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Accelerated muscle contractility and decreased muscle steadiness following sauna recovery do not induce greater neuromuscular fatigability during sustained submaximal contractions

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ABSTRACT

Acute whole-body hyperthermia (WBH) increases blood markers concentration of stress, impairs motor drive to exercising muscles, and decreases resistance to neuromuscular fatigability. The functional natural residual consequences of WBH on neuromuscular functions remain unclear. We aimed to investigate the effects of residual WBH on voluntary and electrically induced ankle plantar flexor contractility properties, motor drive transmission (reflexes), muscle torque steadiness, resistance to neuromuscular fatigability, and markers of stress as the body temperature recovers naturally to normothermia. WBH was induced by Finnish sauna bathing in 16 apparently healthy young (24 ± 4 years) adult men. Motor performance was monitored before and 2 h after the sauna, and immediately after submaximal exercise (120 s at 50% of maximal voluntary contraction). Markers of stress were monitored before and 2 h after the sauna. Finnish sauna exposure induced moderate to severe WBH (rectal temperature, 38.5–39.6 °C). At 2 h after the sauna, rectal temperature had recovered to the preheating level (preheating 37.11 ± 0.33 °C versus postheating 37.00 ± 0.29 °C, $p > .05$). Post-sauna recovery was accompanied by slowed salivary free cortisol diurnal kinetics, whereas noradrenaline, dopamine, and serotonin did not persist into the 2 h recovery after the sauna. Although recovery to normothermia after a sauna led to a greater acceleration of muscle contractility properties and decreased muscle steadiness, sustained isometric submaximal contraction did not provoke greater neuromuscular fatigability.

1. Introduction

Use of a sauna is described as a method for inducing muscle, nerve, and blood vessel relaxation (Mero, Tornberg, Mäntykoski, & Puurtinen, 2015) and for attenuating pain and improving joint mobility (Hannuksela & Ellahham, 2001). Sauna bathing is associated with activation of the sympathetic nervous system (Vuori, 1988), which regulates muscle contractile force (Bowman, 1981), blood perfusion to the muscle (Thomas & Segal, 2004) and proprioceptive feedback to motoneurons in the spinal cord (Hellström, Roatta, Thunberg, Passatore, & Djupsjöbacka, 2005). However, little is known about the residual consequences for neuromuscular function after recovery of body temperature from moderate whole-body hyperthermia (WBH) in a thermoneutral environment.

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Hot air in the sauna (80 °C) affects the skin and the respiratory system; as a result, body core temperature can increase by more than 0.8 °C and body weight can decrease by 0.7–0.9 kg (Kauppinen, 1989; Leppäluoto, Tuominen, Väänänen, Karpakka, & Vuor, 1986). Increased internal body temperature (hyperthermia) is a physiological stress (Moran, Shitzer, & Pandolf, 1998). Physiological stress activates the hypothalamic–pituitary–adrenal hormonal axis (Hannuksela & Ellahham, 2001). The cortisol level may remain elevated during recovery after a sauna in a cooling room (Kukkonen-Harjula et al., 1989). Also after an induced (with sprint interval cycling exercise) stress, cortisol concentration remains elevated for longer than noradrenaline (NA) concentration (Skurvydas et al., 2017). The elevated level of free cortisol causes mobilization of glucose, which increases glucose availability for muscle metabolism (Pääkkönen & Leppäluoto, 2002). A greater supply of energy can have a positive effect on muscle contraction and relaxation. Acute stress may increase force fluctuation during sustained muscle contractions because of elevated sympathetic outflow (Keller-Ross, Schlinder-Delap, Doyel, Larson, & Hunter, 2014) and motor unit synchronization during fatiguing exercise (Mesin, Dardanello, Rainoldi, & Boccia, 2016). Development of muscle fatigue can be defined as a decrease in muscle steadiness, increase in the force fluctuation (Missenard, Mottet, & Perrey, 2009), and decline in maximal force immediately after a fatiguing contraction (Enoka & Duchateau, 2008; McNeil, Murray, & Rice, 2006).

Hormones such as dopamine (DA), NA, and serotonin (5-HT) are implicated in the control of thermoregulation (Bridge, Weller, Rayson, & Jones, 2003), and changes in their concentrations play an important role in determining central fatigue during and after exposure to high temperature (Roelands & Meeusen, 2010; Zhao et al., 2015). NA level increases during heat exposure and remains elevated after cooling following a sauna (Hannuksela & Ellahham, 2001; Kukkonen-Harjula et al., 1989). Exposure to a cold stimulus after heat activates the sympathetic nervous system (Kauppinen, 1989). Cooling during exercise improves trial performance in the heat without affecting fatigue perception (Sunderland, Stevens, Everson, & Tyler, 2015), improves the subjective rating of thermal comfort (Tyler & Sunderland, 2011), and with increase body temperature cold or cool stimuli perceive as being comfortable and increased alertness (Cabanac, 1971). As a result forced external cooling after hyperthermia may blunt the natural recovery after heat exposure.

