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Effect of Intensive and Repetitive Heat Exposure on the Sudomotor Activity

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Abstract

The sweating response is modulated in two different ways depending on adaptation conditions. In this work, we examined sudomotor activities before and after intensive and repetitive heat exposure. Nine male volunteers were exposed to 30-min half-body immersion in hot water (42 ± 0.5°C) at the same time of day on alternate days for 3 weeks. All experiments were performed in an automated climate chamber. Tympanic (Tty) and skin (Ts) temperatures were measured. Mean body temperature (mTb) was calculated. Sudomotor activities, including sweat onset time, sweat rate and volume, activated sweat gland density (ASGD) and output (ASGO), were tested in four regions of the skin: chest, abdomen, upper back and thigh. Basal Tty and mTb were found to decrease by 0.15°C ($P < 0.05$) and 0.16°C ($P < 0.05$), respectively. As a typical data (upper back), sweat onset time increased by 33.6% ($P < 0.05$) after heat acclimation. After heat acclimation, sweat rate decreased by 14.7% ($P < 0.05$), sweat volume decreased by 15.5% ($P < 0.05$) and ASGO also decreased by 11.1% ($P < 0.05$). ASGD decreased by 4.1% after heat acclimation without statistical significance. The data suggest that intensive and repetitive heat exposure induces suppression of sudomotor activities within 3 weeks.

Key Words: heat acclimation, heat exposure, sweat gland density, sweat gland output, sweat rate

Introduction

Heat acclimation is an adaptive physiological process that is induced by repeated exposure to high ambient temperature. When fully acclimated, the sweating response is modulated in two steps. First, sweating begins much sooner following the onset of exercise. Second, sweat production is enhanced so that a larger volume of sweat is produced (8, 27). On the contrary, for tropical inhabitants, heat tolerance is characterized by suppressed sweating (12, 17, 18), which is distinguishable from short-term heat adaptation. In humans, thermal stress can be a challenge to the function of cardiovascular and temperature regulation, body fluid balance and exercise performance (3). Prolonged exposure to hyperthermic conditions can induce water deficits due to excessive sweating and can eventually lead to dehydration. Therefore, in the process of heat acclimation, sweating activity is attenuated on repetitive and intensive heat exposure after getting temporary heat acclimation.

The process of heat acclimation is thought to be more complicated than simply dividing the acclimation process into short- and long-term processes. In general, a longer-term heat expsoure of at least a few months does not seem to be required for the suppression of sweat gland sensitivity to heat. Seasonal acclimation is long-term heat acclimation. We have previously documented that central and

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peripheral sudomotor activity decreases after the summer season (11, 14). Based on seasonal acclimation, we hypothesized that more intensive and repetitive passive heat stress could more rapidly reduce sudomotor activity in response to the heat. To test the hypothesis, we measured sudomotor activity including sweat onset time, sweat rate and sweat volume, activated sweat gland density (ASGD) and activated sweat gland output (ASGO) before and after 3 weeks of 30-min half-body immersion in hot water (42 ± 0.5°C) in human volunteers.

Materials and Methods

Subjects

This protocol was approved by the Soonchunhyang University Research Committee. Each subject gave his written informed consent after being informed of the purpose and experimental procedures and potential risks. Nine healthy male college students (age, 21.9 ± 2.7 years; height, 177.4 ± 3.2 cm; weight, 74.1 ± 5.8 kg; body mass index (BMI), 20.1 ± 2.0 kg/m^2; body fat, 16.5 ± 3.1%; maximum oxygen uptake (VO_2max), 54.6 ± 4.4 ml/kg/min) were enrolled. The subjects refrained from alcohol consumption, smoking, medications and vigorous physical activity during the study period.

Heat Load

All experiments were undertake in a thermoneutral climate chamber with mean air temperature and relative humidity of 26 ± 0.5°C and 60 ± 3% relative humidity, respectively, and an air velocity <1 m/sec between 2 and 5 PM. The subjects were exposed to a 30-min half-body immersion in hot water (42 ± 0.5°C) at the same time of day on alternate days for 3 weeks. The subjects wore short pants and relaxed for 60 min to become conditioned to the chamber atmosphere prior to beginning each experiment. After 60 min of rest, the aforementioned 30-min heat load was applied. Due to the uncomfortable hot water temperature, subjects were allowed to take one-minute breaks at 5, 10 and 20 min check points during the immersion. Drinking water during the immersion experiments was not permitted.

