

The effect of exercise induced hyperthermia on muscle fibre conduction velocity during sustained isometric contraction

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1 INTRODUCTION

2

3 It is likely that a major cause of fatigue during endurance exercise in a hot
4 environment is reduced power output which is regulated by the central nervous
5 system (Hunter et al., 2002c; Nybo 2007). It has also been suggested that this
6 fatigue acts as a protective mechanism to protect the body from **extreme**
7 damage which could occur with excessive **heat retention**. In support of this it
8 has been shown that voluntary muscular activation is reduced during
9 hyperthermic conditions and a greater rate of fatigue is observed during a
10 sustained isometric contraction (Nybo and Nielsen 2001). This increased fatigue
11 was suggested to be caused by a reduction in global **neural activation** as shown
12 by a decline in EMG amplitude. However, an additional important neuromuscular
13 control factor is muscle fibre conduction velocity (MFCV) which is a **potential**
14 indicator of both **central factors such as motor unit recruitment** and
15 **peripheral factors such as fibre membrane properties** (Andreassen and
16 Arendt-Nielsen 1987). It has been previously demonstrated that passively heated
17 muscle results in elevated MFCV (Farina et al., 2005; Gray et al., 2006). It has
18 been proposed that this occurrence is as a result of the higher temperature
19 accelerating opening and closing of the voltage-gated Na⁺ channels which allows
20 less Na⁺ to enter the cell (Rutkove et al., 1997). Consequently, action potential
21 amplitude and duration declines resulting in an increased capacity for the
22 commencement of depolarization which produces faster MFCV. However, during
23 prolonged submaximal exercise **at a fixed intensity** in the heat there will be an

1 increased accumulation of lactate (Galloway and Maughan 1997) from an
2 increase rate of muscle energy metabolism (Edwards et al., 1972; Febbraio et
3 al., 1996). This increase in lactate concentration will result in a greater decline in
4 extracellular pH (Fitts 1994) which will result in a concomitant decline in MFCV
5 (Brody et al., 1991). Recently, we (Hunter et al., 2009) manipulated pH by
6 inducing alkalosis and following prolonged submaximal exercise showed an
7 increase in MFCV during a sustained isometric contraction when compared to
8 placebo ingestion. Therefore, it is likely that accumulation of lactate from
9 submaximal exercise in the heat will indirectly attenuate the increased muscle
10 temperature effect on MFCV.

11

12 It would therefore appear that there are inherently contradictory responses in
13 MFCV during exercise in the heat which may cause it to increase but with a
14 greater accumulation in lactate will conversely produce slower values. This is an
15 important factor to consider when inducing hyperthermia by exercising in a hot
16 environment as opposed to just passive heating. Todd et al (2005) induced
17 hyperthermia by submerging subjects in a warm bath and found that the elbow
18 flexors produced less **absolute** force **with a greater decline** during a sustained
19 **2 minute maximal** isometric contraction. The authors concluded that greater
20 central fatigue was observed **during this contraction** despite a faster **rate of**
21 **motor neuron discharge into the** muscle. Therefore, exercise associated
22 changes will have implications for neuromuscular control strategies, which would
23 need to take into account the level of change in MFCV when delivering activity-

1 controlling coded action potentials to the peripheral muscle. The situation is
2 made more complex from a control perspective in that, as described above, there
3 are two different strategies available for regulating peripheral muscle activity
4 namely; 1) peripheral alterations in the conducting properties of the muscle
5 fibres; and/or 2) altering global recruitment strategy to all or some of the motor
6 units controlling skeletal muscle function. As far as we are aware no study has
7 explored the effect of exercise induced hyperthermia on the relationship between
8 RMS, MFCV and force during isometric fatigue.

9

10 Accordingly, the aim of this study was to determine the effect of inducing
11 hyperthermia by using a submaximal cycle protocol in a hot environment which
12 will increase lactate concentration and heat storage. We therefore used **two**
13 **main** interventions to determine this effect: 1) cycling in a hot environment; and
14 2) cycling in a thermoneutral environment. **In addition to control for the effect**
15 **of cycling in a hot environment we had a third intervention which was**
16 resting in a hot environment. Following this, the relative change of both RMS and
17 MFCV during a sustained maximal isometric fatiguing contraction was observed;
18 in order to determine which neuromuscular recruitment strategy operates
19 principally in controlling peripheral muscle activity in a hyperthermic environment.

20

21

22

1 METHODS

2

3 Seven, healthy, well trained club level cyclists volunteered for this study. The
4 mean (\pm SD) age, $\dot{V} O_{2max}$, height and **mass** of the subjects were 35 ± 9.9 years,
5 57.4 ± 6.6 ml kg⁻¹.min⁻¹, 178.6 ± 6.6 cm and 78.4 ± 9.6 kg respectively. All
6 subjects gave their written informed consent. The study was performed according
7 to the Declaration of Helsinki and was approved by the local research ethics
8 committee.

9

10 *Preliminary testing*

11 To determine peak power output (PPO), a modified protocol as described by
12 Hawley and Noakes (Hawley and Noakes 1992) was used. Subjects performed a
13 10-minute warm up on an electrically braked cycle ergometer (Lode, Groningen,
14 Netherlands). The starting power output was determined by multiplying the
15 subject's body weight by 2.5 W. The load was subsequently increased every
16 150s by first 50 W and then 25 W until the subjects were unable to maintain force
17 output or pedaling frequency dropped from 90 to < 50 revolutions. min⁻¹. PPO
18 was defined as the last completed work rate in watts plus the fraction of time
19 spent in the final non-completed work rate multiplied by 25 W.

