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Possible Biphasic Sweating Response during Short-term Heat Acclimation Protocol for Tropical Natives

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Abstract The aim of the present study was to evaluate the sweat loss response during short-term heat acclimation in tropical natives. Six healthy young male subjects, inhabitants of a tropical region, were heat acclimated by means of nine days of one-hour heat-exercise treatments (40±0°C and 32±1% relative humidity; 50% $\dot{V}O_{2peak}$ on a cycle ergometer). On days 1 to 9 of heat acclimation whole-body sweat loss was calculated by body weight variation corrected for body surface area. On days 1 and 9 rectal temperature (T_{re}) and heart rate (HR) were measured continuously, and rating of perceived exertion (RPE) every 4 minutes. Heat acclimation was confirmed by reduced HR (day 1 rest: 77±5 b·min^{-1}; day 9 rest: 68±3 b·min^{-1}; day 1 final exercise: 161±15 b·min^{-1}; day 9 final exercise: 145±11 b·min^{-1}, $p<0.05$), RPE (13 vs. 11, $p<0.05$) and T_{re} (day 1 rest: 37.2±0.2°C; day 9 rest: 37.0±0.2°C; day 1 final exercise: 38.2±0.2°C; day 9 final exercise: 37.9±0.1°C, $p<0.05$). The main finding was that whole-body sweat loss increased in days 5 and 7 (9.49±1.84 and 9.56±1.86 g·m^{-2}·min^{-1}, respectively) compared to day 1 (8.31±1.31 g·m^{-2}·min^{-1}, $p<0.05$) and was not different in day 9 (8.48±1.02 g·m^{-2}·min^{-1}) compared to day 1 ($p>0.05$) of the protocol. These findings are consistent with the heat acclimation induced adaptations and suggest a biphasic sweat response (an increase in the sweat rate in the middle of the protocol followed by return to initial values by the end of it) during short-term heat acclimation in tropical natives. *J Physiol Anthropol 25(3): 215–219, 2006* http://www.jstage.jst.go.jp/browse/jpa2
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Introduction

Sweating is the main via for heat dissipation during exercise performed in hot environments and is elicited by both local factors and stimuli from the pre-optic area of the hypothalamus, which receives and integrates afferent information from central and peripheral thermal receptors. The present study focused on tropical native's whole-body sweat loss response during short-term heat acclimation.

Heat acclimation is known to reduce resting and exercise heart rate (HR), internal temperature and the threshold for the onset of sweating (Buono et al., 1998; Patterson et al., 2004; Machado-Moreira et al., 2005). These centrally mediated physiological adaptations reduce heat strain and increase exercise tolerance in hot environments. Armstrong and Stoppani (2002) proposed that the brain's homeostatic control regions are modified during successive days of heat acclimation, resulting in an improved ability to exercise in a hot environment.

Recent investigations have described heat acclimation as a biphasic process, in which an initial transient phase is characterized by accelerated autonomic activity to rapid control heat dissipation, followed by a reduction of this activity due to improved effector organs efficiency (Horowitz et al., 1996; Horowitz, 1998). Some studies have shown possible evidence for the biphasic process during heat acclimation that can be found in changes in plasma volume (Wyndham et al., 1968; Shapiro et al., 1981), extracellular volume (Wyndham et al., 1968), plasma aldosterone concentration (Armstrong et al., 1989) and in cardiac output and stroke volume responses (Wyndham et al., 1976). Nevertheless, the possible biphasic sweat rate behavior during short-term heat acclimation has not been measured.

Many authors have described sweating differences between tropical natives and non-tropical natives (Thomson, 1954; Kuno, 1956; Fox et al., 1974; Hori et al., 1976; Nguyen and Tokura, 2003). These studies have described how tropical natives sweat less for a given stimulus in comparison to non-tropical natives; however, no differences in internal temperature were noticed. It has been suggested that tropical natives have, therefore, a more efficient thermoregulatory system (Hori, 1995). Furthermore, tropical natives might respond in a different manner to heat acclimation (Hellon et al., 1956; Wyndham et al., 1952) in comparison to those who are not heat acclimatized.

Thus, the aim of the present study was to evaluate the sweat loss response during short-term heat acclimation in tropical natives.