Tympanic Temperature (Tty) Measurement

Measurements were performed at the first and tenth 30-min half-body immersion. Tty was assessed continuously at 10-s intervals for 30 min in the left ear using a model TSK7+1 thermistor probe (Songkitopia, Incheon, Korea) with a small spring (TAKARA Instrument Co. Ltd., Yokohama, Japan) connected to a model CF-T1 personal computer, which was in turn connected to a data logger (K-720; Technol Seven, Yokohama, Japan) (13, 19).

Mean Body Temperature (mTb) and Mean Skin Temperature (mTs) Measurements

Measurements were performed at the first and tenth 30-min half-body immersion. Skin temperature (Ts) were measured at the 60-s break after 5 min, 10 min and 20 min, and at the end of the water exposure session. Ts on the chest (T1), upper arm (T2), thigh (T3) and leg (T4) were measured using the model TSK7+1 thermistor probe with a model PXK-67 thermistor thermometer (Technol Seven, Yokohama, Japan) connected to the model K-720 data logger (28). mTs was calculated using the Ramanathan equation (28): mTs = 0.3 × (T1 + T2) + 0.2 × (T3+ T4). mTb was calculated from the Tty and mTs using the following equation: mTb = 0.9 × Tty + 0.1×mTs (13).

Measurements of Sweat Rate, Sweat Volume and Sweat Onset Time

Measurements were performed at the first and tenth 30-min half-body immersion. During heat loading, sweat rate at the chest, abdomen, upper back and thigh were continuously recorded by a capacitance hygrometer-ventilated capsule method. In brief, dry nitrogen gas was set to flow into a capsule attached to the skin (13). The humidity of the effluent gas was measured with a model H211 hygrometer (Technol Seven). Sweat rate was captured every 30s with a model CF-T1 personal computer and was expressed as mg/cm^2/min (13). Sweat volume was calculated from sweat rate for 30 min, while local sweat onset time was calculated from sweat rate on the chest, upper back, abdomen and thigh.

Measurements of ASGD and ASGO

Measurements were also performed at the first and tenth 30-min half-body immersion. ASGD was measured with a starch-iodide paper to evaluate qualitative glandular activities. Starch-iodide paper were attached to the right side of the abdomen, chest, upper back and thigh to obtain blue-black colored marks at the 60-s break, after 5, 10 min and 20 min, and at the end of the bathing. To obtain an average ASGD, three sectors (0.5 × 0.5 cm) were marked on the paper strip, and the number of marks was counted (12). Counting was conducted by a single experienced researcher who counted each sector in 10–15 s. The average ASGD (count/cm^2) represented the sum of the three sectors/3 × 4 (12). The average ASGO

(μg/min/single gland) was obtained by dividing the sweat rate (mg/cm^2/min) by ASGD (12, 16).

Statistical Analysis

Values are expressed as mean ± standard deviation (SD) using the SPSS for Windows software (SPSS Inc., Chicago, IL, USA). A normal distribution could be assumed for the Pre-Post differences. The paired *t*-test was used to compare mean values (two-tailed). The effect size (using Cohen's d) was calculated. The level of significance was set at $P < 0.05$.

Results

As shown in our previous study with the same protocol, after heat acclimation, basal Tty decreased by 0.15°C from 36.78 ± 0.13°C to 36.65 ± 0.11°C ($P < 0.05$, $d = 1.08$), and mTb also decreased by 0.16°C from 36.36 ± 0.16°C to 36.22 ± 0.08°C ($P < 0.05$, $d = 1.11$) (34). In this work, local sudomotor activities for sweat onset time, sweat rate, sweat volume, ASGD and ASGO in the four target regions of the body of the participants were measured (Figs. 1-5). There was an increase in local sweat onset time and decreases in local sweat rate, sweat volume and ASGO (Figs. 1-3 and 5). Mean ASGD calculated from the four skin regions was decreased without statistical significance (Fig. 4).

