Temperature regulation during acute heat loads in rats after short-term heat exposure

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Shido, Osamu, Sohtaro Sakurada, Minoru Tanabe, and Tetsuo Nagasaka. Temperature regulation during acute heat loads in rats after short-term heat exposure. J. Appl. Physiol. 71(6): 2107-2113, 1991.—Eleven rats were kept at an ambient temperature of 33.5°C (HC) for 4-5 consecutive days, 9 additional rats were subjected to 33.5°C for ~5 h daily (HI) for the same period, and 12 controls (Cn) were kept at 24°C. After the exposure, the rats were placed in a direct calorimeter, where the wall temperature was set at 24°C, and subjected to direct internal heating (6.2 W·kg⁻¹·30 min) through an intraperitoneal electric heater. After the first heat load and when thermal equilibrium had been attained again, the rats were subjected to indirect external warming by raising the jacket water temperature. The onsets of the rises in tail skin temperature and evaporation of the urine were used as indicators of thermoregulatory responses to heat loads. During heat loads, the latent times for the onset of the rises in tail skin temperature and evaporation were significantly longer, and Tₚₑₑ and Tₑₑ, at the start of increases in heat losses tended to be higher, in the HC than in the Cn. M significantly decreased in all groups, but the magnitude and duration of reduction in M were significantly greater in the HC than in the Cn. There were no differences between the thermoregulatory responses to heat loads of the III and Cn. These results suggest that in HC the threshold core temperature for heat loss response and the upper critical temperature have already shifted to a higher level and that HC respond to heat stress more strongly with the reduction of M than Cn. Short-term intermittent heat exposure had little effect on the thermoregulatory mechanisms in rats.

Direct and indirect calorimetry; evaporative and nonevaporative heat loss; threshold core temperature; metabolic heat production

Acclimation to high environmental temperatures (28-43°C) affects the resting body core temperature (2, 6, 16, 19, 20), threshold core temperatures for evaporative (9, 10) and nonevaporative (10, 15, 17, 18) heat losses, the upper critical temperature (7), and body fluid distributions (8, 21) in mammals. Such physiological changes in association with heat acclimation have been shown to depend partly on the time that heat was loaded (10-12, 19). Horowitz (8) showed that the plasma volume of the heat-exposed rats was less than that of the controls at the 5th and 10th days of heat exposure and, after 28 days, the values were restored to the control level. Horowitz et al. (11, 12) reported that in rats the submaxillary glands showed an increased growth rate and activity for the first 5 days after the start of heat exposure. In freely moving rats, an upward shift of the resting core temperature was accomplished after 10 days of heat exposure but not within 5 days of heat exposure (19). It seems that in rats the thermoregulatory system greatly changes after 5-10 days of heat exposure.

In addition to the period of heat exposure, differences in temperature acclimation schedules affect thermoregulatory mechanisms in rodents. According to our previous observations in rats (15, 19), the mean hypothalamic temperature per day and threshold hypothalamic temperature for the tail skin vasodilation increased after exposure to constant heat for >10 days and decreased after acclimation to heat loaded intermittently for 5 h once a day for ≥10 consecutive days. However, it is not yet known how different methods of short-term (~5 days) (10) heat exposure affect the thermoregulatory mechanisms in rats. In the present study, therefore, we adopted two heat-acclimation regimens (17, 18), i.e., exposure to a high ambient temperature either all day or 5 h·day⁻¹ for 4-5 consecutive days, and investigated the differences of heat loss and metabolic responses to acute heat loads among rats subjected to those heat exposure schedules.

Methods

Animals and preparations. Male Wistar rats (Std: Wistar/ST), initially weighing 280-290 g, were used. They were individually housed in wire mesh cages and given laboratory rat chow and tap water ad libitum with a 12:12-h light-dark cycle (light on at 0600 h) at an ambient temperature (Tᵦ) of 24 ± 1°C. A 22-gauge stainless steel guide cannula was stereotaxically implanted into the median preoptic-anterior hypothalamus under pentobarbital sodium anesthesia (50 mg·kg⁻¹·ip). From 2 days after the surgery, the rats were loosely restrained in cylindrical wire cages individually for 5-6 h (5-6 times a week) to accustom them to the experimental situations. The procedure was repeated at least eight times. Then they were divided into three groups: the control (Cn) group was kept at Tᵦ of 24.0°C throughout the experiment, and heat-exposed groups were constantly subjected to Tᵦ of 33.5 ± 0.4°C (HC) or to the same Tᵦ for 5.2-5.3 h·day⁻¹ (III). In the III, the room temperature was quickly raised to 0900 h from 24.0 to 33.5°C in 40 50 min and maintained and then lowered to 24.0°C at 1400 h. The heat exposures lasted for 4-5 consecutive days (10).

