Effect of Thermal Acclimation on Blood Pressure and Stress-induced Elevation of Core Temperature in Spontaneously Hypertensive Rats and Wistar Rats

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Abstract: Male adult rats of the three strains, spontaneously hypertensive rats (SHR), Wistar-Kyoto rats (WKY) and Wistar rats (WIS) were used. Rats of each strain, at 1 month old, were randomly divided into three groups and chronically exposed to 3 different temperatures, 10℃, 30℃ and 22–24℃. Systolic blood pressure (BP) measured by the tail-cuff method and stress-induced elevation of core temperature were compared. In cold-acclimated WKY and WIS, BP was significantly higher than that in the heat-acclimated rat. In SHR, genetically hypertensive rats, there was no significant difference in BP between cold- and heat-acclimated young adult rats, and BP in heat-acclimated older adult rats was higher than that in cold-acclimated SHR. In normotensive rats, WKY and WIS, thermal acclimation affected systemic BP. In normotensive strains, WKY and WIS, cold-acclimation increased BP, whereas heat-acclimation decreased BP. Mean body weights in cold-acclimated rats were greater than those of heat-acclimated ones in all strains except for older adult WKY. For restraint stress, the conscious rat was paced in a loosely fitting small cage. Cold acclimated SHR (SHR-C) and WKY (WKY-C) were restrained at 10℃ and heat-acclimated SHR (SHR-H) and WKY (WKY-H) were restrained at 30℃. At the start of restraint, there was no significant difference among the rectal temperatures of these four groups. During the restraint stress, elevated core temperature was sustained and tail skin temperature decreased. Both values of rectal temperature (Tre) at the 30th min and increase of Tre (dTre) in the first 30th min during the restraint stress were in the order SHR-C>SHR-H>WKY-C>WKY-H. Values of Tre and dTre in cold-acclimated SHR were significantly greater than those in cold-acclimated WKY, and those values in heat-acclimated SHR were significantly greater than those in heat-acclimated WKY. Thermal acclimation, which started in one month old rats, scarcely affected development of hypertension in SHR, whereas thermal acclimation produced a significant difference between BPs in cold- and heat-acclimated normotensive strains of rats. As for the influence of thermal acclimation on the responsiveness of stress-induced elavation of core temperature to the restraint stress, the strain difference was greater than the influence of thermal acclimation.

Key words: Spontaneously hypertensive rats, Thermal acclimation, Blood pressure, Restraint stress, Core temperature

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INTRODUCTION

It is well known that psychological stress, such as handling, the transfering of rats from the home cage to the open field, restraint stress, etc., induces an elevation of the core temperature and a peripheral vasoconstriction. The stress-induced elevation of core temperature has been called stress-induced hyperthermia (emotional hyperthermia). Kluger and his coworkers (Singer *et al.*, 1986) have suggested that this phenomenon is not stress-induced hyperthermia but fever. Briese and Cabanac (1991) supported this opinion by their experiment. This opinion is supported by following reasons, ① preliminary administration of antipyretic drugs, such as indomethacin or sodium salicylate reduced the stress-induced elevation of core temperature (Singer *et al.* 1986; Kluger *et al.*, 1987), and ② the magnitude of this response is the same in cold and warm environmental temperature (Long *et al.*, 1990; Briese and Cabanac, 1991).

Spontaneously hypertensive rats (SHR) were developed from normotensive Wistar-Kyoto rats (WKY) which had been separated from Wistar rats (WIS) (Okamoto and Aoki, 1963). It is well known that genetical hypertensive rats, SHR show an enhanced stress-induced response, such as elevation of core temperature (Hajós and Engberg, 1986; Tsuchiya *et al.*, 1989) and cardiovascular response to air jet stress in the rat (Ludin and Thorén, 1982; Ely *et al.*, 1985). On the other hand, it was reported that chronic exposure to cold environment enhanced the stress-induced elevation of core temperature and developed the increased systemic blood pressure in Wistar rats (Tsuchiya and Kosaka, 1989; Fregly *et al.*, 1989). There are many reports about genetic relationships among the development of hypertension, enhanced cardiovascular reactivity and behavioral hyperactivity (Tucker and Jonson, 1981; Knardahl and Hendley, 1990; Hendley *et al.*, 1983)

This study was designed to examine the influence of thermal acclimation on the development of hypertension and on stress-induced elevation of core temperature to restraint stress in SHR.

