

The effect of passively induced heat acclimation on its symptoms

Marius Brazaitis^{1*},

Ieva Lukošūtė-Stanikūnienė¹,

Albertas Skurvydas¹,

Laura Daniusevičiūtė²,

Dalia Mickevičienė¹

¹ *Laboratory of Human Motorics,
Department of Applied
Physiology and Physiotherapy,
Lithuanian Academy
of Physical Education,
Sporto 6,
LT-44221 Kaunas,
Lithuania*

² *Physical Education and Health Centre,
Department of Physical Education,
Kaunas University of Technology,
Kaunas, Lithuania*

Thirteen healthy subjects (7 males and 6 females) were passively acclimated by means of 7 sessions with passive lower body heating repeated every other day for two weeks. On every successive occasion, the subjects were sitting for 45 min immersed up to the waistline in a water bath at ~44 °C (air temperature 23 °C, rh 40%). Rectal (T_{re}), skin (T_{sk}) temperatures at three sites (back, thigh and forearm) and body mass were measured before and after passive heating. Heart rate (HR), ratings of thermal sensation (RTS) and comfort (RTC) were collected every 5 min during passive heating. During the first and the last HA session, participants performed a 2-min muscular voluntary contraction (MVC) of knee extensors. After acclimation, both the resting T_{re} prior to heat exposure and the final temperature measured at the end of passive heating were significantly reduced, but had no effect on T_{sk} . Heat acclimation had a significant effect on increased sweating and decreased HR, RTS, RTC and the physiological stress index. Lower body heating resulted in a greater torque fatigue compared to the thermoneutral condition during 2-min MVC. Following HA, MVC torque was uninfluenced. We have concluded that passively induced heat acclimation is reliable for lowering T_{re} at rest. Although passively induced heat acclimation for two weeks improved the symptoms of acclimation, it did not change MVC torque fatigue. Changes in T_{re} , HR and RTC during passive heating are closely related to the shift of T_{re} at rest, following heat acclimation.

Key words: rectal temperature, heat stress, symptoms of acclimation, thermoregulation

INTRODUCTION

It is well known that repeated exposure of the body to heat stress improves the tolerance level while physically working under stress conditions in a natural environment [1, 2]. The physiological response to heat acclimation (HA) includes an increase in sweating and a decrease in heart rate (HR), core temperature (T_{co}), skin temperature (T_{sk}), perceived exertion [3], rectal temperature (T_{re}) at rest (during exercise-induced HA) [4] and oxygen consumption at a given work rate [1].

There are many studies to show successful HA with an significant reduction in resting T_{re} after repeated exercise-

induced heat exposure [4–6]. However, we have not come across any publications showing that reduction in T_{re} at rest is possible during repeated passive body heating following HA. Recently, it has been concluded that a repeated physical exercise is the main stimulus for lowering the resting T_{co} following acclimation [5–8]. In contrast, it is well established that a repetitive exposure to passively induced heat usually results in acclimation [9–12]. For example, Henane and Valatx [10] have shown that during a repeated passively induced heat exposure over 9 days in a climatic chamber there was a marked improvement in sweating capacity and sweating performance, but no improvement in resting T_{co} was reported. The lower resting T_{re} may simply allow an acclimated individual to exercise for a longer period of time in the heat before a critical temperature is reached [5, 13].

* Corresponding author. E-mail: kku712@yahoo.com

Additionally, that in most studies the final T_{co} was higher during exercise-induced HA protocols and ranged between 38.1 and 39.9 °C, while during passively induced HA protocols it did not exceed 38.2 °C. Changes in T_{co} seem to be a reliable indicator of physical performance in the heat as well as in HA. It is possible, however, that a repeated stimulus of critical T_{co} (~39.5 °C) induced by passive high heat stress exposure plays a separate role in HA. It is not clear, however, whether reduction in resting T_{re} can be attained by repeated passive heating following a two-week acclimation.

Many heat-strain indexes for evaluating human physiological strain were introduced during the last century [14, 15]. More recently, Moran et al. [16] have introduced probably a universal physiological strain index (PSI) for humans, based on changes in HR and T_{re} during exposure to exercise heat stress, which can be used on-line measurements. Meanwhile, only one study in exercise-heat-stressed rats used PSI following acclimation [17]. No data are currently available regarding PSI improvement in humans following passively induced HA, therefore, we decided to examine it in more detail.

It is well known that high environmental temperatures are associated with impaired exercise performance and accelerated muscular fatigue [18]. The mechanisms limiting exercise performance in hyperthermia are not well understood. Accelerated fatigue in isometric exercise could be associated with the metabolic inhibition of muscle contraction as the ATP turnover accelerates at high temperatures [19, 20]. However, an elevated core temperature plays an important role as well [18]. It is, however, possible that improved symptoms of passively induced HA, especially in the case of a lowered core temperature of the body, can play a separate role in maximal voluntary contraction torque during sustained exercise in hyperthermia.

There were three main hypotheses: 1) T_{re} at rest should decrease following a two-week heat acclimation induced by repeated passive heating; 2) therefore, we expected that passively induced HA would reduce MVC fatigue while performing exercise in hyperthermia; 3) a decrease in T_{re} at rest may be related to changes in different symptoms (HR, PSI, T_{re} , loss of body mass, ratings of thermal comfort and sensation) of heat acclimation induced by passive heating. However, no clear evidence existed to support these hypotheses, and we decided to examine them in more detail.

