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Non-thermal modulation of sudomotor function during static exercise and the impact of intensity and muscle-mass recruitment

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ABSTRACT

Aim: Static muscle activation elicits intensity-dependent, non-thermal sweating that is presumably controlled by feedforward (central command) mechanisms. However, it is currently unknown how the size of the recruited muscle mass interacts with that mechanism. To investigate the possible muscle-size dependency of that non-thermal sweating, the recruitment of two muscle groups of significantly different size was investigated in individuals within whom steady-state thermal sweating had been established and clamped. **Methods**: Fourteen passively heated subjects (climate chamber and water-perfusion garment) performed 60-s, static handgrip and knee-extension activations at 30% and 50% of maximal voluntary force, plus a handgrip at 40% intensity (143.4 N) and a third knee extension at the same absolute force. Local sweating from four body segments (averaged to represent whole-body sudomotor activity), three deep-body and eight skin temperatures, heart rates and perceptions of physical effort were measured continuously, and analyzed over the final 30 s of exercise. **Results**: In the presence of thermal clamping and low-level, steady-state sweating, static muscle activation resulted in exercise-intensity dependent changes in the whole-body sudomotor response during these handgrip and knee-extension actions ($P < 0.05$). However, there was no evidence of a dependency on the size of the recruited muscle mass ($P > 0.05$), yet both dependencies were apparent for heart rate, and partially evident for the sensations of physical effort. **Conclusion**: These observations represent the first evidence that exercise-related sudomotor feedforward is not influenced by the size of the activated muscle mass, but is instead primarily dictated by the intensity of the exercise itself.

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Introduction

Human eccrine sweating is controlled by thermal and non-thermal mechanisms.[1,2,3] Although our understanding of the latter is much less clear, exercise is known to be a powerful, non-thermal mediator of sweating. Accordingly, following thermally induced sudomotor priming, sweating increases immediately upon initiating either dynamic[4] or static (isometric) muscle activations,[5] while skin blood flow is simultaneously reduced.[6,7] Those sudomotor responses are cholinergically driven,[8] with most super-imposed, non-thermal influences resulting in ubiquitous sweat secretions across both the glabrous (non-hairy) and non-glabrous skin surfaces.[9,10] At present, the exercise-induced sweating responses are also known to be related to the intensity of the muscle activation,[7,11] which also appears to determine the number of activated sweat glands.[7] In this experiment, however, attention was directed to the possibility that the size of the recruited muscle mass during static exercise might also modulate the strength of the sweating response.

During extended dynamic exercise, temperatures within the active-muscle and deep-body tissues are significantly elevated, with feedback from thermosensitive elements driving sweat secretion.[12-14] However, experimentally induced, static muscle activations are typically of a short duration (<2 min), so changes in the

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deep-body tissues are not generally anticipated. Indeed, stable deep-body and skin temperatures are generally reported during similarly transient, static muscle recruitment,[11,15] although most deep-body measurements have a phase delay of at least the same duration as the muscle-activation period,[16] and invariably exclude observations from the activated muscles.[14] Nevertheless, this apparent thermal stability supports the hypothesis that the resulting sudomotor activity may be dominated by non-thermal mechanisms. This possibility is particularly pertinent during low-level thermal loading,[17] and is now well recognized.[1,2]

Several putative mechanisms have been implicated for the non-thermal modulation of sweating.[1,2,18] These include both feedforward (central command[6,17]) and feedback mechanisms.[15,19] In the current experiment, the role of feedforward was evaluated, for which a dependency on muscle-activation intensity has already been established during static exercise.[7,11] A similar dependency exists for the cardiovascular chronotropic response.[20,21] Moreover, both the chronotropic and blood-pressure responses are heightened as the size of the recruited muscle mass is elevated.[21,22] Yet to our knowledge, the latter relationship has not been thoroughly explored with regard to sudomotor activity, and this represents a significant gap in our understanding of the control of human eccrine sweating. It was therefore hypothesized that sudomotor activity, when compared during the independent and static activation of muscles varying in size (volume), would be elevated when a larger muscle mass was recruited with the same relative exercise intensity, but not when those muscles were required to generate the same absolute force.

