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Original research

A passive increase in muscle temperature enhances rapid force production and neuromuscular function in healthy adults

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ABSTRACT

Objective: To test the effects of hot-water immersion on the rapid force production and parameters of neuromuscular function in healthy adults.
Design: Cross-sectional study.
Methods: Fifteen healthy adults (24.9 ± 5.6 years; 178 ± 11.4 cm; 72.8 ± 16.2 kg) performed neuromuscular assessments before, after and ~15 min after either 90 min of 42 °C (hot) or 36 °C (sham-condition) water immersion (lower body). Knee extensors rate of torque development (RTD) was measured during explosive voluntary contraction in the interval of 0–50 ms (RTD_{V50}) and 0–150 ms (RTD_{V150}) and during electrically-evoked contractions by single twitches (RTD_{twitch}) and low- and high-frequencies doublets (RTD_{20Hz} and $_{100Hz}$). Rate of EMG rise (RER) was calculated for voluntary contractions and half-relaxation time (HRT) and electromechanical delay (EMD) was measured during single twitches.
Results: After the hot-water immersion (when rectal and muscle temperature were elevated [↑1 °C and ↑2.4 °C, respectively]), RTD_{V50}, RTD_{20Hz} and RTD_{100Hz} significantly increased and HRT decreased when compared to baseline and sham-condition ($p < 0.05$). Approximately 15 min after the hot-water immersion (when muscle temperature was still higher [↑1.4 °C], but rectal temperature at baseline level), RTD_{V50} remained higher and RTD_{twitch} presented higher values than baseline and sham-condition. The RTD_{20Hz} and RTD_{100Hz} showed further increases compared to post hot-water immersion trials. HRT showed no changes compared to post water immersion, but the EMD presented lower values than baseline and sham-condition. No changes were observed for RTD_{V150} and RER at any moment.
Conclusion: Increased muscle temperature provoked by 42 °C hot-water immersion increases the early phase of the RTD (<70 ms) (voluntary and evoked) and decreases HRT and EMD of the knee extensors.

Practical implications

- Skeletal muscle passive heating increases the rapid force production (voluntary and electrically-evoked contractions) and enhances neuromuscular function by altering specific neuromuscular mechanisms (*i.e.*, increase RTD and decrease muscle half-relaxation time and electromechanical delay).
- The neuromuscular improvements evoked by passive heating in this study may be applied for sports performance and help during the sports injury recovery.
- Hot-water immersion for 90 min of 42 °C may acutely improve physical function requiring rapid contraction force, including falls prevention in older persons and patient populations.

1. Introduction

Passive increases in muscle temperature (T_{mu}) can enhance voluntary muscle contractility, strength and power[1,2] and concomitantly electrically-evoked muscle twitch response (*i.e.*, reduced

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time to peak torque and half-relaxation time).[3] These favourable effects may be explained by temperature regulated Ca^{2+} influx in the muscle cells[4] and increased intracellular fluid that improves Ca^{2+} sensitivity.[5] Contrastingly; however, any parallel increase in core temperature (T_c) may impair neural drive transmission during maximal force efforts and voluntary activation (as measured by the superimposed twitch method),[6,7] masking the potential effect of the muscle contractile responses during voluntary contractions. These decrements in force have been associated with declines in the central neural drive[8] or partly linked to peripheral neural transmission failures to the working muscles.[9] While the detrimental effects of overall heat strain are apparent, the independent effects of T_{mu} and T_c on neuromuscular responses remain ambiguous. Understanding site-specific influences seem critical to inform potentially therapeutic passive heating interventions.

