consistent across both running protocols investigated. The absence of benefits from $CWI_{11°}$ or $HWI_{41°}$ on performance fatigability metrics aligns with [7, 9], but contrasts with previous studies [6, 19]. These discrepancies likely arise from differences in exercise protocols (e.g., simulated soccer match vs fatiguing isokinetic contractions). Such variations may influence the underlying mechanisms and characteristics of fatigability [3], thereby affecting how CWI and HWI interact with them [12]. Additionally, neither $CWI_{11°}$ nor $HWI_{41°}$ alleviated mood disturbance following either of the running protocols. We speculate that the potential mood-enhancing effects of these recovery interventions may have been masked by the experimental procedures employed in the investigation, such as prolonged laboratory stays (~5 h after exercise), repeated PNS, and repeated physical testing. Notably, PWI was rated as the "most helpful" by approximately 20% of participants and the "least helpful" by approximately 40% in each study. Despite a carefully designed intervention to match participant beliefs, preexisting biases regarding $HWI_{41°}$ and $CWI_{11°}$ (mainly) appear to be difficult to compensate for when assessing the efficacy of these recovery methods. Therefore, although the muscular thermal effects and their secondary mechanisms reported in the literature when using HWI [8, 26, 27] and CWI [17, 18, 46], likely underpinned the benefits evidenced herein, other mechanisms cannot be ruled out. These include changes in brain functioning [67], increased release of endorphin [68], and the placebo effect for $CWI_{11°}$ [17], as well as relaxation and stress reduction for $HWI_{41°}$ [23].

4.5 | Limitations

The absence of core and intramuscular temperature recordings, alongside measurements of muscle microtrauma and inflammation, must be acknowledged as a limitation of the current study. The lack of these data prevents a more thorough understanding of the mechanisms of $CWI_{11°}$ and $HWI_{41°}$ in the present context. Nevertheless, this limitation does not preclude an interpretation of our findings to be made. Despite this limitation, the data provided by our study might still be highly valuable for tailoring the use of these interventions to mitigate running-induced impairments in applied settings. Additionally, while dynamic contractions are known to be more sensitive to exercise-induced fatigability [3], we assessed knee-extensor function using isometric contractions due to PNS procedures and the widespread use of isometric contraction assessments in neuromuscular research, which enhances reproducibility and facilitates cross-study comparisons. Although CMJ performance was measured to assess neuromuscular function in dynamic contractions, neither running protocol impaired it. The results of the current study were obtained from male participants only, which does not take into account known sex-based differences in exercise-induced fatigability [3]. Future studies are needed to determine potential sex-based differences in the use of CWI and HWI on exercise performance. Finally, we must acknowledge that participant attrition due to repeated absence from testing sessions ($n = 2$) and injuries unrelated to the study protocols ($n = 2$) reduced the final sample sizes (Study 1: $n = 12$; Study 2: $n = 10$). As our a priori power analysis indicated a minimum of $n = 12$ participants per study was required to achieve an 80% power for f tests, Study 2 fell below this threshold. Consequently, both studies, particularly Study 2, may be underpowered to detect small effect sizes, and caution should be exercised when generalizing our outcomes to larger populations.

4.6 | Conclusion

Following $CONT_{100\%RCP}$ and $HIIT_{150\%RCP}$, neither $CWI_{11°}$ nor $HWI_{41°}$ mitigated impairments in knee-extensor voluntary or involuntary peak force, mood disturbance, or changes in

perceptual responses during subsequent submaximal runs. However, $CWI_{11°}$ increased cardiac vagal modulation, while $HWI_{41°}$ reduced cardiorespiratory demand in subsequent submaximal runs. Both interventions also enhanced *vastus lateralis* activation during MVCs, but this benefit was observed with $CWI_{11°}$ following $HIIT_{150\%RCP}$ and with $HWI_{41°}$ following $CONT_{100\%RCP}$.

