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## ABSTRACT

**Purpose:** In recent years, there has been significant advancement in the guidelines for recovery protocols involving heat or cold water immersion. Yet, comparison between the effects of hot and cold water immersion on key markers of neuromuscular recovery following exercise-induced muscle damage (EIMD) is lacking.

**Methods:** Thirty physically active males completed an individualized and tailored EIMD protocol immediately followed by one of the following recovery interventions: cold water immersion (11°C, CWI<sub>11</sub>), hot water immersion (41°C, HWI<sub>41</sub>) or warm-bath control (36°C, CON<sub>36</sub>). Gastrointestinal temperature was tracked throughout HWI<sub>41</sub>. Knee extensors' maximal isokinetic strength [peak torque ( $T_{\text{peak}}$ )] and explosive strength [late-phase rate of force development, (RFD<sub>100-200</sub>)] were measured prior to EIMD (pre-), 24h (post-24h) and 48h (post-48h) post-EIMD. In addition, pressure pain threshold (PPT) was measured to quantify the recovery from muscle soreness. Surface electromyography signals (sEMG) from the *vastus lateralis* were captured to extract the rates of electromyography rise (REMGR) and the spectral power in the low-frequency band.

**Results:** At post-48h,  $T_{\text{peak}}$  returned to baseline values following both CWI<sub>11</sub> (-8.3±6.8 %,  $p=0.079$ ) and HWI<sub>41</sub> (-1.4±4.1%,  $p=1$ ). In contrast, RFD<sub>100-200</sub> (-2.3 ± 29.3%,  $p=1$ ) and PPT (+5.6±14.6%,  $p=1$ ) returned to baseline values at post-48h only following HWI<sub>41</sub>. Spectral analysis of the sEMG signal revealed that the low-frequency band was significantly increased following CWI<sub>11</sub> (+9.0 ± 0.52%,  $p=0.012$ ). REMGR was unchanged regardless of the

condition (all  $p > 0.05$ ).

**Conclusions:** A single session of HWI<sub>41</sub>, rather than CWI<sub>11</sub>, improved the recovery of the late-phase rate of force development following EIMD in physically active males. This suggests that in athletic contexts where a rapid force development is a key performance determinant, hot bath should be preferred over cold bath.

**Key Words:** RECOVERY, IMMERSION TEMPERATURE, NEUROMUSCULAR FATIGUE, MAXIMAL STRENGTH, EXPLOSIVE STRENGTH, LATE-PHASE RATE OF FORCE DEVELOPMENT

ACCEPTED

## INTRODUCTION

Neuromuscular fatigue is usually defined as a reduced ability to generate strength or power (1). In the context of exercise-induced muscle damage (EIMD), muscle fatigue coincides with structural damage of myofibers and the occurrence of delayed onset muscle soreness (DOMS) (2). Following the insult, a finely regulated immuno-inflammatory response is observed (3), serving as a key step in the initiation of muscle repair (3). In animal model involving non-physiological (i.e. severe) damaging protocols, this step is frequently referred to as “secondary damage” (4), owing to observations that the decline in force-generating capacity is linked to the extent of the immuno-inflammatory response (5, 6). Conversely, a slightly enhanced immuno-inflammatory response alongside blunted markers of EIMD were observed following a second bout of eccentric contractions in humans (7). Taken together, there is no strong evidence that a sustained or exaggerated response would exacerbate indirect markers of secondary muscle damage (e.g. functional recovery or DOMS) in humans.

Recovery interventions involving core and/or muscle temperature manipulation are hypothesized to modulate the dynamic of the immuno-inflammatory response (8-10), with the hope to accelerate neuromuscular recovery (11). Cryotherapy, particularly through cold water immersion (CWI), is nowadays widely embraced by athletes and coaches (12, 13). The rationale for cryotherapy is to maintain a reduction in intramuscular temperature in the immediate stage following the insult, aiming to inhibit the proliferation of the hypothesized secondary damage (14). Often claimed for its anti-inflammatory properties, the use of cryotherapy to accelerate the recovery of muscle function has been challenged on several occasions (8, 15, 16). Although animal studies demonstrated the effectiveness of cryotherapy

in lowering inflammation response (17), these outcomes remain unclear in humans (9, 18). In a recent meta-analysis, pooled data highlighted contrasting results on neuromuscular recovery: whereas CWI induced positive effects on functional parameters related to muscular power (e.g. jump, sprint) 24 h following EIMD, it lacked efficacy in the recovery of maximal strength, i.e. muscle peak torque ( $T_{\text{peak}}$ ) values (19). Furthermore, all studies that included a placebo in their design revealed that CWI was as effective as the placebo condition on neuromuscular recovery, thus raising questions about the effectiveness of this strategy (20).

More recently, thermotherapy in the form of passive hot water immersion (HWI) has attracted interest to promote neuromuscular recovery, since heat might play a key role in modulating the immune-inflammatory response (21). The effects of HWI during the early recovery phase (i.e., within hours post-exercise) appear promising, whether employed in a laboratory-based experimental setting (22) or implemented between training sessions within the same day (23). Conversely, when HWI is applied in a context where late recovery phase is of interest – 24 h to 72 h following a fatiguing task – contrasting outcomes have been observed in terms of neuromuscular recovery, muscle damage or DOMS (24-27). The recent study by Sautillet and colleagues demonstrated that two closely spaced HWI temperature (40°C and 41°C) elicited divergent effects on neuromuscular recovery following EIMD: the 41°C condition succeeded in alleviating DOMS and restoring neuromuscular function to baseline values, whereas the 40°C condition did not (28). In the aforementioned studies that failed to show clear benefits of HWI (24-27), it is worth noting that the temperature used was within the range of 36°C-40°C, a heat stress most likely insufficient for improving neuromuscular recovery. Taken together, the existing literature suggests a potential dose-

response relationship, where low-to-moderate levels of HWI temperature (36°C-40°C) have limited effects on neuromuscular recovery, whereas a more severe strain ( $\geq 41^\circ\text{C}$ ) could yield beneficial outcomes. Finally, studies directly comparing CWI and HWI during the late recovery phase showed conflicting results on neuromuscular recovery (25, 26, 29), preventing a conclusive determination of the superiority of one method over the other (30).