Although previous studies have explored the effects of passive heating on maximal force production,[6,7] less attention has been given to explosive (rapid) contractions. Compared to maximal strength, rapid force production may be a more relevant measure of neuromuscular function when time available to produce force is limited[10,11] and is associated with functional daily tasks.[12,13] In particular, the early phase of the rate of force (or torque) development (RTD) (<75$_{ms}$) appears especially relevant considering its greater contribution to the total kinetic impulse produced in a time-restricted movement (*e.g.*, when reversing a fall).[14] Given the context that T_{mu} increases can increase muscle power[1,2] intracellular fluid and Ca^{2+} sensitivity[5], it is reasonable to suggest that a passive increase in T_{mu} is likely to increase the RTD. However, to the best of our knowledge, this effect is yet to be tested. The possible increase of Ca^{2+} influx and muscle blood flow caused by passive heating could increase fibre rotation during contraction, increasing muscle shortening velocity and force for a given muscle shortening distance.[5] The force-velocity properties during contraction may also be optimised by fluid (water) dependent increases in stiffness.[15]

Passive heating seems promising in improving rapid force capacity. However, while isolated increases in T_{mu} might increase muscle contractility[1,2], decreased neural drive transmission owing to a systemic elevated T_c could nullify this positive effect.[6,7] Accordingly, this study aimed to test the effect of passive heating on the RTD and other parameters of neuromuscular function responses during explosive and electrically-evoked contractions (*i.e.*, rate of EMG rise, half-relaxation time, and electromechanical delay). To better understand this effect, the neuromuscular assessments were completed after the passive heating in two arrangements: firstly, with a concomitant increase of T_c and T_{mu}, and subsequently, with a cooled T_c but increased T_{mu}. It was hypothesised that the elevated T_c would conceal the rapid contraction force response after heat exposure. However, once T_c have returned to baseline level and T_{mu} remains elevated, the RTD and the rate of EMG rise will increase, and half-relaxation time and electromechanical delay will decrease.

2. Methods

Fifteen healthy adults (9 males, 6 females) volunteered for this study (mean ± SD: 24.9 ± 5.6 years; 178.2 ± 11.4 cm; 72.8 ± 16.2 kg; 17.1 ± 5.4% body fat). Exclusion criteria included: (i) medical conditions contraindicating heat stress (*e.g.*, multiple sclerosis, spinal cord injuries, heart and respiratory diseases); (ii) musculoskeletal injury; (iii) thermoregulation altering medication; and (iv) pregnancy. The experimental procedures were approved by the University Human Research Ethics Committee (Reference: 1,800,000,977).

Participants visited the laboratory three times; initially, for familiarisation and subsequently for two experimental trials seven days apart. The randomised, single-blind experimental design involved neuromuscular function testing before and after either hot-water (42 °C) or thermoneutral-water immersion (36 °C, sham-condition). Participants did not know the real water temperature in either visit. Moreover, they did not know that the thermoneutral water (36 °C, sham-condition) was used for control. They received standardised information that the study aimed to compare the effects of two different warm-water immersion on neuromuscular function. All participants avoided vigorous exercise and alcohol consumption for 24 h and caffeine for 12 h before any testing session.

Neuromuscular assessments were undertaken before (Pre) and after water immersion (Post-1), and once the T_c returned to baseline (≤0.2 °C; Post-2) (15.7 ± 4.4 min after Post-1). Hastened reductions in T_c were achieved after Post-1 via the consumption of refrigerated water (~5 °C) ad libitum (11.0 ± 4.3 mL kg^{-1}).[16] Localised heating pads (EP5000. Sunbeam Corporation, Sydney, Australia) were positioned on the thigh to maintain the T_{mu} for the Post-2 neuromuscular test. The Post-1 and Post-2 time-points were interspersed by a standardised 15-min period during the sham-condition.

All testing procedures commenced with adjustment of the stimulation intensity required to induce maximal twitch torque (see electrically-evoked twitch). Next, a resting evoked single twitch was delivered followed by two doublets (one low- (20_{Hz}) and one high-frequency (100_{Hz})) in randomised order, with a 30-s interval between evoked twitches. Three maximal voluntary rapid contraction forces were then performed, with participants instructed to contract 'as fast and hard as possible' while strong verbal encouragement was provided (90-s rest between contractions). The highest value of the three voluntary trials was used in the analysis. Peak torque (PT) data in this study were used only with the purpose of voluntary RTD (50 and 150 ms) normalisation (RTD/PT ratio). Visual feedback was provided during each trial to avoid pretension before the evoked twitches, and knee flexion before the voluntary contractions.

