EXERCISE TRAINING AND 3-DAY HEAD DOWN BED REST DECONDITIONING: EXERCISE THERMOREGULATION

Bed rest (BR) deconditioning causes excessive increase of exercise core body temperature, while aerobic training improves exercise thermoregulation. The study was designed to determine whether 3 days of 6° head-down bed rest (HDBR) affects body temperature and sweating dynamics during exercise and, if so, whether endurance training before HDBR modifies these responses. Twelve healthy men (20.7±0.9 yrs, VO₂max: 46±4 ml·kg⁻¹·min⁻¹) underwent HDBR twice: before and after 6 weeks of endurance training. Before and after HDBR, the subjects performed 45 min sitting cycle exercise at the same workload equal to 60% of VO₂max determined before training. During exercise the VO₂, HR, tympanic (Ttymp) and skin (Tsk) temperatures were recorded; sweating dynamics was assayed from a ventilated capsule on chest. Training increased VO₂max by 12.1% (p<0.001). Resting Ttymp increased only after first HDBR (by 0.22 ± 0.08 °C, p<0.05), while exercise equilibrium levels of Ttymp were increased (p<0.05) by 0.21 ± 0.07 and 0.26 ± 0.08 °C after first and second HDBR, respectively. Exercise mean Tsk tended to be lower after both HDBR periods. Total sweat loss and time-course of sweating responses were similar in all exercise tests. The sweating threshold related to Ttymp was elevated (p<0.05) only after first HDBR. In conclusion: six-week training regimen prevents HDBR-induced elevation of core temperature (Ttymp) at rest but not during exercise. The post-HDBR increases of Ttymp without changes in sweating rate and the tendency for lower Tsk suggest an early (<3d) influence of BR on skin blood flow.

Key words: core temperature, skin temperature, sweating rate, microgravity
INTRODUCTION

Endurance-trained subjects have greater thermoregulatory capacity than sedentary individuals (1). This is evidenced by attenuation of the core body temperature elevation and faster activation of sweating during submaximal exercise (2-4). The thresholds of increase in skin blood flow and sweating are shifted toward lower internal temperature (5, 6). The training-induced improvement of thermoregulation in men can be attributed to expanded plasma volume (7), enhanced cutaneous vasodilatation (6), and increased sensitivity of sweat glands to cholinergic stimulation (8).

There are few data on the effect of microgravity with restricted physical activity on thermoregulation indicating that horizontal (9) or head down bed rest (HDBR) (10-12) results in elevation of exercise core temperature. This effect occurred already after 6 hours of HDBR (11). After 24 hours of HDBR also resting internal body temperature was increased (10). These increases were suggested to be due to the HDBR-induced hypovolemia. In addition, Crandall et al. (13) recently demonstrated that maximal vasodilatation induced by local heating of the forearm and the maximal sweating rate in response to acetylcholine were both diminished after two weeks of HDBR, indicating that peripheral thermoregulatory mechanisms may be also affected.

Greenleaf and Reese (9) reported that both aerobic (68% of VO$_2$ max) and isometric exercise, performed in the supine body position for 1hr daily during 14 days of BR attenuated deterioration of thermoregulatory function. Moreover, Shibasaki et al. (14) demonstrated, that 90 min per day of exercise at 75% of maximal heart rate during 13 days of HDBR prevented both hypovolemia and an excessive increase in body temperature during exercise. It seems, therefore, that applying rather heavy and prolonged exercise during BR is necessary to reduce adverse thermoregulatory changes. It is unknown, however, if physical training preceding HDBR influences its effect on thermoregulation during exercise.

The present study was designed to determine 1) whether 3 days of 6° HDBR affects body temperature and sweating dynamics during submaximal exercise, and if so 2) whether 6 weeks of endurance training applied before HDBR modifies its effect on exercise thermoregulation. Head down BR was used since remaining in this position is the best model of microgravity. In comparison with horizontal BR, HDBR at 4-6° causes greater foot-to-head hydrostatic gradient resulting in greater and accelerated changes in plasma volume (15). Three days were selected since exposure to a few days of inactivity is sufficient to attenuate exercise performance (16). It was hypothesized that the "impairment" of thermoregulation induced by HDBR before training might be attenuated after training.

