

# The Effect of Acute and Prolonged Cooling and Heating on Muscle Force and Recovery

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## ABSTRACT

**Background:** It has been observed that temperature manipulation has different effect on skeletal muscle function. We hypothesise that both acute and prolonged heating will reduce muscle force more than cooling and recovery will be slower in the heating.

**Methods:** Ten participants (age  $27.1 \pm 6.8$  years) performed acute and prolonged thermal manipulations and neuromuscular testing. We measured muscle, rectal temperatures, P20, P100, and maximal voluntary isometric contraction (MVIC) after acute and prolonged cold water immersion (CWI) and hot water immersion (HWI) and control (CON) sitting.

**Results:** Muscle temperature ( $T_{mu}$ ) significantly increased following both acute and prolonged HWI and significantly decreased after acute and prolonged CWI, compared to baseline and CON conditions,  $p < 0.05$ . Rectal temperature ( $T_{rec}$ ) increased after HWI and declined significantly only during prolonged CWI,  $p < 0.05$ . Electrically evoked torques (P20 and P100) and MVIC decreased significantly after fatiguing stimulation across all conditions,  $p < 0.05$ , with greater post-recovery impairments in HWI compared to CWI, especially in the prolonged phase,  $p < 0.05$ . Central activation ratio (CAR) declined significantly only in the prolonged HWI condition after bath and following 3 min of recovery, compared to baseline and other conditions ( $p < 0.05$ ).

**Conclusions:** Although HWI effectively increases body temperature, it may significantly impair neuromuscular recovery more than CWI or passive rest.

**Keywords:** cold water immersion, hot water immersion, muscle force, temperature

## 1. INTRODUCTION

Cooling and heating, such as hot or cold water immersions (HWI or CWI) are applied before and after exercise to enhance recovery (Roberts et al., 2014; Cheng et al., 2017; Sautillet et al., 2024), reduce muscle soreness (Machado et al., 2016), extend endurance performance, and help to delay the development of heat-related fatigue (Baláš et al., 2024). However, prolonged cold and heat exposures can lead to impaired physical performance due to the risk of developing hypothermia or hyperthermia, which in turn impairs muscle function (Fudge et al., 2015; Periard et al., 2021).

Changes in core and muscle temperatures during heating and cooling interventions can also influence skeletal muscle function (Bergh & Ekblom, 1979; Brazaitis et al., 2011; Periard et al., 2021; Rodrigues et al., 2021). The extent of these adjustments can be modulated by exposure duration, water temperature, and the specific type of immersion (Brazaitis et al., 2011; Castellani & Young, 2016). Most studies typically use single water immersions to assess muscle force, with few examining the prolonged effects of temperature changes (Edwards et al., 1972; Davies et al., 1982; Brazaitis et al., 2011;

Rodrigues et al., 2021, 2023). The literature reports inconsistent findings on the effects of local cooling on muscle function, with some studies showing no significant impact (Petrofsky & Phillips, 1986), while others report a slight reduction (Asmussen et al., 1976; Brazaitis et al., 2011) on maximal voluntary isometric contraction (MVIC) force. Local muscle cooling impairs the ability to quickly generate force in both electrically evoked muscle twitches (Drinkwater & Behm, 2007) and voluntary contractions (Bergh & Ekblom, 1979). Accordingly, passive heating impairs voluntary activation (VA) of the muscles during sustained isometric contractions (Todd et al., 2005; Thomas et al., 2006) and is not always evident during brief maximal isometric contractions (Nybo & Nielsen, 2001). Increased muscle temperature ( $T_{mu}$ ) can enhance voluntary and involuntary (electrically evoked) muscle force production (Davies & Young, 1983; Brazaitis et al., 2011; Rodrigues et al., 2021).