After heat acclimation, local onset time of sweating was delayed by 28.72% in the chest, from 5.71 ± 2.29 to 7.35 ± 2.06 min ($P < 0.01$, $d = -0.75$), by 36.33% in the abdomen from 5.89 ± 2.51 to 8.03 ± 2.11 min, ($P < 0.01$, $d = -0.92$), by 33.62% in the upper back from 5.86 ± 2.42 to 7.83 ± 2.04 min, ($P < 0.01$, $d = -0.88$), and by 20.88% in the thigh from 5.94 ± 2.27 to 7.18 ± 2.10 min ($P < 0.01$, $d = -0.57$) compared to those before heat acclimation (Fig. 1). Local sweat rate decreased, after heat acclimation, by 17.11% in the chest from 0.76 ± 0.19 to 0.63 ± 0.17 mg/cm^2/min ($P < 0.01$, $d = 0.72$), by 6.82% in the abdomen from 0.44 ± 0.20 to 0.41 ± 0.15 mg/cm^2/min ($P < 0.05$, $d = 0.17$), by 14.77% in the upper back from 0.88 ± 0.24 to 0.75 ± 0.21 mg/cm^2/min ($P < 0.05$, $d = 0.58$), and by 17.74% in the thigh from 0.62 ± 0.18 to 0.51 ± 0.14 mg/cm^2/min ($P < 0.05$, $d = 0.68$) compared to those before heat acclimation (Fig. 2). Local sweat volume decreased, after heat acclimation, by 17.69% in the chest from 22.78 ± 6.51 to 18.75 ± 5.38 mg/cm^2 ($P < 0.01$, $d = 0.67$), by 8.51% in the abdomen from 13.28 ± 7.04 to 12.15 ± 4.82 mg/cm^2 ($P < 0.05$, $d = 0.19$), by 15.45% in the upper back from 26.47 ± 8.91 to 22.38 ± 6.28 mg/cm^2 ($P < 0.01$, $d = 0.53$) and by 17.70% in the thigh from 18.47 ± 6.15 to 15.20 ± 3.73 mg/cm^2 ($P < 0.01$, $d = 0.64$) compared to those before heat acclimation (Fig. 3). After heat acclimation, ASGD decreased by 8.61% in the chest from 65.17 ± 15.84 to 59.56 ± 13.19 count/cm^2 ($P > 0.05$, $d = 0.38$), by 2.75% in the abdomen from 76.40 ± 16.30 to 74.30 ± 13.24 count/cm^2 ($P > 0.05$, $d = 0.14$), by 4.14% in the upper back from 79.45 ± 19.33 to 76.16 ± 17.10 count/cm^2 ($P > 0.05$, $d = 0.18$), and by 13.51% in the thigh, from 46.57 ± 13.19 to 40.28 ± 11.70 count/cm^2 ($P > 0.05$, $d = 0.50$) (Fig. 4). ASGO decreased, after heat acclimation, by 8.65% in the chest from 11.56 ± 2.28 to 10.56 ± 2.04 μg/min/single ($P < 0.05$, $d = 0.46$), by 4.17% in the abdomen from 5.76 ± 1.21 to 5.52 ± 1.16 μg/min/single ($P > 0.05$, $d = 0.20$), by 11.10% in the upper back from 11.08 ± 2.19 to 9.85 ± 2.27 μg/min/single ($P < 0.01$, $d = 0.55$), and by 4.52% in the thigh from 13.26 ± 1.96 to 12.66 ± 1.50 μg/min/single ($P < 0.01$, $d = 0.34$) compared to those before heat acclimation (Fig. 5).

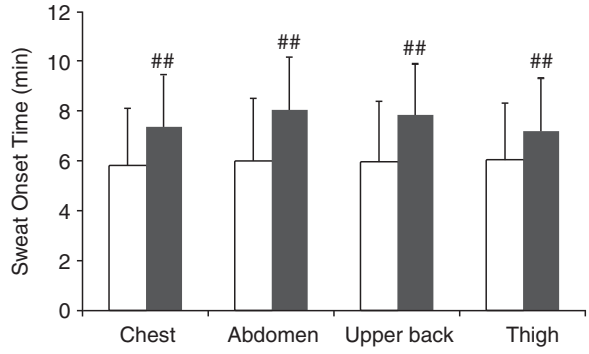

Fig. 1. Local sweat onset time during heat load before (white bars) and after (black bars) heat acclimation. Values (n = 9) are mean ± SD. $^{\#\#}P < 0.01$ indicates a significant difference between before and after heat acclimation.

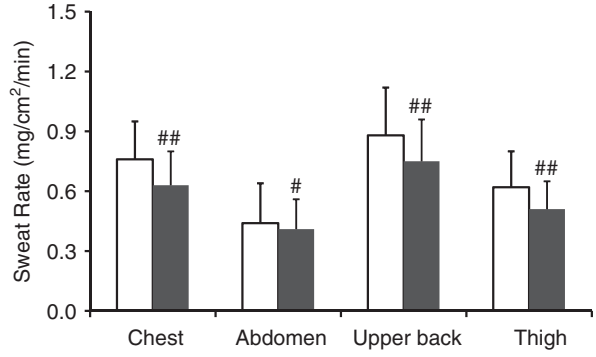

Fig. 2. Local sweat rate during heat load before (white bars) and after (black bars) heat acclimation. Values (n = 9) are mean ± SD. $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ indicates a significant difference between before and after heat acclimation.

