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Mechanism involved of post-exercise cold water immersion: Blood redistribution and increase in energy expenditure during rewarming

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ABSTRACT

Thermogenesis is well understood, but the relationships between cold water immersion (CWI), the post-CWI rewarming and the associated physiological changes are not. This study investigated muscle and systemic oxygenation, cardiorespiratory and hemodynamic responses, and gastrointestinal temperature during and after CWI. 21 healthy men completed randomly 2 protocols. Both protocols consisted of a 48 minutes heating cycling exercise followed by 3 recovery periods (R1-R3), but they differed in R2. R1 lasted 20 minutes in a passive semi-seated position on a physiotherapy table at ambient room temperature. Depending on the protocol, R2 lasted 15 minutes at either ambient condition (R2_AMB) or in a CWI condition at 10°C up to the iliac crest (R2_CWI). R3 lasted 40 minutes at AMB while favoring rewarming after R2_CWI. This was followed by 10 minutes of cycling. Compared to R2_AMB, R2_CWI ended at higher $\dot{V}O_2$ in the non-immersed body part due to thermogenesis (7.16(2.15) vs. 4.83(1.62) $ml.min^{-1}.kg^{-1}$) and lower femoral artery blood flow (475(165) vs. 704(257) $ml.min^{-1}$) ($p < 0.001$). Only after CWI, R3 showed a progressive decrease in vastus and gastrocnemius medialis O_2 saturation, significant after 34 minutes ($p < 0.001$). As blood flow did not differ from the AMB protocol, this indicated local thermogenesis in the immersed part of the body. After CWI, a lower gastrointestinal temperature on resumption of cycling compared to AMB (36.31(0.45) vs. 37.30(0.49) °C, $p < 0.001$) indicated incomplete muscle thermogenesis. In conclusion, the rewarming period after CWI was non-linear and metabolically costly. Immersion and rewarming should be considered as a continuum rather than separate events.

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Introduction

Cold-water Immersion (CWI) is a common recovery method used in various sports, such as contact sports [1], sports with high mechanical stress [2] and endurance sports [3]. Recent research [4] found that 86% of a panel of 111 athletes, coaches and support practitioners used CWI, and 78% found it effective for recovery. Several mechanisms are proposed to explain the beneficial effects of CWI on recovery including hydrostatic pressure and cold-induced analgesic effect and vasoconstriction [5]. However, controversial results remain in the literature, largely due to the variability in CWI protocols between studies [4]. Environmental conditions (e.g. room temperature, humidity) are another source of discrepancies between studies. The recommendation for or against the use of CWI is influenced by a number of factors, including the duration and temperature of CWI, the degree of hyperthermia prior to CWI, the duration of the post-CWI event, the control protocol used, the potential benefit on inflammation, and the type of exercise [6]. The depth of immersion also plays an important role in the CWI protocol [7]. On the other hand, the energy expenditure due to thermoregulation accounts for a significant proportion of our total energy expenditure under normal conditions [8]. As the cooling/rewarming couple appears to be inseparable, a number of studies have focused on temperature changes during different rewarming protocols. It has been shown that body

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temperature continues to fall after leaving a cold bath, known as the "afterdrop." This has been observed at three measurement points with similar kinetics but different timing in rectal, esophageal and ear canal temperatures [9,10]. It was initially proposed that afterdrop was due to increased perfusion of the extremities as a result of post-immersion vasodilation, increasing the amount of cooled blood returning to cool the core, but this hypothesis was refuted by a study focusing on blood flow variation during rewarming [10]. Instead, this study suggested that this phenomenon was related to heat conduction along a thermal gradient toward a cold inner shell (including convective heat exchange between tissues of different temperatures) and a reduction in heat production due to shivering inhibition. The conduction and convection phenomena were validated by Romet [9]. Giesbrecht et al. [11] reported a much lower energy expenditure during shivering suppression, starting 5 min after injection of the suppressant (meperidine) and continuing between 30 and 35 min after immersion. Another study investigated different modalities of rewarming [12] and found approximately the same kinetics of metabolic rate (calculated from oxygen consumption, $\dot{V}O_2$) as Giesbrecht et al. [11]. This demonstrates the significant metabolic impact of the rewarming period. It is therefore interesting to quantify the energetic cost of a cold bath and its subsequent rewarming. Indeed, as part of a recovery strategy, it may be important to compensate for the energy deficit caused by CWI with an adequate energy intake.

Few studies have focused on the energy costs of CWI. Four studies have found an increase in VO_2 during CWI [11,13–15], reflecting an increase in body heat production. Three thermoregulatory mechanisms can explain this heat production. The first two mechanisms refer to shivering thermogenesis (ST) and non-shivering thermogenesis (NST). ST can be defined as involuntary rhythmic muscle contractions controlled by the sympathetic nervous system to produce heat [16]. The ATP production required to sustain shivering comes from glucose, lipids and proteins, depending on the nutritional status of the participant prior to cold exposure, the shivering intensity and variation in muscle fiber recruitment [17,18]. Giesbrecht et al. [11] highlighted the energy cost of ST, during CWI and during the rewarming period. NST is divided into two parts: non-shivering muscle thermogenesis (NST-M) and non-shivering thermogenesis due to the use of brown (and beige) adipose tissue (NST-B). NST-M is based on the activity of the SERCA protein in the sarcoplasmic reticulum of muscle fibers, which allows the active transport of Ca^{2+} from the cytosol to the lumen of the sarcoplasmic reticulum [19]. In the case of NST-M, sarcolipin induces a structural change in the Ca^{2+} transport site that prevents Ca^{2+} from being transported during each catalytic cycle and pushes it back into the cytosol. ATP is hydrolyzed without Ca^{2+} transport, thereby producing only heat [20]. Another poorly understood mechanism is mitochondrial proton leak, which could be mediated by the UCP3 protein or by changes in mitochondrial morphology [21]. Concerning NST-B, brown adipose tissue (BAT) contains mitochondria and uncoupling protein 1 (UCP1) [22]. When this protein is activated, the mitochondrial proton gradient converts the energy of fat oxidation into heat rather than creating ATP [23,24]. However, VO_2 changes due to cold water immersion must be interpreted with caution, as cold shock responses occur within the first 30 sec and stabilize after 3 min in most cases [25]. These responses include an inspiratory gasp of 2 and 3 liters, depending on water temperature [26]. Increased ventilation due to cold shock causes hypocapnia [27], which is partially offset by an increase in metabolic rate, leading to an increase in CO_2 release [26]. This increase in metabolic rate logically leads to an increase in VO_2. At the same time, there is an increase in cardiac output [28] due to tachycardia and the increase of cutaneous vasoconstriction [26]. All these mechanisms are due to the cold shock response and are not thermoregulatory mechanisms.

Vasoconstriction is another important thermoregulatory mechanism to conserve the heat, as blood plays an essential role in transporting heat to tissues, both to regulate deep body temperature and to rewarm tissues. Vasoconstriction leads to a reduction in the transport of heat by the blood, resulting in a drop in muscle and skin

temperature. This event has two effects: First, less cold blood flows back to the central core, helping to protect deep body temperature. Second, it reduces the heat flow between the body and the water, thereby reducing the heat loss from the body by reducing the temperature difference between the muscles and the skin with water. Superficial and deep body tissues continue to cool by conduction, but the body reduces heat delivery to the submerged limb, preserving heat for the central core.

These thermoregulatory mechanisms are primarily activated by the transient receptor potential cation channel subfamily M (melastatin) member 8 (TRMP8), which transmits information to the preoptic area. TRMP8 is a cation channel whose conductance is activated by mild cooling (<28°C) [29]. A feedback loop related to the deep body temperature involves detection of brain, spinal cord and visceral temperature [30]. During acute cold stress, these different sensors elicit different responses according to environmental and deep body temperature. The periods of exposure to cold and heat are likely to result in different energy expenditure responses. Therefore, to understand the impact of CWI, it is necessary to understand the time course of thermoregulatory mechanisms during the exposure and rewarming periods.

The present study was designed to investigate the specific cooling-rewarming pair after a heating exercise, which reflects a common type of recovery in high performance sports. In this context, we investigated the time course of central or local heat production and vasoconstriction during the subsequent CWI and rewarming period after a heating cycling exercise. We hypothesized that the different thermoregulatory mechanisms that occur during rewarming compared to CWI may be an additional cause of the different effects reported in the literature on increased energy expenditure during and after CWI. To test this hypothesis, several physiological parameters were measured pre-, per- and post CWI.