Methods

Subjects

Fourteen healthy and physically active adults (ten male and four female; age 25.6 y [standard deviation (SD) 5.3], height 176.2 cm [SD 6.8], body mass 72.3 kg [SD 10.5]) participated in a single experimental trial involving static muscle activations of the forearm flexors and knee extensors. Females were tested in the first 10 d of the follicular phase to minimize thermoregulatory responses associated with the menstrual cycle.[23] None of the participants was heat adapted, taking medication, or had a history of cardiovascular or thermal illness. All subjects provided written, informed consent to experimental procedures approved by the Human Research Ethics Committee (University of Wollongong) in accordance with the regulations of the National Health and Medical Research Council (Australia).

Experimental procedures

Procedural overview

Participants completed a single familiarization, training and pre-experimental testing session, followed by one experimental trial that occurred at least 48 h later. That trial was performed in a whole-body heated and thermally clamped state (water-perfusion garment and climate chamber), such that a low level of steady-state sweating was established within every individual. Following this sudomotor-priming and thermal-clamping period (60 min), subjects performed three bouts of static muscle activation of varying intensity within each of two exercise modes: handgrip at 30%, 40% and 50% of each individual's mode-specific maximal voluntary activation; and knee extension at both 30% and 50% of maximal activation, as well as at the same absolute force developed during the 40% handgrip activation (143 N). All muscle activations were performed on the right side, and all participants were right-hand dominant.

Pre-experimental familiarization, testing and standardization

To establish the maximal voluntary handgrip and knee-extension forces, each individual participated in familiarization and training before the pre-experimental determination of those right-side muscle forces. That value was taken as the average of three maximal efforts, separated by at least a 2-min rest period. Each activation was performed in an upright, seated posture on a chair mounted within a steel support frame, and with the feet above the floor. The height of the chair was adjusted to meet the following requirements: knees flexed at 90°; forearms fully supported at heart level with the shoulders in a neutral position and the elbows flexed at 90°. The handgrip task was performed using a custom-made, adjustable dynamometer connected to a load cell (SBA-200L, CAS corporation, Seoul, Korea). The size of the handgrip gap was adjusted for each person so that pressure was taken on the heel of the hand and the middle phalanges of the fingers. For the knee extension task, a 10-cm wide strap was secured around the ankle (malleolar level),

with the leg vertical, and connected at 90° to a second load cell that was secured to the support frame (LC1205-K100, A&D Co. Ltd., Seoul, Korea). Extraneous body movements were prevented using straps secured over the mid-thigh (to prevent hip flexion) and around the hips (iliac crest: to prevent movement on the seat).

Subjects acted as their own controls while the within-trial, muscle-activation order was balanced among individuals. To standardize each experimental trial, and to minimize the impact of changes in breathing pattern on cutaneous sympathetic activity,[24] breathing frequency was controlled at 12 breaths.min^{-1}, with each inspiration coinciding with an auditory signal provided via headphones. In addition, the laboratory lights were dimmed. For the 24 h prior to the experimental trials, subjects were instructed to refrain from strenuous exercise, and to avoid alcohol and caffeine consumption during the preceding 12 h. During each trial, neither food nor fluids were consumed.

Experimental routine

On presentation, subjects voided, inserted a rectal temperature sensor and entered an air-conditioned laboratory (air temperature 21.4°C) for preliminary instrumentation and the collection of pre-experimental baseline data. Wearing only a swimming costume, they then entered a climate-regulated chamber (air temperature 36.8°C, water vapor pressure 3.60 kPa [relative humidity 58%]). Each participant sat on the test chair (described above) and a water-perfusion garment was fitted (Paul Webb Associates, Yellow Springs, USA), remaining *in situ* throughout testing. Subjects were secured in the same upright position that was used during the pre-experimental testing. The perfusion garment covered most skin surfaces, except for the neck, head, left dorsal forearm, hands and feet, and was adjusted and secured to ensure good skin contact. This garment did not include a cloth layer, and so it provided maximal skin exposure to the air. It was comprised only of tubing (140 m: Tygon®: internal diameter 1.58 mm, outside diameter 3.0 mm) arranged into 1-m, parallel lengths to form separate jacket and trousers components that were independently perfused through separate manifolds (water temperature 36.4°C [SD 0.1], flow 2.2 L.min^{-1}), ensuring uniform flow across both body regions.[25] Water was provided from a temperature-regulated water bath (38-liter, Grant Instruments [Cambridge] Ltd., UK). Pilot testing was used to establish the air and water-bath temperatures required for sudomotor priming in both the male and female participants. This passive heating continued for ~60 min, and until low-level, steady-state sweating was established. Thereafter, the ambient environment and perfusion garment were used to clamp deep-body, skin and mean body temperatures.[26,27] This was an important design feature, since it ensured sweat-gland priming without imposing a high thermal loading that would minimize the likely impact of the anticipated non-thermal control mechanisms.[17]

