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**Hyperthermia reduces electromechanical delay via accelerated
electrochemical processes**

Running title: Heat therapy hastens muscle contraction

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28 ABSTRACT

29 The present study aimed to determine the effect of hyperthermia on both electrochemical and
30 mechanical components of the electromechanical delay (EMD), using very-high-frame-rate
31 ultrasound. Electrically evoked peak twitch force, EMD, electrochemical (D_m , *i.e.*, delay
32 between stimulation and muscle fascicle motion) and mechanical (T_m , *i.e.*, delay between
33 fascicle motion and force production onset) components of EMD were assessed in sixteen
34 participants. Assessments were conducted in a control ambient environment (CON: 26°C,
35 34% relative humidity) and in hot ambient environment (HOT: 46-50°C, 18% relative
36 humidity, after ~127 min of heat exposure). Following heat exposure, *gastrocnemius medialis*
37 temperature was $37.0 \pm 0.6^\circ\text{C}$ in HOT vs. $34.0 \pm 0.8^\circ\text{C}$ in CON ($P < 0.001$). EMD was shorter
38 (9.4 ± 0.8 ms) in HOT than CON (10.8 ± 0.6 ms, $P < 0.001$). Electrochemical processes were
39 shorter in HOT than CON (4.0 ± 0.8 ms vs. 5.5 ± 0.9 ms, respectively, $P < 0.001$), while
40 mechanical processes were unchanged ($P = 0.622$). These results demonstrate that
41 hyperthermia reduces electromechanical delay via accelerated electrochemical processes
42 while force transmission along the active and passive parts of the series elastic component is
43 not affected following heat exposure. The present study demonstrates that heat exposure
44 accelerates muscle contraction thanks to faster electrochemical processes. Further
45 investigations during voluntary contractions would contribute to better understand how these
46 findings translate into motor performance.

47 **New & Noteworthy**

- 48 • Hyperthermia (targeted core temperature: 38.5°C) reduces the time between
49 *gastrocnemius medialis* stimulation and the onset of plantar flexor force
50 production *in vivo*.
- 51 • This reduction in electromechanical delay is concomitant to an earlier motion of
52 muscle fascicle compared to thermoneutral environment.
- 53 • However, hyperthermia has no impact on the duration of force transmission along
54 aponeurosis and tendon, thereby reflecting different effects of heat exposure on
55 contractile and elastic properties of the muscle-tendon unit.

56 INTRODUCTION

57

58 A rise in core or muscle temperature affects muscle metabolism (2, 14), neural drive (31),
59 contractile properties (10) and overall force production (33). Indeed, tissue temperature is
60 recognized to influence mechanical and biochemical properties of skeletal muscle (5, 13). *In*
61 *vitro*, a rise in temperature increases electrically-evoked rate of force development (RFD) (9,
62 36), with an inverse relationship between temperature and contraction time (CT; *i.e.*, the time
63 between the onset of force rise and peak twitch force) and half-relaxation time (HRT; *i.e.*, the
64 time to obtain half of the decline in twitch maximal force) inferred from a muscle twitch (41).
65 *In vivo*, a rise in muscle temperature has been reported to consistently shorten CT and HRT of
66 peak twitch, while its amplitude increased (11) or was not altered with heat (29, 32, 34).
67 Although these findings reflect an improvement in neuromuscular function during explosive
68 motor tasks, the origins of this effect remain unsolved.

69 The ability to generate force rapidly is influenced by the neural drive transmission to the
70 muscle, the propagation of the action potential, the excitation-contraction coupling processes
71 and the muscle force transmission along the series elastic component (25, 28). On the one
72 hand, it is well established that axonal conduction velocity increases in hot environments (38,
73 43). On the other hand, little is known regarding the impact of hyperthermia on force
74 transmission efficiency by the contractile and elastic components. Diverse effects have been
75 reported on electromechanical processes involved in force transmission efficiency in response
76 to an increase in temperature. Heat increases ATPase activity of the myosin head (3), while it
77 promotes calcium retention by the sarcoplasmic reticulum (42). In addition, heat may
78 differently impact the mechanical properties of the active myofibrils (39) and the passive
79 aponeurosis and tendon within the series elastic components (18). Because the respective
80 contributions of contractile and elastic materials may be challenging to dissociate *in vivo*,

81 further evidence is required to clarify the acute effects of heat exposure on the muscle-tendon
82 unit components.