20

21 *Experimental Procedure*

22 After the preliminary testing each subject reported to the laboratory on **four**
23 separate occasions one week apart, and were instructed to record their dietary
24 intake and physical activity 24 hours before the first visit. The subjects were then

1 instructed to replicate these conditions for the subsequent visits. During the first
2 visit, subjects familiarized themselves with the equipment and laboratory
3 conditions. Thereafter, they completed a familiarization trial by performing muscle
4 function tests before and after the 50 minute cycle ride at 60% of their PPO
5 (Figure 1). The cycle ride during the familiarization trial was in the hot condition of
6 40°C and 35% humidity to ensure that all subjects could complete the full 50
7 minute duration. The subjects then had to return to the laboratory on three
8 subsequent occasions where in random order they were required to either cycle
9 (HOT) or rest (PASS) in 40°C and 35% humidity or cycle in 19°C and 20%
10 humidity (NEUTRO) (Figure 1). At each visit the subjects arrived at the laboratory
11 at the same time of day and their nude body weight was recorded, resting blood
12 samples taken and rectal thermometer (Mon-a-therm, Mallinckrodt, OH, USA)
13 inserted 10cm beyond their anal sphincter. Following this, the subjects were then
14 prepared for the recording of muscle temperature by lying them down in the
15 supine position and injecting 5ml of local anesthetic into the mid distal section of
16 the Vastus Lateralis muscle with the flexed lower limb. After allowing a 5 minute
17 period a needle temperature probe was inserted (Fluke 80PK-5A Type K, Fluke
18 Corporation, USA) 5cm into the same position to record the temperature (Fluke
19 52, Series II thermometer recorder, USA). A surface thermistor (YSI 400, Yellow
20 Springs, OH, USA) was then attached to the Vastus Lateralis muscle for the
21 recording of skin temperature. The subjects then completed three maximal
22 voluntary contractions (MVC), the highest of which was used to normalize
23 subsequent EMG amplitude (RMS) recordings. Following this a further blood

1 sample was taken before embarking on a 50 minute cycle on the electrically
2 braked ergometer (Excalibur Sport, Lode, The Netherlands) at 60% of PPO,
3 during which heart rate, sEMG, $\dot{V} O_2$ and rating of perceived exertion (RPE)
4 where recorded every 10 minutes. Upon completion of the cycle ride **the muscle**
5 **temperature recording procedure was then repeated followed** by a 100
6 second sustained isometric contraction (SMC) (Figure 1). **The total time taken**
7 **from completion of the cycle ride to the start of SMC was approximately ~4**
8 **minutes. To undertake this contraction protocol**, subjects were instructed to
9 contract maximally at the commencement of producing force and attempt to
10 sustain it for the full duration of the 100s trial.

11

12 *Torque Measurement*

13 The strength of the subjects' right knee extensors was measured on an isokinetic
14 dynamometer (Biodex Medical Systems USA) as described previously (Hunter et
15 al., 2002b). Subjects sat on the dynamometer with their hips, thighs and upper
16 bodies firmly strapped to the seat. In this position their hip angle was 100° angle
17 of flexion. The right lower leg was attached to the arm of the dynamometer at a
18 level slightly above the lateral malleolus of the ankle joint and the axis of rotation
19 of the dynamometer arm aligned with the lateral femoral condyle. The
20 dynamometer arm was set at angle of 60° from full leg extension. Each subject
21 performed 3 x 5 second MVC's with a minute recovery in between. The highest
22 torque recorded from these 3 MVC's was used for subsequent analyses.

1 Following the cycle the subjects then performed the SMC using the same torque
2 measurement positions for all three conditions.

3

4 *Electromyography analyses*

5 Four Ag-AgCl EL258S shielded electrodes (Biopac, USA) were inserted into a
6 hard plastic mould in a straight line next to one another. This allowed a distance
7 of 12.5mm between each electrode from the signal detection area and was
8 configured to record 3 parallel EMG signals as described by Lowery et al (Lowery
9 et al., 2002). The electrode array was then positioned on cleansed and shaven
10 skin along the major axis of the muscle fibres half way between the main belly of
11 the Vastus Lateralis and its distal end. Initially the electrodes were inserted with
12 dry round silver inserts for ease of multiple placements. A variety of different
13 locations on the muscle was used until there was a clear propagation in one
14 direction of the action potentials without change in shape all of the 3 sEMG
15 signals. Then the electrode array position was marked with a permanent marker
16 pen, after which the dry silver inserts were subsequently removed and filled with
17 conductive gel (20-30 μ l) and returned and attached to the marked section of
18 skin. The electrode array was firmly secured with 2 sections of Tegaderm. This
19 electrode array was linked to the BioPac EMG apparatus (Biopac Systems, USA)
20 and host computer. The EMG data **were** automatically anti-aliased by the
21 hardware (Biopac Systems, USA). Each activity was sampled at a 2000 Hz
22 capture rate. The raw signal was processed to give root mean square (RMS) of

1 the sEMG power, which was used for subsequent analyses. All post cycle RMS
2 signals were normalized to the pre intervention MVC.

3

4 MFCV was estimated by applying a cross-correlation function between the
5 temporal sEMG signals measured at the three electrodes as previously
6 described (Lowery et al., 2002). The cross-correlation of two signals x and y is
7 given by

8

$$9 \quad R_{xy}(m) = E\{x_{n+m}y_n^*\} = E\{x_n y_{n-m}^*\}$$

10

11 where $E\{\cdot\}$ is the expectation operator. The cross-correlation function of two
12 similar signals will peak where the two signals are maximally similar. In our case
13 the signals are pulsed signals where one signal is a delayed and path distorted
14 version of the other signal, so that their cross-correlation should peak at a
15 number of samples equivalent to the time-delay between them. We make use of
16 this property to estimate the delay between the signals measured at the
17 electrodes 1 and 2, 2 and 3 and 1 and 3. With the distance between the
18 electrodes known, we found the velocities of the signals from:

19

$$20 \quad \text{velocity}_{nm} = \text{electrode pair distance}_{nm} / \text{estimated time delay}_{nm} \quad (\text{m/s})$$

21

22 The 3 sEMG signals were first processed through 2 double differential (DD)
23 amplifiers for the final MFCV estimation. Both DD signals were upsampled to

1 20kHz and an extra 0.5 seconds either side of the epochs are upsampled to
2 avoid possible end-effects from using the Matlab interpft function. MFCV was
3 estimated using the xcorr function of Matlab where xcorr estimates the cross-
4 correlation sequence of a random process. The maximum cross-correlation is
5 noted as the delay for the signal to travel from one electrode to another as
6 determined from the upsampled DD signals. Estimates of MFCV were accepted
7 only when cross-correlation values were higher than 0.8.