MATERIALS AND METHODS

Subjects

Thirteen healthy subjects (7 males and 6 females) participated in the study which was approved by the Human Research Ethics Committee (Kaunas University of Medicine, Protocol No. 150/2008; BE-2-60, Kaunas, Lithuania). All female participants had regular ovulatory menstrual cycles

and followed “normal” hormone fluctuations throughout the menstrual cycle. Regular ovulatory women have an increase in T_{re} of approximately 0.4 °C after ovulation, which is sustained throughout the luteal phase [21, 22]. Additionally, every woman that participated in this study was encouraged to measure T_{re} every day following overnight for one month. Using this method, all the females participated in the study on their early-follicular phase when estrogen and progesterone concentrations are low and the body core temperature is at the lowest level of the menstrual cycle [22]. The findings of this study revealed that there was no significant gender-related difference in body temperatures following heat acclimation, suggesting that similar mechanisms are involved in both genders. Based on this assumption, males and females were analysed as one group. The age, height and body mass of the subjects in this group were (mean (SD)) 22.8 (3.1) yr, 1.77 (0.4) m height, 71.7 (9.7) kg mass. Written informed consent was obtained from the volunteers prior to their participation in this study. All the volunteers might be considered as physically active as they took part in recreational activities two or three times per week.

Heat acclimation protocol

The protocol consisted of 7 sessions (HA1; HA2;... HA7, respectively) of passive lower body heating, which were repeated every other day for two weeks. Testing was conducted from December to March to limit the initial heat acclimation through casual exposure to high ambient temperatures. A passive heating was conducted indoors at the same time of the day (10 : 00 am to 12 : 00). The subjects could not take any food at least 12 hours prior to the experiment, but they could drink water when they wanted. Subjects were also instructed not to drink any liquid 60 min before the first body mass measurement was taken. This was done in order to standardize the state of hydration and the feeling of thirst. The experiment was carried out at 23 °C (room temperature) and relative humidity (rh) = 40%. On arrival to the laboratory, subjects were weighed in the nude and after light dressing (T-shirt, shorts and socks) were asked to rest in a chair for 30 min. Then the rectal and skin temperature as well as HR stabilization and the control measurements of T_{re} , T_{sk} and HR were collected. Then the volunteers sat for 45 min immersed up to the waistline in the water bath at ~44 °C as described previously by Sargeant [23]. During this procedure, HR, RTS and RTC were measured every 5 min. Within ~3 min after leaving the bath, the volunteers were towed dry and the temperature measurements were repeated again. After light dressing, the subjects were asked to rest in the chair. The nude body mass was recorded within 30 min after exit from the water bath when body sweating ended. The same experimental procedure was repeated every other day during two weeks.

Measurements

Core body and skin temperature. Rectal temperature (T_{re}) and skin temperature (T_{sk}) were measured before and at the end of passive heating. T_{re} was measured using a thermocouple (Rectal Probe, Ellab, Hvidovre, Denmark) inserted to a depth of 12 cm past the anal sphincter as recommended [24]. T_{sk} was measured at three sites: back, thigh and forearm (DM852, Ellab, Hvidovre, Denmark), and the average was calculated using the following formula [25]:

$$T_{sk} = 0.5T_{back} + 0.36T_{thigh} + 0.14T_{forearm}$$

The mean body temperature (T_b) was calculated using a T_{re} and T_{sk} temperature weighted equation [26]:

$$T_b = 0.65T_{re} + 0.35T_{sk}$$

Cardiovascular responses. HR was measured before and every 5 min during passive heating. HR was measured with a heart rate monitor (Polar Electro Inc., Lake Success, N. Y.).

Perceptual measurements. Ratings of thermal sensation (RTS) and comfort (RTC) for the whole body were answered every 5 min using scales modified from Gagge et al. [27]. Thermal sensation ranged from 0 (unbearably cold) to 10 (unbearably hot), with 5 being neutral. Thermal comfort ranged from 1 (comfortable) to 5 (very uncomfortable), with increments of 0.5.

Physiological stress index (PSI). To evaluate heat stress on a universal scale of 0–10 and to overcome the limitations of continually getting higher values during rest or recovery periods, we used an index that enabled us to calculate the physiological strain online at any time. The index was based on the same maximal rise values for T_{re} and HR (according to the Human Use Review Committee Limits). Thus, the following normalized physiological stress index is suggested [16]:

$$PSI = 5 (T_{ref} - T_{re0}) \times (39.5 - T_{re0})^{-1} + (HR_t - HR_0) \times (180 - HR_0)$$

The measurements for PSI were taken before (T_{re0} and HR_0) and at the end (45 min) of passive heating (T_{ret} and HR_t), respectively. T_{re} and HR, which depict the combined load of the cardiovascular and thermoregulatory systems, were assigned with the same weight by using a constant of 5. Thus, the index was scaled to a range of 0–10 within the limits of the following values: $36.5 \leq T_{re} \leq 39.5$ °C and $60 \leq HR \leq 180$ beats/min.

State of hydration. The body mass of each subject was determined by taking their nude mass before and 30 min after the passive heating (TBF-300, Tanita UK Ltd. Philpots

Close, UK). The state of hydration was calculated by the formula: Loss of Body Mass (LBM) (%) = (body mass before passive heating – body mass after passive heating) / body mass before passive heating × 100 per cent.

Exercise protocol

The isometric torque of knee extensor muscles was measured using an isokinetic dynamometer (System 3; Biodex Medical Systems, Shiley, New York). The subjects sat upright in the dynamometer chair with the knee joint positioned at an angle of 120 degrees (180 degrees – a full knee extension). Afterwards, the subjects were asked to perform maximal voluntary contraction of the knee extensors for 2 min (MVC 2-min) with the maximal effort. The fatigue index (FI) of MVC torque was calculated as follows: FI = (beginning 2-min MVC – end 2-min MVC) / beginning 2-min MVC × 100 per cent.

