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Short communication

Attenuation of the cutaneous blood flow response during combined exercise and heat stress

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Abstract. Skin blood flow (SkBF) was measured in six male subjects using laser-Doppler velocimetry, with zero-gradient auditory canal temperature (T_{ac}) used as an index of body core temperature (T_c). Subjects performed incremental, upright cycling commencing at 40% peak power ($\dot{W}_{peak}$: 10 min), increasing every 4 min by 5% $\dot{W}_{peak}$ thereafter. Trials were conducted in hot (ambient temperature (T_a) 36.7 ±0.2°C, relative humidity (rh) 46.1 ±3.2%; $\bar{X}$ ±S.D.), and neutral environments (T_a 19.6 ±0.3°C, rh 50.2 ±1.4%). SkBF increased with T_{ac} in all subjects. Attenuation of SkBF occurred at the same T_{ac}, relative SkBF and cardiac frequency (f_c) between environments, but at a lower exercise intensity (40.8 ±0.8% versus 55.8 ±3.0% $\dot{W}_{peak}$) in the hot environment ($p<0.05$). Data indicate that T_c thresholds for SkBF attenuation may exist. However, it is suggested that attenuation thresholds coincided with a reduced central blood volume, which may occur at a critical level of cutaneous blood pooling.

Key words: Exercise - Laser-Doppler velocimetry - Skin blood flow - Temperature regulation

Introduction

At the initiation of exercise, the demand for blood at the active muscles increases immensely, resulting in a reduction in blood flow to the inactive muscles, skin and viscera in an approximately inverse proportion to exercise intensity (Rowell 1983). An increase in cutaneous vasoconstrictor tone at the commencement of exercise causes body heat accumulation, eventually resulting in active vasodilation. The exercise intensity governs the core temperature (T_c) threshold for vasodilation, which is greater at higher intensities (Mack et al. 1994). The resulting vasodilation allows an increase in heat flow from core to skin, where heat exchange takes place with the environment, permitting thermal homeostasis during submaximal exercise.

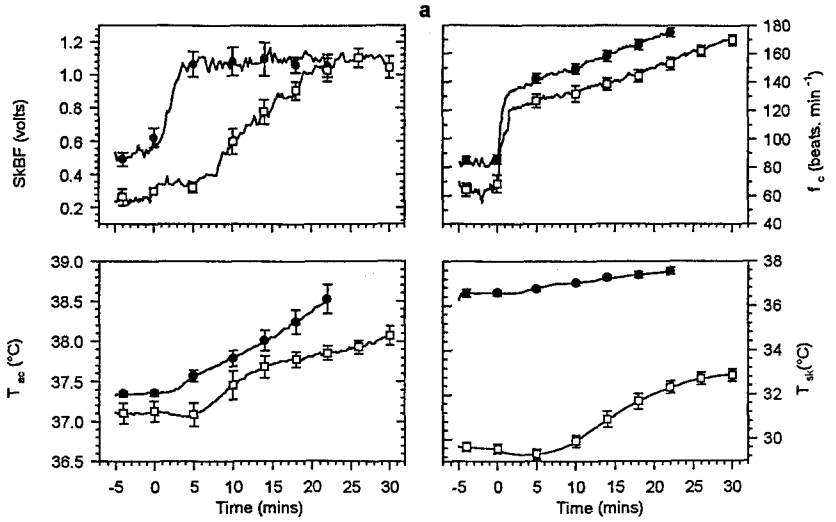
Further elevation in exercise intensity increases demand for blood flow at the active muscles, and elevates metabolic heat production. Cardiac output will increase to service both metabolic and cutaneous thermal needs (Nadel et al. 1979). However, when cardiac output peaks, skin blood flow (SkBF) will be reduced to maintain systemic blood pressure (Rowell 1983). This SkBF attenuation will compromise heat dissipation, allowing core temperature to escalate. It was the aim of this study to identify relative exercise intensities at which SkBF attenuation occurs during incremental exercise, with reference to variations between thermal environments.

Methods

Six active, healthy males, without a history of recent heat exposure, volunteered as subjects: age 21.3 ±1.7 yr (mean ±S.D.), height 181.1 ±4.5 cm, weight 74.86 ±9.21 kg, aerobic power ($\dot{V}_{O2peak}$) 4.92 ±0.23 l·min^{-1}. Following provision of informed consent, subjects performed two exercise trials, seven days apart, at the same time of day, in neutral (T_a 19.6 ± 0.25°C, rh 50.2 ± 1.35%) and hot (T_a 36.7 ± 0.15°C, rh 46.1 ± 3.17%) environments. $\dot{V}_{O2peak}$ cycle tests were conducted using a ramp protocol (36 W·min^{-1}), to obtain experimental work rates.