Methods

Twenty-one male volunteers (mean(SD); age: 24(5) years, body mass: 68(8) kg; height: 177(6) cm; $\dot{V}O_{2max}$: 55(6) $ml.kg^{-1}.min^{-1}$, maximal aerobic power: 300(50) W, body fat percentage: 14.7(3) %) who regularly participated in endurance sports such as cycling or trail running, were recruited for this study. All participants were fully informed of the potential risks and requirements of the study and provided written informed consent. The study was approved by the national ethical committee for STAPS (Science and Technique of Physical activity and Sport) Research (number IRB00012476–2022-21-04-176). To be eligible, participants were required to have completed an incremental cycling exercise test providing data on their power at the first and second ventilatory thresholds (P_{VT1} and P_{VT2}, respectively), maximal aerobic power and $\dot{V}O_{2max}$. Any participant who failed to complete the test protocol or sustained an injury during the process was excluded from the study.

Incremental exercise test

The incremental protocol consisted of a 2 min warm-up at 100 W, followed by increments of 30 W every 2 min. The first ventilatory threshold ($\dot{V}T1$) was defined as the time at which an initial deviation from expiratory volume (VE) linearity was observed, and when a systematic increase in the ventilatory equivalent for O_2 ($\dot{V}E/\dot{V}O_2$) and the fraction of expired O_2 first appeared. The second ventilatory threshold ($\dot{V}T2$) was defined based on a secondary increase in VE and VE/VO_2, and a marked increase in the ventilatory equivalent for CO_2 ($\dot{V}E/\dot{V}CO_2$) combined with a decrease in the fraction of expired CO_2 [31]. Participants were required to abstain from alcohol (48 h), caffeine, tea or guarana shot (24 h) and strenuous exercise (24 h) before and during the experiment. Additionally, participants were requested to arrive well-hydrated.

Experimental design

To ensure familiarity with the protocol for measuring cardiac output ($\dot{Q}c$), all participants underwent a familiarization session. This was followed by two experimental protocols, randomized 48 hours apart.

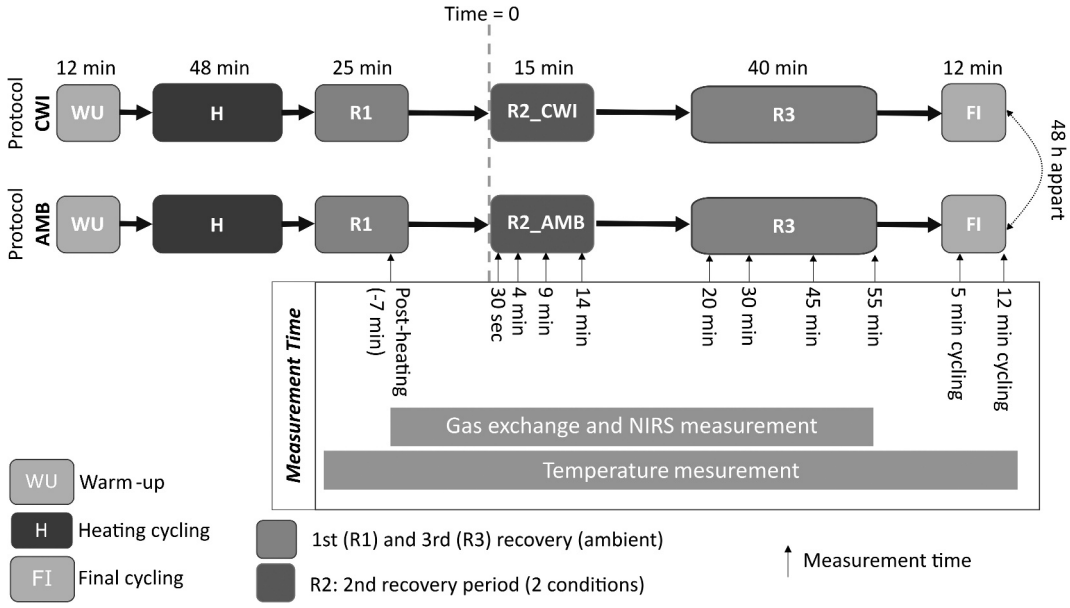


Figure 1. Test protocols and measurement schedule. CWI, cold-water immersion at 10(0.5) °C; AMB, ambient room condition at 20.8 (1.2) °C.

As shown in Figure 1, both protocols session started with a 5 min warm-up cycle exercise (WU), followed by a 48 min heating cycling exercise (H), 3 recovery periods (R1-R3), and a final cycling exercise (FI). The protocols only differed in R2. More precisely, WU started at 80% of the power at VT1, followed by 4 × 30s at the power at VT1 plus 90% of the difference between the power at VT1 and the power at VT2, interspaced by 1 min passive recovery [32]. It ended with 2 all-out isokinetic sprints at a mechanical cadence of 120 rpm [33]. The 48 min H exercise consisted of 8 sets of 5 min at a fixed cycling workload (90% of the power reach at VT1) with 1 min recovery at 75W. Cognitive tasks were performed during sets 1, 2, 4, 5, 7 and 8. These data were used in another study investigating cognitive decision making before, during and after exercise [34]. As the data for this period are not covered in a way that is consistent with the topic of this article, you will find the data for the heating period in the supplementary material. All recovery periods (R1–R3) were carried out in a standardized semi-seated position at 20.8(1.2) °C room temperature and 40.7 (8.9) % humidity. In R1, participants rested passively on a physiotherapy table for 25 min while some measurements were taken and some equipment was placed on the participant (as described in the Measurement section), as in the Peiffer et al. [35] study. During R1, the recorded physiological variables returned to relatively stable values, while rectal temperature continued to fall thereafter [36]. Depending on the protocol, R2 lasted 15 min in either ambient condition (R2_AMB) or cold water immersion (R2_CWI) conditions, which determined the name of the protocol (either CWI or AMB) (Figure 1). The order between these two protocols was randomized and well-balanced between participants. More precisely, in the R2_CWI condition, the participant sat down in a bathtub filled-up with water at 10°C for 15 min [37]. The instruction was to sit down as soon as possible on a stool adjusted so that water would rise up to the iliac crest. The bathtub was located 1 meter from the physiotherapy table where the passive R1 recovery was completed. In the R2_AMB condition, participants mimicked getting in and out of the bathtub to ensure that the observed effects were related to cold and not to movement. For both protocols, in R3 the participant sat on the physiotherapy table at ambient room temperature for a further 40 min [37]. In the protocol including the R2_CWI condition, the participant dried off with their towel, kept their bathing suit and put on a dry T-shirt. For them only, R3 corresponded to a rewarming period. Both protocols ended with a FI similar to the exercise used in WU to examine the changes in deep body temperature during a cycling activity following CWI.

Measurements

1h15 before the start of the CWI, participants were asked to swallow a pill (e-Celsius Performance, BodyCap, France) which was connected to their monitor via Bluetooth to record their gastrointestinal temperature (T_{GI}) every 30 s throughout the experiment. For ease of reading, we have set T = 0 as the start of the R2(CWI and AMB) period (Figure 1). Cardiorespiratory variables including VO_2, VCO_2, VE and respiratory exchange ratio (RER) were recorded with breath-by-breath acquisition and cardiac output (Qc) by rebreathing [38] from −7 to 55 min (end of R3) using the Innocor system (Cosmed, Italy). To reflect the local energy expenditure of the targeted muscles, muscle oxygen saturation of the vastus medialis (MSO_2VM) and gastrocnemius medialis (MSO_2GM) of the left lower limb were recorded with a NIRS system (MOXY, Moxymonitor, United States) at 2 Hz as previously described [39,40]. To monitor the vasoconstriction mechanisms, the changes in superficial femoral artery blood flow (FABF) complemented the Qc recordings. FABF was measured using a Doppler ultrasound device (Echowave, Telemed, Italy) at each of the measurement times described in Figure 1. The probe was 6 cm wide and the sampling frequency set at 9 MHz (HL9.0/60/128Z–2, Telemed, Italy). FABF Data were collected at an insonation angle of 60 ° [41]. The room humidity and temperature were measured with one thermo-hygro-barometer (RS-1161, RS Pro, United Kingdom). The water temperature was continuously monitored and kept at 10(0.5) °C using a water temperature controller (CryopackPerf, Cryo control, Castanet Tolosan, France). The water was not stirred manually, but the cooling system necessarily involved suction and discharge of water into the basin, resulting in a movement of water in the bath.