MATERIAL AND METHODS

Animals

Male rats of the three strains, spontaneously hypertensive rats (SHR, Charles River, Japan), Wistar-Kyoto rats (WKY, Charles River, Japan) and Wistar rats (WIS, Shizuoka Laboratory Animal Center) were used in this experiment. Two or three rats were kept in a plastic cage on wood shavings. Food and water were given *ad lib.* under constant room temperature (22–24℃), constant humidity (50%, r.h.) and alternating 12 hr light/dark cycle (08:00–20:00). Experiments were done during the daytime (10:00–17:00). Rats of each strain, at one-month old (M.), were randomly divided into three groups. Rats were chronically exposed to 3 different ambient temperatures, 10℃ (cold-acclimated rats), 30℃ (heat-acclimated rats) and 22–24℃ of thermoneutral temperature (control) for more than 2 months.

Blood pressure measuring

Blood pressure and body weight were measured in adult rats {about 4 months old (M.) for SHR and WKY and 7 M. for WIS} and in older adult rats (6 M.–9 M. for SHR and WKY, 11 M. for WIS). After exposure to hot environment of 30℃–35℃ for about 20 min, systemic blood pressure (BP) was measured by tail cuff method using a sphygmomanometer (PE-300, Narco Bio Systems, USA).

Restraint experiment

Each conscious rat of SHR and WKY (4–5 M.) was placed in a loosely fitting cage, which was suspended in air in a climatic chamber. Rats were treated carefully to avoid psychological disturbance before the restraint experiment. Its air temperature was kept at a constant 30℃ (60%, r.h.) in case of heat-acclimated rats and at 10℃ in case of cold-acclimated rats. A thermistor probe was inserted more than 5 cm beyond the anus, and another one was attached on the dorsal surface of the tail at the middle to base of the tail and the thermistor sensors were loosely wrapped by adhesive surgical tape. The thermistor sensors were coated by a thin polyethylene, diameter was 2 mm for the rectum, 1 mm for the tail skin. During the restraint, rectal temperature and the tail skin temperature were recorded continuously as indices of change in core temperature and the tail blood flow, respectively.

Statistics

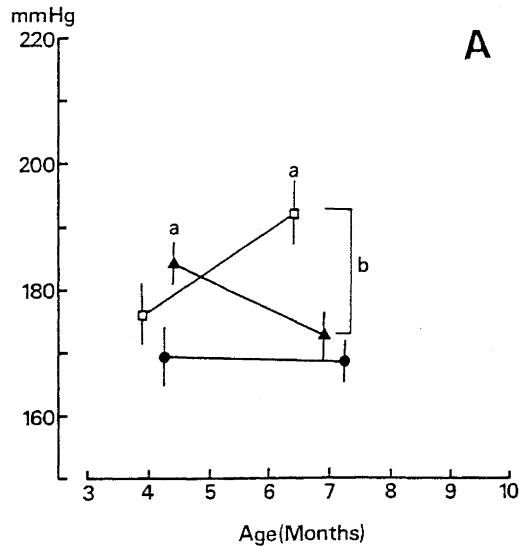
Values are represented as means and standard error of means (Mean±S.E.). Statistical significance was determined by a two tailed Mann Whiteneys U-test.

