

ORIGINAL ARTICLE

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Intense exercise increases the post-exercise threshold for sweating

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Abstract We demonstrated previously that esophageal temperature (T_{es}) remains elevated by $\approx 0.5^\circ\text{C}$ for at least 65 min after intense exercise. Following exercise, average skin temperature (T_{avg}) and skin blood flow returned rapidly to pre-exercise values even though T_{es} remained elevated, indicating that the T_{es} threshold for vasodilation is elevated during this period. The present study evaluates the hypothesis that the threshold for sweating is also increased following intense exercise. Four males and three females were immersed in water (water temperature, $T_w = 42^\circ\text{C}$) until onset of sweating (Immersion 1), followed by recovery in air (air temperature, $T_a = 24^\circ\text{C}$). At a T_a of 24°C , 15 min of cycle ergometry (70% $\dot{V}O_{2max}$) (Exercise) was then followed by 30 min of recovery. Subjects were then immersed again ($T_w = 42^\circ\text{C}$) until onset of sweating (Immersion 2). Baseline T_{es} and T_{skavg} were 37.0 (0.1) $^\circ\text{C}$ and 32.3 (0.3) $^\circ\text{C}$, respectively. Because the T_{skavg} at the onset of sweating was different during Exercise [30.9 (0.3) $^\circ\text{C}$] than during Immersion 1 and Immersion 2 [36.8 (0.2) $^\circ\text{C}$ and 36.4 (0.2) $^\circ\text{C}$, respectively] a corrected core temperature, T_{es} (calculated), was calculated at a single designated skin temperature, $T_{sk(\text{designated})}$, as follows: $T_{es(\text{calculated})} = T_{es} + [\beta/(1-\beta)][T_{skavg} - T_{sk(\text{designated})}]$. The $T_{sk(\text{designated})}$ was set at 36.5°C (mean of Immersion 1 and Immersion 2 conditions) and β represents the fractional contribution of T_{skavg} to the sweating response (β for sweating = 0.1). While $T_{es(\text{calculated})}$ at the onset of sweating was significantly lower during exercise [36.7 (0.2) $^\circ\text{C}$] than during Immersion 1 [37.1 (0.1) $^\circ\text{C}$], the threshold of sweating during Immersion 2 [37.3

(0.1) $^\circ\text{C}$] was greater than during both Exercise and Immersion 1 ($P < 0.05$). We conclude that intense exercise decreases the sweating threshold during exercise itself, but elicits a subsequent short-term increase in the resting sweating threshold.

Key words Warm-water immersion · Esophageal temperature · Cutaneous vasodilation · Thermoregulation · Heat loss

Introduction

We demonstrated previously that esophageal temperature (T_{es}) remains elevated by $\approx 0.5^\circ\text{C}$ for at least 65 min after intense exercise (Thoden et al. 1994). In addition, the plateau value was equal to the threshold T_{es} at which active skin vasodilation was initiated during exercise. The post-exercise elevation was not of a metabolic origin since oxygen consumption ($\dot{V}O_2$) returned to baseline values within 5–10 min of exercise termination. Skin blood flow and mean skin temperatures (T_{skavg}), at all sites except over the exercised muscle (i.e., thigh and calf), also returned to control values within 10–15 min after exercise, despite the sustained increase in T_{es} . The reduction of T_{skavg} and skin blood flow throughout the prolonged elevated plateau in T_{es} is consistent with an increase in the vasodilation threshold during recovery from exercise. The post-exercise elevations in T_{es} and vasodilation threshold could be a function of either: (1) residual exercise-related factor(s), or (2) a significant elevation in body heat content (Houdas et al. 1973; Webb 1995).

In a previous effort to address the latter mechanism we immersed subjects in warm water (42°C) until T_{es} increased to levels similar to those induced by 15 min of heavy exercise (Kenny et al. 1996b). Following exit from the warm water, T_{es} , T_{skavg} , and skin blood flow all returned to control values within 10 min. Therefore the post-exercise increases in T_{es} and vasodilation threshold do not seem to be solely a consequence of increased

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body heat content. In addition, successive exercise/recovery cycles, performed at progressively increasing pre-exercise T_{es} , resulted in further and parallel increases of the vasodilation threshold during exercise and the post-exercise plateau in T_{es} , as well as an increased post-exercise vasodilation threshold (Kenny et al. 1996a). These results lend further support to a residual exercise-related effect on the warm thermoregulatory response of active cutaneous vasodilation.