Three days before the experiments, a laparotomy was performed under pentobarbital sodium anesthesia (50 mg·kg⁻¹·ip) with a 1.5-cm median incision for inserting an intraperitoneal electric heater into the peritoneal cavity.

ity. The special heater was made of constantan wire (0.2 mm diam, 2 m long) passed through a double-lumen polyethylene tube (1.0 mm OD, 0.4 mm ID; DP4, Nataume, Tokyo) that was loosely coiled (30 cm long). A copper-constantan thermocouple (0.1 mm diam) was inserted into the hypothalamic cannula and fixed to the cannula with dental cement. Lead wires were passed subcutaneously and exteriorized at the nape.

At the termination of experiments, the rats were killed with a large dose of anesthetics and the location of the hypothalamic cannula was checked.

Measurements. All tests were performed in the light phase of a day. The core temperature of rats has been shown to fall profoundly (below 36.5°C) for several hours immediately after the end of heat exposure, which might have been attributed to uncontrolled heat dissipation, and to increase thereafter (19). To avoid such an unstable period in body core temperature, we performed measurements ~15 h after the termination of the heat exposure schedule. Each rat was loosely restrained in a cylindrical wire cage with dimensions that were the same as those of the cage used for habituation to restraint. A thermocouple (0.2 mm diam) covered with a polyethylene tube was inserted 6 cm into the colon and fixed with vinyl tape wrapped around the tail. Another thermocouple was placed longitudinally along the tail at the middle portion of the ventral side and was held in place by one turn of the tape, which covered the thermocouple junction. Thereafter the rat in the cage was transferred to a gradient layer-type direct calorimeter (13). An acrylic pan filled with vegetable oil was set under the cage to collect the wastes in order to prevent evaporation. The calorimeter wall temperature (Twall) was initially set at 24°C with an accuracy of ±0.02°C.

Fresh dry air was introduced into the calorimeter chamber at a constant rate of 1.8 l·min⁻¹. Nonevaporative heat loss (R + C + K) was measured by the calorimeter, and evaporative heat loss (E) was calculated from the flow rate and humidity of the air monitored with a humidity sensor (MHI-11, Vaisala, Helsinki). A fraction of air (100 ml·min⁻¹) withdrawn from the calorimeter chamber was sent into a Zirconia O₂ analyzer (LC-700B, Toyara, Tokyo), and O₂ consumption was calculated from measurements of O₂ content. Metabolic heat production (M) was calculated from O₂ consumption and the caloric equivalent for O₂ (4.823 kcal/l). Hypothalamic (T₁h), colonic (Tcoli), and tail skin (Tsk) temperatures were measured through the thermocouples. Twall and temperatures of jacket water and outlet air from the calorimeter were also detected by the other thermocouples. All parameters were sampled every 5 s through an analog-to-digital converter (ADC-121B, Kanazawa Control Kiki, Kanazawa) connected to a personal computer (PC-9801VX, NEC, Tokyo).

Heat loads. After a 60-min control period, the rats were subjected to an acute heat load of 6.2 W·kg⁻¹ (1.68–1.99 W per rat) for 30 min through the heater chronically implanted in the peritoneal cavity (direct internal heating). The amount of heat load was based on our preliminary observation, showing that heat load below 1.5 W per rat sometimes failed to induce a further rise in Tsk after the tail skin vasodilation had occurred (in this case, we cannot assess (R + C + K) responses to the rise in body core temperature). At least 60 min after an internal heating and when thermoregulatory parameters had been restored to initial levels, an indirect external warming was performed by raising the jacket water temperature surrounding the calorimeter chamber. Water temperature was continuously raised for 90 min at a constant rate of 0.16°C·min⁻¹. The details of (R + C + K) estimation during external warming have been described elsewhere (17).