The 90-min water immersion occurred in an inflatable bath (iBody, iCoolsport, Miami, Australia) connected to an external generator/pump (Dual Temp Unit, iCoolsport, Miami, Australia). Participants were submerged to the waist in 42.0 ± 0.2 °C (hot)[17] and 36.0 ± 0.1 °C (sham-condition)[18] water in a room where environmental conditions were 24 ± 1 °C and 55% relative humidity. Muscle temperature was measured immediately before each neuromuscular test (Pre, Post-1 and Post-2) (Appendix A, Fig A1) using a needle thermo-sensor (26-gauge, 4-cm length) (MT-26/4 H T, Physitemp, Clifton, USA) connected to a digital thermometer (Thermalert TH-5, Physitemp, Clifton, USA). The needle was inserted in the *vastus lateralis* midway between, and lateral to, a line joining the anterior superior iliac spine and patella at two depths estimated by ultrasound imaging: superficial (once the needle breached the muscle fascia: ~0.5 cm for males and ~1 cm for females, and deep (~3.5 cm below the skin).[17] Rectal temperature (T_{re}) was measured using a flexible thermistor (YSI, 400 DeRoyal, Knox, USA) inserted to the depth of ~12 cm beyond the rectal sphincter.

Knee extensor torque was measured using an isokinetic dynamometer (Biodex Medical Systems 3, Shirley, New York, USA). Participants were seated in an upright position with the backrest of the chair adjusted to support the lower back fully. They were then secured into place via straps across the chest and hips and instructed to cross their arms across the chest. The fulcrum of the dynamometer lever was aligned with the right lateral epicondyle (knee). The lower leg was attached to the lever arm via a velcro strap. Isometric contractions were performed at 70º of knee flexion (0º= full extension). Torque and surface electromyography (sEMG)