Data management

FABF was determined from superficial femoral artery diameter (FAD) and blood velocity (FABV) data using an Echowave (Echo wave II, version 4.1.0, Telemed, Italy). FAD was defined as the average of measurements in 4 directions and FABV was the average peak blood velocity during 5 heartbeats before the FAD image was taken. FABF was calculated as follows:

$$FABF = \frac{\pi \times \frac{FAD^2}{2}}{1000} \times \frac{FABV}{100} \times 10^5$$

where FAD is measured in mm, FABV in $cm.s^{-1}$ and FABF in $ml.min^{-1}$.

The recorded T_{GI}, MSO_2 and cardiorespiratory data were imported into Matlab (R2020a, MathWorks, United States). Gas exchange data were smoothed with 11-point rolling average (between *n*-5 and n + 5), and then averaged over the 30-s period prior to each Qc (the 9 recording times are shown in Figure 1). T_{GI} was measured at 5 min intervals from the end of the last heating exercise intensity until the first Qc measurement, then at each Qc measurement. Finally, data were collected after 5 min of WU after R3, and at the end of WU (12 min cycling).

Statistics

A linear mixed model was performed using the lme4 [42] and lmerTest [43] packages in R. To assess the protocol effect as a function of time, protocol and time were used as fixed effects and the random effects included test day and subject. Time corresponds to each measurement instant as described earlier in the method. A pairwise comparison with Holm's adjustment was then used for the interaction. The following data were included in the analysis: VO_2, VCO_2, VE, RER, T_{GI}, Qc and FABF. The statistical method was similar to that used in the study by Macchi et al. [44]. The effect size (ES) of mean differences was determined using Cohen's d coefficient, with effect sizes equal to or greater than 0.2, 0.6, and 1.2 interpreted as small, moderate, and large effects, respectively [45]. Partial eta squared (η_p^2) was calculated for each interaction. For MSO_2VM and MSO_2GM, a one-dimension statistical parametric mapping (SPM) [46] was used to examine the influence of the protocol. This method applied the test directly to the original (recorded) curves and was analyzed using the open-source spm1d code (version 0.4.8, www.spm1d.org) in Matlab (R2020a, The MathWorks Inc, Natick, USA). Normality was checked using an SPM Shapiro Wilk test before analyzing the NIRS data. The statistical calculations are described in Pataky [47]. A 5% risk was used for all statistical tests.

Results

As all the parameters studied, except muscle O_2 saturation, showed a significant interaction between the effects of the protocol (CWI vs. AMB) and the time of measurement, only the interactions are detailed in their case.

Cardio-respiratory parameters

VO_2, VE, VCO_2 and RER showed a significant time x protocol (CWI vs. AMB) interaction ($p < 0.001$ for all variables; VO_2 $\eta_p^2 = 0.09$; VCO_2 $\eta_p^2 = 0.18$; VE $\eta_p^2 = 0.30$; RER $\eta_p^2 = 0.30$). The group mean values (±SD) are shown for each protocol in Figure 2 together with the statistics related to the protocol effect. The time effect statistics are detailed (p-values and effect sizes) in Tables 1 and 2.

Time effect in each protocol

Only the CWI protocol showed a significant time effect, due to an overall increase in cardiorespiratory parameters during the R2_CWI condition compared to the R1 and R3 recovery periods.

During R2_CWI, VO_2 at 30 sec was significantly higher than in R1 and R3 periods. VO_2 then decreased up to 9 min before increasing again at 14 min. At 4 min VO_2 was still higher than in R3 (up to 45 min), at 9 min not different from R1 and R3, and at 14 min significantly higher than in R3.

VCO_2 clearly peaked at 30 sec, being significantly higher than at any other time point in R1, R2 and R3. Due to its subsequent decrease, at 4 min VCO_2 was only higher than in R3 (up to 45 min). A slight increase in both VO_2 and VCO_2 was observed at 55 min in R3.

VE also peaked at 30 sec before decreasing progressively. At both 30 sec and 4 min VE was significantly higher than at any other time point in R1, R2 and R3. At 9 min, VE was not different from R1 and R3. At 14 min, due to a slight increase, VE was again higher than in R3.

RER also peaked at 30 sec before decreasing. At 30 sec RER was higher than at any other time point in R1, R2 and R3. At 4 min, RER was still higher than at 9 min and 14 min in R2, and at 20 min in R3. Compared to R1, RER was also higher at 9 min and 14 min in R2, and at 55 min in R3.

Protocol effect (CWI vs. AMB)

As shown in Figure 2, the R2_CWI condition was mostly characterized by higher cardiorespiratory values than the R2_AMB condition. VO2 was significantly higher at all times (at 30 sec: $p = 0.003$, d = 0.96; 4 min: $p < 0.001$, d = 1.16; 9 min: $p = 0.007$, d = 0.90; 14 min: $p < 0.001$, d = 1.23). VCO2 was also significantly higher, except at 9 min (at 30 sec: $p < 0.001$, d = 1.58; 4 min: $p = 0.001$, d = 1.25; 14 min: $p = 0.011$, d = 0.96). VE was also significantly higher at all time points (at 30 sec: $p < 0.001$, d = 2.31; 4 min: $p < 0.001$, d = 1.66; 9 min: $p = 0.029$, d = 0.67; 14 min: $p < 0.001$, d = 1.06). RER was only significantly higher at 30 sec ($p < 0.001$, d = 2.02) and was even lower at 14 min ($p = 0.019$, d = 0.91).

Hemodynamic parameters

A significant interaction time x protocol (CWI vs. AMB) was found for FABF and Qc ($p < 0.001$; FABF $\eta_p^2 = 0.06$; Qc $\eta_p^2 = 0.09$). The group mean values (±SD) are shown in Figure 3 together with the statistics related to the protocol effect. The time effect statistics are detailed in Table 3.

Time effect in each protocol

Only the CWI protocol showed a significant time effect on FABF and on Qc:

During R2-CWI, FABF decreased sharply and remained significantly lower than in R1 throughout. During the subsequent rewarming period R3, FABF was not significantly different from R1 or R2, except at 30 min, which was significantly higher than at 14 min in R2.

Qc peaked at 30 sec, being significantly higher than at any other time point in R1, R2 and R3. It then decreased progressively until the beginning of R3 at 20 min, being below R1 and R2 up to 4 min. A slight decrease occurred during R3 at 40 min resulted in a difference with R1 and R2 at 30 sec.

Protocol effect

As shown in Figure 3, the R2_CWI condition was characterized by lower FABF values than the R2_AMB condition (at 30 sec: $p < 0.001$, d = 0.80; 4 min: $p < 0.001$, d = 0.95; 9 min: $p < 0.001$, d = 0.97; 14

