

Research article

Temperature Control of Hypertensive Rats during Moderate Exercise in Warm Environment

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Abstract

The control of body temperature in Spontaneously Hypertensive Rat (SHR) subjected to exercise in warm environment was investigated. Male SHR and Wistar rats were submitted to moderate exercise in temperate (25°C) and warm (32°C) environments while body and tail skin temperatures, as well as oxygen consumption, were registered. Total time of exercise, workload performed, mechanical efficiency and heat storage were determined. SHR had increased heat production and body temperature at the end of exercise, reduced mechanical efficiency and increased heat storage ($p < 0.05$). Furthermore, these rats also showed a more intense and faster increase in body temperature during moderate exercise in the warm environment ($p < 0.05$). The lower mechanical efficiency seen in SHR was closely correlated with their higher body temperature at the point of fatigue in warm environment ($p < 0.05$). Our results indicate that SHR exhibit significant differences in body temperature control during moderate exercise in warm environment characterized by increased heat production and heat storage during moderate exercise in warm environment. The combination of these responses result in aggravated hyperthermia linked with lower mechanical efficiency.

Key words: Thermoregulation; physical exercise; hypertension; heat stress; exercise performance.

Introduction

The practice of physical exercise, as well as other activities of daily living, in warm environment has gained importance in recent decades mainly because of the progressive increases in environmental temperature, which is directly related to hospitalizations due to cardiovascular disease and heat stroke (Easterling et al., 2000; Ebi et al., 2004; Nybo, 2008). During exercise performed in the heat, hypertensive patients experience an elevated blood pressure response and a greater thermal strain than normotensive individuals (Kenney et al., 1984). Since low-to-moderate exercise is currently prescribed as a non-pharmacological treatment for hypertension (Carneiro-Júnior et al., 2013), and warm environment may represent additional cardiovascular stress during physical activity, the study of body temperature (T_b) control in hypertension emerge as a focus of research.

The Spontaneously Hypertensive Rat (SHR) has been extensively used to study cardiovascular disease since it is

considered a good animal model of human essential or primary hypertension (Folkow, 1987; Okamoto and Aoki, 1963). Both species share pathophysiological cardiovascular features of hypertension such as increased total peripheral resistance and hyperresponsiveness of the sympathoadrenal system to stressful stimuli (Folkow, 1987; Okamoto and Aoki, 1963; Trippodo and Frohlich, 1981).

In order to maintain T_b constant at $\sim 37^\circ\text{C}$, the balance between heat production and heat loss is accurately coordinated by the central nervous system (Romanovsky et al., 2007; Webb, 1995). During exercise, the inherent increase in heat production in response to greater metabolic rate leads to the activation of heat loss mechanisms to avoid critically high T_b (Leite et al. 2007; Pires et al., 2007). It is well accepted that homeothermic animal species are able to control T_b efficiently in a range of cool to moderate ambient conditions (Galloway and Maughan, 1997). However, this capacity has been shown to be impaired during exercise conducted in warm environment (Fuller et al., 1998; Walters et al., 2000). In such situation, increased blood flow to maintain the energy demand of active muscles occurs simultaneously with increases of cutaneous blood flow required to meet the demand of T_b regulation (Folkow, 1987; González-Alonso et al., 2008). These combined demands for blood flow can result in a competition for the available cardiac output (Rowell, 1974), resulting in increased T_b and reduced physical capacity. In fact, it has been shown that high T_b and increased rates of body heating and heat storage are limiting factors for physical performance during prolonged exercise (Fuller et al., 1998; Lacerda et al., 2005; Leite et al., 2006).