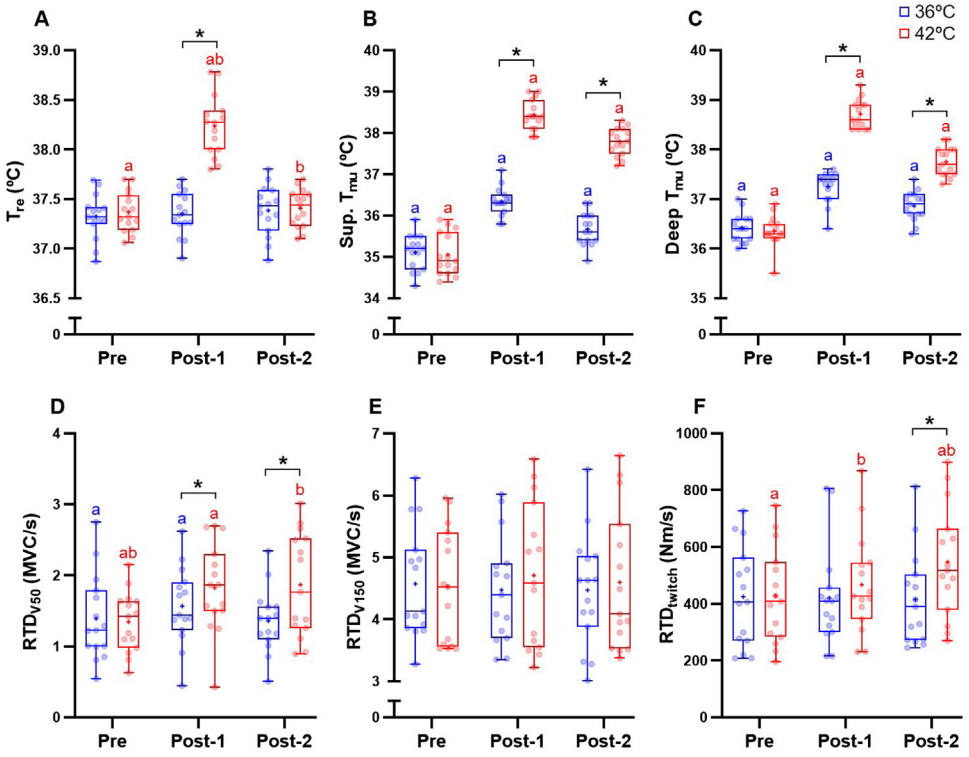

Fig. 1. Rectal temperature (A), superficial (B) and deep muscle temperature (C), normalised voluntary rate of torque development (RTD) at 50 ms (RTD_{V50}) (D) and 150 ms (RTD_{V150}) (E), RTD during single twitch (RTD_{twitch}) (F).
Data are presented as box and whiskers plots with individual values (light blue and red dots). Same letter means significant difference between time-points within water immersion treatments (42 °C or 36 °C). *statistical difference between water immersion treatments (42 °C vs. 36 °C). $p < 0.05$.

signals were simultaneously recorded at 2000_{Hz} (16-bit PowerLab 26T; AD Instruments, Sydney, Australia) and processed using LabChart 8.0 (AD Instruments, Sydney, Australia). The torque signal was filtered (low-pass: 500_{Hz}) and gravity-corrected before data were extracted. The torque onset of the rapid contraction forces (voluntary and involuntary) was visually identified when the torque increased above baseline using the unfiltered signal.[19]

The electrically-evoked twitch was assessed using a constant-current stimulator (DS7AH; Digitimer Ltd., Welwyn Garden City, England) by a single square-wave pulse ($200_{\mu s}$ width). Two self-adhesive surface gel electrodes were used; a cathode (3.2-cm diameter; Pals, Axelgaard Manufacturing Co. Ltd., Fallbrook, USA) was placed and pressed over the femoral nerve (femoral triangle) by a rubber ball and the dynamometer's hips strap, and an anode (5 × 9 cm; Pals, Axelgaard Manufacturing Co. Ltd., Fallbrook, USA) was placed under the gluteal fold. Maximal resting twitch was determined by delivering a series of single stimuli of increasing intensity until a plateau in action potential (M-wave) amplitude and peak twitch torque were concurrently obtained. The final current was increased by 20% to ensure supramaximal stimulation. Using the same intensity, doublets (two pulses) were elicited at low- (20_{Hz}) and high-frequencies (100_{Hz}) and the $20{:}100_{Hz}$ ratio was calculated to estimate changes in myoplasmic Ca^{2+} concentration.[20] Rate of torque development was calculated as the average change in torque per time interval from torque onset to 50 ms during single evoked twitch (RTD_{twitch}) and 0 to 70 ms during doublets for low- (RTD_{20Hz}) and high-frequencies (RTD_{100Hz}). Voluntary RTD was measured from torque onset to 50 and 150 ms (RTD_{V50}, RTD_{V150}) and normalised to peak torque.

The half-relaxation time was considered to be the time taken for the torque of the resting single twitch decreased by 50% its peak torque amplitude. The electromechanical delay (EMD) was measured during the single twitches, defined as the time elapsed between the start of M-wave (*vastus lateralis* and *vastus medialis*: VL and VM) and torque onset.

The sEMG data were recorded during the twitches and voluntary contractions. Skin preparation involved shaving, exfoliation, and cleaning with 70% ethanol. Bipolar electrodes (30 × 22 mm; N-00-S; Ambu A/S, Ballerup, Denmark) were placed over the *vastus lateralis* and *vastus medialis* following the expected muscle fibre direction and marked with an indelible ink pen to ensure matched placement between trials. After being amplified (2000x), the EMG signal received a bandpass filter ($10\text{-}1000_{Hz}$) and the root means square was calculated. The rate of EMG rise was calculated as the average increase in the EMG amplitude per time interval from the EMG onset to 50 ms during the voluntary contraction. The EMG onset was defined as the first point where EMG amplitude exceeded 0.01_{mV} from baseline. The rate of EMG rises of *vastus lateralis* and *vastus medialis* were summed and considered as an indicator of vastii muscle excitation.