8

9 *Blood sampling*

10 An 18-gauge Teflon cannula (Jelco, Johnson and Johnson, Halfway house,
11 South Africa) was positioned in an antecubital vein and connected to a three way
12 stop cock (Uniflex, Mallinckrodt, Hennef-Seig, Germany). This cannula was
13 flushed periodically with 2-3 ml of sterile saline containing heparin (5 IU ml^{-1}) and
14 was used for the collection of venous blood samples (10 ml) at rest and during
15 exercise. Venous blood samples (10ml) were drawn at rest, at the end of each 15
16 min work rate and at exhaustion. The samples were then divided into aliquots,
17 which were put into an ice-cold tube containing potassium oxalate and sodium
18 fluoride for later determinations of lactate concentrations. The tubes were
19 centrifuged at 3000-x g for 10 minutes at 4°C immediately after the completion of
20 the trial and the supernatants were stored at -20°C for later analyses of plasma
21 lactate. Plasma lactate concentrations were measured with spectrophotometric
22 (Beckman Model 35, Beckman Instruments Inc., Fullerton, Ca, USA) enzymatic
23 assays (Lactate PAP, BioM (rioux, Lyon, France; NEFA half-micro test;

1 Boehringer Mannheim, Germany). This procedure was the same as described
2 previously (Hunter et al., 2002a).

3

4 *Recordings of heart rate and perceived exertion*

5 Heart rate was recorded at rest and then recorded along with **rating** of perceived
6 exertion (RPE) (Borg 1973) every 10 minutes for the full 50 minute ride (Figure
7 1).

8

9 *Statistical Analyses*

10 All data are expressed as means \pm SD. A (time-by-trial) repeated measures

11 ANOVAs were performed to evaluate differences between and within trials.

12 **These data were** analyzed by: 1) 3 (condition) x 5 (time [**25s epochs**]) for SMC;

13 2) % delta change for each variable throughout SMC 3 (condition) x 3 (**variables**

14 [**torque, RMS and MFCV**]) and; 3) a 3 (condition) x 2 (time [**pre and post**]) for

15 the 50 minute intervention. Post hoc analyses of the main effect of time were

16 done using a Tukey's **HSD**. Significance was accepted at $P \leq 0.05$.

17

1 RESULTS

2

3 ***Sustained Maximal Contraction***

4 Delta change over the SMC for all 3 conditions and variables showed a
5 significant ($p < 0.01$) difference between the conditions with a significant
6 interaction effect (Figure 2). Post hoc analyses showed that within HOT both
7 torque and RMS declined by ~37% but MFCV was reduced significantly ($p < 0.05$)
8 less by just ~9% (Figure 2). While within NEUTRO (torque: ~21%; RMS: ~36%,
9 MFCV: ~20%) and PASS (torque: ~10%, RMS: ~20% MFCV: ~17%) no
10 statistical differences were shown between the decline in variables.

11

12 Torque significantly ($p < 0.01$) declined for all three conditions at significantly
13 ($p < 0.01$) different rates with reductions of ~39% for HOT, ~22% for NEUTRO and
14 ~4% for PASS (Figure 3A). This resulted in significantly ($p < 0.01$) different final
15 torque values between conditions with HOT being the lowest followed by
16 NEUTRO and then by PASS (Figure 3A). MFCV significantly ($p < 0.01$) declined
17 over the 100 s during SMC for all three conditions (Figure 3B). No differences
18 were observed between HOT and NEUTRO and a group effect revealed that
19 PASS was significantly ($p < 0.05$) less than HOT (Figure 3B). RMS also
20 significantly ($p < 0.01$) declined over the same contraction at the same rate for all
21 three conditions. There was a tendency ($p = 0.077$) for a difference between
22 groups with the biggest differences shown from the reduced RMS of HOT
23 compared to NEUTRO (Figure 3C).

1

2 ***50 minute intervention***

3 Core temperature rose significantly ($p < 0.01$) over the duration of the cycling
4 intervention for HOT and NEUTRO with PASS remaining unchanged throughout
5 the 50 minute intervention (Figure 4A). HOT rose to significantly ($p < 0.01$) higher
6 values than NEUTRO by the end of the cycle (Figure 4A). Skin temperature
7 significantly ($p < 0.01$) rose similarly during the 50 minute intervention for HOT and
8 PASS with NEUTRO remaining unchanged (Figure 4B). No differences existed
9 for skin temperature between HOT and PASS (Figure 4B). Muscle temperature
10 significantly ($p < 0.01$) rose following the intervention for all three conditions
11 (Figure 4C). Following the intervention HOT was significantly ($p < 0.01$) higher
12 than the other two conditions and NEUTRO had a tendency ($p = 0.062$) to be
13 higher than PASS (Figure 4C).

14

15 Heart rate significantly ($p < 0.01$) increased for both HOT and NEUTRO with HOT
16 rising to a significantly ($p < 0.01$) higher level than NEUTRO (Figure 5A). PASS
17 also significantly ($p < 0.01$) rose to a higher peak value over the 50 minutes at a
18 slower rate than the other two conditions and was significantly lower ($p < 0.01$)
19 than NEUTRO by the end of the intervention (Figure 5A). Lactate rose to a
20 significantly ($p < 0.01$) higher level for HOT than NEUTRO while PASS remained
21 unchanged at the end of the intervention (Figure 5B).

22

23 RPE was significantly ($p < 0.05$) higher for HOT than NEUTRO with both
24 conditions rising at a similar and significant ($p < 0.01$) rate over the 50 minutes

1 with PASS remaining unchanged (Figure 5C). Thermal comfort showed a
2 significant ($p < 0.01$) main effect for all 3 conditions with HOT increasing the most,
3 followed by PASS and then by NEUTRO (Figure 5D).

4

5 DISCUSSION

6

7 As expected cycling in the heat resulted in significantly greater reduction in
8 torque output during a sustained isometric contraction than that found in cycling
9 in a **thermoneutral** environment, or sitting passively in hot conditions; The novel
10 finding from this study was that during the hyperthermic conditions MFCV did not
11 decline in proportion to the torque and RMS as it did in the other two conditions.

12 .