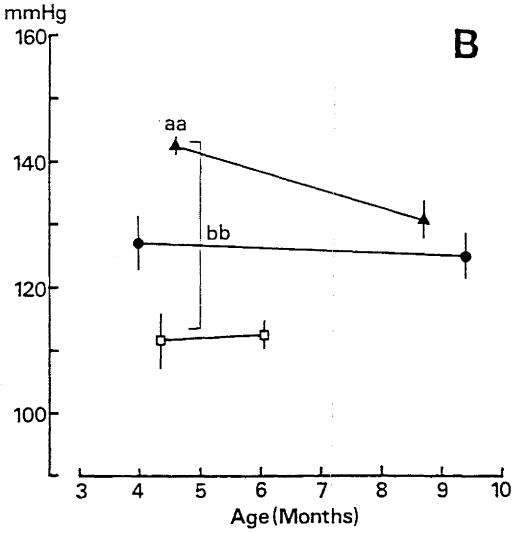
RESULTS

I) *BP and BW in adult rats*

a) BP

In SHR (3.9 M.–4.4 M.), mean value of systolic blood pressure (BP, M. ±S.E.) was 184 ±4 mmHg in cold-acclimated SHR (SHR-C, 4.4 M., N=10), 176±5 mmHg in heat-acclimated SHR (SHR-H, 3.9 M., N=9), 170±5 mmHg in contol SHR (SHR-N, 4.2 M. N=8). In WKY (4.0 M.–4.6 M.), mean value of BP was 143±2 mmHg in cold acclimated WKY (WKY-C, 4.6 M, N=5), 115±3 mmHg in heat-acclimated (WKY-H, 4.3 M., N=8) and 127±4 mmHg in control WKY (WKY-N, 4.0 M., N=6). In WIS (7.4 M), mean value of BP was 135±3 mmHg in cold-acclimated WIS (WIS-C, N=6), 110±4 mmHg in heat-acclimated WIS (WIS-H, N=6) and 118±5 mmHg in control WIS (WIS-N, N=6). In these three strains, BPs of cold-acclimated groups were significantly ($p<0.05$ for SHR and WIS, $p<0.01$ for WKY) higher than the value of each control. There were no significant differences between BP of SHR-C and SHR-H. In both WKY and WIS, BPs of cold-acclimated rats were significantly ($p<0.01$) higher than those of heat-acclimated rats. Changes in BP of adult and older adult rats of the three strains are illustrated in Figs. 1-A, B and C.





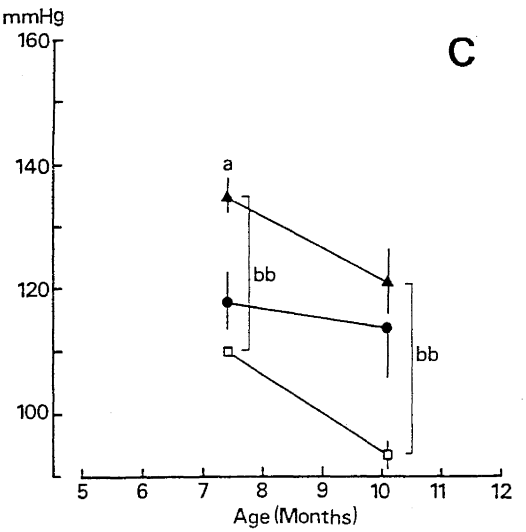


Fig. 1. Systolic blood pressure measured by the tail cuff method in thermally acclimated rats of the three strains, *A*; spontaneously hypertensive rats (SHR), *B*; Wistar-Kyoto rats (WKY), *C*; Wistar rats (WIS). Mean values of systolic blood pressure (Mean±S.E.) were shown for cold-acclimated rats with solid triangles, heat-acclimated rats with open rectangles and control rats with solid circles. Symbols for statistical significance are as follows; a; $p<0.05$, aa; $p<0.01$ as compared to control, b; $p<0.05$, bb; $p<0.01$ comparison between heat- and cold-acclimated rats.

b) BW

In SHR, mean value of BW was 344±7 g in SHR-C, 264±17 g in SHR-H, 308±11 g SHR-N. Value of BW in SHR-C was significantly greater ($P<0.01$) and that in SHR-H was significantly smaller ($p<0.05$) than that of control. In WKY, mean value of BW was 349±6 g in WKY-C, 314±7 g in WKY-H and 324±15 g of WKY-N. Values in WKY-C and WKY-H were not significanly different to that in control. In WIS, mean value of BW was 618±13 g in WIS-C, 506±20 g in WIC-H and 677±28 g in WKY-N. Value in WIS-H was significantly ($p<0.05$) smaller than that of control. In the three strains, BWs in cold-acclimated groups were significantly greater ($p<0.01$ for SHR and WIS, $p<0.05$ for WKY) than that in heat-acclimated rats of each strain.

In the three strains, BPs and BWs in adult rats of three types of the thermal acclimations are summarized in Table 1-(I).

