Core temperature and sweating onset in humans acclimated to heat given at a fixed daily time

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Shido, Osamu, Naotoshi Sugimoto, Minoru Tanabe, and Sotaro Sakurada. Core temperature and sweating onset in humans acclimated to heat given at a fixed daily time. Am. J. Physiol. 276 (Regulatory Integrative Comp. Physiol. 45): R1095–R1101, 1999.—The thermoregulatory functions of rats acclimated to heat given daily at a fixed time are altered, especially during the period in which they were previously exposed to heat. In this study, we investigated the existence of similar phenomena in humans. Volunteers were exposed to an ambient temperature (Tₐ) of 46°C and a relative humidity of 20% for 4 h (1400–1800) for 9–10 consecutive days. In the first experiment, the rectal temperatures (Tᵣₑₐₙ) of six subjects were measured 24 h at a Tₐ of 27°C with and without heat acclimation. Heat acclimation significantly lowered Tᵣₑₐₙ only between 1400 and 1800. In the second experiment, six subjects rested in a chair at a Tₐ of 28°C and a relative humidity of 40% with both legs immersed in warm water (42°C) for 30 min. The Tᵣₑₐₙ and sweating rates at the forearm and chest were measured. Measurements were in the morning (0900–1100) and afternoon (1500–1700) on the same day before and after heat acclimation. Heat acclimation shortened the sweating latency and decreased the threshold Tᵣₑₐₙ for sweating. However, these changes were significant only in the afternoon. The results suggest that repeated heat exposure in humans, limited to a fixed time daily, alters the core temperature level and thermoregulatory function, especially during the period in which the subjects had previously been exposed to heat.

human heat acclimation; sweating latency; heat stress; circadian rhythm; metabolic hormone

ACCLIMINATION TO HEAT has repeatedly been shown to modify various thermoregulatory functions in various species of animals, such as the core temperature level (1, 6, 20), thermoeffector thresholds (10), and magnitude of thermoregulatory responses to a given level of central drive (10). Our previous studies (19, 21, 22) showed that when rats were subjected to daily heat exposure, limited to ~5 h at a fixed time of the day for more than 5 consecutive days, and then transferred to a constant ambient temperature (Tₐ) of 24°C, the pattern of day-night variations in core temperature was altered so that the core temperature of the rats fell in the same period during which they had been previously exposed to heat. This drop in core temperature lasted for several days after the termination of the daily heat exposure (19). In association with the drop in core temperature, threshold temperatures for nonevaporative heat loss and cold-induced thermogenesis shifted to low levels (23). Because the core temperature may be preferably controlled within a range between thresholds for heat loss and heat production, the drop in core temperature is considered to be a result of regulation by the thermoregulatory system. Based on these observations, we postulated that a time memory for heat exposure is established in heat-acclimated rats, and that their thermoregulatory function changes to maintain their core temperature at a low level, especially during the period in which they were previously exposed to heat.

Physiological changes in thermoregulation due to heat acclimation are also well documented in humans, especially with respect to the sweating response (5–7, 17, 18, 30). Acclimation to heat given for a few hours per day for more than 9 days has been shown to lower the body core temperature (1, 6, 7, 20), shorten the time lag of sweat onset (13, 14), shift threshold temperatures for sweating (7, 17, 20) and skin vasodilation (1) to low levels, and increase the sweating capacity (5, 14, 30). However, it is not known whether such thermoregulatory changes in heat-acclimated humans are consistent throughout a day or, as observed in the heat-acclimated rats, seen clearly during the period in which subjects were exposed to heat. The present study was therefore conducted to determine how the core temperature level and sweating response to a mild heat load differs between the period in which subjects had previously been exposed to heat and during other parts of the day in humans acclimated to heat given daily at a fixed time. It was hypothesized that even in the human autonomic thermoregulatory system, a memory for timed heat exposure could be formed and, according to the time memory, the core temperature and sweating threshold might be lowered during the period in which subjects had been previously exposed to heat.

METHODS

Subjects. Healthy male and female subjects volunteered for the present experiments, giving informed consent. Before participating, they were well familiarized with the test procedures and with the equipment to be affixed to their bodies, but were not informed of the purpose of the study.