Research design

The subjects were required to attend the laboratory on nine separate occasions. The initial visit involved familiarization with experimental procedures and equipment. A week later, the participants returned to the laboratory for the experimental session. The subjects were required to exercise in a thermoneutral environment (see the section “Exercise protocol”) at 23 °C. A week after the last visit, participants arrived to the laboratory and the HA protocol began (see the section “Heat acclimation protocol”). The experimental sessions were performed on day 1 (heat-acclimation sessions 1 (HA1)) and day 14 (heat-acclimation sessions 7 (HA7)) of HA. Initially, body mass, T_{sk} , T_{re} and HR were measured as described above. Afterwards, however, a volunteer sat for 45 min immersed up to the waistline in the water bath at a ~44 °C. Immediately after this procedure, the volunteer was towelled dry and the temperature measurement was repeated. Within ~3 min after leaving the bath, the volunteers were seated in the chair of the isokinetic dynamometer, dressed in the sports wear. Without delay, the experiment was terminated by a 2-min MVC. Nude body mass was recorded within 30 min after leaving the water bath.

Statistical analysis

Data are presented as mean values and the standard deviation of the mean. One-way (heat-acclimation (HA1; HA2;... HA7)) repeated-measures analysis of variance (ANOVA) was used to analyse changes in PSI and LBM. A two-factor (heat-acclimation (HA1 and HA7) × time (5, 10, 15... 45 min; the beginning and the end of MVC 2-min)) ANOVA was used to compare changes in HR, RTS, RTC, MVC torque and (heat-acclimation (HA1; HA2;... HA7) × time (before and after passive heating)) to analyse differences in temperature (T_{re} , T_{sk} , and T_b).

Table 1. The changes in different symptoms following heat-acclimation

ΔT_{re} at rest, (°C)	Δ PSI	$\Delta\Delta T_{re}$ (°C)	$\Delta\Delta$ HR, (b./min ⁻¹)	$\Delta\Delta$ RTC, (points)	$\Delta\Delta$ RTS, (points)	$\Delta\Delta$ LBM, (%)
-0.3 (0.2)	-1.0 (0.7)	-0.02 (0.3)	-5.3 (13.6)	-0.5 (1.2)	-0.2 (1.6)	-0.4 (0.2)

Values are means \pm SD. T_{re} , rectal temperature; PSI, physiological stress index; HR, heart rate; RTC, rating of thermal comfort; RTS, rating of thermal sensation; LBM, loss of body mass. For delta (Δ) and delta–delta ($\Delta\Delta$) explanations see section “statistical analysis”.

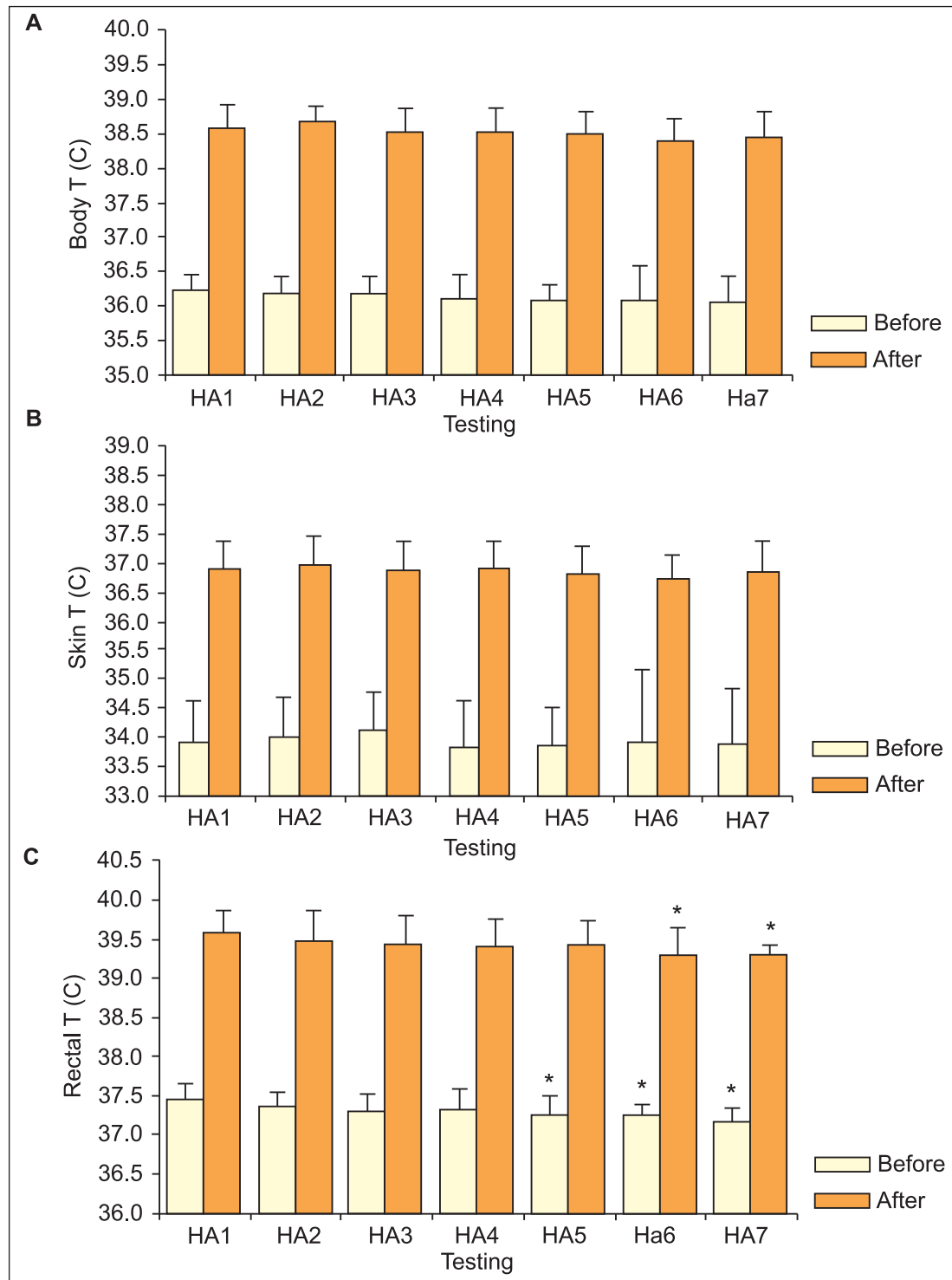


Fig. 1. Change in resting and final body (A), skin (B) and rectal (C) temperature in each of the 7 sessions of heat acclimation (HA). * Significant difference compared with the value of HA1 session, $p < 0.05$. Values are means \pm SD

When a significant main effect was found, a *post hoc* test with the Bonferroni correction was applied to locate the difference. For all statistical analyses, the 0.05 level of significance was used.