Data analysis and statistics. The point of a sharp bend in Tsk curve was determined by a person who did not know the purpose of the present study. The latent time and core temperatures at the onset for the tail skin vasodilation were estimated from the point (17), because a very good association between tail skin blood flow and temperature over the ventral vascular bundle has been observed during an increase in tail blood flow of rats (14). Similarly, the putative threshold Tco1 and Tco for the onset of E response were determined by a point, signaling a distinct rise in E (Fig. 1).

All values of parameters were obtained as averages of 1 min, and initial resting values were estimated as the means of 10-min data before heat loading at Tco of 24°C. Tco values of one rat in the HC were discarded from the results because the colonic thermocouple had been dislocated. Total thermal conductance was calculated as (R + C + K)/(Tco - Twall). Heat storages were obtained as M - (R + C + K) - E during external warming and as M - (R + C + K) - E + HL during internal heating, where HL was the amount of heat delivered by the intraperitoneal heater. HL was known by electric current and voltage with a digital ammeter and a voltmeter. The linear regression lines showing the relationships between (R + C + K) or thermal conductance and Tco after the tail skin vasodilation had occurred were obtained by the method of least squares from the mean values. The results are presented as means ± SE, and statistical evaluations among mean values were assessed by one-way analysis of variance and paired Student’s t test or Scheffe’s multiple comparison test where appropriate. The statistical significance of the difference between regression coefficients was determined by F test. P < 0.05 was considered significant.

RESULTS

The mean body weights just before the heat loads were 310 ± 4, 304 ± 6, and 318 ± 2 g in the Cn, HC, and III,
TABLE 1. Values at the onset of tail skin vasodilation during internal heat load

\[
\begin{array}{cccc}
\text{n} & \text{T_{onset}, min} & \text{T_{by}, \degree C} & \text{T_{co}, \degree C} & \text{T_{sk}, \degree C} \\
\text{Cn} & 11 & 9.33 & 38.35 & 38.63 & 24.81 \\
 & & \pm 1.08 & \pm 0.06 & \pm 0.14 & \pm 0.04 \\
\text{HC} & 10 & 15.32 & 38.52 & 38.57 & 24.71 \\
 & & \pm 1.88 & \pm 0.12 & \pm 0.13 & \pm 0.10 \\
\text{HI} & 9 & 9.48 & 38.29 & 38.72 & 24.54 \\
 & & \pm 0.85 & \pm 0.10 & \pm 0.12 & \pm 0.11 \\
\end{array}
\]

Values are means \( \pm \) SE; \( n \), no. of rats; \( T_{by}, T_{co}, \) and \( T_{sk} \), hypothalamic, colonic, and tail skin temperatures, respectively; Cn, control group kept at \( 24^\circ \text{C} \) ambient temperature; HC, heat-exposed group constantly subjected to \( T_{by} \) of \( 33.5 \pm 0.4^\circ \text{C} \); HI, heat-exposed group subjected to \( T_{by} \) of \( 33.5 \pm 0.4^\circ \text{C} \) for 5.2-5.3 h once a day. * Significantly different from Cn and HI values. \( p \), \( n = 9 \).

respectively. The initial values of all parameters measured at \( T_{by} \) of \( 24^\circ \text{C} \) immediately before internal heating and external warming did not differ among the three groups.

Thermoregulatory responses to direct internal heating. Table 1 summarizes mean latent time, \( T_{by}, T_{co}, \) and \( T_{sk} \) at the onset of the tail skin vasodilation observed during the internal heating in the Cn, HC, and HI. The latent time for the onset of the tail skin vasodilation in the HC was significantly increased by \( -6 \) min compared with the other two groups. In addition, threshold \( T_{by} \) and \( T_{co} \) for the cutaneous vasodilation in the HC tended to be higher by \( -0.2^\circ \text{C} \) than those of the Cn and HI. Because internal heating did not induce a clear rise in \( E \), we could not determine the thresholds of \( E \) response.

The internal heating for 30 min significantly elevated \( T_{by}, T_{co}, \) and \( T_{sk}, \) and thermal conductance and decreased \( M \) in all groups tested. Figure 2 shows mean changes in \( T_{by}, T_{co}, \) and \( T_{sk} \) before and during internal heating in the three groups. \( T_{by} \) promptly increased after the start of internal heating, and the rate of rise in \( T_{by} \) was attenuated when tail skin vasodilation occurred. There were no significant differences in \( T_{by} \) among the Cn, HC, and HI throughout the experiment. Similar changes were shown in \( T_{co} \). However, \( T_{co} \) increased more rapidly and greatly than \( T_{by} \) during internal heating, because the intraperitoneal heater may primarily have increased temperatures of the intraperitoneal viscera. \( T_{sk} \) of the HC was significantly lower than that of the other two groups for the last 18 min during the internal heating. The level to which \( T_{by} \) and \( T_{co} \) rose after the 30 min of internal heating did not significantly differ among the three groups.