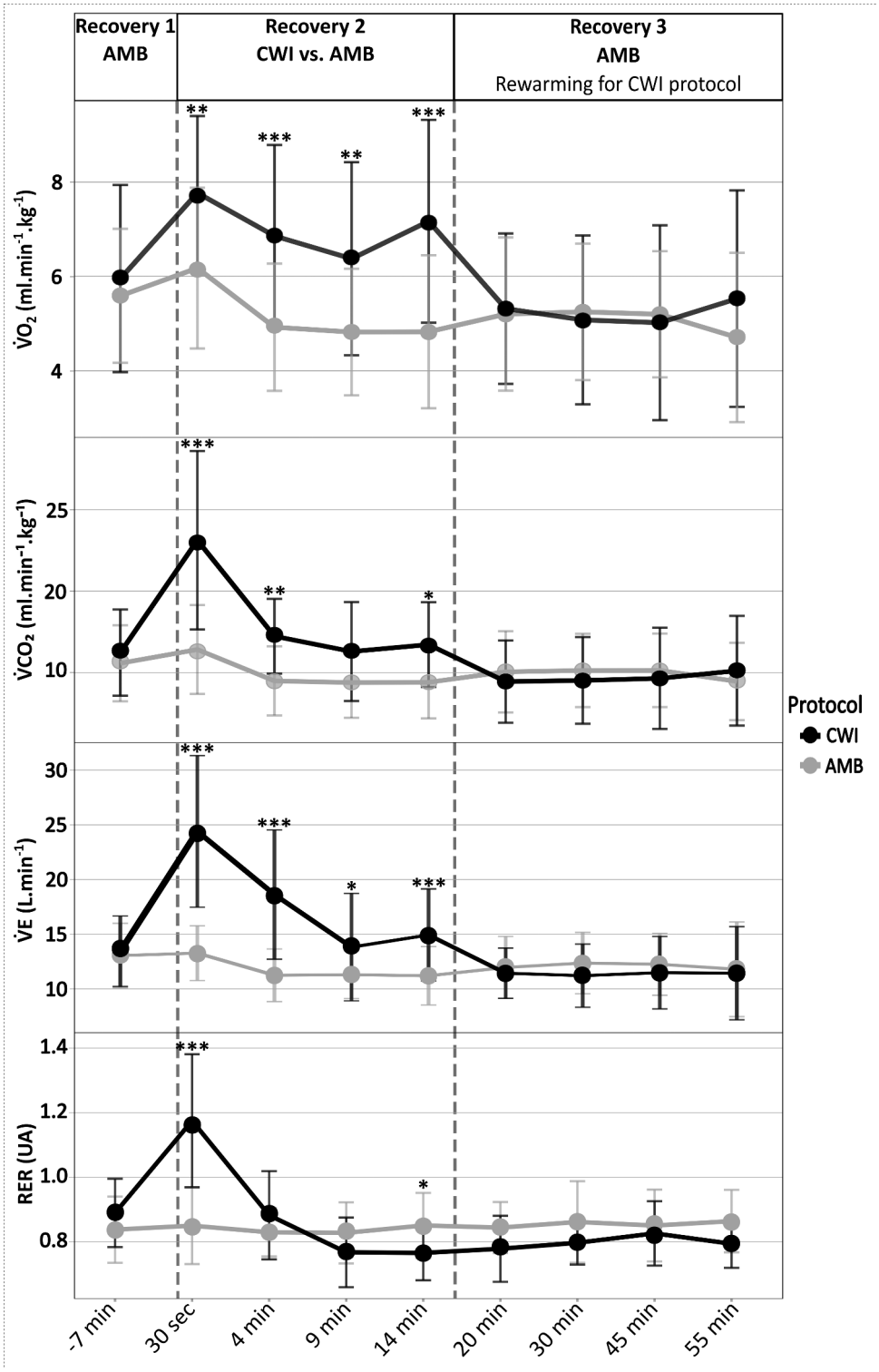


Figure 2. Influence of the protocol (CWI vs. AMB) on the time course of the relative oxygen uptake (VO_2), relative carbon dioxide output (VCO_2), ventilation (VE) and respiratory exchange ratio (RER) during the 3 recovery periods (R1–R3) following a heating cycling exercise. Time was set at 0 at the beginning of R2. The black and grey lines correspond to the protocol with the R2 recovery in either cold water immersion (CWI) or at ambient room temperature (AMB). The statistical values of the time effect in each protocol are detailed in Tables 1 and 2. *$p < 0.05$, **$p < 0.01$ between protocols, ***$p < 0.001$ between protocols.

Table 1. P-values (p) and effect size (Cohen's d value) for the significant time effect on VO_2 and VCO_2.

				R1	R2				R3				
				-7 min	30 sec	4 min	9min	14 min	20 min	30 min	45 min	55 min	
$\dot{V}O_2$	R1	-7 min	p		0.004								CWI protocol
			d		1.00								
	R2	30 sec	p						<0.001	<0.001	<0.001	<0.001	
			d						1.52	1.57	1.48	1.13	
		4 min	p						0.043	0.006	0.003		
			d						0.87	0.96	0.92		
		9 min	p										
			d										
		14 min	p						<0.001	<0.001	<0.001	0.003	
			d						0.98	1.06	1.02	0.74	
	R3	20 min	p										
			d										
		30 min	p										
			d										
		45 min	p										
			d										
		55 min	p										
			d										
$\dot{V}CO_2$	R1	-7 min	p		<0.001								CWI protocol
			d		1.63								
	R2	30 sec	p			<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	
			d			1.42	1.56	1.52	2.07	2.03	1.93	1.78	
		4 min	p						0.009	0.013	0.024		
			d						1.15	1.09	0.95		
		9 min	p										
			d										
		14 min	p										
			d										
	R3	20 min	p										
			d										
		30 min	p										
			d										
		45 min	p										
			d										
		55 min	p										
			d										
				AMB protocol									

The table only shows the statistically significant differences in the relative values of oxygen uptake (VO_2) and carbon dioxide output (VCO_2) between different measurement times in each protocol. The upper right (smoothed) corresponds to the CWI protocol, and the lower left (hatched) corresponds to the AMB protocol. R1, R2 and R3 correspond to the first, second and third recovery periods after a heating cycling exercise. The two protocols only differed in R2, which was performed in either cold water immersion (CWI) or at ambient room temperature (AMB). From lightest to darkest coloring: from no difference or small effect size, to moderate effect size, and large effect size for the darkest.

min: $p < 0.001$, d = 1.06) while Qc was only initially higher (at 30 sec: $p < 0.001$, d = 0.84 and 4 min: $p = 0.048$, d = 0.55). During the subsequent rewarming period R3, FABF was again significantly lower than in the AMB protocol at 45 min ($p = 0.049$, d = 0.52) and 55 min ($p = 0.017$, d = 0.57).

Gastrointestinal temperature

A significant interaction time x protocol (CWI vs. AMB) was found for T_{GI} ($p < 0.001$; $\eta_p^2 = 0.25$). The group mean values (±SD) are shown in Figure 4 together with the statistics related to the protocol effect. The time effect statistics are detailed in Table 4.

Time effect in each protocol

During R1, in both protocols, T_{GI} dropped during the second half of R1 (being lower at −10 and −7 min and thus 15 to 18 min after the end of the heating exercise). During the subsequent CWI and R3 periods, T_{GI} remained rather stable, decreasing only in the CWI protocol at 55 min in R3. During FI, T_{GI} changed in an opposite way

Table 2. P-values (p) and effect size (Cohen's d value) for the significant time effect on VE and RER.

				R1	R2				R3				
				-7 min	30 sec	4 min	9 min	14 min	20 min	30 min	45 min	55 min	
V̇E	R1	-7 min	p		<0.001	<0.001							CWI protocol
			d		2.18	1.10							
	R2	30 sec	p			<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	
			d			0.91	1.81	1.74	2.76	2.69	2.55	2.37	
		4 min	p				<0.001	0.009	<0.001	0.015	<0.001	<0.001	
			d				0,88	0.73	1.,64	1.62	1.51	1.41	
		9 min	p										
			d										
		14 min	p						0.011	0.004	0.027	0.011	
			d						1.03	1.03	0.91	0.82	
	R3	20 min	p										
			d										
		30 min	p										
			d										
		45 min	p										
			d										
		55 min	p										
			d										
RER	R1	-7 min	p		<0.001		0.001	<0.001	0.004			0.036	CWI protocol
			d		1.84		1.07	1.29	0.93			1.14	
	R2	30 sec	p			<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	
			d			1.73	2.59	2.77	2.58	2.66	2.28	2.64	
		4 min	p				0.006	0.004	0.021				
			d				0.86	1.03	0.86				
		9 min	p										
			d										
		14 min	p										
			d										
	R3	20 min	p										
			d										
		30 min	p										
			d										
		45 min	p										
			d										
		55 min	p										
			d										
				AMB protocol									

The table only shows the statistically significant differences in ventilation (VE) and respiratory exchange ratio (RER) between different measurement times in each protocol. The upper right (smoothed) corresponds to the CWI protocol, and the lower left (hatched) corresponds to the AMB protocol. R1, R2 and R3 correspond to the first, second and third recovery periods after a heating cycling exercise. The two protocols only differed in R2, which was performed in either cold water immersion (CWI) or at ambient room temperature (AMB). From lightest to darkest coloring: from no difference or small effect size, to moderate effect size, and large effect size for the darkest.

in the two protocols: In the CWI protocol, it decreased, being significantly lower than at any other time point in R1, R2 and R3. In the AMB protocol, it was lower compared to the 10 first min of R1, but its value after 12 min of cycling was significantly higher than at 45 min and 55 min in R3.

Protocol effect

As shown in Figure 4, T_{GI} varied similarly in both recovery protocols throughout the recovery period. However, the final cycling exercise was associated with significantly different T_{GI} changes: in the CWI protocol, T_{GI} decreased so that it was significantly lower than in the AMB protocol after 5 min ($p < 0.001$, d = 2.05) and 12 min ($p < 0.001$, d = 2.73) of cycling.