From 2 wk before the start of measurements, the subjects’ clock times for waking up and going to bed were controlled (0700–0730 and 2300–2330, respectively), with the exact times left up to the subjects. During this period, we instructed all subjects not to participate in any strenuous exercise or to take a J Japanese-style bath (hot water immersion) to avoid a rise in core temperature. In addition, the subjects were not allowed to stay in a Tₐ above 28°C, except during the heat exposure period.

For more than 3 days before the first measurements, we recorded all food that the subjects consumed and the time of

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ingestion. The subjects had the same food at the same time for the same period before and during the second measurements. When the subjects consumed beverages containing calories, the amount and the time of the ingestion were also checked and repeated as with the food control. In addition, the subjects were not allowed to have any food or beverage that contained caffeine, alcohol, or a large amount of capsaicin for at least 1 wk before the measurements. Water intake (without calories) was not limited throughout the experiment. The food control was performed to avoid the possible influence of calorie intake on heat-loss responses (8).

Heat exposure. The subjects wore T-shirts and shorts and stayed in a temperature-controlled chamber (TBR-2HAG2A; Tabai Espec, Osaka, J. Japan) in which the air temperature was set at 46.0 ± 0.2°C and the relative humidity was 20 ± 3% for 4 h (1400–1800). The subjects could change their posture (e.g., lie down or sit in a chair), read books, or study in the chamber. The heat exposure was repeated for 9–10 consecutive days in experiment 1 and 10 consecutive days in experiment 2. Body mass was measured before and after the heat exposure a few times in each subject. The amount of decrease in body mass during the 4-h heat exposure was ~1.1–1.5 kg.

Experiment 1. Four male subjects and two female subjects (mean age, height, and body mass were 25 yr, 167 cm, and 64.9 kg, respectively) volunteered for this experiment. On the day of the measurements, the subjects were instructed to arrive at the laboratory by 0800 after having had breakfast. Each subject wore a T-shirt and shorts and entered a climatic chamber. The heat exposure was repeated for 9–10 consecutive days in experiment 1 and 10 consecutive days in experiment 2. Body mass was measured before and after the heat exposure a few times in each subject. The amount of decrease in body mass during the 4-h heat exposure was ~1.1–1.5 kg.

Experiment 2. Six male subjects (mean age, height, and body mass were 24 yr, 174 cm, and 70.9 kg, respectively) were used. All measurements were conducted in a climatic chamber (TBL-6-S; Tabai Espec) at a Ta of 28.0 ± 0.5°C and a relative humidity of 40 ± 5%.

The measurements in the CN, consisting of the morning and afternoon tests, were made on the day before commencement of the 10-day heat-exposure schedule. The subjects were instructed to arrive at the laboratory by 0800 without breakfast. They wore only shorts. All devices for measurement were fitted on the subjects. The subjects rested in the chamber, seated in a chair in an upright position, for 60 min (0900–1000). A blood sample (~20 ml) was then taken from the vein at the right cubital region. About 30 min after the blood sampling, the subjects immersed their legs in a water bath (LTP-112; Tabai Espec) in which the water temperature was controlled (42.0 ± 0.1°C) for 30 min (1030–1100). The same measurements were repeated in the afternoon, i.e., the subjects rested between 1500 and 1600, a blood sample was taken at 1600, and the subjects' legs were immersed in warm water between 1630 and 1700. Between the two tests, the subjects were not allowed to have any food or beverage that contained calories and all sensors except the skin thermistor probes were kept in place.

Sweating rates (m:\text{\text{sw}}) at the forearm and chest were measured by the ventilation method (26) with 0.79-cm² capsules (1.0-cm diameter). The capsules were fixed to the skin of the left forearm and to the center of the sternum with adhesive tape and highly ventilated with dry air taken from a pressurized air tank at a constant flow of 500 ml/min. The outlet air was then sent into a capacitance hygrometer (HMP23UT; Vaisala, Helsinki, Finland) and water content was computed from the flow and relative humidity of the air. The left forearm skin blood flow was measured by a laser-Doppler flowmeter (ALF-2100; Advance, Tokyo, J. Japan). The probe was fixed to the skin surface and held in place with adhesive tape. After the measurements were taken, the subjects immersed their forearms in warm water (42°C) for 30 min to obtain maximal forearm skin blood flow (28).