In order to evaluate the relationship between changes in different symptoms of HA (Table 1) after passive body heating, the Pearson correlation coefficient was calculated. The decrease of resting T_{re} (ΔT_{re}) that occurred during the first and the last acclimation sessions was calculated by subtracting the resting T_{re} at HA1 from the T_{re} at HA7 sessions before passive heating. The change in PSI (ΔPSI) was calculated by subtracting the PSI at HA1 from the PSI at HA7. The changes following HA in different symptoms (HR, PSI, T_{re} , RTS, RTC and LBM) were calculated as follows: $\Delta \Delta$ symptom of HA = HA1 (value before – value after passive heating) – HA7 (value before – value after passive heating).

RESULTS

Body temperatures in all experimental sessions increased significantly ($p < 0.05$) after passive lower body heating. However, there were no significant changes in T_b (Fig. 1A) and T_{sk} (Fig. 1B) ($p > 0.05$) during the 7 sessions of heat exposure, but T_{re} ($p < 0.05$) lowered from HA5 in pre- and HA6 in post-passive heating measurements. Heat acclimation (HA1 compared to HA7) resulted in a decrease by ~ 0.3 °C in T_{re} during pre- and post- passive heating measurements (Fig. 1C). No significant reduction in body temperatures was found between genders following HA ($p > 0.05$).

The relative LBM during passive lower body heating increased significantly ($p < 0.05$) from the HA5 experimental session (Fig. 2). Besides, there was a significant ($p < 0.05$) effect of HA on PSI which decreased from the HA6 session (Fig. 3).

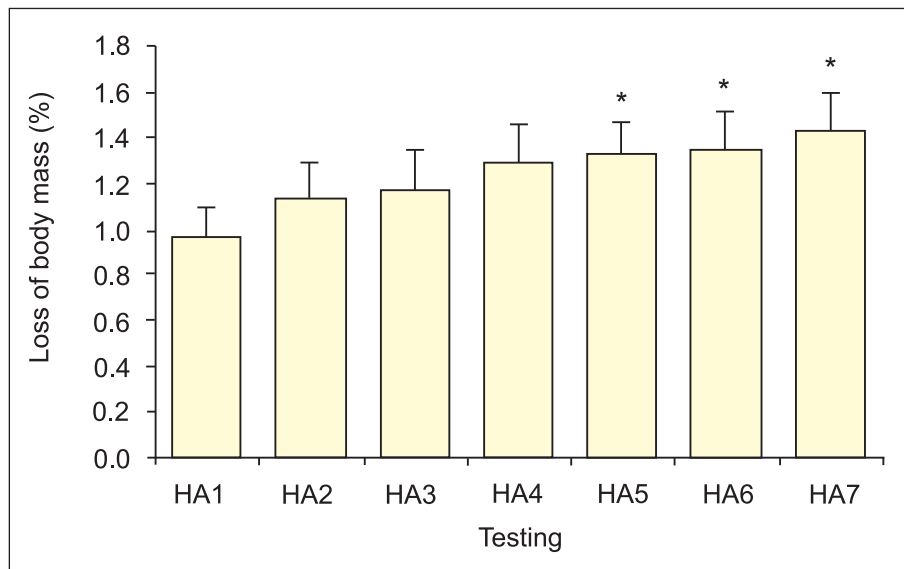


Fig. 2. Change in relative loss of body mass in each of the 7 sessions of heat acclimation (HA).

* Significant difference compared with the value of HA1 session, $p < 0.05$. Values are means \pm SD

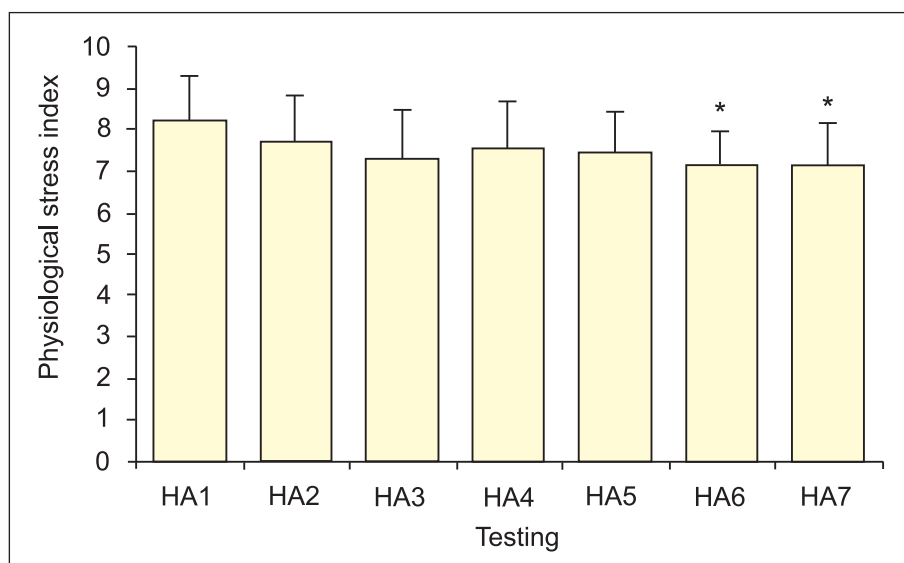


Fig. 3. Change in physiological stress index in each of the 7 sessions of heat acclimation (HA).