Muscle O_2 saturation

As the statistical test is different, the results are presented in a different way. We will only discuss the

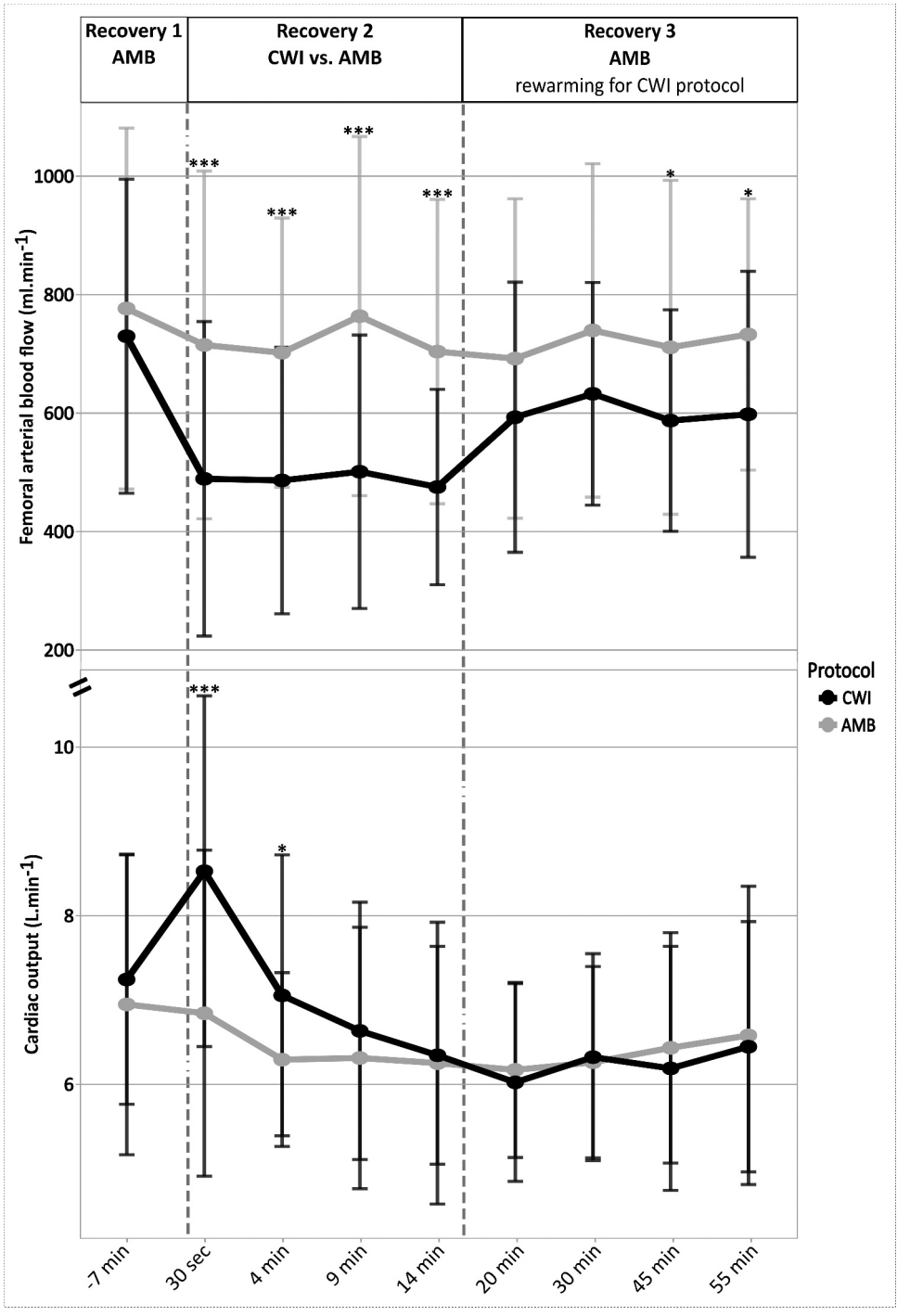
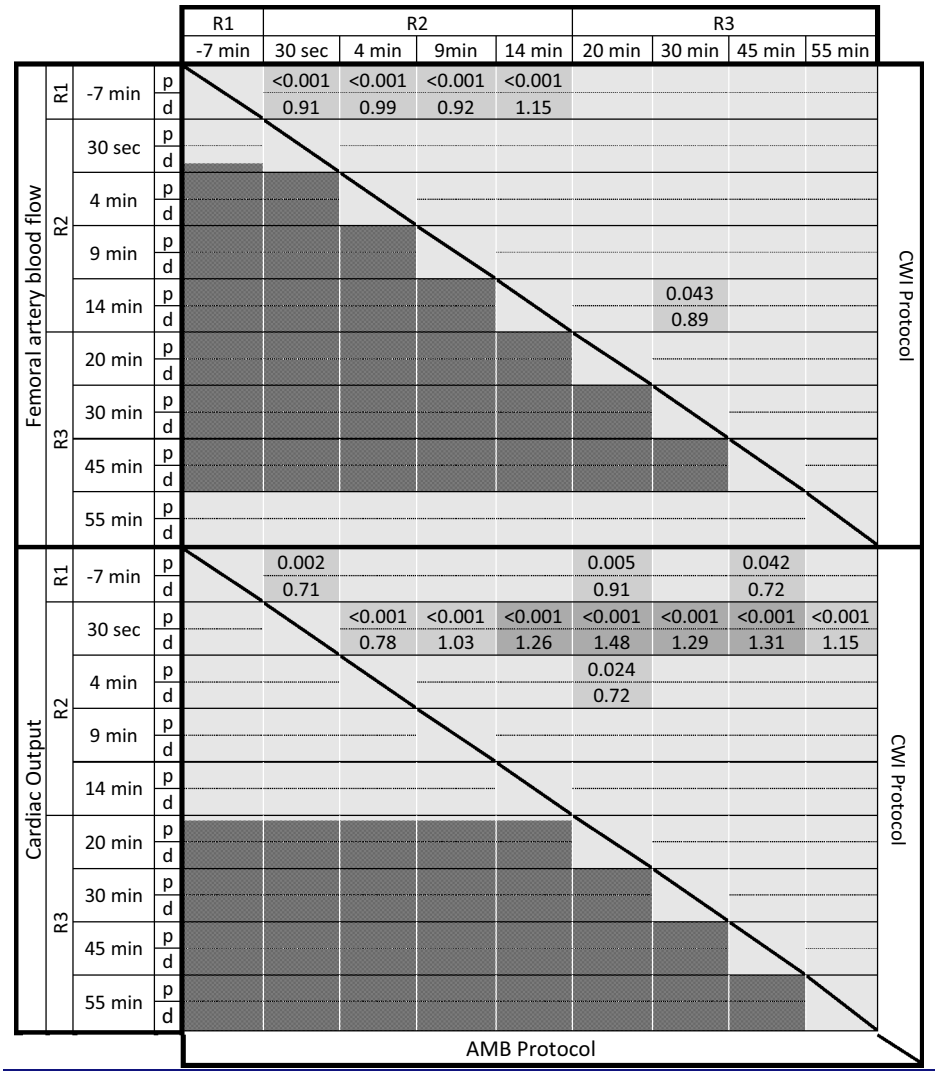


Figure 3. Influence of the protocol (CWI vs. AMB) on the time course of the femoral artery blood flow and cardiac output during the 3 recovery periods (R1–R3) following a heating cycling exercise. Time was set at 0 at the beginning of R2. The black and gray lines correspond to the protocol with the R2 recovery in either cold water immersion (CWI) or at ambient room temperature (AMB). *$p <$ 0.05, ***$p < 0.001$ between protocols.

difference between CWI and AMB measurements over time.

During R2, MSO_2VM et MSO_2GM did not differ significantly between protocols. During R3 in the CWI protocol, MSO_2VM and MSO_2GM decreased progressively after 22 min (7 min after the cold bath) (Figure 5A,B). Compared to the AMB protocol (Figure 5C), in the CWI protocol MSO_2VM was significantly lower from 31 min (16 min after the cold bath) to the end of R3, and

Table 3. P-values (p) and effect size (Cohen's d value) for the significant time effect on femoral artery blood flow and cardiac output.



The table only shows the statistically significant differences in the femoral artery blood flow and cardiac output between different measurement times in each protocol. The upper right (smoothed) corresponds to the CWI protocol, and the lower left (hatched) corresponds to the AMB protocol. R1, R2 and R3 correspond to the first, second and third recovery periods after a heating cycling exercise. The two protocols only differed in R2, which was performed in either cold water immersion (CWI) or at ambient room temperature (AMB). From lightest to darkest coloring: from no difference or small effect size, to moderate effect size, and large effect size for the darkest.

MSO_2GM from 38 min (23 min after the cold bath) to the end of R3.