Much of the research examining the influence of CWI or HWI on neuromuscular function has primarily focused on the recovery kinetics of peak torque values ( $T_{\text{peak}}$ ), typically obtained through maximal isometric contractions (19, 30). However, the ability to generate a high amount of force (or torque) within a short interval is considered functionally more relevant than a single peak value such as  $T_{\text{peak}}$  (31). This capacity is defined as the rate of force development (RFD) and is assessed by measuring the slope of the force vs. time curve during brief contractions, usually within a 250 ms timeframe (32). Interestingly, RFD emerged as a more sensitive indirect marker of muscle damage than  $T_{\text{peak}}$ , since previous studies pointed out that RFD was more impacted by EIMD than  $T_{\text{peak}}$  (33, 34). Taken together, the exploration of RFD – rather than relying solely on  $T_{\text{peak}}$  – holds potential interest in discerning the effects of CWI and HWI on neuromuscular recovery (35). Additionally, the analysis of surface electromyography (sEMG) signals in the frequency domain can complement the examination of RFD. Neuromuscular fatigue is known to induce distinct changes in the spectral power across various sEMG frequencies. To date, examination of the spectral density of low- and high-frequency sEMG components separately has facilitated the identification of the differential contributions of slow- and fast-twitch muscle fibers, with recent applications in studying neuromuscular fatigue (36, 37). Specifically, the power

spectral density of the low-frequency band emerged as particularly sensitive to neuromuscular fatigue (38).

Therefore, the main aim of this study was to compare pre- and post-EIMD the effects of CWI or HWI on: quadriceps *femoris*  $T_{peak}$ , RFD, DOMS and power spectral density of the low-frequency band, while ensuring that both CWI and HWI were administered in accordance with the latest guidelines for improving neuromuscular recovery. Given the limited evidence suggesting that blunting the inflammatory response would be advantageous for muscle repair, we hypothesized that HWI would alleviate EIMD-decline in RFD and soreness, whereas CWI would not. Consistent with this hypothesis, CWI would result in an elevated spectral density in the low-frequency band derived from sEMG signals, indicative of a more pronounced neuromuscular fatigue.

## MATERIALS AND METHODS

### Participants

Thirty healthy, physically active young male individuals (see statistical section for sample size calculation) were recruited and split into 3 groups: CWI<sub>11</sub> (11°C, n = 10), HWI<sub>41</sub> (41°C, n = 10) and a warm-bath control, *i.e.* CON<sub>36</sub> (36°C, n = 10). Groups were matched for age (CWI<sub>11</sub>: 21.5 ± 1.3 years; HWI<sub>41</sub>: 20.5 ± 1.3 years; CON<sub>36</sub>: 20.5 ± 1.8 years), body mass index (CWI<sub>11</sub>: 23.6 ± 2.7 kg.m<sup>-2</sup>; HWI<sub>41</sub>: 22.4 ± 2.1 kg.m<sup>-2</sup>; CON<sub>36</sub>: 22.3 ± 4.7 kg.m<sup>-2</sup>) and training level (> 6 hours of training per week). Participants were asked to refrain from exercising or using any additional recovery strategy throughout the protocol. The study received institutional ethical approval (ETH-23IRFC-I). Before participating, each subject

provided written consent after being fully informed.

## **Experimental design**

This randomized controlled study was designed to assess and compare the effects of two specific water temperatures (11°C and 41°C) on key markers of neuromuscular recovery. Participants visited the laboratory on three consecutive days (figure 1). On day 1, baseline measurements were taken, and subsequently, the EIMD protocol was executed. Participants were randomly assigned to one of the following recovery interventions: CWI<sub>11</sub>, HWI<sub>41</sub> or CON<sub>36</sub>, within 30 minutes post-EIMD. During the subsequent two visits on post-24h (day 2) and post-48h (day 3), the same set of parameters as those measured on day 1 were assessed.

## **Hot water Immersion and Warm-bath control protocols**

An individually tailored HWI protocol was proposed to the participants. Briefly, participants were immersed in the hot bath until their core body temperature reached and remained within the range of 38.5-39°C for ~25 minutes. This methodology has recently demonstrated its effectiveness in accelerating neuromuscular recovery (28). Immersion up to the waist was preferred over whole-body immersion, as the former represents a relevant compromise, ensuring adequate and safe increase in core body temperature with minimized thermal discomfort. Core body temperature was continuously tracked during immersion with a telemetric temperature capsule (e-Celsius, BodyCap, Caen, France), which was ingested by the participants at least 6 hours prior to the onset of the immersion. Participants were immersed in an inflatable bath (Arebos, France) to the waist level, seated, legs fully extended and forearms non-submerged. Throughout immersion, water temperature was meticulously

maintained at 41°C to ensure a significant rise in core body temperature within the specified target range. After achieving a stable core body temperature of 38.5-39°C for ~25 minutes, participants exited the bath and subsequently rested on a chair until their core body temperature returned to baseline values. They were encouraged to drink water throughout immersion and following immersion, a fluid ingestion equivalent to 120% of body mass loss. The warm-bath control (*i.e.* CON<sub>36</sub>) replicated the identical procedure except that the water temperature was set at 36°C. Since there was no increase in core body temperature during CON<sub>36</sub>, the immersion time during CON<sub>36</sub> was adjusted to match the expected duration of HWL<sub>41</sub> (28).

### **Cold water immersion protocol**

The CWI protocol consisted of full-body immersion (head-out, seated) in water maintained at a temperature of 11°C for a duration of 11 minutes, using a dedicated ice bath system (Cryo Control TEAM, Cryo Control, Toulouse, France). The immersion duration, water temperature and depth of immersion adhered to contemporary recommendations designed to accelerate neuromuscular recovery and reduce soreness (19, 39).

### **Data acquisition**

EIMD protocol and assessment of the quadriceps *femoris* neuromuscular function were performed using an isokinetic dynamometer (Cybex Norm<sup>®</sup>, division of Lumex, Inc., Ronkonkoma, New York, USA). Before testing, participants followed a standardized warm-up made up of 8 min of cycling at 75-100 Watts in addition to 20 warm-up contractions on the dynamometer. Participants were seated in an upright position and then fastened using straps to

secure the chest, hip, and ankle of the dominant (working) leg. The range of motion was set from 0° (full knee extension) to 90° knee flexion for all participants. The lateral epicondyle of the knee joint was aligned with the dynamometer's axis of rotation.