83 The measurement of time between the muscle activation onset and force production (EMD;
84 *i.e.*, electromechanical delay) reflects both electrochemical processes (*i.e.*, synaptic
85 transmission, action potential propagation through the sarcolemma, excitation-contraction
86 coupling) and mechanical processes (*i.e.*, force transmission along the active and passive parts
87 of the series elastic component) (6). Thus, the investigation of EMD would provide more
88 information about the mechanisms and muscle-tendon components involved in force
89 transmission efficiency. EMD has been shown to be sensitive to cooling (7, 46). Indeed, the
90 decrease in muscle or skin temperature, subsequent to the localized application of cold,
91 induces an increase in EMD in humans (21, 26, 46). Using mechanomyography, Cè et al. (7)
92 reported a lengthening of the electrochemical but not the mechanical processes in response to
93 muscle cooling. Using very high-frame-rate ultrasound, it is possible to detect the onset of
94 muscle fascicle and myotendinous junction motions in response to myostimulation (22, 23,
95 24, 28). This technique offers the opportunity to determine the respective contribution of
96 electrochemical and mechanical processes to heat-induced changes in EMD in humans. Thus,
97 the present study may contribute to better identify the steps of force production and
98 transmission potentially responsible for the improvement in explosive strength reported in the
99 literature following heat exposure.

100 The present study aimed to determine (i) the influence of hyperthermia on EMD; and (ii) the
101 respective heat-mediated alterations in electrochemical and/or force transmission processes.
102 We hypothesized that EMD would decrease in response to heat exposure likely due to faster
103 electrochemical processes.

104

MATERIALS AND METHODS

Participants

Sixteen recreationally active participants (9 men and 7 women, age: 24.9 ± 5.7 yrs, height: 174.8 ± 7.5 cm and body mass: 69.8 ± 9.9 kg), with no recent history of ankle disorder or injury, participated in this study. Participants completed a pre-inclusion medical visit consisting of a clinical examination, a medical history questionnaire and anthropometric measurements before entering to the study. They were informed regarding the nature, aims and risks associated with the experimental procedures before providing written consent. This study was approved by the Sud-Ouest et Outre-mer III ethics committee (approval reference: 3849, ID-RCB: 2019-A00596-51) and conformed to the standards of the Declaration of Helsinki.

Experimental design

Two to four days after a familiarization session allowing the participants to be accustomed to the procedures, the participants performed two identical testing sequences during a single visit, including electrically-evoked peak twitch and EMD assessment. The tests were first performed in control ambient environment (CON; $25.8 \pm 1.8^{\circ}\text{C}$, $33.6 \pm 8.6\%$ relative humidity) and then in hot ambient environment (HOT; $47.4 \pm 1.84^{\circ}\text{C}$, $18.5 \pm 4.7\%$ relative humidity) after the participants passively reached a core temperature of 38.5°C (*i.e.*, after 127 ± 33 min from the onset of heat exposure). After heat exposure, muscle temperature increased from $34.0 \pm 0.8^{\circ}\text{C}$ to $37.0 \pm 0.6^{\circ}\text{C}$ ($P < 0.001$) in a subsample of six participants. The hyperthermia was controlled by an environmental chamber (Thermo-training room, Paris,

France) and participants could drink *at libitum*. Body mass was measured before and after heat exposure to evaluate the weight loss due to dehydration.

Twitch force

A custom-built ergometer composed of a specific footplate (Bio2M, Compiègne, France) was used to measure plantar flexor force evoked by electrical nerve stimulations. Participants were laid prone with their legs fully extended and their ankle flexed at 90° (*i.e.*, foot perpendicular to the tibia). Their right foot was firmly fixed on the footplate connected to a force sensor (2712-100 daN-0.02-B, Sensy, Charleroi, Belgium). The force signal was digitized at a sampling rate of 2 kHz using an analog-to-digital converter designed in our laboratory. The tibial nerve was electrically stimulated using a constant current stimulator (Digitimer DS7AH, Digitimer, Letchworth Garden City, UK) delivering a single electrical pulse (1000 µs, 400 V) through a cathode placed in the popliteal cavity and an anode placed distally to the patella. The intensity was adjusted for each participant by progressive increase in current (*i.e.*, incremental step of 10 mA) until plantar flexor force reached a plateau. Thereafter, five stimulations were delivered at the electrical intensity required to elicit peak force multiplied by 1.5.

EMD

Participants laid prone with their legs fully extended on a second home-made ergometer previously used to measure the EMD on plantar flexor muscles (22, 23, 28). Their right foot was firmly attached in a rigid cycling shoe (chosen to avoid possible dynamics in coupling between the shoe and the force sensor) fixed on an adjustable system connected to a force sensor (2712-50 daN-0.02-B, Sensy, Charleroi, Belgium) near the metatarsal joint.

