

1 Abstract:

2 *Purpose:* The smaller muscle mass of the upper body compared to the lower body may elicit a
3 smaller thermoregulatory stimulus during exercise and thus produce novel training induced
4 thermoregulatory adaptations. Therefore, the principal aim of the study was to examine the effect
5 of arm training on thermoregulatory responses during submaximal exercise. *Methods:* Thirteen
6 healthy male participants (Mean \pm SD age 27.8 \pm 5.0yrs, body mass 74.8 \pm 9.5kg) took part in 8
7 weeks of arm crank ergometry training. Thermoregulatory and calf blood flow responses were
8 measured during 30 minutes of arm cranking at 60% peak power (W_{peak}) pre, and post training and
9 post training at the same absolute intensity as pre training. Core temperature and skin
10 temperatures were measured, along with heat flow at the calf, thigh, upper arm and chest. Calf
11 blood flow using venous occlusion plethysmography was performed pre and post exercise and calf
12 volume was determined during exercise. *Results:* The upper body training reduced aural
13 temperature ($0.1 \pm 0.3^{\circ}\text{C}$) and heat storage ($0.3 \pm 0.2 \text{ J}\cdot\text{g}^{-1}$) at a given power output as a result of
14 increased whole body sweating and heat flow. Arm crank training produced a smaller change in
15 calf volume post training at the same absolute exercise intensity ($-1.2 \pm 0.8\%$ compared to -2.2
16 $\pm 0.9\%$ pre training; $P < .05$) suggesting reduced leg vasoconstriction. *Conclusion:* Training improved
17 the main markers of aerobic fitness. However, the results of this study suggest arm crank training
18 additionally elicits physiological responses specific to the lower body which may aid
19 thermoregulation.

20 Keywords: Thermoregulation, upper body exercise, training, calf volume

21

22

23 **Abbreviations:**

24 Analysis of Variance	ANOVA
25 Blood lactate	Bla
26 Degrees Centigrade	$^{\circ}\text{C}$
27 Haemoglobin	Hb

1	Haematocrit	Hct
2	Heart Rate	HR
3	Kilogram	Kg
4	Microlitres	μl
5	Mid training trial	MID
6	Minute ventilation	VE
7	Peak Oxygen Consumption	$\dot{V} O_{2\text{peak}}$
8	Post training absolute intensity	POST-ABS
9	Post training relative intensity	POST-REL
10	Peak Power	W_{peak}
11	Pre training trial	PRE
12	Ratings of perceived exertion	RPE
13	Revolutions per minute	$\text{rev}\cdot\text{min}^{-1}$
14	Standard Deviation	SD
15	Years	yrs

Introduction

Previous research examining responses to upper body exercise training have mainly investigated whether adaptations are muscle specific (Volianitis et al., 2004; Bhambhani et al., 1991; Stamford et al., 1978), whether training benefits can be transferred to lower body exercise performance (Loftin et al., 1988) and the use of upper body exercise for rehabilitation (Mostardi et al., 1981; Tew et al., 2009). The literature though appears to be in conflict with regards to the specific causes of the improvement in aerobic capacity with upper body exercise training. Some studies suggest that aerobic improvements are dependent on central adaptations such as cardiac output and stroke volume (Loftin et al., 1988) whereas other studies suggest peripheral circulatory changes such as arterial – venous oxygen difference are predominant (Volianitis et al., 2004). However, training is limb specific (Loftin et al., 1988) which implies that a substantial proportion of the conditioning response to training is attributed to extracardiac or peripheral factors such as alterations in blood flow and cellular and enzymatic adaptations in the trained limb alone (Volianitis et al., 2004).

In addition to cardiorespiratory adaptations lower body exercise training also causes adaptations to thermoregulatory responses such as initiating sweating and cutaneous vasodilation at lower core and skin temperatures (Armstrong and Maresh, 1998). However, there are no reported studies regarding the effects of upper body exercise training on thermoregulatory responses to exercise. Differences between modes may exist due to that fact that upper body exercise involves a smaller muscle mass with potentially smaller increases in core temperature when compared to lower body exercise at the same relative exercise intensity (Sawka et al., 1984). Regular, smaller increases in core temperature and thus lower thermal strain during each training session could result in different training induced thermoregulatory responses when compared to lower body exercise.