Muscle force recovery following cooling and heating interventions can be influenced by both central and peripheral fatigue mechanisms. Fatigue caused by hyperthermia is related to the central nervous system (CNS), which includes decreased cerebral blood flow and disruptions in the neurotransmitter activity of the brain (Nybo et al., 2014). Research suggests that alterations in body temperature during prolonged exercise may, in part, influence performance through central mechanisms, as evidenced by a decrease in VA (Nybo & Nielsen, 2001) with increased core temperature. The results obtained by Brazaitis and others (2011) indicate that elevated core and muscle temperatures reduce VA and increase force variability of the exercised muscles; however, local muscle cooling decreases force variability and has only a minor effect on central fatigue. In contrast to cooling,  $T_{mu}$  increased due to heating can exacerbate fatigue during exercise (Periard et al., 2011; Bailey et al., 2012). Our research provides evidence-based insights how different recovery strategies, such as cooling and heating, affect muscle performance, fatigue, and recovery. Understanding these effects would help coaches and athletes understand how to apply thermal interventions to maximise athletes' performance while reducing the risk of fatigue related injuries. We aimed to investigate the effect of acute and prolonged heating and cooling on skeletal muscle fatigability and recovery. We hypothesise that both acute and prolonged heating will reduce muscle force during fatiguing exercise more than cooling and recovery will be slower in the heating.

## 2. METHODS

The current study builds on earlier investigations by Treigyte et al. (2023, 2024), which assessed the impact of single and intermittent/prolonged hot water immersion (HWI) and cold water immersion (CWI) on muscle force production and contractile characteristics under both unfatigued and fatigued states. In order to address the objectives of the current research, methodological procedures related to physiological assessments, neuromuscular testing, the fatiguing protocol, and data processing were applied in a manner consistent with those previously reported.

### 2.1 Participants

The study utilised a randomised cross-over design involving 10 recreationally active males. Participant baseline characteristics are shown in Table 1. Participants were eligible for inclusion if they were between 18 and 45 years of age, in good health, not currently involved in other research studies, physically active (2–3 times per week), not taking any medications, and had a body mass index (BMI) below 30 kg/m<sup>2</sup>. Individuals were excluded if they had a history of health problems: asthma, neurological or cardiovascular diseases, and other conditions that could be aggravated by cold exposure, or any illnesses or physical impairments that might interfere with their ability to complete the neuromuscular assessments.

Table 1. Characteristics of participants

| Males                  | n = 10      |
|------------------------|-------------|
| Age, years             | 27.1 ± 6.8  |
| Height, cm             | 187.5 ± 7.5 |
| Body mass, kg          | 85.9 ± 7.8  |
| Body fat, %            | 18.0 ± 2.5  |
| BMI, kg/m <sup>2</sup> | 24.8 ± 2.2  |

### 2.2 Ethical approval

All experiments took place at Lithuanian Sports University (Kaunas, Lithuania). The Kaunas Regional Biomedical Research Ethics Committee approved the study protocol (no. P1-BE-2-14/2022), which complied with the latest revision of the Declaration of Helsinki. All participants were fully informed of the procedures and provided written consent before participation.

### 2.3 Experimental design

Participants were required to visit the laboratory

six times over the experimental research. During the week prior to the first experiment, participants were familiarised with the protocols, equipment, neuromuscular assessments, and the fatiguing protocol. The study consisted of two phases: acute and prolonged immersions. Participants were randomly assigned to begin the study with one of the protocols. The acute phase, which was completed first, consisted of a 45 min session involving either sitting on a chair (CON) or immersion in cold (CWI) or hot (HWI) water, followed by the prolonged phase of 300 min sitting (CON) or immersions (CWI, HWI). Participants were randomly assigned to the order in which they began the protocols, with a minimum 1-week break between experiments. On each testing day, participants arrived at the laboratory in the morning (9.00 a.m.) following an overnight fast. Baseline measurements, including body mass, body composition, rectal temperature ( $T_{\text{rec}}$ ), and muscle temperature ( $T_{\text{mu}}$ ), were recorded after 15 min of seated rest. Participants then completed the initial neuromuscular assessment, which was followed by a 45 min intervention consisting of either passive sitting in the laboratory (CON), immersion in cold water at  $10.0 \pm 0.2^\circ\text{C}$  (CWI), or immersion in hot water at  $44.5 \pm 0.5^\circ\text{C}$  (HWI). For CWI, ice slush was added to maintain the target temperature, whereas in HWI, hot water was supplemented while cooler water was simultaneously released to stabilise temperature.  $T_{\text{rec}}$  was monitored every 5 min throughout this period. Immediately after the 45 min intervention or control (CON) sitting,  $T_{\text{rec}}$  and  $T_{\text{mu}}$  were measured again, and a second neuromuscular assessment was conducted. During the acute phase, the neuromuscular testing was followed by a fatiguing protocol involving 100 trains of electrical stimulation applied to the knee extensors and after 3 min recovery neuromuscular testing was repeated. During the CWI prolonged phase, two additional bouts of 15 min cold water immersions were added at 165 min and 255 min. While during HWI prolonged phase, eight additional baths for 15 min were added at 75, 105, 135, 165, 195, 225, 255, and 285 min in order to maintain elevated  $T_{\text{rec}}$  and  $T_{\text{mu}}$ . In the prolonged phase, this protocol was extended to ensure  $T_{\text{mu}}$  and  $T_{\text{rec}}$  remained elevated and stable over 300 min while minimising the risk of hyperthermia. During the prolonged CON condition, participants remained seated at room temperature for the full duration.  $T_{\text{rec}}$  was recorded every 15 min from 60 min until the end of the 300 min protocol, whereas  $T_{\text{mu}}$  was only measured before and after both acute and prolonged interventions. After each