T_{re} was measured with a thermistor probe introduced 15 cm into the rectum. Skin temperatures were recorded at seven body sites (forehead, trunk, forearm, hand, thigh, calf, and foot) by skin thermistors held in place with surgical tape. The heart rate (HR) was estimated by the count of R wave in 1 min on an electrocardiogram. All data were sampled every minute and stored with a portable four-channel memory (VM4-064; VINE, Tokyo, J. Japan) for more than 24 h (at least between 1000 and 1000 of the next day).

The measurements were made twice in each subject. For three subjects, we performed measurements in the control condition (CN) before acclimation to heat, whereas for the other three subjects, we made the measurements in the CN 3–4 wk after completing the 9- to 10-day heat-exposure schedule. Measurements in the heat-acclimated condition (HA) began on the next day after the heat-exposure schedule. For the female subjects, we performed the measurements at the same stage of the menstrual cycle.

In an additional study, we measured T_{re} temperatures of the forehead and chest and HR before and during the 4-h heat exposure (1300–1800) on the 4th day of the heat-exposure schedule using the portable memory.

Experiment 2. Six male subjects (mean age, height, and body mass were 24 yr, 174 cm, and 70.9 kg, respectively) were used. All measurements were conducted in a climatic chamber (TBL-6-S; Tabai Espec) at a Ta of 28.0 ± 0.5°C and a relative humidity of 40 ± 5%.

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T_{re} was measured with a thermistor probe introduced 15 cm into the rectum. Skin temperatures were recorded at seven body sites (forehead, trunk, forearm, hand, thigh, calf, and foot) by skin thermistors held in place with surgical tape. The heart rate (HR) was estimated to be within ±0.05°C. Systolic and diastolic arterial blood pressures (BP) and HR at the right arm were monitored every 5 min with an electric sphygmomanometer (MPV-7101; Nihon Kohden, Tokyo, J. Japan).

All data except BP and HR were sampled every 30 s via a computer-based logging system (PC9801VX; NEC, Tokyo, J. Japan). In addition, m:\text{\text{sw}} values were continuously recorded with a potentiometer (INR-6041; TOA Electronic, Tokyo, J. Japan) to determine the time of sweating onset. Mean skin (T_{sk}) and mean body (T_{b}) temperatures were then computed as follows

\[
T_{sk} = 0.07T_1 + 0.35T_2 + 0.14T_3 + 0.05T_4 + 0.19T_5 + 0.13T_6 + 0.07T_7
\]

\[
T_b = 0.7T_{re} + 0.3T_{sk}
\]

where T_{1–7} are the temperatures of the forehead, trunk, arm, hand, thigh, calf, and foot, respectively (9).

Blood was collected into three different types of tubes, one containing NaF for the glucose assay, one containing EDTA-2K for the arginine vasopressin (AVP) and catecholamine assays, and the other empty. After the hematocrit (Hct) was measured, the tubes were centrifuged at 4°C and 1,500 rpm. Plasma and serum samples were frozen and stored at below −20°C until assays. All of the assays except Hct were performed by Sumitomo Metal Industry Bioscience (Tokyo). Briefly, plasma osmolality (P_{osmol}) was determined by freezing point depression and the plasma levels of Na, K, Cl, and glucose (G) were measured by an ion-selective electrode
method. Total protein (TP) and albumin (Alb) concentrations were measured by the Biuret and bromcresol green methods, respectively, and triglyceride (TG) and nonesterified fatty acid (NEFA) were measured by the enzyme method. Plasma concentrations of 3,3',5-triiodo-L-thyronine (T₃), free T₃ (fT₃), thyroxine (T₄), free T₄ (fT₄), AVP, and aldosterone (Ald) were determined by radioimmunoassay, and those of epinephrine (Epi) and norepinephrine (NE) were analyzed by HPLC.

The measurements in the HA were performed on the day after the 10-day heat-exposure schedule. The procedure was repeated exactly the same way as in the CN. The places where the sweating capsules were attached were carefully marked after the control measurements, so that the sweating rates were always measured at the same skin areas.