Previous research examining thermoregulatory responses during upper body exercise has produced interesting results with regards to responses within the lower body. For example, a decrease in calf

skin temperature during arm crank exercise in cool (21°C) conditions and an increase in skin temperature during exercise in the heat (31°C) have been observed (Price and Campbell, 2002; Price and Mather, 2004; Dawson et al., 1994). In addition, calf volume (representing whole limb blood flow from strain gauge plethysmography measurements) has been observed to decrease during arm crank exercise in cool ambient conditions suggesting a sympathetically mediated redistribution of blood away from the lower body (Hopman et al., 1993). Such a response potentially explains the observed decrease in calf skin temperature due to a reduction in limb blood flow and thus delivery of blood. These acute adaptations in calf volume and skin temperature suggests the lower body plays an important thermoregulatory role during upper body exercise by redistributing blood to the more active upper body with further adaptations potentially occurring as a result of upper body exercise training. More importantly, during upper body exercise such adaptations may be local to those areas not specifically involved in force production during exercise per se, i.e. the lower body. Lower body exercise training has been demonstrated to lessen the decrease in blood flow to splanchnic, renal and cutaneous areas at a given power output (Ho et al., 1997; Rowell et al., 1964; 1965) whereas blood flow to muscle remains unaffected (Stolwijk, 1997). Since upper body exercise affects splanchnic, renal and cutaneous blood flow (Ahlborg et al., 1975) it is possible that upper body training may also influence vasomotor responses in other areas such as calf volume changes during exercise. As vasomotor adaptations are linked to thermoregulatory responses (Wakabayashi et al., 2012) specific vasomotor adaptations may occur with training. Therefore, the principal aim of the study was to examine the effect of arm training on thermoregulatory responses, including calf volume, during submaximal exercise.

Methods

Participants

Thirteen healthy male participants (Mean \pm SD age 27.8 \pm 5.0yrs, body mass 74.8 \pm 9.5kg, body fat percentage of 17.8 \pm 5.2%) not specifically upper body trained, volunteered to participate in this study.

University Ethics Committee approval for the study's experimental procedures was obtained and followed the principles outlined in the Declaration of Helsinki. Participants undertook approximately 2 ± 1 hr a week of training in a range of sports such as football, running and general gym work. All participants were given written information concerning the nature and purpose of the study, completed a pre-participation medical screening questionnaire and gave written consent prior to participation.

Preliminary Tests

Participants performed a continuous incremental exercise test ($\dot{V} O_{2\text{peak}}$) to volitional exhaustion using the protocol of Smith et al. (2004). In brief this protocol involved a starting power output of 50W with increases of 20W every two minutes to volitional exhaustion. Cadence was set at 70 rev.min⁻¹. Peak oxygen uptake ($\dot{V} O_{2\text{peak}}$), peak power (W_{peak}) and subsequent exercise intensities for the training programme were determined before, during and after 8 weeks of training. Participants sat at the arm crank ergometer (Lode, Angio, Groningen, the Netherlands) with the crank shaft in line with the shoulder joint (Bar-Or and Swirren, 1975). Expired gas was continuously measured throughout the test using an online breath by breath analyser (Metamax 3B, Leipzig, Germany) calibrated against room air and a calibrating gas. Oxygen consumption (VO_2) and minute ventilation (VE) were subsequently determined. Heart rate (HR) was continuously monitored (Polar Accurex Plus, Kempele, Finland). Central and local ratings of perceived exertion (RPE_{central} and RPE_{local} respectively; Borg Scale) were recorded at volitional exhaustion. Following a ten minute cool down participants were familiarised with the intensity of exercise, which was to be undertaken in the subsequent experimental trials and training sessions for 5 minutes.

Prior to each $\dot{V} O_{2\text{peak}}$ test skin fold measurements were taken using skin fold callipers (Baty International, West Sussex, UK). Measurements were taken at the bicep, tricep, subscapular, abdominal, iliac crest, supraspinale, thigh and calf sites. The sum of eight sites were determined as well as body fat percentage using four sites determined from the equation of Durnin and Womersley

(1974). The circumference of the bicep muscle on the right arm was also measured whilst relaxed and tensed in accordance with the ISAK protocol (Nevil, 2006).