Taking into account the relationship between the cardiovascular system and the regulation of T_b , and that hypertension can affect the mechanisms of heat dissipation due to increased peripheral resistance (Kellogg et al., 1998; O'Leary and Wang, 1994; Wright et al., 1978), studies have been conducted to investigate the thermoregulatory ability of SHR (Barney et al., 1999; Berkey et al., 1990). Berkey et al. (1990) measured core temperature of SHR at ambient temperatures of 5, 15, 25, 35 and 40°C. After 1h of exposure to these different ambient temperatures the only ambient temperature at which SHR had higher core temperature than normotensive controls was at 40°C (Berkey et al., 1990). Additionally, Barney et

al. (1999) exposed SHR to ambient temperature of 25 or 37.5°C for 3.5h, reporting that SHR had both higher core temperature and evaporative water loss during heat exposure than normotensive controls (Barney et al., 1999). Such studies support that SHR has a hyperresponse to stress, which leads to higher core temperature in situations like exposure to warm environment (Berkey et al., 1990) and dehydration due to heat stress (Barney et al., 1999).

Despite these previous results, there are few studies investigating the control of T_b during exercise in warm environment associated with pathological conditions of the cardiovascular system, i.e., hypertension. Thus, the aim of the present study was to investigate temperature control in SHR subjected to moderate exercise in warm environment.

Methods

Animals

Male SHR (systolic blood pressure: 170 ± 2.6 mmHg, 300-370 g, $n = 8$) and normotensive WIS (systolic blood pressure: 104 ± 2.7 mmHg, 350-450 g, $n = 8$) at 16 weeks of age were individually housed at room temperature of 22 ± 2 °C, under 12/12 h light-dark cycles and had free access to water and rodent chow. Taking into account that the development of hypertension in SHR is dependent on age rather than body weight, age was matched in the present experimental paradigm (Hom et al., 2007). All experimental protocols were approved by the Ethics Committee in Animal Use of our Institution (Protocol #74/2010) and were carried out in accordance with the Guide for the Care and Use of Laboratory Animals (2011).

Implantation of intraperitoneal sensor

Anesthesia was induced and maintained with a mixture of ketamine ($2 \text{ mg}\cdot\text{kg}^{-1}$) and xylazine ($2 \text{ mg}\cdot\text{kg}^{-1}$, i.p.). After pre-surgical preparation of the incision site, a G2 E-Mitter temperature sensor (Mini Mitter, USA) was implanted into the peritoneal cavity through a small incision in the linea alba. Prior to surgery, all rats received a prophylactic dose of antibiotic (Enrofloxacin, $10 \text{ mg}\cdot\text{kg}^{-1}$, i.m.). Postoperative analgesia (Tramadol, $4 \text{ mg}\cdot\text{kg}^{-1}$, every 8 h, i.m.) was provided as soon as animals awoke from anesthesia and was continued for 24 hours.

Familiarization period

After a 48 h recovery period following surgery, animals were gradually adapted to exercise on a motor-driven treadmill (Panlab, Harvard Apparatus, Spain) by running 5 min per day at 5° inclination for five consecutive days. During the first two days, the running speed was $10 \text{ m}\cdot\text{min}^{-1}$, increasing $1 \text{ m}\cdot\text{min}^{-1}$ per day. At the third day, animals also started to be familiarized with a skin temperature thermocouple fixed to the tail. This preliminary exercise did not constitute training, but intended to teach them which direction to run (Primola-Gomes et al., 2007). Electrical stimulation was determined according to each animal's tolerability (Leite et al., 2012).

Exercise capacity test

After adaptation, each rat was submitted to an incremental workload running test until the point of fatigue to determine their individual exercise capacity. The test was performed in temperate environment, beginning at a velocity of $10 \text{ m}\cdot\text{min}^{-1}$ (5° inclination), with increments of $1 \text{ m}\cdot\text{min}^{-1}$ every 3 min until fatigue. Fatigue was defined as the point where animals were no longer able to keep pace with the treadmill for 10 s (Soares et al., 2004).