Figure 3 shows mean changes in \( M \) and \( R + C + K \) in the Cn, HC, and HI before and during internal heating. \( M \) first significantly decreased 7, 3, and 6 min after initiation of internal heating in the Cn, HC, and HI, respectively. The significant reduction of \( M \) continued to the end of internal heating in the HC and HI but not for the last 5 min in the Cn. For the last 17 min during internal heating, \( M \) of the HI was significantly lower than that of the Cn. The minimum level of \( M \) during internal heating in the HC \((42.1 \pm 1.6 \text{ W} \cdot \text{m}^{-2}) \) was significantly less than that of the Cn \((47.1 \pm 0.7 \text{ W} \cdot \text{m}^{-2}) \) and the HI \((46.1 \pm 0.5 \text{ W} \cdot \text{m}^{-2}) \). \( R + C + K \) showed no significant changes for the first 6, 8, and 7 min in the Cn, HC, and HI, respectively. \( R + C + K \) increased after tail skin vasodilation. As expected from the results of \( T_{sk} \), the HC showed significantly lower \( R + C + K \) and thermal conductance from 16 min after the start of internal heating to the end of the experiment than the Cn and/or HI.

Thermoregulatory responses to indirect external warming. Table 2 summarizes mean latent times, \( T_{by}, T_{co}, \) and \( T_{sk} \) at the onset for tail skin vasodilation and the increase in \( E \) observed during the external warming in the Cn, HC, and HI. In the HC, the latent time for the onset of tail skin vasodilation was significantly longer than that of the Cn and HI. The threshold of \( T_{by}, T_{co}, \) and \( T_{sk} \) for cutaneous vasodilation tended to be higher in the HC than in the other two groups. In the HI, the latent time for the onset of cutaneous vasodilation varied greatly, ranging from 39 to 82 min after the start of external warming. The obvious increases in \( E \) were observed in all rats in the Cn and HI. However, 2 of 10 rats in the HC showed no increase of \( E \) during the 90-min external warming. When those examples were discarded from the results, mean values of latent time (81.5 \( \pm \) 2.1 min), \( T_{by} \) (38.28 \( \pm \) 0.06°C), \( T_{co} \) (38.26 \( \pm \) 0.05°C), and \( T_{sk} \) (34.16 \( \pm \) 0.77°C) at the onset of the \( E \) response were underestimated (even in this case, the start of the \( E \) response was obviously delayed in the HC). In those two rats, the minimum value of the onset time for the rise in \( E \) was 90 min, which corresponded to the value at the end of external warming experiments. If such minimum values were tentatively adopted, the latent time for the start of the rise in \( E \) of the HI would become significantly greater than that of the Cn and HI. Similarly, \( T_{co} \) at the onset of the
increase in $E$ would be significantly higher in the IIC than in the Cn.

The 90 min of external warming significantly increased $T_{by}$, $T_{co}$, $T_{sk}$, thermal conductance, and $E$ and significantly decreased $M$ and $(R + C + K)$ in all groups. Figure 4 shows mean changes in $T_{by}$, $T_{co}$, and $T_{sk}$ for the last 50 min during external warming in the Cn, HC, and HI. $T_{by}$ significantly increased from the initial levels 58, 56, and 56 min after the start of external warming in the Cn, HC, and HI, respectively. The rate of the rise in $T_{by}$ in the HC seemed to be slower than the rates of the other two groups especially from 70 min after the start of external warming. When the rises in $T_{by}$ for the last 20 min of external warming were assessed as a function of the time, the rate of HC (0.0247°C·min⁻¹) was significantly slower than that of Cn (0.0379°C·min⁻¹) and HI (0.0378°C·min⁻¹). Similar changes in $T_{co}$ were seen in the three groups. For a while, $T_{sk}$ gradually increased according to the rise in $T_{a}$, and then it increased sharply at a certain point where the tail skin vasodilation had occurred. The changes of $T_{sk}$ during external warming in the HI were similar to the changes in the Cn. In the HC, however, $T_{sk}$ was lower than those of the other groups from ~60 min after the start of external warming. At the end of the experiments, the degree that $T_{by}$ of the HC had risen (0.90 ± 0.06°C) was significantly smaller than that of the Cn (1.22 ± 0.10°C) and HI (1.25 ± 0.07°C), and the degrees that $T_{co}$ had increased differed significantly among the three groups (1.21 ± 0.11, 0.92 ± 0.07, and 1.28 ± 0.09°C in the Cn, HC, and HI, respectively).