Percutaneous electrical stimulations were applied over the *gastrocnemius medialis* (GM) to elicit contraction by a single electrical pulse (1000 μ s, 400 V) through two electrodes, one placed on the motor point (previously determined as the location inducing the strongest twitch with a low electrical stimulation) and the other placed on the distal portion of GM. The stimulation intensity corresponded to the intensity necessary to obtain peak force. An ultrafast ultrasound scanner (v. 12, Aixplorer, Supersonic Imagine, Aix en Provence, France) coupled with a linear transducer array (4-15 MHz, SuperLinear 15-4, Vermon, Tours, France) was used to acquire raw radiofrequency signals at 4 kHz. Muscle stimulations were delivered 50 ms after the onset of ultrasound images acquisition through an automatized trigger signal transmitted from the ultrasound scanner to the stimulator, thereby ensuring a consistent synchronization between mechanical and ultrasound data. For each participant, two bouts (muscle and tendon trials) composed of three electrically-evoked contractions with 1 min rest between each trial were performed. During the muscle trials, the ultrasound probe was placed over the GM muscle belly to detect the onset of muscle fascicle motion, reflective of electrochemical processes. During tendon trials, the ultrasound probe was maintained over the distal myotendinous junction of GM to detect the onset of myotendinous junction motion, reflective of mechanical processes. These two bouts were performed alternating the order, which was the same in CON and HOT. Participants were instructed to fully relax before each stimulation.

Temperature monitoring

Prior to testing, participants were instrumented to control their core and skin temperatures. Core temperature was monitored rectally using an electronic capsule (e-Celcius, BodyCap, Caen, France) self-inserted by the length of a gloved finger. Participants were instrumented

with four data loggers (iButtons, Maxim Integrated, USA) measuring skin temperature at the arm, chest, thigh and shin to calculate the average skin temperature, according to the following equation (35):

$$0.3 \times \text{chest temperature} + 0.3 \times \text{arm temperature} + 0.2 \times \text{thigh temperature} + 0.2 \times \text{shin temperature}$$

Core and skin temperatures were continuously measured throughout the testing sequences and heat exposure. To obtain temperatures values in HOT, the mean value of core and skin temperatures were averaged during the testing sequence after heat exposure. In addition, muscle temperature of contralateral GM was measured immediately before each testing sequence in a subsample of six participants using a needle intramuscular thermistor (MKA08050-A, Ellab, Roedovre, Denmark) inserted under local anesthesia after skin disinfection.

Data analysis

Data were analysed using custom-written scripts (Origin 2020, OriginLab corporation, USA and Matlab 2010a-2017b, The Mathworks, Natick, MA).

Twitch force

First, force signal was low-pass filtered (20 Hz, zero lag 3rd order Butterworth). Then, the mechanical response to the five electrically-evoked stimulations were analysed and averaged to determine peak twitch amplitude (PT; *i.e.*, the highest value of twitch force production), CT and HRT. The average RFD was calculated as PT/CT (34).

EMD

Force signals were low-pass filtered (200 Hz, zero lag 3rd order Butterworth). We defined the EMD as the delay between electrical stimulation and the force production onset. For each

198 participant, six values of EMD were obtained in each test sequence: three muscle trials and
 199 three tendon trials. As previously described by Nordez et al. (28) and Lacourpaille et al. (23),
 200 ultrasound B-mode images were used to determine the region of interest for each stimulation
 201 in order to detect the motion onset for GM fascicle during muscle trials (*i.e.*, between the two
 202 aponeuroses) and myotendinous junction during tendon trials (*i.e.*, on the myotendinous; Fig.
 203 1). The displacements of the regions of interest along the ultrasound beam axis were
 204 calculated using a one-dimensional cross correlation of the windows of consecutive
 205 ultrasound images. Thus, the tissue motion between two consecutive images (*i.e.*, particle
 206 velocity) was measured with micrometric precision. Absolute particle velocities were
 207 averaged within previously determined regions of interest and then used to detect the onset of
 208 GM fascicle (Fig. 1A) and myotendinous junction (Fig. 1B) motion. The onset of motion was
 209 detected as the first point with a negative derivative of tissue motion in the reverse direction
 210 time (28). The same method was used to automatically detect the onset of the force
 211 production over time. Visual inspection was performed to check and validate the onset
 212 detection for each signal. Then we determined the delay (in ms) between the onset of
 213 electrical stimulation (which corresponded to the trigger output signal collected from the
 214 electrical stimulator) and either the onset of muscle fascicle motion (D_m , for muscle fascicle
 215 trials) or the onset of myotendinous junction motion (D_t , for tendon trials). The delay between
 216 the onset of GM fascicle motion and the onset of force production is attributed to the force
 217 transmission (T_m , time delay to force transmission). The difference between D_m and D_t and
 218 between D_t and EMD were calculated to respectively compute the delay of force transmission
 219 along aponeurosis and tendon. Using the same technique as described elsewhere, Nordez et al.
 220 (28) and Lacourpaille et al. (24) respectively demonstrated a good repeatability [standard
 221 error measurement (SEM) < 0.88 ms; coefficient of variation (CV): 5.0-11.6%] and a good
 222 inter-day reliability of EMD, D_m and D_t (SEM < 0.79 ms; CV: 6.8-12.5%). In the current

study, SEM were < 0.40 ms and CV values ranged from 8.2 to 15.2% across EMD, D_m and D_t .

