The Effect of Arm Training on Thermoregulatory Responses and Calf Volume during Upper Body Exercise

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Abstract:

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

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

Abbreviations:

Analysis of Variance	ANOVA
Blood lactate	Bla
Degrees Centigrade	°C
Haemoglobin	Hb

Haematocrit	Hct
Heart Rate	HR
Kilogram	Kg
Microlitres	µl
Mid training trial	MID
Minute ventilation	VE
Peak Oxygen Consumption	$\dot{V}O_{2peak}$
Post training absolute intensity	POST-ABS
Post training relative intensity	POST-REL
Peak Power	W_{peak}
Pre training trial	PRE
Ratings of perceived exertion	RPE
Revolutions per minute	$rev.min^{-1}$
Standard Deviation	SD
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 (Stolwijik ,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 ±SD age 27.8 ±5.0yrs, body mass 74.8 ±9.5kg, body fat percentage of 17.8± 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 ($\dot{V}O_{2peak}$) 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 ($\dot{V}O_{2peak}$), 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 $\dot{V}O_{2peak}$ 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 µl capillary blood samples were also taken for measurement of haematocrit (Hct) using a micro haematorcrit 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. VO_2, 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.

Results

Peak physiological responses

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

Physiological and Thermoregulatory Responses during Submaximal Exercise Trials: =

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

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

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

Core Temperature during Exercise and Passive Recovery

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

Skin temperature Responses during Exercise and Passive Recovery

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

Heat Storage

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

Heat Flow during Exercise and Passive Recovery

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

Calf Volume and Blood Flow during Exercise and Passive Recovery

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

Discussion

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

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

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

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

(1966) and Gant et al. (2004) noted a correlation between exercise intensity (%VO_{2max}) and rectal temperature during lower body exercise suggesting that rectal temperature was dependent on exercise intensity. However, the present study showed no correlation between the percentage of $\dot{V}O_{2peak}$ with core temperature responses pre and post training suggesting that there may be differences in heat dissipation between upper and lower body exercise and core temperature estimates.

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

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

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

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

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

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

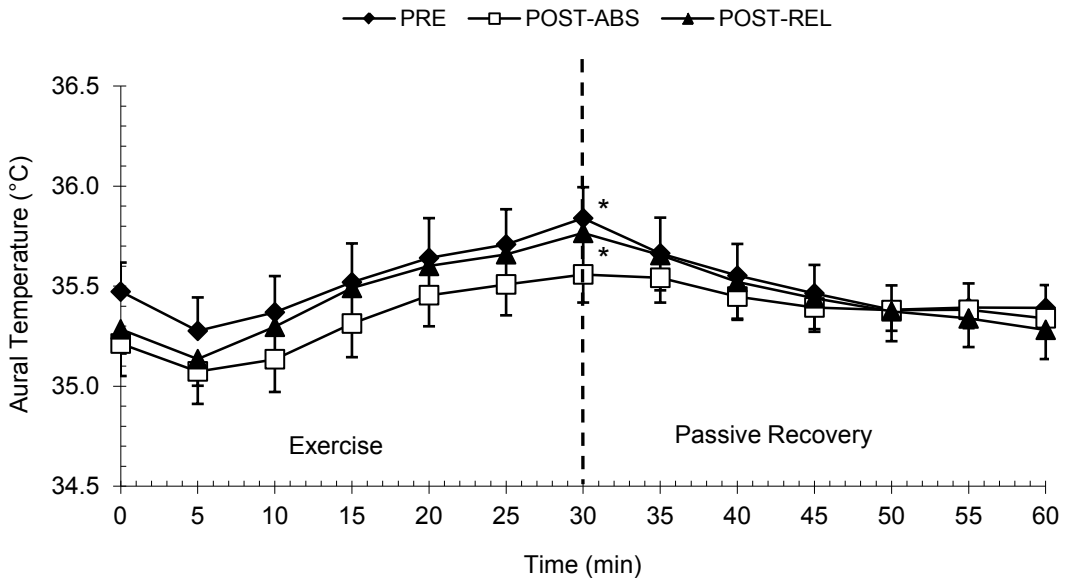
	PRE	MID	POST	% change (POST-PRE)
$\dot{V}O_{2peak}$ ($l.min^{-1}$)	2.03 (±0.41)	2.23 (±0.48) †	2.43 (±0.50)* ‡	18.9 (±14.6)
$\dot{V}O_{2peak}$ ($ml.kg.min^{-1}$)	27.4 (±5.6)	27.9 (±10.0) †	32.1 (±5.6)* ‡	18.9 (±14.6)
HR_{peak} ($beats.min^{-1}$)	178 (±12)	180 (±14)	183 (±11)	2.7 (±5.7)
W_{peak} (W)	120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