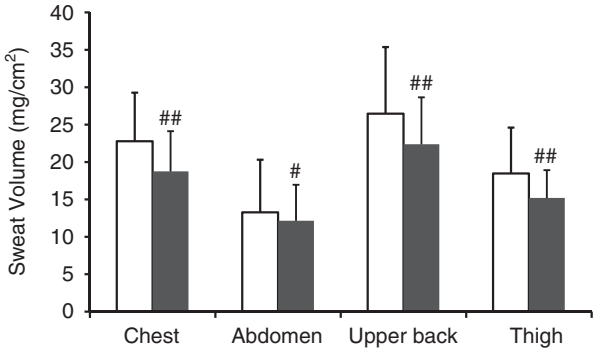

Fig. 3. Local sweat volume during heat load before (white bars) and after (black bars) heat acclimation. Values (n = 9) are mean ± SD. $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ indicates a significant difference between before and after heat acclimation.

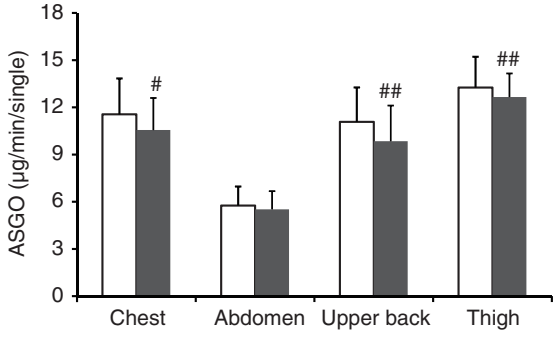

Fig. 5. Local ASGO during heat load before (white bars) and after (black bars) heat acclimation. Values (n = 9) are mean ± SD. $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ indicates a significant difference between before and after heat acclimation.

Discussion

To our knowledge, the present results are the first demonstration that suppression of sudomotor activity can be induced by repetitive and intensive passive heat loading within 3 weeks. Our results showed that resting body temperature decreased, sweat onset time increased, and local sweat rate, sweat volume and ASGO decreased after heat acclimation. ASGD also decreased, but to a degree without statistical significance. These changes may have been induced by an enhanced thermotolerance and defense system to minimize dehydration during the prolonged repetitive heat strain. In addition, reduced sweating activity may be attributable to desensitized acetylcholine (ACh) stimulation. In one study, the administration of ACh four times a day for 9 days suppressed local sweating under passive heating (2). Suppressed sudomotor function in response to ACh has been documented in tropical conditions (17, 18). The

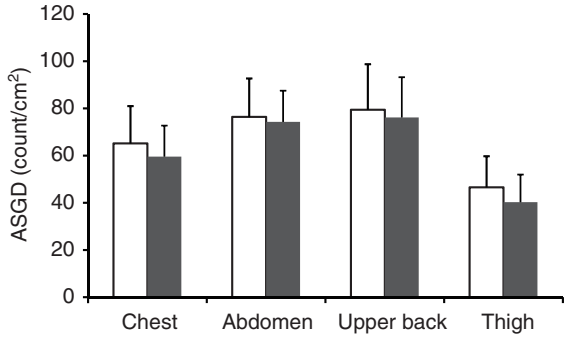

Fig. 4. Local ASGD during heat load before (white bars) and after (black bars) heat acclimation. Values (n = 9) are mean ± SD.

results imply that repetitive stimulation of endogenous ACh worsens peripheral sensitivity to ACh. Our results are consistent with these studies.

The present study showed a decrease in resting body temperature after heat acclimation. Previously, our studies also demonstrated that resting body temperature and basal metabolic rate (BMR) reduced after the summer season (10, 11). BMR plays a role in maintaining body temperature. Metabolic heat produced in the body must be dissipated to maintain a constant body temperature (4). There is evidence supporting the apparent reduction of BMR during extended exposure to hot climates (21, 36, 38). Because a lower basal metabolism is more favorable for maintaining both a lower body temperature in a hot environment and tolerance to heat (4), a reduction in resting body temperature after intensive and repetitive heat loading is reasonable in a preset study.