13 It is clear that hyperthermia was induced to a greater degree during HOT as
14 evident by higher core and muscle temperature values compared to NEUTRO
15 and PASS. The elevated heart rate found in HOT could be explained in some
16 part as being caused by thermoregulatory compensation (Gonzalez-Alonso et al.,
17 1999a; Hunter et al., 2002c), where the higher skin temperature observed is
18 representative of an increase in skin blood flow (Nielsen et al., 1993). This
19 increase in skin blood flow would cause reduced cardiac return, therefore
20 decreasing stroke volume (Rowell et al., 1968). Although there was an increase
21 in thermoregulation in HOT, it is evident from the increase in core temperature
22 that this was ineffective in reducing heat storage to similar levels as was found in
23 NEUTRO. Although muscle and skin temperature increased in PASS,
24 thermoregulatory processes appeared to be effective in attenuating an increase

1 in core temperature **during** passive resting in the heat condition. This clearly
2 demonstrates that it is the combination of exercise in the heat which is the cause
3 of the increased core and muscle temperature in HOT shown in this study.

4

5 There were no significant differences in absolute MFCV between the conditions
6 during the sustained isometric contraction between HOT and NEUTRO despite
7 higher muscle temperature and greater fatigue for HOT. In contrast to this finding
8 Gray et al (2006) demonstrated faster MFCV during 6 seconds of maximal sprint
9 in hot conditions. However, our study examined the MFCV response during
10 isometric fatigue following 50 minutes of cycling at 60% of peak power output
11 which resulted in elevated blood lactate concentrations for both HOT and
12 NEUTRO. These blood lactate concentrations were higher following HOT which,
13 combined with a likely lower muscle blood flow (Nybo 2007), should lower
14 extracellular pH to such an extent to slow down MFCV (Hunter et al., 2009) and
15 attenuate regulation of the temperature effect on MFCV described by Gray et al
16 (2006). Therefore it is likely that the altered muscle energy metabolism would
17 have offset any increase in MFCV brought about by higher muscle temperature
18 values.

19

20 However, RMS was reduced to a similar level as **the** torque output in the HOT
21 compared to the other two conditions which suggests that the reduction in RMS
22 may explain in part the lower torque production during SMC. Previous studies
23 (Farina et al., 2005; Rutkove et al., 1997) have also shown reduced EMG

1 amplitude during hot conditions with Nybo and Nielsen (2001) concluding that the
2 decline in recruitment following submaximal exercise in the heat was a result of
3 reduced drive from the CNS. However, Rutkove et al (1997) heated just the lower
4 limb and fatigued it with tetanic stimulation and showed a reduction in RMS with
5 no alteration in neurotransmission, and concluded that peripheral mechanisms
6 such as nerve and muscle ion channel function were partly responsible for these
7 findings. This therefore suggests that there may be both central and peripheral
8 influences associated with the reduction of RMS in HOT. Nevertheless, when
9 **examining** the level of RMS and MFCV decline in relation to torque decrement it
10 becomes apparent that RMS **rather than** MFCV in our study is likely to be mainly
11 responsible for the fatigue observed in HOT. This is an interesting finding given
12 that it is well established that during normal conditions SMC to fatigue both RMS
13 and mean power frequency spectrum (MPFS) will both decline **in a similar**
14 **fashion** (Moritani et al., 1986). However, the limitation of MPFS measurement is
15 that it is representative of both firing rate and MFCV which makes it difficult to
16 differentiate between neuromuscular recruitment strategies and peripheral
17 mechanisms altering MFCV. As our study measured MFCV we are able to
18 elucidate that RMS declined to similar levels as torque unlike MFCV despite an
19 increase in lactate accumulation. Therefore, global motor unit recruitment,
20 **including the firing frequency and degree of synchronization for single**
21 **motor units**, as opposed to slowing of MFCV appears to be the main factor
22 responsible for the significantly greater reduction in torque output observed in the
23 HOT.

1
2 As a result of exercising in the heat during the HOT it is likely that there was an
3 increase in muscle metabolism (Edwards et al., 1972; Febbraio et al., 1996) as is
4 evident from the higher lactate values. This increase in lactate accumulation is
5 therefore likely to be from an increase in production without any concomitant
6 elevation in lactate clearance (Gonzalez-Alonso et al., 1999a). Generally,
7 previous studies (Edwards et al., 1972; Febbraio et al., 1996) that have
8 measured muscle energy metabolism elevated just muscle and not core
9 temperature. However, Drust et al (2005) elevated both core and muscle
10 temperature by having subjects perform 40 minutes of high intensity intervals
11 followed by 5 maximal 15 second efforts in a hot environment. Interestingly, it
12 was concluded that the impaired performance was not as a result of any increase
13 in metabolites, but rather from CNS down regulation (Drust et al., 2005; Nybo
14 2007). However, the subjects produced less power over the 40 minutes during
15 the hot condition which will inevitably reduce muscle energy metabolism. It can
16 therefore be proposed that this occurrence is likely a consequence of, rather than
17 a direct cause of the increased core and muscle temperature. This protocol
18 however is unlike our study which used the same submaximal work rate for both
19 conditions during the cycle. Therefore, the greater plasma lactate accumulation
20 during HOT does indicate that there was an increase of metabolic products in the
21 muscle (Febbraio et al., 1996) which may have had direct effects on the MFCV
22 (Hunter et al., 2009) as well as indirect effects on neuromuscular recruitment
23 strategies (St Clair Gibson et al., 2001).

1

2 RPE was higher in HOT than NEUTRO which concurs with previous findings
3 (Gonzalez-Alonso et al., 1999b; Nielsen et al., 2001). Nybo et al (2001) also took
4 electroencephalogram (EEG) alongside RPE measures and both showed a linear
5 increase alongside core temperature. Although the authors suggested that these
6 variables were associated they acknowledged that the impact of altered brain
7 activity on RPE was not necessarily causal. Given that RPE is perception of
8 effort caused from a variety of cues (Hampson et al., 2001) there may well be
9 additional mechanisms affecting this perceptual response. Thermal comfort
10 (discomfort) was higher in HOT which were expected given the higher core
11 temperature values. However, it was interesting to note that thermal comfort
12 values were higher in PASS than NEUTRO when the core temperatures did not
13 reflect this. This suggests that despite a higher rate of heat storage in NEUTRO
14 the perception of the environment is unrelated to the effectiveness of the
15 thermoregulatory processes.