Training Study:

All participants completed an eight week upper body exercise training programme which involved three sessions each week. More specifically, two training sessions involved exercising for 30 min at 60% W_{peak} with the third session comprising of 50 minute of interval training (Figure 1). The interval session involved 10 min at 60% W_{peak} followed by alternating bouts of 2 min unloaded arm cranking (0 W setting) followed by 2 min at 75% W_{peak} repeated 10 times. All exercise was performed at 70 rev.min⁻¹ with HR recorded throughout all sessions. The VO_{2peak} test was undertaken initially (PRE) to determine the training intensities undertaken and was repeated at the start of week 5 (MID) to adjust the training intensity. Each participants VO_{2peak} was measured again at the end of the eight week training period (POST) to determine overall effects of the training on aerobic fitness.

Submaximal Exercise Trials

To determine baseline thermoregulatory responses in cool conditions (22.0 ±0.5°C and 64.4% rh) at the beginning of training (week 1, session 1) participants performed a submaximal exercise trial (PRE) at 60% W_{peak} for 30 min followed by 30 min of passive recovery. Two submaximal exercise trials were undertaken at the end of training, one at the original absolute work load (POST-ABS) and one at the new relative work load (POST-REL). These trials enabled the comparison of:

- 1) Absolute workloads before and after training (PRE vs POST-ABS)
- 2) Relative workloads before and after training (PRE vs POST-REL)
- 3) Absolute vs relative workloads post training (POST-ABS vs POST-REL).

All submaximal exercise trials during the training period were performed at an ambient temperature of 22.3 ±2.1°C and 64.2 ±7.5% relative humidity. No fluid was consumed during exercise. On arrival at the laboratory body mass was recorded using electronic scales (Seca, Hamburg, Germany).

Participants wore shorts, socks, and training shoes and rested for 20 min while temperature thermistors (Grant, Cambridge, UK) and heat flow sensors (Data Harvest Easy sense Advanced, Bedfordshire, UK) were attached. Aural, rectal and skin temperatures (calf, thigh, chest, upper arm and back) were measured using a data logger (Squirrel 2020 series, Cambridge, UK) and provided values for calculation of heat storage (Havenith et al., 1995). Heat flow at the calf, thigh, chest and upper arm (same landmarks as skin thermistors) and gas analysis were measured throughout, rest, exercise and recovery.

Baseline data for all measures were obtained during the final five min of seated rest prior to exercise. Resting blood pressure was measured at the left arm using a sphygmomanometer (Accoson Ltd, London, UK). A resting capillary blood sample was taken from the left earlobe for measurement of blood lactate (Bla) (Analox GM7, London, UK). Three 80 μ l capillary blood samples were also taken for measurement of haematocrit (Hct) using a micro haematocrit reader (Hawksley, Surrey, UK) along with three cuvettes for analysis of haemoglobin concentration (Hb; Hemocue, Clandon, Sheffield). Plasma volume was subsequently calculated using the equation of Dill and Costill (1974). Calf blood flow and volume were measured at rest and throughout both exercise and recovery using standard procedures for venous occlusion plethysmography (Fehling et al., 1999; Hopman et al., 1993). A contoured cuff was placed on the left thigh and connected to a rapid cuff inflator (Hokanson E20, Bellevue, USA) set to inflate to 50mmHg and held for five seconds and rapidly deflated over eight seconds. A 1% calibration was performed on the plethysmograph after 5 min resting followed by a resting blood flow measurement in triplicate. Calf volume change was measured from pre to post exercise by measuring the resistance change in the strain gauge throughout exercise. The change in resistance was then converted to a percentage change in calf volume by comparing the value with the 1% calibration.

