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Title

The effect of head and neck per-cooling on neuromuscular fatigue following exercise in the heat

Running title

Cooling and central fatigue in the heat

Authorship

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Author contributions

The present investigation was conducted at the Sports and Exercise Science Research Centre physiology laboratory, located on the Whitelands campus at the University of Roehampton. Ralph Gordon, Neale Tillin and Christopher Tyler contributed to the conception and design of the study. Ralph Gordon, Neale Tillin and Christopher Tyler contributed to the acquisition, analysis and interpretation of the data. Ralph Gordon drafted the manuscript and Neale Tillin and Christopher Tyler made critical revisions. Ralph Gordon, Neale Tillin and Christopher Tyler have approved the final version of the manuscript. Ralph Gordon, Neale Tillin and

Christopher Tyler agree to be accountable for all aspects of the presented work. Ralph Gordon, Neale Tillin and Christopher Tyler qualify for authorship.

Abstract

The effect of localised head and neck per-cooling on central and peripheral fatigue during high thermal strain was investigated. Fourteen participants cycled for 60 min at 50% $\dot{V}O_{2peak}$ on three occasions: CON (18°C), HOT (35°C) and HOT with cooling ($HOT_{cooling}$). Maximal voluntary force (MVF) and central activation ratio (CAR) of the knee extensors were measured every 30s during a sustained maximal voluntary contraction (MVC). Triplet peak force was measured following cycling, pre-and post the MVC. Rectal temperatures were higher in $HOT_{cooling}$ (39.2 ± 0.6°C) and HOT (39.3 ± 0.5°C) than CON (38.1 ± 0.3°C; $P < 0.05$). Head and neck thermal sensation was similar in $HOT_{cooling}$ (4.2 ± 1.4) and CON (4.4 ± 0.9; $P > 0.05$) but lower than HOT (5.9 ± 1.5; $P < 0.05$). MVF and CAR were lower in HOT than CON throughout the MVC ($P < 0.05$). MVF and CAR were also lower in $HOT_{cooling}$ than CON at 5, 60, and 120s, but similar at 30 and 90s into the MVC ($P > 0.05$). Furthermore, they were greater in $HOT_{cooling}$ than HOT at 30s, whilst triplet peak force was preserved in HOT post-MVC. These results provide evidence that central fatigue following exercise in the heat is partially attenuated with head and neck cooling, which may be at the expense of greater peripheral fatigue.

Novelty

- Central fatigue was greatest during hyperthermia
- Head and neck cooling partially attenuated the greater central fatigue in the heat
- Per-cooling led to more voluntary force production and more peripheral fatigue

Key words

Hyperthermia, cooling, central activation, maximal voluntary contraction, peripheral fatigue, exercise.

Introduction

Sub-maximal endurance exercise performance is impaired in hot environmental conditions (Galloway & Maughan, 1997). The reasons for the impaired performance are yet to be fully elucidated but may be partly due to neuromuscular fatigue. Neuromuscular fatigue is measured as a decline in maximal voluntary force (MVF) production and may be caused by mechanisms distal (peripheral fatigue; Allen et al., 2008) and/or proximal (central fatigue) to the neuromuscular junction (Gandevia, 2001). Peripheral fatigue is typically measured as a decrease in the involuntary contractile forces, reflecting a reduction in the available force capacity of muscle (Allen et al., 2008). Central fatigue is often measured as a larger decline in maximal voluntary- relative to involuntary contractile forces (Todd et al., 2005), representing a reduced ability of the central nervous system to drive the available force capacity of muscle.

Neuromuscular fatigue is exacerbated when thermal strain increases (core body temperature ≥ 38.5°C) and evidence suggests that this is due to increased central fatigue (Nybo & Nielsen, 2001a, Périard et al., 2014). Specifically, observations of larger reductions in MVF during sustained (45-120s) maximal voluntary contractions (MVC) following exercise-induced hyperthermia compared to control conditions, have been accompanied by larger declines in the central activation ratio (CAR; ratio of MVF- to the sum of MVF and superimposed-involuntary forces; Shield & Zhou, 2004) during the sustained MVCs (Nybo & Nielsen, 2001a, Périard et al., 2014). Similar evidence of reductions in MVF and CAR during sustained MVCs have also been observed during passively induced thermal strain (Todd et al., 2005, Périard et al., 2014, Racinais et al., 2008). Despite consistent evidence of greater central fatigue during high thermal strain compared to control conditions, it is unclear how hyperthermia effects the development of peripheral fatigue during fatiguing exercise. The degree of peripheral fatigue recorded in a fatiguing contraction to task failure is directly proportional to the absolute force task (Burnley et al., 2012). It is therefore conceivable that the hyperthermia-induced reduction in neural drive leading to lower force outputs during fatiguing contractions would result in lower peripheral fatigue, but this hypothesis has not been tested.

Hyperthermia-induced central fatigue may be attenuated by externally cooling the head and/or neck region. Neck cooling has been demonstrated to improve time trial running performance and time to exhaustion (by ~6 – 13%; Tyler & Sunderland, 2011b) in hot environmental conditions, without influencing thermoregulatory or cardiovascular strain (Tyler & Sunderland, 2011b, Tyler et al., 2010, Tyler & Sunderland, 2011a). The improvement may be due to the neck cooling reducing the temperature of the thermoregulatory centre at the brain (Racinais et al., 2008), but is more likely to be improved perception of thermal strain (Tyler & Sunderland, 2011b, Nielsen & Jessen, 1992), permitting the participant to tolerate higher core temperatures and/or select a faster pace for the same core temperature (Tyler & Sunderland, 2011b). By improving perceptions of thermal strain with head and neck cooling, it is conceivable that central fatigue may also be reduced, which may attenuate the decline in MVF during a sustained MVC following exercise in the heat. Racinais et al., (2008) observed no effect of head and neck cooling on central fatigue during a sustained contraction when hyperthermic; however, hyperthermia was induced passively, and rectal temperature remained < 39°C. Given the detrimental effects of hyperthermia on neuromuscular fatigue appear greater following exercise- vs. passively-induced hyperthermia (Périard et al., 2011), and greater at core temperatures > 39 °C vs < 39°C (Périard et al., 2014, Thomas et al., 2006), it is possible that the benefits of head and neck cooling may only be measurable at exercise-induced core temperatures > 39°C.

The aim of this study was to investigate the effects of cooling the head and neck whilst cycling in the heat to core body temperatures > 39°C on central and peripheral fatigue. We hypothesized that: (i) hyperthermia induced by cycling in the heat would augment neuromuscular fatigue due to greater central fatigue, but this would reduce peripheral fatigue; and (ii) that head and neck cooling during and after cycling in the heat would attenuate the greater central fatigue caused by hyperthermia, but at the expense of greater peripheral fatigue.