Experimental protocol

On the day of the experiment and according to the exercise capacity test results, animals were individually submitted to running exercise until fatigue at a constant speed and inclination (60% maximal running speed and 5° inclination). The experimental protocol was performed between 7:00 and 12:00 am and conducted in two ambient temperature situations: 1) temperate environment (TE, 25°C) and 2) warm environment (WE, 32°C). The experimental situations were randomized and balanced. A 48 h interval was allowed for recovery between tests.

Intraperitoneal temperature and tail skin temperatures (T_{tail}) were recorded during moderate exercise. Intraperitoneal temperature was used as an index of body T_b , and measured by an implanted telemetry sensor using the VitalView software package (VitalView, Mini Mitter, USA). T_{tail} was measured using a thermometer (THR-140, Instrutherm Instruments, Brazil) and a thermocouple (S-09K, Instrutherm Instruments, Brazil) taped with a waterproof adhesive tape to the lateral surface of the tail, 20 mm from the base of the tail (Young and Dawson, 1982).

In another experimental setup the metabolic rate was measured by means of oxygen consumption (VO_2) with an open-flow indirect calorimeter (Panlab, Harvard Apparatus, Spain), and was continuously recorded during exercise until fatigue using a computerized system (Metabolism, Harvard Apparatus, Spain) (Leite et al., 2007).

Calculations

Workload (W, kgm) was calculated as follows: body weight (kg) x TTE (min) x treadmill speed ($\text{m}\cdot\text{min}^{-1}$) x $\sin\alpha$ (treadmill inclination) (Brooks and White, 1978).

Mechanical efficiency (ME, %) was calculated using the formula: $\text{ME} = (\text{workload}/\text{energetic cost}) \times 100$ (Brooks et al., 1984).

Heat storage (HS, cal) was calculated as $\text{HS} = (\Delta T_b) \times \text{body weight (g)} \times c$, where ΔT_b represents the change in T_b ($T_f - T_i$) and T_f and T_i represent the T_b at the fatigue point and prior to exercise, respectively, and c represents specific heat of the body tissues ($0.826 \text{ cal}\cdot\text{g}^{-1}\cdot\text{°C}^{-1}$) (Gordon, 1993). HS was normalized per 100 g of body weight.

Statistical analysis

Data are reported as mean \pm SEM. Two-way ANOVA was used for determining differences between groups and the effect of environment temperature. This was done in order to evaluate the differences in T_b , T_{tail} and VO_2 . Significant interactions observed by ANOVA were further evaluated by Tukey *post hoc* analysis to locate significant differences between means. To evaluate T_b , T_{tail} and VO_2 during experiments compared to baseline we used one-

way ANOVA with repeated measures followed by Tukey *post hoc*. Total time of exercise, workload, mechanical efficiency and heat storage were analyzed by two-way ANOVA followed by the Newman-Keuls *post hoc*. The correlations were assessed using Pearson's correlation coefficient. Significance level was set at $p < 0.05$.

Results

During the exercise capacity test, the WIS and SHR showed similar physical performance (47.03 ± 5.06 min WIS vs. 54.21 ± 5.21 min SHR). The maximal running speed was also similar between groups (22.3 ± 0.8 m·min⁻¹ WIS vs. 24.0 ± 0.8 m·min⁻¹ SHR). Thus, the exercise speed corresponding to 60% of maximal running speed was equal between groups (13.4 ± 0.2 m·min⁻¹ WIS vs. 14.4 ± 0.2 m·min⁻¹ SHR).

During the moderate exercise protocol, it was not observed differences in physical performance between groups in both temperate (47.03 ± 7.06 min WIS-TE vs. 54.21 ± 5.21 min SHR-TE) and warm environments (15.78 ± 0.95 min WIS-WE vs. 16.96 ± 1.32 min SHR-WE). On the other hand, there was an environment effect during exercise that led to a reduction in physical performance of 66% and 68% for WIS and SHR, respectively ($p < 0.05$) (Figure 1). Similarly, WIS and SHR had their workload reduced by 69% and 67%, respectively, when comparing the environments. The workload calculated was 25.7 ± 2.67 kgm for WIS-TE, 6.93 ± 0.45 kgm for WIS-WE, 23.04 ± 4.13 kgm for SHR-TE and 8.29 ± 0.75 kgm for SHR-WE. The workload was not different between WIS and SHR groups.