Data are presented as box and whiskers plots (Figs. 1 and 2) and mean±95% confidence interval (CI) (Table1). Mauchly's test was used for data sphericity and Greenhouse-Geisser correction was adopted when appropriate. Levene's test was used for equality of error variances and Shapiro-Wilk test for data distribution. The comparison between treatments was completed using a two-way repeated measure ANOVA (time vs. water immersion treatment (36 °C vs. 42 °C)) and Bonferroni *post hoc* testing on SPSS 25.0 (IBM, Chicago, USA). Repeated-measures Bland-Altman within-subject correlation coefficients were computed using RStudio (Version 1.3.1056) using rmcorr package to determine the relationship between T_{mu} (superficial and deep) and RTD_{V50}.[21] Statistical significance was accepted when $p \leq 0.05$.

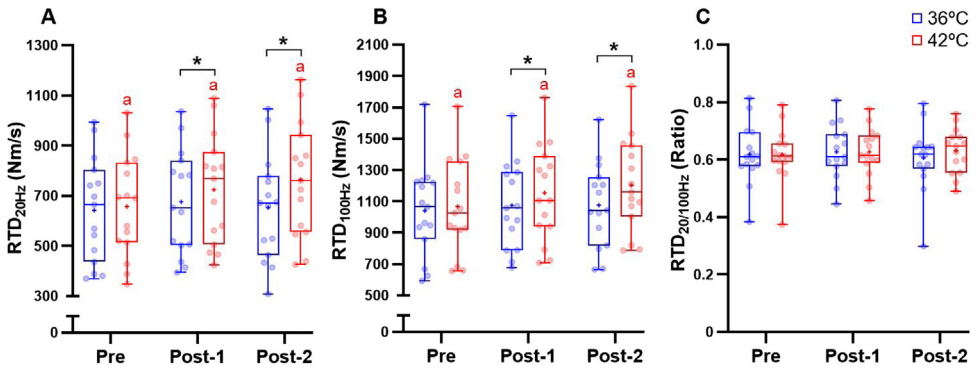

Fig. 2. RTD during tetanic contraction by 20 Hz (RTD_{20Hz}) (A) and 100 Hz (RTD_{100Hz}) (B), and RTD 20:100 Hz ratio ($RTD_{20/100Hz}$) (C).
Data are presented as box and whiskers plots with individual values (light blue and red dots). Same letter means significant difference between time-points within water immersion treatments (42 °C or 36 °C). *statistical difference between water immersion treatments (42 °C vs. 36 °C). $p < 0.05$.

Table 1
Neuromuscular parameters responses before (Pre), after (Post-1) and ~15 min after (Post-2) the water immersion treatments.

	Hot-water (42 °C)			Sham-condition (36 °C)		
Parameter	Pre	Post-1	Post-2	Pre	Post-1	Post-2
RER (mV)	3.49 ± 0.65	3.92 ± 0.64	4.15 ± 0.88	3.75 ± 0.69	3.89 ± 0.66	3.65 ± 0.78
HRT (ms)	71.24 ± 5.59 ^{ab}	62.44 ± 7.14 ^{a}	65.36 ± 6.14 ^{b}	69.68 ± 6.17	69.67 ± 6.01*	71.70 ± 6.47 *
EMD-VL (ms)	15.82 ± 1.56 ^{a}	13.87 ± 1.22 ^{a}	12.87 ± 1.25 ^{a}	15.13 ± 1.46	14.33 ± 1.42	14.93 ± 1.73 *
EMD-VM (ms)	15.80 ± 1.61 ^{a}	13.97 ± 1.43 ^{a}	12.94 ± 1.28 ^{a}	15.32 ± 1.45	14.23 ± 1.37	15.28 ± 1.54 *

Abbreviations: EMD, electromechanical delay; HRT, half-relaxation time; RER, rate of EMG rise; VL, vastus lateralis; VM, vastus medialis. Data are presented as mean ± 95%CI. Same letter means significant difference between time-points within water immersion treatments (42 °C or 36 °C). *statistical difference between water immersion treatments (42 °C vs. 36 °C). $p < 0.05$.