Statistical analysis

All statistical analyses were performed with Statistica (v. 13.0; StatSoft, Tulsa, Oklahoma, USA). Values were therefore reported as mean \pm standard deviation (SD). The assumptions of normality of the data were verified using a Shapiro-Wilk's test. Paired t-tests were used to compare the effect of the environmental condition (CON vs. HOT) on core, skin and muscle temperatures, mechanical responses to the electrically-evoked stimulation (PT, CT, HRT and RFD) and EMD. A two-way analysis of variance (ANOVA) for repeated measures [condition (CON, HOT) \times delay (D_m , T_m)] was used to test whether heat exposure affected, absolute (in ms) and relative (in % of EMD) values of D_m and T_m . Then, a two-way ANOVA for repeated measures [condition (CON, HOT) \times delay (aponeurosis, tendon)] was used to test the effect of heat exposure on force transmission independently of electrochemical process, in absolute and relative values. When the sphericity assumption in repeated measures ANOVAs was violated (Mauchly's test), a Geisser-Greenhouse correction was used. When appropriate, *post hoc* analyses were performed using Bonferroni tests. Effect sizes were described in terms of partial eta-squared (η^2_p , with $\eta^2_p \geq 0.06$ representing a moderate effect and $\eta^2_p \geq 0.14$ a large effect). Statistical significance was set as $P < 0.05$.

RESULTS

Heat exposure

Mean core and skin temperatures were significantly higher in HOT ($38.4 \pm 0.3^{\circ}\text{C}$ and $38.8 \pm 0.4^{\circ}\text{C}$, respectively) than CON ($37.0 \pm 0.3^{\circ}\text{C}$ and $34.7 \pm 0.7^{\circ}\text{C}$, respectively; all P values < 0.001). Shin skin temperature was evenly significantly higher in HOT ($38.8 \pm 0.9^{\circ}\text{C}$) than CON ($34.3 \pm 0.9^{\circ}\text{C}$; $P < 0.01$). Muscle temperature measured in a subsample ($n = 6$) from the 16 participants was $37.0 \pm 0.6^{\circ}\text{C}$ in HOT vs. $34.0 \pm 0.8^{\circ}\text{C}$ in CON ($P < 0.001$). Heat exposure, which lasted 127 ± 33 min, had no statistical effect on body mass measured in HOT vs. CON ($P = 0.89$).

Twitch force

No difference was found in PT amplitude between HOT and CON ($P = 0.301$, $\eta^2_p = 0.036$; Table 1). Both CT and HRT were shorter in HOT than CON ($-9.0 \pm 6.5\%$ and $-15.1 \pm 10.1\%$ respectively; $P < 0.001$, $\eta^2_p \geq 0.419$; Table 1). RFD was higher in HOT than CON ($+16.9 \pm 32.8\%$; $P = 0.029$, $\eta^2_p = 0.149$; Table 1).

EMD

EMD was 1.37 ± 0.87 ms ($-12.6 \pm 7.7\%$) shorter in HOT than CON ($P < 0.001$, $\eta^2_p = 0.523$; Fig. 1). This reduction in EMD consistently occurred for all participants (range: -0.08 to -2.98 ms). We observed an effect of heat exposure on absolute D_m and T_m (condition effect: $P < 0.001$, $\eta^2_p = 0.509$). *Post hoc* analysis revealed that D_m was significantly shorter in HOT than CON ($-26.5 \pm 18.2\%$, $P < 0.001$, $\eta^2_p = 0.416$; Figs. 1-2), whereas no significant difference was found for T_m between HOT and CON ($P = 0.622$, $\eta^2_p = 0.008$; Fig. 1). Two-way ANOVA revealed a main effect of hyperthermia on relative contribution of D_m and T_m to EMD ($P = 0.015$, $\eta^2_p = 0.182$). *Post hoc* analysis showed that the relative contribution of D_m to EMD was smaller in HOT than CON ($-8.0 \pm 11.1\%$, $P = 0.015$, $\eta^2_p = 0.182$). Conversely, the relative

contribution of T_m to EMD was higher in HOT than CON ($+8.0 \pm 11.1\%$, $P = 0.015$, $\eta^2_p = 0.182$). We found no significant main effect of heat exposure on absolute force transmission delays (aponeurosis and tendon; $P = 0.270$, $\eta^2_p = 0.086$). However, two-way ANOVA revealed a main effect of hyperthermia on relative contribution of force transmission to EMD (aponeurosis and tendon, $P = 0.034$, $\eta^2_p = 0.208$). *Post hoc* showed that the relative contribution of force transmission along aponeurosis to EMD was not altered by heat exposure ($P = 0.294$, $\eta^2_p = 0.037$), whereas relative contribution of force transmission along tendon to EMD was greater in HOT than CON ($+10.8 \pm 11.5\%$, $P = 0.012$, $\eta^2_p = 0.192$).