prolonged immersion or CON sitting, neuromuscular testing was followed by the same fatiguing protocol as in the acute phase, with subsequent testing after a 3 min recovery. During the prolonged phase, participants were provided with a strawberry-flavoured breakfast cereal bar, whereas no food was given during the acute sessions. Participants were not allowed to drink water during any experimental session but were permitted to rinse their mouths with cool water. For immersion sessions, participants wore only swimming shorts and were quickly dried after each bath before neuromuscular testing. Between immersion periods or CON sitting, participants wore shorts and a T-shirt.

#### 2.4 Anthropometric measurements

Body mass and body fat percentage were assessed using a body composition analyser (*Tanita, TBF-300, IL, USA*). Participant height was recorded with a height gauge, and BMI was subsequently calculated.

#### 2.5 Body temperature measurements

$T_{\text{rec}}$  was measured using a thermocouple probe (*Ellab, Hvidovre, Denmark;  $\pm 0.01^\circ\text{C}$* ) inserted 12 cm past the anal sphincter.  $T_{\text{mu}}$  was assessed with a needle microprobe (*Ellab, DM-852*) inserted 3 cm into the vastus lateralis of the right thigh at mid-thigh, slightly lateral to the femur.

#### 2.6 Water immersions

Cold water immersion (CWI) was applied during both the acute and prolonged protocols, with participants seated in a  $10.0 \pm 0.2^\circ\text{C}$  water cold bath, immersed their bodies from the legs up to the waist. During the hot water immersion (HWI) protocols, participants similarly immersed their bodies from the legs up to the waist in a water bath at  $44.5 \pm 0.5^\circ\text{C}$ .

#### 2.7 Neuromuscular testing

Neuromuscular function was assessed via electrically evoked torque and maximal voluntary isometric contraction (MVIC) of the knee extensors. Participants were seated upright in an isokinetic dynamometer (*System 4, Biodex Medical Systems, Shirley, NY, USA*), calibrated according to the manufacturer's instructions, with gravity correction applied using the Biodex Advantage software (*Version 4.X*). The trunk, shank, and shoulders were stabilised using straps, and the knee joint was fixed at a  $90^\circ$  angle. Electrical stimulation was delivered through three  $12 \times 8$  cm carbonised rubber electrodes (*MARP Electronic*), coated with conductive

gel (*ECG-EEG Gel, MediGel, Modi'in, Israel*). Two electrodes were positioned on the proximal quadriceps (vertically and transversely), while a third covered the distal quadriceps above the patella. Electrode sites were marked for consistency across all six experimental sessions. A Digitimer DS7A stimulator (*Digitimer, Hertfordshire, UK*) delivered 0.5 ms square-wave pulses at 100 mA with a 200 V voltage limit, which ensured full quadriceps activation (Eimantas et al., 2022). The testing protocol began with 1 s electrical stimulations at 20 Hz (P20) and 100 Hz (P100), separated by 3 s of rest. This was followed by two MVICs, each separated by 1 min rest. During MVIC, participants were instructed to exert maximal effort for approximately 5 s, and a 250 ms superimposed 100 Hz stimulation was applied 3–4 s into the contraction to calculate the central activation ratio (CAR). CAR, indicating voluntary muscle activation, was calculated by expressing MVIC torque as a percentage of the total peak torque measured during the superimposed 250 ms test train. The highest MVIC torque achieved across attempts was used for analysis. Immediately following neuromuscular testing and after either immersion or control (CON) sitting,

participants performed a fatiguing protocol consisting of 100 trains of electrical stimulation of the knee extensor muscle (250 ms test train stimulation at 100 Hz (TT) interspaced with 1 s break). After a 3 min recovery, electrical stimulations and MVIC measurements were repeated following the same sequence.