Trials started with a 25 min adaptation, with data collection commencing 5 min prior to exercise. Using an electronically-braked ergometer (Excalibur, Quinton, USA), cycling began at 40% $\dot{W}_{peak}$ (10 min), increasing every 4 min by 5% $\dot{W}_{peak}$. Aerodynamic bars were used to control body position and minimise forearm movement. Tests ended after 30 min, if auditory canal temperature (T_{ac}) reached ≥39°C, or at the subject's request. All completed the neutral trials. In the hot condition, 5 subjects completed 26 min of exercise, with the 6th terminating after 22 min due to fatigue.

T_{ac} (zero-gradient aural thermometry; Keatinge and Sloan 1975) was continuously measured as an indicator of core temperature. Skin temperatures were measured at eight sites (YSI EU mini-thermistors, USA) and recorded using a Squirrel data logger (Grant, 1200 series, UK) at 0.2 Hz. Mean skin temperature ($\bar{T}_{sk}$) was calculated as the area-



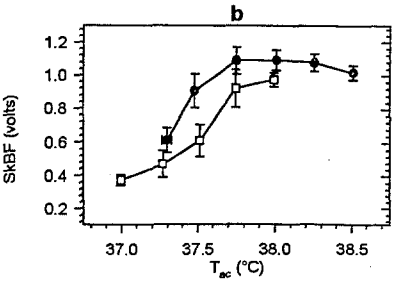


Fig 1. Skin blood flow (SkBF), auditory canal temperature (T_{ac}), cardiac frequency (f_c) and mean skin temperature ($\overline{T}_{sk}$) as functions of time (a), and T_{ac} (b). Data are means ±S.E.M. (□ = neutral and ● = hot environment).

weighted average of the 8 sites. SkBF was measured at the right forearm on the mid-radial ventral surface (laser-Doppler velocimetry, TSI Laserflo BPM^2, USA; λ=780nm, fibre separation of 0.5mm; SkBF expressed in voltage units). SkBF and T_{ac} data were converted to digital equivalents (Computerboards Inc, PPIO-A108, USA) and recorded at 1 Hz on a portable computer. Cardiac frequency (f_c) was recorded at 0.2 Hz (Polar Electro, Sports Tester model PE3000, Finland). Thermal sensation and perceived exertion (RPE; 15 point-Borg Scale) votes were recorded at 2 min intervals.

SkBF, T_{ac}, $\overline{T}_{sk}$ and f_c were averaged at 2 min intervals, and analysed using a repeated-measures ANOVA, to evaluate between-trial differences. Paired *t*-tests were used to compare differences between variables at SkBF vasodilatory thresholds and attenuation levels.

Results

Throughout the hot trial, f_c, T_{ac} and $\overline{T}_{sk}$ were significantly elevated ($p<0.05$; Fig. 1a) at each point in time, in comparison to levels achieved in the neutral environment. Thermal sensation and RPE were elevated (RPE 16.5 ±2.2 versus 18.3 ±0.6; sensation 8.9 ±0.9 versus 11.2 ±0.7; $p<0.05$; mean ±S.E.M), and exercise duration was reduced (30.0 ±0.0 versus 25.3 ±0.7 min; $p<0.001$). T_{ac} vasodilation thresholds were similar in both trials, although the onset time was earlier in the heat (Table 1). Exercise intensity at these thresholds did not differ, since thresholds occurred in the first 10 min (40% $\dot{W}_{peak}$). SkBF gain (relative to T_{ac}) was greater in the heat (Table 1).

SkBF attenuation was observed in all subjects in both conditions, occurring at the same T_{ac} between environments (Fig. 1b), while the exercise intensity at attenuation was lower in the hot environment (Table 1). Work rates at attenuation were 158.7 ±7.8 and 213.7 ±13.4 watts in the hot and neutral states respectively ($p<0.05$), while SkBF and f_c did not differ between conditions at this point ($p>0.05$).

Table 1. Cutaneous vasodilatory and attenuation thresholds during incremental exercise in neutral and hot environments.