It is unclear whether this exercise-related effect is limited to the warm thermoregulatory response of cutaneous vasodilation, or if a similar effect on the sweating response also occurs. The core temperature threshold for sweating has been reported to increase (Johnson and Park 1981), decrease (Tam et al. 1978; Lopez et al. 1995), or remain unchanged (Kellogg et al. 1991) from baseline during exercise itself. Similarly, no change was reported to occur during recovery in a protocol involving hyperhydration (Lopez et al. 1995). Since hyperhydration itself decreases the sweating threshold (Greenleaf and Castle 1971), the present study evaluates the hypothesis that, at normal hydration status, the sweating threshold decreases during intense exercise, but that residual exercise-related factor(s) increase the subsequent post-exercise sweating threshold relative to baseline values.

Methods

Subjects

With approval from our Faculty Human Ethics Committee, seven healthy subjects (four males, three females) with no history of cardiovascular or respiratory disease, participated in this study after providing their written, informed consent. Subjects were physically active, although none engaged in daily or intensive training programs. Subjects were 28 (6) (mean \pm SD) years old, 1.8 (0.1) m tall and weighed 69 (10) kg. Female subjects were eumenorrheic, and reported regular, approximately 28-day-long, menstrual cycles. To control for hormonal effects female subjects were studied within 9 days after starting menstruation (follicular phase).

Instrumentation

Core temperature was measured using an esophageal thermocouple inserted through a nostril to the level of the heart. Skin temperatures were monitored at nine sites and the area-weighted mean was calculated by assigning the following regional percentages: head 6%, upper arms 9%, forearms 9%, fingers 2%, back 22%, chest 11%, abdomen 11%, thigh 17%, and calf 13%.

Sweat rate was measured using a ventilated capsule ($\approx 5.0 \times 3.5$ cm) placed on the forehead. Anhydrous compressed air was passed through the capsule over the skin surface at a rate of $11 \cdot \text{min}^{-1}$ (Brooks 5850 Mass Flow Controller, Emerson Electric, Hetfield, Pa., USA). The vapor density of the effluent air was determined according to the relative humidity and temperature of the air, as measured by an Omega HX93 humidity and temperature sensor (Omega Engineering, Stamford, Conn., USA). Sweat rate was defined as the product of the difference in water content between effluent and influent air, and the flow rate. This value was adjusted for the skin surface area under the capsule and expressed in $\text{g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$. Sweating was monitored throughout the study.

Peripheral vessel tone was measured to determine the time course of the return to baseline values following warm-water immersion or exercise. Accordingly, fingertip blood flow was assessed using a perfusion index (PI_{avg}) derived from an Ohmeda Biox 3700 pulse Oximeter (Ohmeda, Louisville, Colo., USA) with an oximeter probe placed on the middle digit. This method correlates well with blood flow as measured by volume plethysmography ($r^2 = 0.88$) (Ozaki et al. 1993).

The subjects' $\dot{V}O_2$ was determined using an open-circuit method from measurements of expired minute volume and inspired and mixed expired gas concentrations sampled from a 10-l fluted mixing box.

Analog data from the thermocouples and gas analyzers were acquired using an electrically isolated Macintosh IIci computer (Apple Computer, Cupertino, Calif., USA) equipped with a NB-MIO-16L 16-channel analog-to-digital converter (National Instruments, Austin, Tex., USA). Data were digitized asynchronously at 2 Hz and displayed graphically at 30-s intervals.

Experimental protocol

Subjects performed one incremental maximal $\dot{V}O_2$ test on a cycle ergometer on the first day. These data were used to select the work load for the submaximal experimental exercise trial. The experimental trial was conducted in the morning following a 24-h period without heavy or prolonged physical activity, the last 12 h of which included abstinence from stimulants and alcohol, 8 h of sleep and a minimum of 0.25 l of water during each waking hour. On each study day care was taken to avoid major thermal stimuli or any substantial increase of metabolic rate between awakening and the start of the experiment. Subjects were clothed in bathing suits and were instrumented appropriately. All experimental trials were conducted in the morning. Baseline data were collected over 30 min at an ambient air temperature (T_a) of 24°C. These subjects were then immersed to the level of their clavicles in water at a temperature (T_w) of 42°C (Immersion 1) until 3–5 min following initiation of sweating. Subjects then rested (20–35 min) in air ($T_a = 24^\circ\text{C}$) until T_{es} , T_{skavg} and finger tip blood flow returned to baseline values. Subjects then exercised on a cycle ergometer at 70% $\dot{V}O_{2\text{max}}$ for 15 min (Exercise) and then rested for 30 min. The exercise bout was sufficient to initiate sweating. The rest period was sufficiently long that T_{skavg} and finger tip blood flow returned to baseline (Kenny et al. 1996a). Subjects were then again immersed in water ($T_w = 42^\circ\text{C}$) (Immersion 2) until 3–5 min after initiation of sweating.