## 2.8 Data analysis

Data were analysed using GraphPad Prism 9.0.2. Normality was confirmed with the Shapiro–Wilk test. Results are presented as mean  $\pm$  SD. Two-way repeated-measures ANOVA assessed the effects of condition, time, and their interaction, with Sidak's post hoc tests applied when significant interactions were observed. Significance was defined as  $p < 0.05$ .

## 3. RESULTS

The baseline physiological and muscle force parameters of the subjects are presented in Table 2. No differences were found between acute and prolonged groups in all three temperature conditions.

Table 2. Baseline temperature and muscle force indicators of the subjects

| Indicators             | Baseline     |              |               |              |              |              |
|------------------------|--------------|--------------|---------------|--------------|--------------|--------------|
|                        | CON          |              | CWI           |              | HWI          |              |
|                        | Acute        | Prolonged    | Acute         | Prolonged    | Acute        | Prolonged    |
| Muscle temperature, °C | 35.7 (0.8)   | 35.6 (0.6)   | 35.6 (0.6)    | 35.6 (0.6)   | 36.0 (0.6)   | 35.6 (0.6)   |
| Rectal temperature, °C | 36.9 (0.2)   | 36.8 (0.4)   | 36.9 (0.2)    | 36.9 (0.3)   | 36.9 (0.3)   | 36.9 (0.2)   |
| 20 Hz torque, (Nm)     | 137.2 (48.0) | 141.3 (57.3) | 145.9 (56.3)  | 144.7 (52.7) | 142.2 (52.7) | 144.4 (52.5) |
| 100 Hz torque, (Nm)    | 195.2 (80.4) | 193.6 (93.3) | 203.6 (97.6)  | 198.7 (80.5) | 199.2 (85.2) | 198.5 (75.7) |
| MVIC torque (Nm)       | 317.0 (86.2) | 304.5 (94.3) | 317.3 (118.0) | 294.0 (74.8) | 295.1 (96.8) | 302.8 (85.8) |
| CAR (%)                | 95.8 (6.0)   | 94.7 (5.3)   | 97.2 (3.8)    | 95.8 (4.7)   | 95.6 (4.4)   | 95.7 (5.6)   |

### Effect of acute and prolonged thermal manipulations on body temperature responses

Muscle temperature ( $T_{mu}$ ) changes were similar after acute ( $+2.5 \pm 0.63^\circ\text{C}$ ) and prolonged ( $+2.9 \pm 0.56^\circ\text{C}$ ) HWI, with a significant increase compared to baseline and CON conditions ( $p < 0.05$ ). Both acute and prolonged CWI led to similar decreases in  $T_{mu}$  ( $-3.7 \pm 2.09^\circ\text{C}$  and  $-4.3 \pm 1.15^\circ\text{C}$ ), with a significant decrease compared to baseline and CON