However, McMorris et al. (2006) showed that NA and 5-HT levels return to the preheating level after recovery. Morrison, Sleivert, and Cheung (2004) and Thomas, Cheung, Elder, and Sleivert (2006) reported that maximal voluntary contraction (MVC) and voluntary activation are restored to the baseline level when the core temperature returns to normal after passive hyperthermia. In McMorris et al. (2006) and Morrison et al. (2004) studies participants were rehydrated. However, Hoffman et al. (1994) and Melin et al. (1994, 1997) suggested that the extent of dehydration is probably more important than heat exposure in increasing plasma NA and cortisol concentrations.

Cernych, Satas, and Brazaitis (2018) investigated the natural post-sauna recovery responses without cooling stimuli and without restoration of lost fluid, and showed enhanced relaxation in the brain neural network (increase in alpha oscillations). Increased alpha band activity is associated with increased 5-HT level (Yu et al., 2011). DA and NA levels are critical for regulation of cortical arousal (Chaouloff, 1989; Ouyang, Hellman, Abel, & Thomas, 2004). Reduced activity in neurons of the locus coeruleus (the largest adrenergic nucleus) and reduced level of NA decrease arousal (Berridge & Foote, 1991). Inhibition of NA reuptake is associated with greater central/supraspinal fatigue, but inhibition of DA reuptake does not affect central fatigue (Klass et al., 2012). Whereas an increase in alpha oscillations is observed after post-sauna recovery (Cernych et al., 2018), increased 5-HT level (Yu et al., 2011) and decreased NA and DA levels (Chaouloff, 1989; Ouyang et al., 2004) can be expected investigating post-sauna recovery effect on stress hormones changes, and as a result greater central fatigue will be induced.

In this study, we aimed to induce moderate WBH (rectal temperature, $T_{re} \sim 38.5$ °C (Lucas, Sarma, Schlader, Pearson, & Crandall, 2015)) in young men by using a traditional Finnish sauna and to evaluate the residual effect of hyperthermia on neuromuscular function as body temperature recovered naturally to the initial level. We hypothesized that during recovery 2 h after passive hyperthermia induced by a sauna without cooling or fluid replacement, cortisol concentration would remain elevated and that this elevated cortisol level would improve muscle contractile properties (Pääkkönen & Leppäluoto, 2002) during MVC and electrically induced muscle contractions. Decreased NA level (Berridge & Foote, 1991) and increased 5-HT level (Yu et al., 2011) may induce greater central fatigability (Roelands & Meeusen, 2010; Zhao et al., 2015). These factors can provoke greater force fluctuation (Keller-Ross et al., 2014; Mesin et al., 2016) during the contraction of ankle plantar flexor at 50% of an MVC for 120 s, and lower muscle steadiness should lead to greater overall (central and peripheral) neuromuscular fatigability.

2. Methods

2.1. Participants

The participants were 16 young men with mean (± SD) age of 24 ± 4 years; their anthropometric measures were: 184.19 ± 8.28 cm height, 84.27 ± 15.28 kg mass, 24.73 ± 3.24 $kg \cdot m^{-2}$ body mass index, and 15.74% ± 4.03% body fat. They were considered healthy and physically active but did not participate excessively in sport activities (i.e., exercised < 3 times per week). They had had no involvement in any temperature-manipulation program for ≥3 months, were nonsmokers, and took no medications that could affect natural thermoregulation and/or tolerance to fatigue. The participants were asked to avoid any physical work on the testing day, food and drink (except water) for 4 h before testing, and eating or drinking during all experimental sessions.

After being informed of the purpose, experimental procedures, and known risks of the study, the participants signed a written informed consent form to participate in this study and were then familiarized with all experimental procedures. All procedures were approved by LUHS Kaunas Region Biomedical Research Ethics Committee.