16

17 It must be noted that, as described above, there were different patterns of torque
18 output, RMS and MFCV changes in the HOT, NEUTRO and PASS. Despite
19 these differences in patterns of changes, or perhaps because of them, all
20 subjects were able to complete the trials and none terminated either the cycling
21 or sustained isometric contraction components of the tests prematurely. This
22 indicates that there must be some degree of intelligent or strategic processing,
23 which takes into account all the peripheral and central effects of the prior cycling

1 bout and directs the subsequent alteration to the neuromuscular control
2 pathways to maintain the fidelity of the control processes regulating muscle
3 contraction (St Clair Gibson and Noakes 2004).

4

5 In conclusion this study has shown that hyperthermia induced by cycling in the
6 heat resulted in exacerbated fatigue during sustained isometric contraction of
7 maximal effort. It is likely that this was caused mainly from decrements in global
8 motor unit recruitment as opposed to slowing of muscle fibre conduction velocity.
9 However the cause of, or control strategies regulating, the different patterns of
10 relative decline in MFCV, RMS and torque for the three conditions is difficult to
11 interpret due to the complex afferent signalling to the CNS resulting in altered
12 efferent responses to the neuromuscular control strategy.

13

14

15

16 Acknowledgements

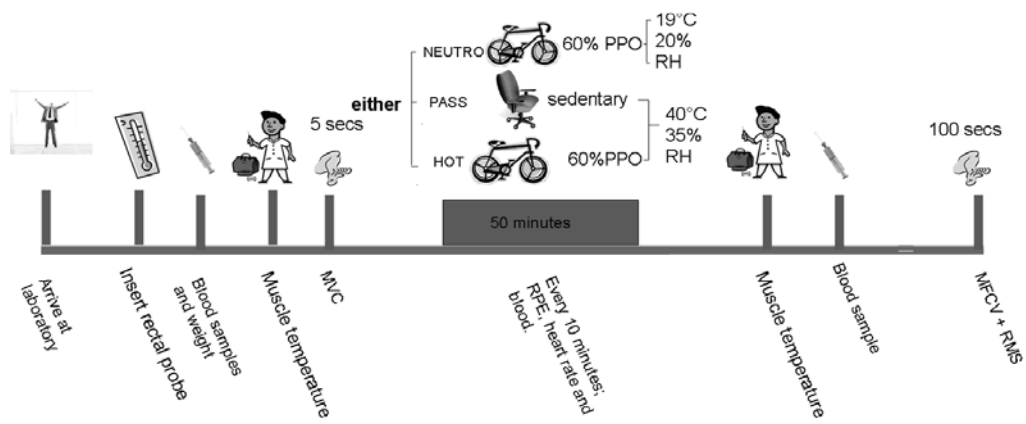
17 Dr Andi Johnson for her help and observations

18

1 FIGURES

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5 Figure 1. Time sequence of protocol

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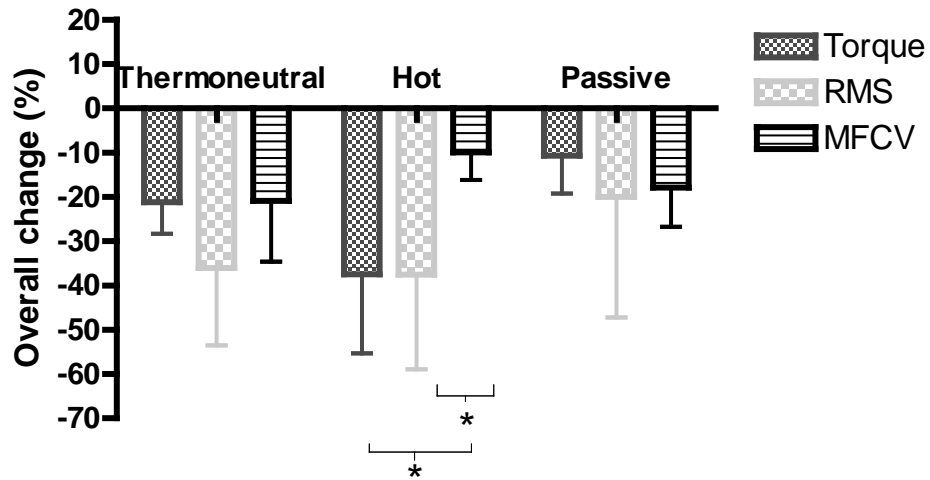


Figure 2. Delta change from peak value to the end of the contraction for torque, RMS and MFCV for the thermoneutral, hot and passive conditions where there was a significant ($p < 0.01$) difference between conditions and a significant interaction effect ($p < 0.05$). * $p < 0.01$