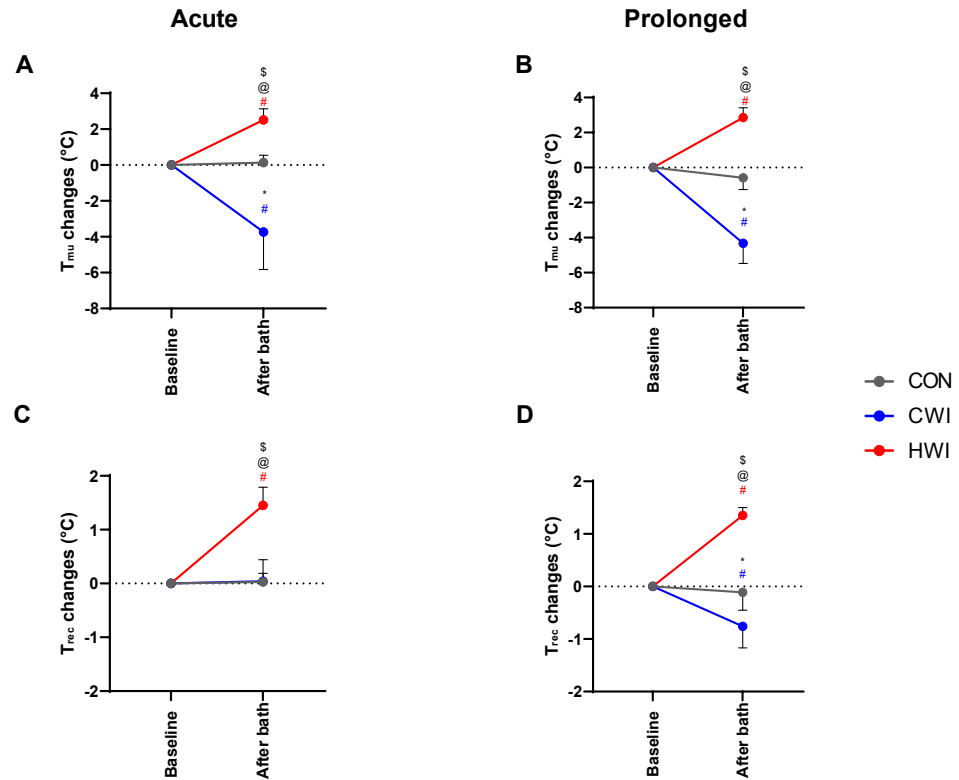
conditions ( $p < 0.05$ ). During acute and prolonged CON condition  $T_{mu}$  remained similar. Significant differences in  $T_{mu}$  changes after bath were observed between the HWI and CWI groups during both acute and prolonged phases ( $p < 0.05$ ) (Figure 1A; 1B).

Rectal temperature ( $T_{rec}$ ) changes were similar after acute ( $+1.5 \pm 0.34^\circ\text{C}$ ) and prolonged ( $+1.4 \pm 0.15^\circ\text{C}$ ) HWI, with a significant increase compared to baseline and CON conditions ( $p < 0.05$ ). During

acute CWI and both CON conditions,  $T_{rec}$  remained similar to baseline, while it decreased during prolonged CWI ( $-0.76 \pm 0.41^{\circ}\text{C}$ ), with a significant change compared to baseline ( $p < 0.05$ ). Statistically

significant differences in  $T_{rec}$  changes after bath were observed between the HWI and CWI groups during both acute and prolonged phases ( $p < 0.05$ ) (Figure 1C; 1D).

Figure 1. Changes in muscle ( $n = 9$ ) and rectal ( $n = 10$ ) temperatures from baseline to post-immersion following acute and prolonged baths under different temperature conditions



# – significant differences between baseline and after HWI bath; # – significant differences between baseline and after CWI bath; \* – significant differences between CON and CWI; @ – significant differences between CON and HWI; \$ – significant differences between HWI and CWI;  $p < 0.05$ ;  $T_{mu}$  – muscle temperature;  $T_{rec}$  – rectal temperature.

### Impact of acute and prolonged thermal manipulations on electrically evoked muscle torques

Relative P20 and P100 torques during electrically evoked contractions are presented in Figure 2A; 2B; 2C; 2D. P20 remained unchanged at the end of the bath following acute and prolonged exposure in all experimental conditions. However, P20 torques significantly decreased in all acute (CON – 46.8%, CWI – 41.4%, HWI – 61.5%) and prolonged (CON – 55.6%, CWI – 47.6%, HWI – 60.4%) conditions after 100 stimuli (in fatigue state), compared to baseline ( $p < 0.05$ ). After 100 stimuli (in fatigue state), significant differences were obtained between CON and CWI (prolonged phase), and between HWI and CWI (acute and prolonged phases),  $p < 0.05$ . After 3 min of recovery, relative P20 torques remained significantly reduced from baseline in all groups (acute phase: CWI – 17.5%, HWI – 37.6%,

prolonged phase: CON – 29.2%, CWI – 23.3%, HWI – 34.4%),  $p < 0.05$ , with the exception of the acute CON condition. Significant differences after 3 min recovery were observed between the HWI and CWI conditions ( $p < 0.05$ ).