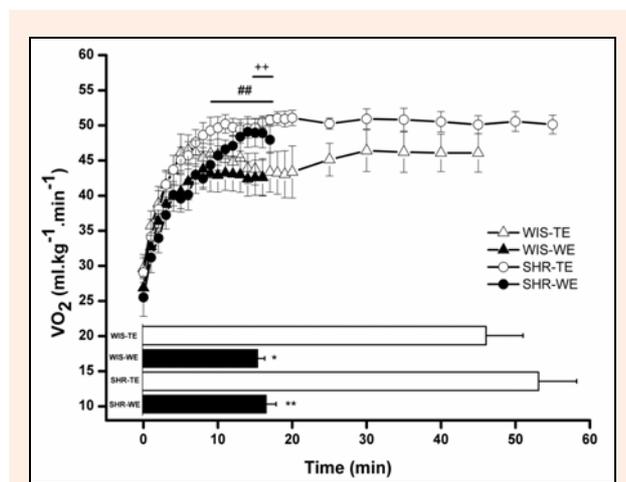


Figure 1. Oxygen consumption (VO_2) during moderate exercise. Total time of exercise is indicated by the horizontal bar at the bottom. Values are expressed as mean \pm SEM, $n = 8$ each group. * $p < 0.05$: WIS-WE vs. WIS-TE. ** $p < 0.05$: SHR-WE vs. SHR-TE. ++ $p < 0.05$: SHR-WE vs. WIS-WE. ## $p < 0.05$: SHR vs. WIS.

As seen in Figure 1, moderate exercise induced an increase in metabolic rate (heat production) for all groups. For all situations, it was not observed any environment effects in heat production. Regardless of the environment, from the 11th until the 17th min of exercise, the SHR group showed greater heat production in comparison to WIS group. Moreover, from the 15th until the 17th min of

exercise the SHR-WE had greater heat production in comparison to WIS-WE.

As seen in Figure 2A, moderate exercise induced a gradual increase in T_b in all groups. Compared to baseline, WIS-TE, WIS-WE, SHR-TE and SHR-WE had higher T_b values at minutes 7, 6, 7 and 3, respectively ($p < 0.05$). Related to the environment, animals exercising in warm environment presented higher T_b values from the 4th min until the end of exercise ($p < 0.05$). Moreover, SHR-WE showed higher T_b compared to SHR-TE after 8 min of exercise ($p < 0.05$), while WIS-WE was able to maintain its heat balance and showed T_b similar to WIS-TE at the same time. As seen in Fig. 2B, during the moderate exercise T_{tail} was higher for both groups in warm environment ($p < 0.05$).