Methods

Participants

Fourteen healthy, physically active males volunteered to participate. Their mean (± SD) age, body mass, percentage body fat, stature, and relative peak oxygen uptake ($\dot{V}O_{2peak}$) were; 25.3 ± 3.2 years, 77.4 ± 11.0 kg, 15.9 ± 5.8%, 180.6 ± 6.6 cm, and 52.9 ± 5.8 $mL \cdot kg^{-1} \cdot min^{-1}$, respectively. Participants were informed of any risks and discomforts associated with the experiment before giving their written and oral informed consent. Participants visited the laboratory on five occasions (two familiarisations and three experimental sessions) at the same time of day, each separated by 7 ± 2 days. A health screening procedure was repeated prior to each laboratory visit to assess the health status of the participant (ACSM, 1998). All experimental procedures were approved by the Ethical Advisory Committee of the University of Roehampton and in accordance with the declaration of Helsinki.

Pre-experimental sessions

In the first familiarisation session participants had their stature and body mass recorded and underwent body composition assessment using air plethysmography (BodPod, Cosmed, Italy) before performing an incremental maximal power test (Kuipers et al., 1985) on a cycle ergometer (Monark, 874E, Monark, Vansbro, Sweden) to determine maximum power output (W_{max}) and $\dot{V}O_{2peak}$. Participants were then familiarised with the neuromuscular function measurements (isometric MVCs and electrically evoked involuntary contractions of the knee extensors of the preferred leg). The second familiarisation session was identical to the experimental session completed in hot environmental conditions without cooling (see *Experimental sessions*), i.e. participants exercised in the heat and performed the neuromuscular function measurements whilst in a hyperthermic state. This second familiarisation session was deemed necessary from our pilot testing, which showed several participants were unable to complete a HOT protocol without prior familiarisation with exercising in the heat.

Experimental sessions

Participants wore the same exercise attire (shorts and T-shirt) for each session and were asked to abstain from strenuous physical activity and alcohol consumption 24h prior to each experimental visit. Upon arrival at the laboratory, participants were seated in the strength testing chair (see *Force section*) for instrumentation of electromyography (EMG) and electrical stimulation of the femoral nerve (see *Electrical stimulation*). Participants completed a series of warm-up contractions at incremental intensities from 20 – 90% of maximum perceived effort, followed by four MVCs (separated by ~1 min to allow adequate recovery) in which they were instructed to push as ‘hard’ as possible for 3 – 5s. A single twitch and triplet contraction (see *Electrical stimulation section*), separated by 2s, were superimposed at the plateau of the force-time curve (~1s after contraction onset) during the second and fourth MVCs.

Following the MVCs, the participants remained at rest whilst a train of involuntary contractions were elicited, consisting of one twitch, one triplet, one twitch, and one triplet, each separated by 2s. This same train of four involuntary contractions was then used throughout the protocol where electrical stimulation occurred (Figure. 1).

All neuromuscular assessments were performed outside the environmental chamber, in a thermoneutral laboratory (~22°C). After completion of the initial (pre-cycling) neuromuscular assessments, participants emptied their bladders, recorded nude body mass (Seca, Robusta 813, Seca, Birmingham, UK), self-inserted a rectal thermistor, and moved into the walk-in environmental chamber (Weiss Technik Ltd, Wales, UK). When in the chamber, participants sat quietly in an upright position for ~5 min while being instrumented with skin thermistors. Once resting temperature, heart rate (HR), and perceptual measurements were recorded, participants began cycling at 50% W_{max} for 60 min, in one of three conditions (a different condition in each experimental session conducted in a randomized order); a thermoneutral control (CON; 18°C, 50% relative humidity (Rh)), hot (HOT; 35°C, 50% Rh), and HOT with head and neck cooling ($HOT_{cooling}$). Head and neck cooling was achieved through a customised water-perfused hood and neck cooling system with inlet water temperature set to 3°C (Active Ice and Cool Flow Cooling System, Polar Product Inc., USA).

Once the 60 min cycling bout was completed, participants put on an impermeable rain jacket to restrict heat loss before leaving the climatic chamber and returned to the isometric strength testing chair (located ~5 m from the walk-in environmental chamber). During $HOT_{cooling}$, participants continued to wear the head and neck cooling garments while performing the post cycling neuromuscular assessments. Participants were seated; securely fastened and re-instrumented as quickly as possible (transition time: ~5 min). Following this preparation, the stimulation train of four involuntary evoked contractions were elicited at rest to determine a change in baseline involuntary contractile properties following the cycle exercise (Figure. 1).

Five seconds after the last electrically evoked contraction, participants performed a sustained MVC in which they were instructed to push as hard as possible for 123s. The stimulation train was superimposed during the 123s MVC at 2, 27, 57, 87 and 117s (centre of the train coinciding with 5, 30, 60, 90, and 120s), and evoked again at rest 5s after the 123s MVC. Strong verbal encouragement was provided throughout, and participants were blinded to time during their efforts to avoid any pacing strategies. Refrigerated water was provided ad libitum throughout the trials.

Measurements

Force

All voluntary and involuntary isometric contractions of the knee extensors were conducted in a custom-built isometric strength testing chair (Maffiuletti et al., 2016). Participants were securely fastened with a waist belt and shoulder straps, with hip and knee angles fixed at 100º and 105º respectively (180° was full extension). An ankle strap, in series with a strain gauge load cell (Force Logic, FSB-1.5kN Universal Cell 1.5kN, Force Logic, Reading, UK), was secured 4 cm proximal to the medial malleolus with the load cell aligned perpendicular to the tibia during knee extension. The force signal was amplified (x375), interfaced with an analogue-to-digital converter (CED, Mirco3 1401, Cambridge Electrical Design, Cambridge, UK), and sampled at 2000 Hz with a personal computer using Spike2 software (Spike 2 Version 8, Cambridge Electrical Design, Cambridge, UK). Real-time biofeedback of the force response was provided on a 127 cm television screen, directly in front of the isometric strength testing chair.

EMG

Surface EMG signals were recorded from the rectus femoris (RF), vastus lateralis (VL) and vastus medialis (VM) (Noraxon, TeleMYO DTS, Noraxon, Arizona, USA). Following preparation of the skin (shaving, light abrasion and cleaning using 70% ethanol) two bipolar silver-silver-chloride gel-electrode configurations (2 cm diameter, and 2 cm inter-electrode distance; Noraxon, Dual Electrode, Noraxon, Arizona, USA) were placed: over the belly of each muscle (i.e., two EMG signals per muscle); in parallel to the presumed orientation of the muscle fibres; and at 60 ± 4% (RF1), 47 ± 3% (RF2), 74 ± 15% (VL1), 64 ± 9% (VL2), 83 ± 19% (VM1) and 75 ± 13% (VM2) of the distance from the greater trochanter to the lateral knee-joint space. Once attached to the skin the electrodes remained in place for the duration of the experimental trial, with placement conducted by the same investigator throughout all trials. Each EMG signal was amplified (x500; 10-500 Hz bandwidth) and sampled (2000 Hz) in synchronisation with force via the same analogue-to-digital converter utilising Spike2 software. In off-line analysis, the EMG signals were band-pass-filtered between 5 and 500 Hz using a fourth-order Butterworth digital filter and corrected for the 156 ms delay inherent in the Noraxon, TeleMYO DTS system. Signals collected during voluntary contractions were smoothed with a root mean squared (RMS) moving time window with a 500 ms epoch.