DISCUSSION

The aim of this study was to determine the influence of hyperthermia induced by heat exposure on EMD and the subsequent alterations in electrochemical and mechanical processes involved in EMD. In accordance with our initial hypothesis, hyperthermia reduced the delay between muscle stimulation and the onset of plantar flexor force production *in vivo*. This reduction was concomitant with shorter electrochemical processes (*i.e.*, decreased delay between muscle stimulation and the onset of muscle fascicle motion) compared with control conditions. The delay between muscle fascicle motion and force production was unchanged in hot environment, reflecting no impact of hyperthermia on force transmission along elastic components.

In line with previous reports, contraction time and half-relaxation time were shortened and rate of force development increased after heat exposure (11, 34). In comparison with Racinais et al. (34), the smaller reduction in contraction time (-9.0% vs. -36.5%) and half relaxation time (-15.1% vs. -25.2%) and the smaller increase in rate of force development ($+16.9\%$ vs. $+64.7\%$) observed in the current study was probably due to the smaller amplitude of core

temperature increase with heat exposure (+1.4 vs. +2.6°C). The main aim of the present experimental protocol was to identify the phases of force production and transmission that could account for this heat-mediated improved explosive performance.

The delay between muscle stimulation and the onset of force production measured under thermoneutral environment (10.8 ± 0.6 ms) was in the range of EMD reported in the literature on plantar flexors muscles [*i.e.*, 7.9 ms to 18.8 ms (16, 17, 27, 28)]. With muscle temperature increase, EMD consistently decreased in all participants by 1.37 ± 0.87 ms on average (*i.e.*, -12.6 \pm 7.7%) as compared to the values obtained in a thermoneutral environment. In line with the present findings, a previous study reported a 16% decrease in EMD in malignant hyperthermia susceptible participants [*i.e.*, +4.3% of muscle temperature (2)]. Conversely, Kubo et al. (21) observed an increase in EMD of plantar flexors after a 30 min immersion in hot water of the lower limbs. The partial immersion, the difference in heat medium (water vs. ambient air) or the contraction modality and the shorter duration of heat application compared to the present study (*i.e.*, 30 min vs. 127 min) may partly explain this discrepancy, although it is not possible to attest the impact of hot water immersion on core or muscle temperature in the aforementioned study. Using local application of heat (*i.e.*, hot packs), Zhou et al. (46) measured EMD during voluntary contractions of quadriceps muscle at muscle temperatures between 30°C and 38°C. EMD decreased from 30°C to 36°C, then it increased up to 36-38°C. Our results showed a decrease in EMD from a muscle temperature of 34°C in thermoneutral environment to 37°C in hot environment, suggesting that the impact of heat on EMD may differ between voluntary and electrically-evoked contraction and between muscle groups. Given the different properties between knee extensor and plantar flexor muscles, these findings may also illustrate a possible muscle-dependent effect of heat exposure (18, 34, 39). Overall, our findings merged with previous works collectively suggest a continuum of temperature-mediated effect on EMD *in vivo*, reflected by a negative relationship between

318 muscle temperature and EMD. Further investigations exploring EMD under various muscle
319 temperatures are required to conclude with certainty on the nature of this potential relation.

320 By coupling ultrafast ultrasound to EMD assessments, we aimed to distinguish the
321 contribution of the electrochemical processes from the muscle force transmission along the
322 series elastic component to EMD. In line with our hypothesis, we observed substantial
323 differences in the delay between muscle stimulation and the onset of muscle fascicle motion
324 in hot compared to thermoneutral environment (-1.46 ± 1.12 ms, $-26.5 \pm 18.2\%$). This
325 reduction could be attributed to faster synaptic transmission, propagation of action potential
326 through the sarcolemma and/or excitation-contraction coupling (6, 8, 28). Interestingly, when
327 normalized to EMD, the relative contribution of electrochemical processes also decreased
328 with hyperthermia ($-8.0 \pm 11.1\%$), demonstrating that the electrochemical processes were
329 accelerated in a greater extent than the impact appraised on the global EMD. At a muscle
330 temperature of 23°C achieved through localized cold application, Cè et al. (7) reported a
331 lengthening of electrochemical processes which were multiplied by ~ 1.5 . These variations in
332 the duration of electrochemical processes upon hot and cold environments could be related to
333 temperature-mediated effects on axonal conduction velocity. Indeed, axonal conduction
334 velocity can decrease with cold and inversely increase with heat (12, 38). On the one hand, a
335 decrease in muscle temperature is recognized to slow down both the opening and closing of
336 Na^{+} channels, while an increase in muscle temperature accelerates synaptic transmission (33,
337 37). However, the depolarization of muscle fibres depends on the influx of Na^{+} ions through
338 cell membrane. Increasing ambient temperature reduces the amplitude and the duration of
339 depolarization, due to reduced Na^{+} influx. This effect in turn challenges the production of a
340 muscle fibre action potential (37), which could suggest an increase in the duration of
341 electrochemical processes with heat. On the other hand, an increase in ambient temperature
342 additionally increases ATPase activity of myosin heavy chains (3) and reduces Ca^{2+} uptake in