The sweating threshold was defined as the onset of a sustained sweat rate exceeding $50 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$ (Lopez et al. 1995). To compare thresholds at different T_{skavg} and T_{es} , the following equation (Matsukawa et al. 1995) was used to correct the T_{es} ($T_{es(\text{calculated})}$) for a designated skin temperature ($T_{\text{sk}(\text{designated})}$):

$$T_{es(\text{calculated})} = T_{es} + [\beta/(1-\beta)][T_{\text{skavg}} - T_{\text{sk}(\text{designated})}] \cdot T_{\text{sk}(\text{designated})}$$

was set as the mean T_{skavg} of Immersion 1 and Immersion 2 conditions (i.e., 36.5°C) and β represents the fractional contribution of the skin to the sweating response ($\beta = 0.1$) (Nadel et al. 1971a).

Analysis of results

An analysis of variance for repeated measures was used to compare:

1. T_{es} , T_{skavg} , and finger blood flow values for pre-Immersion 1 (average of the final 5 min), post-Immersion 1 (average of the final 5 min), end-Exercise, and post-Exercise (average of the final 5 min).
2. T_{es} , T_{skavg} , and $T_{es(\text{calculated})}$ at the threshold for sweating for the three conditions (i.e., Immersion 1, Exercise, and Immersion 2).

In the event of statistical significance ($P < 0.05$), a Tukey's test was used to identify significant differences.

Results

Immersion 1

Baseline T_{es} , and T_{skavg} , were 37.0 (0.1)°C and 32.3 (0.3)°C, respectively. During transfer to the water tank and immersion in water ($T_W = 42^\circ\text{C}$) there was a transient liminal decrease in T_{es} of 0.2°C followed by a steady increase to 37.3°C at the end of immersion (average immersion time was 17.2 min). On average, sweating onset occurred 9.8 min after immersion at a T_{es} of 37.0 (0.1)°C (Table 1). At the onset of sweating the $T_{es(\text{calculated})}$ at a designated T_{skavg} of 36.5°C was 37.1 (0.1)°C. During recovery T_{es} decreased to 37.1°C within an average of 20 min and remained constant for the last 10 min of recovery. Recovery T_{es} was not significantly different from baseline. T_{skavg} and finger blood flow returned to baseline values within 15–20 min of recovery (Fig. 1).

Exercise

Upon initiation of exercise T_{es} increased at a rate of $0.2^\circ\text{C} \cdot \text{min}^{-1}$ during the first 7.5 min after which T_{es} either remained stable or rose only slightly reaching an end-exercise temperature of 38.0 (0.2)°C. $T_{es(\text{calculated})}$ at the sweating threshold [$36.7 (0.2)^\circ\text{C}$] was lower than during Immersion 1 ($P < 0.05$), (Fig. 2). Following exercise termination T_{es} decreased from 38.0°C to 37.44°C within 15 min with only a slight further decrease to 37.4°C after 30 min. This plateau was significantly higher than the pre-exercise value ($P < 0.05$). T_{skavg} and finger tip blood flow returned to baseline values by the 25th min of the 30-min recovery period.

Immersion 2

Upon immersion in water ($T_W = 42^\circ\text{C}$), T_{es} decreased transiently by 0.1°C followed by a steady increase to 37.4 (0.1)°C at the end of immersion (11 min). Sweating onset occurred 7.2 min after immersion at a $T_{es(\text{calculated})}$ of 37.3 (0.1)°C that was greater than following both Immersion 1 ($P < 0.05$) and Exercise ($P < 0.05$) (Fig. 2).