Electrical stimulation

Electrical square-wave pulses (0.2 ms duration) delivered over the femoral nerve (Digitimer, DS7AH Constant Current Stimulator, Digitimer, Hertfordshire, UK) were used to evoke twitch contractions, compound muscle action potentials (M-waves), and triplet contractions (3 pulses at 100 Hz). The anode (Rubber electrode 10 x 7 cm, EMS Physio Ltd, Oxfordshire, UK) was secured by surgical tape (Transpore 2.5 cm x 5 cm, 3M, UK) to the skin over the greater trochanter. The cathode stimulation probe (1 cm diameter tip; Digitimer, S1 Compex Motor PointPen, Digitimer, Hertfordshire, UK), which protruded 2 cm from the centre of a custom-built plastic base (4 x 3 cm) was placed over the femoral nerve in the femoral triangle. The greatest evoked peak twitch force in response to a submaximal current determined the precise placement of the cathode, where it was taped in place. The intensity of stimulation was then progressively increased, until there was a plateau in both twitch peak force and peak-to-peak M-wave amplitude (M_{max}) at each EMG site. This intensity was increased by a further 20% (supra-maximal) to ensure all stimulations were eliciting a maximal involuntary response and kept constant thereafter for all twitch and triplet contractions. The cathode position was marked on the skin with permanent ink prior to the 60 min of cycling to ensure accurate relocation in the post-cycling neuromuscular function assessment.

Skin and rectal temperature

To assess rectal temperature (T_{re}), a rectal thermistor (REC-U-VL30, Grant Instruments, Cambridge, UK) was self-inserted ~10 cm past the anal sphincter. Four skin thermistors (EUS-U-VL3-0, Grant Instruments, Cambridge, UK) were applied to the skin with a transparent dressing (Tegaderm, 6 x 7 cm, 3M, Minnesota, USA) and secured with surgical tape for the assessment of local skin temperature. Mean weighted skin temperature ($\overline{T}_{sk}$) was calculated from the four skin sites located on the right side of the body (suprasternal notch and one each on the belly of the following muscles, flexi carpi radialis, gastrocnemius and rectus femoris) using the equation of Ramanathan (1964). Mean neck skin temperature ($\overline{T}_{neck}$) was obtained from two thermistors placed either side of the spinal midline at approximately the 3rd/4th cervical vertebrae. All temperature measurements were recorded at: baseline immediately prior to the cycling, 5 min intervals during cycling, and immediately before and after the 123s MVC.

Perceptual measurements and heart rate

Rating of perceived exertion (RPE), whole body thermal sensation (TS), thermal sensation of the head and neck (TS_{neck}) and HR were recorded at the same time as temperature data. RPE was rated using a fifteen-point scale from 6 (at rest) to 20 (maximal exertion; Borg, 1982). Thermal sensation was rated using a nine-point scale from 0 (unbearably cold) to 8 (unbearably hot) with 4 as neutral (Young et al., 1987). HR was recorded with a heart rate monitor, secured with strap and worn by the participant in contact with the skin (Polar F3, Polar Electro, UK, Ltd).

Neuromuscular Data Analysis

Pre-cycling

Pre-cycling MVF was defined as the greatest voluntary (i.e., not due to superimposed twitch or triplet) force recorded in any of the MVCs performed prior to the 60 min cycling. To assess central drive at/near MVF, the CAR was determined as voluntary force at the point of triplet stimulation divided by the sum of voluntary force at triplet stimulation and superimposed triplet force (total muscle force; Kent-Braun & Le Blanc, 1996), and averaged across the two MVCs in which superimposed stimulation occurred. Central drive was also assessed from RMS EMG at MVF (or at the point closest to MVF without influence of artefact from electrical stimulation), normalised to the maximal M-wave (M_{max}; determined from the average of the two M-waves evoked during the MVCs), and averaged across the six EMG sites to give a value for the whole quadriceps muscle (EMG_{MVF}).

Stimulation at Rest

For each stimulation train elicited at rest (i.e., pre-cycling, pre-123s MVC, and post-123s MVC) the following variables were averaged across the two twitch or two triplet contractions in that stimulation train: M_{max} (from the twitch); triplet peak force (PF), triplet peak rate of force development (pRFD; determined with a 50 ms epoch) and triplet half-relaxation time (HRT).

123s MVC

CAR was averaged from the two superimposed triplets, and M_{max} from the two superimposed twitch contractions in each stimulation train elicited during the 123 s MVC (i.e., at 5, 30, 60, 90 and 120s). MVF and EMG_{MVF} were also recorded at 5, 30, 60, 90 and 120s, where EMG_{MVF} was obtained by normalising RMS EMG at the superimposed M_{max}, before averaging across the six EMG sites.

Statistical analyses

Descriptive data are reported as mean ± standard deviation (SD). Data were assessed for normality of distribution with the Sharipo-Wilk test. Two-way repeated measures ANOVAs evaluated the effect of condition by time on all dependent variables. Specifically, ANOVAs for: MVF, CAR, and EMG_{MVF} included 3 conditions (CON, $HOT_{cooling}$ and HOT) by 6 time points (baseline pre-cycling, and at 5, 30, 60, 90, and 120s during the 123s MVC). ANOVAs for triplet variables and M_{max} evoked at rest included 3 conditions x 3 time points (baseline pre-cycling, pre-123s MVC, and post-123s MVC). ANOVAs for TS, TS_{neck}, HR, T_{re}, $\overline{T}_{neck}$, and $\overline{T}_{sk}$ included 3 conditions by 13 time points (12 time points for RPE; baseline pre-cycling and 5 min intervals throughout cycling). Violations of sphericity were corrected for using the Greenhouse-Geisser adjustment when appropriate. Following a significant F value, pairwise differences between conditions were identified using stepwise Bonferroni-corrected paired t-tests, at each individual time point for all the above dependant variables. The significance level was set at $P < 0.05$. Statistical analysis was completed using SPSS version 21 (SPSS Inc., Chicago, IL). Cohen's Effect size (*d*) for paired comparisons were calculated (Cohen, 1988).