5 | Perspective

From a practical standpoint, these findings enable the tailored use of $CWI_{11°}$ and $HWI_{41°}$ to mitigate fatigability following running. Irrespective of the preceding running protocol, $CWI_{11°}$ is recommended to enhance cardiac vagal modulation, while $HWI_{41°}$ may be used to improve cardiorespiratory recovery. To address the postexercise reduction in muscle activation capacity, the choice between $CWI_{11°}$ and $HWI_{41°}$ must be guided by the preceding running protocol (i.e., $CONT_{100\%RCP}$ or $HIIT_{150\%RCP}$). Future studies should further investigate the mechanisms underlying the effects of $CWI_{11°}$ and $HWI_{41°}$ reported here, as well as whether such benefits apply to females and larger populations.

Author Contributions

Y.M.D. and A.M.Z. conceived and designed research. Y.M.D. and P.T.M. performed experiments. Y.M.D. and P.T.M. analyzed data. Y.M.D., P.T.M., A.J.C., J.M.M., and A.M.Z. interpreted results of experiments. Y.M.D. prepared figures. Y.M.D. and A.M.Z. drafted manuscript. Y.M.D., P.T.M., A.J.C., J.M.M., and A.M.Z. edited and revised manuscript. Y.M.D., P.T.M., A.J.C., J.M.M., and A.M.Z. approved final version of manuscript.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

References

1. M. Buchheit and P. B. Laursen, "High-Intensity Interval Training, Solutions to the Programming Puzzle: Part I: Cardiopulmonary Emphasis," *Sports Medicine* 43 (2013): 313–338.

2. M. J. MacInnis and M. J. Gibala, "Physiological Adaptations to Interval Training and the Role of Exercise Intensity," *Journal of Physiology* 595 (2017): 2915–2930.

3. M. Behrens, M. Gube, H. Chaabene, et al., "Fatigue and Human Performance: An Updated Framework," *Sports Medicine* 53 (2023): 7–31.

4. R. M. Enoka and J. Duchateau, "Translating Fatigue to Human Performance," *Medicine & Science in Sports & Exercise* 48 (2016): 2228–2238.

5. M. Kellmann, M. Bertollo, L. Bosquet, et al., "Recovery and Performance in Sport: Consensus Statement," *International Journal of Sports Physiology and Performance* 13 (2018): 240–245.

6. M. Bouchiba, N. L. Bragazzi, S. Zarzissi, et al., "Cold Water Immersion Improves the Recovery of Both Central and Peripheral Fatigue Following Simulated Soccer Match-Play," *Frontiers in Physiology* 13 (2022): 860709.

7. J. Gustafsson, D. Montiel-Rojas, M. G. A. Romare, et al., "Cold- and Hot-Water Immersion Are Not More Effective Than Placebo for the Recovery of Physical Performance and Training Adaptations in National Level Soccer Players," *European Journal of Applied Physiology* 125 (2025): 3179–3194.

8. S. Benoît, B. Nicolas, M. P. Grégoire, et al., "Hot but Not Cold Water Immersion Mitigates the Decline in Rate of Force Development Following Exercise-Induced Muscle Damage," *Medicine and Science in Sports and Exercise* 56 (2024): 2362–2371.

9. A. J. Richards, R. Malekzadeh, M. E. Elghobashy, et al., "Cold Water Immersion Does Not Enhance Recovery and Performance After High-Intensity Interval Dorsiflexion Exercise," *Scandinavian Journal of Medicine & Science in Sports* 35 (2025): e70061.

10. A. F. Machado, P. H. Ferreira, J. K. Micheletti, et al., "Can Water Temperature and Immersion Time Influence the Effect of Cold Water Immersion on Muscle Soreness? A Systematic Review and Meta-Analysis," *Sports Medicine* 46 (2016): 503–514.

11. P. Rodrigues, L. B. R. Orssatto, G. S. Trajano, L. Wharton, and G. M. Minett, "Increases in Muscle Temperature by Hot Water Improve Muscle Contractile Function and Reduce Motor Unit Discharge Rates," *Scandinavian Journal of Medicine & Science in Sports* 33 (2023): 754–765.

12. G. M. Minett and J. T. Costello, "Specificity and Context in Post-Exercise Recovery: It Is Not a One-Size-Fits-All Approach," *Frontiers in Physiology* 6 (2015): 130.