Figure 5 shows mean changes in $M$ and $(R + C + K)$ for 40-90 min after the start of external warming in the Cn, HC, and HI. $M$ gradually and significantly decreased during external warming in all groups. The significant reductions of $M$ were observed between 24 and 78, 17 and 90, and 35 and 81 min after the start of external warming in the Cn, HC, and HI, respectively. Between 73 and 88 min after the start of external warming, $M$ in the HC was significantly lower than that of the Cn. The minimum level of $M$ during external warming in the HC (36.6 ± 1.4 W·m⁻²) was lower than that of the Cn (40.2 ± 0.8 W·m⁻², significant) and the HI (39.7 ± 0.5 W·m⁻²). $(R + C + K)$ progressively decreased during external warming in all groups according to the gradual reduction of temperature gradient between body core and environment. The rate of the decrease in $(R + C + K)$ was attenuated ~60-70 min after the start of external warming where tail skin vasodilation had occurred.

**DISCUSSION**

The present results clearly show that the thermoregulatory responses to acute heat loads in rats after exposure to a high $T_{a}$ for 4-5 days can vary according to differences in heat exposure regimens to which they were subjected. Short-term continuous heat exposure significantly prolonged the onsets of heat loss responses and produced more pronounced metabolic reduction during acute heat loads compared with the control rats. This is, however, not the case in the HI; i.e., intermittent heat exposure for 5 h·day⁻¹ for 4-5 consecutive days appeared to have virtually no effect on the thermoregulatory mechanisms in rats.

During external warming, the latent time for the onset of the rise in $E$ was significantly longer in the HC than in the Cn and HI (Table 2). Because the water temperature

**TABLE 2. Values at the onset of heat loss response during external body warming**

<table>
<thead>
<tr>
<th></th>
<th>Tail Skin Vasodilation</th>
<th>Evaporative Heat Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$n$</td>
<td>Time, min</td>
</tr>
<tr>
<td>Cn</td>
<td>11</td>
<td>66.56</td>
</tr>
<tr>
<td>HC</td>
<td>10</td>
<td>74.63†</td>
</tr>
<tr>
<td>HI</td>
<td>9</td>
<td>62.18</td>
</tr>
<tr>
<td>Cn</td>
<td>70.17</td>
<td>38.16</td>
</tr>
<tr>
<td>Cn</td>
<td>83.10†</td>
<td>38.34</td>
</tr>
<tr>
<td>HC</td>
<td>70.34</td>
<td>38.24</td>
</tr>
</tbody>
</table>

Values are means ± SE; $n$, no. of rats. * Significantly different from Cn; † significantly different from HI. ‡ $n = 9$. 

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surrounding the direct calorimeter was raised at a constant rate, the latent time was closely related to \(T_a\) to which the rats were subjected; e.g., \(T_{\text{wall}}\) at the start of the rise in \(E\) in the HC (37.02 ± 0.30°C) was significantly higher than that in the Cn (35.11 ± 0.45°C) and HI (35.13 ± 0.67°C). The results may therefore suggest the upward shift of the upper critical temperature in the HC. In addition, \(T_{by}\) and \(T_{es}\) at the onsets for heat loss responses tended to be higher than those of the Cn (Tables 1 and 2). The observations were consistent, whether heat loss responses were induced with direct internal heat load or indirect external body warming. Although statistically significant difference was shown only in \(T_{es}\) at the onset of evaporative heat loss response, the observation may suggest an upward shift of threshold core temperatures for heat loss responses in rats after short-term heat exposure for 4–5 days.

