

Sweating responses to a sustained static exercise is dependent on thermal load in humans

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ABSTRACT

The purpose of this project was to test the hypothesis that internal temperature modulates the sweating response to sustained handgrip exercise. Ten healthy male subjects immersed their legs in 43 °C water for 30–40 min at an ambient temperatures of 30 °C and a relative humidity of 50%. Sweating responses to 50% maximal voluntary contraction isometric handgrip exercise (IH) were measured following the onset of sweating (i.e. following slight increases in internal temperature), and after more pronounced increases in internal temperature. Oesophageal temperature (T_{es}) was significantly lower during the first bout of exercise (37.54 ± 0.07 °C) relative to the second bout (37.84 ± 0.12 °C; $P < 0.05$). However, the increase in mean sweating rate (SR) from both the chest and forearm (non-glabrous skin) was significantly greater during the first IH bout relative to the second bout ($P < 0.05$). Increases in mean arterial blood pressure and palm SR (glabrous skin) did not differ significantly between exercise bouts, while heart rate and rating of perceived effort were significantly greater during the second bout of IH. As T_{es} and mean skin temperature did not change during either bout of exercise, the changes in SR from non-glabrous skin between the bouts of IH were likely because of non-thermal factors. These data suggest that sweating responses from non-glabrous skin during IH vary depending on the magnitude of thermal input as indicated by differing internal temperatures between bouts of IH. Moreover, these data suggest that the contribution of non-thermal factors in governing sweating from non-glabrous skin may be greatest when internal temperature is moderate (37.54 °C), but has less of an effect after greater elevations in internal temperature (i.e. 37.84 °C).

Keywords cutaneous vascular response, isometric handgrip exercise, non-thermal factors, thermal factors.

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During dynamic exercise, increased heat loss associated with evaporation, is an important factor in maintaining a stable internal temperature within a limited range. This is necessary because excessive increases in internal temperature could evoke central nervous system failure. During dynamic exercise, sweating rate (SR) from non-glabrous skin such as forearm and chest is controlled by both thermal (e.g. internal and skin temperatures) (Benzinger 1959, Nielsen 1969, Nadel *et al.* 1971, Johnson & Park 1981, Kondo *et al.* 1998) and non-thermal factors (e.g. central command, mechanoreceptors and metaboreceptors) (Van Beaumont & Bullard 1963, 1966, Gisolfi & Robinson 1970, Yamazaki *et al.*

1994, Kondo *et al.* 1997, 1999). However, the interaction between these thermal and non-thermal factors in the control of sweating response remains unclear.

After sweating has been activated following a heat load, SR from non-glabrous skin increases immediately upon the onset of dynamic exercise, and this increase is independent of changes in internal and skin temperatures (Van Beaumont & Bullard 1963). Similarly, after sweating has been initially activated, isometric handgrip exercise (IH) causes further increases in SR, with essentially no accompanying change in internal temperature (Van Beaumont & Bullard 1966, Kondo *et al.* 1999, 2000, Shibasaki *et al.* 2001). Finally, increases

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in forearm sweating have been observed in normo-thermic subjects when acetylcholine hydrolysis was inhibited via local administration of neostigmine (Shibasaki *et al.* 2001). These results clearly demonstrate that SR can be altered by non-thermal factors, however, sweating must either be activated, or acetylcholine hydrolysis must be inhibited, to observe this response. Also, in these studies the sweating response to non-thermal factors was shown when sweating was induced by increasing skin temperature without a marked change in internal temperature from rest. Moreover, the effect of activation of the muscle metaboreflex on sweating varies between non-glabrous skin and glabrous skin such as the palm (Kondo *et al.* 1999).

After increasing internal temperature during dynamic exercise, two variables (the internal temperature threshold for the onset of sweating and the sensitivity of the response) for control of sweating were not influenced by exercise compared with passive heating (Johnson & Park 1981, Kellogg *et al.* 1991). As dynamic exercise involves the integrated responses of simultaneous thermal and non-thermal reflexes and internal temperature is a predominant thermal input to sudomotor centre compared with skin temperature (Nadel *et al.* 1971), internal temperature (thermal factor) appears to predominate over non-thermal factors in the control of sweating during dynamic exercise. Also, it has been reported that forearm blood flow is decreased from rest at the initiation of exercise by non-thermal factors, and that this reduction can be attenuated by sufficient hyperthermia (Johnson & Park 1982).