3. Results

The thermoregulatory responses measured before and after the water immersion treatments showed significant interaction for T_{re} ($p < 0.001$), superficial T_{mu} ($p < 0.001$), and deep T_{mu} ($p < 0.001$). *Post hoc* analysis within and between water immersion treatments are displayed in Fig. 1. Thermoregulatory responses (*i.e.*, rectal and skin temperature, thermal comfort and sensation, heart rate and blood pressure) during the 90-min water immersion (42 °C vs. 36 °C) are shown in Appendix A.

Significant interaction (time vs. water immersion condition) was observed for RTD_{V50} ($p = 0.01$), RTD_{twitch} ($p = 0.001$), RTD_{20Hz} ($p < 0.001$), RTD_{100Hz} ($p = 0.005$), HRT ($p = 0.03$) and EMD (VL and VM, $p = 0.002$). However, no significant interaction was observed for RTD_{V150} ($p = 0.26$) (Fig. 1E), $RTD_{20/100Hz}$ ratio ($p = 0.36$) (Fig. 2C), and RER ($p = 0.25$) (Table 1). *Post hoc* analyses are presented in Table 1 and Figs. 1 and 2. ANOVA results (F values, degrees of freedom and η_p^2) are reported in Appendix B.

After the passive heating (Post-1), RTD_{V50}, RTD_{20Hz}, and RTD_{100Hz} increased compared to pre ($p = 0.002$; $p = 0.001$; $p < 0.001$) and to sham-condition ($p = 0.04$; $p = 0.003$; $p = 0.001$, respectively). Half-relaxation time decreased compared to pre and sham-condition (pre, $p = 0.006$; sham, $p = 0.02$), and EMD decreased when compared to pre (VL, $p = 0.02$; VM, $p = 0.03$), but was similar to sham-condition ($p > 0.05$). RTD_{V50} also increased after sham-condition (pre, $p = 0.05$).

Fifteen minutes after the passive heating (Post-2), RTD_{V50} remained higher than pre and sham-condition (pre, $p = 0.006$; sham, $p = 0.007$), RTD_{twitch} increased compared to pre ($p = 0.01$) and sham-condition ($p < 0.001$) while RTD_{20Hz} and RTD_{100Hz} increased compared to Post-1 ($p = 0.003$; $p = 0.03$; respectively). The EMD reduced compared to Post-1 (VL, $p = 0.02$; VM, $p = 0.03$) and presented lower values than sham-condition (VL and VM, $p < 0.001$). RTD_{V50} after sham-condition did not show any difference ($p > 0.05$).

A large repeated measures correlation was observed between superficial T_{mu} and RTD_{V50} across time-points for hot-water immersion ($r = 0.68$, $p < 0.001$), but not for sham-condition ($p = 0.18$). However, there was a large correlation between deep T_{mu} and RTD_{V50} for hot-water immersion ($r = 0.60$, $p < 0.001$) and a moderate correlation for sham-condition ($r = 0.38$, $p = 0.04$) (Plots are shown in Appendix B).

4. Discussion

This study aimed to investigate the effects of a passive increase in T_{mu} by hot-water immersion on RTD (voluntary and electrically-evoked), rate of EMG rise, twitch torque half-relaxation time, and electromechanical delay. Although previous studies have reported impaired maximal force production aligned with moderate (T_c 38.5 °C)[7] and severe (T_c 39.4 °C)[6] hyperthermia, the present data suggest that the moderate hyperthermia (T_{re} 38.3 ± 0.2 °C) caused by 42 °C hot-water immersion does not impair rapid contraction force responses or cause neural drive attenuation (no differences found in RTD_{V50}, RTD_{V150} and RER between Post-1 and Post-2). Therefore, the enhanced neuromuscular responses (*i.e.*, ↑RTD_{V50}, ↑RTD_{20Hz}, ↑RTD_{100Hz}, ↓HRT, ↓EMD) apparent with an increased T_{mu} after passive heating seem to be influenced by peripheral rather than central responses. Interestingly, some peripheral variables (*i.e.*, RTD_{twitch}, RTD_{20Hz}, RTD_{100Hz}, EMD) demonstrated better outcomes approximately 15 min after the passive heating (*i.e.*, Post-1 < Post-2), indicating a possible beneficial timing effect of hot-water immersion on muscle contractile properties. Although this study was not able to show the effect of severe hyperthermia on rapid force production, the passive heating protocol adopted is safer and more tolerable than other protocols reaching T_c 39.5 °C.