Table 1. Systolic blood pressure (BP) and body weight (BW) in thermally-acclimated spontaneously hypertensive rats (SHR), Wistar-Kyoto rats (WKY) and Wistar rats (WIS). SHR-C, SHR-H and SHR-N represent cold-acclimated SHR, heat-acclimated SHR and control reared under thermoneutral condition, respectively

(I) Adult

	BP (mmHg)	BW (g)	N	Age (Months)
(A) SHR				
SHR-C	184±4 a	344±7 aa ┐bb	10	4.4
SHR-H	176±5	264±17 a ┘	9	3.9
SHR-N	170±5	308±11	8	4.2
(B) WKY				
WKY-C	143±2 aa ┐bb	349±6 ┐b	5	4.6
WKY-H	115±5 ┘	314±7 ┘	8	4.3
WKY-N	127±4	324±15	6	4.0
(C) WIS				
WIS-C	135±3 a ┐bb	618±13 ┐bb	6	7.4
WIS-H	110±4 ┘	506±20 a ┘	6	7.4
WIS-N	118±5	677±28	6	7.4

(II) Older adult

	BP (mmHg)	BW (g)	N	Age (Months)
(A) SHR				
SHR-C	173±3 ┐b	390±5 a ┐b	10	6.9
SHR-H	192±6 a ┘	302±16 aa ┘	8	6.4
SHR-N	169±3	423±10	6	7.3
(B) WKY				
WKY-C	131±4 ┐bb	372±14	5	8.7
WKY-H	111±6 ┘	376±9	5	6.0
WKY-N	125±4	409±19	4	9.4
(C) WIS				
WIS-C	121±6 ┐bb	648±12 ┐bb	6	11.0
WIS-H	93±6 ┘	547±25 a ┘	6	11.0
WIS-N	114±8	694±47	6	11.0

N; numbers of rats. a; $p<0.05$, aa; $p<0.01$ compared to values in control rats. b; $p<0.05$, bb; $p<0.01$ compared between cold- and heat-acclimated rats.

Ⅱ) *BP and BW in the older adult rats*

a) BP

In SHR (6.9 M.–7.3 M.), mean value of BP was 173±3 mmHg in SHR-C (6.9) M., N=10), 192±6 mmHg in SHR-H (6.4 M., N=8), 169±3 mmHg in SHR-N (7.3 M., N=6). Value of BP in SHR-H was significantly ($p<0.05$) higher than that of control. In WKY (8.7 M. −9.4 M.), mean value of BP was 131±4 mmHg in WKY-C (8.7 M., N=5), 111±6 mmHg in WKY-H (6.0 M., N=5) and 125±4 mmHg in WKY-N (9.4 M., N=4). In WIS (11.0 M.), mean BP was 121±6 mmHg in WIS-C (N=6), 93±6 mmHg in WIS-H (N=6) and 114±8 mmHg in WKY-N (N=6). Value of BP in SHR-H was significantly higher ($P<0.05$) than that of SHR-C, and values in WKY-H and WIC-H were significantly ($p<0.01$ for WKY and WIS) lower than that of cold-acclimated group in each strain.

b) BW

In SHR, mean value of BW was 390±5 g in SHR-C, 302±16 g in SHR-H, 423±10 g SHR-N. Value of BW in SHR-C was significantly greater ($p<0.05$) and BW in SHR-H was significantly smaller ($p<0.01$) than that in control. In WKY, mean value of BW was 372±14 g in WKY-C, 376±9 g in WKY-H and 409±19 g in WKY-N. Values of BW in WKY-C and WKY-H were not significanly different to each other . In WIS, mean BW was 648±12 g in WIS-C, 547±25 g and 694±47 g in WKY-N. Value of WIS-H was significantly ($p<0.05$) smaller than that of control. In SHR and WIS, BWs in the cold-acclimated group were significantly greater ($p<0.05$ for SHR, $p<0.01$ for WIS) than those in heat-acclimated groups.

In the three strains, Bp and BW in older adult rats of three types of thermal acclimations are summarized in Table 1-(Ⅱ).

Ⅲ) *Change in rectal temperature (Tre) and tail skin temperatue during restraint in thermally-acclimated SHR and WKY*

a) Change in rectal temperature

At the start of restraint, the mean rectal temperarure (Tre) was 37.1±0.2℃ in SHR-C (4.8 M., N=6) and 37.2±0.1℃ in SHR-H (3.9 M., N=8). Mean Tre was 37.1±0.1℃ in WKY-C (5.1 M., N=5), and 37.3±0.3℃ in WKY-H (5.2 M., N=5). There were no significant differences among these four values. Changes in Tre is shown in Fig 2-A, and increases in Tre (dTre) are shown in Fig. 2-B.