Based on these results of the effect of thermal and non-thermal factors on sweating response in humans, what remains unknown is whether the degree of thermal input to thermoregulatory centres (i.e. moderate or high internal temperatures) modulates the influence of the aforementioned non-thermal factors in controlling sweating. Also, it is unclear that there are regional differences in the interaction between thermal and non-thermal factors in the control of sweating response. Thus, the purpose of this study was to test the hypothesis that the influence of non-thermal factors in the control of sweating from non-glabrous skin and glabrous skin is modulated by the magnitude of thermal load (i.e. internal temperature). To test this hypothesis, sweating responses on non-glabrous and glabrous skin were measured during IH when internal temperature was moderate and high by passive heating.

METHODS

Subjects

Ten healthy male subjects with a mean age of 21.8 ± 2.2 years (\pm SD), height 170.6 ± 5.3 cm, and

weight 62.4 ± 7.5 kg participated in the study. None of the subjects was taking any medications and all were non-smokers. Each subject was informed of the purpose and procedures involved in the study and written consent was obtained. Subjects were prohibited from eating at least 2 h before each test. This study was approved by Human Committee in our department.

Experimental protocol

All experiments were conducted in an environmental chamber (SR-3000, Nagano Science Co., Osaka, Japan). Figure 1 shows a schematic of the first experimental protocol. Ambient temperature was maintained at 30 °C, with a relative humidity of 50% and minimal air movement. After entering the chamber each subject rested in the sitting position for ~60 min, while wearing only shorts. During this time, each subject was instrumented for data collection, followed by each subject performing two maximal voluntary contractions (MVC) with the right arm using a handgrip dynamometer. The higher of the two values were used to determine the relative workload (i.e. % MVC). Baseline data were recorded for 5 min at rest, then the subject immersed both legs, below the knee, into a 43 °C water bath. Upon the onset of sweating, 50% MVC IH was performed for 60 s using the right hand. After this bout, the subject rested, with his legs remaining in the warm water, until SR and internal temperature reached a plateau; this was followed by a second bout of IH at the same intensity as the first IH bout, also using the right hand. For both exercise bouts the subject used a visual feedback system to maintain the force of the handgrip. As the movements of the thoracic cage (respiration) influence skin sympathetic nervous activity (Delius *et al.* 1972) and thus may influence SR, respiratory frequency was maintained at 12 breaths min^{-1} for 1 min rest (prior to IH bouts) and during both IH bouts. After the second IH bout, room temperature was elevated to 38 °C, while SR was continually monitored. This procedure was performed to confirm that sweating responses observed during the second bout of IH were not saturated and sweating rate reached a maximal value.

On a different day, a second experiment was performed in four subjects to investigate whether differences in sweating responses between the first and second bouts of IH in the first experiment were affected by the order of the procedure. We measured the sweating response to 1 min IH of 50% MVC twice, separated by 20 min. Ambient temperature was 35 °C and relative humidity was 50% in both cases. Subjects had rested for 60 min to allow SR to reach a steady-state before the first IH. The interval between the two bouts of IH was determined from the average interval for the two bouts of IH in the first experiment. We