Prior to the start of exercise resting values for HR, heat flow, calf volume, blood flow, aural, rectal and skin temperatures were recorded. Participants then performed arm crank exercise at 60% W_{peak} for

30 min at 70 rev.min⁻¹. Participants remained seated post exercise for a further 30 min. Heat flow, and core and skin temperatures were recorded every 5 min during exercise and passive recovery. Changes in calf volume were recorded continuously throughout exercise. Ratings of perceived exertion using the Borg Scale were determined for overall fatigue (RPE_{central}) as well as local arm fatigue (RPE_{local}). RPE was recorded at 5, 15, and 30 min during exercise. VO₂, and VE were determined at five minute intervals during exercise, and passive recovery. Calf blood flow was recorded at rest, on the cessation of exercise and every 5 min during passive recovery. Blood samples were taken from the earlobe for Bla concentration at 5, 15, and 30 min, as well as for Hb and Hct at the end of exercise. Body mass was recorded after passive recovery to calculate whole body sweat losses (l) and extrapolated to sweat rate (l.hr⁻¹).

Statistical Analysis

The Shapiro-Wilk statistic confirmed that the normal distribution assumption was met for all variables. Paired T-tests were performed on the pre and post anthropometric data. All other independent variables were analysed using a repeated measures two-way (Trial X Time) analysis of variance (ANOVA; SPSS v20). Post hoc analyses (Bonferroni pairwise comparisons) were performed on significant ANOVA results to control for type I error. Data are presented as mean ± standard deviation in tables and figures. Significance was set at p<0.05. Where appropriate, Pearsons correlations were undertaken to determine relationships between variables. A post hoc statistical power analysis was conducted using the Hopkins method, and it was found that the sample size was sufficient to provide more than 80% statistical power.

1 Results

2
3 *Peak physiological responses*

4 The peak physiological responses obtained during PRE, MID and POST incremental tests for $\dot{V} O_{2peak}$
5 as well as anthropometric changes determined PRE and POST training are shown in Table I. Both \dot{V}
6 O_{2peak} and W_{peak} increased with training ($P<0.05$) being greatest POST compared to both PRE and MID
7 ($P<0.05$), although HR_{peak} remained the same ($P>0.05$). Both $RPE_{overall}$ and RPE_{local} at volitional
8 exhaustion increased POST ($P<0.05$). Whole body fat percentage, sum of 8 skin fold sites and body
9 mass PRE and POST training remained the same ($P>0.05$). Bicep circumference when relaxed
10 remained the same following training ($P>0.05$), however, when tensed values increased by $3.7 (\pm 2.8)\%$
11 ($P<0.05$). There was a positive correlation between the percentage increase in bicep circumference
12 tensed and increase in W_{peak} ($r=0.78$; $P<0.05$).

13
14
15 ***Table I near here***
16

17 *Physiological and Thermoregulatory Responses during Submaximal Exercise Trials:*

18 ***Table II near here***

19
20 The physiological responses at the cessation of each trial are shown in Table II. Significant trial \times time
21 interactions were noted for HR and VO_2 with values being lowest during POST-ABS ($P<0.05$). No
22 differences in HR were observed between PRE and POST-REL whereas VO_2 was greatest during
23 POST-REL when compared to PRE. There was a significant time \times trial interaction for blood lactate
24 concentration ($P<0.05$). Blood lactate concentration increased from rest and reached a plateau by 15
25 min during exercise in all trials. Values were lowest during POST-ABS and greatest during POST-REL
26 ($P<0.05$; Table II).

27
28 Participants perceived RPE_{local} to be greater than $RPE_{central}$ during PRE and POST-REL ($P<0.05$)
29 however, there were no differences between RPE_{local} and $RPE_{central}$ during POST-ABS ($P>0.05$). Both
30 $RPE_{central}$ and RPE_{local} increased at 5, 15 and 30 min of exercise ($P<0.05$) with POST-ABS being lower
31 compared to PRE and POST-REL (Table II). Sweat rate was significantly greater POST-REL compared

1 to PRE ($P < 0.05$; main effect for trial). Sweat rates for PRE, POST-ABS and POST-REL were 0.6 ± 0.6 ,
2 0.8 ± 0.6 and 1.0 ± 0.6 l.hr⁻¹.