Surface electromyography signal (sEMG) was recorded using a wireless sEMG apparatus (MyoSystem, Noraxon Inc., Scottsdale, AZ, USA). Two self-adhesive Ag-AgCl electrodes 10 mm in diameter (Noraxon dual Elektrodes) were placed with a constant inter-electrode distance of 20 mm at a site corresponding to the distal one-third of the belly of the *vastus lateralis*. Prior to electrode attachment, the skin was prepared by shaving, abrading (Nuprep) and cleaning with an alcohol-based solution to ensure low skin impedance ( $< 5 \text{ k}\Omega$ ). The raw sEMG signal was sampled at 1000 Hz. All electrode positions were carefully measured in each participant to ensure identical pre- and post- recording sites.

Raw dynamometer signals (torque, angular velocity and range of motion) and sEMG signal were simultaneously collected at 1000 Hz with a powerLab 16/35 data acquisition system and later analysed with the Labchart Pro software package (version 8.1, ADInstruments, Bella Vista, NSW, Australia).

### **Exercise-induced muscle damage**

For each participant, the EIMD protocol consisted in repeated quadriceps *femoris* eccentric contraction in the dominant leg. More precisely, an individually tailored EIMD protocol was chosen given the wide inter-individual response to a standardized eccentric bout. Therefore, the primary criterion to assess EIMD was based on force-generating capacity

rather than a predefined number (sets) of eccentric contractions. A moderate reduction in  $T_{\text{peak}}$  was considered in the present study, i.e. a 15 to 25% loss in  $T_{\text{peak}}$  from baseline values. Briefly, all participants completed 7 sets of 10 maximal unilateral, isokinetic ( $30^{\circ} \cdot \text{s}^{-1}$ ), eccentric contractions with their quadriceps *femoris* muscle. At the completion of each eccentric contraction, the leg was passively returned to the starting position ( $90^{\circ}$ ), and another eccentric contraction was immediately initiated. Then, participants were asked to complete a single set of 3 maximal, isokinetic, concentric knee extension at  $60^{\circ} \cdot \text{s}^{-1}$ , with the best trial retained to assess reduction in  $T_{\text{peak}}$ . If strength loss criterion was not reached, 2 to 3 sets of 10 maximal eccentric contractions were proposed until strength loss  $> 15\%$ .

### Peak Torque

Two set of 3 maximal, concentric knee-extensions were performed at a slow isokinetic angular velocity ( $60^{\circ} \cdot \text{s}^{-1}$ ) to measure  $T_{\text{peak}}$ . The mean of the two highest  $T_{\text{peak}}$  values was retained for the analysis. Prior to analysis, a low-pass filter with a cut-off frequency of 10 Hz was applied to the raw torque signal. The intra-individual coefficient of variation between the two highest  $T_{\text{peak}}$  values was  $< 2\%$ . A decrease in  $T_{\text{peak}}$  was interpreted as an indicator of overall neuromuscular deficit, i.e. a combination of both central and peripheral factors.

### Rate of Force Development

Late-phase RFD was determined from the two concentric contractions that resulted in the highest quadriceps *femoris*  $T_{\text{peak}}$ . This calculation involved analysing the force vs. time relationship, specifically the slope ( $\Delta\text{force}/\Delta\text{time}$ ) of the linear function within the timeframe of 100 to 200 ms relative to the onset of contraction ( $\text{RFD}_{100-200}$ ).  $\text{RFD}_{100-200}$  was measured

twice, and the mean of the two RFD values was retained for the analysis. A reduced RFD<sub>100-200</sub> was interpreted as diminished peripheral factors, such as muscle contractile properties and muscle architecture. The onset of contraction (i.e. 0 ms) was identified when the shift in the direction of the lever arm coincided with the angular displacement starting to decrease.

### **Rate of Electromyography Rise (sEMG temporal analysis)**

sEMG signal was first smoothed using a moving root-mean-square filter with a time constant interval of 50 ms (RMS-sEMG). Subsequently, the rate of sEMG rise (REMGR) was calculated as the slope of the RMS-sEMG time curve analysed at time intervals of 0-30 (REMGR<sub>30</sub>), 0-50 (REMGR<sub>50</sub>) and 0-75 ms (REMGR<sub>75</sub>) relative to the onset of the RMS-sEMG signal. The onset was initiated 70 ms prior to muscle contraction onset to account for the presence of electromechanical delay. Each REMGR was determined from the two concentric contractions that resulted in the highest  $T_{peak}$ , and the mean of the two values was retained for the analysis. A decrease or increase in REMGR was interpreted as an indication of decreased or increased neural drive, respectively (40).

### **Spectral Power of the Low-frequency band (sEMG spectral analysis)**

Discrete wavelet transform was used to assess spectral changes throughout the protocol. The discrete wavelet analysis is a suitable approach especially when the signal under investigation is non-stationary, as in the present study (38). Specifically, a *rbio.3.1* 6-level wavelet analysis was used to characterize the spectral dynamics of the sEMG signal. The center frequencies and widths for each level were as follows: level 1:  $12 \pm 4$ ; level 2:  $24 \pm 8$ ; level 3:  $48 \pm 16$ ; level 4:  $94 \pm 30$ , level 5:  $187 \pm 62$  and level 6:  $350 \pm 100$  Hz. Subsequently, a

Fast Fourier Transform was performed to obtain the power spectral density of each level. The low-frequency band encompassed levels 1 and 2 while the high-frequency band was comprised of levels 5 and 6 (41). Finally, the power contained in the low-frequency band was expressed as a percentage of the overall power spectrum, which encompasses the sum of all levels. The calculation of the low-frequency band was determined from the two concentric contractions that resulted in the highest  $T_{\text{peak}}$ , and the mean of the two values was retained for the analysis. An increase in the power spectral density of the low-frequency band would suggest a state of neuromuscular fatigue (38). Discrete wavelet transform was performed using the MATLAB wavelet toolbox version 4.11 (R2013a, The MathWorks Inc., Natick, Massachusetts).

### **Pressure Pain Threshold**

Pressure pain threshold (PPT) was assessed by means of a hand-held digital algometer (FDX-50, Wagner Instruments, Greenwich, USA) to quantify the recovery from DOMS (42, 43). The participants adopted a seated position while remaining relaxed. The pressure ( $\text{N}\cdot\text{cm}^{-2}$ ) was applied gradually and perpendicularly to the mid-belly sites of the *vastus lateralis*. A 1- $\text{cm}^2$  hard rubber tip was used to apply the pressure. At each time point, 3 serial measurements were taken at three sites (spaced 3 cm apart) by a single trained examiner (BS) and subsequently averaged for further analysis. For each trial, the selected value corresponded to the threshold at which the participant deemed the pain to be unbearable.