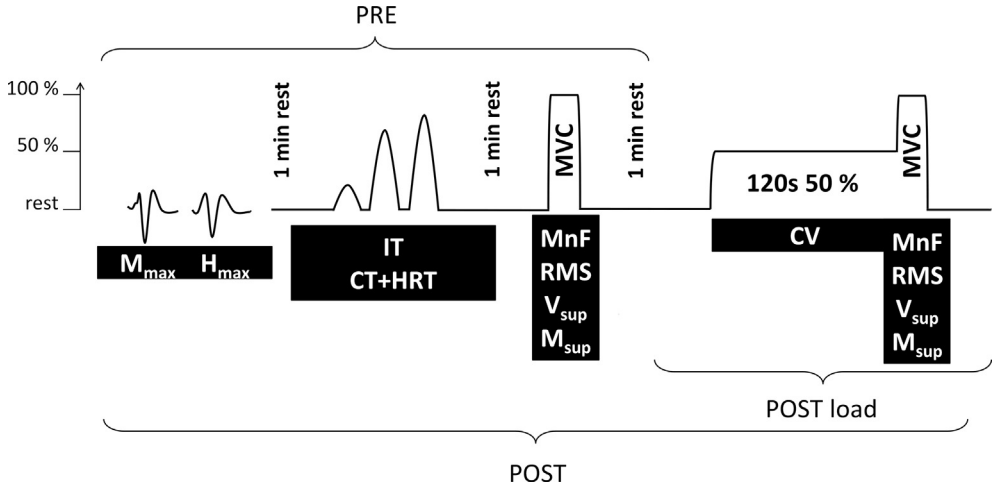

Fig. 1. Schematic representation of the experimental protocol. M_{max} = M-wave; H_{max} = H-reflex; IT = involuntary torque induced with electrical stimulation at 1 Hz, 20 Hz, and 100 Hz; CT + HRT = muscle contraction time plus half-relaxation time (torque induced with 100 Hz stimulation); MVC = maximal voluntary contraction, MnF = mean frequency; RMS = root mean square; V_{sup} = V-wave during MVC; M_{max} = M-wave during MVC; CV = coefficient of variation of force.

2.2. Experimental design

The experiment was designed to evaluate the residual effects on neuromuscular function after WBH induced by a standard sauna. Each participant completed 2 experimental sessions, both at the same time of day (16:00 h) and spaced at least 7 days apart. In the experimental session (EXP), WBH was induced. The other session was a control (CON) without thermal stress. The order of testing was randomized. On arrival at the laboratory, the participant's nude body weight was measured (Tanita UK Ltd., Philpots Close, UK) and he was asked to rest in semi-recumbent posture for 20 min in a thermoneutral (T_a 23 °C) ambient condition (Fig. 1). Resting rectal temperature (T_{re}) and heart rate (HR) were recorded, and saliva and blood samples were collected. Within ~3 min after these measurements, the participant was seated in an isokinetic dynamometer chair for neuromuscular function testing.

Upon completion of the neuromuscular function recording, the participant entered the sauna. WBH was induced by Finnish sauna bathing (temperature 80–90 °C, relative humidity 30%). The sauna bathing included 4 sets: the first for 15 min and the next 3 sets for 10 min each, with 15 min rest between each set in a thermoneutral ambient condition. At the end of the fourth sauna set, T_{re} and HR were recorded.

After the sauna protocol, the participant took a warm shower and rested in a thermoneutral ambient condition for 2 h. At the end of the 2 h, nude body weight was measured to quantify body weight loss, T_{re} and HR were measured, saliva and blood samples were collected, and neuromuscular function was tested. In the CON condition, the participant spent time in a thermoneutral ambient condition for the same duration as the sauna bathing protocol.

2.3. Physiological heat stress evaluation

T_{re} and HR were measured to assess the physiological strain index (PSI). T_{re} was measured using a rectal thermocouple (Rectal Probe, Ellab, Hvidovre, Denmark; accuracy ± 0.01 °C) inserted by each participant to 12 cm past the anal sphincter. HR was measured using an S-625X Polar Electro HR monitor (Kempele, Finland). The PSI was calculated using the equation of Moran et al. (1998):

$$PSI = 5(T_{ret} - T_{re0}) \times (39.5 - T_{re0})^{-1} + 5(HR_t - HR_0) \times (180 - HR_0)^{-1}$$

T_{re0} and HR_0 refer to the T_{re} and HR values obtained before the sauna; T_{ret} and HR_t refer to the T_{re} and HR values obtained before leaving the sauna/after 2 h recovery. The PSI value was scaled to the range from 1 (no heat stress) to 10 (very high heat stress) within the limits of the following values: $36.5 \leq T_{re} \leq 39.5$ °C and $60 \leq HR \leq 180$ beats min^{-1}.

2.4. Saliva and blood samples

The samples of saliva and blood were collected at 2 time points: before and 2 h after the sauna (EXP) or rest (CON) before neuromuscular function testing. No saliva stimulants were used. The participant rinsed his mouth with water 5 min before sample collection. The participants had no oral disease, inflammation, or lesions. A minimum of 1 mL of saliva was collected into special tubes (IBL SaliCap, Germany). Samples were stored at –24 °C for later analysis. The concentration of free cortisol in saliva was measured using an enzyme-linked immunosorbent assay (ELISA) using a Gemini analyzer (Stratec Biomedical GmbH, Birkenfeld, Germany). The ELISA is based on the principle of competitive binding and was performed in accordance with the manufacturer's

instructions (Labor Diagnostika Nord GmbH & Co. KG, Nordhorn, Germany).

Blood samples were obtained immediately after saliva samples and were used to measure the concentrations of NA, 5-HT, and DA. Blood samples were collected by venipuncture into vacuum tubes with EDTA as the anticoagulant (EDTA-K3, 3 mL) to measure NA and DA concentrations in blood plasma and into vacuum tubes for serum with a gel separator (5 mL) to measure 5-HT concentration. The blood samples were centrifuged at 1200 × *g* for 15 min at 4 °C. Samples for plasma were centrifuged immediately after blood collection, and the samples for serum were allowed to clot before being centrifuged. Plasma and serum were separated from the pelleted cells immediately after centrifugation. All samples were stored at −70 °C until analysis. NA, DA, and 5-HT concentrations were measured using an ELISA kit on a Gemini analyzer (Stratec Biomedical GmbH, Birkenfeld, Germany). The assays were performed in accordance with the manufacturer's instructions.