	Cool	Hot	*p* value
T_{ac} threshold (°C)	37.31 ±0.04	37.27 ±0.13	*NS*
$\overline{T}_{sk}$ threshold (°C)	29.54 ±0.24	36.59 ±0.12	<0.001
Onset vasodilation (seconds)	761.8 ±48.3	318.3 ±26.6	0.001
SkBf:T_{ac} gain (Volts·°C^{-1})	1.01 ±0.15	3.65 ±1.04	0.05
T_{ac} attenuation (°C)	37.8 ±0.04	37.6 ±0.09	*NS*
SkBF attenuation (Volts)	1.11 ±0.04	1.07 ±0.03	*NS*
$\overline{T}_{sk}$ attenuation (°C)	32.01 ±0.38	36.85 ±0.13	<0.001
f_c attenuation (beats·min^{-1})#	151.8 ±2.0	141.2 ±14.0	*NS*
Exercise intensity attenuation (%$\dot{W}_{peak}$)	55.8 ±3.0	40.8 ±0.8	0.007

Data are means ±S.E.M., $n=6$ ($^{\#}n=5$); *p* values from paired *t*-test; *NS*=$p>0.05$.

Discussion

In the hot trials, there was a 27% reduction in the relative exercise intensity at which SkBF attenuation occurred. While our aim was to determine these relative exercise intensities, results indicate that it may be more appropriate to conclude that SkBF attenuation occurs at a common T_c, regardless of exercise intensity. This supports observations of Brengelmann et al. (1977), who observed a reduction in SkBF gain at a T_c of 38°C, and of Smolander et al. (1987), who observed a similar T_c at SkBF attenuation in two thermal environments. This phenomenon may be

explained by reduced cutaneous vasodilator gain to a given T_c elevation, or baroreceptor-induced vasoconstriction.

There were no between-condition differences in SkBF at attenuation, and there was an elevated SkBF gain in the heat. It is therefore assumed that SkBF attenuation was equivalent between conditions, and occurred to maintain arterial pressure. If we assume forearm SkBF is an accurate indicator of whole body SkBF, then the blood volume displaced to the periphery would be approximately equal between conditions. Therefore, it seems that a critical level of cutaneous blood pooling is achieved prior to attenuation; this being relatively independent of metabolic demands, since exercise intensity differed at attenuation between trials. Since SkBF remained constant after attenuation, with a further 71.5 watt increase in work rate in the heat, additional muscle blood flow would be drawn from inactive tissues other than the skin.

Three questions concerning the validity of the laser-Doppler technique require discussion. First, since Smolander et al. (1987) observed a greater SkBF at attenuation in hot compared to cool environments, it may be argued that signal saturation occurred, producing equivalent SkBF between trials. Saturation was unlikely, since SkBF at attenuation was less than 50% of the output range of the apparatus, with larger SkBF being observed following the application of an exogenous vasodilatory agent (unpublished observations). Second, while laser-Doppler velocimetry is sensitive to changes in both blood volume and cell velocity, it may be less sensitive to small fluctuations in blood volume (Smolander and Kolari 1985). If blood volume and SkBF increase, without significant changes in red cell velocity, then SkBF may increase without being detected. This effect is based upon conjecture, and its presence and influence on the current data are unknown. Finally, it may be suggested that SkBF attenuation was simply a reflection of region-specific, maximum SkBF being attained within each condition. Evidence against this possibility comes from the work of Brengelmann et al. (1977), who observed a further increase in SkBF when local heating was applied to one forearm, after SkBF attenuation had been induced experimentally.

The autonomic control of SkBF during thermal stress has been shown to be primarily dependent on changes in T_c, with $\overline{T}_{sk}$ playing a relatively minor role (Wyss et al. 1974). The present data appear to extend this observation, since, at attenuation, SkBF and T_{ac} were equivalent between trials, while $\overline{T}_{sk}$ differed significantly. Therefore, T_c was either a major contributory factor, or coincided with another factor which caused displacement of blood from the periphery to the core. However, $\overline{T}_{sk}$ also plays a role in this control, since the greater gain in SkBF in the hot environment may be attributed to a higher $\overline{T}_{sk}$. The increased exercise duration, and greater exercise intensity attained at attenuation in the neutral state was probably due to a reduced SkBF gain and increased core:skin:air temperature gradients.

It is suggested that attenuation of SkBF may result from baroreflex control, rather than simply a redistribution of blood flow to supply the metabolic demands of the active muscles, or simply the influence of T_c. It is difficult to conceive how the attainment of a critical T_c could result in a reduction of the SkBF response to the rising T_c. Instead, it is more likely that the T_c attenuation thresholds coincided with reduced central blood volume, and its resultant baroreceptor activation. It is suggested that a reduction in central blood volume may have occurred at a critical level of cutaneous blood pooling. Therefore, the differences in exercise intensity at SkBF attenuation within this study appear to be a reflection of environmental temperature, and its influence on central blood volume and the volume of blood in the cutaneous vessels.

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