Table II: Mean (±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.

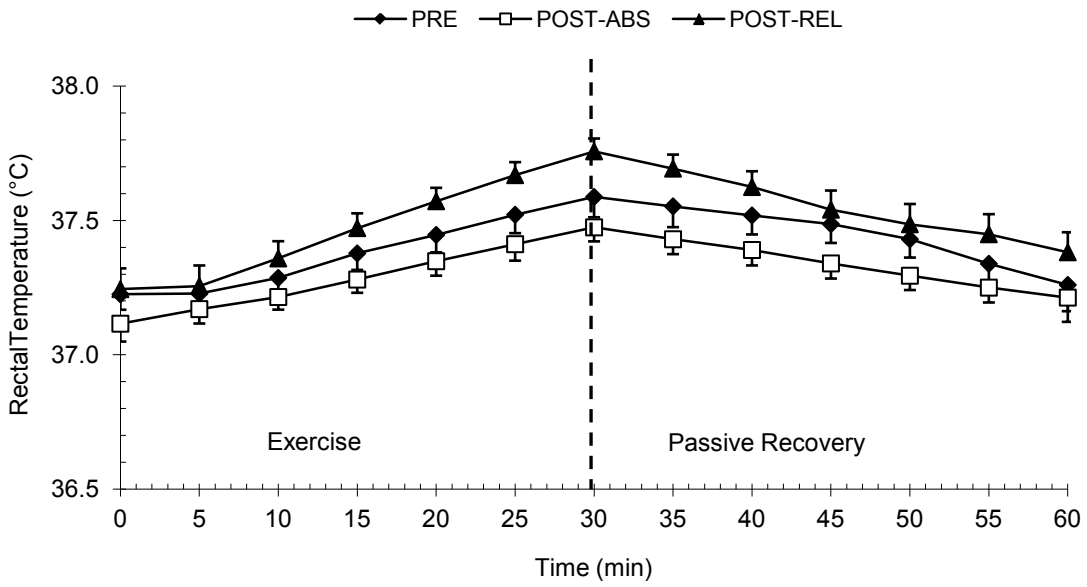
	PRE	POST-ABS	POST-REL
$\dot{V}O_2$ (l.min^{-1})	1.67 (±0.26)	1.40 (±0.27)*	1.80 (±0.37)* †
HR (beats.min^{-1})	155 (±12)	127 (±13)*	156 (±12) †
Bla (mmol.l^{-1})	4.5 (±1.1)	2.9 (±1.5)*	5.2 (±1.4) †
$RPE_{central}$ (Borg Scale)	15 (±2)	12 (±1)*	15 (±2) †
RPE_{local} (Borg Scale)	17 (±2)	12 (±2)*	16 (±2)*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