2.5. Neuromuscular function

All preparation procedures, recordings, and analyses were made according to the method of Brazaitis et al. (2016).

The torque of the ankle plantar flexor muscles was measured using an isokinetic dynamometer (System 3; Biodex Medical Systems, Shirley, NY, USA). The subject was seated in the dynamometer chair with the trunk inclined at 45° with respect to the vertical, and with hip, knee, and ankle joint angulations of 90°, 100° (full knee extension = 180°), and 90°, respectively. Stimulating electrodes were fixed over the tibial nerve, electromyography (EMG) electrodes were placed over the soleus (SOL) muscle (~13 cm above the calcaneus and below the muscle fibers of the gastrocnemius) of the right leg, and the electrode position was marked. The ground electrode was positioned on the tarsus of the same leg. The EMG signal was recorded on the biometrics memory card and analyzed using MATLAB (MathWorks, Inc., USA).

Peak involuntary torque (measured in N m) induced by a single electrical stimulation at 1 Hz, and by 5-electrical-stimuli at 20 Hz, and 100 Hz was measured with a 3 s rest interval between electrical stimulation. Contraction plus half-relaxation time (CT + HRT, in ms) was calculated as the time taken for the 100 Hz torque to increase to the peak value and then to decrease to half of that value.

In the soleus muscle (SOL), the resting H-reflex (H_{max}) and M-wave (M_{max}) were evoked by 0.5 ms square-wave pulses stimulated by a cathode placed in the popliteal cavity and an anode placed distal to the patella over the posterior tibial nerve with an inter-electrode distance of ~4 cm (Aagaard, Simonsen, Andersen, Magnusson, & Dyhre-Poulsen, 2002; Palmieri, Ingersoll, & Hoffman, 2004). Increasing the electrical intensity by 3 V every 10 s in the 30–150 V range caused the H-reflex response to increase progressively and then to decrease and disappear. By contrast, the M-wave achieved its maximum and remained stable.

After the resting measurements were obtained, the participant performed 4 brief (3–4 s) MVCs of the plantar flexor muscles with at least 1 min rest between. The highest MVC value was used for further analysis.

The same electrical stimulus as that used to induce the H-reflex and M-wave was induced during the MVC M-wave (M_{sup}) and V-wave (V_{sup}). From the EMG signal, the peak-to-peak amplitude (in mV) and latency (in ms) of the electrical evoked action potentials of the H_{max}, M_{max}, M_{sup} and V_{sup} were calculated. The M_{max} and M_{sup} amplitudes were used to normalize the amplitude of the reflex waves recorded (H_{max}/M_{max}, V_{sup}/M_{max}, and V_{sup}/M_{sup}) (Aagaard et al., 2002; Solianik, Skurvydas, Mickevičienė, & Brazaitis, 2014). EMG signals such as the root mean square (RMS, in mV) and mean frequency (MnF, in Hz) were extracted from a 1 s epoch coinciding with the 1 s force interval just before each 100 Hz superimposed on an MVC.

After the EXP and CON measurements, upon completion of the MVC and after the 1 min rest, the participant was asked to achieve and maintain a contraction of the ankle plantar flexors at 50% of his MVC for 120 s. The coefficient of variation for force (CV, as a %) was calculated by applying the equation:

$$CV = (SD/x) \times 100\%,$$

where SD is the standard deviation and x is the mean torque during the 120 s contraction.

Immediately after the 120 s load (POST load) imposed after the sauna or rest, the participant performed an MVC. The M_{sup} and V_{sup} were evoked by 0.5 ms square-wave pulses during the MVC. The RMS and MnF were extracted from a 1 s epoch coinciding with the 1 s force interval just before the 1 Hz superimposed on an MVC.

2.6. Statistical analysis

The data were tested for normal distribution using the Kolmogorov–Smirnov test, and all were found to be normally distributed. The data are presented as the mean and SD. One-sample *t* test was performed to compare the effects of the sauna visit at 2 times (before and 2 h after EXP) on the factor T_{re}.

Two-way ANOVA (general linear model) was used to identify any residual effects of sauna bathing on hormone concentrations (before vs 2 h after) as within-subject factors at 2 levels and trial (CON vs EXP) as within-subject factors at 2 levels. If significant effects were found, Tukey's post hoc adjustment was used for multiple comparisons within each repeated-measure ANOVA. A dependent-sample *t* test was used to locate any differences in time points.