Following establishment and verification of the thermal clamp, testing commenced. This took the form of 60-s, static handgrip and knee-extension muscle activations. Based on the average maximal data obtained during the pre-experimental session, each participant performed single activations at 30%, 40% and 50% his or her own maximal handgrip force, as well as knee extensions at both 30% and 50% of that mode-specific maximal force. In addition, knee extension was performed at an absolute force that corresponded to that which was developed during the 40% handgrip activation (143.4 N [SD 28.7]). While this represented the same absolute force, it was only 25.3% of the maximal voluntary knee-extension force. Each voluntary effort was achieved using a visual feedback display mounted at eye level. Subjects were required to generate sufficient force so that either an analog (handgrip: QM-1020 DCV multimeter, Digitech, Shanghai, China) or digitally displayed target (knee extension: AD-4329 A&D Co. Ltd., Seoul, Korea) was rapidly achieved, and then sustained. Those targets were set to match the six, participant-specific experimental forces, although subjects were not informed of those intensities or their presentation sequence. Successive efforts occurred at 10-min intervals, ensuring restoration of both the thermal and sudomotor steady states, although no evidence was found that the former had changed during exercise. Across subjects, those target forces were administered in a counter-balanced order. In this way, there was no prior knowledge of the required force generation. Moreover, breathing frequency was controlled (12 breaths.min^{-1}), the lights were dimmed and verbal encouragement was not provided. At the completion of each activation, subjects provided a subjective rating of the effort required (perceived exertion) to achieve and sustain the target force.

Physiological measurements

Sudomotor responses

Local sweat rates were measured continuously from the head, upper torso, and the upper and lower limbs using ventilated capsules (3.16 cm^2) attached to the skin: central forehead, left pectoral surface, mid-dorsal forearm (left side) and the mid-anterior thigh (left). To provide a generalized evaluation (whole-body surrogate) of the changes in sudomotor function, those simultaneously recorded data were averaged during each muscle activation. The peak sweat secretion, averaged across the four sites, during the final 30 s of each static exercise period was then used as the primary index of central sudomotor drive.

A surgical adhesive was used to attach the sweat capsules to the skin, and to prevent leakage and pressure-induced artifacts (Collodion USP., Mavidon Medical Products, FL, USA). Air flowing through the four capsules was collected over a saturated lithium chloride solution to control its relative humidity at 12%. That flow was independently regulated to each capsule (300-500 $mL.min^{-1}$) to maximize the evaporation of sweat secreted beneath all capsules. Post-capsular airflows were directed past capacitance hygrometers (MiniCap 2, Panametrics Pty. Ltd., Gymea, Australia) within an integrated sweat-monitoring system (Clinical Engineering Solutions, Sydney, Australia), with air temperature and relative humidities simultaneously sampled from four channels at 1-s intervals (DAS1602, Keithley Instruments, Inc., Cleveland, OH, USA). Those data were used to compute local sweat rates.[28] A three-point hygrometer calibration preceded experimentation, and involved two saturated salt-solution standards and two ambient temperatures.