3

4 *Core Temperature during Exercise and Passive Recovery*

5 There were no differences in resting aural or rectal temperature between trials ($P > 0.05$). Aural
6 temperature increased by 0.3 ± 0.2 , 0.4 ± 0.3 and 0.5 ± 0.4 °C, for PRE, POST-ABS and POST-REL,
7 respectively ($P < 0.05$; main effect for time; Figure 1a) from rest to the end of exercise. Rectal
8 temperature increased from rest during exercise in all trials by 0.4 ± 0.2 , 0.4 ± 0.2 and 0.5 ± 0.3 °C for
9 PRE, POST-ABS and POST-REL, respectively; $P < 0.05$; main effect for time; Figure 1b). These
10 increases for both aural and rectal did not correlate with the percentage $\dot{V} O_{2max}$ at 30 min of exercise
11 ($r = 0.03$ and $r = 0.06$ respectively). Absolute aural temperature was significantly lower during POST-ABS
12 compared to both PRE and POST-REL with no differences between PRE and POST-REL during
13 exercise ($P < 0.05$; main effect for trial). Aural and rectal temperature both decreased towards resting
14 values by 30 min of passive recovery in all trials ($P > 0.05$).

15

16 ***Figure I near here***

17

18 *Skin temperature Responses during Exercise and Passive Recovery*

19 There were no effects of training on resting skin temperature for any site ($P > 0.05$). Upper arm, back
20 and thigh skin temperatures were coolest during POST-ABS with no differences between PRE and
21 POST-REL ($P < 0.05$, main effect for trial). Conversely, when compared to the other skin temperature
22 sites calf skin temperature decreased during exercise in all trials ($P < 0.05$; Figure 2a). The greatest
23 decrease occurred post training in POST-ABS. Calf skin temperature had a tendency to be lower
24 ($P = 0.08$) at rest during PRE and was significantly cooler throughout exercise when compared to both
25 post training trials ($P < 0.05$). During passive recovery calf skin temperature decreased further by -1.7
26 ± 0.8 , -1.2 ± 0.5 , and -1.4 ± 0.7 °C in PRE, POST-ABS and POST-REL respectively with no differences
27 between trials ($P > 0.05$).

28

29 ***Figure II near here***

30

31 *Heat Storage*

1 Heat storage increased from rest in all trials until the end of exercise ($P<0.05$; main effect for time).
2 Heat storage increased by 0.95 ± 0.55 , 0.75 ± 0.62 and 1.03 ± 0.39 $J \cdot g^{-1}$ for PRE, POST-ABS and POST-
3 REL respectively. Heat storage was lower during POST-ABS when compared to PRE and POST-REL
4 ($P<0.05$; main effect for trial). Heat storage decreased during recovery in all trials ($P<0.05$).

5

6 *Heat Flow during Exercise and Passive Recovery*

7 Heat flow significantly increased during exercise in all trials at the upper arm, chest and thigh sites
8 whereas it remained unchanged at the calf (Figure III). During passive recovery heat flow decreased
9 at all sites ($P<0.05$; main effect for time). Heat flow was greater during POST-REL than for POST-ABS
10 at the upper arm, chest and thigh ($P<0.05$; main effect for trial) and greater during POST-REL than PRE
11 for the upper arm, chest and calf ($P<0.05$). Heat flow was greater during POST-ABS compared to PRE
12 for the calf and chest ($P<0.05$). Calf heat flow produced a weak correlation with calf skin temperature
13 during exercise ($r=0.46$; $P<0.05$).

14

15 ***Figure III near here***

16

17 *Calf Volume and Blood Flow during Exercise and Passive Recovery*

18 Calf volume decreased during exercise when compared to rest for each trial (-2.2 ± 0.9 , -1.2 ± 0.8 , -1.8
19 $\pm 1.0\%$ for PRE, POST-ABS and POST-REL respectively; $P<0.05$; Figure IVa). Training resulted in a
20 smaller decrease in calf volume during POST-ABS compared to PRE ($P<0.05$; main effect for trial).
21 There were no differences between PRE and POST-REL ($P>0.05$). There was no correlation between
22 change in calf volume and calf skin temperature ($r=0.08$; $P>0.05$). There were no differences in calf
23 blood flow at rest or at the end of exercise between trials ($P>0.05$; Figure IVb). However, blood flow for
24 the remainder of recovery was lowest post training in POST-REL ($P<0.05$; main effect for trial).