During restraint, Tre increased gradually, peaked in the 20th min to the 30th during the restraint. Mean Tre at the 30th min (Tre (30)) and mean increase of Tre in the first 30 min (dTre (30)) were summarized in Table 2. Tre (30) and dTre (30) were 39.4±0.1℃ and 2.3±0.3℃ in SHR-C, 39.0±0.1℃ and 1.8±0.2℃ in SHR-H and 38.4±0.1℃ and 1.2±0.2℃ in WKY-C and 37.9±0.2℃ and 0.6±0.2℃ in WKY-H, respectively. In both Tre (30) and dTre (30), there were no significant differences between values of heat-acclimated rats and cold-acclimated rats in both strains, SHR and WKY. Tre (30) of SHR-C was significantly higher ($p<0.01$) than that of WKY-C, and Tre (30) of SHR-H was significantly higher than that of WKY-H. Value of dTre (30) in SHR-H was significantly ($p<0.01$) greater than that in WKY-

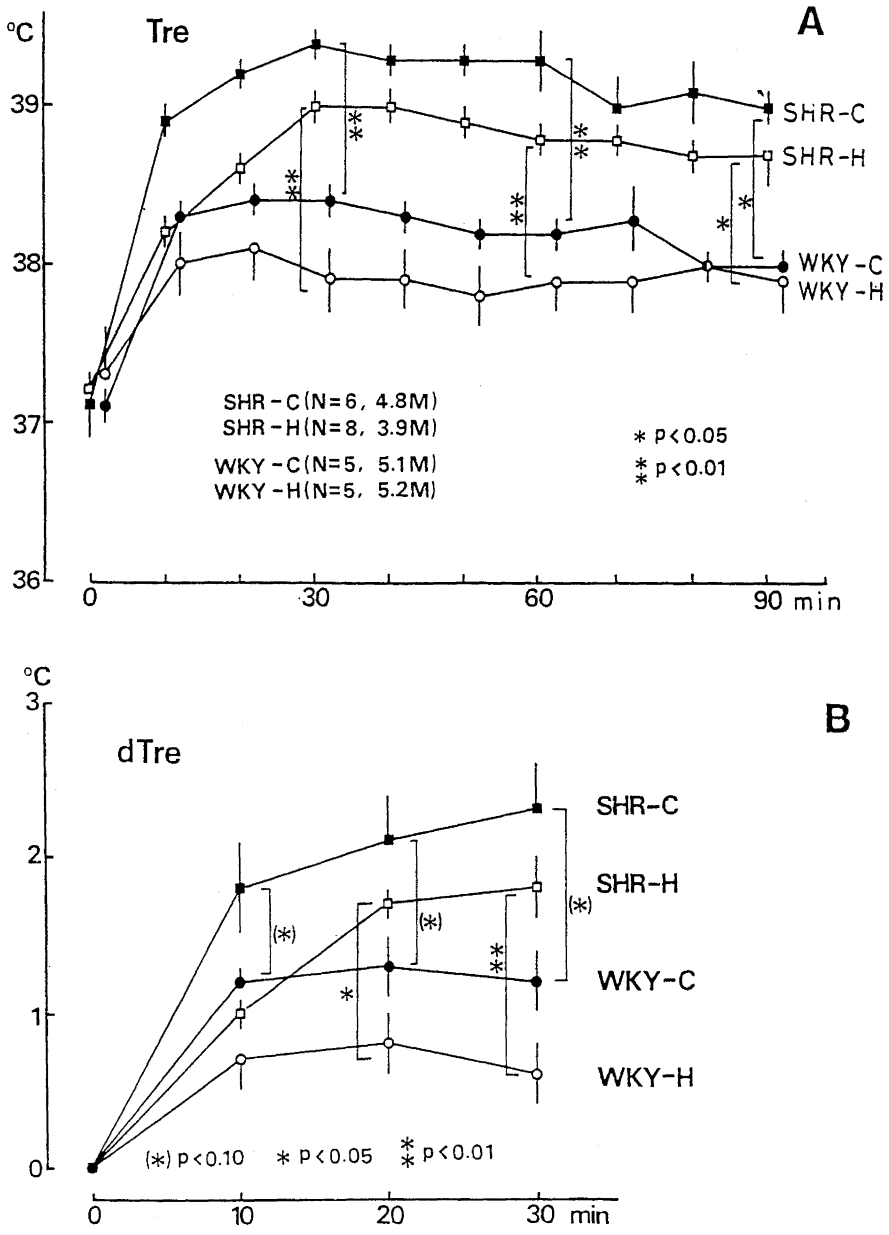


Fig. 2. Change in rectal temperature (*A*) and increase of rectal temperature (*B*) during the restraint in thermally-acclimated SHR and WKY. Mean values were shown for cold-acclimated SHR (SHR-C) with solid rectangles, heat-acclimated SHR (SHR-H) with open rectangles, cold-acclimated WKY (WKY-C) with solid circles and heat-acclimated WKY (WKY-H) with open