Body-tissue temperatures

Three deep-body temperature indices (esophageal, auditory canal and rectal) were simultaneously measured, from which was derived an unweighted average. That approach provides a superior estimation of whole-body heat storage and deep-body temperature,[16] which is presumed to drive central thermoafferent flow. Furthermore, we have demonstrated a phase delay of <50 s between a dynamic, exercise-provoked temperature change in skeletal muscle (*vastus lateralis*) and the corresponding change in esophageal temperature,[14] thereby allowing for peripheral heat production to be detected centrally. The esophageal thermistor (Edale instruments Ltd., Cambridge, UK) was inserted trans-nasally, and positioned behind the heart (after[29]). Rectal thermistors were self-inserted (YSI type 401, Yellow Springs Instruments Co. Ltd., Yellow Springs, OH, USA) and positioned 12 cm beyond the anal sphincter.[30] The auditory canal sensors were also self-inserted. Those devices took the form of an ear-molded plug with a protruding thermistor (Edale instruments Ltd., Cambridge, UK), which was positioned within the external auditory meatus and shielded from the thermal environment (cotton-wool pad). This procedure results in a valid, reliable and very responsive measurements with minimal artifactual influences.[14,31,32]

Skin temperatures were simultaneously measured from eight sites using thermistors fastened to the skin with a single layer of waterproof tape (YSI type-EU, Yellow Springs Instruments, Yellow Springs, OH, USA). To approximate mean skin temperature, measurements were taken from the forehead, right chest (pectoral), right upper back (scapula), left lateral upper arm, right mid-dorsal forearm, right dorsal hand, right mid-anterior thigh and the right lateral leg. Mean skin temperature was approximated using an eight-site equation.[33] Mean body temperature was calculated as the weighted sum of the average deep-body and mean skin temperatures using a deep-body mixing coefficient of 80% (after[34]). All tissue temperatures were sampled at 5-s intervals (1206 Series Squirrel, Grant Instruments Ltd, Shepreth, Cambridgeshire, UK), with thermistors calibrated across physiologically relevant temperatures in a stirred water bath, and against a certified reference thermometer (Dobros total immersion, Dobbie Instruments, Sydney, Australia).

Cardiovascular function and perception of effort

To evaluate feedforward control over cardiac function, heart rates were measured. Those data were recorded continuously and sampled at 5-s intervals (Polar Electro Sports Tester, Kempele, Finland). As an established surrogate of the strength of the feedforward signal,[35] subjective effort sensations (perceived exertion) were assessed using the 15-point Borg scale,[36] as direct measurement of sympathetic traffic was not available. After each muscle

activation, for which subjects had no prior knowledge concerning either the desired force or the required activation intensity, participants were asked to assess the physical exertion required to achieve each target (scale 6-20).

Statistical analyses

Data were analyzed over three periods: the 60 s prior to commencing each activation (baseline), during the final 30 s of static exercise and during the first 60 s of recovery. One-way, repeated-measures analysis of variance was used for comparing the physiological responses accompanying the two modes of static exercise and the activation intensities. The Greenhouse-Geisser correction was used to adjust degrees of freedom for violations of sphericity (Mauchly's test). Significant effects were tested using the Least Significant Difference *post hoc* procedure. Changes in sudomotor function from baseline to peak secretion rates were compared across the two exercise modes using paired *t*-tests. The level of significance was set at 5% for all analyses, and data are presented as means with standard errors of the means (±), except when describing independent variables or highlighting data distributions (standard deviations [SD]).

Results

Precision of the thermal clamp

All physiological variables were stable both before and following every muscle activation (Table 1), regardless of the exercise intensity. Of particular relevance to this investigation were the three deep-body temperatures and the mean skin temperature, for their stability confirmed the integrity of the thermal clamp across all trials (Table 1). Indeed, the resulting mean body temperatures, when averaged across the six static-exercise periods, were 37.1°C (SD 0.2) at baseline, and during both the exercise and recovery periods. Most variables, and certainly all of the body-tissue temperatures, also remained stable during the first 60 s of the recovery period, although the sudomotor responses, particularly those accompanying the two 50% activation intensities, took longer to recover.

Table 1. Physiological data during thermal clamping (air temperature: 36.8°C; perfusion garment water temperature 36.4°C) prior to (baseline), during (exercise) and following (recovery) static handgrip and leg-extension exercise performed at three activation intensities ($N = 14$). Data are means obtained across both exercise modes and all activation intensities, with standard deviations provided within parenthesis. None of the between- or within-mode differences were significantly different ($P > 0.05$). Significant differences between the baseline and static exercise periods are indicated: $^{\dagger}(P < 0.05)$.