Sustained Maximal Contraction

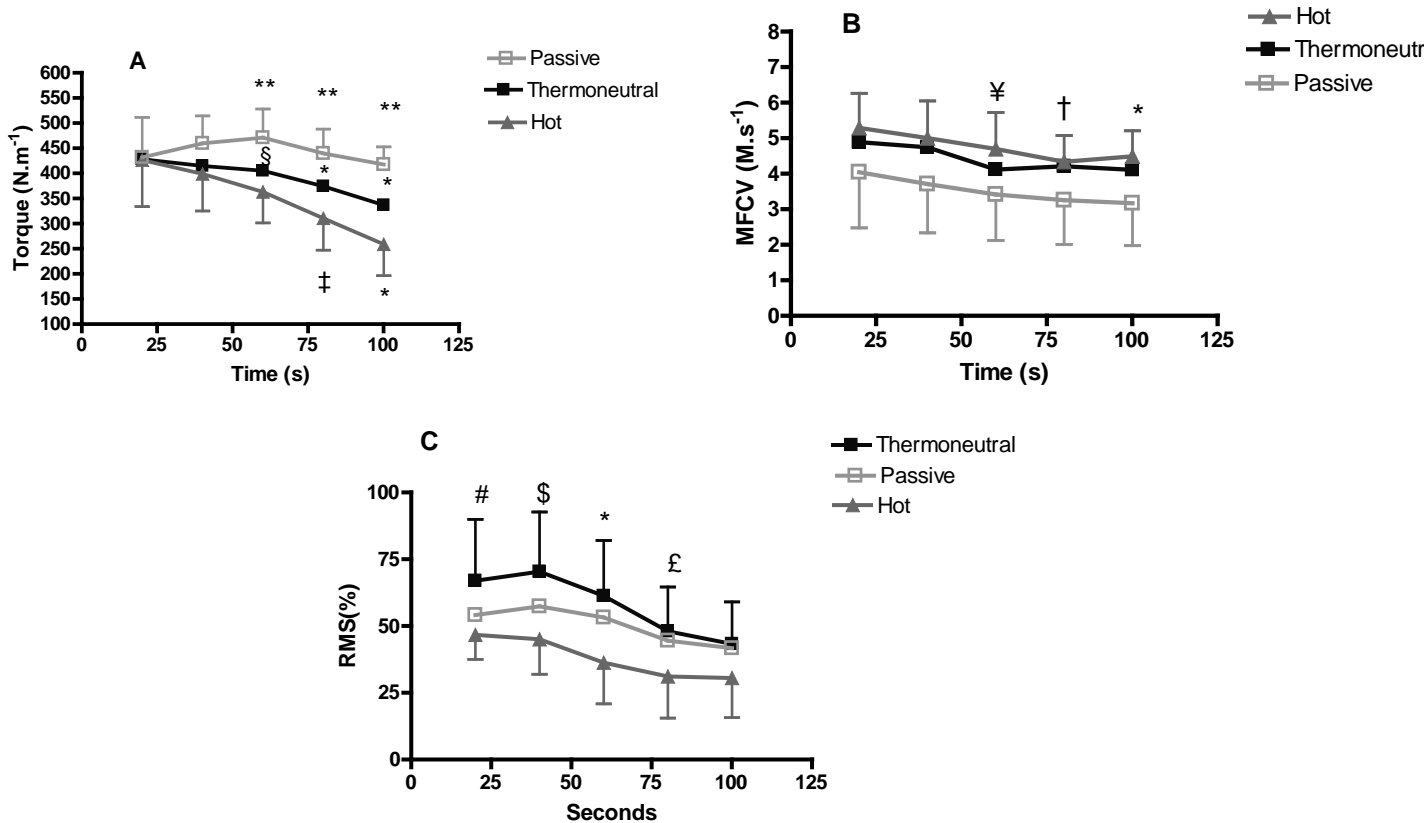


Figure 3 100s MVC post 50minute intervention for; **A.** Torque where all 3 conditions had a significant ($p < 0.01$) interaction effect. **B** Muscle Fibre Conduction Velocity where all conditions significantly ($p < 0.01$) declined at the same rate. A significant ($p < 0.05$) group effect was shown; with differences shown between just hot and passive conditions. **C** RMS where all conditions significantly ($p < 0.01$) declined over the contraction at the same rate with a tendency ($p = 0.077$) for a difference between groups which was shown between thermoneutral and hot conditions. The top, middle and bottom line of symbols represent passive vs. hot, passive vs. thermoneutral and thermoneutral vs. hot respectively. # - $p = 0.083$, ‡ - $p = 0.081$, \$ - $p = 0.076$, £ - $p = 0.063$, § - $p = 0.057$, * - $p < 0.05$, ** $p < 0.01$.

1 **50 minute intervention**

2

3 **Body Temperature**

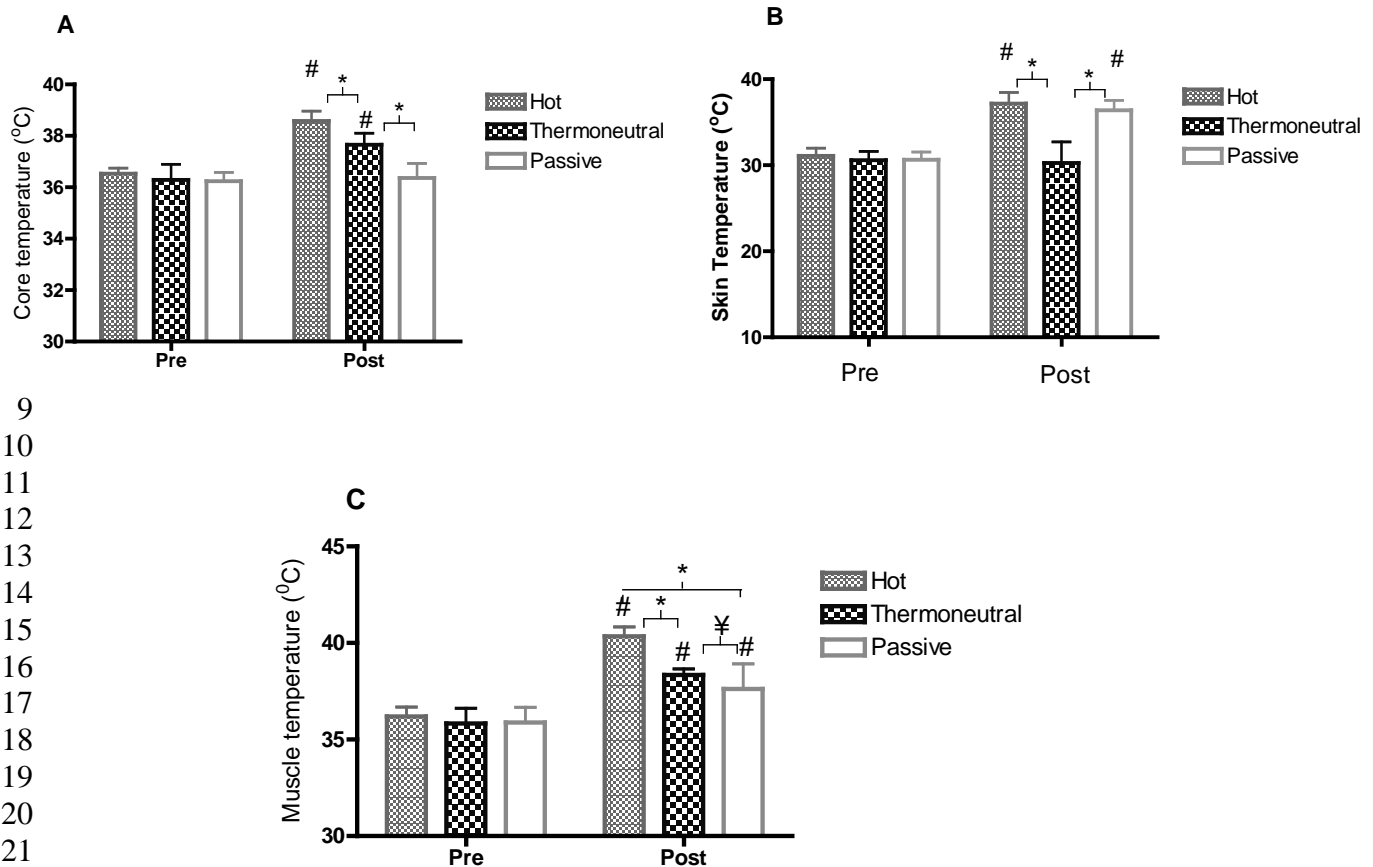
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24 **Figure 4** Rectal (A), skin (B) and muscle (C) temperatures taken before and after