the sarcoplasmic reticulum, thereby increasing the rate of cross-bridge cycling (40, 42). Thus, literature reports heat-mediated effects that would inversely impact the duration of electrochemical processes after a heat exposure. The present experimental approach does not allow to dissociate the respective contribution of each process involved in hyperthermia-induced changes. However, Nordez et al. (28) suggested a minor role of the active part of the series elastic component to electrochemical processes. It is therefore likely that synaptic transmission and excitation-contraction coupling may be primarily affected by hyperthermia. One could note that an acceleration of synaptic transmission is associated with a lower amplitude of action potential, which could impact the action potential response (37). A faster synaptic transmission would reduce electrochemical processes duration, which has yet been shown to be associated with a lower amplitude of the action potential response with heat. Hyperthermia could thus likely reduce the amplitude of the action potential. However, our results showed a faster response.

Our findings showed that absolute delay for force transmission along the aponeurosis and the tendon was not affected by ambient temperature. In their systematic review, Bleakley et al. (4) reported an equivocal effect of heat or cold on passive stiffness in soft tissues. *In vitro*, tendon viscosity and temperature follow an exponential relationship (18), suggesting an increase in tendon compliance following heat exposure, confirmed by animal studies (44, 45) reporting a greater viscous mechanical behavior with heat. *In vivo*, local application of heat on lower limbs does not change the stiffness of tendinous tissues, assessed in passive and active conditions, despite an extended range of motion (15, 21). Such effect of hyperthermia on the mechanical properties of the series elastic component could impair the effectiveness of force transmission. In contrast, cooling resulted in increasing tendon stiffness, *in vitro* (19) and *in vivo* after thirty minutes of local icing (1) or four sets of four minutes, with one minute recovery in between, of cryotherapy (30). These results suggest that absolute force

transmission processes are unchanged with heat. In accordance with the current scientific literature, this advocates that tendinous stiffness could thus be unchanged or slightly diminished with heat.

Methodological considerations

Our findings showed a decrease in electro-induced EMD with hyperthermia. Given that viscoelastic responses of muscle and tendon may differ between electro-induced and voluntary contractions, these results could be slightly different during voluntary contractions. However, one may assume that EMD measurements during voluntary contraction also present some drawbacks. As suggested by Hug et al. (20), EMD measurements obtained during voluntary contractions could be influenced by methodological artefacts and should be interpreted with caution. Indeed, EMD measured during voluntary contraction, with the use of electromyography, is insufficient to precisely detect the onset of activation of the studied muscle fibres. While the assessment of EMD is challenging in voluntary conditions, voluntary RFD has been shown to increase in hot environment. While electromechanical processes appeared to be the main responsible for the reduction in EMD *in vivo*, the relative contributions of the synaptic transmission, the excitation-contraction coupling, and the active part of the series elastic component could not be directly quantified in the present study. Further assessments of EMD and its various components in several cold and hot conditions may contribute to determine the nature of the relationships between temperature and EMD suggested in the literature (46). Given that EMD plays a major role in explosive motor task performance, the present findings demonstrate that ambient temperature should be reported with explosive strength evaluations and EMD delays measurements. The present study allowed us to locate the phases of force production and transmission potentially altered

following heat exposure which could impact explosive strength and in turn sport performance. With an electromechanical delay reduction of 1.37 ms (*i.e.*, 12.6% of EMD in GM muscle), the effect of hyperthermia can be important for performance and make a difference in many sports. Further investigations during voluntary contractions are warranted to improve our understanding of the effects of heat exposure on the different components of the muscle-tendon unit in hot conditions and their consequences on motor performance.

Conclusion

The present study showed that hyperthermia decreased the time elapsed between the onset of muscle activation and force production, mainly through an acceleration of electrochemical processes. However, mechanical processes involved in force transmission during electromechanical delay were unchanged following heat exposure. These findings contribute to elucidate the mechanisms involved in the enhanced contractile properties previously reported following an acute heat exposure. While future research is required to further describe the role of muscle-tendon dynamics in heat-mediated effects, the present results suggest a major role of synaptic transmission and excitation-contraction coupling in electromechanical delay shortening. This study allowed to determine the respective contributions of electromechanical and mechanical processes to heat-induced changes in EMD. A shortening of electromechanical delay and electrochemical processes might explain the improvement in explosive strength reported in the literature. Future investigations of muscle-tendon dynamics involved during voluntary contractions are warranted for a better understanding of motor performance following hyperthermia.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

A.M., S.R., F.B. and G.G. conceived and designed the research; A.M., S.R., F.B., M.A., R.H., Y.D. and G.G. performed the experiments; A.M., R.H. and G.G. analysed the data; A.M., S.R., F.B., and G.G. interpreted the results; A.M. S.R., F.B. and G.G. prepared the figures; A.M. and G.G. drafted the manuscript; A.M., S.R., F.B., M.A. and G.G. edited and revised the manuscript; A.M., S.R., F.B., M.A., R.H., Y.D. and G.G. reviewed and approved the final version of the manuscript.