Table 1 Mean temperatures for sweating onset during Immersion 1, Exercise, and Immersion 2. Values are expressed as the mean (SD). (T_{skavg} Average skin temperature, T_{es} esophageal temperature, $T_{es(\text{calculated})}$, T_{es} corrected for a designated T_{skavg} of 36.5°C)

Variables	Temperatures at sweating thresholds		
	Immersion 1	Exercise	Immersion 2
T_{skavg} (°C)	36.76 (0.2)	30.93 (0.3)*	36.38 (0.2)**
T_{es} (°C)	37.04 (0.1)	37.30 (0.1)*	37.34 (0.1)*
$T_{es(\text{calculated})}$ (°C)	37.07 (0.1)	36.69 (0.2)*	37.33 (0.1)**

*Temperature at sweating threshold significantly different from Immersion 1 ($P < 0.05$)

**Temperature at sweating threshold significantly greater than for Exercise ($P < 0.05$)

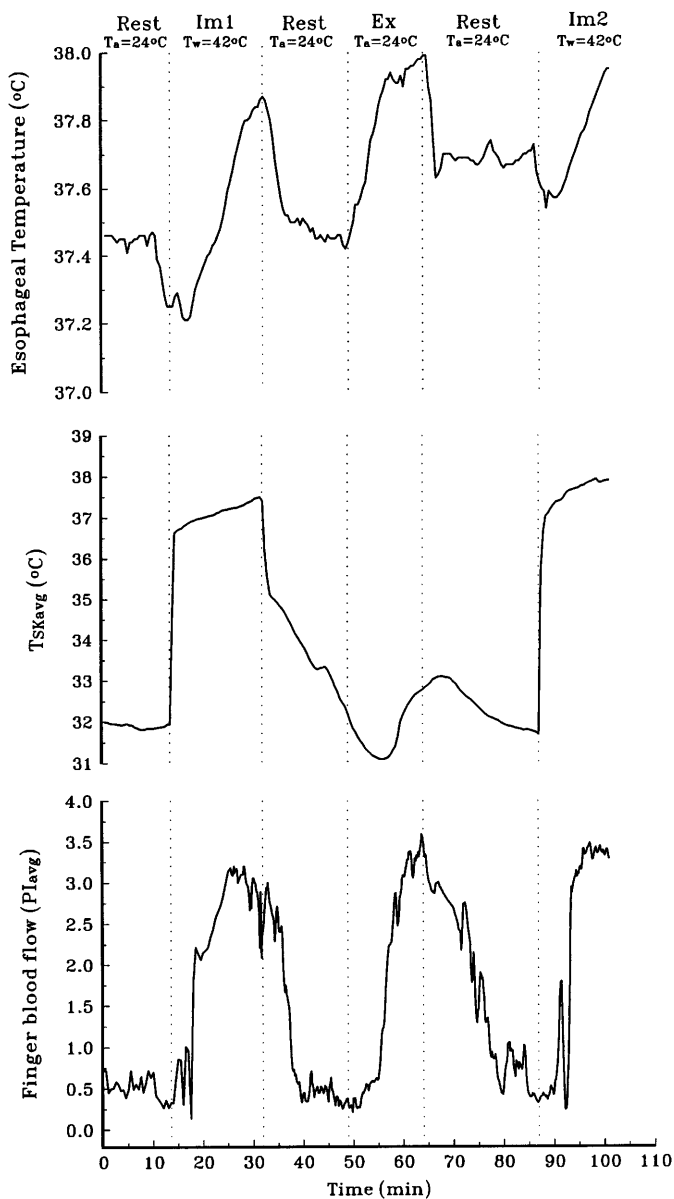


Fig. 1 Esophageal (T_{es} , top), and average skin temperatures (T_{skavg} , middle) finger blood flow (PI_{avg} , bottom) and sweating for one subject during baseline, Immersion 1 (*Im 1*), post-Immersion 1 (*Rest*), Exercise (*Ex*), post-exercise recovery (*Rest*) and Immersion 2 (*Im 2*). The dashed line indicates the start and end of each period. (T_a Air temperature, T_W Water temperature)

The absolute sweating thresholds for females were 0.5 (0.1)°C higher than for males. However, the sweating thresholds consistently decreased from Immersion 1 to Exercise and subsequently increased during Immersion 2, with Immersion 2 thresholds being greater than for Immersion 1 for all but one subject.