25 the 50 minute intervention of cycling in a hot (40°C) and thermoneutral (18°C)

26 environment as well as passively resting in the same environment as hot. All

27 temperatures showed a significant ($p < 0.01$) time, group and interaction effect.28 *= $p < 0.01$ and ¥= $p = 0.062$ difference between conditions; #= $p < 0.01$ difference

29 within condition to pre value.

30

1 **50 minute intervention**

2
3 **Other physiological and subjective responses**

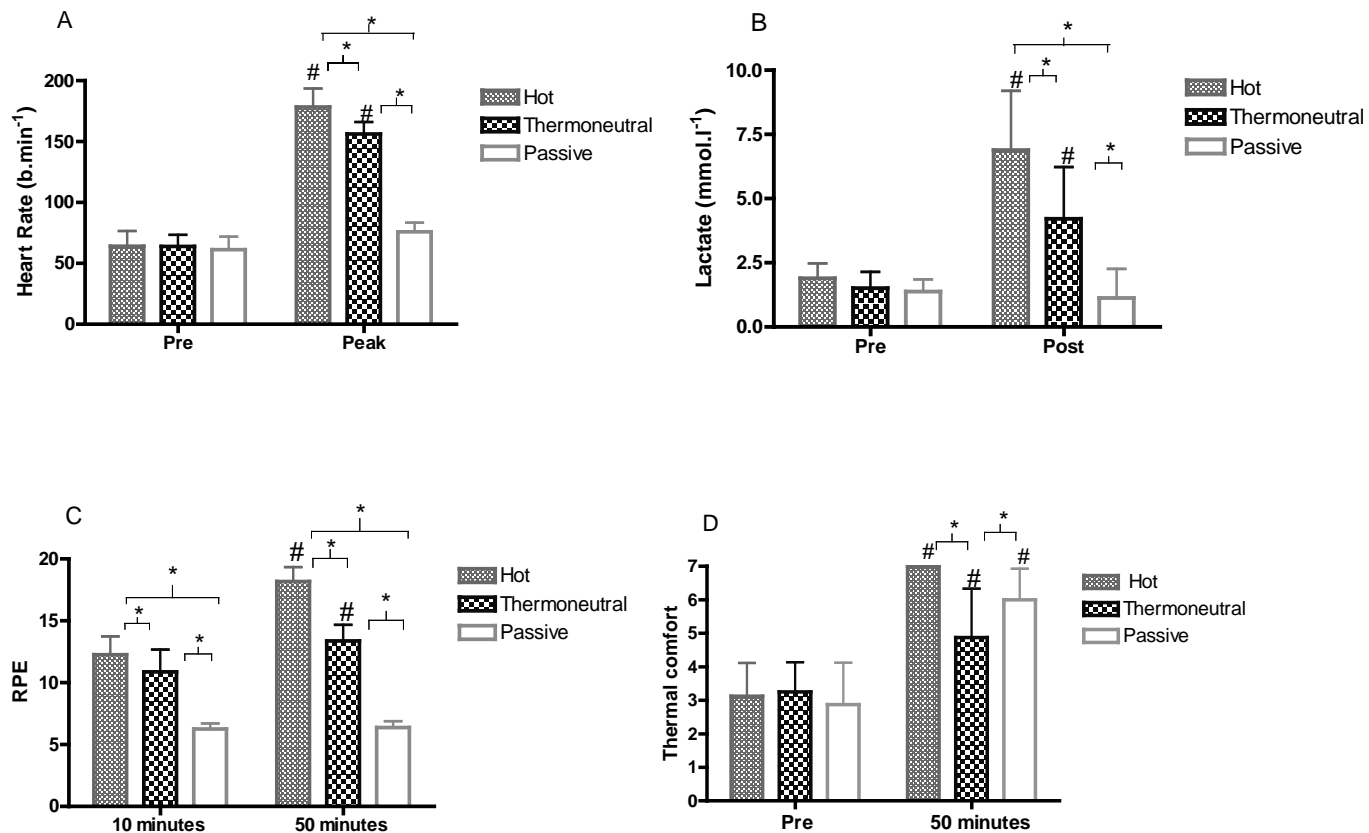


Figure 5. Heart rate (A), Lactate (B), RPE (C) and Thermal comfort (D) taken before and at the peak value (40 minutes); pre and post; 10 and 50 minutes; and pre and 50 minutes of the intervention respectively. All temperatures showed a significant ($p < 0.01$) time, group and interaction effect. $*$ = $p < 0.01$ difference between conditions; $\#$ = $p < 0.01$ difference within condition to pre value.