Discussion

The aim of the study was to define the time course of central or local heat production and vasoconstriction during the subsequent CWI and rewarming period after a heating cycling exercise. The observation of an increase in VO_2 with associated peripheral vasoconstriction during CWI, followed by a decrease in MSO_2 (indicating increased O_2 consumption in lower limb muscles) during rewarming, supports our hypothesis that different thermoregulatory mechanisms may contribute to the increase in energy expenditure during and after CWI. Specifically, we distinguished non-thermoregulatory responses during early CWI from different thermoregulatory responses during and after CWI. The physiological thermoregulatory changes over time demonstrated that the rewarming period as an integral part of the

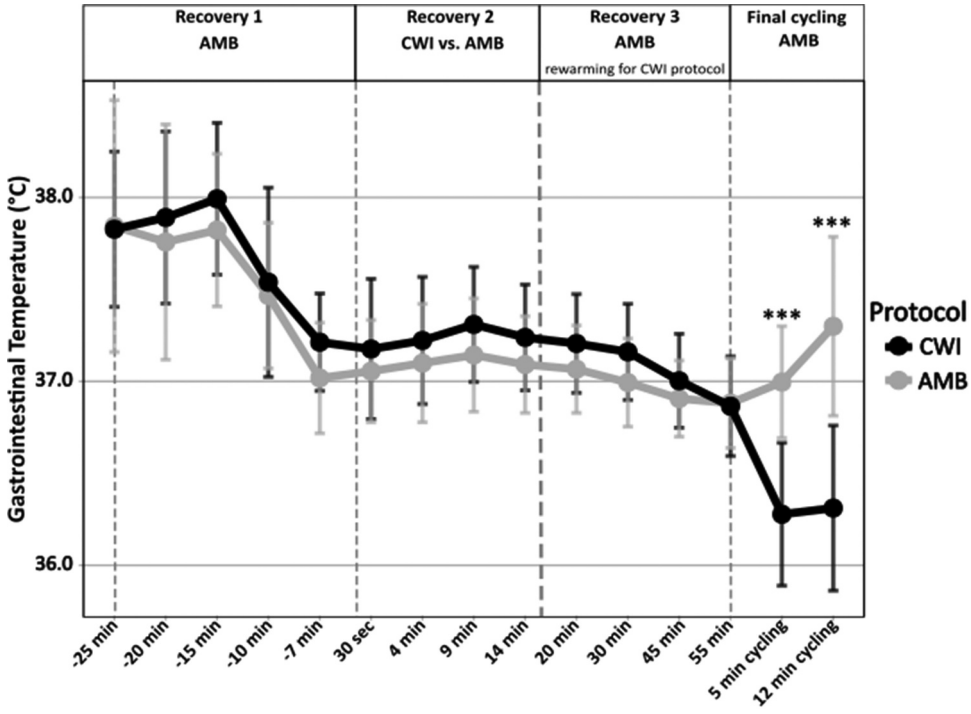


Figure 4. Influence of the protocol (CWI vs. AMB) on the time course of stomach temperature during the 3 recovery periods (R1–R3) following a heating cycling exercise. Time was set at 0 at the beginning of R2. The black and gray lines correspond to the protocol with the R2 recovery in either cold water immersion (CWI) or at ambient room temperature (AMB). ***$p < 0.001$ between protocols.

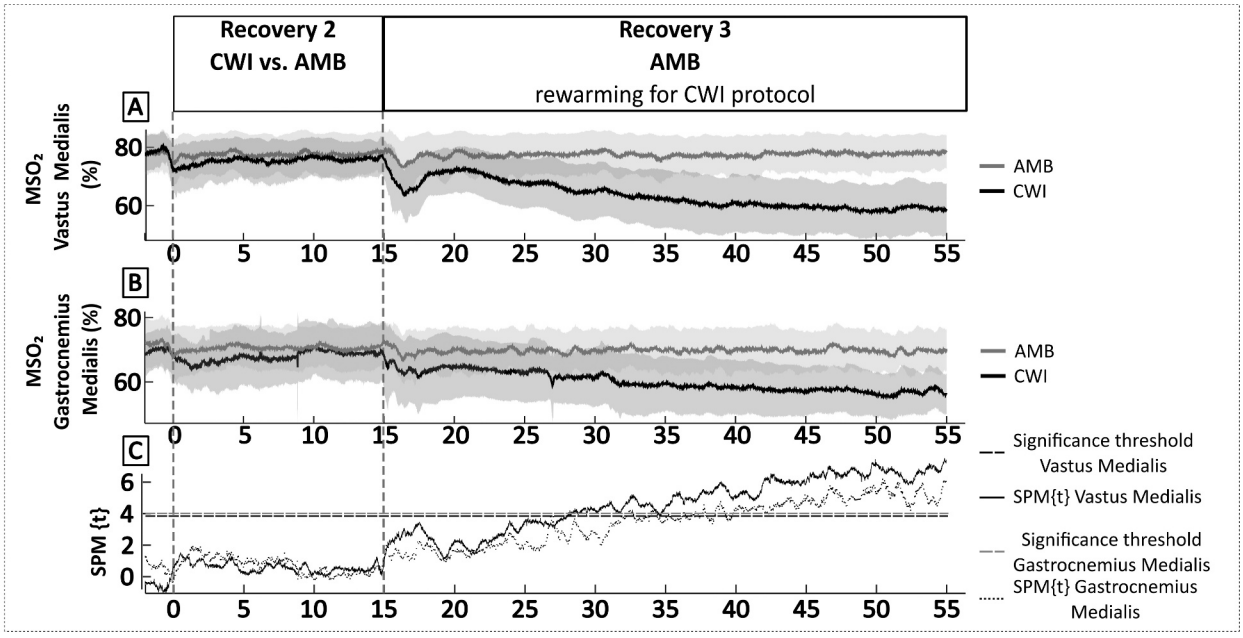


Figure 5. Muscle saturation in O_2 of vastus medialis (A) and gastrocnemius medialis (B) and t-test results including the raw SPM{t} with the random field theory computed critical t threshold (C). Muscle O_2 saturation in the CWI protocol is significantly lower than in the AMB protocol when its SPM{t} is higher than the significance threshold (horizontal cut line). CWI, cold water immersion protocol; AMB, ambient protocol.

Table 4. P-value (p) and effect size (Cohen's d value) of the time effect on gastrointestinal temperature.

Gastrointestinal temperature				R1					R2				R3				FI	
				-25 min	-20 min	-15 min	-10 min	-7 min	30 sec	4 min	9 min	14 min	20 min	30 min	45 min	55 min	5 min cycling	12 min cycling
R1	-25 min	p						<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		d						1.74	1.62	1.58	1.41	1.65	1.77	1.93	2.39	2.73	3.82	3.48
	-20 min	p					0.044	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		d					0.71	1.77	1.68	1.63	1.47	1.69	1.81	1.94	2.37	2.68	3.74	3.44
	-15 min	p					<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		d					0.97	2.23	2.06	2.03	1.87	2.13	2.27	2.42	2.90	3.23	4.28	3.91
	-10 min	p	0.047		0.017				0.013				0.041	0.007	<0.001	<0.001	<0.001	<0.001
		d	0.69		0.88				0.80				0.81	0.93	1.32	1.63	2.75	2.53
	-7 min	p	<0.001	<0.001	<0.001	<0.001										<0.001	0.021	<0.001
		d	1.61	1.47	2.21	1.27										1.30	2.82	2.47
R2	30 sec	p	<0.001	<0.001	<0.001	0.002											<0.001	<0.001
		d	1.58	1.44	2.17	1.21											2.34	2.09
	4 min	p	<0.001	<0.001	<0.001	0.016										0.013	<0.001	<0.001
		d	1.45	1.31	1.95	1.02										1.15	2.58	2.30
	9 min	p	<0.001	<0.001	<0.001											<0.001	<0.001	<0.001
		d	1.38	1.24	1.86											1.52	2.95	2.62
	14 min	p	<0.001	<0.001	<0.001	0.011										0.007	<0.001	<0.001
		d	1.52	1.38	2.10	1.12										1.34	2.84	2.51
R3	20 min	p	<0.001	<0.001	<0.001	0.004										0.025	<0.001	<0.001
		d	1.60	1.45	2.24	1.23										1.26	2.81	2.47
	30 min	p	<0.001	<0.001	<0.001	<0.001											<0.001	<0.001
		d	1.75	1.60	2.45	1.46											2.70	2.37
	45 min	p	<0.001	<0.001	<0.001	<0.001											<0.001	<0.001
		d	1.96	1.81	2.80	1.79											2.23	1.94
	55 min	p	<0.001	<0.001	<0.001	<0.001											<0.001	<0.001
		d	1.95	1.81	2.75	1.78											1.76	1.52
FI	5 min cycling	p	<0.001	<0.001	<0.001	<0.001												
		d	1.62	1.49	2.24	1.32												
	12 min cycling	p	<0.001	<0.001	<0.001										0.018	0.007		
		d	0.91	0.79	1.17										1.90	1.85		

AMB Protocol

CWI protocol

The table only shows the statistically significant differences in the gastrointestinal temperature between different measurement times in each protocol. The upper right (smoothed) corresponds to the CWI protocol, and the lower left (hatched) corresponds to the AMB protocol. R1, R2 and R3 correspond to the first, second and third recovery periods after a heating cycling exercise. The two protocols only differed in R2, which was performed in either cold water immersion (CWI) or at ambient room temperature (AMB). From lightest to darkest coloring: from no difference or small effect size, to moderate effect size, and large effect size for the darkest.

recovery protocol using CWI could potentially explain some of the contradictions found in the literature. As most studies in the literature have examined each of these variables separately, we will discuss them in turn before discussing their potential relationships.