1 Discussion

2 The principal aim of the study was to examine the effect of arm training on thermoregulatory responses
3 during submaximal exercise. The main findings were; reduced aural temperature, skin temperature
4 and heat storage during exercise at the same absolute intensity post training when compared to PRE
5 training. Sweat rate increased post training at the same relative exercise intensity when compared to
6 PRE. There was also a blunted calf volume response suggesting less blood flow is redistributed during
7 exercise at the same absolute intensity post training compared to pre training.

8
9 Training improved $\dot{V} O_{2peak}$ by 18.9% which is similar to that of Magel et al. (1978; 16.5%) after 10
10 weeks of arm interval training. Furthermore, when performing exercise at the same absolute exercise
11 intensity as pre-training $\dot{V} O_2$, HR and blood lactate concentration were lower and indicative of
12 improved exercise economy. Increasing leg strength in untrained participants has been demonstrated
13 to improve leg cycling economy (Loveless et al., 2005) therefore suggesting that in the present study
14 the lower $\dot{V} O_2$ obtained during POST-ABS could have been a result of improved arm strength and
15 increased stroke volume most likely due to increased left ventricle chamber dimensions (Gates et al.,
16 2003).

17
18 Although there is evidence in the current data for improved central factors on performance (i.e. an
19 increase in SV as noted above) there also appears to be some involvement of peripheral factors at the
20 muscle level. For example, support for peripheral limitations in the present study includes the increase
21 in W_{peak} of ~30%, which was much greater than for $\dot{V} O_2$ peak. Peripheral adaptations are evident by
22 the fact that the increase in W_{peak} was significantly correlated with the increased bicep circumference
23 when flexed ($r=0.78$; $P<0.05$), therefore hypertrophy of the biceps in part is likely to have produced the
24 increase in peak power.

25
26 The present study demonstrated a lower aural temperature during POST-ABS when compared to PRE
27 but there were no differences in rectal temperature. The difference between sites is possibly due to
28 differences in local heat dissipation between sites and, with regards to rectal temperature, some heat
29 gain from nearby intrapelvic muscles (Aulick et al., 1981). Both Saltin and Hermansen (1966) and Gant

1 et al. (2004) noted a correlation between exercise intensity ($\%VO_{2max}$) and rectal temperature during
2 lower body exercise suggesting that rectal temperature was dependent on exercise intensity. However,
3 the present study showed no correlation between the percentage of $\dot{V}O_{2peak}$ with core temperature
4 responses pre and post training suggesting that there may be differences in heat dissipation between
5 upper and lower body exercise and core temperature estimates.

6

7 Heat storage during exercise at the same absolute power output was significantly reduced with training.
8 This is likely due to heat storage being a combination of the reduced core temperature responses noted
9 earlier and decreased individual and mean skin temperatures during POST-ABS. The reduced heat
10 storage during POST-ABS was most likely due to more efficient heat dissipation as a result of a more
11 rapid cutaneous vasodilation (Boegli et al. 2003) and an earlier onset of sweating (Yamazaki et al. 1994;
12 Pilardeau et al. 1988). Although cutaneous blood flow was not measured in the present study, heat
13 flow, which has been considered indicative of cutaneous blood flow (Sawka et al., 1984), was generally
14 greater for the upper arm chest and calf during POST-ABS compared to PRE suggesting increased dry
15 heat exchange with training. In addition, whole body sweat rate increased following training (POST-
16 REL) suggesting an accompanying increase in evaporative heat loss. Increased sweating together with
17 increased dry heat exchange would have resulted in more efficient heat dissipation and subsequently
18 reduced heat storage post training.