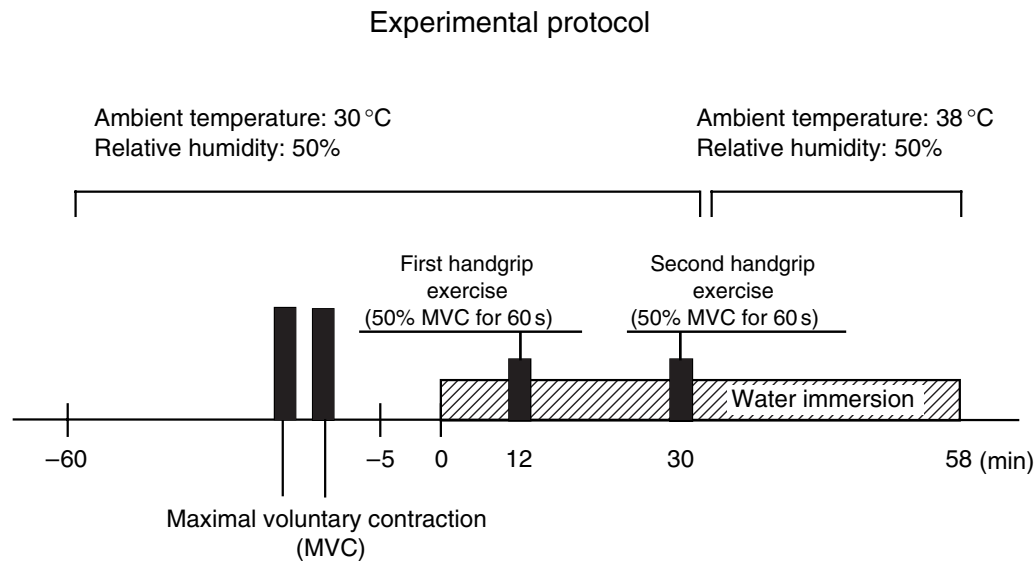


Figure 1 Schematic of the experimental protocol of this study. Each subject immersed their legs in 43 °C water, followed by performing a bout of isometric handgrip exercise at the onset of sweating and after further increases in internal temperature. After the second bout of IH, the temperature of the climatic chamber was elevated from 30 to 38 °C. This figure represents the timing of events for one subject, which does not necessarily depict the timing of the events for all subjects.

selected the environmental conditions to cause sudomotor activity at rest.

Measurements

In all experiments, the following variables were recorded: oesophageal temperature (T_{es}), local skin temperature at eight sites (chest, forearm, palm, forehead, abdomen, thigh, lower leg and foot), SR from the left forearm and chest (non-glabrous skin) and from the left palm (glabrous skin), skin blood flow from the left forearm and chest, heart rate (HR), systolic and diastolic arterial blood pressure, and rating of perceived effort (RPE). The T_{es} and local skin temperature were measured with thermocouples. The tip of the T_{es} thermocouple was inserted in the esophagus to a distance equal to one-quarter of the subject's height. Mean skin temperature (T_{sk}) was calculated according to the method of Hardy & DuBois (1938). The SR was measured continuously using the ventilated capsule method. Dry nitrogen gas was supplied to each capsule (forearm and chest: 7.06 cm², palm: 1.53 cm²) at a rate of 1.5–2.0 L min⁻¹ and the relative humidity of the effluent nitrogen was measured using a capacitance hygrometer (HMP 133Y, Vaisala, Helsinki, Finland). Time delay of our system for measuring SR was 1 s and was counted for calculating SR. Changes in skin blood flow were indexed continuously using laser Doppler velocimetry (ALF21, Advance, Tokyo, Japan). Cutaneous vascular conductance (CVC) was calculated for each site from the ratio of skin blood flow to mean arterial pressure (MAP). The SR and CVC from

non-glabrous skin were determined from the mean value of the chest and forearm sites to compare the regional difference in the responses clearly. The probes for measurement of skin blood flow were placed within 1 cm of the ventilated capsule. Temperatures, SR and skin blood flow were recorded at 1 s intervals, and stored on a personal computer (PC9801RA, NEC Co., Tokyo, Japan) using a data logger (HR2300, Yokogawa Co., Tokyo, Japan), and were later reduced to 5-s averages.

The HR was recorded continuously from the electrocardiogram. Arterial blood pressure was measured from the middle finger of the non-exercising hand by the Penaz method (Finapres, Ohmeda Co., Madison, WI, USA). The MAP was calculated as the sum of diastolic pressure plus one-third of the pulse pressure. At the end of each IH bout, subjects were asked to rate their RPE on a scale from 6 to 20 (Borg 1970). It is reported that the RPE was linear to exercise intensity during IH (Kondo *et al.* 2000) and the reported RPE value was used as an index of central command (Vissing *et al.* 1991, Vissing & Hjorts 1996).

Data processing and statistical analysis

Responses throughout the first IH bout were compared with responses during the second IH bout via a one-way repeated measures analysis of variance (ANOVA), followed by a Scheffe's post hoc test when *F*-values were significant. In addition, data were analysed from a 30-s period preceding the exercise bout (baseline), and from the final 30 s of the IH bout via a paired *t*-test.