* Significant difference compared with the value of HA1 session, $p < 0.05$. Values are means \pm SD

The effect of HA on RTS and RTC during passive heating is presented in Fig. 4. There was a significant increase in RTS and RTC by time during passive heating in HA1 and HA7 sessions ($p < 0.05$). The two-way ANOVA revealed that HA reduced significantly the RTS and RTC ($p < 0.05$). The RTS changed from “a little warm” to “very hot” in HA1 and from “neutral and a little warm” to “hot” in HA7 (Fig. 4A). The RTC changed from “comfortable” and “a little uncomfort-

able” to “uncomfortable” and “very uncomfortable” in HA1 and from “neutral” to “uncomfortable” in HA7 (Fig. 4B).

The effect of HA on HR during passive heating is shown in Fig. 5. There was a significant increase in HR by time during passive heating in HA1 and HA7 sessions ($p < 0.05$). The two-way ANOVA revealed HA to reduce HR ($p < 0.05$).

The effect of HA on knee extension peak torque during a 2-min MVC is presented in Table 2. The values of

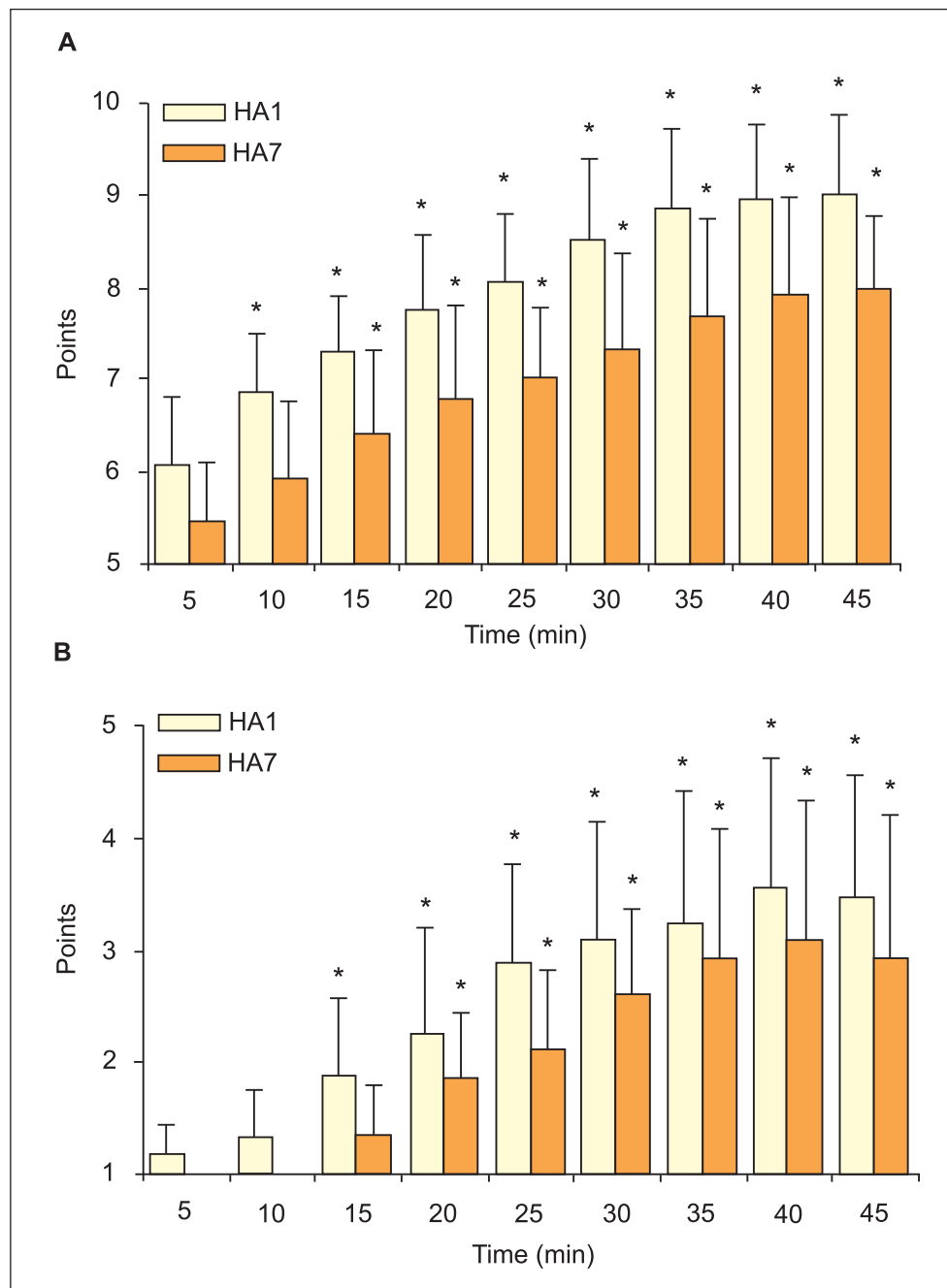


Fig. 4. Change in ratings of thermal sensation (A) and comfort (B) during passive heating in heat-acclimation sessions 1 (HA1) and 7 (HA7). * Significant difference compared with the value established at 5-min time point of passive heating, $p < 0.05$. Values are means \pm SD