13. R. T. Thorpe, "Post-Exercise Recovery: Cooling and Heating, a Periodized Approach," *Frontiers in Sports and Active Living* 3 (2021): 707503.

14. T. Wiewelhove, J. Fernandez-Fernandez, C. Raeder, et al., "Acute Responses and Muscle Damage in Different High-Intensity Interval Running Protocols," *Journal of Sports Medicine and Physical Fitness* 56 (2016): 606–615.

15. D. Iannetta, J. Zhang, J. M. Murias, and S. J. Aboodarda, "Neuromuscular and Perceptual Mechanisms of Fatigue Accompanying Task Failure in Response to Moderate-, Heavy-, Severe-, and Extreme-Intensity Cycling," *Journal of Applied Physiology* 133 (2022): 323–334.

16. J. Stanley, J. M. Peake, and M. Buchheit, "Cardiac Parasympathetic Reactivation Following Exercise: Implications for Training Prescription," *Sports Medicine* 43 (2013): 1259–1277.

17. M. Ihsan, G. Watson, and C. R. Abbiss, "What Are the Physiological Mechanisms for Post-Exercise Cold Water Immersion in the Recovery From Prolonged Endurance and Intermittent Exercise?," *Sports Medicine* 46 (2016): 1095–1109.

18. S. Y. Kwiecien and M. P. McHugh, "The Cold Truth: The Role of Cryotherapy in the Treatment of Injury and Recovery From Exercise," *European Journal of Applied Physiology* 121 (2021): 2125–2142.

19. M. Pointon and R. Duffield, "Cold Water Immersion Recovery After Simulated Collision Sport Exercise," *Medicine and Science in Sports and Exercise* 44 (2012): 206–216.

20. C. Galvez-Rodriguez, P. Valenzuela-Reyes, S. Fuentealba-Sepúlveda, C. Farias-Valenzuela, and A. E. Salinas, "Cold Water Immersion, Heart Rate Variability and Post-Exercise Recovery: A Systematic Review," *Physiotherapy Research International* 30 (2025): e70033.

21. T. H. Lei, I. L. Wang, Y. M. Chen, et al., "Critical Power Is a Key Threshold Determining the Magnitude of Post-Exercise Hypotension in Non-Hypertensive Young Males," *Experimental Physiology* 108 (2023): 1409–1421.

22. T. Peçanha, C. L. M. Forjaz, and D. A. Low, "Additive Effects of Heating and Exercise on Baroreflex Control of Heart Rate in Healthy Males," *Journal of Applied Physiology (1985)* 123 (2017): 1555–1562.

23. C. Menzies, N. D. Clarke, C. J. A. Pugh, C. J. Steward, C. D. Thake, and T. Cullen, "Passive Heating in Sport: Context-Specific Benefits, Detriments, and Considerations," *Applied Physiology, Nutrition, and Metabolism* 50 (2025): 1–15.

24. A. J. Cheng, S. J. Willis, C. Zinner, et al., "Post-Exercise Recovery of Contractile Function and Endurance in Humans and Mice Is Accelerated by Heating and Slowed by Cooling Skeletal Muscle," *Journal of Physiology* 595 (2017): 7413–7426.

25. W. Chidnok, J. Fulford, S. J. Bailey, et al., "Muscle Metabolic Determinants of Exercise Tolerance Following Exhaustion: Relationship to the 'Critical Power'," *Journal of Applied Physiology (1985)* 115 (2013): 243–250.

26. P. Rodrigues, G. S. Trajano, L. Wharton, and G. M. Minett, "Muscle Temperature Kinetics and Thermoregulatory Responses to 42°C Hot-Water Immersion in Healthy Males and Females," *European Journal of Applied Physiology* 120 (2020): 2611–2624.

27. S. H. Faulkner, S. Jackson, G. Fatania, and C. A. Leicht, "The Effect of Passive Heating on Heat Shock Protein 70 and Interleukin-6: A Possible Treatment Tool for Metabolic Diseases?," *Temperature (Austin)* 4 (2017): 292–304.