Results

Temperature

There was a main effect of time ($P < 0.001$) on T_{re}, $\overline{T}_{sk}$ and $\overline{T}_{neck,}$ which all increased throughout the cycling in all conditions. There were also main effects of condition and condition by time interaction effects on these variables ($P < 0.001$). T_{re} was lower in CON than HOT and HOT$_{cooling}$ after 30 min of cycling ($P < 0.05$; $0.38 \geq d \leq 7.96$; Figure. 2A), and $\overline{T}_{sk}$ was lower in CON than HOT and HOT$_{cooling}$ at all measured time points throughout the trial ($P < 0.05$; $0.37 \geq d \leq 7.38$; Figure. 2B). However, T_{re} and $\overline{T}_{sk}$ were similar throughout HOT and HOT$_{cooling}$ ($P > 0.05$; $0.01 \geq d \leq 0.65$). $\overline{T}_{neck}$ was similar in CON and HOT$_{cooling}$ ($P > 0.05$; $0.01 \geq d \leq 0.41$; Figure. 2C) at all measured time points, except at baseline where it was lower in CON ($P < 0.001$; $d = 2.11$), and post 123s MVC where it was lower in HOT$_{cooling}$ ($P = 0.004$; $d = 1.36$). $\overline{T}_{neck}$ was greater in HOT than both CON ($P < 0.05$; $3.42 \geq d \leq 6.66$) and HOT$_{cooling}$ ($P < 0.05$; $2.48 \geq d \leq 6.00$; Figure. 2C) at all measured time points.

Perceptual measures and Heart Rate

There was a main effect of time ($P < 0.001$) on TS and TS$_{neck}$, which both increased throughout all conditions. There were also main effects of condition ($P < 0.001$) for both variables, but not condition by time interaction effects ($P > 0.05$). TS was lower throughout CON than both HOT ($P < 0.05$; $0.73 \geq d \leq 3.03$) and HOT$_{cooling}$ ($P < 0.05$; $0.72 \geq d \leq 2.29$), but similar in HOT and HOT$_{cooling}$ ($P > 0.05$; $-0.84 \geq d \leq -0.03$) at all measured time points (Figure. 3C). On the other hand, TS$_{neck}$ was similar in CON and HOT$_{cooling}$ ($P > 0.05$; $0.04 \geq d \leq 0.85$), but lower in both these conditions compared to HOT ($P < 0.05$; $0.30 \geq d \leq 2.19$), at all measured time points (Figure. 3D).

RPE and HR were effected similarly by time ($P < 0.001$), condition ($P < 0.001$), and condition by time ($P < 0.05$). Specifically, RPE and HR increased throughout the cycling in all conditions but were both greater in HOT ($P < 0.05$; $0.56 \geq d \leq 2.34$) and HOT$_{cooling}$ ($P < 0.05$; $0.50 \geq d \leq 2.10$) than CON after the first 5 min, and similar for HOT and HOT$_{cooling}$ ($P > 0.05$; $0.01 \geq d \leq 0.20$) at all measured time points (Figure. 3A and 3B).

MVC Measures

There was a main effect of time ($P < 0.001$) on MVF, which declined throughout the 123s MVC in all conditions. There was also a main effect of condition ($P < 0.001$) and a condition by time interaction effect ($P = 0.043$). Whilst MVF was similar in all conditions pre-cycling ($P > 0.05$; $0.06 \geq d \leq 0.17$; Figure. 4A), it was 14-35% greater in CON than HOT ($P < 0.05$; $0.42 \geq d \leq 0.97$; Figure. 4A) at all measured time points during the 123s MVC post-cycling. Whilst MVF in CON was 9-38% greater than in $\text{HOT}_{\text{cooling}}$ at 5, 60, and 120s ($P < 0.05$; $0.27 \geq d \leq 1.34$), it was similar between these conditions at 30s ($P = 0.39$; $d = 0.23$) and 90s ($P = 0.74$; $d = 0.47$; Figure. 4A) into the 123s MVC. MVF in $\text{HOT}_{\text{cooling}}$ was 4-12% greater than HOT throughout the 123s MVF and although these differences were not significant at any time point ($P > 0.05$; $0.12 \geq d \leq 0.37$), there was a small beneficial effect at 30s ($P = 0.072$; $d = 0.32$).

Similar to MVF, there was a main effect of time ($P < 0.001$) on CAR, which decreased throughout the 123s MVC, condition ($P < 0.001$), and a condition by time interaction effect ($P = 0.017$). At baseline pre-cycling, CAR was similar between conditions ($P > 0.05$; $0.02 \geq d \leq 0.10$); however, during the 123s MVC post-cycling CAR was 10-30% greater in CON than HOT at all measured time points ($P < 0.05$; $0.63 \geq d \leq 1.01$; Figure. 4B). In contrast, CAR during the 123s MVC was only greater (6-24%) in CON than $\text{HOT}_{\text{cooling}}$ at 5, 60 and 120s into the 123s MVC ($P < 0.05$; $0.47 \geq d \leq 0.79$; Figure. 4B), but similar between these conditions at 30s ($P = 0.99$; $d = 0.20$) and 90s ($P = 0.174$; $d = 0.51$). Furthermore, CAR during the 123s MVC in $\text{HOT}_{\text{cooling}}$ was 4-15% greater than HOT at each time point, and this difference was statistically significant at 30s ($P = 0.04$; $d = 0.38$; Figure. 4B).

EMG_{MVF} was similar in all conditions at baseline pre-cycling ($P > 0.05$; $0.07 \geq d \leq 0.28$; Figure. 4C) but there was a main effect of time ($P < 0.001$) and EMG_{MVF} decreased throughout the 123s MVC. There was also a main effect of condition ($P < 0.001$), but no condition by time interaction effect (P = 0.27), caused by EMG_{MVF} in CON being greater than HOT at 5 and 30s ($P < 0.05$; $0.76 \geq d \leq 0.78$) and greater than $HOT_{cooling}$ at 5 and 60s ($P < 0.05$; $0.56 \geq d \leq 0.85$; Figure. 4C) during the 123s MVC. EMG_{MVF} in $HOT_{cooling}$ and HOT was similar throughout the 123s MVC ($P > 0.05$; $0.03 \geq d \leq 0.32$).

Resting Evoked Measurements

There was a main effect of time on PF, pRFD, and HRT ($P < 0.05$ for all). There was also a main effect of condition ($P < 0.05$) on these variables and a condition by time interaction effect for pRFD ($P < 0.001$). The pattern of change for PF, pRFD and HRT was similar for all three conditions. Specifically, PF ($P < 0.001$; $-1.60 \geq d \leq -1.25$) and pRFD ($P < 0.001$; $-1.35 \geq d \leq -0.54$), decreased, and HRT was unchanged from pre- to post-cycling, pre-123s MVC (P = 0.12; $-0.68 \geq d \leq -0.29$; Table 1). From pre- to post-123s MVC, PF decreased (P = 0.002; $-1.10 \geq d \leq -0.76$), and HRT increased ($P < 0.001$; $1.02 \geq d \leq 1.53$), whilst pRFD (P = 0.054; $-0.74 \geq d \leq -0.48$), was unchanged, in all conditions. Between conditions, PF, pRFD, and HRT were similar at baseline ($P > 0.05$; $0.01 \geq d \leq 0.16$). However, pRFD was lower in CON compared to either HOT or $HOT_{cooling}$ following cycling, both pre- ($P < 0.05$; $0.45 \geq d \leq 0.70$) and post- the 123s MVC ($P < 0.05$; $0.75 \geq d \leq 0.90$). PF was greater and HRT shorter in HOT compared with CON post-123s MVC (PF; $P < 0.05$; d = 0.47; HRT; $P < 0.05$; d = -1.01). No other differences between conditions were observed ($P > 0.05$).