FIGURES LEGENDS

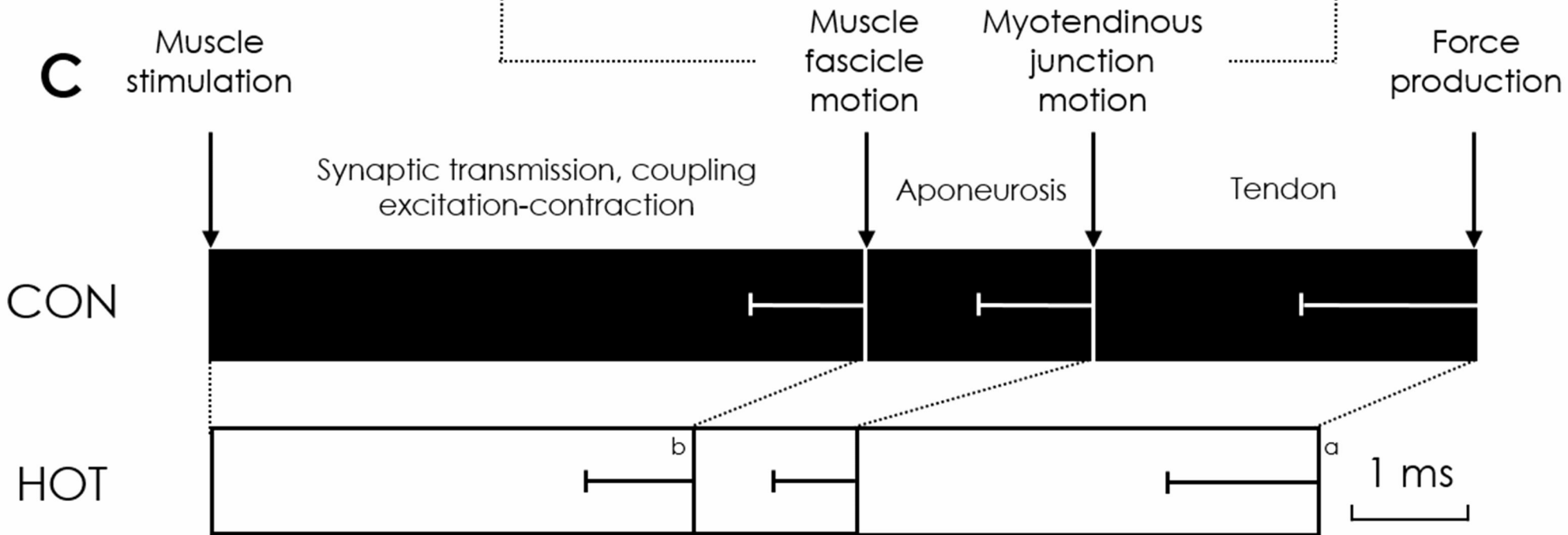
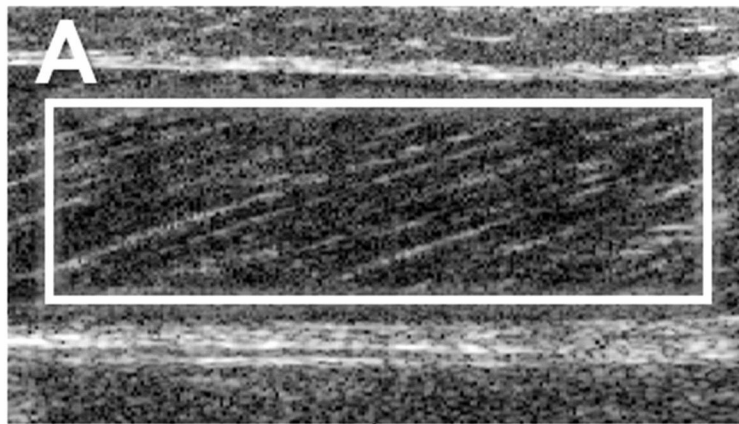
Figure 1. Ultrasound images showing the regions of interest delineated with white squares for the GM muscle (A) and myotendinous junction (B). Representation of the electromechanical delay (EMD) and its components in control ambient (CON) and hot ambient (HOT) environment (C). Horizontal stacked bar plots represent means \pm SD. The delay between electrical muscle stimulation and the onset of muscle fascicle motion is attributed to electrochemical processes (D_m). The delay between the onset of fascicle motion and the onset of force production is attributed to force transmission (T_m). The delay between muscle electrical stimulation and the onset of myotendinous junction motion (D_t) reflects the time required for electrochemical processes and force transmission along aponeurosis. ^a and ^b: Significant difference between HOT and CON for EMD and D_m (ms); $P < 0.001$.