P100 torques decreased ( $-15.4\%$ ) at the end of the bath only in acute CWI, compared to baseline, and significant differences were found at the end of the bath between CON and CWI, HWI and CWI conditions in acute phase ( $p < 0.05$ ). P100 torques significantly decreased in all acute and prolonged conditions after 100 stimulus (in fatigue state) (acute: CON – 36.8%, CWI – 40.9%, HWI – 46.3%, prolonged: CON – 40.2%, CWI – 37.4%, HWI – 43.9%), compared to baseline ( $p < 0.05$ ). After 3 min of recovery, relative P100 torques remained significantly reduced from baseline only in HWI conditions (acute phase – 23.6%; prolonged phase – 16.6%) ( $p < 0.05$ ). Significant differences

after 3 min recovery were observed between the HWI (–23.6%) and CWI (–12.3%) conditions only in acute phase ( $p < 0.05$ ).

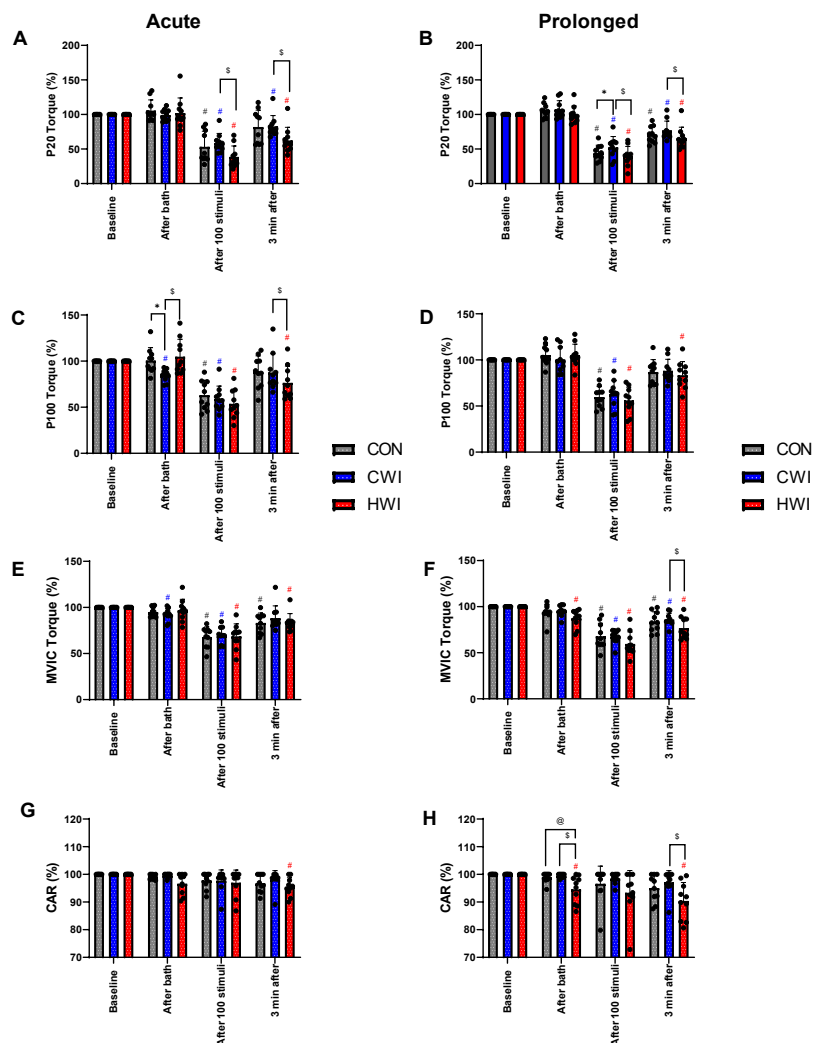
#### *Impact of acute and prolonged thermal manipulations on voluntary muscle contraction*

Relative MVIC torques and CAR are presented in Figure 2E; 2F; 2G; 2H. MVIC torques significantly decreased at the end of the bath in acute CWI (–8.0%) and in prolonged HWI (–12.6%), compared to baseline ( $p < 0.05$ ). Additionally, MVIC torques were significantly reduced in all acute (CON – 32.2%, CWI – 30.6%, HWI – 31.3%) and prolonged (CON – 32.1%, CWI – 32.3%, HWI – 40.5%) phases after 100 stimuli (in fatigue state), compared to baseline ( $p < 0.05$ ). After 3 min of recovery, MVIC torques remained significantly lower than baseline in the acute CON (–16.9%) and HWI (–16.3%) conditions, while during the prolonged

phase, reductions were observed across all groups (CON – 16.6%, CWI – 14.0%, HWI – 23.2%) ( $p < 0.05$ ). Statistically significant differences after 3 min recovery were observed between the HWI and CWI conditions only in the prolonged phase ( $p < 0.05$ ).