There was a main effect of time ($P < 0.001$) on M_{max} at rest, which decreased progressively at each time point (pre-cycling, pre-MVC and post-MVC; Table 1). There was no main effect of condition (P = 0.73) or condition by time interaction (P = 0.18).

Discussion

The present study assessed the effects of head and neck per-cooling whilst cycling in the heat on central and peripheral fatigue during subsequent fatiguing exercise. As expected, cycling during compensable heat stress (T_{re} ~39.3°C at the start of the 123s MVC) resulted in greater declines in MVF associated with greater central fatigue (reduced CAR) during the 123s MVC following cycling, compared to CON. Our results provide some, albeit in-conclusive, evidence that head and neck cooling may attenuate the effects of hyperthermia on central fatigue. Whilst MVF during the 123s MVC in $HOT_{cooling}$ was not statistically different to HOT, it was statistically similar to CON at 30 and 90s. Furthermore, CAR was greater in $HOT_{cooling}$ than HOT at 30s and similar between $HOT_{cooling}$ and CON at 30 and 90s. The potential attenuation of central fatigue with head and neck cooling may be due to improved perception of thermal strain of the head and neck, evidenced by lower TS_{neck}, in $HOT_{cooling}$ compared with HOT, despite almost identical responses between these conditions in perceived (TS) and actual (T_{re}) thermal strain, cardiovascular strain (HR), and RPE. Interestingly, whilst evoked PF and HRT were similar between all three conditions pre- the 123s MVC; immediately after the 123s MVC, PF was lower and HRT longer in CON compared to HOT but similar between CON and $HOT_{cooling}$. This suggests there was lower peripheral fatigue in HOT but not $HOT_{cooling}$, compared to CON, likely due to the greater central fatigue and thus lower forces in HOT.

As reported elsewhere (Tyler & Sunderland, 2011b, Tyler et al., 2010, Tyler & Sunderland, 2011a, Sunderland et al., 2015), cooling the head and neck had no effect on physiological (T_{re}, $\overline{T}_{sk}$, HR) or whole body perceptual (TS, RPE) strain and exertion, which were similar between HOT and $HOT_{cooling}$, and greater in both HOT conditions compared to CON. However, the head and neck cooling was effective at reducing $\overline{T}_{neck}$ and TS_{neck} to CON values in $HOT_{cooling}$

at all time points except baseline, where $\overline{T}_{neck}$ was lower in CON, and post MVC when $\overline{T}_{neck}$ was lower in HOT$_{cooling}$. Reductions in $\overline{T}_{neck}$ and TS$_{neck}$ with head and neck cooling when exercising in the heat have been shown to benefit endurance performance (Tyler & Sunderland, 2011b), so it is conceivable they may have benefited neuromuscular performance in the current study.

Both MVF and CAR were similar between conditions at baseline, pre-cycling, but both decreased following the cycling, at 5s into the 123s MVC in all conditions. Thus, the cycling induced central fatigue, which likely contributed to the decline in MVF, in all conditions. However, both MVF and CAR at 5s into the 123s MVC post-cycling were greater in CON than either HOT or HOT$_{cooling}$, demonstrating greater central fatigue and thus a greater reduction in MVF, induced by hyperthermia. Furthermore, MVF and CAR continued to decline throughout the 123s MVC in all conditions but remained greater in CON than HOT at all measured time points, and greater in CON than HOT$_{cooling}$ at 5, 60, and 120s. These results are consistent with previous studies showing greater central fatigue causing greater reductions in MVF in hyperthermic vs. control conditions, where hyperthermia was induced either actively (Nybo & Nielsen, 2001a, Périard et al., 2014, Périard et al., 2011) or passively (Todd et al., 2005, Périard et al., 2014, Racinais et al., 2008, Périard et al., 2011). However, in the current study, MVF and CAR were similar between HOT$_{cooling}$ and CON at 30 and 90s and there were small effects for them to be larger in HOT$_{cooling}$ compared to HOT at 30s, during the 123s MVC. While these effects were small, the authors acknowledge there is some variability to the data, and it is not clear why per-cooling had an effect specifically at 30 and 90s during the sustained isometric contraction and not at other discrete time-points. It is plausible, however, over time during a long-distance event the cumulative small effect of per-cooling could accumulate to provide some benefit to performance.

The mechanisms of increased central fatigue in hyperthermic conditions are thought to be multifaceted with increases in brain temperature (Caputa et al., 1986), reductions in cerebral blood flow (Nybo & Nielsen, 2001b), inability to increase motor unit firing rate to accommodate faster muscle relaxation (Todd et al., 2005), and reductions in cerebral dopamine (Meeusen & Roelands, 2017) all potential contributing factors . Hyperthermia progressively impairs neuromuscular performance (Morrison et al., 2004) but the present study shows that cooling the head and neck may attenuate this reduction without effecting core body temperature. The exact mechanisms of the improved neuromuscular performance with head and neck cooling remain unclear but may be associated with reducing the temperature of the carotid blood destined for the brain (Zhu, 2000); however, others have suggested direct cooling of the brain is unlikely (Nybo et al., 2002). In the present study, improved thermal sensation of the head and neck from cooling, may have attenuated typical hyperthermia-induced reductions in brain activity (Xue et al., 2018), cortical somatosensory processing (Nakata et al., 2017) and/or dopamine neuron activation (Hasegawaa et al., 2000). In addition, head cooling can protect some functions of cognition in the heat (Racinais et al., 2008) and collectively, these factors may attenuate reductions in arousal (Nielsen et al., 2001). An increased state of arousal by alleviating local thermal sensation, could have translated into higher levels of motivation and greater voluntary neural activation of the central nervous system.

EMG_{MVF} (normalised to M_{max}) decreased throughout the 123s MVC in all conditions, which is consistent with the declines in CAR, and demonstrates central fatigue during the sustained contractions. However, the condition effects on normalised EMG_{MVF} were not as noticeable as they were for CAR. Specifically, EMG_{MVF} was only greater in CON than HOT (5 and 30s) or $HOT_{cooling}$ (5 and 60s) at 2/5 time points during the 123s MVC and was similar between HOT and $HOT_{cooling}$ at all measured time points. Périard et al., (2014) also reported more noticeable effects of hyperthermia on CAR compared to EMG_{MVF} normalised to M_{max}, observing greater reductions in CAR during a sustained contraction in hyperthermia vs. control conditions, but no condition effects on normalised EMG_{MVF}. Thus, EMG amplitude does not appear to be as sensitive as CAR to the effects of hyperthermia (or head and neck cooling when hyperthermic) on central drive during fatiguing exercise. This is likely due to the large variability inherent in EMG amplitude (Buckthorpe et al., 2012), in spite of the steps taken in the current study to improve reliability, such as recording EMG amplitude from two distinct sites on each muscle (Balshaw et al., 2017), and normalising EMG amplitude to M_{max} (Buckthorpe et al., 2012).