Two-way ANOVA (general linear model) was used to identify the residual effects of sauna bathing on neuromuscular function (before, 2 h after the sauna, Post-load) as the within-subject factors at 3 levels and trial (CON vs EXP) as within-subject factors at 2 levels. If significant effects were found, Tukey's post hoc adjustment was used for multiple comparisons within each repeated-measure ANOVA. A dependent-sample *t* test was used to locate the difference in time points. For all tests, statistical significance was defined as $p < .05$. Statistical analyses were performed using IBM SPSS Statistics software (v. 22; IBM Corp., Armonk, NY, USA).

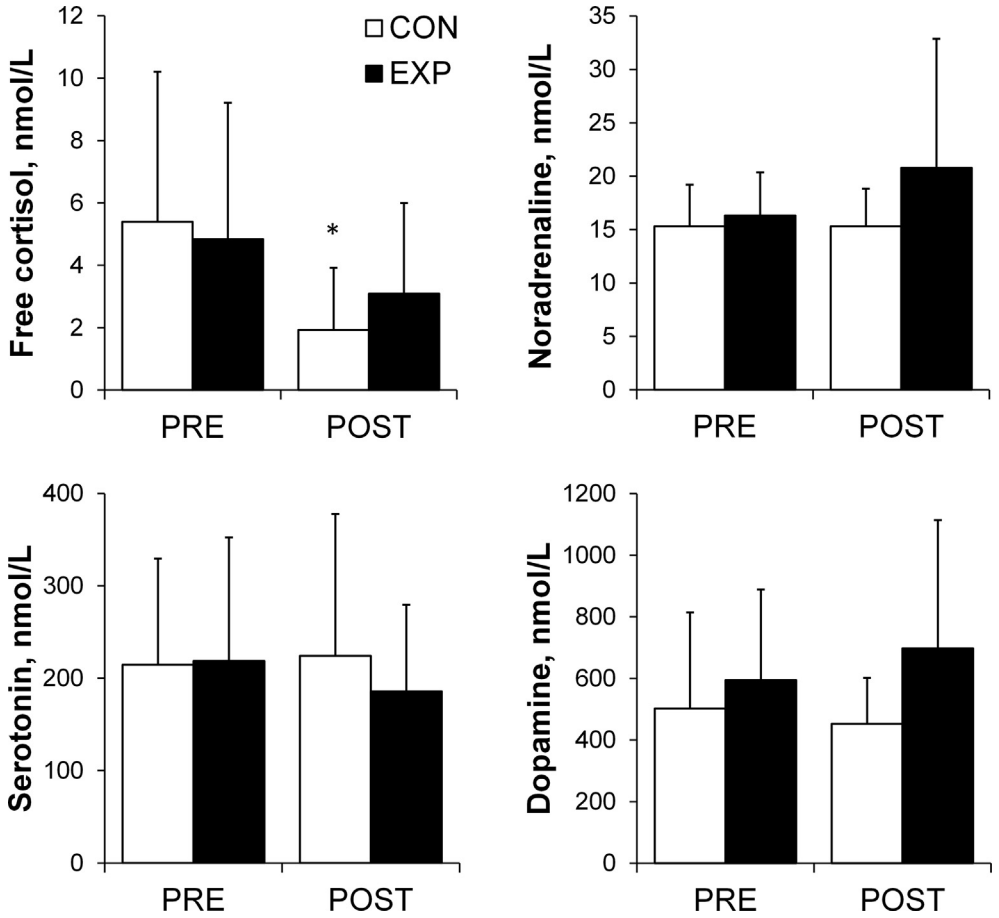

Fig. 2. Blood variables before (Pre) and after (Post) the control (CON) and experimental (EXP) trials. Values are shown as mean ± SD. $^{*}p < .005$ compared with before.

3. Results

3.1. Physiological responses

Participants experienced moderate to high thermal strain, as indicated by a PSI of 6.45 ± 1.47, during the last minute in the sauna. At 2 h after the sauna, the participants had recovered from the thermal strain (PSI 0.94 ± 0.64). T_{re} recovered to the pre-heating level (Pre 37.11 ± 0.33 °C, Post 37.00 ± 0.29 °C). Participants lost 1.30 ± 0.33 kg by sweating.

3.2. Hormone concentrations

In CON, the free cortisol concentration was significantly lower after compared with before the rest ($p < .05$) (Fig. 2). Free cortisol concentration did not change significantly after EXP. DA, NA, and 5-HT concentrations did not differ from before to after the trials in both CON and EXP (Fig. 2).

3.3. Neuromuscular function

The sauna significantly decreased the electrically induced muscle torque evoked with 1 Hz stimulation ($p < .05$) (Fig. 3). No other significant differences ($p > .05$) in involuntary muscle torque amplitude after the rest or sauna were found (Fig. 3). In EXP, CT + HRT was significantly shorter 2 h after heating than before heating ($p < .05$) (Fig. 3). In CON, CT + HRT did not change significantly from before to after rest.