1 REFERENCES

2

3

4

5 Andreassen, S., Arendt-Nielsen, L. Muscle fibre conduction velocity in motor
6 units of the human anterior tibial muscle: a new size principle parameter. *J*
7 *Physiol* 1987;391(561-571

8 Borg, G. A. Perceived exertion: a note on "history" and methods. *Med Sci Sports*
9 1973;5(2):90-93

10 Brody, L. R., Pollock, M. T., Roy, S. H., De Luca, C. J., Celli, B. pH-induced
11 effects on median frequency and conduction velocity of the myoelectric
12 signal. *J Appl Physiol* 1991;71(5):1878-1885

13 Drust, B., Rasmussen, P., Mohr, M., Nielsen, B., Nybo, L. Elevations in core and
14 muscle temperature impairs repeated sprint performance. *Acta Physiol*
15 *Scand* 2005;183(2):181-190

16 Edwards, R. H., Harris, R. C., Hultman, E., Kaijser, L., Koh, D., Nordesjo, L. O.
17 Effect of temperature on muscle energy metabolism and endurance during
18 successive isometric contractions, sustained to fatigue, of the quadriceps
19 muscle in man. *J Physiol* 1972;220(2):335-352

20 Farina, D., Arendt-Nielsen, L., Graven-Nielsen, T. Effect of temperature on spike-
21 triggered average torque and electrophysiological properties of low-
22 threshold motor units. *J Appl Physiol* 2005;99(1):197-203

23 Febbraio, M. A., Carey, M. F., Snow, R. J., Stathis, C. G., Hargreaves, M.
24 Influence of elevated muscle temperature on metabolism during intense,
25 dynamic exercise. *Am J Physiol* 1996;271(5 Pt 2):R1251-R1255

26 Fitts, R. H. Cellular mechanisms of muscle fatigue. *Physiol Rev* 1994;74(1):49-94

27 Galloway, S. D., Maughan, R. J. Effects of ambient temperature on the capacity
28 to perform prolonged cycle exercise in man. *Med Sci Sports Exerc*
29 1997;29(9):1240-1249

30 Gonzalez-Alonso, J., Calbet, J. A., Nielsen, B. Metabolic and thermodynamic
31 responses to dehydration-induced reductions in muscle blood flow in
32 exercising humans. *J Physiol* 1999a;520 Pt 2(577-589

33 Gonzalez-Alonso, J., Teller, C., Andersen, S. L., Jensen, F. B., Hyldig, T.,
34 Nielsen, B. Influence of body temperature on the development of fatigue
35 during prolonged exercise in the heat. *J Appl Physiol* 1999b;86(3):1032-
36 1039

- 1 Gray, S. R., De, Vito G., Nimmo, M. A., Farina, D., Ferguson, R. A. Skeletal
2 muscle ATP turnover and muscle fiber conduction velocity are elevated at
3 higher muscle temperatures during maximal power output development in
4 humans. *Am J Physiol Regul Integr Comp Physiol* 2006;290(2):R376-
5 R382
- 6 Hampson, D. B., St Clair, Gibson A., Lambert, M. I., Noakes, T. D. The influence
7 of sensory cues on the perception of exertion during exercise and central
8 regulation of exercise performance. *Sports Med* 2001;31(13):935-952
- 9 Hawley, J. A., Noakes, T. D. Peak power output predicts maximal oxygen uptake
10 and performance time in trained cyclists. *Eur J Appl Physiol Occup Physiol*
11 1992;65(1):79-83
- 12 Hunter, A. M., De, Vito G., Bolger, C., Mullany, H., Galloway, S. D. The effect of
13 induced alkalosis and submaximal cycling on neuromuscular response
14 during sustained isometric contraction. *J Sports Sci* 2009;1-9
- 15 Hunter, A. M., St Clair, Gibson A., Derman, W. E., Lambert, M., Dennis, S. C.,
16 Noakes, T. D. The effect of selective beta1-blockade on EMG signal
17 characteristics during progressive endurance exercise. *Eur J Appl Physiol*
18 2002a;88(3):275-281
- 19 Hunter, A. M., St Clair, Gibson A., Lambert, M., Noakes, T. D. Electromyographic
20 (EMG) normalization method for cycle fatigue protocols. *Med Sci Sports*
21 *Exerc* 2002b;34(5):857-861
- 22 Hunter, A. M., St Clair, Gibson A., Mbambo, Z., Lambert, M. I., Noakes, T. D. The
23 effects of heat stress on neuromuscular activity during endurance
24 exercise. *Pflugers Arch* 2002c;444(6):738-743
- 25 Lowery, M., Nolan, P., O'Malley, M. Electromyogram median frequency, spectral
26 compression and muscle fibre conduction velocity during sustained sub-
27 maximal contraction of the brachioradialis muscle. *J Electromyogr Kinesiol*
28 2002;12(2):111-118
- 29 Moritani, T., Muro, M., Nagata, A. Intramuscular and surface electromyogram
30 changes during muscle fatigue. *J Appl Physiol* 1986;60(4):1179-1185
- 31 Nielsen, B., Hales, J. R., Strange, S., Christensen, N. J., Warberg, J., Saltin, B.
32 Human circulatory and thermoregulatory adaptations with heat acclimation
33 and exercise in a hot, dry environment. *J Physiol* 1993;460:467-485
- 34 Nielsen, B., Hyldig, T., Bidstrup, F., Gonzalez-Alonso, J., Christoffersen, G. R.
35 Brain activity and fatigue during prolonged exercise in the heat. *Pflugers*
36 *Arch* 2001;442(1):41-48
- 37 Nybo, L. Hyperthermia and fatigue. *J Appl Physiol* 2007;104(3):871-878

- 1 Nybo, L., Nielsen, B. Hyperthermia and central fatigue during prolonged exercise
2 in humans. *J Appl Physiol* 2001;91(3):1055-1060
- 3 Rowell, L. B., Brengelmann, G. L., Blackmon, J. R., Twiss, R. D., Kusumi, F.
4 Splanchnic blood flow and metabolism in heat-stressed man. *J Appl*
5 *Physiol* 1968;24(4):475-484
- 6 Rutkove, S. B., Kothari, M. J., Shefner, J. M. Nerve, muscle, and neuromuscular
7 junction electrophysiology at high temperature. *Muscle Nerve*
8 1997;20(4):431-436
- 9 St Clair, Gibson A., Lambert, M. L., Noakes, T. D. Neural control of force output
10 during maximal and submaximal exercise. *Sports Med* 2001;31(9):637-
11 650
- 12 St Clair, Gibson A., Noakes, T. D. Evidence for complex system integration and
13 dynamic neural regulation of skeletal muscle recruitment during exercise
14 in humans. *Br J Sports Med* 2004;38(6):797-806
- 15 Todd, G., Butler, J. E., Taylor, J. L., Gandevia, S. C. Hyperthermia: a failure of
16 the motor cortex and the muscle. *J Physiol* 2005;563(Pt 2):621-631
17
18
19
20
21
22
23
24