Data are expressed as mean \pm SEM. The *P*-value level of significance was set at 0.05.

RESULTS

Oesophageal temperature, T_{es} (37.55 ± 0.18 °C) was significantly lower before the first bout of exercise relative to the period before the second bout of exercise (37.87 ± 0.13 °C; $P < 0.05$). The HR was also significantly lower before the first exercise bout relative to before the second bout of exercise. Baseline T_{sk} and

Table 1 Various body temperatures (oesophageal temperature: T_{es} , mean skin temperature: T_{sk} and local skin temperature from non-glabrous skin: T_{sl}), rating of perceived effort (RPE), sweating rate (SR) from non-glabrous skin and glabrous skin, and cutaneous vascular conductance (CVC) from non-glabrous skin at baseline, during both bouts of isometric handgrip exercise (IH)

Variables	First IH	Second IH
T_{es} (°C)		
Baseline	37.55 \pm 0.18	37.87 \pm 0.13*
Exercise	37.54 \pm 0.17	37.84 \pm 0.12*
T_{sk} (°C)		
Baseline	37.02 \pm 0.12	36.86 \pm 0.11
Exercise	37.04 \pm 0.12	36.87 \pm 0.12
T_{sl} (°C)		
Non-glabrous skin		
Baseline	35.72 \pm 0.14	35.71 \pm 0.14
Exercise	35.76 \pm 0.14	35.77 \pm 0.16
RPE		
Exercise	14.1 \pm 0.5	16.0 \pm 0.5*
HR (beats min ⁻¹)		
Baseline	79.7 \pm 3.1	86.4 \pm 2.7*
Exercise	90.5 \pm 3.5†	102.1 \pm 3.7*†
Delta	10.8 \pm 2.5	15.8 \pm 2.7*
MAP (mmHg)		
Baseline	82.1 \pm 1.7	80.9 \pm 1.8
Exercise	101.7 \pm 5.2†	105.6 \pm 4.6†
Delta	19.6 \pm 4.5	24.8 \pm 3.8
SR (mg cm ⁻² min ⁻¹)		
Non-glabrous skin		
Baseline	0.144 \pm 0.028	0.461 \pm 0.070*
Exercise	0.288 \pm 0.027†	0.534 \pm 0.065*†
Delta	0.144 \pm 0.027	0.073 \pm 0.012*
Glabrous skin (palm)		
Baseline	0.054 \pm 0.015	0.077 \pm 0.025
Exercise	0.106 \pm 0.025†	0.119 \pm 0.022†
Delta	0.052 \pm 0.034	0.042 \pm 0.018
CVC (mv mmHg ⁻¹)		
Non-glabrous skin		
Baseline	1.06 \pm 0.08	1.89 \pm 0.14*
Exercise	1.22 \pm 0.07	1.74 \pm 0.12*
Delta	0.16 \pm 0.10	-0.15 \pm 0.07*

Delta was analysed from a 30-s period preceding each exercise bout (baseline) relative to the final 30 s of the each IH. *Significantly different from the first IH bout for each variable (i.e. baseline, exercise and delta) ($P < 0.05$). †Indicates the responses during exercise were significantly different relative to the responses during baseline ($P < 0.05$). Data are mean \pm SEM.

MAP did not differ between exercise bouts (Table 1). There were no significant increases in T_{es} or T_{sk} during either IH bout (Fig. 2 and Table 1). Moreover, local skin temperature from non-glabrous skin was not different between the two exercise bouts (Table 1). The RPE and increases in HR during the second IH were significantly higher than during the first exercise bout (Table 1), while no significant differences in the change in MAP were observed between bouts.