circles. Standard errors were shown with vertical bars. SHR-C and WKY-H were restrained at 10℃ while SHR-H and WKY-H were restrained at 30℃. In Fig. 2-A, statistical significances of differences between values in SHR-C and WKY-C and those in SHR-H and WKY-H at time 0, 30, 60, and 90 min are indicated in these figures. Symbols for statistical significance as follows; * * ; $p<0.01$. *; $p<0.05$, (*); $p<0.10$

Table 2. Rectal temperature (Tre), change in rectal temperature (ΔTre) and change in the difference (ΔTtail,) between tail skin (Ttail) and environmental air temperature (Ta) during the restraint stress (ΔTtail=Ttail−Ta) in cold− and heat− acclimated rats

	Tre(℃) at time 0 min	Tre(℃) at time 30 min	dTre(℃) in the first 30 min	dTtail(℃) at time 30 min	dTtail(℃) at time 60 min	N	Age (Month)
SHR							
SHR-C	37.1±0.2	39.4±0.1	2.3±0.3	0.5±0.1	0.6±0.2	6	4.8
SHR-H	37.2±0.1	39.0±0.1	1.8±0.2	0.2±0.3	2.2±0.5	8	3.9
WKY							
WKY-C	37.1±0.1	38.4±0.1	1.2±0.2	0.3±0.1	0.4±0.4	5	5.1
WKY-H	37.3±0.3	37.9±0.2	0.6±0.2	0.7±0.6	0.9±0.5	5	5.2

Values are represented as mean±S.E.. N; number of rats. Symbols for statistical significance are shown as follows **; $p<0.01$, *; $p<0.05$, (*); $p<0.10$.