Methods

Subjects

Six healthy and physically active young males (21±3 years; 72.56±7.82 kg; 175±5 cm; 1.9±0.1 m^2 of body surface area (Dubois and Dubois, 1916); $\sum_{skinfolds}$ 65±16 mm), inhabitants of a tropical region (latitude 19.5°S and longitude 43°W), underwent a heat acclimation protocol during October and November of 2003 (Oct: 22.6±2.4°C; Nov: 22.6±2.1°C). All the procedures were approved by the Human Ethics Research Committee of the Federal University of Minas Gerais, and subjects provided written informed consent.

Procedures

Heat acclimation was induced using nine treatment exposures (exercise + heat stress). The subjects exercised for one hour on a cycle ergometer (Monark, 824E, Varberg, Sweden) at 50% $\dot{V}O_{2peak}$ in a hot and dry environment (40±0°C, 32±1% relative humidity and 0.0 m/s of air velocity). The work rate during exercise was calculated from a $\dot{V}O_{2peak}$ test (ACSM, 2000) conducted in the same hot and dry environment 4 days prior to the commencement of the acclimation protocol. The test consisted of a graded exercise (cycling), starting with 50W of work rate followed by 25W increases each 2 minutes until the onset of volitional fatigue. All procedures took place in a climatic chamber (Russels Technical Products, WMD 1150-5, Holland, MI, USA).

Two days before and 2 days after the heat acclimation protocol a $\dot{V}O_{2peak}$ test (ACSM, 2000) was conducted in a temperate environment (21±0°C; 64±1% relative humidity; and 0.0 m/s of air velocity) to access training status.

To calculate sweat loss, the subjects voided and were weighed (Filizola® MF-100 scale, precision of 0.02 kg, São Paulo, SP, Brazil) before and after exercise in days 1 to 9 of heat acclimation. Body weight variation, uncorrected for respiratory and metabolic losses, was divided by the time between measurements and corrected for body surface area. In addition, HR (Polar Vantage NV, Kempele, Finland) was measured continuously and Borg's rating of perceived exertion (RPE) was measured every 4 minutes. On the 1st and 9th days of heat acclimation rectal temperature (T_{re}) was measured using a disposable probe (YSI, series 4400–4491-E, Yellow Springs, OH, USA) inserted 10–12 cm beyond the anal sphincter. Besides, the ratio whole body sweat rate/rectal temperature variation was calculated.

Hydration status

The volunteers were asked to drink 500 ml of water 2 hours before the experiment (ASCM, 1996). During the first heat acclimation exposure water ingestion was provided *ad libitum* and equal volumes were given during the subsequent exposures (Houmard et al., 1990; Machado-Moreira et al., 2005). The subjects were considered eu-hydrated (urine specific gravity (G_u)<1030; Armstrong, 2000) before and after all trials. G_u was measured by means of a portable refractometer (model JSCP-Uridens, São Paulo, SP, Brazil) previously calibrated with distilled water. The volunteers always wore shorts, socks and tennis shoes.

Statistical analysis

A one-way ANOVA with repeated measures followed by the *post-hoc* Bonferroni t-test was performed to compare the sweat loss among heat-exercise exposures. A two-way ANOVA with repeated measures followed by the *post-hoc* Student Newman Keuls test was performed to compare HR and T_{re} between days 1 and 9. A paired student *t-test* was performed to compare the ratio sweat loss/ΔT_{re} between days 1 and 9. RPE medians were compared using the Wilcoxon test. Alpha was set at 5% for all analyses. Data are present as mean±SD.

Results

Heat acclimation was confirmed by reduced HR (day 1 rest: 77±5 $b \cdot min^{-1}$; day 9 rest: 68±3 $b \cdot min^{-1}$; day 1 final exercise: 161±15 $b \cdot min^{-1}$; day 9 final exercise: 145±11 $b \cdot min^{-1}$, $p<0.05$), reduced RPE (13 vs. 11, $p<0.05$) and lower T_{re} (day 1 rest: 37.2±0.2°C; day 9 rest: 37.0±0.2°C; day 1 final exercise: 38.2±0.2°C; day 9 final exercise: 37.9±0.1°C, $p<0.05$) (Table 1).