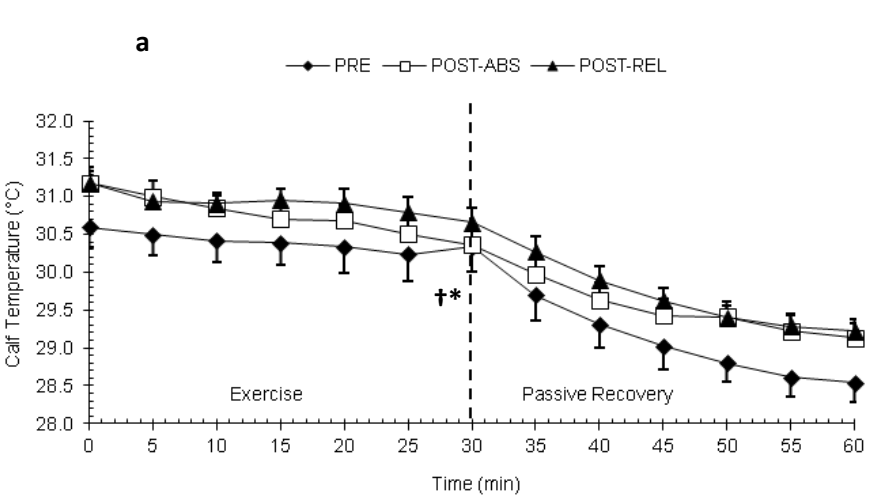
List of Figures:

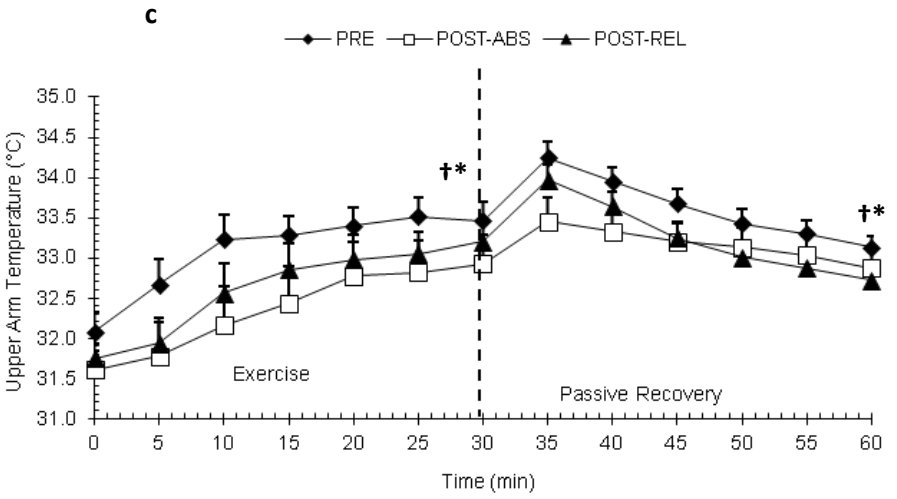
a


PRE
POST-ABS
POST-REL
Aural Temperature (°C)
36.5
36.0
35.5
35.0
34.5
*
*
Exercise
Passive Recovery
0
5
10
15
20
25
30
35
40
45
50
55
60
Time (min)

b


PRE
POST-ABS
POST-REL
RectalTemperature (°C)
38.0
37.5
37.0
36.5
Exercise
Passive Recovery
0
5
10
15
20
25
30
35
40
45
50
55
60
Time (min)


a
PRE
POST-ABS
POST-REL
Calf Temperature (°C)
32.0
31.5
31.0
30.5
30.0
29.5
29.0
28.5
28.0
†*
Exercise
Passive Recovery
0
5
10
15
20
25
30
35
40
45
50
55
60
Time (min)