Dependent variable	Baseline	Exercise	Recovery
Esophageal temperature (°C)	37.1 (0.3)	37.1 (0.3)	37.1 (0.3)
Auditory canal temperature (°C)	37.3 (0.3)	37.3 (0.3)	37.3 (0.3)
Rectal temperature (°C)	37.6 (0.2)	37.6 (0.2)	37.6 (0.2)
Mean skin temperature (°C)	36.0 (0.3)	36.0 (0.3)	36.1 (0.3)
Forehead sweat rate ($mg.cm^{-2}.min^{-1}$)	0.64 (0.26)	0.88 (0.25)†	0.69 (0.29)
Chest sweat rate ($mg.cm^{-2}.min^{-1}$)	0.32 (0.18)	0.48 (0.20)†	0.34 (0.19)
Forearm sweat rate ($mg.cm^{-2}.min^{-1}$)	0.22 (0.14)	0.38 (0.16)†	0.25 (0.15)
Thigh sweat rate ($mg.cm^{-2}.min^{-1}$)	0.30 (0.16)	0.39 (0.14)†	0.30 (0.15)
Heart rate ($beats.min^{-1}$)	80 (15)	95 (18)†	81 (16)

Baseline sudomotor activity

To increase the possibility of detecting non-thermal sudomotor influences, four objectives needed to be satisfied. Firstly, it was necessary to adequately prime the sweat glands. Secondly, localized sudomotor responses from several different body segments needed to be simultaneously evaluated to provide a highly sensitive, whole-body surrogate of sudomotor activity. Thirdly, the resulting sweat rates must be stable prior to commencing each muscle activation. Finally, the size of the thermal load must remain small. Each of those criteria was satisfied (Table 1), and relative to the mean body temperature obtained before entering the climate chamber, passive heating and thermal clamping induced an overall mean body temperature change of 1.4°C (SD 0.2; $P<0.05$) across participants. In that state, the intra-individual, regional variations in the baseline sweat rates conformed with data described in the literature.[3] When collectively evaluated with data derived during thermal clamping, those outcomes led to the interpretation that differences in sudomotor activity within the six exercise periods were unlikely to be of a thermal, but of a non-thermal, origin.

Exercise-induced sudomotor and cardiac activity

During every static muscle activation, regardless of its intensity, sweat secretion rates were significantly increased (Table 1; $P < 0.05$). These data were averaged across the four body regions to represent whole-body sweating during each phase of static exercise, and are provided in Figure 1. With the exception of the 50% knee-extension condition (Fig. 1F), the pre-activation sudomotor baselines were restored within the first