Table 1 also shows that there were no differences in rectal temperature variation nor in sweat loss/ΔT_{re} between days 1 and 9.

As shown in Fig. 1, sweat loss was higher in days 5 and 7 (9.49±1.84 and 9.56±1.86 $g \cdot m^{-2} \cdot min^{-1}$, respectively) compared to day 1 (8.31±1.31 $g \cdot m^{-2} \cdot min^{-1}$; $p<0.05$), however, it was not different in day 9 (8.48±1.02 $g \cdot m^{-2} \cdot min^{-1}$) compared to day 1 ($p>0.05$) of the protocol. These results can be interpreted as a biphasic sweating response.

Table 2 presents the results of the $\dot{V}O_{2peak}$ tests conducted before and after the heat acclimation protocol in a temperate

Table 1 Resting and final exercise rectal temperatures (T_{re}), rectal temperature variation (ΔT_{re}) and the ratio whole-body sweat loss/rectal temperature variation (Sweat loss/ΔT_{re}) on days 1 and 9 of heat acclimation. (*) Smaller than day 1, $p<0.05$.

	T_{re} rest (°C)	T_{re} final exercise (°C)	ΔT_{re} (°C)	Sweat loss/ΔT_{re} ($g \cdot m^{-2} \cdot min^{-1} \cdot °C^{-1}$)
Day 1	37.2±0.2	38.2±0.2	0.94±0.16	0.9±0.2
Day 9	37.0±0.2*	37.9±0.1*	0.88±0.27	0.9±0.3

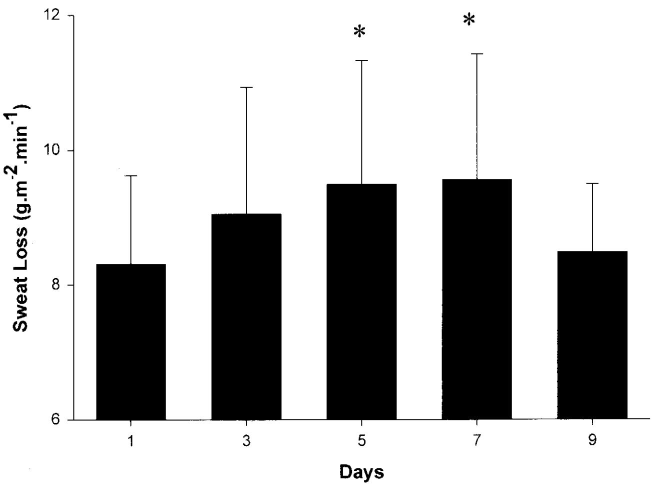

Fig. 1 Sweat loss in days 1, 3, 5, 7 and 9 of the heat acclimation protocol. (*) $p<0.05$; different from day 1.

Table 2 Physiological variables measured during the graded exercise test before and after heat acclimation. $\dot{V}O_{2peak}$: aerobic capacity; HR_{max}: maximal heart rate; W_{max}: maximal power; RPE: rating of perceived exertion; TET: total exercise time.

	$\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	HR_{max} (bpm)	W_{max} (Watts)	Final RPE (min)	TET
Before	54±6	194±10	300±42	20	22±4
After	53±5	189±7	296±49	20	22±4

environment. As shown, none of the measured parameters changed after the protocol.

Discussion

The main and original finding of the present study was the increased sweat loss in the middle of the heat acclimation protocol (days 5 and 7) and its returning to initial values by day 9 in tropical native subjects. This result is in agreement with the model proposed by Horowitz (1998), which suggests a biphasic process during acclimation as a consequence of a transient mechanism for heat dissipation in the early days of heat acclimation. According to this model, once acclimation is achieved, the evaporation rate can be maintained probably due to a reduction in sweat electrolyte concentration (Kuno, 1956; Fox et al., 1974; Hori et al., 1976) and higher skin temperature (Thomson, 1954; Nguyen and Tokura, 2002) despite a reduction in sweat rate; i.e. increased efficiency. Therefore, the sweating augmentation observed in the middle of the heat acclimation protocol could be interpreted as an emergency mechanism for maintenance of thermal homeostasis, showing a biphasic sweating response. However, our interpretation is based on the difference between day 1 and days 5 and 7 and the non-difference between days 1 and 9. Hence, the biphasic sweating response during heat acclimation in tropical natives must be better investigated to confirm our findings.