19

20 Calf skin temperature at rest and during exercise was warmer post training and accompanied by a
21 greater heat flow when compared to pre training. The warmer calf skin temperature post training at
22 the same absolute intensity may be a result of repeated redistribution of blood flow in the legs during
23 training with more blood directed to the skin and less blood flow directed to the relatively inactive
24 muscles. It has been shown that training increases cutaneous blood flow at a lower core temperature
25 during exercise (Johnson, 1998). It is therefore possible that the warmer calf skin temperature during
26 the POST-ABS trial is a result of increased skin blood flow transferring warm blood to the skin for heat
27 dissipation. This corresponds with the increase in heat flow occurring at the calf during the POST-ABS
28 trial compared to PRE. When examining the decreases in calf skin temperature from rest in all trials
29 during exercise ($0.4 \pm 0.8^{\circ}C$, $0.8 \pm 0.6^{\circ}C$ and $0.5 \pm 0.9^{\circ}C$ for PRE, POST-ABS and POST-REL

1 respectively), there was a greater decrease in calf skin temperature from rest at the same absolute
2 intensity post training indicating improved heat loss.

3

4 The decrease in calf volume during exercise observed during each trial is most likely a result of
5 increased vasoconstriction in the calf vasculature (Hopman et al., 1993) which increases venous return
6 to the central circulation. This reasoning is based on the assumption that the decrease in calf volume
7 is due to an increase in muscle sympathetic nerve activity (MSNA) causing vasoconstriction in the non
8 active muscle. Saito et al. (1990) demonstrated a delay in the decrease in blood flow to the calf region
9 during static handgrip exercise which coincided with a delay in the increase in sympathetic nerve
10 activity. This is further supported by the findings of Seals (1989) which demonstrated that MSNA and
11 vascular resistance were tightly coupled during exercise. The findings of Hopman et al. (1993) are also
12 of interest as they found that in spinal cord injured participants, with no sympathetic activity in their
13 lower limbs, had no change in calf volume during arm cranking. The current study demonstrated that
14 the reduced calf volume decrease during POST-ABS when compared to PRE and POST-REL is likely
15 due to reduced sympathetic activity acting directly on the blood vessels as a result of the POST-ABS
16 exercise intensity representing a lower proportion of the post training W_{peak} .

17

18 Although there was a decrease in calf volume during exercise in all trials there was a concomitant
19 increase in whole limb calf blood flow on the cessation of exercise compared to rest. This increase was
20 most likely due to increased skin blood flow during exercise, which supports the work of Theisen et al.
21 (2000, 2001a, 2001b). Since the calf is relatively metabolically inactive during arm exercise any
22 changes in blood flow using venous occlusion plethysmography is likely a result of skin blood flow
23 (Johnson and Rowell, 1975). Therefore, the greater blood flow at the cessation of exercise could be
24 indicative of an increase in skin blood flow during exercise, a response which has been demonstrated
25 by Theisen et al. (2001a, 2001b) using Laser Doppler Flowmetry. In addition increases in calf heat flow
26 which were noted during exercise could reflect increases in calf skin blood flow allowing greater dry
27 heat exchange as suggested by Sawka et al. (1984). The increase in core temperature observed during
28 exercise in the present study could have stimulated an increase in skin blood flow at the calf suggesting
29 that the vasoconstriction in the calf, as demonstrated by the decrease in calf volume, may be related to
30 increasing venous return to support increases in skin blood flow.

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In conclusion, upper body training increased the traditional whole body markers of aerobic fitness. Upper body exercise training reduced aural temperature and heat storage at an absolute exercise intensity as a result of increased whole body sweating and increased heat flow. However, the results of this study suggest upper body exercise training elicits different localised physiological responses to that of lower body training studies, specifically in the lower leg. Training elicited a warmer calf skin temperature at rest and during exercise possibly due to changes in calf skin blood flow and heat flow. Upper body aerobic exercise training produced an attenuated reduction in calf volume change during POST-ABS demonstrating less blood flow being redirected away from the lower body, which was most likely a result of a reduced response to sympathetic nervous activity and reduced vasoconstriction at a lowered relative exercise intensity.