28. J. S. Petrofsky, I. A. Khowailed, H. Lee, et al., "Cold vs. Heat After Exercise-Is There a Clear Winner for Muscle Soreness," *Journal of Strength and Conditioning Research* 29 (2015): 3245–3252.

29. B. Sautillet, N. Bourdillon, G. P. Millet, et al., "Hot Water Immersion: Maintaining Core Body Temperature Above 38.5°C Mitigates Muscle Fatigue," *Scandinavian Journal of Medicine & Science in Sports* 34 (2024): e14503.

30. R. Solsona, T. Méline, F. Borrani, et al., "Active Recovery vs Hot- or Cold-Water Immersion for Repeated Sprint Ability After a Strenuous Exercise Training Session in Elite Skaters," *Journal of Sports Sciences* 41 (2023): 1126–1135.

31. I. K. Brenner, S. Thomas, and R. J. Shephard, "Spectral Analysis of Heart Rate Variability During Heat Exposure and Repeated Exercise," *European Journal of Applied Physiology and Occupational Physiology* 76 (1997): 145–156.

32. T. Shushan, S. J. McLaren, M. Buchheit, T. J. Scott, S. Barrett, and R. Lovell, "Submaximal Fitness Tests in Team Sports: A Theoretical Framework for Evaluating Physiological State," *Sports Medicine* 52 (2022): 2605–2626.

33. A. K. A. McKay, T. Stellingwerff, E. S. Smith, et al., "Defining Training and Performance Caliber: A Participant Classification Framework," *International Journal of Sports Physiology and Performance* 17 (2022): 317–331.

34. H. C. Choo, M. Lee, V. Yeo, W. Poon, and M. Ihsan, "The Effect of Cold Water Immersion on the Recovery of Physical Performance Revisited: A Systematic Review With Meta-Analysis," *Journal of Sports Sciences* 40 (2022): 2608–2638.

35. J. Cohen, *Statistical Power Analysis for the Behavioral Sciences* (Routledge, 2013), 567.

36. F. Faul, E. Erdfelder, A. G. Lang, and A. Buchner, "G*Power 3: A Flexible Statistical Power Analysis Program for the Social, Behavioral, and Biomedical Sciences," *Behavior Research Methods* 39 (2007): 175–191.

37. Y. M. Dutra, J. P. F. Lopes, J. M. Murias, and A. M. Zagatto, "Within- and Between-Day Reliability and Repeatability of Neuromuscular Function Assessment in Females and Males," *Journal of Applied Physiology (1985)* 135 (2023): 1372–1383.

38. B. Ferguson, "ACSM'S Guidelines for Exercise Testing and Prescription 9th ed. 2014," *Journal of the Canadian Chiropractic Association* 58 (2014): 328.

39. B. Kingma, A. Frijns, and W. van Marken Lichtenbelt, "The Thermoneutral Zone: Implications for Metabolic Studies," *Frontiers in Bioscience (Elite Edition)* 4 (2012): 1975–1985.

40. E. S. Malta, V. H. F. Lopes, M. R. Esco, and A. M. Zagatto, "Repeated Cold-Water Immersion Improves Autonomic Cardiac Modulation Following Five Sessions of High-Intensity Interval Exercise," *European Journal of Applied Physiology* 123 (2023): 1939–1948.

41. A. M. Zagatto, J. A. M. González, R. A. B. de Poli, F. A. Barbieri, L. d. L. S. Bloedow, and L. Peyré-Tartaruga, "Mechanical Energy on Anaerobic Capacity During a Supramaximal Treadmill Running in Men: Is There Influence Between Runners and Active Individuals?," *Physiological Reports* 11 (2023): e15564.

42. D. J. Bentley, J. Newell, and D. Bishop, "Incremental Exercise Test Design and Analysis: Implications for Performance Diagnostics in Endurance Athletes," *Sports Medicine* 37 (2007): 575–586.

43. E. T. Howley, D. R. Bassett, and H. G. Welch, "Criteria for Maximal Oxygen Uptake: Review and Commentary," *Medicine and Science in Sports and Exercise* 27 (1995): 1292–1301.