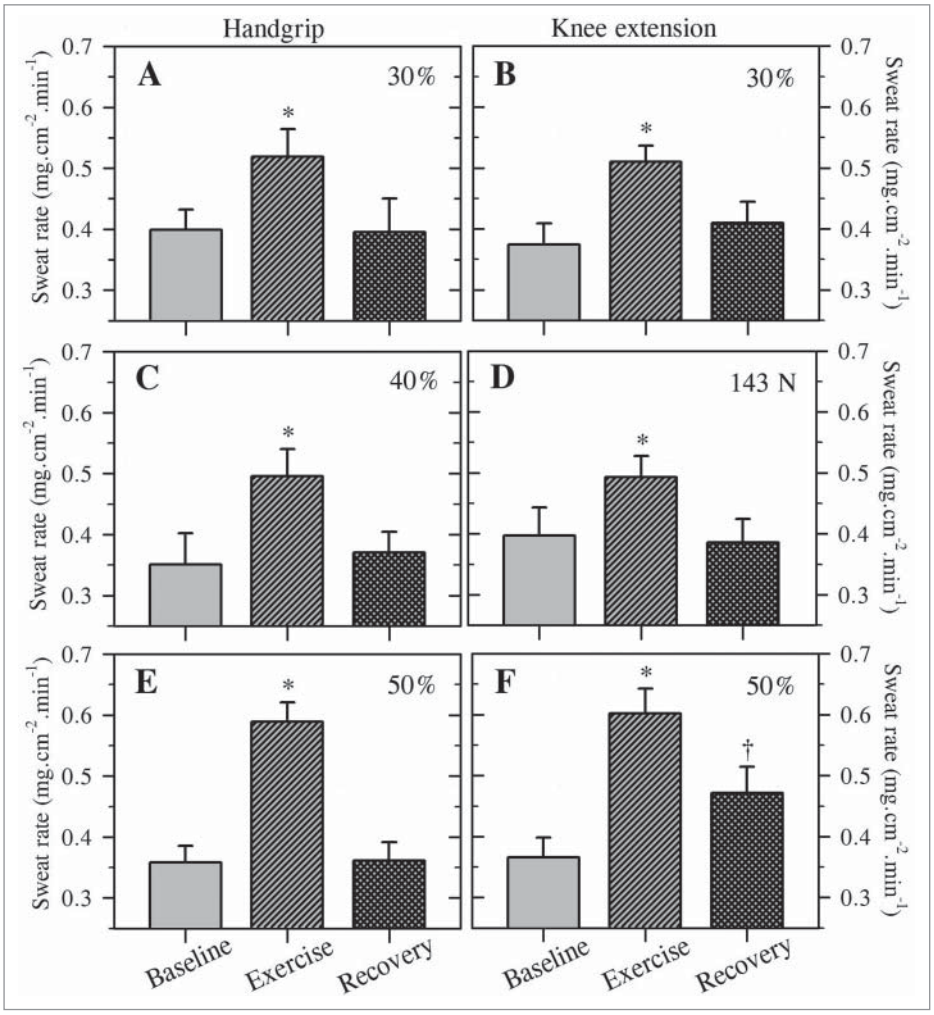

Figure 1. Average sweat secretion rates before (baseline), during (exercise) and following (recovery) two static muscle activations (60 s: handgrip and knee extension). These sudomotor responses are averages derived from four simultaneously collected local sweat rates (forehead, chest, forearm and thigh) and collected from thermally clamped individuals ($N = 14$; air temperature: 36.8°C; perfusion garment water temperature 36.4°C). Data were collected at three exercise intensities relative to each participant's maximal voluntary activation, and are presented as means with standard errors of the means. Two activations within each muscle group were at the same relative intensities (30%: A and B; 50%: E and F), while the third activation was at the same absolute force for both muscle groups (143 N: C and D). Exercise data represent sweating peaks collected over the final 30-s phase of static exercise. Significant differences are indicated by the symbols ($P < 0.05$): * for the baseline and exercise comparison; and † for baseline relative to recovery.

60 s of each 10-min recovery period ($P > 0.05$), ensuring that the impact of every subsequent muscle activation could be evaluated against equivalent, steady-state baselines. In all cases, static exercise evoked a significant elevation in sweating from each of the four body regions investigated (Table 1), along with an elevation in chronotropic drive (Fig. 2).

The chronotropic effect revealed an intensity dependency for both the handgrip and knee-extension modes, as well as a muscle-mass dependency (Fig. 2), for both muscle activity modes, as respectively demonstrated by Seals[20] and Seals et al.[22] Similarly, the average sweat rate and perceptions of the required effort were tightly coupled with the muscle-activation intensity (Table 2), as reported by Kondo et al.[11] and Amano et al. [7] for sudomotor function.

When analyzed with respect to the size of the recruited muscle mass, there was no evidence for an influence of muscle size on sudomotor function (Fig. 1 and Table 2), there was also no evidence of a gender effect on the relative changes in either muscle and sudomotor function, so data from both groups were treated collectively. That is, regardless of whether the forearm flexors and knee extensors