CAR remained unchanged across all time points in the acute phase, except for a significant decrease found after 3 min of recovery (–4.5%), compared to baseline ( $p < 0.05$ ). In the prolonged phase, CAR decreased at the end of the bath (–5.5%) and after 3 min of recovery (–9.6%) in HWI condition, compared to baseline ( $p < 0.05$ ). At the end of the bath (prolonged phase), significant differences in CAR were observed between HWI (–5.5%) and CWI (–0.5%) conditions ( $p < 0.05$ ). After 3 min of recovery (prolonged phase), CAR was also lower in HWI (–9.6%) than in CWI (–2.9%) ( $p < 0.05$ ).

Figure 2. Isometric torque during electrically induced and voluntary muscle contraction ( $n = 10$ )



# – significant differences between baseline and after HWI bath; @ – significant differences between baseline and after CWI bath; \$ – significant differences between baseline and CON; \* – significant differences between CON and CWI; @ – significant differences between CON and HWI; \$ – significant differences between HWI and CWI;  $p < 0.05$ ; P20 – 20 Hz torques; P100 – 100 Hz torques; MVIC – maximal voluntary isometric contraction; CAR – central activation ratio.

## 4. DISCUSSION

The present study investigated the effects of acute and prolonged cooling and heating on skeletal muscle force resistance to electrically induced fatiguing exercise and recovery. The findings indicated that acute and prolonged hot water immersion significantly increased muscle and core temperatures, while cold water immersion led to notable reductions in muscle temperature ( $T_{mu}$ ) and a moderate drop in rectal temperature ( $T_{rec}$ ) during prolonged exposure. Neuromuscular performance, including electrically evoked and voluntary muscle contractions, declined significantly after all conditions following fatiguing stimulation. MVIC was reduced more prominently in prolonged heating and CAR declined only in heating, especially during the prolonged phase. These findings suggest that while heating effectively elevates body temperature, it may negatively affect neuromuscular recovery more than cooling or control (CON) conditions.

As we expected, P20 and P100 were not affected at the end of a hot bath. Our findings are in line with earlier research that reported similar results in males (Brazaitis et al., 2019; Baranauskiene et al., 2023). However, significant changes were found for P100 at the end of the bath in acute cooling, and for P20 after exercise in prolonged cooling, compared to CON. This agrees with the study of Brazaitis et al. (2016), where they found that cooling impairs force production, especially at high frequency stimulations. Cooling slows nerve conduction velocity, reduces synaptic transmission efficiency, therefore these changes are more visible at high stimulation frequencies (Brazaitis & Skurvydas, 2010). Regardless, prolonged or intense cooling can also affect low-frequency torque, especially after fatiguing exercise (Solianik et al., 2015; Brazaitis et al., 2016).

Relative MVIC of knee extensors was not affected by cooling. Although the study of Petrofsky & Phillips (1986) reported no effect of local cooling on muscle force, our findings are inconsistent with other results, which reported a reduction in MVIC of knee extensors after cooling (Bergh & Ekblom, 1979; Brazaitis et al., 2011). Acute and prolonged heating also did not affect MVIC torques, however prolonged heating showed greater decrease in MVIC force after exercise and reduced force during recovery. This can be supported by the study results of Bergh & Ekblom (1979), where they found that elevated  $T_{mu}$  variations have minimal to no effect during maximal isometric contractions. In addition to that, a reduction in maximal voluntary

contraction (MVC) torque occurs during both moderate (approximately 38.5°C) and severe hyperthermia (around 39.5°C) (Morisson et al., 2004; Todd et al., 2005; Periard et al., 2014).