In EXP, MVC was significantly lower 2 h after heating than before heating ($p < .05$) In CON, MVC did not differ significantly from before to after rest (Fig. 4). MnF and RMS did not change from before to after rest (CON) or heating (EXP) (Fig. 4).

The spinal and supraspinal reflex excitability amplitudes and V_{sup}/M_{max} and V_{sup}/M_{sup} ratios did not change (Table 1). M_{max} latency decreased significantly after heating ($p < .05$). M_{sup} latency decreased significantly after the sauna ($p < .05$) and was lower

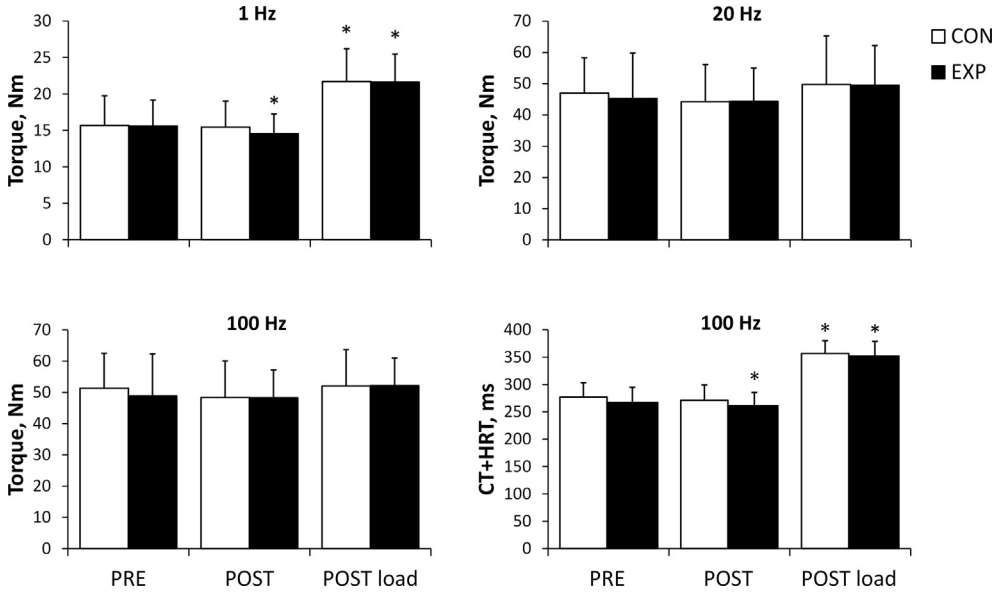

Fig. 3. Electrically induced ankle plantar flexor muscle torque induced by stimulation at 1 Hz, 20 Hz, and 100 Hz, and the sum of muscle contraction and half-relaxation time (CT + HRT, lines) before (Pre) and after (Post) the rest control (CON) and experimental (EXP) trials, and immediately after the 120 s contraction at 50% of MVC (Post-load). Values are shown as mean ± SD. $^{*}p < .005$ compared with before.

after EXP than after CON (Table 1).

All participants maintained the contraction at 50% of MVC for 120 s: CON 61.83 ± 9.86 N m, EXP 64.74 ± 11.95 N m. However, the CV of force was significantly higher 2 h after heating in EXP (2.77% ± 1.78%) compared with the same time after CON (2.14% ± 0.79%) ($p < .005$).

The 120 s load significantly increased the electrically induced muscle torque induced with 1 Hz stimulation ($p < .005$). The 120 s load significantly increased CT + HRT in both trials, but the values did not differ significantly between CON and EXP (Fig. 3).

MVC was significantly lower after the 120 s load compared with before (Fig. 4) in both trials ($p < .005$). MnF and RMS were also significantly lower after ($p < .05$) compared with before the test (Fig. 4). However, the MnF and RMS did not differ significantly between CON and EXP. V_{sup} and M_{sup} amplitudes, V_{sup}/M_{sup} ratio, and V_{sup} latency did not change significantly after the 120 s load in either the CON or EXP trial. By contrast, in CON, M_{sup} latency was significantly lower after than before the 120 s load; M_{sup} latency was also significantly lower after EXP than after CON (Table 1).

4. Discussion

The aim of this study was to assess the effects of residual hyperthermia induced by a traditional sauna on neuromuscular function in young men during natural recovery of body temperature. When participants recovered after heat stress and returned to normothermia 2 h after the sauna, the properties of muscle excitation–contraction coupling (as shown by 1 Hz-induced torque), CT + HRT and sarcolemmal excitability (M_{max}, M_{sup}) remained faster. However, SOL muscle voluntary torque (MVC) decreased after recovery from hyperthermia but the electrophysiological activity (RMS and MnF) did not change. Force variability (CV) was higher during 120 s submaximal sustained contraction after hyperthermia, but this increase did not cause any additional changes in neuromuscular function, except for the change in M_{sup} latency. The free cortisol concentration remained unchanged from the preheating level (EXP), although it declined after rest (CON) because of circadian rhythm (Horrocks et al., 1990).