Figure 2. Delay between the onset of electrical stimulation and the onset of muscle fascicle motion (D_m , A) and delay between the onset of muscle fascicle motion and the onset of force production (T_m , B), obtained in control ambient (CON) and hot ambient (HOT) environment. The bold traces represent the mean change, and box charts correspond to standard deviation. *** Significant difference between HOT and CON, $P < 0.001$.

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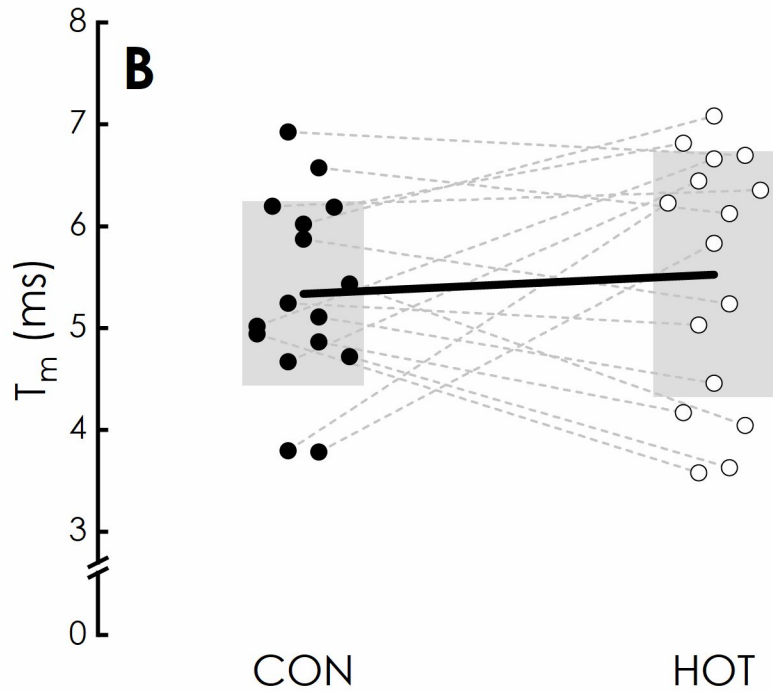
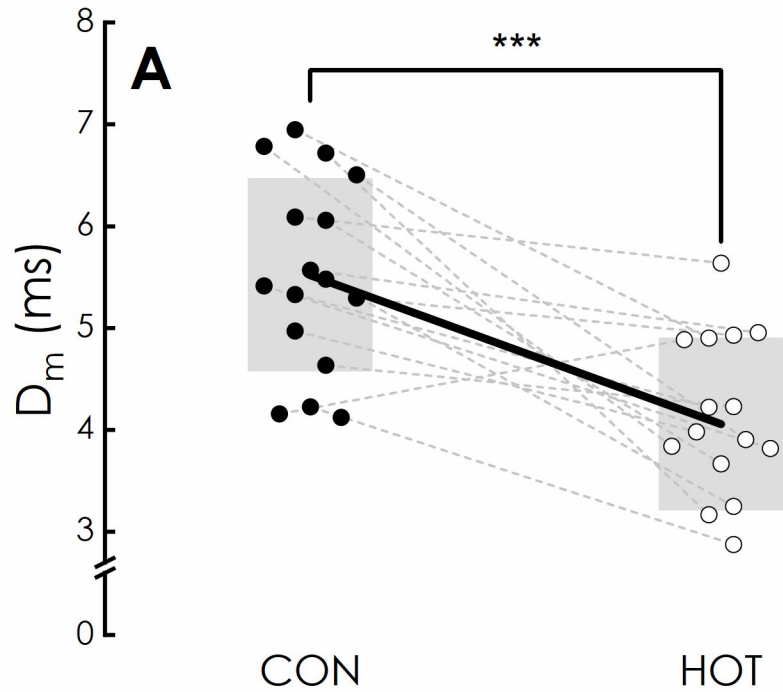


Table 1. Main characteristics of the nervous electrically-evoked twitch in control ambient (CON) and hot ambient (HOT) environment.

	CON	HOT	<i>P</i>	η^2_p
PT (N)	66.9 ± 12.9	71.8 ± 13.9	0.301	0.036
CT (ms)	121.2 ± 7.5	110.2 ± 5.7***	< 0.001	0.419
HRT (ms)	88.8 ± 6.1	75.4 ± 8.1***	< 0.001	0.485
RFD (N.ms⁻¹)	0.56 ± 0.12	0.65 ± 0.11*	0.029	0.149

Values are presented as mean ± SD. PT, peak twitch amplitude; CT, contraction time; HRT, half relaxation time; RFD, rate of force development. * Significant difference between HOT and CON, $P < 0.05$; ***, $P < 0.001$.