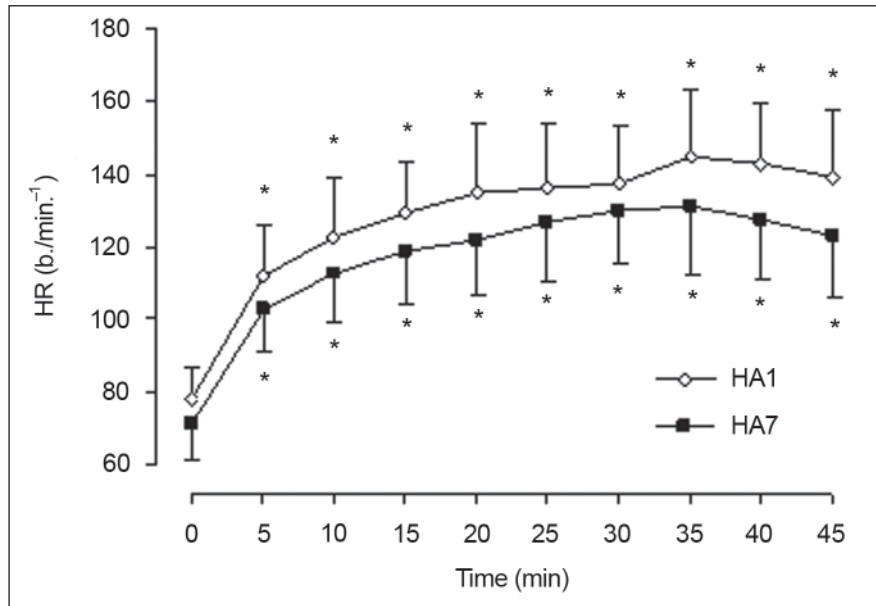


Fig. 5. Change in heart rate (HR) during passive heating in heat-acclimation sessions 1 (HA1) and 7 (HA7). Values for time point 0 are taken from initial measurements in these experiments. * Significant difference compared with the initial value, $p < 0.05$. Values are means \pm SD

peak torque decreased ($p < 0.05$) during a 2-min MVC in CON, HA1 and HA7 experiments. Those decreases were significantly greater in HA1 and HA7 compared to CON ($p < 0.05$); however, the differences between HA1 and HA7 sessions were not significant.

There was a significant correlation between ΔT_{re} at rest and $\Delta \Delta T_{re}$ ($r = 0.75$; $P < 0.01$), as well as between ΔT_{re} at rest and $\Delta \Delta HR$ ($r = 0.60$; $p < 0.05$) following acclimation. We have established a significant relationship between ΔT_{re} at rest and $\Delta \Delta RTC$ ($r = 0.64$; $p < 0.05$). There was no significant relationship between ΔT_{re} at rest and $\Delta \Delta LBM$ ($r = 0.29$), $\Delta \Delta RTS$ ($r = -0.12$) as well as ΔPSI ($r = -0.02$).

DISCUSSION

The most interesting finding is that the stimulus of critically elevated T_{re} during repeated passively induced hyperthermia for two weeks is reliable for T_{re} reduction at rest. In the present study, we observed that the 7-session HA protocol repeated every other day for two weeks was suc-

cessful, as was evident from a significant decrease in T_{re} in pre- and post- passive heating measurements, PSI, increase in sweating rate and HR, as well as the improved rating of thermal sensation and comfort following the acclimation. Although the passively-induced heat acclimation for two weeks improved the symptoms of acclimation, it did not change torque fatigue during 2-min MVC in hyperthermia.

The lowering of the resting core temperature indicates an improvement of HA [4–6]. The significant ~ 0.3 °C decrease in resting and final T_{re} following acclimation is in agreement with other works [4, 5, 28, 29]. The reported reduction of the resting and final T_{co} can range from 0.2 to 0.8 °C, which might be influenced by different exercise-induced HA protocols and climatic conditions [5, 6]. It seems that a repeated passively heat stress elevated to the critical level can reduce the resting T_{re} following acclimation, as well as it was reported in exercise-induced heat exposure [5–8]. However, it should be noted that our study used a passive method with two days' rest interval to induce HA, while others applied daily exercise-induced heat exposure

Table 2. The changes in torque during 2-min MVC in control (CON) experiment and experiments with body heating in heat-acclimation sessions 1 (HA1) and 7 (HA7)

	CON	HA1	HA7
MVC torque (N·m)	162.6 (10.1)	176.7 (9.8)*	174.5 (8.7)*
FI of MVC (%)	69.6 (4.0)	78.6 (2.2)*	76.7 (2.5)*

Values are means \pm SD. FI of MVC – fatigue index of maximal voluntary contraction torque; MVC – maximal voluntary contraction.

* Significant difference compared to CON value, $p < 0.05$

protocols. It could be concluded, however, that the passively induced heat stress stimulus is reliable for HA improvement even with a 48-hour rest duration inbetween the heating sessions. Simply the effect of a decrease in T_{re} at rest after HA would be to reduce T_{re} at any given point during exercise in the heat, if the same amount of heat is generated [5, 13]. Specifically, precooling studies [30, 31] that lowered resting T_{co} by $\sim 0.2\text{--}0.3$ °C have shown this to have a beneficial effect on exercise performance. Several researchers have suggested that a reduction in the resting metabolic rate is responsible for reducing the resting T_{re} with HA [32, 33]. It was established that thyroid function decreased with HA [34].

There is some evidence that a reduction in T_{re} at rest significantly correlates with the final T_{re} during exercise-induced heat exposure following HA, suggesting that the final level of the core temperature depends on its initial value [4–6]. However, we failed to find any other study to describe the relation in changes between T_{re} (ΔT_{re}) during exposure to passively induced heat and T_{re} (ΔT_{re}) at rest following HA (Table 1). Thus, our results showed a strong correlation between these two parameters. This result suggests that changes in T_{re} during passive heating depend on the shift of T_{re} at rest following HA.