The SR and CVC from non-glabrous skin were significantly higher prior to the second IH than prior to the first bout (Fig. 3). The change SR from non-glabrous skin was significantly higher during the first IH bout than during the second bout (Table 1), although SR increased significantly from baseline during both bouts of exercise. However, there was no significant

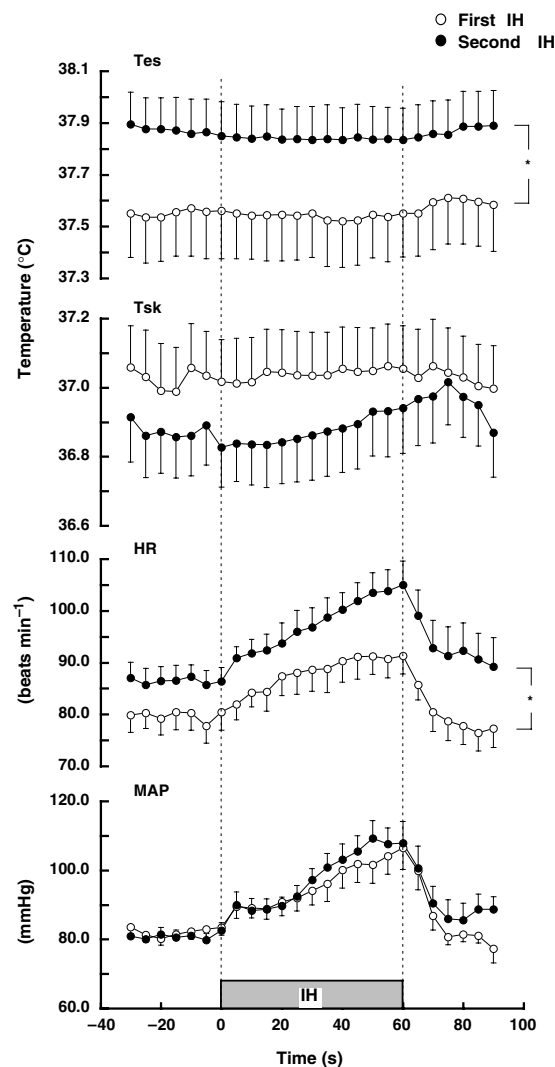


Figure 2 Changes in oesophageal temperature (T_{es}), mean skin temperature (T_{sk}), heart rate (HR) and mean arterial pressure (MAP) during the first (open circles) and second (closed circles) bouts of isometric handgrip exercise (IH). * $P < 0.05$ between bouts of IH. Data are mean \pm SEM.

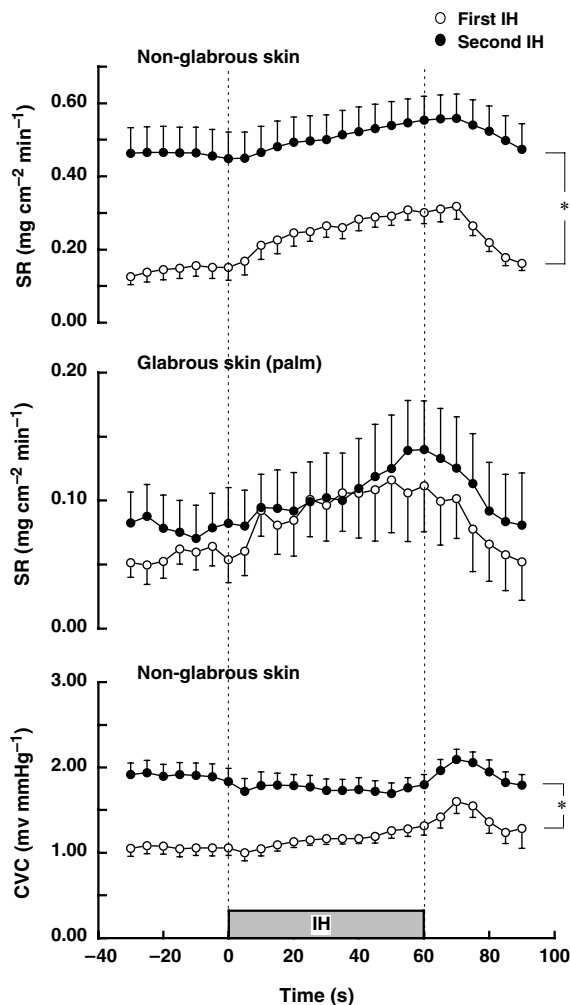


Figure 3 Changes in sweating rate (SR) from non-glabrous skin (derived from the average SR from the chest and forearm), SR from glabrous skin (palm), and cutaneous vascular conductance (CVC) from non-glabrous skin (derived from the average CVC from the chest and forearm) during the first (open circles) and second (closed circles) bouts of isometric handgrip exercise (IH). * $P < 0.05$. Data are mean \pm SEM.