The RTD_{V50} increased after hot-water immersion (26.3%) and sham-condition (11.3%) (Post-1) showing association with changes in deep T_{mu} across the time-points (Fig. 1C), potentially suggesting a dosage effect of deep T_{mu} on the early phase of voluntary rapid torque (see Appendix B, Fig B1, for repeated-measures correlations plot). At Post-2, when T_{re} returned to baseline, the RTD_{V50} after hot-water immersion remained higher than pre with no further

increase compared to Post-1, evidencing that T_{re} had no negative influence on RTD. The early phase of voluntary RTD has been mostly explained by neural contribution (EMG activity).[11,22] Thus, it was expected that increases in RTD_{V50} would be accompanied by increases in neuromuscular activity,[22] as the rate of EMG increase represents the neural equivalent of the RTD.[23] However, the rate of EMG rise in this study did not show any difference between conditions or pre and post time-points, indicating a potential muscle contractile property contribution to increases in RTD after passive heating rather than neural mechanisms. Nevertheless, gains in the early phase of the RTD are associated with an increase in stiffness of series elastic structures of the muscle-tendon unit (~40%) as the speed of force transmission through a material is influenced by the material's stiffness.[23] The initial phase of muscle activation force is low and has a high compliant series of elastic structures; however, when muscle force increases toward maximum during the late phase of the RTD the elastic structures are stretched, and their stiffness increase.[24] This may also explain why no change was observed on the RTD_{V150} after hot-water immersion (Fig. 1E), once changes in the late-phase of RTD are not related to shifts in the muscle-tendon unit stiffness.[24]

The early phase of the RTD (twitch and voluntary) in this study might have been positively influenced by the muscle-tendon unit stiffness effect caused by an increase in muscle fluid. Localised passive heating (T_{mu} ~37.4 °C) can increase muscle blood flow up to 60.9%, and evoke metabolically-induced vasodilation.[25] While not measured here, the high T_{mu} achieved (deep T_{mu} at Post-1 ~38.7 °C and Post-2 ~37.7 °C) makes it plausible that a muscle fluid shift would have occurred. Increases in muscle fluid have been associated with increased muscle fibre force and shortening velocity.[5] Increased muscle fluid requires muscle fibres to expand when they shorten to maintain a constant volume,[15] described as intramuscular spring property.[26] Consequently, the incompressible nature of the fluid within the muscle cells has an important mechanical role in muscle force transmission.[15,26] The intramuscular spring governs the muscle fibre shape decreasing the resistance in muscle thickness and increasing muscle width.[26] Because intramuscular spring is influenced by fluid pressure, the magnitude of muscle fluid load and intramuscular spring response will change the force transmission and contraction.[26] Moreover, intramuscular spring controls the muscle fibre rotation by resisting muscle thickness compression. Therefore, muscle water content could increase the RTD by inducing the fibres to rotate further during the contraction and creating additional longitudinal passive fibre shortening force.[5] The involuntary RTD did not present any statistical differences at Post-1, only at Post-2 (higher values than baseline and sham-condition, Fig. 1F). This response in the RTD_{twitch} may be explained by a timing effect of passive heating on muscle fluid (continuous increase throughout time-points), which would cause a further increase in the muscle muscle-tendon unit stiffness caused by intramuscular spring property. Although muscle fluid was not tested in the present study, this premise may also explain the better results in evoked doublets (RTD_{20Hz} and RTD_{100Hz}) found at Post-2 when compared to Post-1.