In our study, CAR was not affected by acute cooling and heating, while a significant reduction was observed after the bath in the prolonged heating condition, compared to CON. Studies that assessed CAR after cooling also found no effect on voluntary activation (VA) during MVIC (Brazaitis et al., 2014). However, elevated core temperature can also impair central drive to the working muscles (Racinais & Oksa, 2010). Rodrigues and colleagues (2023) found that elevated core temperature during immersion in 42°C water for 2 h reduces maximal voluntary contraction (MVC) torque and VA level. According to Morrison et al. (2004), passive hyperthermia reduces VA due to CNS impairment rather than peripheral changes. This CNS impairment involves altered function in the motor cortex and spinal pathways, and potentially impaired neurotransmission due to increased core temperature.

The results of this study demonstrate that HWI effectively increases  $T_{mu}$ , however, it may impair neuromuscular recovery more than CWI or passive rest. These findings are consistent with Roberts et al. (2014), where they found that CWI at 10°C water improved the recovery of submaximal muscle function. In contrast, Jackman et al. (2023) observed that post-exercise HWI did not accelerate recovery and was less effective than passive rest for restoring muscle function. Elevated temperature may affect the responsiveness of the central nervous system and impair VA, leading to greater decrements in CAR (Sautillet et al., 2024).

### 4.1 Practical application and perspectives

Our results indicated the effect of thermal manipulations, such as HWI and CWI, on muscle strength and recovery in recreationally active males. This is especially relevant for sports coaches, athletes who seek to improve their performance and recovery, or people who are constantly exposed to temperature changes at work or in daily activities (military officers, skiers, marathon runners). CWI can effectively reduce muscle and core temperatures; however, it may impair neuromuscular function, particularly high-frequency force production, after application. Coaches and athletes should be careful when using CWI right before activities that need high muscle power. HWI, commonly used to promote relaxation and circulation, resulted in more pronounced and sustained impairments in torque

and CAR, particularly in the prolonged phase. Prolonged heating should be avoided immediately prior to training or competition. These results can help optimise recovery and fatigue management, performance and training modulations, improve rehabilitation processes.

#### 4.2 Limitations

The study involved only recreationally active men as participants. The sample size in this study was relatively small (only 10 subjects), therefore, the findings should be confirmed in future studies with a larger sample size. Our findings demonstrate significant effects of hot and cold water immersion (HWI and CWI) on muscle and core temperatures in recreationally active males using passive heating and cooling protocols. However, these results may not directly translate to other populations, such as elite athletes, or situations involving other heating or cooling protocols. It is unclear whether similar results would be observed in females, who may indicate different thermoregulatory responses, muscle fatigue profiles, or recovery dynamics due to physiological and hormonal differences. Therefore, caution should be exercised when applying these findings to performance-based recommendations. Another aspect to be aware of is that the findings from our study are based only on water immersion methods (HWI and CWI), limiting the applicability of the results to different thermal interventions. It remains unclear whether using other recovery methods, such as cryotherapy chambers, dry sauna, or contrast therapy, would have led to similar results. The prolonged cooling and, especially, heating protocols were not easy for the subjects, thus applicability to real-world conditions may be challenging. In our study,  $T_{mu}$  was measured only at 3 cm depth, thus variability in  $T_{mu}$  may be attributed to technical issues or reliability of the probes, as well as the inability to fully control participants' motivation during MVIC. Finally, the acute phase preceded the prolonged phase, but the three conditions were randomised, which should have limited any adaptation effects. However, some differences between phases may still be observed.

## 5. CONCLUSIONS

Both acute and prolonged hot water immersion (HWI) significantly increased muscle and core temperatures, while cold water immersion (CWI) led to substantial reductions in muscle temperature ( $T_{mu}$ ) and a moderate decrease in core temperature during prolonged exposure in recreationally active males. Although thermal manipulations, such as HWI and CWI, induced comparable shifts in tissue temperature, neuromuscular performance declined across all experimental conditions following fatiguing stimulation. Importantly, maximal voluntary isometric contraction (MVIC) was more markedly reduced following prolonged heating, and reductions in central activation ratio (CAR) were observed exclusively under hot water conditions, particularly during the prolonged phase. This impairment appears to result partly from central and partly from peripheral effects. Involuntary force P20, P100 are more closely linked to  $T_{mu}$ , while MVIC is influenced by core and muscle temperatures. These results indicate that although HWI effectively increases body temperature, it may significantly impair neuromuscular recovery when compared to CWI or passive control.

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