### **Statistical analysis**

A prospective power analysis was used to calculate the required sample size (G\*Power

software version 3.1). In the present study, the primary outcome variable of interest was the change in RFD<sub>100-200</sub> in a two-way mixed-design ANOVA. The effect size ( $\omega^2 = 0.12$ ) from Sautillet and colleagues (2023) were used to calculate the sample size. To maintain a type I error rate of 0.05 while ensuring a statistical power of 0.80, 30 participants (n=10 per group) were required. This calculation ensured an 80% likelihood of detecting the expected effect size, given the actual level of significance, while also accounting for a 20% drop-out rate.

The samples were first tested for normality, homogeneity of variance and sphericity with Shapiro-Wilk's, Levene's and Mauchly's test, respectively. If Mauchly's test was significant, the degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity. A one-way repeated-measures ANOVA was conducted to track changes in gastrointestinal temperature throughout HWL<sub>41</sub>. For variables collected pre- and post-EIMD, two-way mixed ANOVA was conducted to investigate the main effect for condition (CON<sub>36</sub> vs. CWI<sub>11</sub> vs. HWL<sub>41</sub>), time (pre- vs. post-24h vs. post-48h) and their interaction (condition  $\times$  time). Multiple pairwise comparisons adjusted with the Holm correction were conducted whenever the main effect or interaction was significant. Pearson's correlations were performed to assess the relationship between  $\Delta T_{\text{peak}}$ ,  $\Delta \text{RFD}_{100-200}$  and  $\Delta \text{PPT}$ . Statistical analysis and graphs were performed using JASP (version 0.16.10, JASP team, 2022) and OriginPro software (version 2022, Origin-Lab), respectively. Statistical significance for all analyses was accepted at  $p < 0.05$ . Effect sizes associated with ANOVA (omega-squared,  $\omega^2$ ) and subsequent particular comparison (Cohen's  $d$ ) are reported in the results section. Threshold values for  $\omega^2$  were  $\geq 0.01$ , small;  $\geq 0.06$ , medium;  $\geq 0.14$ , large; for Cohen's  $d$ ,  $\geq 0.2$ , small;  $\geq 0.5$ , medium and  $\geq 0.8$ , large. Changes reported in the results section refer to

mean  $\pm$  SD.

## RESULTS

### EIMD-induced neuromuscular fatigue

Four participants, two from each of the CWI<sub>11</sub> and CON<sub>36</sub> groups, were excluded from analysis since they did not satisfy strength loss criterion (*i.e.* a drop in  $T_{peak} > 15\%$  post-EIMD) despite undergoing 100 eccentric contractions. Among the remaining participants ( $n=26$ ), EIMD induced a similar acute neuromuscular fatigue (*i.e.* drop in  $T_{peak}$  values) across the 3 conditions (CON<sub>36</sub>:  $-27.9 \pm 7.8\%$ ; CWI<sub>11</sub>:  $-21.2 \pm 6.2\%$ ; HWI<sub>41</sub>:  $-21.2 \pm 5.1\%$ ;  $p = 0.13$ ;  $\omega^2 = 0.009$ ) (figure 2A).

### Core body temperature during HWI

No clinical symptoms occurred either during or after immersion. By the end of HWI<sub>41</sub>, the participant's core body temperature exhibited a notable increase in comparison to pre-immersion values ( $38.82 \pm 0.13$  vs.  $37.47 \pm 0.24$  °C;  $p < 0.001$ ;  $d = 5.58$ ), reflecting a mean increase of  $+ 1.33$  °C ( $\pm 0.23$ ) from pre-immersion values. The duration of elevated core body temperature exceeding  $38.5$  °C was  $22.2$  ( $\pm 8.2$ ) minutes.

### Peak torque, late-phase rate of force development and pressure pain threshold

The mean changes in  $T_{peak}$ , RFD<sub>100-200</sub> and PPT in response to the three conditions are displayed in Figure 2A, B & C. Prior to EIMD, the average time to reach  $T_{peak}$  was  $534$  ( $\pm 71.1$ ) ms. A significant interaction (condition  $\times$  time) was observed for  $T_{peak}$  ( $F_{(3,66)} = 3.152$ ;  $p = 0.009$ ;  $\omega^2 = 0.023$ ). Specifically,  $T_{peak}$  was lower than baseline values at the post-24h time

point following both CON<sub>36</sub> ( $p < 0.001$ ,  $d = 1.193$ ) and HWL<sub>41</sub> ( $p = 0.026$ ,  $d = 0.726$ ). In contrast, following CWI<sub>11</sub>, there was no difference in  $T_{\text{peak}}$  between baseline and the post-24h time point ( $p = 1$ ,  $d = 0.452$ ). At the post-48h time point,  $T_{\text{peak}}$  remained lower compared to baseline following CON<sub>36</sub> ( $p < 0.001$ ;  $d = 1.129$ ), whereas  $T_{\text{peak}}$  returned to baseline following either CWI<sub>11</sub> ( $p = 0.079$ ,  $d = 0.728$ ) or HWL<sub>41</sub> ( $p = 1$ ,  $d = 0.124$ ).

A significant interaction was found for RFD<sub>100-200</sub> ( $F_{(2,44)} = 3.358$ ;  $p = 0.018$ ;  $\omega^2 = 0.036$ ). Specifically, at the post-24h time point, RFD<sub>100-200</sub> remained similar to baseline values following CON<sub>36</sub> ( $p = 1$ ,  $d = 0.583$ ), CWI<sub>11</sub> ( $p = 0.08$ ;  $d = 1.004$ ) and HWL<sub>41</sub> ( $p = 1$ ,  $d = 0.33$ ). However, while RFD<sub>100-200</sub> was decreased at the post-48h time point compared to baseline following both CWI<sub>11</sub> ( $p = 0.027$ ;  $d = 1.222$ ) and CON<sub>36</sub> ( $p = 0.040$ ,  $d = 0.981$ ), it returned to baseline following HWL<sub>41</sub> ( $p = 1$ ,  $d = 0.144$ ).