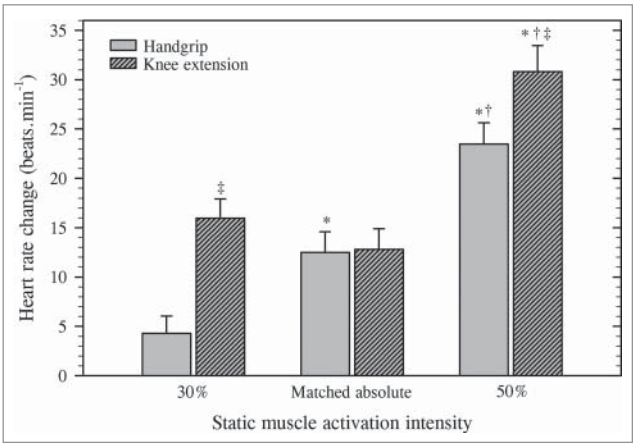

Figure 2. Changes in peak heart rate during two static muscle-activation modes (final 30 s of 60-s bouts: handgrip and knee extension) performed at two relative exercise intensities that were proportional to each participant's maximal voluntary activation, and at one matched absolute intensity (143 N). Subjects were thermally clamped ($N = 14$; air temperature: 36.8°C; perfusion garment water temperature 36.4°C), with data reported as means with standard errors of the means. Significant differences are indicated by the symbols ($P < 0.05$): * for the within-mode comparisons with the lightest intensity (30% of maximal activation); † for the within-mode comparisons between the same absolute (matched) force generation and the highest intensity (50%); and ‡ for the between-mode comparisons within each exercise intensity.

were activated at the same relative intensity or with the same absolute force, between-mode sweat rates did not differ significantly within any exercise intensity ($P > 0.05$). That outcome was at odds with the chronotropic response, which also revealed a muscle-size dependency (Fig. 2). Effort sense, on the other hand, only provided evidence of a size dependency when both exercise modes were performed with the same absolute force (Table 2). In this case, the handgrip action was perceived to require significantly more effort to achieve and hold for 60 s ($P < 0.05$).

Discussion

The experimental design criteria necessary to detect and evaluate feedforward (non-thermal) influences on the generalized sweat secretion were fulfilled. Indeed, thermal clamping was sustained throughout every trial, with the aim of elevating and sustaining a constant thermoafferent flow from the deep-body and cutaneous thermoreceptors. One may therefore exclude those feedback signals from the modulation of sudomotor function during concurrent, short-duration muscle activations. Under these well-controlled conditions, the interactions of both the static muscle-activation intensity and muscle size with sweating could be explored. Our observations have reinforced the previously established tenets of feedforward control, while revealing one novel outcome. From changes in heart rate, the established chronotropic dependency on both exercise intensity and muscle size was again evident.[20-22] Also observable was an activation-intensity impact on the generalized sudomotor response,[7,12] with others showing that this is determined by the number of activated sweat glands when sudomotor activity is modest.[7,37] The novel observation from this experiment was that the previously clamped, whole-body sweat secretion rate was elevated in an exercise intensity-dependent manner, but unlike the heart rate response, it appeared that this feedforward mechanism was unresponsive to variations in the size of the recruited muscle mass. This insensitivity existed even though the activated muscles had a three-fold variation in size, leading to a rejection of the size-dependency hypothesis during static (isometric) muscle activity under these experimental conditions.

We cannot exclude the possibility that thermosensitive tissues elsewhere within the body drove these sudomotor responses. Since heat is liberated during muscle activations, then feedback from the active skeletal muscles may have contributed to these sudomotor

Table 2. Sudomotor change and subjective ratings of the required muscle-activation effort (perceived exertion) during static handgrip and leg-extension exercise (final 30 s of 60-s bouts) performed at four activation intensities (25%, 30%, 40%, and 50%) within each exercise mode ($N = 14$). Subjects were heated and thermally clamped throughout experimentation (air temperature: 36.8°C; perfusion garment water temperature 36.4°C). Data are means with standard errors of the means in parenthesis. Significant differences are indicated by the symbols ($P < 0.05$): * for within-mode comparisons with the lightest exercise intensity; and † for within-mode comparisons with the middle intensity.