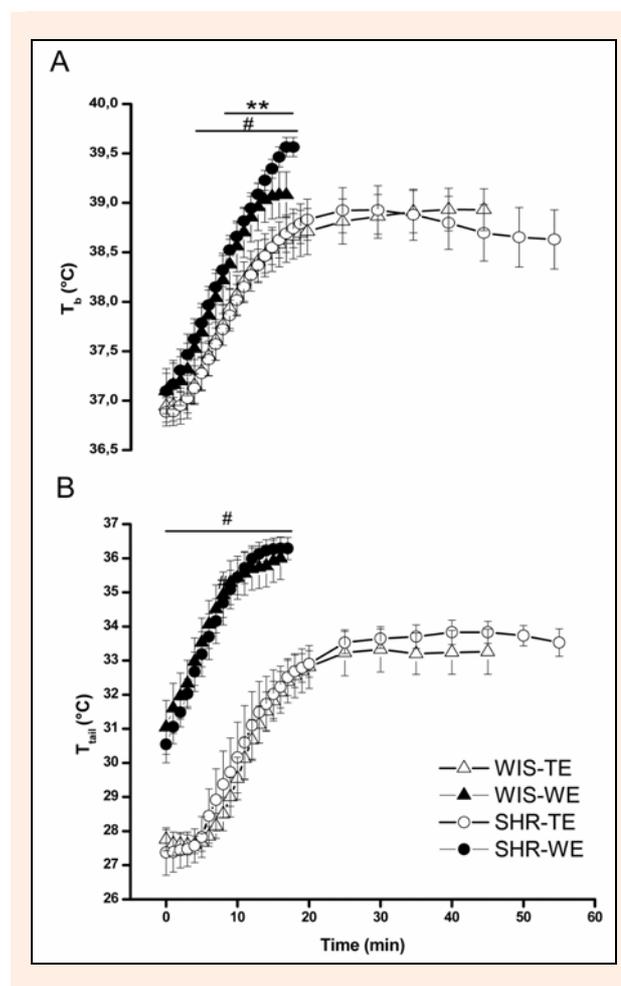


Figure 2. Body (T_b , A) and tail skin (T_{tail} , B) temperatures during moderate exercise until fatigue. Values are expressed as mean \pm SEM, $n = 8$ each group. # $p < 0.05$: WE vs. TE. ** $p < 0.05$: SHR-WE vs. SHR-TE.

As shown in Figure 3A, SHR had lower mechanical efficiency compared to WIS, both in temperate ($22.68 \pm 2.36\%$ WIS-TE vs. $12.09 \pm 0.59\%$ SHR-TE, $p < 0.05$) and warm environment ($24.65 \pm 3.21\%$ WIS-WE vs. $14.24 \pm 0.99\%$ SHR-WE, $p < 0.05$). An environmental effect in mechanical efficiency was not observed. A close correlation was found between mechanical efficiency and T_b at the point of fatigue in the warm environment ($r = 0.82$, $p < 0.05$) (Figure 3C).

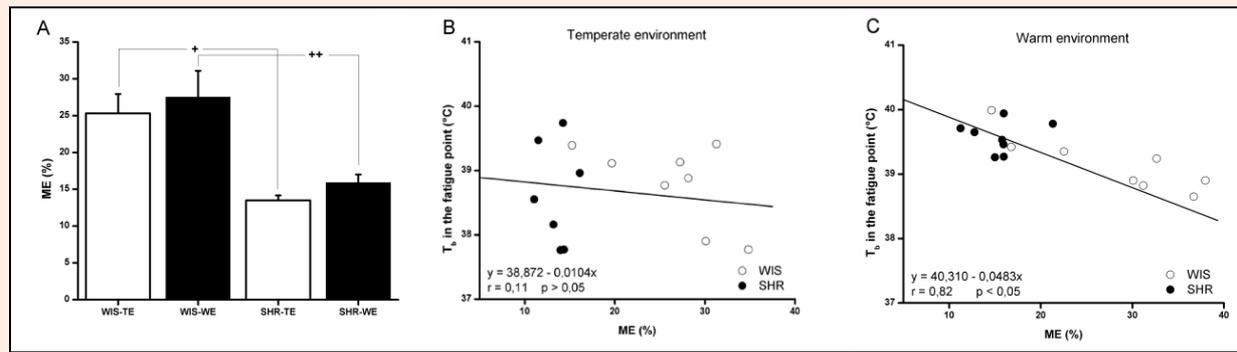


Figure 3. Mechanical efficiency (ME, **A**) during moderate exercise and correlation between ME and T_b at the fatigue point in the temperate environment (**B**) and warm environment (**C**). Values are expressed as mean \pm SEM, $n = 8$ each group. + $p < 0.05$: SHR-TE vs. WIS-TE. ++ $p < 0.05$: SHR-WE vs. WIS-WE.