Additionally, our results revealed no significant difference between males and females in body temperature measurements. This indicates a similar HA effect on both genders. However, our results disagree with data of Kampmann et al. [6]. They showed no significant reduction of resting T_{co} and only a small lowering of the final level in females compared to males following HA. However, the main difference between these two studies is that they did not control the females' menstrual cycle during the study, while we did. It is well established, however, that hormonal fluctuation throughout the menstrual cycle, causing an increase of resting core temperatures by about 0.4 °C during the transition from the follicular to the luteal phase [35], could have interfered with the change of the resting and final T_{re} during HA; also, using oral contraceptives lead to a change of the core temperature between 0.1 and 0.3 °C [36, 37]. Furthermore, it was reported that the relationship between the change in resting T_{co} and the change in final T_{co} did not differ between males and females. Therefore, it was suggested that similar mechanisms (reduced metabolism and increased heat dissipation at rest [35]) influence both genders.

In our study, HR at the 45th min of passive heating dropped significantly ($12 \text{ beats} \cdot \text{min}^{-1}$) following HA. Moreover, $\Delta\Delta\text{HR}$ was significantly correlated with ΔT_{re} at rest, suggesting that impairment in HR during passive heating is associated with the decrease in T_{re} at rest following HA. According to Wyndham et al. [34], the reasons for changes in HR during acclimatization are complex. The ma-

ior advantage attributed to a lower HR following HA is the improvement in aerobic fitness and the ability to tolerate a higher T_{co} at exhaustion [38]. Mainly, it is a result of the increased stroke and plasma volume [5, 39] after exercise-induced HA.

RTS and RTC were significantly lower during passive heating after HA. However, only $\Delta\Delta\text{RTC}$ showed a significant correlation with ΔT_{re} at rest. This indicates that a change in subjective RTC during passive heating is associated with changes in T_{re} at rest following HA. According to Aoyagi et al. [40], the RTS is associated with skin wettedness and the impact of sweat evaporation on skin temperature. Coincidentally, there was no significant correlation between $\Delta\Delta\text{LBM}$ and ΔT_{re} at rest, implying that skin wettedness is independently associated with a decrease in T_{re} at rest. In such conditions, therefore, it could be suggested that the increase in sweating performance during passive heating following HA is mostly due to RTS.

The adjusted PSI was applied to human subjects following a 7-session HA protocol and a 45-min immersion up to the waistline in a water bath at ~ 44 °C (air t 23 °C, rh 40%). During the study, no exercise-induced heat exposure was performed. To our knowledge, it is the first study to show that HA induced by repeated passive heating leads to a decreased PSI in humans. However, there is only one study with animals, which showed a decreased PSI in rats following exercise-induced HA [17]. Our results also demonstrate that the rating of PSI ranged between moderate and high. A similar range in PSI rating was reported by Moran et al. [16] in humans performing exercise in the heat (40 °C, rh 40%; 1.34 m/s at a 2% grade) for 120 min. However, these results indicate that similar ratings of PSI could be attained not only by exercise-induced heat exposure, but also during a passively induced heat exposure. Surprisingly, no correlation was found between ΔPSI and ΔT_{re} at rest following acclimation. It seems that the PSI model is not sensitive to changes in T_{re} at rest following acclimation, despite the fact that there was a strong relationship between ΔT_{re} and $\Delta\Delta T_{re}$, as well as between ΔT_{re} and $\Delta\Delta\text{HR}$.

Surprisingly, we found no significant difference in torque fatigue followed successful HA while performing 2-min MVC in hyperthermia. There is some evidence to that persons with a high physical fitness show a lower resting T_{co} compared to persons with a lower fitness [8, 41]. Notably, in our case the final T_{re} before and after HA at which subjects started exercising was higher or similar to those reported for persons with a high physical fitness (T_{re} 39.2 °C) [8]. However, in this study, the subjects were physically active but not highly fit individuals. Thus, it might be suggested that the final T_{re} at which the exercise was started was too high to enhance MVC torque following HA.

In summary, our results indicate that passively induced heat acclimation is reliable for lowering T_{re} at rest. Although passively induced heat acclimation for two weeks improved symptoms of acclimation, it did not change MVC torque fatigue. The results also show that changes in T_{re} , HR and RTC during passive heating are closely related to the shift of T_{re} at rest, following heat acclimation.