In our previous study, continuous heat exposure for 10 days raised mean \(T_{by}\) in an entire day in freely moving rats (19), and the same exposure for >15 days elevated both the resting level of body core temperature and threshold \(T_{by}\) for the onset of tail skin vasodilation (17). In the present study, the initial level of body core temperature of rats after short-term continuous heat exposure did not differ from that of the control rats. The results may be supported by the observation that 5 days of continuous heat exposure failed to affect mean \(T_{by}\) at the light and dark phases of a day in freely moving rats (19). It is therefore assumed that the upper critical temperature and possibly threshold core temperature for heat loss responses in rats shift to higher levels within a 5-day continuous heat exposure and the resting level of mean body core temperature subsequently increases in a further 5-day heat exposure.

During acute heat loads, \(M\) significantly decreased in all groups tested. However, the minimum level of \(M\) was significantly lower in the HC than in the Cn. In rodents, acclimation to a constant heat load for >2 days has been shown to suppress basal \(O_2\) consumption by decreasing plasma thyroid hormone level (1, 5), metabolic responses to thyroid hormone and norepinephrine (1, 4, 9), and respiratory activity of thermogenic organs such as the liver (3). Such metabolic changes after heat acclimation may partly be responsible for maintaining the lower \(M\) during heat loads in the HC. Especially during external warming, a significant decrease in \(M\) lasted longer in the HC than in the Cn. When \(T_c\) was increased further, \(M\) started to rise, which may have been induced by release of catecholamines into the circulation (5) or a \(Q_{by}\) effect associated with the rise in core temperature. These observations in metabolic changes in rats with short-term continuous heat exposure are consistent with those of rats subjected to long-term heat exposures (17, 18).

The different thermoregulatory responses to acute heat loads between the rats subjected to continuous and intermittent heat exposures might have been brought about by the duration of heat exposure. The total duration of exposure to constant heat for 4–5 days is comparable with that of intermittent heat exposure for 5 h · day⁻¹ for ~2 wk. Our previous study (18) showed that intermittent heat exposure for >15 days resulted in increases in \(T_{by}\) and \(T_{es}\) at the onset of \((R + C + K)\) response during indirect body warming, with results similar to the HC in the present study. However, the same heat exposure significantly lowered resting and mean \(T_{by}\) in an entire day and threshold \(T_{by}\) for the onset of heat loss response induced by internal heating (16, 18, 19). This was obviously not the case in the present IIC. Thus the different heat loss responses between the rats subjected to continuous and intermittent heat exposures may have been induced not by differences in the total duration of heat
exposure but by differences in the pattern of heat exposure schedule.

On the contrary, in metabolic responses to heat loads, there seem to be no marked differences between the HC and rats with long-term acclimation to heat loaded intermittently. For instance, during external warming, the duration during which the low \( M \) remained was longer and the minimum \( M \) was lower in those two groups than in their respective controls. Yahata and Kuroshima (22) suggested that, in cold-acclimated rats, cold tolerance and the magnitude of nonshivering thermogenesis were related to the total duration of cold exposure. In the case of heat acclimation, metabolic adaptation may also depend on the total length of heat exposure.

When the changes in \( (R + C + K) \) after the onset of tail skin vasodilation were plotted as a function of \( T_{by} \), the slope of the regression line showing the relationship between \( (R + C + K) \) or thermal conductance and \( T_{by} \) of the Cn was steeper than that of the HC during internal heating and tended to be steeper during external warming. These results suggest that the rats subjected to short-term continuous heat exposure showed an attenuated \( (R + C + K) \) response to the rise in \( T_{by} \). The observation seems to be paradoxical, because heat-exposed rats can keep their body temperature at a lower level in the heat than nonacclimated subjects (Fig. 4 of Ref. 17). However, simultaneously with the blunted heat loss response, \( M \) decreased more in the HC than in the Cn during acute heat loads. To assess the whole thermoregulatory response to heat stress, heat storage may have to be examined. Figure 6 shows the changes in heat storage during internal heating and external warming in the three groups. Heat storage of the HC tended to be lower than that of the Cn and HI throughout the 30 min of internal heating (insignificant) despite the same internal heat load. During external warming, there were no significant differences in heat storage among the three groups for 69 min after the start of heating. For the last 21 min, however, heat storage of the HC was significantly less than that of the Cn and/or HI. The results clearly indicate that heat was prevented from accumulating in the body more effectively in the HC than in the Cn and HI. In rats after short-term continuous heat exposure, especially during external warming, the reduction of metabolic heat production may have surpassed the attenuated heat loss responses to the rise in core temperature and compensated the delay of the onsets for \( E \) and \( (R + C + K) \) responses.

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