A significant interaction was found for PPT ( $F_{(4,44)} = 6.58$ ;  $p < 0.001$ ;  $\omega^2 = 0.079$ ). More precisely, PPT exhibited a reduction from baseline at the post-24h time point following both CON<sub>36</sub> ( $p = 0.027$ ;  $d = 0.962$ ) and CWI<sub>11</sub> ( $p = 0.005$   $d = 1.369$ ), whereas PPT was unaltered following HWL<sub>41</sub> ( $p = 0.819$ ;  $d = 0.531$ ). The decline in PPT persisted at the post-48h time point following CON<sub>36</sub> ( $p = 0.013$ ,  $d = 1.031$ ) and CWI<sub>11</sub> ( $p < 0.001$ ,  $d = 1.844$ ), whereas PPT remained unaltered following HWL<sub>41</sub> ( $p = 1$ ,  $d = 0.196$ ).

### Rate of Electromyography Rise

The mean changes in REMGR<sub>30</sub>, REMGR<sub>50</sub> and REMGR<sub>75</sub> in response to the three conditions are displayed in figure 3. Changes in REMGR<sub>30</sub> demonstrated a main effect for

condition ( $F_{(2,18)} = 5.951$ ;  $p = 0.010$ ;  $\omega^2 = 0.148$ ). More precisely, the participants from the HWI<sub>41</sub> condition had higher REMGR<sub>30</sub> than the CWI<sub>11</sub> condition regardless of the specific time point examined ( $p = 0.011$ ,  $d = 1.522$ ). Changes in REMGR<sub>50</sub> demonstrated a main effect for condition ( $F_{(2,18)} = 9.874$ ;  $p = 0.002$ ;  $\omega^2 = 0.340$ ). More precisely, the participants from the HWI<sub>41</sub> condition had higher REMGR<sub>50</sub> than the CWI<sub>11</sub> condition ( $p = 0.001$ ,  $d = 1.726$ ) and the CON<sub>36</sub> condition ( $p = 0.021$ ,  $d = 1.127$ ) regardless of the specific time point examined. Changes in REMGR<sub>75</sub> demonstrated a main effect for condition ( $F_{(2,36)} = 9.888$ ;  $p = 0.002$ ;  $\omega^2 = 0.270$ ). More precisely, the participants from the HWI<sub>41</sub> condition had higher REMGR<sub>50</sub> than the CWI<sub>11</sub> condition ( $p = 0.002$ ,  $d = 1.737$ ) and the CON<sub>36</sub> condition ( $p = 0.012$ ,  $d = 1.325$ ) regardless of the specific time point examined.

### **Normalized Low-frequency band of the electromyography signal**

The mean changes in the low-frequency band in response to the three conditions are displayed in Figure 4. A significant interaction was found in the low-frequency band ( $F_{(4,44)} = 10.049$ ;  $p < 0.001$ ;  $\omega^2 = 0.175$ ). More precisely, at the post-48h time point, there was an increase in the low-frequency band from baseline following both CON<sub>36</sub> ( $p = 0.034$ ,  $d = 1.287$ ) and CWI<sub>11</sub> ( $p = 0.012$ ,  $d = 1.434$ ), while no such increased was observed following HWI<sub>41</sub>.

### **Correlation between pressure pain threshold, peak torque, and late-phase rate of force development**

Bivariate correlations are displayed in figure 5. There was no significant correlation observed between the changes in PPT and  $T_{\text{peak}}$  (figure 5A), while a significant positive

correlation was found between the changes in PPT and RFD<sub>100-200</sub> (figure 5B).

## DISCUSSION

Adhering to the most recent recommendations for both cold and hot water immersion, the key findings of the present study, performed with physically active males, were as follows: (i) A single session of hot water immersion effectively mitigated the decline in the rate of force development and soreness after exercise-induced muscle damage, in contrast to both warm-bath control and cold water immersion; (ii) This impairment in rapid force production after cold water immersion or the warm-bath control was concomitant to an increase in low-frequencies band in the sEMG signal, while the rate of sEMG rise remained unaffected; and (iii) At the post-48h time point, a dissociation in the recovery patterns of maximal and explosive strength was apparent following cold water immersion.

The current findings emphasize that HWI<sub>41</sub> elicited an adequate physiological stimulus to counteract the reduction in explosive strength of the knee extensors, as supported by the return of RFD<sub>100-200</sub> to baseline values at the post-48h time point (figure 2B). Therefore, our results align with recent research indicating that heat can effectively facilitate the recovery of explosive strength after EIMD (28). Maximal force, denoted as  $T_{peak}$ , is commonly viewed as the primary indicator for assessing the kinetics of neuromuscular recovery subsequent to EIMD (44). However, recent evidence suggest that this evaluation may be incomplete, and RFD should also be considered for a more comprehensive evaluation (31, 45). Although not directly assessed, it is anticipated that the present eccentric regimen led to structural damage to the myofibers, with a specific emphasis on fast-twitch fibers more prone to damage than

slow-twitch fibers (46). Consequently, eccentric exercise is expected to particularly alter the late stage of RFD (*e.g.* within 100-200 ms) due to a greater recruitment inertia of fast-twitch compared to slow-twitch fibers (47). Consistent with this hypothesis, the study by Peñailillo and colleagues demonstrated that  $RFD_{100-200}$  assessed on the same muscle group (quadriceps *femoris*) was more sensitive to EIMD than  $T_{peak}$ , as well as any other RFD calculated from the force vs. time curve (33). It is worth noting that regardless of the immersion condition, EIMD did not seem to have impacted the neural drive, as quantified with REMGR (figure 3). Since only  $HWI_{41}$  was effective in mitigating the decline in  $RFD_{100-200}$ , our results indirectly suggest that  $HWI_{41}$ , rather than  $CWI_{11}$ , yielded favourable outcomes in enhancing muscle repair (*i.e.* intrinsic muscular factors) following EIMD.

The mechanisms by which heat attenuated the decline in explosive strength likely involved the expression of heat shock proteins (HSP). Senf and colleagues demonstrated that extracellular HSP(70) had a significant role in muscle repair and fiber adaptation by restoring the recruitment of muscle cells involved in the inflammatory response (48). In the present study, the heat protocol succeeded in maintaining the core body temperature of each participant above 38.5°C (mean peak value: 38.8 °C), a pivotal threshold required for the upregulation of extracellular HSP (49). Notably, the study by Sautillet and colleagues demonstrated that maintaining a core body temperature above this threshold also correlates with accelerated neuromuscular recovery following EIMD, while an increase close to, albeit lower than 38.5°C, had no significant effect (28). Previous studies examining the effects of HWI have merely proposed immersion durations and temperatures that are not associated with sufficient endogenous thermal stress on core/muscle temperature, which could thus

explain their inconclusive findings on neuromuscular recovery (26, 27).