The absence of difference in the sweat loss between days 1 and 9 could have been due to the short period of heat acclimation, since Armstrong and Maresh (1991) suggested that the sweat rate improvement takes place at 8 to 14 days of continuous heat exposure. However, in the present study, it seems that there was a transient enhancement in the sweat loss and a return to pre-heat-acclimation values, showing that there was sufficient time for adaptation in our subjects.

Some studies have shown other possible evidence for a biphasic process during heat acclimation that can be found in changes in plasma volume (Wyndham et al., 1968; Shapiro et al., 1981), extracellular volume (Wyndham et al., 1968), plasma aldosterone concentration (Armstrong et al., 1989) and in cardiac output and stroke volume responses (Wyndham et al., 1976). However, the present study is the first one to find such a response for sweating.

It is important to note that short-term and long-term heat acclimation may induce quite different responses. Also, the subjects' previous exposure to heat should lead to different responses. It is well established that short-term heat acclimation induces a higher sweat rate (Wyndham, 1967; Nadel et al., 1974; Nielsen et al., 1993), higher sweat gland flow and no change in the number of activated sweat glands (Peter and Wyndham, 1966). In contrast, tropical natives, who have presumably been subjected to long-term heat acclimatization, have been shown to have a lower sweat rate (Wyndham et al., 1952; Thomson, 1954; Kuno, 1956; Fox et al., 1974; Hori et al., 1976; Nguyen and Tokura, 2003), reduced sweat electrolyte concentration (Kuno, 1956; Fox et al., 1974; Hori et al., 1976), a higher number of activated sweat glands (Kuno, 1956) and higher skin temperature (Thomson, 1954; Nguyen and Tokura, 2002). Therefore, our findings contradict the current belief that short-term heat acclimation enhances the sweat rate.

In the present study, we investigated tropical natives. Hence, the maintenance of sweat loss without disturbing thermal homeostasis following heat acclimation, as noticed in our data, can be seen as an efficient mechanism of adaptation to heat. It is interesting to note that Mitchell et al. (1976) heat acclimated subjects from a temperate region for ten days and observed a sweating augmentation after heat acclimation. However, even though the evaporated rate increased by approximately 10%, the unevaporated rate increased almost 200%, which can be interpreted as wasteful sweating. Those findings have been corroborated by others (Avellini et al., 1980).

It is not known whether ethnic differences in heat adaptation are due to genotypic or phenotypic adaptation. Kuno (1956) has suggested that the number of activated sweat glands is dependent on the environmental heat stress that one is subjected to before the age of two, and Hori (1995) proposed that due to repeated heat exposures, the sweating center becomes habituated to stimulation by heat, reducing its response. These studies support a phenotypic adaptation view. On the other hand, it is possible that chronic exposure to heat stress for many generations might have selected genetic adaptations that allow more efficient thermoregulation.

Another result of the present study was that similar ΔT_{re} induced similar sweat loss after acclimation. Although many studies have reported increases in sweat production after heat acclimation (Taylor, 1986), the effect of ΔT_{re} on whole-body sweat loss after a short-term heat acclimation protocol has not been well investigated. In the present study ΔT_{re} did not change after heat acclimation as already found by others (Buono et al., 1998). In addition, it was found that there was similar sweat loss/ΔT_{re} showing that the whole-body sweating sensitivity did not change after the acclimation process, although others have shown increased local sensitivity (Patterson et al., 2004). Probably, the sweat rate sensitivity increases in some body regions and decreases in others resulting in a redistribution of the sweat across body regions, which leads to higher efficiency.

In the present study the use of the same work rate throughout the heat acclimation protocol might have been a limitation because of the possible training effect and, therefore, the same absolute work rate could have represented a lower relative work rate, which would elicit smaller strain (Taylor, 1986; Havenith, 2001). However, the variables measured in the $\dot{V}O_{2peak}$ tests conducted before and after heat acclimation were not different. Studies that have employed similar heat acclimation protocols also did not report a training effect (Nadel et al., 1974; Houmard et al., 1990).

In conclusion, the results of the present study showed a possible biphasic sweating response during a short-term heat acclimation protocol in tropical natives.

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