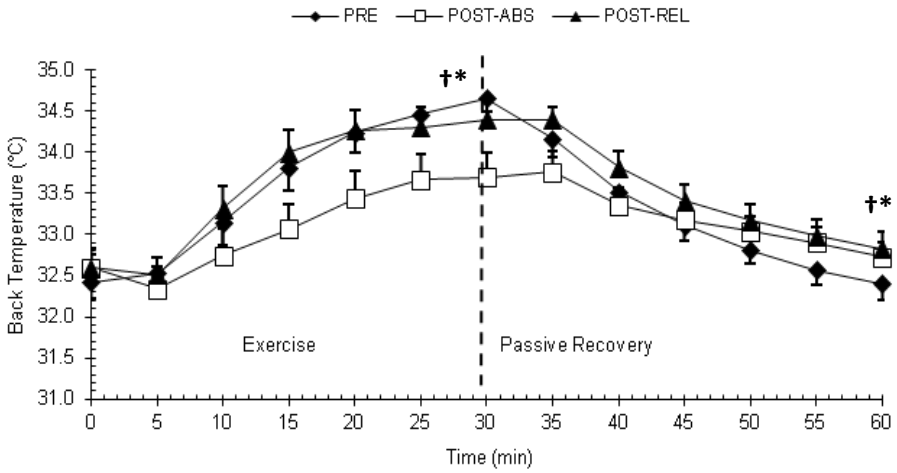

c
PRE
POST-ABS
POST-REL
Upper Arm Temperature (°C)
35.0
34.5
34.0
33.5
33.0
32.5
32.0
31.5
31.0
†*
†*
Exercise
Passive Recovery
0
5
10
15
20
25
30
35
40
45
50
55
60
Time (min)