Extensive intra- (skeletal muscle fiber) and extra- (fascial connective tissue) muscular damage resulting from repeated eccentric contractions initiates the activation of nociceptor pathways, subsequently leading to the development of DOMS (46). Interestingly, PTT was reduced following CWI<sub>11</sub> and CON<sub>36</sub>, indicating increased pressure pain sensitivity, whereas this remained unchanged following HWI<sub>41</sub> (Figure 2C). Thus, the current findings indicate that immersion at 41°C reduced the severity of DOMS, whereas neither CWI<sub>11</sub> nor CON<sub>36</sub> exhibited similar alleviating effects. These results confirm the analgesic properties of HWI on muscle soreness (50, 51), potentially mediated by heat-induced increases in blood flow/metabolism, HSP upregulation, metabolite alteration, and/or desensitization of peripheral pain nociceptor pathways (*e.g.* TRPV channels) (52-55). With regard to cold exposure, our results contradict current meta-analyses, which found a decline in DOMS beginning from 48 hours after EIMD (19). These discrepancies could be attributed to the methodology used to assess DOMS. Specifically, previous studies predominantly relied on an overall perception of pain intensity, typically quantified using subjective visual analogue scales (56). In contrast, the present findings were based on the stimulation of high-threshold mechanosensitive receptors, quantified through PPT (42). Interestingly, a small yet significant positive correlation was found between the changes in PPT and RFD<sub>100-200</sub> (figure 5B), indicating that the greatest decrease in PPT corresponded with the highest decrease in RFD<sub>100-200</sub>. In line with this observation, Fleckenstein and colleagues demonstrated that PPT, rather than pain intensity (quantified using a visual analogue scale), was correlated to neuromuscular dysfunction. Taken together, PTT could offer a more sensitive marker of soreness compared

to the visual analogue scale during the post-EIMD recovery (42). Moreover, PTT is regarded as a semi-objective measure of pain, unlike the visual analogue scale, more prone to subjective interpretation (43). Considering the prevalent belief in the benefits of CWI among athletes (12), relying solely on subjective measures could bias pain perception due to a placebo effect, as previously demonstrated (20).

CWI<sub>11</sub> succeeded in mitigating the decline in maximal force production, since  $T_{peak}$  returned to baseline values at both the 24 h and 48 h time points compared to CON<sub>36</sub> (figure 2A). However, at the 48 h time point, RFD<sub>100-200</sub> remained depressed, thus demonstrating a clear uncoupling between maximal and explosive strength recovery kinetics following CWI<sub>11</sub> (Figure 2A & B). Interestingly, we noted that low-frequency band derived from sEMG *vastus lateralis* signal was increased after CWI<sub>11</sub>, a trend which was not observed after HWI<sub>41</sub> (Figure 4). In response to accumulated neuromuscular fatigue, the spectral power profile of the *Vastus lateralis* shifts towards a clear dominance of lower frequencies (37). Evidence suggests that the low frequencies may be partly associated with motor unit synchronization (36). Considering that alterations in motor unit synchronization may play a role in peak force production (57), one may suggest that CWI<sub>11</sub> contributed to maintain peak force output – but not explosive strength – through compensatory neural mechanisms, potentially including an elevation in motor unit synchronization.

The ability to produce force rapidly is considered a primary physical requirement, particularly in situations where the time to generate force is limited (< 250-300 ms), such as in sprinting, jumping (58) or stabilizing joints to prevent injury (59). In our participants, the

mean time taken to achieve peak force output by the knee extensors was 534 ms, consistent with findings from prior reports (40). This indicates that RFD serves as a more appropriate proxy for neuromuscular recovery than  $T_{\text{peak}}$  in situations involving rapid athletic movements (45). Consequently, the return of  $T_{\text{peak}}$  back to baseline values following CWI should not be systematically viewed as a positive outcome, as commonly assumed. Instead, it should be interpreted by considering the athletic context in which the recovery takes place.

### Limitations

The primary limitation of the current study lies in the utilization of two fundamentally different approaches to execute the HWI and CWI protocols. Specifically, the thermal dose of HWI relied on endogenous criteria, which involved maintaining a target internal temperature for a specific duration, whereas the CWI dose was guided by exogenous criteria, namely a predetermined water temperature (11°C) and immersion duration (11 minutes), albeit consistent with current practices. During CWI, the rate of convective heat loss from the muscles to the skin is influenced by anthropometric characteristics, with adipose tissue thickness being a critical determinant (60). As subcutaneous body fat increases, the magnitude of reduction in core body temperature (61) and intramuscular temperature (60) decreases. Consequently, in the present study, it is likely that CWI induced disparate tissue cooling across individuals compared to HWI. As achieving a minimum threshold of muscle/core body temperature is likely paramount for triggering the expected physiological changes (*e.g.* reduced inflammation) (62), the variability in tissue cooling in response to CWI may have obscured the potential benefits of the cold stressor on neuromuscular recovery. However, the degree to which reduction in muscle temperature is necessary to optimally accelerate recovery

in humans remains unknown (14), thus explaining the reliance of current guidelines on external dosing criteria (19).

## CONCLUSIONS

In healthy physically active males, a single session of HWI<sub>41</sub> applied immediately following a mild EIMD provided the required physiological stimulus to accelerate the recovery of explosive strength (i.e., RFD<sub>100-200</sub>). In contrast, CWI<sub>11</sub> only mitigated the loss of maximal strength (i.e., T<sub>peak</sub>), thereby questioning the relevance of the cold stressor in athletic scenarios where the ability to rapidly generate force is a key performance factor.

## Perspectives

From a practical standpoint, this study underscores the effectiveness of waist-level HWI for athletes seeking quick recovery of explosive strength between bouts of damaging exercise. However, in some participants, the time required to raise body (and muscle) temperature to levels relevant for recovery purposes could approach 60 minutes, a duration that may conflict with the inherent time constraints frequently reported by athletes (13). In contrast, local heat therapy methods such as microwave diathermy have demonstrated substantial increases in muscle temperature within just 20 minutes (63). Whether these methods, which specifically target muscle temperature, are also relevant for accelerating neuromuscular recovery remains to be determined.