Discussion

This study is the first to demonstrate a residual increase in the resting sweating threshold following intense ex-

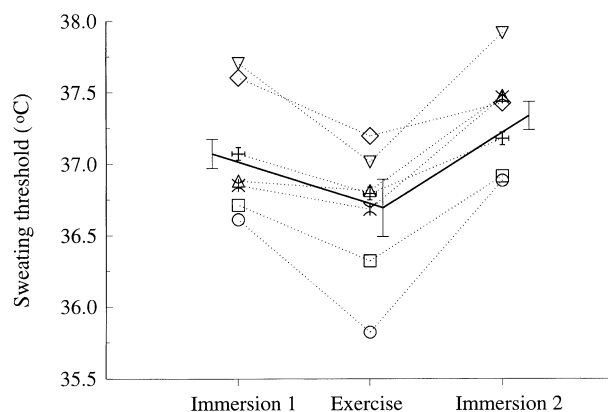


Fig. 2 Mean and individual sweating thresholds ($n = 7$), during *Immersion 1*, *Exercise*, and *Immersion 2*. The 0.4°C decrease in the sweating threshold during *Exercise*, and the 0.3°C increase in sweating threshold during *Immersion 2* were significantly different from that of *Immersion 1* ($P < 0.05$). The solid line represents the mean (SD) of all subjects

ercise. There was a 0.4°C decrease in sweating threshold during exercise itself, but a subsequent 0.6°C increase during recovery with values significantly higher than resting pre-exercise conditions.

Other studies have demonstrated a reduction in the sweating threshold during exercise in dogs (Jackson and Hammel 1963) and humans (Jequier 1970; Tam et al. 1978; Lopez et al. 1995); the effect being greater as exercise intensity increases from 40 to 70% of maximum $\dot{V}\text{O}_2$ (Tam et al. 1978). However, some studies have demonstrated no decrease in the sweating threshold during exercise in humans (Benzinger et al. 1961; Kellogg et al. 1991), while others report an increase (Johnson and Park 1981; Mekjavic and Bligh 1989).

Lopez et al. (1995) conducted the only other study of post-exercise sweating thresholds. Although they also found a decreased sweating threshold during exercise, the post-exercise sweating threshold did not differ from pre-exercise values.

Possible mechanism for results obtained during exercise

The mechanism by which exercise reduces the sweating threshold remains unclear. Previous work has demonstrated that the thermoregulatory system maintains core temperature within a narrow range (i.e., $\approx 0.2^{\circ}\text{C}$) in which a small change in core temperature does not trigger a thermoregulatory response. This interthreshold range in core temperature between thresholds for heat loss and heat gain mechanisms may indicate either: 1) a set-point control with a tolerance for small core temperature displacement before heat loss or heat gain responses are initiated (Sawka and Wenger 1988), or 2) separate reference temperatures for each thermoregulatory response (Lopez et al. 1994). It is possible that the decrease in sweating threshold represents either a decrease in the set-point, a specific decrease in the reference temperature for sweating onset, or an increase in the gain

of the central controller (Jackson and Hammel 1963; Nadel et al. 1971b; Tam et al. 1978). Such a decrease could represent a teleological advantage since a feed-forward mechanism acts to prevent or minimize, in this case, the increase in core temperature during exercise.

It is possible that the decrease in sweating threshold during exercise was merely a result of an increase in the integrated thermal signal (from the core, skin and muscles) at a given T_{es} , since muscle temperatures were likely to still be elevated following Immersion 1. However, Lopez et al. (1995) reduced core temperature prior to exercise by central venous infusion of cold lactated-Ringer's solution at 3°C and observed a similar decrease in sweating threshold during exercise. The fact that the sweating threshold during exercise was decreased in both studies favors an actual shift in central thermoregulatory control.

It may be that during exercise circulating catecholamines (Sato and Sato 1981) or increased sympathetic activity (Stolwijk and Nadel 1973) could reduce the sweating threshold. The reduction in the sweating threshold that occurred during exercise could also have been a function of a greater rate of warming of T_{es} or T_{skavg} during exercise compared to Immersion 1. Changes of T_{skavg} in the order of $1^{\circ}\text{C} \cdot \text{min}^{-1}$ are required to exert a significant effect on the output from the central controller (Wyss et al. 1975). Since our rates of increase for T_{skavg} were 0.10 and $0.17^{\circ}\text{C} \cdot \text{min}^{-1}$ for Immersion 1 and Exercise, respectively, our results are not likely to be due to dynamic changes in skin temperature.