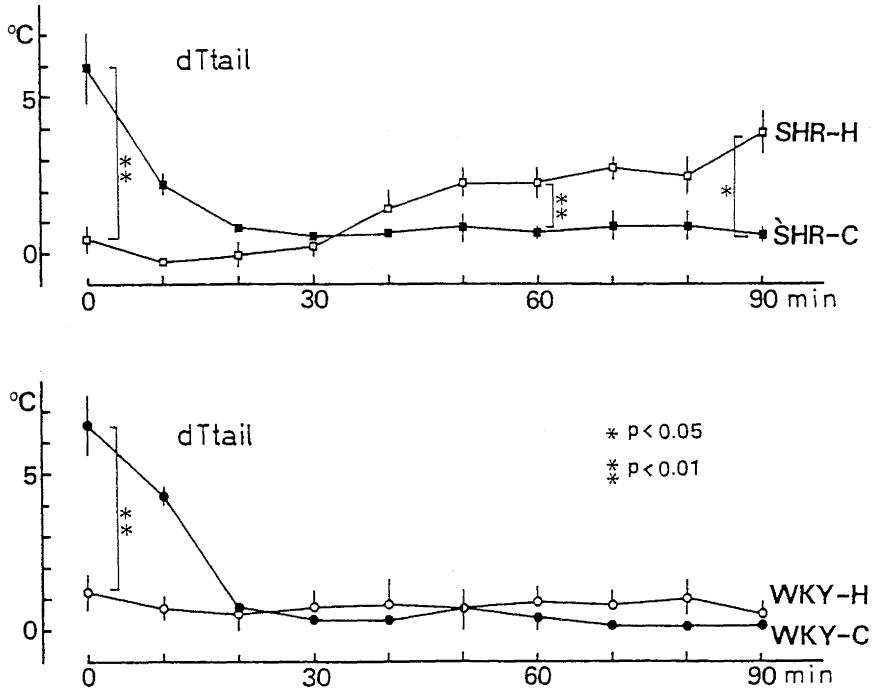


Fig. 3. Changes in differneces (dTtail) between tail skin temperature (Ttail) and environmental temperature (Ta), (dTtail=Ttail−Ta). *A;* Comparison between cold-acclimated SHR (SHR-C) and heat-acclimated SHR (SHR-H), *B;* comparison between cold-acclimated WKY (WKY-C) and heat-acclimated WKY (WKY-H). SHR-C and WKY-C were restrained at 10℃, and SHR-H and WKY-H were restrained at 30℃. Statistical significances of values between values at time 0, 30, 60 and 90 min in SHR and WKY are shown. Other abbreviations and symbols for statistical significance are the same as shown in Fig. 2.

H, and Tre (30) of SHR-C was greater than that of WKY-C ($P<0.10$).

b) Change in the tail skin temperature (Ttail)

As shown in Fig. 3, in SHR-C and WKY-C, dTtail were more than 5℃ at the start of restraint. In cold-and heat-acclimated rats of both strains, Ttail continuously decreased during the first 30 min during the restraint. In the cold-acclimated rat of both strains and WKY-H, low value of dTtail was kept through out the restraint. This indicates the peripheral vasoconstriction in the tail skin. In heat-acclimated SHR, dTtail began to increase at after 30 min. Differences (dTail) between tail skin temperature (Ttail) from ambient temperature (Ta) are summarized in Table 2 (dTtail=Ttail−Ta) and illustrated in Figs. 3-A and 3-B. At 30th min from the beginning of the restrain, mean value of dTtail was 0.5±0.1℃ in SHR-C, 0.2±0.3℃ in SHR-H, 0.3±0.1℃ in WKY-C and 0.7±0.6℃ in WKY-H. There were no significant differences among these four values. Mean values of dTtail at 60th min and dTtail (60) were in the same order 0.6±0.2℃, 2.2±0.2℃, 0.4±0.4℃ and 0.9±0.5℃. dTtail (60) of SHR-H was significantly ($p<0.01$) greater than that of SHR-C.

DISCUSSION

Systemic BP

In this study, rats of the three strains, SHR, WKY and WIS were used. In two normotensive strains, WKY and WIS, systemic BPs in cold-acclimated groups (10℃) were significantly higher than those in heat-acclimated ones (30℃). Hypertension of SHR was similarly developed both in cold and warm environment. In hypertensive strains, SHR, BPs in both cold-and heat-acclimated groups of young adult (4M.) were not significantly different from each other, whereas BP of heat-acclimated SHR was higher than that of cold acclimated SHR in the older adult group (9 M.). There was a tendency for systemic BPs to decrease with age in cold acclimated rats in the three strains.

Fregly and his coworkers have studied the effect of chronic exposure to cold (5℃–6℃) on the cardiovascular system in rats. They reported that after chronic exposure to cold, the responsiveness of the heart to β-adrenergic stimulation increased (Fregly *et al.*, 1977; Barney *et al.*, 1980) and systemic BP increased (Fregly *et al.*, 1989). It was suggested that the sympathetic nervous system is involved in cold-induced hypertension from the fact that the concentration of plasma noradrenaline was high in cold-acclimated rats (Papanek *et al.*, 1991).

It was reported that the function of the cardiac sympathetic varicosity and a higher vascular resistance of SHR compared with WKY were already established at 4 weeks (Dyke *et al.* 1989; Adams *et al.*, 1989). Tucker *et al.* (1984) studied that effect of environmental stress on genetically programmed development of autonomic nervous control of the heart in the rat. They revealed the fact that the early stimulation by handling decreased sympathetic tones on the heart in neonates of the both SHR and WKY.

From these facts, it is suggested that rats in a prehypertensive stage of less than 4 weeks may be susceptible to the influence of chronic exposure to various environmental temperatures. In this study, the chronic exposure of rat to heat or cold environment started at 4 weeks of age.