44. D. Iannetta, R. de Almeida Azevedo, C. P. Ingram, D. A. Keir, and J. M. Murias, "Evaluating the Suitability of Supra-POpeak Verification Trials After Ramp-Incremental Exercise to Confirm the Attainment of Maximum O_2 Uptake," *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology* 319 (2020): R315–R322.

45. E. Moore, J. T. Fuller, J. D. Buckley, et al., "Impact of Cold-Water Immersion Compared With Passive Recovery Following a Single Bout of Strenuous Exercise on Athletic Performance in Physically Active Participants: A Systematic Review With Meta-Analysis and Meta-Regression," *Sports Medicine* 52 (2022): 1667–1688.

46. W. Gregson, M. A. Black, H. Jones, et al., "Influence of Cold Water Immersion on Limb and Cutaneous Blood Flow at Rest," *American Journal of Sports Medicine* 39 (2011): 1316–1323.

47. J. R. Broatch, A. Petersen, and D. J. Bishop, "Postexercise Cold Water Immersion Benefits Are Not Greater Than the Placebo Effect," *Medicine and Science in Sports and Exercise* 46 (2014): 2139–2147.

48. D. G. Moreira, J. T. Costello, C. J. Brito, et al., "Thermographic Imaging in Sports and Exercise Medicine: A Delphi Study and Consensus Statement on the Measurement of Human Skin Temperature," *Journal of Thermal Biology* 69 (2017): 155–162.

49. D. A. Keir, D. Iannetta, F. Mattioni Maturana, J. M. Kowalchuk, and J. M. Murias, "Identification of Non-Invasive Exercise Thresholds: Methods, Strategies, and an Online App," *Sports Medicine* 52 (2022): 237–255.

50. V. Strojnik and P. V. Komi, "Neuromuscular Fatigue After Maximal Stretch-Shortening Cycle Exercise," *Journal of Applied Physiology (1985)* 84, no. 1 (1998): 344–350.

51. G. Y. Millet, V. Martin, A. Martin, and S. Vergès, "Electrical Stimulation for Testing Neuromuscular Function: From Sport to Pathology," *European Journal of Applied Physiology* 111 (2011): 2489–2500.

52. N. P. Linthorne, "Analysis of Standing Vertical Jumps Using a Force Platform," *American Journal of Physics* 69 (2001): 1198–1204.

53. M. R. Esco, A. A. Flatt, and F. Y. Nakamura, "Agreement Between a Smartphone Pulse Sensor Application and Electrocardiography for Determining lnRMSSD," *Journal of Strength and Conditioning Research* 31 (2017): 380–385.

54. C. J. Beedie, P. C. Terry, and A. M. Lane, "The Profile of Mood States and Athletic Performance: Two Meta-Analyses," *Journal of Applied Sport Psychology* 12 (2000): 49–68.

55. G. A. Borg, "Psychophysical Bases of Perceived Exertion," *Medicine and Science in Sports and Exercise* 14 (1982): 377–381.

56. E. Borg, G. Borg, K. Larsson, M. Letzter, and B. M. Sundblad, "An Index for Breathlessness and Leg Fatigue," *Scandinavian Journal of Medicine & Science in Sports* 20 (2010): 644–650.

57. C. Froyd, F. G. Beltrami, G. Y. Millet, and T. D. Noakes, "Central Regulation and Neuromuscular Fatigue During Exercise of Different Durations," *Medicine & Science in Sports & Exercise* 48 (2016): 1024–1032.

58. C. Nicol, J. Avela, and P. V. Komi, "The Stretch-Shortening Cycle," *Sports Medicine* 36 (2006): 977–999.

59. C. G. Brownstein, M. Metra, F. Sabater Pastor, R. Faricier, and G. Y. Millet, "Disparate Mechanisms of Fatigability in Response to Prolonged Running Versus Cycling of Matched Intensity and Duration," *Medicine and Science in Sports and Exercise* 54 (2022): 872–882.

60. S. Racinais, O. Girard, J. P. Micallef, and S. Perrey, "Failed Excitability of Spinal Motoneurons Induced by Prolonged Running Exercise," *Journal of Neurophysiology* 97 (2007): 596–603.