Data analysis and statistics. In experiment 2, resting levels of thermoregulatory and cardiovascular parameters were obtained as means for 10 min before the 30-min leg water immersion. The forearm skin blood flow is expressed as the percentage of maximal output of the flowmeter (%Qsk) measured when the arm was immersed in water at 42°C (28). The results are presented as means ± SE. The effects of heat acclimation and time of day on all parameters measured were evaluated by two-way ANOVA. Significant changes in thermoregulatory and cardiovascular parameters during leg water immersion were also assessed by two-way ANOVA. P < 0.05 was considered to be significant.

RESULTS

Experiment 1. Figure 1 shows changes in Tₑₑ and HR before and after the 4-h heat exposure. The heat exposure significantly increased the Tₑₑ (−0.75°C) and the forehead and chest skin temperatures (data not shown). HR tended to increase during the heat exposure, but because of the movement of the subjects in the climatic chamber, the changes were not significant.

There were clear nycthemeral variations in Tₑₑ, skin temperatures, and HR in all subjects. Figure 2 shows

![Fig. 1. Changes in rectal temperature (Tₑₑ; top) and heart rate (HR; bottom) before and during 4-h heat exposure (1400–1800). Data are means of 10-min periods ± SE (vertical bars). Dotted lines on abscissae indicate period of heat exposure. bpm, Beats/min.](http://www.ajpregu.physiology.org/)

![Fig. 2. Changes in Tₑₑ (top), forehead skin temperature (Tf; middle), and HR (bottom) during a 24-h period (1000–1000 of next day) in control (○) and heat-acclimated (●) conditions. Data are means of 2-h periods ± SE (vertical bars). Filled bars on abscissae indicate period of lights off. Spaces between dotted lines indicate period of previous heat exposure (1400–1800). * Significant difference between the 2 conditions.](http://www.ajpregu.physiology.org/)

the mean changes in Tₑₑ, forehead skin temperature, and HR over 24 h in the CN and HA conditions. In the CN, as is well known, Tₑₑ increased in the morning and early afternoon and reached the maximal level around the evening. It then fell gradually and started to rise just before subjects woke up. Similar changes were seen in the forehead skin temperature and HR. In the HA, however, Tₑₑ did not increase in the afternoon. The Tₑₑ levels in the HA were significantly lower than those in the CN, especially between 1400 and 1800, when the subjects had been previously exposed to heat. The Tₑₑ levels during sleep and in the morning were the same between the CN and HA conditions. The skin temperature and HR did not differ significantly between the two conditions, regardless of the time of the day.

Experiment 2. Table 1 shows the resting levels of the thermoregulatory and cardiovascular parameters before the start of the leg water immersion in the morning and afternoon tests in the CN and HA. In the afternoon test, Tₑₑ fell significantly after heat acclimation, whereas in the morning test, Tₑₑ fell in three subjects, increased in two subjects, and did not change in one subject, resulting in an insignificant influence of heat acclimation on the Tₑₑ level. Tₑₑ appeared to be lowered by heat acclimation, but again, the change was more evident in the afternoon test than in the morning test. There were
no significant differences in the resting $T_{sk}$ and $\%Q_{sk}$ between the periods of the day and acclimation conditions. Similarly, the BP and HR values were not significantly affected by heat acclimation or the time of day.

The 30-min leg water immersion increased the $T_{re}$, $T_b$, $T_{sk}$, $%Q_{sk}$, and $m_{sw}$ but did not affect BP or HR in all subjects tested. The onset of thermal sweating was determined by a prompt increase of $m_{sw}$ in each measurement (Fig. 3). The time at the onset of thermal sweating after the commencement of the leg water immersion and the $T_{re}$ corresponding to the sweating onset were defined as a sweating latency and threshold $T_{re}$ for sweating, respectively.

Figure 4 shows the sweating latency and threshold $T_{re}$ for forearm sweating in each subject in control (CN) and heat-acclimated (HA) conditions. In the morning test, heat acclimation elongated the sweating latency in two subjects, shortened it in three subjects, and had no effect in one subject. In the afternoon test, the sweating latency was shortened after heat acclimation in all subjects. Thus a significant effect on the latency was seen only in the afternoon. The sweating latency in the afternoon was significantly longer than that in the morning in the CN. However no such difference was noted in the HA.