In addition to central fatigue, peripheral fatigue was also induced in all conditions, with decreases in evoked PF and pRFD from baseline to pre-123s MVC, following the cycling. Peripheral fatigue increased during the 123s MVC, as evidenced by the further declines in PF and the increase in HRT from pre- to post 123s MVC, in all conditions. These are typical responses known to occur in fatiguing exercise, due to metabolic perturbation interrupting excitation-contraction coupling (Allen et al., 2008). The effects of such metabolic perturbation on pRFD were mitigated in the HOT conditions, as evidenced by greater pRFD in HOT and $HOT_{cooling}$ compared to CON, both pre- and post- the 123s MVC, likely due to the higher muscle temperatures which are thought to improve the rate of myosin-actin cross bridge attachment (de Ruiter et al., 1999). The similar PF and HRT between conditions both pre-cycling and pre-123s MVC following the cycling, suggest the cycling induced similar peripheral fatigue in all conditions. However, post- the 123s MVC, PF was greater and HRT shorter in HOT, but not $HOT_{cooling}$, compared to CON. Thus, the 123s MVC induced less peripheral fatigue in HOT than CON, likely due to the greater central fatigue during the MVC in HOT, resulting in less force output and thus logically less metabolic perturbation. Furthermore, whilst head and neck cooling mitigated the effects of hyperthermia on central fatigue during the MVC, this appears to be at the expense of greater peripheral fatigue given the similarities in peripheral fatigue between $HOT_{cooling}$ and CON. Work from Amann and colleagues (Amann & Dempsey, 2008) suggests that during fatiguing self-paced exercise, central drive to the muscles is inhibited to limit peripheral fatigue to a task and individually specific critical threshold. Based on the results of the current study, we speculate that hyperthermia lowers this critical threshold of peripheral fatigue, though head and neck cooling may override this mechanism.

The M_{max} evoked at rest declined in all conditions from pre- to post-cycling and declined further from pre- to post-123s MVC. A decline in M_{max} with fatiguing exercise is well documented (Allen et al., 2008) and likely reflects an efflux of cellular K^+ from the muscle fibres causing reduced muscle fibre excitability (Clausen et al., 2004). However, there were no condition effects (i.e., no effects of hyperthermia) on M_{max}, which is inconsistent with studies showing M_{max} to decrease with increased muscle or whole-body temperature (Périard et al., 2014, Racinais et al., 2008, Dewhurst et al., 2005), possibly due to reduced muscle fibre depolarisation time and associated decrease in cellular Na^+ influx (Rutkove, 2001). It is possible the effects of fatiguing exercise on the M_{max} in the three conditions of the current study may have masked any subtle effects of temperature on M_{max}, and thus further research is required to better understand these mechanisms and their interactions.

One possible limitation of the current study was the need to assess neuromuscular function outside of the environmental chamber in temperate conditions. Core body temperature (T_{re}) was stable within each condition during the 123s MVC (Figure. 2A) but there was a decline in $\overline{T}_{neck}$ (-2.4 ± 1.1°C) and TS_{neck} (-1.7 ± 1.4) in HOT during the 123s MVC. $\overline{T}_{neck}$ and TS_{neck} remained higher in HOT (35.0 ± 1.1°C; 5.8 ± 1.5; CON: 34.0 ± 0.6°C; 5.1 ± 0.8; $HOT_{cooling}$: 32.5 ± 1.1°C; 3.8 ± 0.7); however, because the effectiveness of any cooling intervention is dependent on the interaction between the magnitudes of cooling provided and thermal strain experienced (for meta-analysis see Tyler et al., 2015) the natural reductions observed may have masked some of the cooling benefits.

In conclusion, our results provide evidence that head and neck cooling may attenuate some of the greater neuromuscular fatigue caused by hyperthermia, likely due to reduced central fatigue, although effects were small and not observable at all measured time points during a fatiguing activity. We also found that the greater central fatigue in hyperthermic conditions appears to reduce peripheral fatigue, but this response is mitigated with head and neck cooling.

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Compliance with ethical standards

Conflicts of interest

The authors declare that they have no conflict of interest.

Funding

The authors have no funding to declare.

Research involving human participants

Ethical approval

All procedures performed in this study involving human participants were in accordance with the ethical standards of the Ethical Advisory Committee of the University of Roehampton and in accordance with the 1964 Helsinki declaration and its later amendments.

Informed consent

Informed consent was obtained from all individual participants included in the study.

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Table

Table 1. Evoked triplet properties (PF (peak force); pRFD (peak rate of force development); TPT (time to peak tension); HRT (half-relaxation time)) and maximal M-wave (M$_{max}$) recorded at different time points (pre-60 min cycling and pre- and post- a 123s MVC) in three environmental conditions: control (CON), hot (HOT) and HOT with head and neck cooling (HOT$_{cooling}$). Data are means ± SD (*n* = 14).

	PF (N)	**pRFD (N·s^{-1})**	**HRT (ms)**	**M$_{max}$ (mV)**
Pre-cycling				
CON	432 ± 82	12060 ± 3475	75.0 ± 22.5	3.6 ± 0.9
HOT$_{cooling}$	440 ± 65	11601 ± 3400	71.4 ± 22.3	3.6 ± 1.0
HOT	437 ± 70	11637 ± 2864	71.5 ± 23.4	3.7 ± 0.9
Pre-123s MVC				
CON	324 ± 77 †	8024 ± 2413 †	69.2 ± 17.1	3.1 ± 0.8 †
HOT$_{cooling}$	326 ± 77 †	9214 ± 2891 †*	63.7 ± 16.9	3.0 ± 0.8 †
HOT	342 ± 81 †	9994 ± 3188 †*	58.7 ± 12.6	2.9 ± 0.7 †
Post-123s MVC				
CON	253 ± 51 †‡	6553 ± 1467 †	97.3 ± 19.7 ‡	3.1 ± 1.0 †‡
HOT$_{cooling}$	265 ± 57 †‡	7874 ± 2021 †*	84.3 ± 23.2 ‡	2.8 ± 0.8 †‡
HOT	283 ± 75 †‡*	8563 ± 2798 †*	78.8 ± 16.6 ‡*	2.8 ± 0.7 †‡

†; within condition paired differences ($P < 0.05$), different from pre-cycling

‡; within condition paired differences ($P < 0.05$), different from pre- 123s MVC

*; between condition paired differences at the same time point ($P < 0.05$), different from CON