To compare the total thermal effect of exercise in both groups, heat storage was calculated (Figure 4). Heat storage in SHR-WE (176.88 ± 25.42 cal/100 g body weight) was higher when compared to WIS-TE (158.12 ± 14.68 cal/100 g body weight), WIS-WE (176.88 ± 25.42 cal/100 g body weight) and SHR-TE (139.00 ± 19.14 cal/100 g body weight) ($p < 0.05$) (Figure 4).

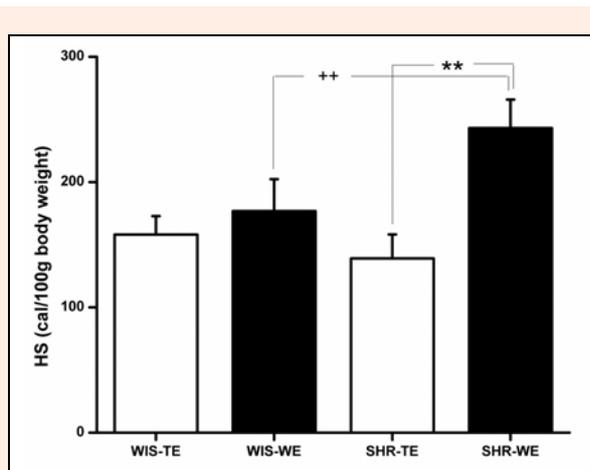


Figure 4. Heat storage (HS) during moderate exercise. Values are expressed as mean \pm SEM, $n = 8$ each group. ** $p < 0.05$: SHR-WE vs. SHR-TE. ++ $p < 0.05$: SHR-WE vs. WIS-WE.

Discussion

In the present study, the control of temperature in hypertensive rats subjected to moderate exercise in warm environment was investigated. In this condition, we observed that SHR presented increased heat production and heat storage that resulted in higher T_b at the end of exercise, associated with a reduction in mechanical efficiency. Additionally, a more intense and faster increase in T_b during moderate exercise in the warm environment was shown by SHR. The lower mechanical efficiency seen in SHR was closely correlated with their higher T_b at point of fatigue in warm environment. Taken together, these results demonstrate that when exercising in a warm environment the SHR exhibit differences in temperature control.

To maintain heat balance, the heat produced by exercising muscles should be counteracted by increased heat dissipation, otherwise, activity results in greater T_b . The increase in T_b that occurs in response to exercise is the consequence of the increase in metabolic rate coupled with impaired heat dissipation, which does not keep pace with heat production (Webb, 1995). Our results showed that SHR have higher values of T_b at the end of moderate exercise in warm environment. Such response is probably the consequence of higher metabolic heat production shown by SHR, evidenced by a higher VO_2 during moderate exercise, associated with no change in heat dissipation through tail vasodilation, as indicated by similar tail temperature.

Impaired heat balance in SHR has been well documented in other models of heat stress (Barney et al., 1999; Wright et al., 1977). These studies have shown that when exposed to warm environment, the SHR survival time is shorter in relation to normotensive animals (Wright et al., 1977). Moreover, it has been evidenced that SHR exhibit sympathoadrenal hyperresponsiveness during exposure to cold environment (Kirby et al., 1999), and in condition of dehydration due to heat stress (Barney et al., 1999). Based on measurements of VO_2 , previous results showed that during continuous exposure to cold heat production was deficient in SHR (Kirby et al., 1999), indicating a possible deficiency in the activation of metabolic heat production in SHR.