In the morning test, the threshold $T_{re}$ for sweating shifted to higher levels in two subjects and to lower levels in three subjects after heat acclimation. In the afternoon test, the threshold was lowered by heat acclimation in five of the six subjects. Again, the change was significant only in the afternoon test. Similar effects of heat acclimation on the sweating response were seen in the chest (Fig. 5).

Because $%Q_{sk}$ varied greatly and the onset of the rise in $%Q_{sk}$ was not reliably identified, we could not assess the effects of heat acclimation on the onset for skin vasomotor response.

Tables 2 and 3 summarize the data for Hct, $P_{osmol}$, plasma levels of electrolytes, energy substrates, and various hormones in the CN and HA in the morning and afternoon tests. Although Hct in the HA appeared to be lower than that in the CN in the afternoon test, heat acclimation and the time of day had no significant influence on Hct. $P_{osmol}$ in the afternoon test was significantly lower than that in the morning test regardless of the acclimation conditions. However, heat acclimation did not affect $P_{osmol}$ in the two periods of the day. The plasma G level slightly decreased and the plasma TG and NEFA levels significantly increased in the
core temperature level in humans (1, 6, 20), e.g., heat exposure for 0.5–3 h daily for more than 12 days decreased oral temperature by 0.19°C (6), and repeated exercise under heat for 1.5–2.0 h daily for 9 days reduced Tre by ~0.3°C (1). The present results are comparable with these observations, i.e., after subjects were acclimated to heat given for 4 h daily at a fixed time for 9–10 consecutive days, their Tre was significantly decreased (0.19–0.24°C) (Fig. 2 and Table 1). However, the fall in core temperature was not consistent throughout the day. The Tre in the HA were kept at lower levels than those in the non-acclimated condition in the afternoon, especially around the period when the subjects had been previously exposed to heat, whereas heat acclimation had no significant influence on the Tre levels in the morning, at night, or during sleep (Fig. 2). Thus, similar to the observations of rats (19, 21–23, 25), our data suggest that even in humans, daily heat exposure for several hours limited to a fixed time alters the pattern of nycthemeral variations in core temperature so that the core temperature falls during the period in which the subjects were previously exposed to heat.

It is also well documented that in humans with short-term heat acclimation, the latency for thermal sweating is shortened (6, 14) and threshold core temperatures for heat-loss responses are shifted to low levels (1, 17, 30). Such thermoregulatory changes, especially the downward shift of the thermoeffector threshold, may contribute to keeping core temperature at a low level (23). The present study confirmed these observations (i.e., in the HA, the sweating latency was significantly shortened and the threshold Tre for thermal sweating was significantly lowered). However, the changes in evaporative heat-loss response were evident only when measurements were made in the afternoon (between 1500 and 1700), the period when the subjects had previously been exposed to heat. Again, in agreement with results in the heat-acclimated rats (23, 25), heat exposure of several hours limited to a fixed time daily may modify thermoregulatory function, especially during the period corresponding to the previous heat-exposure time in humans.

There are several possible explanations for the early onset of thermal sweating in the HA. The onset of local thermal sweating is known to be regulated by both

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**Table 2. Hematocrit, plasma osmolality, and plasma levels of electrolytes and energy substrates**

<table>
<thead>
<tr>
<th></th>
<th>Morning</th>
<th>HA</th>
<th>Afternoon</th>
<th>HA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hct, %</td>
<td>44.5 ± 1.6</td>
<td>44.0 ± 1.1</td>
<td>45.1 ± 1.5</td>
<td>43.7 ± 1.0</td>
</tr>
<tr>
<td>P_{osmol}, mosmol/kg</td>
<td>286 ± 1</td>
<td>285 ± 1</td>
<td>283 ± 1*</td>
<td>282 ± 1*</td>
</tr>
<tr>
<td>Na, meq/l</td>
<td>140 ± 1</td>
<td>140 ± 1</td>
<td>140 ± 1</td>
<td>138 ± 1</td>
</tr>
<tr>
<td>K, meq/l</td>
<td>4.2 ± 0.1</td>
<td>4.1 ± 0.1</td>
<td>4.1 ± 0.1</td>
<td>4.1 ± 0.1</td>
</tr>
<tr>
<td>Cl, meq/l</td>
<td>106 ± 2</td>
<td>103 ± 1</td>
<td>104 ± 1</td>
<td>102 ± 2</td>
</tr>
<tr>
<td>TP, g/dl</td>
<td>7.3 ± 0.1</td>
<td>7.4 ± 0.1</td>
<td>7.5 ± 0.1</td>
<td>7.4 ± 0.1</td>
</tr>
<tr>
<td>Alb, g/dl</td>
<td>4.4 ± 0.1</td>
<td>4.5 ± 0.1</td>
<td>4.5 ± 0.1</td>
<td>4.5 ± 0.1</td>
</tr>
<tr>
<td>G, mg/dl</td>
<td>92 ± 2</td>
<td>91 ± 2</td>
<td>88 ± 1</td>
<td>88 ± 1</td>
</tr>
<tr>
<td>TG, mg/dl</td>
<td>68.2 ± 11.5</td>
<td>66.5 ± 6.5</td>
<td>76.8 ± 11.2*</td>
<td>74.2 ± 11.2*</td>
</tr>
<tr>
<td>NEFA, meq/l</td>
<td>0.34 ± 0.07</td>
<td>0.35 ± 0.07</td>
<td>0.69 ± 0.11*</td>
<td>0.58 ± 0.08*</td>
</tr>
</tbody>
</table>