The Ca^{2+} release/sequestration kinetics might also have an important contribution to the effect of increased T_{mu} on the muscle's shortening velocity. Passive heating increases the myoplasmic Ca^{2+} accumulation[4,27] when muscle cells are exposed to temperatures ranging from 37–42 °C, temperature-dependently,[28] which increase the myofibrillar Ca^{2+} sensitivity and isometric muscle rate of force production.[29] The sarcoplasmic ryanodine receptors (RyR) (voltage sensitivity Ca^{2+} release gate) regulates the slope of the force-frequency relation responses in the muscle.[29] However, although the present study showed increased RTD after hot-water immersion during the electrically-evoked doublets, no difference was observed in the force-frequency slope relationship between low- and high-frequencies activation as RTD_{100Hz} and RTD_{20Hz} demonstrated similar response after passive heating (Fig. 2A,B). Moreover, the force-frequency relationship ratio ($RTD_{20/100Hz}$) showed no difference in this study, suggesting that increased T_{mu} by passive heating does not affect the RyR regulation. Alternatively, heat stimulates the knockdown of transient receptor potential vanilloid 1 (Trpv1),[28] which mediates the increase of [Ca^{2+}] from the sarcoplasmic reticulum into the muscle cells.[27] Consequently, Trpv1 rather than RyR appears to play a role in increasing sarcoplasmic [Ca^{2+}] after passive heating. Nevertheless, the present study data could not prove such hypothesis. On the other hand, our findings showed decreases in half-relaxation time at Post-1 and Post-2 time-points (Table 1), indicating that a passive increase in T_{mu} can accelerate the sarcoplasmic Ca^{2+} reuptake. These findings align with Périard et al.,[30] who reported improved twitched half-relaxation time after moderate hyperthermia (T_c ~38.5 °C; T_{mu} ~38.7 °C). The excitation-contraction model is governed by the Ca^{2+} sequestration back into the sarcoplasm after dissociation from troponin.[29] Together, these results indicate that passive heating may improve the Ca^{2+} sequestration kinetics.

This study also found decreased electromechanical delay (electrically-evoked) after passive heating for *vastus medialis* and *lateralis* (Table 1). This response can be related to the increases in evoked RTD after hot-water immersion, including the lower values at Post-2 compared to Post-1. The electromechanical delay reflects the synaptic transmission, action potential propagation,[31] Ca^{2+} release and its excitation-contraction coupling, and force transmission along with elastic components.[32] Therefore, electromechanical delay after passive heating can be decreased by increased muscle-tendon stiffness mediate by increases in muscle fluid content. Moreover, the possible increase of muscle fluid at Post-2 compared to Post-1 would plausibly explain the electromechanical delay response after passive heating by increasing the force transmission.

5. Conclusion

After a passive heating session of hot-water immersion for 90 min at 42 °C (when T_{mu} and T_{re} were elevated [↑2.4 °C and ↑1 °C, respectively]) increases in the early phase of voluntary explosive contraction was observed. Approximately 15 min after the hot-water immersion (elevated T_{mu} [↑1.4 °C], but T_{re} at baseline level), voluntary explosive contraction remained higher and, at this time, the involuntary RTD also increased. The voluntary explosive contraction responses seem to have a positive dose-response relationship effect with T_{mu} (positive correlation at Post-1 and Post-2). Furthermore, the hot-water immersion decreased half-relaxation time and electromechanical delay of knee extensor. It is suggested that the main positive effects of passive heating on rapid force contraction are likely caused by muscle-tendon unit stiffness and perhaps partly related to Ca^{2+} kinetics. These mechanisms may have also decreased the half-relaxation time and electromechanical delay. Rapid force production is not impaired by moderate hyperthermia (T_{re} ~38.3 °C), and a passive increase in T_{mu} seem to have a timing effect on muscle contractility properties (*i.e.*, RTD_{twitch}, RTD_{20Hz}, RTD_{100Hz}, EMD) by continuous increasing muscle fluid.

Conflict of interest

None.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.jsams.2021.01.003.

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