The shorter M-wave latency accompanied by the decreased CT + HRT shows that the main changes after post-heat recovery occurred in muscle. The elevated concentration of free cortisol mobilizes glucose, thereby increasing its availability for muscle metabolism (Pääkkönen & Leppäluoto, 2002). Changes in crucial metabolites such as ATP can improve muscle contractile properties. A possible explanation is that, because the participants remained dehydrated, changes in electrolyte distribution across the sarcolemma affected muscle contractile properties, and calcium release and reuptake had not recovered by 2 h after hyperthermia. The M-wave may have changed because of alterations in ion distribution, transport through the plasmalemma, sarcolemma permeability, or activity of the ion pump (Piitulainen, Komi, Linnamo, & Avela, 2008).

Changes in the muscle fiber conductance velocity are generally explained by a shorter depolarization time, which allows less Na^+ to enter cells and lowers the M-wave amplitude (Rutkove, 2001). At the same time, the ATP-requiring Na^+–K^+ pump (Sejersted & Sjøgaard, 2000) may be affected by changes in cortisol level or disturbance of ion distribution across the sarcolemma, secondary to fluid loss by sweating, and these may alter the resting and threshold membrane potentials (Piitulainen et al., 2008). For these reasons, decreased conductance velocity was not accompanied by reduced M-wave amplitude.

It seems that the loss of muscle strength observed here, as measured by MVC, did not involve the spinal and/or supraspinal

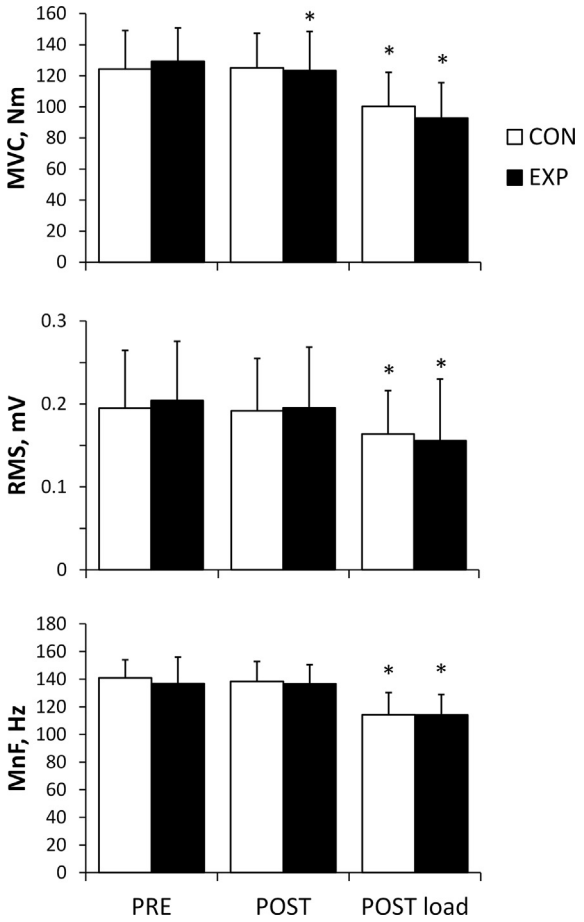

Fig. 4. Changes in maximal voluntary contraction (MVC) torque of the plantar flexor muscles, and mean frequency (MnF) and root mean squire (RMS) of electromyogram of the soleus muscle before (Pre) and after (Post) the rest control (CON) and experimental (EXP) trials, and immediately after the 120 s contraction at 50% of MVC (Post-load). Values are shown as mean ± SD. $^{*}p < .005$ compared with before.

Table 1
Changes in spinal and supraspinal reflex excitability before (Pre) and after (Post) rest (CON) and sauna (EXP), and immediately after a 120 s contraction at 50% of maximal voluntary contraction (MVC) torque (Post-load). Resting H-reflex (H_{max}) and M-wave (M_{max}); V-wave (V_{sup}) and M-wave (M_{sup}) during MVC.

	CON			EXP		
	PRE	POST	After 2 min load	PRE	POST	After 2 min load
Amplitude, mV						
H_{max}	3.18 ± 0.97	3.24 ± 0.99		3.19 ± 0.90	3.15 ± 1.02	
M_{max}	4.34 ± 0.24	4.31 ± 0.38		4.28 ± 0.59	4.33 ± 0.25	
H_{max}/M_{max}	0.73 ± 0.22	0.75 ± 0.21		0.75 ± 0.20	0.73 ± 0.23	
V_{sup}	2.68 ± 1.18	2.86 ± 1.21	2.13 ± 1.31	2.79 ± 1.12	2.68 ± 0.92	1.67 ± 0.88
M_{sup}	3.86 ± 0.87	3.87 ± 0.91	3.56 ± 1.09	3.88 ± 0.95	4.05 ± 0.66	3.98 ± 0.80
V_{sup}/M_{max}	0.62 ± 0.28	0.67 ± 0.28		0.66 ± 0.24	0.61 ± 0.21	
V_{sup}/M_{sup}	0.75 ± 0.42	0.79 ± 0.44	0.70 ± 0.62	0.80 ± 0.42	0.67 ± 0.22	0.50 ± 0.47
Latency, ms						
H_{max}	38.13 ± 2.66	38.31 ± 2.75		37.94 ± 2.32	37.63 ± 2.55	
M_{max}	11.63 ± 1.41	11.63 ± 1.36		11.63 ± 1.20	11.19 ± 1.42*	
V_{sup}	36.81 ± 2.20	37.19 ± 2.71	37.67 ± 2.66	36.56 ± 2.28	36.44 ± 1.86	37.36 ± 2.06
M_{sup}	9.25 ± 1.29	9.69 ± 1.62	10.00 ± 1.71*	9.56 ± 1.41	9.19 ± 1.28$^{*,\#}$	9.47 ± 1.30$^{\#}$