Body weight

In cold-acclimated groups, BWs were significantly greater than those in heat-acclimated rats of the three strains, except for BW in older adult WKY. In older adult rats, BWs in controls which were reared under thermoneutral condition was greatest in each strain. This fact suggests that chronic exposure to cold as well as heat might not be suitable for growth of the rat.

Yamauchi *et al.* (1981) studied the effect of rearing temperature on the growth of the rat, BW of the rat in 3 weeks of age was significantly smaller at 12℃, 14℃, 30℃ and 32℃. Peeters *et al.* (1989) also reported that a growth rate was smaller in rats chronically exposed to 4℃ than that in the control.

Influence of themal acclimation on increase of BW is similar among the three strains, whereas its influence on the systemic BP was different between the hypertensive SHR and the normotensive WKY and WIS.

Restraint stress

It was widely believed that SHR have higher core temperature than that of control strain, WKY. However, it was suggested that high core temperature of the SHR might be due to the stress-induced elevation of the core temperature in the measuring procedure of the rectal temperature (Hajós and Engberg, 1986). Recently, the core temperatures of SHR and WKY were compared by measuring with the radiotelemetry method which had negligible psychological disturbance in the measuring procedure. It was shown that there were no significant differences between core temperature of SHR and WKY even after the exposure to the ambient temperatures ranging from 5℃ to 35℃ during 1 hr as well as in 24 hr recording under thermoneutral condition (Berkey *et al.*, 1990; Morley *et al.*, 1990).

In this study, before the start of the restraint experiment, rats were carefully treated to minimize the psychological disturbance. There was no significant difference in the rectal temperature just after the start of the restraint (at time 0 in Fig. 2 and Table 2) in four groups of rats, SHR-C, SHR-H, WKY-C and WKY-H. These rectal temperatures at the start of the restraint stress may be near the resting core temperature.

Enhanced responses of SHR to the various environmental stress were reported, for example, cardiovascular responses to air jet on the face (Lundin and Thorén, 1982; Ludin *et al.*, 1984; Ely *et al.*, 1985), temperature responses of the body core and of the tail skin to the restraint stress (Tsuchiya *et al.*, 1989) etc. It has been emphasized that stress activates the pituitary-adrenal system and the sympathoadrenal system. Stress-induced activation of the sympathetic nerves which regulate the activity of organs, such as brown adipose tissue (BAT) (Shibata and Nagasaka, 1982, 1984) as well as sympathetic vasoconstrictor nerve in the tail skin (Tsuchiya *et al.*, 1989; Tsuchiya and Kosaka, 1989) were reported.

BAT of rat is the major site of nonshivering thermogenesis which is controlled by the sympathetic nerves. Hayashi *et al.* (1988) showed that nonshivering thermogenesis capacity of the SHR does not differ from that of the WKY. Therefore, greater elevation of core temperature of SHR compared with WKY may not be due to differences of thermogenesis capacity of BAT but to the activity of the sympathetic nerves. Restraint stress reduced dT-tail (difference between tail skin temperature and ambient temperature, Fig. 3). This fact indicates peripheral vasoconstriction due to activation of the sympathetic vasoconstrictor nerves (O'Leary *et al.*, 1985; Kondo, 1972). Both in SHR and WKY, values of dTre in cold-acclimated groups were greater those in heat-acclimated ones. Both values of Tre at the 30th min and dTre in the first 30 min were in the order SHR-C>SHR-H>WKY-C>WKY-H. One of the possible explanation for the difference between heat- and cold-acclimation is considered as follows; Thermal acclimation affects on the BAT functionally and morphologically (Smith and Roberts, 1964; Al-Hilli and Wright, 1980; Himms-Hargen, 1986; Bertin *et al.*, 1990), this may result in a change of nonshivering themogenesis capacity during the stress.

In conclusion, cold- and heat-acclimation in rats, which started at one month of age, had only a weak effect on the development of hypertension in SHR, whereas there were significant difference between BPs in cold-and heat-acclimated normotensive strains. As for the magnitude of the temperature response to the restraint stress, the strain difference was

greater than the difference due to thermal acclimation. It still remains to be solved whether the genetically programmed functional changes in SHR is more susceptible to influence of environmental temperature in the prehypertension phase than in the hypertension-developmental phase.

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