difference in the increase in palm SR between the two exercise bouts (Table 1). Also, there is no significant difference in the time delay for increase in SR on non-glabrous skin between the exercises bouts (Fig. 3). The CVC from non-glabrous skin during the first IH bout tended to be greater relative to baseline, while CVC tended to decrease from baseline during the second bout of IH. These responses resulted in significant differences in the change in CVC between the two exercise bouts (Table 1).

The SR ($0.722 \pm 0.113 \text{ mg cm}^{-2} \text{ min}^{-1}$) from non-glabrous skin when room temperature was elevated to 38°C immediately following the second bout of exercise was significantly greater than the highest absolute SR attained during either bout of IH (Fig. 3). This procedure confirmed that the level of SR was sub-

maximum during the bouts of IH. Also, increases in SR were not different between two IH with 20-min interval in ambient temperature of 35°C and relative humidity of 50% in the second experiments.

DISCUSSION

This study investigated sweating responses from non-glabrous skin during IH exercise when internal temperature was moderate (i.e. just after onset of sweating during passive heating) and at higher internal temperatures. The primary finding of this study was that when internal temperature was high (37.84°C) the elevation in SR from non-glabrous skin during IH was diminished relative to the elevation of SR during IH when internal temperature was moderate (37.54°C). These data suggest that sweating responses from non-glabrous skin during non-thermal stimulation (i.e. IH) likely vary depending on the magnitude of thermal input (i.e. internal temperature), and that this response can be attenuated by sufficient hyperthermia. Also, this indicates that SR may be controlled by non-thermal factors until internal temperature increased and then sweating rate would be modulated mainly by thermal factors as internal temperature increases more.

In this study, SR from non-glabrous skin increased during both bouts of IH (Fig. 3). As T_{es} and T_{sk} remained constant throughout both IH bouts (Fig. 2), increases in SR during IH were primarily mediated via non-thermal factors. Key to the present findings is the observation that SR returned to pre-IH levels after IH was completed. If sweating responses during IH were solely the result of thermal factors associated with passive heating, SR would not have returned to the pre-exercise level immediately following the bout of exercise. Thus, it is clear that changes in SR from non-glabrous skin during both bouts of exercise were due to non-thermal factors associated with the exercise. Also, changes in SR during the first IH bout were greater relative to the second bout. This suggests that the level of thermal input (primarily T_{es}) may govern the degree that non-thermal inputs modulate the control of sweating from non-glabrous skin. These non-thermal inputs are likely a combination of central command (Van Beaumont & Bullard 1963, 1966, Vissing *et al.* 1991, Vissing & Hjorts 1996), as well as mechano- (Gisolfi & Robinson 1970, Kondo *et al.* 1997) and metabosensitive (Kaufman *et al.* 1983, Victor *et al.* 1988, Sinoway *et al.* 1989, Nishiyasu *et al.* 1994, Kondo *et al.* 1999) receptors in the exercising muscle.

Central command has been reported to be the primary mechanism that stimulates sympathetic outflow (including vasoconstrictor, vasodilator and sudomotor) to the skin during IH (Vissing *et al.* 1991, Vissing & Hjorts 1996). Moreover, SR from non-glabrous skin is

greater in response to active limb movement in which central command is engaged relative to passive movement in which central command is not engaged (Kondo *et al.* 1997). Taken together, this suggests that central command contributes to the modulation of sweating during dynamic exercise. In the present study, HR and RPE were significantly greater during the second bout of IH (Table 1). Given that these variables have been reported to be indicators of central command (Vissing *et al.* 1991, Vissing & Hjorts 1996), it is likely that the central command signal was greater during the second bout of exercise relative to the first. If central command was the primary modulator of sweating in humans during IH, the increase in SR from non-glabrous skin during the second IH bout should have been greater when compared with the first bout, which was not the case. Thus, the greater increase in SR associated with the first IH bout is not likely because of differences in central command between bouts. Moreover, as the same workload was performed during both bouts of IH, coupled with the observation that SR from non-glabrous skin increased to a greater extent during the first bout of exercise, it is unlikely that the differences in sweating responses between bouts were because of mechanisms associated with mechanoreceptor stimulation.