Values are means ± SE. Hct, hematocrit; P_{osmol}, plasma osmolality; TP, protein; Alb, albumin; G, glucose; TG, triglyceride; NEFA, nonesterified fatty acid. *Significantly different from corresponding value in morning test.

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**Table 3. Plasma hormone levels**

<table>
<thead>
<tr>
<th></th>
<th>Morning</th>
<th>HA</th>
<th>Afternoon</th>
<th>HA</th>
</tr>
</thead>
<tbody>
<tr>
<td>T3, ng/dl</td>
<td>145 ± 10</td>
<td>150 ± 12</td>
<td>131 ± 10</td>
<td>136 ± 9</td>
</tr>
<tr>
<td>FT3, pg/ml</td>
<td>3.4 ± 0.1</td>
<td>3.4 ± 0.2</td>
<td>3.4 ± 0.2</td>
<td>3.4 ± 0.1</td>
</tr>
<tr>
<td>T4, µg/dl</td>
<td>7.6 ± 0.3</td>
<td>8.0 ± 0.4</td>
<td>8.3 ± 0.4</td>
<td>7.9 ± 0.3</td>
</tr>
<tr>
<td>FT4, ng/dl</td>
<td>1.4 ± 0.1</td>
<td>1.3 ± 0.1</td>
<td>1.4 ± 0.1</td>
<td>1.4 ± 0.1</td>
</tr>
<tr>
<td>Epi, pg/ml</td>
<td>27 ± 4</td>
<td>21 ± 5</td>
<td>23 ± 3</td>
<td>26 ± 3</td>
</tr>
<tr>
<td>NE, pg/ml</td>
<td>192 ± 40</td>
<td>188 ± 41</td>
<td>180 ± 32</td>
<td>178 ± 34</td>
</tr>
<tr>
<td>AVP, pg/ml</td>
<td>2.3 ± 0.3</td>
<td>2.2 ± 0.3</td>
<td>2.3 ± 0.6</td>
<td>2.2 ± 0.4</td>
</tr>
<tr>
<td>Ald, pg/ml</td>
<td>34 ± 18</td>
<td>38 ± 25</td>
<td>31 ± 19</td>
<td>31 ± 18</td>
</tr>
</tbody>
</table>

Values are means ± SE. T3, 3,3’5-triiodo-L-thyronine; FT3, free T3; T4, thyroxine; FT4, free T4; Epi, epinephrine; NE, norepinephrine; AVP, arginine vasopressin; Ald, aldosterone.

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afternoon test compared with the morning test in both the CN and HA, which may have been attributable to fasting. Again, heat acclimation had no significant effect on the levels of plasma energy substrates. The plasma levels of Na, K, Cl, TP, and Alb, and the concentrations of all plasma hormones tested, were not affected by heat acclimation in either the morning or the afternoon test.