Figure Legends

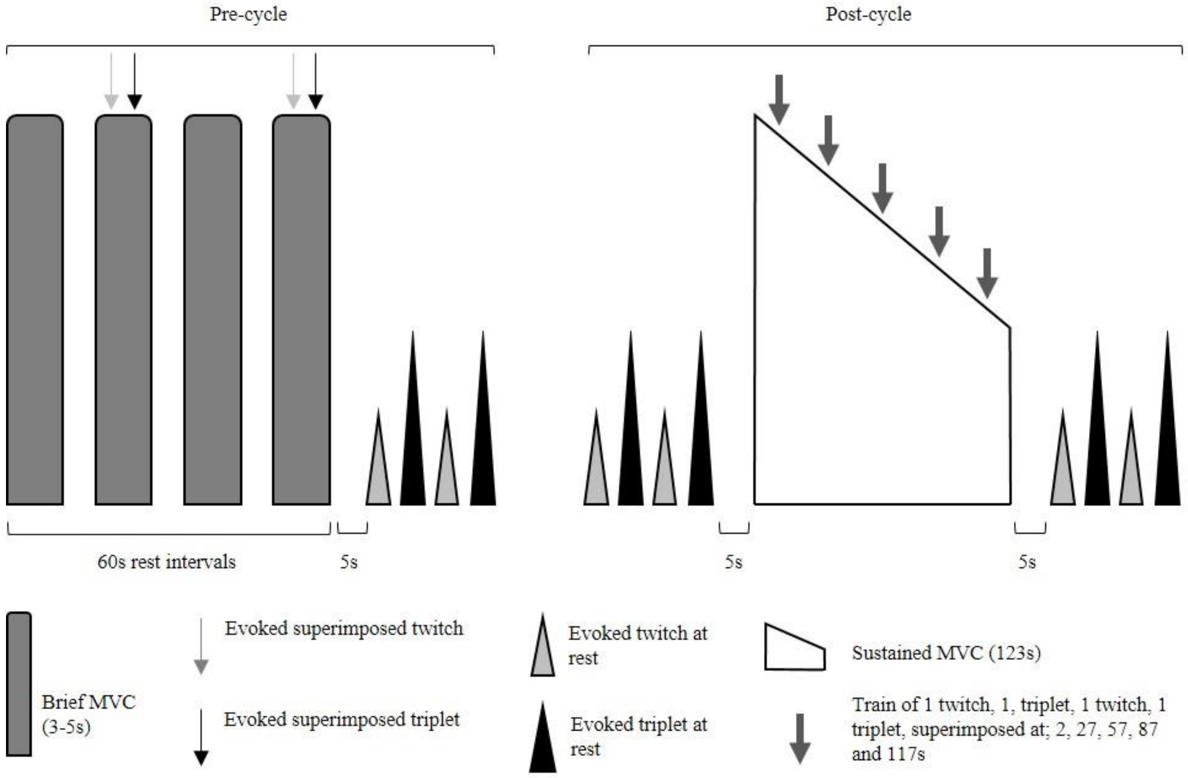

Figure. 1. Schematic of the protocol conducted in three separate environmental conditions in 50% relative humidity: hot (HOT, 35°C), HOT with head and neck cooling (HOT$_{cooling}$), and control (CON, 18°C). Participants cycled for 60 min on a cycle ergometer at 50% $\dot{V}O_{2peak}$, between pre- and post-cycle assessments.

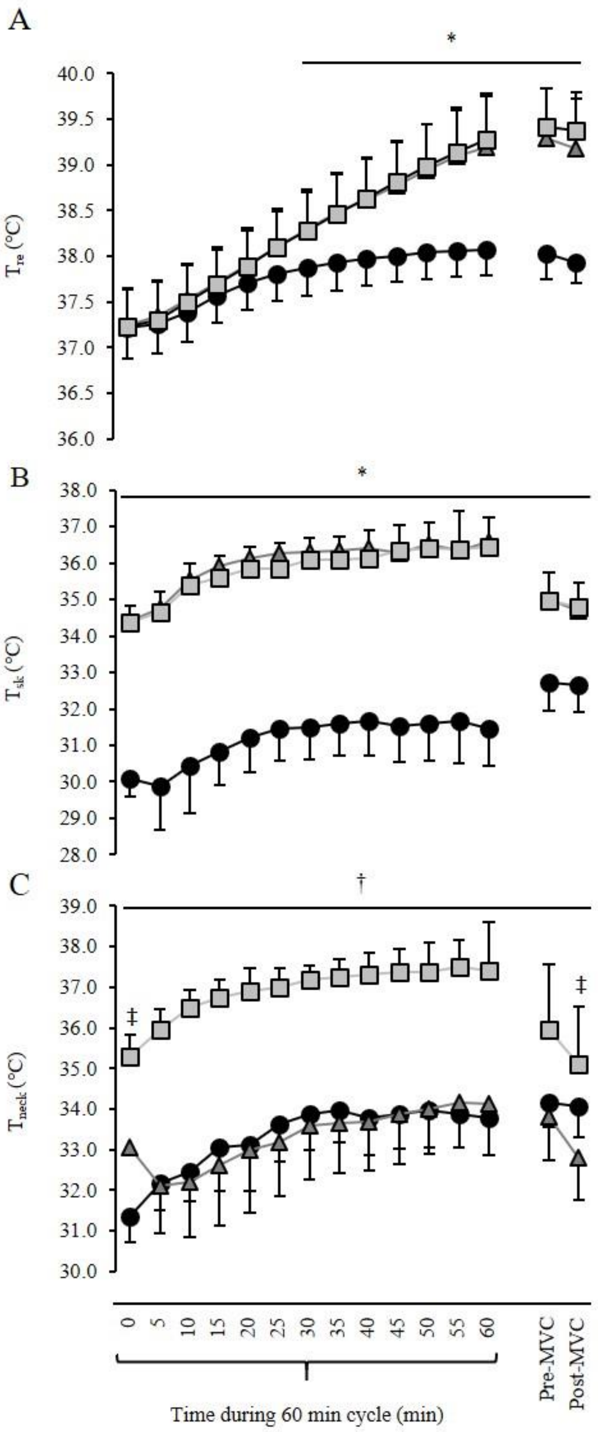

Figure. 2. Rectal (A), skin (B), and neck temperatures (C) recorded during and post 60 min of cycling in three separate environmental conditions: hot (HOT; light grey squares), HOT with head and neck cooling ($\text{HOT}_{\text{cooling}}$, dark grey triangles), and control (CON, black circles). Data are mean ± SD (*n* = 14). Between condition paired differences are denoted by * (CON < HOT and $\text{HOT}_{\text{cooling}}$), † (CON and $\text{HOT}_{\text{cooling}}$ < HOT), and ‡ (CON different from $\text{HOT}_{\text{cooling}}$).