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## FIGURE LEGENDS

**Figure 1:** The parallel group, randomized controlled trial depicting the timeline of the experiment and the variables under investigation. Assessments were conducted on the damaged leg prior to (pre-), at post-24h and at post-48h following single-leg exercise-induced muscle damage (EIMD). Assessments included (i) measuring peak torque ( $T_{\text{peak}}$ ) and late-phase rate of force development ( $\text{RFD}_{100-200}$ ) of the knee extensors, (ii) measuring pressure pain threshold of the vastus *lateralis*, (iii) conducting a temporal analysis of surface electromyographic (sEMG) signals from the vastus *lateralis* to calculate the rate of electromyography rise (REMGR), and (iv) examining the low-frequency band from the vastus *lateralis* by means of wavelet spectral analysis.

**Figure 2:** Individual data points and mean ( $\pm$  SD) for: peak torque ( $T_{\text{peak}}$ , **panel A**), late-phase rate of force development ( $\text{RFD}_{100-200}$ , **panel B**) and pressure pain threshold (PPT, **panel C**) in response to warm-bath control (36°C), cold water immersion (11°C) or hot water immersion (41°C). In panel A, note that the acute phase refers to the acute neuromuscular fatigue (i.e. drop in  $T_{\text{peak}}$ ) immediately after exercise-induced muscle damage.

\* $p < 0.05$  indicates a significant difference from pre-.

**Figure 3:** Mean changes in the rate of electromyography rise (30, 50 and 75 ms) in response to warm-bath control (36°C), cold water immersion (11°C) or hot water immersion (41°C). Error bars are intentionally omitted to improve visual clarity. Note that no significant difference was observed regardless of the time point, or condition considered.

**Figure 4:** Individual data points and mean changes ( $\pm$  SD) in the low-frequency band, expressed as a percentage of the overall power spectrum, in response to warm-bath control (36°C), cold water immersion (11°C) or hot water immersion (41°C).

**Figure 5:** Bivariate correlation between  $\Delta$ PTT and  $\Delta T_{\text{peak}}$  (**panel A**) and  $\Delta$ RFD<sub>100-200</sub> (**panel B**).