**DISCUSSION**

Heat acclimation brought about by daily heat exposure for a short duration has been shown to lower the
central thermoregulatory drive and local skin temperature (14, 15). The central thermoregulatory drive to the effectors is a function of the total thermal input from a whole body and an excitability or a sensitivity of the central nervous system to the thermal stimuli. In addition, the responsiveness of sweat glands is likely to be modified with heat acclimation (3, 5). Thus the shortening of the sweating latency occurring with heat acclimation could be attributed to an increase in thermal input, an enhanced sensitivity of the central nervous system, a rise in the skin temperature of the area where \( m_{\text{sw}} \) was measured, and/or an increased sweat gland sensitivity to a given level of central drive. In the present observations, \( T_b \), an indicator of total thermal input, did not increase at the onset of thermal sweating, but rather decreased after heat acclimation. Local skin temperatures at the forearm and chest were not higher in the HA than in the CN. It therefore appears that the sensitivity of the central thermoregulatory system to thermal stimuli or a responsiveness of local sweat glands increases in the HA, especially during the period in which the subjects were exposed to heat.

In addition to thermoregulatory factors, the onset of the evaporative heat-loss response is shown to be strongly influenced by the body hydration state and \( P_{\text{osmol}} \) (4, 11, 12, 16, 27). For instance, hyperosmolality delayed a sweating onset (4) and enlarged the amount of rise in core temperature required to elicit thermal sweating (27). In the present study, the plasma \( P_{\text{osmol}} \) was significantly lower in the afternoon test than in the morning test. However, heat acclimation had no significant effect on the \( P_{\text{osmol}} \). The Hct in the afternoon test appeared to be lower in the HA than in the CN, suggesting an occurrence of plasma expansion due to heat acclimation. Again, the change in Hct was not significant. Thus changes in plasma volume or \( P_{\text{osmol}} \) may not have a close association with the shortening of sweating latency and the downward shift of sweating threshold observed in the afternoon test in the HA.

Short-term heat acclimation has been shown to increase the magnitude or capacity of evaporative and nonevaporative heat-loss responses in humans (1, 13, 30). In rats acclimated to heat given daily at a fixed time, their thermoregulatory responses to acute heat load were facilitated only during the period in which the animals had been exposed to heat (25). Thus, in the present study, we expected that the magnitude of sweating and blood flow responses to heat would differ between the periods of the day in the HA. The heat stress (leg water immersion) applied in this study was rather mild because we had to repeat the same heat stress within several hours. Therefore, the amount of increase in \( m_{\text{sw}} \) and skin blood flow was not sufficient to assess their responsiveness and capacities. Whether the magnitude of thermoregulatory responses to heat load is enhanced during the previous heat-exposure time remains to be investigated.

Acclimation to heat has also been shown to bring about several adjustments in endocrine functions related to thermogenic activity and body water balance, e.g., heat acclimation altered the patterns of nycthemeral variations in plasma thyroid hormones (24) and Ald (2) concentrations in animals. We have reported that in rats acclimated to heat given daily at a fixed time, plasma levels of \( T_3 \), FT3, T4, and FT4, which are known to decline in heat-acclimated subjects, paradoxically increased during the period corresponding to the previous heat-exposure time (24). However, in the present heat-acclimated human subjects, we observed no significant changes in plasma thyroid hormone levels during the specific period. The plasma concentrations of energy substrates, catecholamines, AVP, and Ald were also not modified by heat acclimation.

In experiment 1, the control measurements were performed 3–4 wk after the termination of the heat exposure in three subjects. Short-term heat acclimation is known to be transient and to disappear gradually. Although there are great variations in the rate of decay for heat acclimation among the reports (29), the major physiological changes associated with heat acclimation could be lost within 2–3 wk. Thus the residual effect of heat acclimation in those measurements might be minimal. Indeed, we could observe clear differences in the pattern of nycthemeral variations of \( T_{\text{re}} \) between the CN and HA.

In summary, after humans were subjected to daily heat exposure given for 4 h at a fixed time for more than 9 consecutive days, the pattern of day-night variations in core temperature was modified so that the core temperature fell during the period when the subjects had been previously exposed to heat. Additionally, the latency for thermal sweating was shortened and the threshold temperature for sweating was lowered by heat acclimation, especially during the period corresponding to the previous heat exposure. As in heat-acclimated rats, a time memory for heat exposure could be formed in the human thermoregulatory system and, according to the memory, autonomic thermoregulatory function could change during the period of previous heat exposure time without actual temperature stimuli.

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