Mittleman and Mekjavic (1991) demonstrated only a small change in the thermoregulatory response during cold-water immersion when core temperature cooling was increased from 3.0 to $13.8^{\circ}\text{C} \cdot \text{h}^{-1}$. Lopez et al. (1994) demonstrated that small differences in the rate of core temperature change (i.e., 0.7 and $1.7^{\circ}\text{C} \cdot \text{h}^{-1}$) had no effect on the response of the sweating threshold. In our study core temperature was increased at a rate of 3.6 ($0.9^{\circ}\text{C} \cdot \text{h}^{-1}$) during warm-water immersion (Immersion 1), but was not significantly different from the 6.0 ($1.1^{\circ}\text{C} \cdot \text{h}^{-1}$) increase that occurred during Exercise. Thus, it is unlikely that differences in core warming rates affected our results.

Possible mechanism for post-exercise results

Although the results we obtained during Exercise are in agreement with those of Lopez et al. (1995), they differ with respect to the post-exercise period. Lopez and co-workers observed no change in the post-exercise sweating threshold compared to pre-exercise values. On the other hand, following Exercise we measured a 0.3°C increase from pre-Exercise values and a 0.6°C increase over Exercise values. During the study of Lopez et al. (1995), subjects were infused with 3–5 l of fluid over a 2.5-h period. Greenleaf and Castle (1971) demonstrated that sweating threshold decreases with even moderate hyperhydration (i.e., a 1.2% body weight increase).

Therefore, the similarities in pre- and post-exercise sweating threshold observed in the study of Lopez et al. (1995) could be a result of a balance between the threshold-decreasing effect of hyperhydration offsetting the threshold-increasing effect of exercise.

Alternatively, the increase in the post-exercise sweating threshold observed in the present study could be a result of, or accentuated by, relative hypohydration. Numerous studies have shown that hypohydration increases the sweating threshold (Fortney et al. 1984), and that the magnitude of the response is dependent upon the level of hypohydration (Montain et al. 1995). Although we did not quantify the hydration status of our subjects, it is unlikely that any significant hypohydration occurred. Montain and Coyle (1992) demonstrated that 2 h of heavy dynamic exercise in a warm environment (33°C) with no water intake results in a maximum weight loss of 4.2%. Therefore, in our study the short period of exercise (only 15 min) in a cooler environment (24°C) with unrestricted pre-trial water intake is unlikely to have caused more than a 0.5% weight loss. Under these conditions our subjects could be considered to be euhydrated (Greenleaf and Castle 1971).

The prolonged nature of the protocol we used was unlikely to contribute to an elevated sweating threshold during Immersion 2. Brengelmann et al. (1994) made sequential measurements and actually demonstrated a time-dependent *decrease* in the sweating threshold by 0.09 to 0.17°C. Finally, we measured the forehead skin temperature adjacent to the sweat capsule, since it is known that local skin temperature can affect the central control of sweating (Nadel et al. 1971a). There was no significant difference in forehead skin temperature at sweating onset between Immersion 1 [36.0 (0.6)°C] and Immersion 2 [35.8 (0.5)°C]. Data from the study of Nadel et al. (1971b) indicate that, at the range of core and skin temperatures observed in the present study, a decrease in local skin temperature of as much as 1.4°C would decrease the core threshold for sweating onset by only 0.05°C.

In addition to our demonstration of a post-exercise increase in the sweating threshold, we have previously demonstrated increases in post-exercise thresholds for vasoconstriction and shivering (Kenny et al. 1996c). The increases in thresholds for both heat loss and heat gain responses, and the elevated post-exercise plateau in T_{es} while $\dot{V}O_2$, T_{skavg} , and fingertip blood flow return to baseline values (within 10–20 min), are consistent with an increase in the post-exercise set-point or parallel increases in the thresholds for each response. These thermoregulatory changes are likely to be due to some residual exercise-related factors such as endocrine changes (Francesconi 1996), endogenous pyrogen, metabolic byproducts (Cannon and Kluger 1983; Francesconi 1996), or baroreflex activity (Kellogg et al. 1991). In summary, we provide the first quantification of post-exercise sweating thresholds under normal hydration status. One possible advantage of delaying sweating by increasing the post-exercise sweating threshold would be

to conserve body fluid following significant fluid losses during exercise. We conclude that residual exercise-related factor(s) increase the post-exercise sweating threshold.

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