Values are shown as mean ± SD.
* $p < .005$ compared with before.
$^{\#}$ $p < .005$ compared with CON.

exitability because the H-reflex and V-wave remained unchanged. This change in MVC may not reflect central fatigue, as indicated by EMG activity (Gandevia, 2001). The decrease in strength associated with changes in muscle was reflected in changes in M-wave latency and CT + HRT. In the study by Morrison et al. (2004), voluntary activation and MVC returned to baseline after induced passive hyperthermia (39.4 °C) followed by cooling to lower the core temperature to normal. The authors concluded that changes in force and voluntary activation could be attributed directly to changes in core temperature. However, they used cooling and their participants were not dehydrated, and these conditions may have blunted the effects of natural post-heat recovery.

Sustained muscle contraction (120 s at 50% of MVC) was performed with greater force fluctuations after post-heat recovery, but these changes did not relate to changes in DA, NA, and 5-HT concentrations. McMorris et al. (2006) also reported that, with a mean increase in core temperature of 1.10 ± 0.11 °C, the concentrations of DA, NA, and 5-HT returned to the preheating level during post-heat recovery. In the present study, T_{re} was elevated by 1.73 ± 0.38 °C, and cortisol concentration did not change from before heat exposure to after post-sauna recovery. Because of the circadian rhythm of cortisol, the free cortisol level was expected to decrease naturally during this time (Horrocks et al., 1990), as observed in CON. Slowed salivary cortisol diurnal kinetics in young men after the 2 h post-sauna recovery might be one factor that may have influenced the muscle contractile properties. By elevating stress hormone levels, acute stress might decrease muscle steadiness, as shown by excessive and inappropriate motor control (Christou, Jakobi, Critchlow, Fleshner, & Enoka, 2004; Keller-Ross et al., 2014). Greater force fluctuation during a sustained load is an indicator of fatigue because more motor units must be recruited to hold the same torque, which induces greater fluctuations in force (Singh, Arampatzis, Duda, Heller, & Taylor, 2010) and motor unit synchronization during contraction (Mesin et al., 2016). Although the greater force fluctuation during 120 s sustained contraction in EXP led to decreased muscle steadiness, no decline in torque was observed during this contraction or during the maximal effort tasks (MVC, RMS, and MnF) immediately after. These results suggest that the task time to exhaustion is more suitable than sustained 120 s at 50% of MVC load for inducing greater fatigability after post-sauna recovery.

Moreover sustained exercise after CON seemed to have induced peripheral perturbations beyond the sarcolemma (Girard, Bishop, & Racinais, 2013), and the muscle response, as shown by M_{sup}, was slower in this condition. Members of the heat-shock protein family in human muscle have been shown to be activated and to act as protection factors during and 24 h after heat exposure involving an increase in muscle temperature at a 2 cm depth to about 40 °C (Ogura et al., 2007). Possibly because of protection factors activated during heat stress in EXP, sarcolemmal excitability was not affected after sustained muscle contraction.

It is important to note that only young men were included in present study and that the results can be interpreted only in this context. Physiological sex differences, such as differences in muscle mass, muscle morphology, and neuromuscular activation (Hicks, Kent-Braun, & Ditor, 2001) may influence post-heat recovery (Cernych et al., 2017; Kenny & Jay, 2007). Aging reduces the ability for thermoregulation, and older men accumulate and store more heat in the skin and deep muscle than young men because of greater heat transfer delay and inertia (Brazaitis et al., 2017). The results of the present study showing the physiological responses to moderate heat stress should be evaluated in young women, and middle-aged and older men and women.

5. Conclusion

In conclusion, we show that post-sauna recovery was accompanied by slowed salivary free cortisol diurnal kinetics, whereas the heat stress-induced increased NA, DA, and 5-HT serotonin levels (McMorris et al., 2006) did not persist into the 2 h recovery after the sauna. Although recovery to normothermia after the sauna caused greater acceleration of muscle contractile properties and decreased muscle steadiness, sustained (120 s) isometric submaximal contraction (50% of MVC) did not provoke greater neuromuscular fatigability.

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