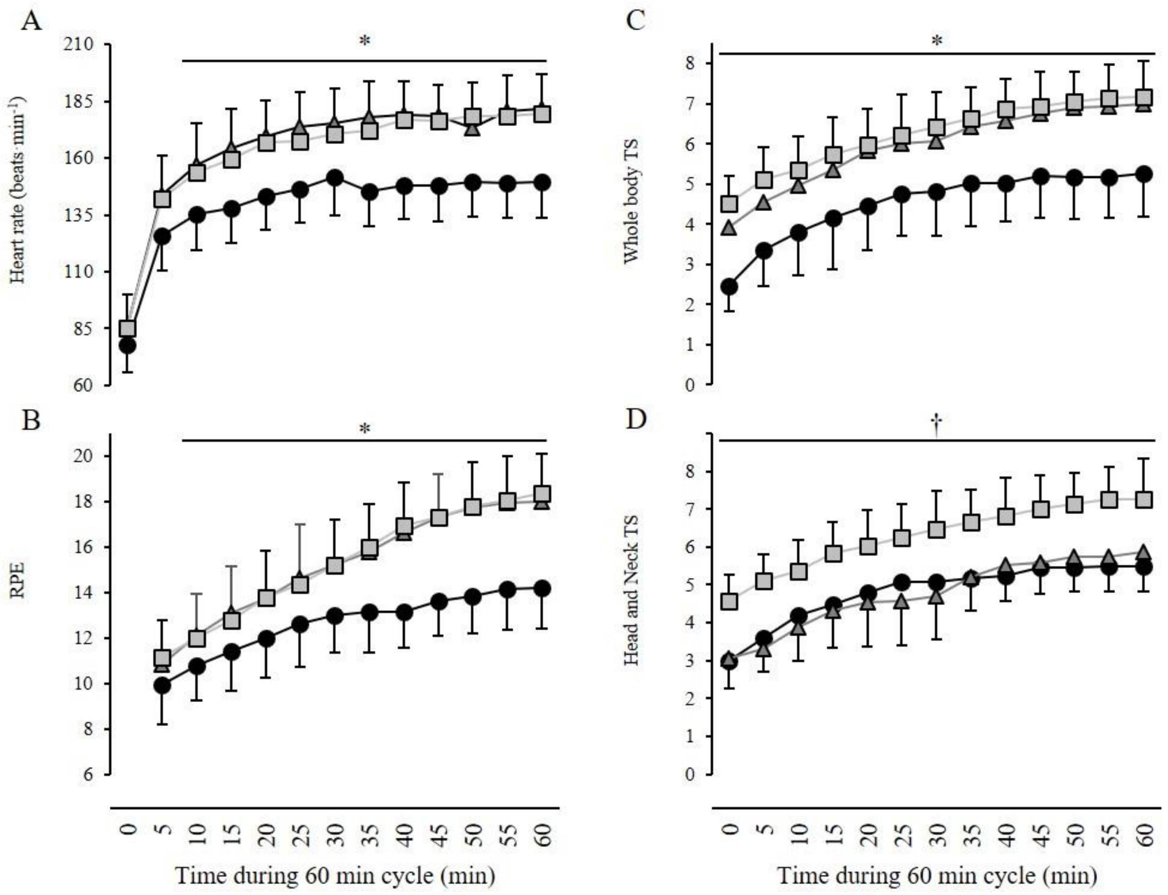

Figure. 3. Heart rate (A), ratings of perceived exertion (RPE; B), whole body thermal sensation (TS; C) and head and neck TS (D), during 60 min of cycling at 50% $\dot{V}O_{2peak}$ in three separate environmental conditions: Hot (HOT; light grey squares), HOT with head and neck cooling ($HOT_{cooling}$; dark grey triangles), and control (CON black circles). Data are mean ± SD *(n* = 14). Between condition paired differences are denoted by * (CON < HOT and $HOT_{cooling}$) and † (CON and $HOT_{cooling}$ < HOT).

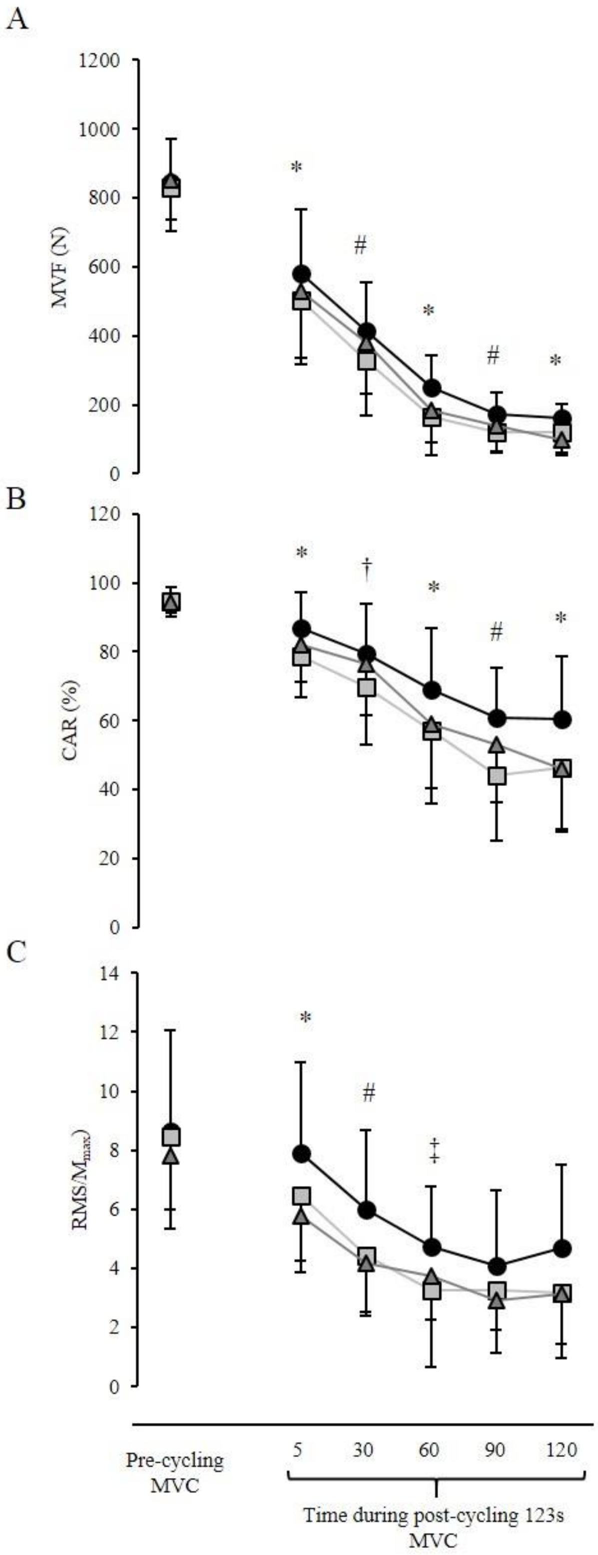

Figure. 4. Maximal voluntary force (MVF; A), central activation ratio (CAR; B) and normalised EMG amplitude at MVF (EMG_{MVF}, C), of the knee extensors pre-, and during a 123s MVC immediately post- a 60 min cycle in three separate environmental conditions: Hot (HOT, light grey squares), HOT with head and neck cooling ($HOT_{cooling}$, dark grey triangles), and control (CON, black circles). Data are mean ± SD (*n* = 14 except for EMG_{MVF} where *n* = 13). Between condition paired differences are denoted by * (CON > HOT and $HOT_{cooling}$), # (CON > HOT), † (CON and $HOT_{cooling}$ > HOT), and ‡ (CON > $HOT_{cooling}$).