While baroreflexes are thought to influence the sweating response during dynamic exercise (Mack *et al.* 1995), we recently reported that elevations in blood pressure during post-isometric exercise ischaemia does not modify sweating responses (Shibasaki *et al.* 2001). Furthermore, in the present study MAP was similar between exercise trials (Table 1). Hence, differences in SR between two exercise bouts were unlikely to be the result of baroreflex modulation of SR.

Sweating responses from glabrous skin (i.e. palmar skin) are primarily influenced by mental and/or emotional stress (Kuno 1956, Ogawa 1975). In the present experiment, the increase in palm SR during IH was similar in both conditions (Table 1), thereby suggesting that the mental and/or emotional response was similar between trials. Therefore, although SR from non-glabrous skin is not believed to be modulated by mental and/or emotional stimuli, a lack of difference in palmar SR during IH between conditions suggests similar degrees of mental and/or emotional stimulation between exercise trials. Also, it may be difficult to separate clearly mental and/or emotional stress from central command even if RPE and palm SR did not show the similar difference between both the bouts in the present study.

As sweating responses are influenced by the local environment around sweat glands (Ogawa 1970), the smaller change in SR on the non-glabrous skin during the second bout of IH may potentially be because of lower local skin temperatures. However, in this study local skin temperature was similar between the two

exercise bouts (Table 1 and Fig. 2), thereby removing this hypothesis. Moreover, differences in SR from non-glabrous skin between the first and second IH trials are unlikely to be the result of SR reaching maximal levels, because SR from non-glabrous skin when room temperature was elevated to 38 °C increased to a level significantly greater when compared with SR at any point during either IH bout. In addition, it is possible that the order of exercise bout in the first experiment would not influence the differences in sweating response between the first bout of IH and second bout of IH because of results in the second experiment.

It is not clear why SR from non-glabrous skin during the first bout of IH was greater than SR during the second bout of IH, even when central command during the second IH was likely greater and other non-thermal factors were not markedly different between exercise bouts. The present data suggest that the effects of non-thermal factors, such as central command and muscle metabo/mechanoreceptor stimulation, in the control of sweating are influenced by the level of internal temperature and there is a possibility that the smaller increment in non-thermal sweating during the second bout of IH might be contributed to decreased sweating responsiveness to a given non-thermal factors at the higher level of internal temperature or sweat activity. However, we should not define the mechanisms that would have induced the greater SR from non-glabrous skin during the first bout of IH in the present study, although sweating responses are also modulated by central mechanisms in thermoregulatory centres and peripheral mechanisms in the sweat glands.

Cutaneous vascular responses from non-glabrous skin between IH bouts were also significantly different. The IH tended to cause reductions in CVC from baseline during the second bout of IH, while slight elevations in CVC from base line were observed during the first bout of exercise (see Table 1). Sympathetic vasoconstrictor and sympathetic active vasodilator systems regulate skin blood flow (Johnson & Proppe 1996). The active vasodilator system predominates in hyperthermia, especially when internal temperature is elevated (Kellogg *et al.* 1991). Prior studies suggest that CVC decreases significantly when IH is performed in more pronounced hyperthermic conditions, and this reduction in CVC was primarily caused by withdrawal of active vasodilator activity (Crandall *et al.* 1995, 1998). In the present study, as T_{es} was significantly higher prior to the second IH bout relative to the first bout (Table 1), it was likely that the active vasodilator system was activated to a greater extent before and inhibited to a greater extent during the second bout of IH relative to the first bout of IH. Thus, differing responses in CVC between IH bouts may be the result of the degree of activation of the active cutaneous vasodilator system.

In conclusion, the present study demonstrates that the level of internal temperature modulates the magnitude of IH-induced sweating from non-glabrous skin. This finding confirms the hypothesis that the effectiveness of non-thermal factors in the control of sweating from non-glabrous skin may be dependent on background thermal load, such that when thermal load is high the effectiveness of non-thermal factors in modulating sweat rate is diminished.

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