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Figure 1

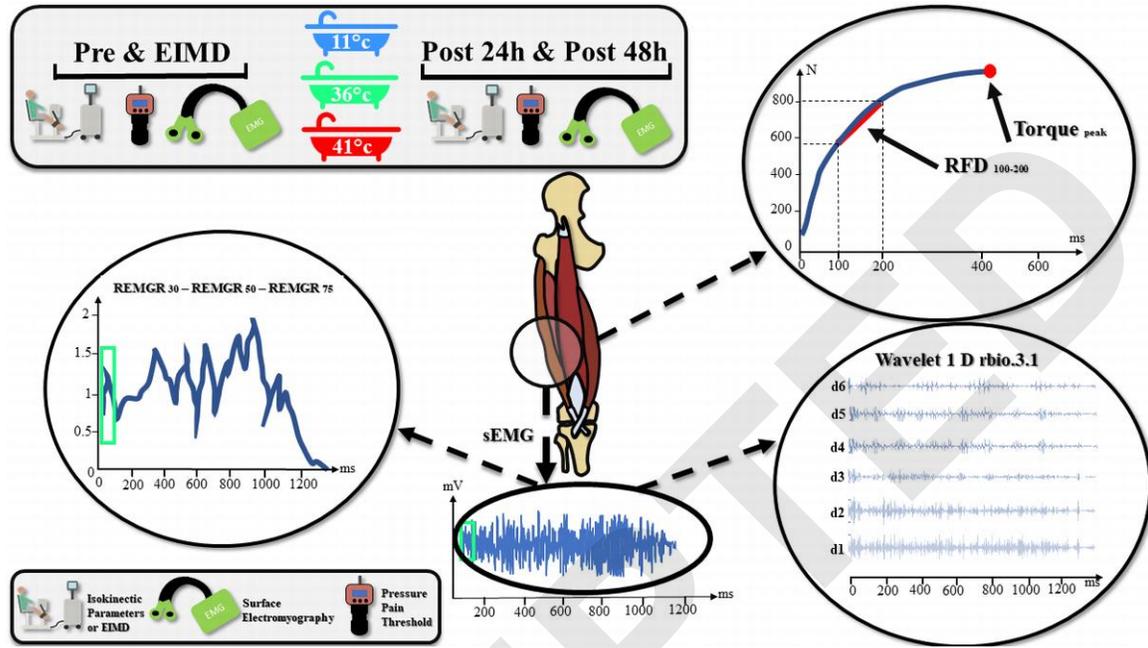


Figure 2

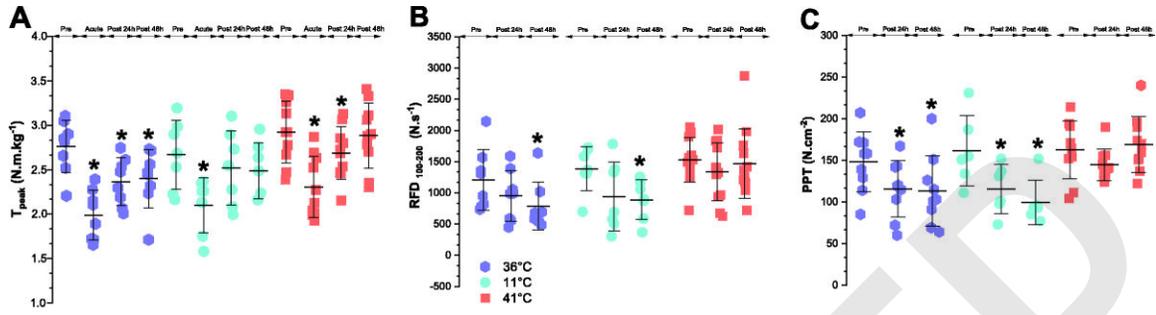


Figure 3

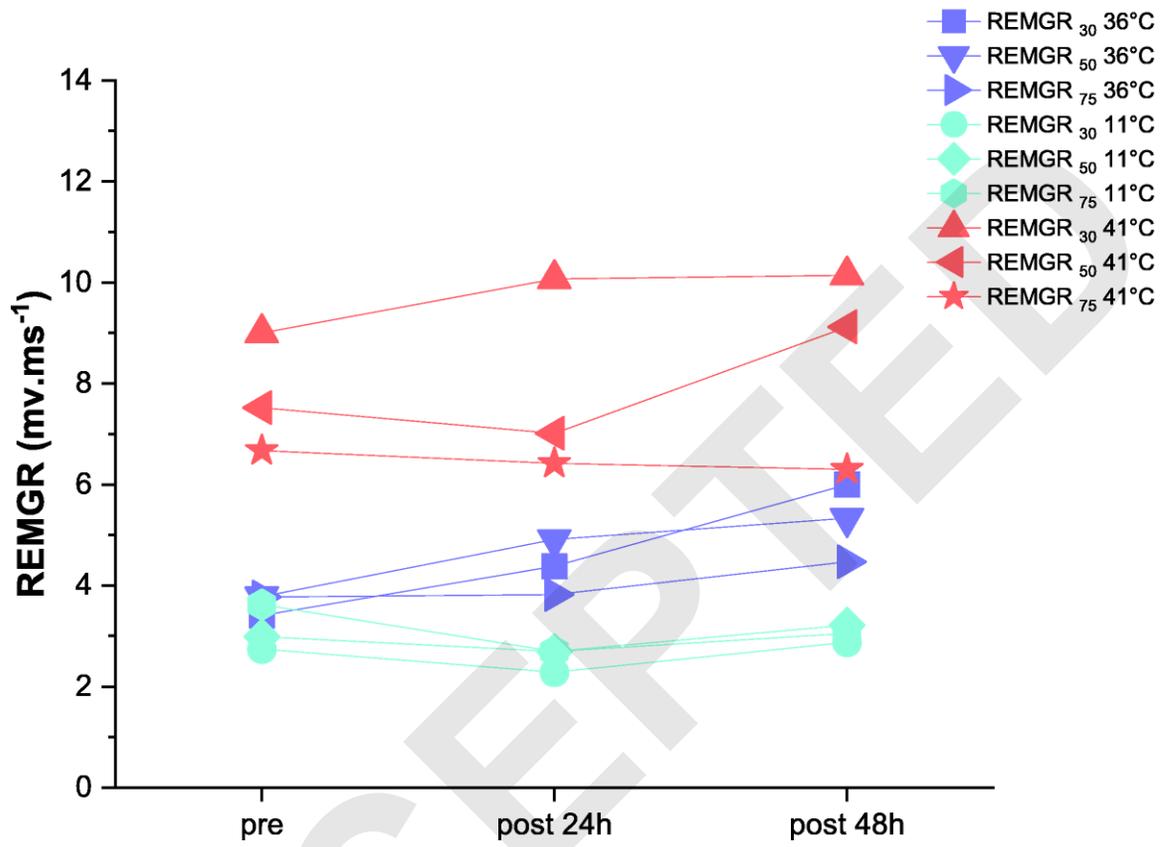


Figure 4

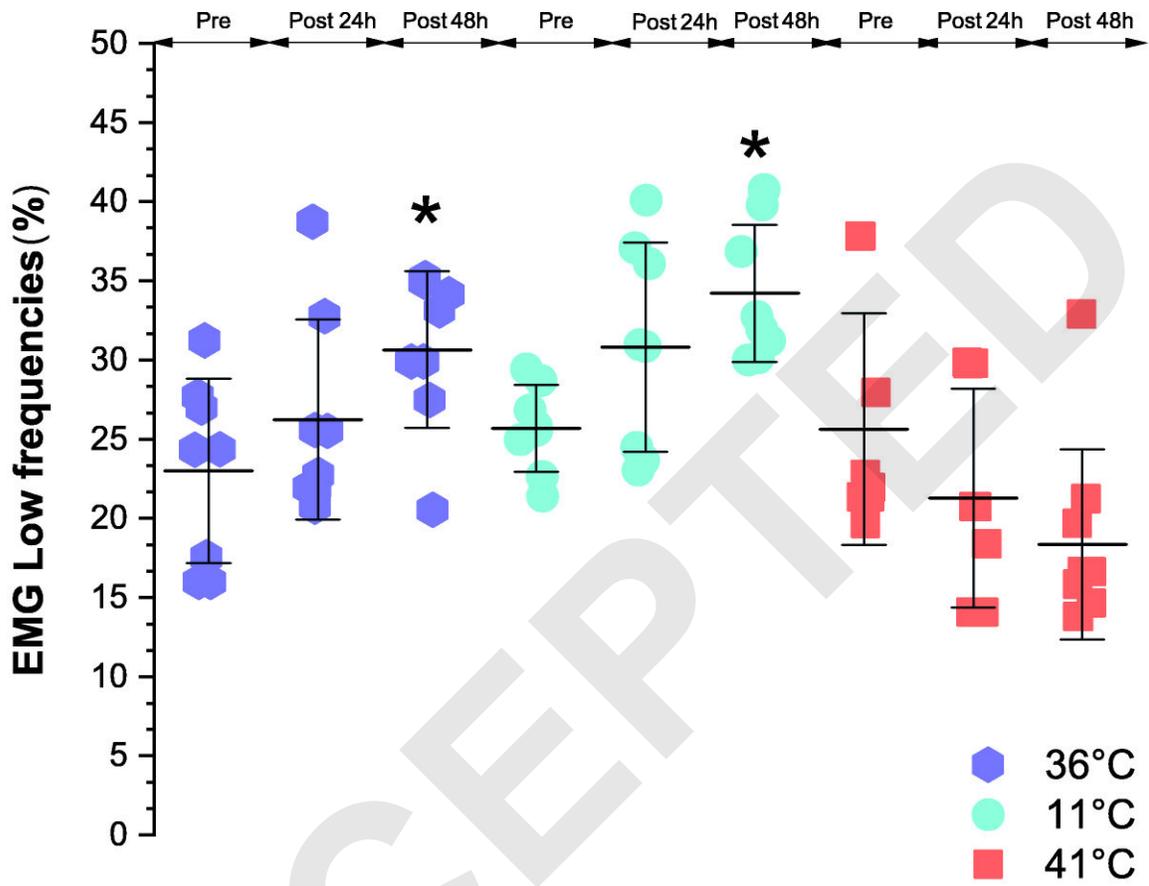


Figure 5