On the other hand, the regulation of cutaneous sympathetic outflow, which acts to facilitate heat dissipation in response to body heating, is critical during moderate exercise in the heat to maintain T_b within safe limits (Webb, 1995). Therefore, in states of high thermoregulatory demand, the higher sympathetic activity seen in SHR seems to lead to heat intolerance by decreasing heat dissipation through mechanisms of tail vasodilation (Kellogg et al., 1998; O'Leary and Wang, 1994). It is worthy to note that rats dissipate nearly 25% of their resting heat production through vasodilation of the tail skin (Young and Dawson, 1982), and during exercise this percentage may increase to 40% (Shellock and Rubin, 1984). In the current study, T_{tail} was considered as an index of the tail artery vasomotor tonus, i.e., the magnitude of vasodilation of the tail artery was reflected by tail skin temperature

(Leite et al., 2012; Pires et al., 2007; Shellock and Rubin, 1984). T_{tail} and, hence, cutaneous heat loss are strongly influenced by ambient temperature (Romanovsky et al., 2002).

Our results showed that T_{tail} was already significantly elevated at the beginning of exercise in warm environment. Furthermore, the degree of tail vasodilation reached by both SHR and WIS during exercise in warm environment was higher in comparison to that in temperate environment, indicating amplified blood volume redistribution to the tail for heat dissipation. Nevertheless, T_{tail} was similar between animals in both environmental temperatures, suggesting equal heat dissipation by this route between strains. However, we cannot overlook the contribution of other routes of heat dissipation, which were not measured in this study, such as the dissipation by the paws and the respiratory tract (Gordon, 1993).

In addition, evidence has shown that SHR has a higher ratio of type IIA to I fibers in the soleus muscle, therefore, there is a reduced number of type I fibers inherently more efficient (Damatto et al., 2013; Nagatomo et al., 2009; Novák et al., 2010). These findings can be related to the lower mechanical efficiency exhibited by SHR, which reflects a higher amount of energy to be dissipated as heat. It is recognized that energy efficiency of the body becomes apparent during exercise, when ~20–27% of the energy expended can be used for external work, whereas the remaining energy production is used for homeostasis or dissipated as heat (Brooks et al., 1984). In this sense, SHR had a bigger part of their consumed metabolic energy during exercise to be dissipated as heat. Taking into account that the dissipation of heat from the body is believed to be more important than the control of heat production in the regulation of T_b , particularly during exercise (Lacerda et al., 2005; Leite et al., 2006), such greater demand of heat dissipation may have overridden the limits of tail vasodilation, directly contributing to the higher T_b and increased heat storage observed in SHR-WE. Thus, the overload of heat dissipation capacity as a result of the lower mechanical efficiency may have jeopardized T_b regulation in SHR during exercise in warm environment.

It is clear that elevated T_b and increased rates of body heating and heat storage are considered limiting factors that reduce the central nervous system drive for exercise performance and precipitate feelings of fatigue, thus protecting the brain from thermal damage (Fuller et al., 1998; Lacerda et al., 2005; Leite et al., 2006). In the present study, animals exercising in warm environment had reduced total time of exercise and workload. This was probably a consequence of the imposed higher heat load, which augmented T_b after 4 min of exercise until fatigue compared to temperate environment. In addition, the higher demand for heat dissipation seen in these animals may have shifted a larger amount of blood volume to the tail skin, resulting in blood flow competition with active muscles, contributing to the reduced running performance in warm conditions.

Conclusion

In conclusion, the results indicate that hypertensive rats

exhibit significant differences in temperature control during moderate exercise in warm environment. The increase in heat production and heat storage during moderate exercise in warm environment resulted in aggravated hyperthermia linked with lower mechanical efficiency.

Acknowledgements

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Key points

- The practice of physical exercise in warm environment has gained importance in recent decades mainly because of the progressive increases in environmental temperature;
- To the best of our knowledge, this is the first study to analyze body temperature control of SHR during moderate exercise in warm environment;
- SHR showed increased heat production and heat storage that resulted in higher body temperature at the end of exercise;
- SHR showed reduced mechanical efficiency;
- These results demonstrate that when exercising in a warm environment the hypertensive rat exhibit differences in temperature control.

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