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Table I: The mean (\pm SD) physiological responses obtained during the last 30 seconds of the preliminary incremental $\dot{V} O_{2\text{peak}}$ tests (n=13). *significant difference between PRE and POST, † between PRE and MID and ‡ between MID and POST (P<0.05)

	PRE	MID	POST	% change (POST-PRE)
$\dot{V} O_{2\text{peak}}$ (l.min ⁻¹)	2.03 (\pm 0.41)	2.23 (\pm 0.48) †	2.43 (\pm 0.50)* ‡	18.9 (\pm 14.6)
$\dot{V} O_{2\text{peak}}$ (ml.kg.min ⁻¹)	27.4 (\pm 5.6)	27.9 (\pm 10.0) †	32.1 (\pm 5.6)* ‡	18.9 (\pm 14.6)
HR _{peak} (beats.min ⁻¹)	178 (\pm 12)	180 (\pm 14)	183 (\pm 11)	2.7 (\pm 5.7)
W _{peak} (W)	120 (\pm 26)	141 (\pm 28) †	158 (\pm 30)* ‡	29.9 (\pm 12.8)
60%W _{Peak}	72 (\pm 16)	85 (\pm 17) †	93 (\pm 17)* ‡	29.9 (\pm 12.8)
75%W _{peak}	90 (\pm 20)	106 (\pm 21) †	116 (\pm 21)* ‡	29.9 (\pm 12.8)
RPE _{central} (Borg Scale)	16 (\pm 2)	17 (\pm 2) †	18 (\pm 2)*	13.5 (\pm 15.8)
RPE _{local} (Borg Scale)	18 (\pm 2)	19 (\pm 1)	20 (\pm 1)*	9.7 (\pm 15.6)
Systolic BP (mmHg)	116 (\pm 7)	112 (\pm 8)	113 (\pm 10)	-2.5 (\pm 7.3)
Diastolic BP (mmHg)	76 (\pm 6)	77 (\pm 5)	74 (\pm 6)	-2.0 (\pm 11.3)
Body Fat (%)	17.8 (\pm 5.2)		17.4 (\pm 4.7)	-1.0 (\pm 9.3)
Sum of 8 sites (mm)	95.0 (\pm 41.9)		92.9 (\pm 39.9)	-1.5 (\pm 6.5)
Biceps Relaxed (cm)	30.9 (\pm 3.0)		31.6 (\pm 2.9)	2.3 (\pm 2.9)
Biceps Tensed (cm)	32.6 (\pm 2.7)		33.8 (\pm 2.7)*	3.7 (\pm 2.8)
Body Mass (kg)	75.9 (\pm 9.8)		74.6 (\pm 9.3)	-0.1 (\pm 2.0)

Table II: Mean (\pm SD) physiological responses at the cessation of arm exercise during each submaximal trial (n=13). * significant difference from PRE. † denotes significant difference between POST-ABS and POST-REL.

	PRE	POST-ABS	POST-REL
$\dot{V} O_2$ (l.min ⁻¹)	1.67 (\pm 0.26)	1.40 (\pm 0.27)*	1.80 (\pm 0.37)* †
HR (beats.min ⁻¹)	155 (\pm 12)	127 (\pm 13)*	156 (\pm 12) †
Bla (mmol.l ⁻¹)	4.5 (\pm 1.1)	2.9 (\pm 1.5)*	5.2 (\pm 1.4) †
RPE _{central} (Borg Scale)	15 (\pm 2)	12 (\pm 1)*	15 (\pm 2) †
RPE _{local} (Borg Scale)	17 (\pm 2)	12 (\pm 2)*	16 (\pm 2)* †
Sweat rate (l.hr ⁻¹)	0.6 (\pm 0.6)	0.8 (\pm 0.6)	1.0 (\pm 0.6)*

List of Figures:

Figure I: Mean (\pm SEM) a) aural and b) rectal temperature during exercise and passive recovery for each submaximal trial (n=13). *significant difference from POST-ABS.

Figure II: Mean (\pm SEM) a) calf, b) thigh, c) Upper arm and d) back skin temperatures during exercise and passive recovery. Δ significant difference from PRE. *significant difference from POST-ABS. †significant difference POST-REL.

Figure III: Mean (\pm SEM) heat flow measurements for a) calf, b) thigh, c) upper arm and d) chest for all three trials (n=13). Δ significant difference from PRE. *significant difference from POST-ABS. †significant difference POST-REL.

Figure IV: a) Percentage change in calf volume during exercise for each trial (mean \pm SEM; n=13). * denotes significant difference from PRE, b) Mean (\pm SEM) calf blood flow measurements at rest and during passive recovery for all trials (n=13).