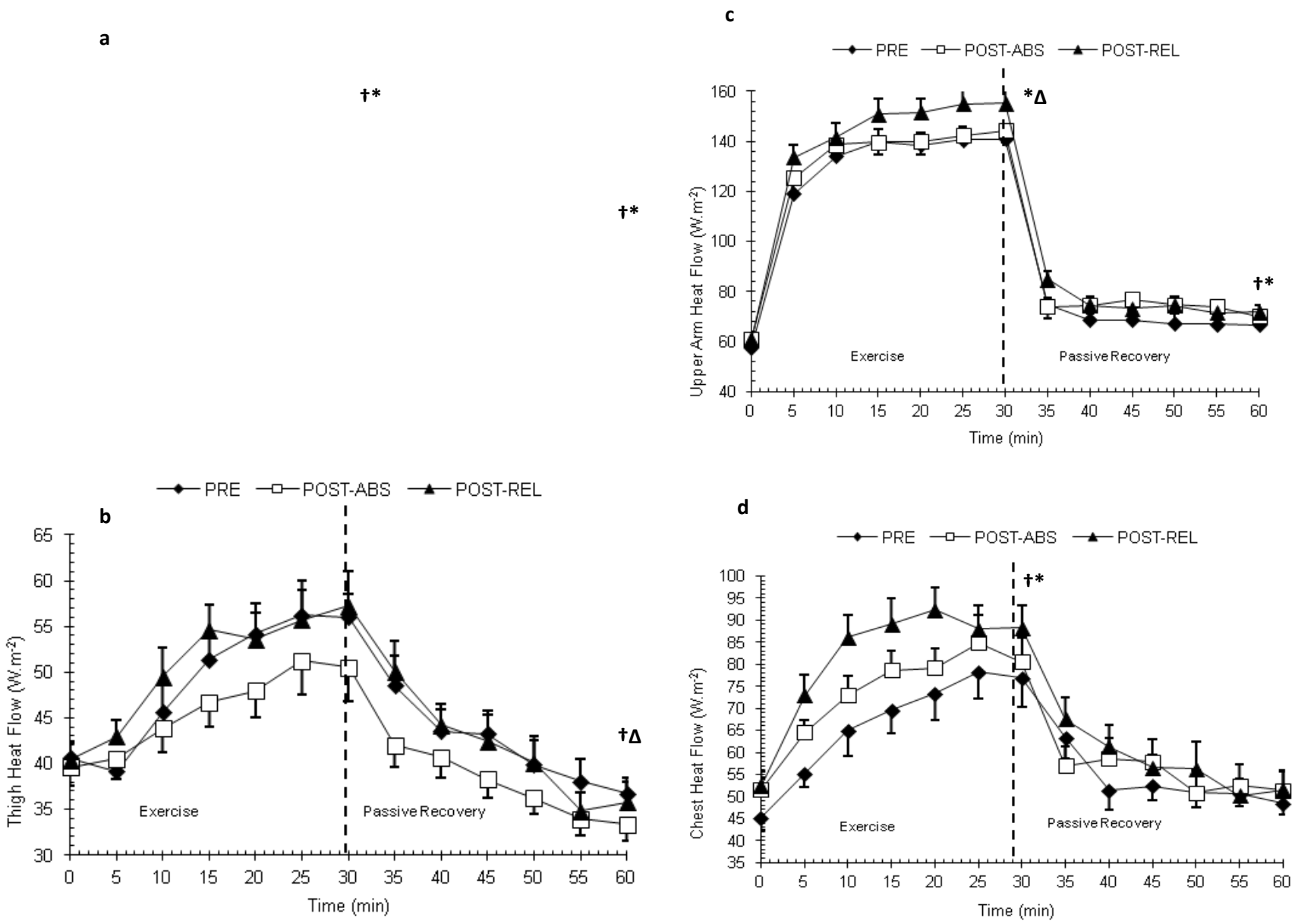
b

†Δ

*Δ

d

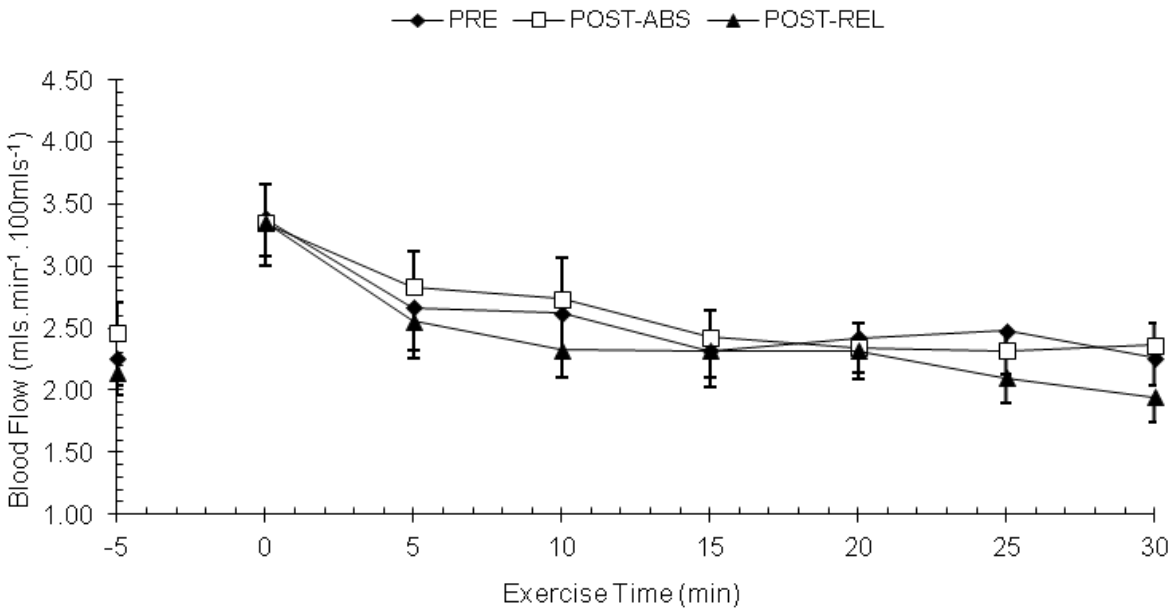

PRE
POST-ABS
POST-REL
Back Temperature (°C)
35.0
34.5
34.0
33.5
33.0
32.5
32.0
31.5
31.0
†*
†*
Exercise
Passive Recovery
0
5
10
15
20
25
30
35
40
45
50
55
60
Time (min)


a
†*
†*
b
PRE
POST-ABS
POST-REL
Thigh Heat Flow (W.m-2)
Exercise
Passive Recovery
Time (min)
†Δ
c
PRE
POST-ABS
POST-REL
Upper Arm Heat Flow (W.m-2)
*Δ
†*
Exercise
Passive Recovery
Time (min)
d
PRE
POST-ABS
POST-REL
Chest Heat Flow (W.m-2)
†*
Exercise
Passive Recovery
Time (min)

a

*

b


PRE
POST-ABS
POST-REL
Blood Flow (mls.min-1.100mls-1)
4.50
4.00
3.50
3.00
2.50
2.00
1.50
1.00
-5
0
5
10
15
20
25
30
Exercise Time (min)