61. S. Mense, "Effects of Temperature on the Discharges of Muscle Spindles and Tendon Organs," *Pflügers Archiv* 374 (1978): 159–166.

62. P. Aagaard, E. B. Simonsen, J. L. Andersen, P. Magnusson, and P. Dyhre-Poulsen, "Increased Rate of Force Development and Neural Drive of Human Skeletal Muscle Following Resistance Training," *Journal of Applied Physiology (1985)* 93 (2002): 1318–1326.

63. M. Coertjens, P. C. Coertjens, M. P. Tartaruga, et al., "Energetic Responses of Head-Out Water Immersion at Different Temperatures During Post-Exercise Recovery and Its Consequence on Anaerobic Mechanical Power," *European Journal of Applied Physiology* 123 (2023): 2813–2831.

64. F. Budini, D. Rafolt, M. Christova, E. Gallasch, and M. Tilp, "The Recovery of Muscle Spindle Sensitivity Following Stretching Is Promoted by Isometric but Not by Dynamic Muscle Contractions," *Frontiers in Physiology* 11 (2020): 905.

65. H. V. Cabral, K. M. Meiburger, L. F. de Oliveira, and T. M. Vieira, "Changes in Supramaximal M-Wave Amplitude at Different Regions of Biceps Brachii Following Eccentric Exercise of the Elbow Flexors," *European Journal of Applied Physiology* 121 (2021): 307–318.

66. J. M. Davis and S. P. Bailey, "Possible Mechanisms of Central Nervous System Fatigue During Exercise," *Medicine and Science in Sports and Exercise* 29 (1997): 45–57.

67. G. M. Minett and R. Duffield, "Is Recovery Driven by Central or Peripheral Factors? A Role for the Brain in Recovery Following Intermittent-Sprint Exercise," *Frontiers in Physiology* 5 (2014): 24.

68. M. J. Tipton, N. Collier, H. Massey, J. Corbett, and M. Harper, "Cold Water Immersion: Kill or Cure?," *Experimental Physiology* 102 (2017): 1335–1355.

69. S. Koga, T. Shiojiri, N. Kondo, and T. J. Barstow, "Effect of Increased Muscle Temperature on Oxygen Uptake Kinetics During Exercise," *Journal of Applied Physiology (1985)* 83 (1997): 1333–1338.

70. J. Gomez-Cambronero, "The Oxygen Dissociation Curve of Hemoglobin: Bridging the Gap Between Biochemistry and Physiology," *Journal of Chemical Education* 78 (2001): 757.

71. S. P. Hoekstra, N. C. Bishop, S. H. Faulkner, S. J. Bailey, and C. A. Leicht, "Acute and Chronic Effects of Hot Water Immersion on Inflammation and Metabolism in Sedentary, Overweight Adults," *Journal of Applied Physiology* 125 (2018): 2008–2018.

72. D. Farina, A. Holobar, R. Merletti, and R. M. Enoka, "Decoding the Neural Drive to Muscles From the Surface Electromyogram," *Clinical Neurophysiology* 121 (2010): 1616–1623.

73. J. Avela, H. Kyröläinen, and P. V. Komi, "Altered Reflex Sensitivity After Repeated and Prolonged Passive Muscle Stretching," *Journal of Applied Physiology* 86 (1999): 1283–1291.

74. G. S. Trajano, L. Seitz, K. Nosaka, and A. J. Blazevich, "Contribution of Central vs. Peripheral Factors to the Force Loss Induced by Passive Stretch of the Human Plantar Flexors," *Journal of Applied Physiology* 115 (2013): 212–218.

75. L. Braschler, P. T. Nikolaidis, M. Thuany, et al., "Physiology and Pathophysiology of Marathon Running: A Narrative Review," *Sports Medicine—Open* 11 (2025): 10.

76. G. Singh, K. J. M. Bennett, L. Taylor, and C. J. Stevens, "Core Body Temperature Responses During Competitive Sporting Events: A Narrative Review," *Biology of Sport* 40 (2023): 1003–1017.

Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Appendix S1:** sms70191-sup-0001-AppendixS1.docx.