With regard to the changes in body temperature during CWI, most studies have recorded rectal temperature [35–37,41] and, more recently, gastrointestinal temperature using a pill [48,49]. Among them, Crampton et al. [48] showed a decrease in intestinal temperature from the beginning to the end of a 30 min CWI in 15°C water. However, the intestinal temperature of their participants had already decreased during the first 5 min of the CWI, which differs from the current results where the gastrointestinal temperature remained stable. However, their participants' intestinal temperature at the beginning of the CWI was around 38°C (above the ideal temperature of the human body), whereas ours was 37.21°C. Another explanatory factor may be the different location of the temperature measurement: In Crampton et al. [48] study, the pill was swallowed 3 hours before immersion, whereas our participants took it 1 hour before. As a result, we measured temperature changes higher up in the body than they did. A third and most likely reason is that their participants were immersed in the bath 5 min after the end of the heating exercise. In the current study, immersion

did not take place until 25 min after the heating exercise. During this intermediate (R1) recovery period at ambient room temperature, a decrease in gastrointestinal temperature began between 10 to 15 min later. Under ambient conditions, Peiffer et al. [36] also reported a decrease in rectal temperature 5 to 10 min later. Therefore, it is possible that the decrease in intestinal temperature reported by Crampton et al. [48] during immersion was due to the effect of CWI combined with natural recovery post-exercise. Similarly, these authors also reported a decrease in intestinal temperature during exercise after leaving the cold bath. In a more recent study [49], intestinal temperature was measured during a 10 min immersion protocol at 8, 15 and 20°C without prior exercise. Consistent with our results, they found no significant difference in intestinal temperature between the beginning and end of the immersion, regardless of water temperature. A maintained gastrointestinal temperature during CWI can be attributed to two phenomena: A vasoconstrictive effect that reduces convective heat loss from the immersed limb by lowering their muscle and skin temperature [48–50] and/or a production of heat to counteract cooling.

In our study, we observed a decrease in FABF during CWI, which is supported by the findings of Vaile et al. [37] at the end of CWI. However, our FABF values were higher than theirs, possibly due to the use of different measurement techniques (venous occlusion plethysmography vs. Doppler ultrasound device). During whole-body cold exposure, a significant decrease in arterial blood velocity has been reported due to blood redistribution to the deep circulation [5,37]. This may be due to an increase in capillary vasoconstriction and hydrostatic pressure due to CWI. Their combination with the increase in heart rate is expected to explain the increase in Qc at 30 sec and 4 min in the CWI protocol [25,26]. These results suggest that during the CWI the muscles cool down, reducing the exchange with the blood and maintaining a stable deep body temperature. This is supported by the large decrease in gastrointestinal temperature when cycling resumed. This is due to the cold being transferred to the deep body by increased blood flow from the cold muscles.

Several studies have reported an increase in VO_2 during CWI. This response is initially neural in origin and is referred to as the cold shock response. In this line, we observed a 35% increase in VO_2 at 30 sec during the R2_CWI period. However, it contrasted with the 400% increase reported during the first minute of immersion by Mekjavic & Bligh [14]. One reason for this could be that their study involved immersion up to the neck, whereas in our study it was only involved up to the iliac crest. Indeed, Ran et al. [51] have shown an increase in the neuronal response in the spinal cord as the number of cold receptors stimulated increased with decreasing temperature. We can therefore hypothesize that an increase in the number of activated receptors due to an increase in the area of immersion will also lead to an increase in neuronal activity, and therefore the intensity of the cold shock response. In addition, our participants first placed their calves in the water before sitting on the stool, which may have attenuated the reflex increase of VO_2 [25]. Staged immersion has been shown to reduce the cold shock response as evidenced by a smaller increase in VE compared to non-staged immersion [52]. As observed in our study, cold shock responses are reported to reach a peak within 30 sec of immersion and adapt over the first 3 min of immersion in most individuals [25]. Similarly, the increase in RER observed 30 sec after entering the cold bath was attributed to the reflex increase in VE, since the induced increase in pulmonary air exchange should lead to an increase in expired VCO_2 associated with a decrease of alveolar CO_2 [27]. This agrees with the hypocapnia reported when entering a cold bath [25]. Therefore, it is suggested that the current increase in RER was due to non-metabolic phenomena.

In a second phase, to maintain its deep body temperature in CWI and after it, the body produces heat through two primary mechanisms: Non-shivering thermogenesis (NST) and shivering thermogenesis (ST), both of which consume O_2 [21,24]. In our study we found a 25% increase in VO_2 after 14 min of immersion at 10°C vs. 130% reported after 15 min at 14°C by Šrámek [15]. In the latter study, immersion was again up to the neck. It appears that VO_2 increases as the surface area of the body exposed to cold increases,

probably due to the increased energy flow exchanged when more of the body is in contact with the water in addition to the neural response. This requires the body to produce more heat, resulting in increased O_2 consumption. In addition, Eyolfson et al. [13] observed a gradual increase in VO_2 during immersion in colder water (from 20°C to 8°C) over 15 min. The plateau in VO_2 observed in our study during the entire CWI is probably due to ST. Indeed, it can increase VO_2 up to 40% of VO_{2max} [17]. Nevertheless, as indicated by the stability of MSO_2GM and MSO_2 VM, this metabolic phenomenon is unlikely to be due to ST or NST in the immersed lower limbs. Moreover, it has been shown that ST suppression during a cold bath results in a decrease in VO_2 compared to a protocol without suppression [11]. Since brown fat is mainly found in different parts of the trunk [53] and inhibition of shivering would only be effective at a core temperature below 31°C [54], ST and NST could have occurred in the trunk and upper limbs, but unfortunately we did not measure them. At this stage, it is concluded that the increase in VO_2 during CWI, first by neural phenomena and then by metabolic phenomena, with a return to the same value as in the AMB protocol during R3, suggests that different mechanisms were involved during CWI, but also after during the rewarming period.

Regarding muscle O_2 consumption, only two studies have examined MSO_2 in the immersed muscles during the CWI period and one during the rewarming period. Therefore, it is difficult to draw conclusions based on the comparison with their results. However, our results during the CWI period are consistent with some findings in the literature (e.g. Roberts et al. [55]), which reported no change in MSO_2 in the vastus lateralis during CWI up to the umbilical level at 10°C for 10 min. In contrast, an increase in MSO_2 in the vastus lateralis has been reported during leg immersion at 10°C for 15 min (Ihsan et al. [56]). This discrepancy is attributed to differences in the test protocol. In the latter study, only one leg was immersed up to the level of the gluteal fold, covered with a polyethylene bag to avoid contact between the NIRS and the water, and the participants were instructed to distribute their weight evenly while standing. Given all these differences in protocols, it seems difficult to compare our results with theirs. If the result of Roberts et al. [55] are closer to ours during CWI, they reported no change in MSO_2 in the vastus lateralis during the rewarming period, unlike our. However, their participants walked to another experimentation room to perform an isometric maximum voluntary contraction. This may explain why Roberts et al. [55] did not observe a decrease in MSO_2 during the rewarming period, due to the increase in muscle temperature caused by the muscle activity through increased heat generation and blood flow. However, it is important to note that one of the limitations of this measurement is the change in muscle temperature during and after immersion. Indeed, it has been shown that the NIRS signal can be used to measure the in vivo temperature of water-containing tissue [57]. Nevertheless, several studies have investigated the evolution of muscle temperature at depths of 1,2,3 cm [41,58]. In 10 min their immersion protocols at 8°C, muscle temperature at 2 cm has already decreased by 3 to 5°C 1 min after exiting the cold bath. We can therefore hypothesize that during 15 min immersion at 10°C, muscle temperature at 2 cm depth starts to decrease during immersion. The NIRS signal is measured at a muscle depth corresponding to 50–60% of transmitter -receiver distance [59]. The farthest receiver on our NIRS was 25 mm from the transmitter, so we measured oxygenation at a depth of about 12 mm. As the current NIRS signal remained extremely stable throughout the immersion and decreased regularly 7 min after exiting, we believe that the observed variation can't be explained by the muscle temperature alone. Nevertheless, this remains a limitation of the current paper because this part of the discussion is partially based on the hypothesis that muscle temperature changes during CWI. The current decrease of MSO_2VM and MSO_2GM during the passive rewarming period could be rather explained by ST or the NST of the muscle (NST-M) since our participants did not perform any muscular activity after CWI but oxygen seems to be consumed. Unfortunately, neither EMG electrodes nor accelerometer were placed on the vastus medialis and gastrocnemius medialis muscles, which would have allowed us to differentiate

between ST and NST-M [25]. At the present stage it can only be said that NST-M and/or ST of the immersed muscles may have occurred more than 10 min after leaving the cold bath.