Received 9 August 2009

Accepted 23 October 2009

References

1. Armstrong LE, Maresh CM. *Sports Med* 1991; 12(5): 302–12.
2. Nielsen B. *Ergonomics* 1994; 37(1): 49–58.
3. Wyndham CH. *Annu Rev Physiol* 1973; 35: 193–220.
4. Buono MJ, Heaney JH, Canine KM. *Am J Physiol Regul Integr Comp Physiol* 1998; 274 (5 Pt 2): R1295–99.
5. Saat M, Sirisinghe RG, Singh R et al. *J Physiol Anthropol Appl Human Sci* 2005; 24(5): 541–9.
6. Kampmann B, Bröde P, Schütte M et al. *Eur J Appl Physiol* 2008; 104(2): 321–7.
7. Armstrong CG, Kenney WL. *J Appl Physiol* 1993; 75(5): 2162–7.
8. Cheung SS, McLellan TM. *J Appl Physiol* 1998; 84(5): 1731–9.
9. Fox RH, Goldsmith R, Kidd DJ et al. *J Physiol* 1963; 166: 548–62.
10. Henane R, Valatx JL. *J Physiol* 1973; 230(2): 255–71.
11. Henane R, Bittel J. *J Appl Physiol* 1975; 38(2): 294–9.
12. Candas V, Libert JP, Vogt JJ. *J Appl Physiol* 1979; 47(6): 1194–200.
13. Garden JW, Wilson ID, Rasch PJ. *J Appl Physiol* 1966; 21(2): 665–9.
14. Belding HS, Hatch TF. *Heat Pip Air Condit* 1955; 27: 129–36.
15. Lee DHK. *Environ Res* 1980; 22: 331–56.
16. Moran DS, Shitzer A, Pandolf KB. *Am J Physiol* 1998; 275 (1 Pt 2): R129–34.
17. Moran DS, Horowitz M, Meiri U et al. *J Appl Physiol* 1999; 86(3): 895–901.
18. Nybo L, Nielsen B. *J Appl Physiol* 2001; 91: 1055–60.
19. Edwards RH, Harris RC, Hultman E, Kaijser L. *J Physiol* 1972; 220, 335–52.
20. Rall JA, Woledge RC. *Am J Physiol* 1990; 259, R197–203.
21. Horvath SM, Drinkwater BL. *Aviat Space Environ Med* 1982; 53(8): 790–4.
22. Bauman JE. *Fertil Steril* 1981; 36(6): 729–33.
23. Sargeant AJ. *Eur J Appl Physiol Occup Physiol* 1987; 56(6): 693–8.
24. Proulx CI, Ducharme MB, Kenny GP. *J Appl Physiol* 2003; 94(4): 1317–23.
25. Burton AC. *J Nutr* 1935; 9: 261–80.
26. Ramanathan NL. *J Appl Physiol* 1964; 19: 531–3.
27. Gagge AP, Stolwijk JA, Hardy JD. *J Environ Res* 1967; 1(1): 1–20.
28. Patterson MJ, Stocks JM, Taylor NA. *Am J Physiol Regul Integr Comp Physiol* 2004; 286(3): R512–8.
29. Shvartz E, Magazanik A, Glick Z. *J Appl Physiol* 1974; 36: 572–6.
30. Lee DT, Haymes EM. *J Appl Physiol* 1995; 79(6): 1971–6.
31. Olschewski H, Bruck K. *J Appl Physiol* 1988; 64(2): 803–11.
32. Shvartz E, Saar E, Meyerstein N et al. *J Appl Physiol* 1973; 34(2): 214–9.
33. Buguet A, Gati R, Soubiran G et al. *Eur J Appl Physiol* 1988; 58(3): 334–9.
34. Wyndham CH, Rogers GG, Senay LC et al. *Appl Physiol* 1976; 40(5): 779–85.
34. Aoyagi Y, McLellan TM, Shephard RJ. *Sports Med* 1997; 23(3): 173–210.
36. Rogers SM, Baker MA. *Eur J Appl Physiol* 1997; 75:34–8.
37. Grucza R, Pekkarinen H, Titov EK. *Eur J Appl Physiol* 1993; 67:279–85.
38. Selkirk GA, McLellan TM. *J Appl Physiol* 2001; 91(5): 2055–63.
39. Nielsen B, Hales JRS, Strange S et al. *J Physiol* 1993; 460: 467–5.
40. Aoyagi Y, McLellan TM, Shephard RJ. *Ergonomics* 1998; 41(3): 328–357.
41. Shvartz E, Shapiro Y, Magazanik A. *Appl Physiol* 1977; 43: 678–83.

Marius Brazaitis, Ieva Lukošiušė-Stanikūnienė,
Albertas Skurvydas, Laura Daniusevičiūtė, Dalia Mickevičienė

KARTOTINIO PASYVAUS ŠILDYMO POVEIKIS AKLIMATIZACIJOS KARŠČIUI POŽYMIŲ KAITAI

Santrauka

Pagrindinis šio tyrimo tikslas buvo ištirti kartotinio pasyvaus šildymo poveikį aklimatizacijos karščiui požymių kaitai. Trylika tiriamųjų (septyni vyrai ir šešios moterys) buvo pasyviai pratinami prie karščio taikant septynių kursų programą. Pasyvaus šildymo kursas kas antrą dieną buvo kartojamas dvi savaites. Kiekvieno pasyvaus šildymo kurso metu tiriamieji 45 min. sėdėjo iki juosmens vonioje su vandeniu, kurio temperatūra buvo ~44 °C (patalpos oro temperatūra 23 °C; santykinis drėgnumas 40 %). Prieš ir po pasyvaus šildymo matuota tiriamųjų kūno masė, rektalinė

(T_{re}) ir odos (T_{odos}) temperatūra. Pasyvaus šildymo metu kas 5 min. fiksuotas širdies susitraukimo dažnis (ŠSD), tiriamųjų šiluminis pojūtis ir komfortas. Per pirmą ir septintą kursą po pasyvaus šildymo tiriamieji atliko 2 min. trukmės maksimalaus valingo izometrinio raumenų susitraukimo krūvį (MVJ – 2 min.). Aklimacija karščiui sumažino ramybės būklės T_{re} ir pasyvaus šildymo pabaigoje, tačiau reikšmingai nepaveikė T_{odos} . Per septintą pasyvaus šildymo kursą nustatytas reikšmingai sumažėjęs ŠSD, fiziologinis karščio stresas, šiluminis pojūtis ir komfortas, taip pat padidėjęs prakaitavimas. Raumenų izometrinio susitraukimo jėga, atliekant MVJ – 2 min., labiausiai sumažėjo po pasyvaus apatinių galūnių šildymo, lyginant

su įprastinėmis sąlygomis. Aklimatizacija karščiui reikšmingai nepaveikė MVJ nuovargio. Nustatyta, kad kartotinė pasyvaus šildymo programa yra patikimas būdas sumažinti ramybės būklės T_{re} , taip pat aklimatizuotis esant karščiui. Nors pasyvaus šildymo kursas, kartojamas dvi savaites, pagerino aklimatizacijos požymius, tačiau reikšmingai nepaveikė MVJ nuovargio krūvio metu. Po aklimatizacijos karščiui T_{re} , ŠSD ir šiluminio komforto kaita pasyvaus šildymo metu priklauso nuo T_{re} kaitos esant ramybės būklei.

Raktažodžiai: rektalinė temperatūra, karščio stresas, aklimatizacijos požymiai, termoreguliacija