Heat acclimation results in whole-body adaptations that improve heat tolerance. It also affects thermoregulatory molecules responses such as prostaglandin E_2 (PGE_2), cyclooxygenase (COX)-2 and orexin. Our previous data suggest that decreased basal body temperature after heat acclimation is associated with decreases in PGE_2, COX-2 and orexin (34). PGE_2 is the principal mediator of fever and exerts its pyrogenic action by binding to receptors on thermoregulatory neurons in the anterior hypothalamus (9). COX-2 is the rate-limiting enzyme for PGE_2 synthesis (20). Orexin has widespread actions, and it influences many physiological functions including body temperature and energy metabolism (35). Reduction of the levels of the above molecules after heat acclimation would reinforce the decrease in resting body temperature after heat acclimation.

Subjects from tropical regions are able to maintain lower core and Ts as compared to subjects from temperate regions (12, 22). Conversely, other studies have found that tropical residents have a higher core temperature in a resting state (23-25, 29), even though they show sweat rate suppression. This

adaptation, which is usually accompanied by increased Ts, affords them enhanced dry heat loss through convection, conduction and radiation, which is beneficial in reducing the water losses associated with sweating (8). Furthermore, regulating the core body temperature at a higher set-point reduces the total amount of heat that must be dissipated (8). The conflicting observation about core temperatures is thought to be related to the different climate zones in the different studies. And heat exposure conditions, such as sunlight *vs*. passive heating, consecutive *vs*. intermittent exposure, and duration of exposure, may all have contributed to the discrepant results. Further studies are needed to clarify the alterations of resting core and Ts after long-term heat acclimation.

ASGD and ASGO are major components of sweat rate. Increased sweat rate could be due to an increased ASGD, an increased ASGO (33), or a combination of both factors (6, 30). Presently, the decreased sweat rate appeared to result from reduced ASGO and, to a lesser extent, from reduced ASGD.

The size and number of activated sweat glands can change upon heat exposure (26). Nielsen reported that increased sensitivity of the sweat glands after acclimation may be obtained through an increase in the size or number of active sweat glands (26). Kondo *et al*. demonstrated that increased sweating rate relies on both ASGD and ASGO during exercise and passive heating (5). Previously, we also observed that 10 days of heat exposure increased ASGO and led to a tendency towards an increase in ASGD (15). And we found that trained athletes showed a greater increase in ASGD and ASGO under a sweating test as compared with normal subjects (33). However, there are few studies regarding the modification of ASGD and ASGO after long-term heat acclimation, which the present study aimed to address. Sweating responses are governed by thermal and non-thermal factors, and ASGD and ASGO are modulated by both factors (1, 6, 7). Kondo *et al*. demonstrated that changes in sweat rate rely on both ASGD and ASGO during the initial period of exercise and passive heating, whereas further increases in sweat rate are dependent on increases in ASGO in both conditions (5). This implies that a further increase in sweat rate is to be primarily mediated by increases in ASGO. On the other hand, sweating decreases in aged mice mainly due to a decline of ASGO and, to a lesser extent, to a decrease in the number of secreting glands. A reduction of the size of sweat glands in aged mice was also found, suggesting that the diminished sweat gland responsiveness with ageing may be attributed to sweat gland atrophy as well as to loss of innervation (37). In summary, sweat rate is primarily modified by an increase or decrease in ASGO, rather than in ASGD, after an initial period of sweating. Our results are consistent with these studies.

Short-term heat acclimation has been demonstrated in many previous studies. However, little is known about the early temporal features of long-term heat acclimation. Sudomotor activity is attenuated in tropical residents and in those who are acclimated to summer conditions. The time course and magnitude of the adaptations are a function of both the frequency and the intensity of exposure to heat conditions (8). Intensive heat load may contribute to rapid establishment of long-term heat acclimation. In the present study, heat load was applied to the subjects by immersion to waist level in a hot water bath maintained at 42°C for 30 min with brief breaks during the immersion period. The data showed that intensive and repetitive heat exposure resulted in the suppression of sudomotor activity within 3 weeks. Given exposure to water temperatures below 40°C, the results might be different.

In this study, sweat activity was measured on the 4 skin regions with different density and capacity of the sweat glands (6, 31, 32). In addition, the patterns of change in ASGD and ASGO differed from one region of the body to another (6). Thus, a detailed study should be undertaken to examine regional differences in the contribution of ASGD and ASGO to changes in sweat rate during heat acclimation.

In conclusion, data of the present study suggest that intensive and repetitive heat exposure induces suppression of sudomotor activity within 3 weeks. These changes may have been induced by an enhanced thermotolerance and defense system to minimize dehydration during prolonged repetitive heat strain.

Acknowledgments

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Conflict of Interest

The authors declared no conflicts of interest.

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