Combining all the results after the heating exercise, i.e. the increase in VO_2 during the cold water immersion with peripheral vasoconstriction, the decrease in MSO_2 during the subsequent rewarming during R3, and the decrease in gastrointestinal temperature during the final cycling period, reveals a continuum of different responses during CWI and rewarming. The first phase after the onset of immersion, the cold shock response, is thought to be neural rather than thermoregulatory in origin. This phase, which lasts approximately 3 min, was observed in our study with reflex vasoconstriction and increased VO_2, VCO_2, RER and Qc in agreement with the literature. The first thermoregulatory responses then occur, with VO_2 stabilizing at a higher value than in the AMB condition and RER decreasing. These phenomena are well described in the literature. The higher VO2 indicates an increase in energy expenditure of the non-submerged part of the body, probably related to ST or NST. Indeed, this does not seem to be due to an increase in metabolic activity of the lower limb muscles as shown by the stagnation of MSO_2VM and MSO_2GM, but rather of the trunk and/or upper limb muscles. There seems to be a transitional period when the participant comes out of the cold bath. Indeed, VO_2 has already returned to the same level as in AMB 5 min after the end of immersion, while peripheral vasodilation also seems to relax, as shown by the return of FABF to a lower but not significantly different level than in AMB. However, during this period of stagnation, MSO_2GM and MSO_2VM start to decrease 7 min after the end of the cold bath, but do not differ significantly from the AMB protocol until 31 min, i.e. 16 min after the end of immersion. At this point, a new phase in the fight against cold seems to emerge. Indeed, the gastrointestinal temperature drops after 30 min. This well-known mechanism is called afterdrop. However, it is interesting to note the stability of gas exchange values, but a slight relapse in FABF may indicate renewed peripheral vasoconstriction. The decrease in MSO_2 tends to be explained by an increase in thermogenesis at the muscle level, although the effect of temperature on the NIRS signal may be a limitation. These different phases are schematized in Figure 6, in relation to the evolution of the different measured variables useful for our discussion. It is also important to note that thermogenesis in the lower limb muscles did not completely warm them. Indeed, the decrease in gastrointestinal temperature on resumption of cycling could be due to an increase in convection link to the increase in blood flow to the legs, which cools on contact with the cold muscles and

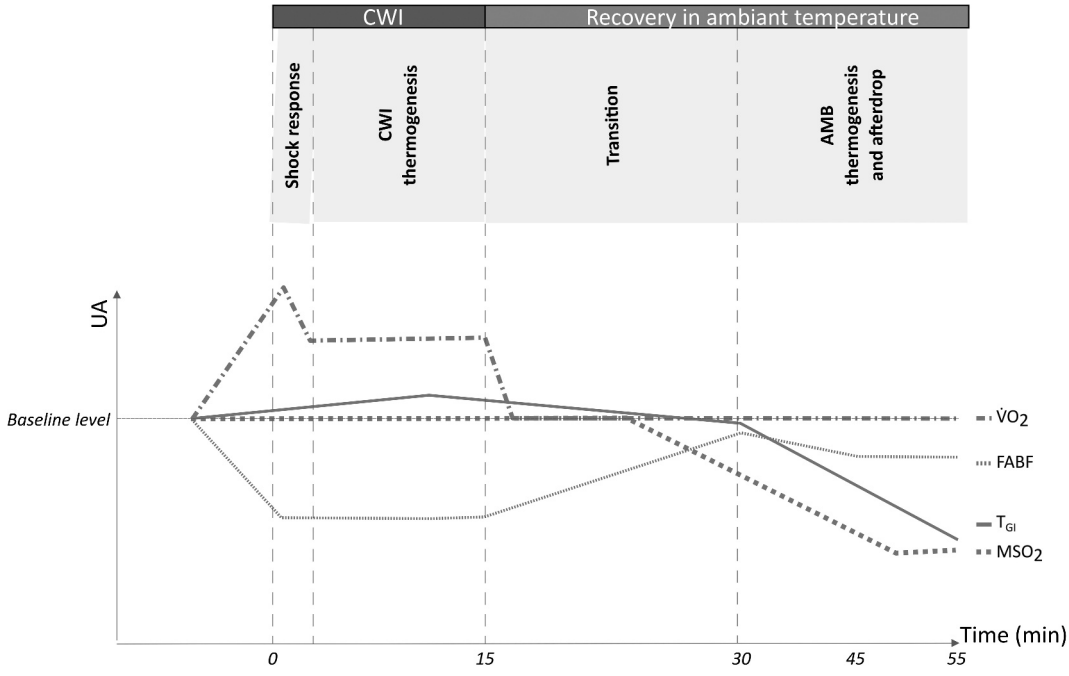


Figure 6. Schematic evolution of oxygen consumption (VO_2), gastrointestinal temperature (T_{GI}), muscle O_2 saturation (MSO_2) and arterial blood flow (FABF) during the CWI and the rewarming.

thus cool the deep body. The main limitation of this work is the lack of measurements to differentiate between ST, NST-B and NST-M. This would have allowed us to deepen our discussion, particularly by expressing the parts of the body where they occur during and after immersion. The second factor to be investigated in future research is the evolution of muscle temperature during the rewarming period.

Conclusion and perspective

This study represents one of the first attempts to combine measurements of stomach temperature, cardiac and arterial output with systemic and local oxygen consumption in immersed muscles during the immersion and rewarming periods, compared to passive recovery in ambient conditions, post cycling exercise. It's important to note that the participants were immersed up to the waist, and the responses might have been different if they hadn't exercised beforehand or if they had been immersed up to the neck. Our results demonstrated the existence of different responses during and after cold immersion, which can be distinguished by gastric temperature kinetics, hemodynamic and metabolic variables. Our main result was that thermogenesis during CWI seemed to take place initially in the non-immersed zones, before shifting to the immersed limbs during the rewarming period. Nevertheless, at 55 min post-CWI, thermogenesis in the lower limbs was still insufficient, given the drop in deep body temperature on resumption of physical activity. This underlines the importance of recording core, muscle and skin temperature changes in parallel, considering immersion and rewarming as a continuum rather than as separate events. Our results also highlight the importance of studying the rewarming state after cold immersion in order to understand the whole effects of this last.

Data accessibility

All the data are available to the following link: https://dataverse.harvard.edu/privateurl.xhtml?token=d5a43e33–8082-48e5-a3cc-cfa68acd7696 If the article is accepted, a permanent link will be created.

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Disclosure statement

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Abbreviation

AMB	Ambient temperature (°C)
ATP	Adenosine triphosphate
BAT	Brown adipose tissue
CWI	Cold-water immersion
FAD	Femoral Artery Diameter (mm)
FABF	Femoral Artery Blood Flow ($ml.min^{-1}$)
FABV	Femoral Artery Blood Velocity ($cm.s^{-1}$)
T_{GI}	Gastrointestinal temperature (°C)
NST	Non-shivering thermogenesis
Qc	Cardiac output ($L.min^{-1}$)
RER	Respiratory exchange ratio
MSO_2GM	Muscle oxygen saturation of the Gastrocnemius Medialis muscle (%)
MSO_2VM	Muscle oxygen saturation of the Vastus Medialis muscle (%)
ST	shivering thermogenesis
UCP1	uncoupling protein 1
VE	Ventilation ($L.min^{-1}$)
VCO_2	Relative carbon dioxide output ($ml.min^{-1}.kg^{-1}$)
VO_2	Relative oxygen uptake ($ml.min^{-1}.kg^{-1}$)
VT1	First ventilatory threshold
VT2	Second ventilatory threshold

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