Dependent variable	Mode	25%	30%	40%	50%
Change in sweat rate ($mg.cm^{-2}.min^{-1}$)	Handgrip		0.12 (0.03)	0.15 (0.03)	0.23 (0.03)*†
	Knee extension	0.10 (0.02)	0.14 (0.02)*		0.24 (0.03)*†
Perceived exertion (scale 6-20)	Handgrip		12.8 (0.4)	15.1 (0.5)*	17.6 (0.4)*†
	Knee extension	12.0 (0.5)	13.5 (0 .4)*		17.4 (0.4)*†

responses,[12-14] with both the group III and IV muscle afferents responding to thermal stimuli.[38,39] Nevertheless, we suspect that influence, if present, would have been minimal, since Vissing et al. [6] reported that sympathetic flow to the skin preceded muscle tension development. On that basis, and in combination with the brevity of the static exercise, one may also eliminate contributions from metaboreceptor feedback.

Instead, it was speculated that both the heart and sweat rates were principally modified through variations in feedforward (sympathetic) flow accompanying neural drive emanating from the motor cortex. It was implicitly assumed that static exercise within muscles of a different size would, when activated at the same relative intensity (*e.g.*, 30% and 50% of maximal voluntary activation), recruit different volumes of skeletal muscle. It was further assumed that, when required to generate an equivalent force (143 N), both muscles would then recruit a muscle volume proportional to the relative intensity of that static activation (25% [knee extensors] versus 40% [forearm flexors]), although those assumptions were not evaluated. When one subsequently compared the resulting chronotropic and sudorific effects, it was anticipated those responses might reveal both activation-intensity and recruited muscle-mass dependencies.

The fact that this did occur at the heart (Fig. 2), but not at the sweat glands (Fig. 1), is possibly indicative of the independent nature of those two feedforward pathways, and the more complex neural control of the heart. Indeed, the early rise in cardiac frequency is biphasic in nature, and brought about by a withdrawal of the parasympathetic brake, with further elevations driven by elevated sympathetic tone.[40] During static handgrip exercise, parasympathetic withdrawal dominates during the first 10 s.[41] Sweat glands, on the other hand, are exclusively activated by sympathetic neurons.[42] One may therefore assume, since the present data were collected only during the final 30 s of static exercise, that these concurrent observations were of an almost entirely sympathetic origin, and that those efferent signals carried different neural information. In the absence of direct measurements of either cardiac or sudomotor sympathetic activity, that possibility remains speculative.

It may follow that these two forms of sympathetic output are encoded differently, as previously demonstrated for efferent signals flowing to the skin and skeletal muscle during static handgrip exercise.[6] Nevertheless, it appears that similar cutaneous sympathetic activity accompanies static forearm-flexor and knee-extensor activation at the same relative intensities.[21] Thus, in the presence of an apparent cutaneous sympathetic homogeneity, there may exist a sympathetic heterogeneity, when signals flowing to different tissues are compared. On a first-principles basis, this divergent possibility is appealing, since the required convective delivery of oxygen is both exercise-intensity and muscle-volume dependent, while heat production, and therefore the heat-loss requirement, may be more closely linked to the exercise intensity, and less tightly associated with the volume of the activated muscle.

Finally, it is important to recognize that, while the current observations appear to be the first confirmation of the independent nature of the sudomotor response with regard to the size of the activated muscle mass, there is clear evidence that cutaneous sympathetic activity displays such autonomy. Indeed, Ray and Wilson,[21] using the same types of static exercise, but within normothermic individuals exercising at one relative intensity only, found that the relative exercise intensity, but not the recruited muscle mass, determined the intensity of cutaneous sympathetic activity. Nevertheless, that group did not observe a consistent sudorific response, and interpreted that to signify an absence of sympathetic flow to the sweat glands. The current data, when combined with the well-established evidence that non-thermal sweating is a ubiquitous phenomenon, but sometimes requires sweat-gland priming to be revealed,[9,10,43] imply that conclusion was a misinterpretation that was a function of the experimental design.

Conclusion

This experiment appears to have provided the first clear evidence that generalized (whole-body) sweating during short-duration, static exercise is not influenced by the size of the muscle mass recruited. Instead, it appears to be primarily dictated by the intensity of the exercise. This outcome is consistent with direct observations of sympathetic nerve activity during these same modes of exercise, and is therefore assumed to have been mediated by a non-thermal, feedforward mechanism that may be of a different form to that innervating the heart and skeletal muscles.

Disclosure of potential conflicts of interest

No potential conflicts of interest were disclosed.

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