MATERIAL AND METHODS

Subjects. Twelve healthy male students of the Military Academy (age: 20.7±0.9 yr, body mass: 74.4±(SD)8.4 kg, height: 176.2±(SD)4.6 cm, VO$_2$max: 46 ± (SD)4 ml·kg$^{-1}$·min$^{-1}$) volunteered to take
part in the study after giving informed consent. All lived in the Students’ Hostel, had similar daily activities and the same controlled diet. The study protocol was approved by Ethical Committee of the Academy of Physical Education, in Poznan, Poland.

**Study protocol.** The subjects were subjected twice to the 6° HDBR for 3 days before and after six weeks of endurance training (HDBR1 and HDBR2, respectively). During HDBR the subjects were under medically supervised conditions in specially configured rooms in the Students’ Hostel. They drank 1L of carbonate free mineral water per day, and their total energy intake was 12,000 kJ/day (50 % carbohydrates, 35% fat and 15% protein). On the first day of HDBR following an overnight fast, and then after 3 days of HDBR, venous blood samples were taken for hematocrit and hemoglobin determinations.

The training program included 5 sessions per week lasting 60-90 min. The training sessions consisted of 10 min jogging at heart rate (HR) of 120-130 beats/min, 30 min of constant rate running at 60-70% of VO\(_{2\max}\), interval running with the maximal speed for 150-200 m, and 30 min of swimming or soccer. To evaluate the effectiveness of training, three days before both HDBR periods the subjects performed an incremental exercise test until volitional exhaustion during which VO\(_{2\max}\) was determined.

One day before and after completion of both HDBR the subjects were submitted to 45 min submaximal exercise tests on cycloergometer in the sitting position. The workload approximated 60% of VO\(_{2\max}\) determined before HDBR1. These tests were carried out between 15.00 and 18.00 hr, three hours after lunch at an ambient temperature of 22.5 ± 0.5°C and a relative humidity of 35 ± 4%. The subjects were dressed in shorts and shoes. Before and after exercise body mass (BM) was measured to the nearest 10g. During exercise \(\text{O}_2\) uptake, \(\text{CO}_2\) production, HR, tympanic temperature (Ttymp) and skin (Tsk) temperatures were recorded every 5 min. Skin temperature was measured on the forehead, arm, trunk and thigh. Local sweating rate was assessed on the basis of relative humidity of nitrogen flowing at the rate of 2.0 L/min through the capsule fixed on the posterior chest. The surface of the capsule was 20.5cm\(^2\).

**Methods.** Pulmonary ventilation, \(\text{O}_2\) uptake, \(\text{CO}_2\) production and HR were recorded using the Vmax 29 system (SensorMedics,USA). Tympanic temperature was measured with a thermocouple (Ellab,Copenhagen,Denmark) and skin temperature with an infra-red thermometer (Infrapro 1, Oacton, USA). The relative humidity of ambient air and nitrogen flowing through the capsule was measured with Rotronic AG Hygrometer-Control 3 (Switzerland) computerized system.

**Calculations.** Mean Tsk, and mean body temperature (Tmb), were calculated according to the following formulas:

\[
\text{Mean Tsk} = 0.6 \times \text{Tsk trunk} + 0.1 \times \text{Tsk arm} + 0.2 \times \text{Tsk tigh} + 0.1 \times \text{Tsk forehead};
\]

\[
\text{Tmb} = 0.8 \times \text{Ttymp} + 0.2 \times \text{mean Tsk}
\]

Skin heat conductance (Hsk), defined as the average heat flow from body core to skin surface related to the temperature gradient , was calculated using data obtained in the last 10 min of exercise with the equation of Winslow et al. (17) as modified by Greenleaf and Reese (9):

\[
\text{Hsk} (\text{kJ} \cdot \text{m}^{-2} \cdot \text{h}^{-1} \cdot \circ\text{C}^{-1}) = (\text{M} – \text{Eres} – \text{W} – \text{S})/ \text{SA}.(\text{Ttymp} – \text{mean Tsk})
\]

where: M (kJ · h\(^{-1}\)) is total heat production calculated from oxygen uptake and respiratory exchange ratio; Eres (kJ · h\(^{-1}\)) is evaporative heat loss from the respiratory tract ; W (kJ · h\(^{-1}\)) is heat loss due to external work; S (kJ · h\(^{-1}\)) is change in body heat content (3.48 · body mass · Tmb) during the last 10 min of exercise; SA. (m\(^2\)) is body surface area.

Total sweat loss during exercise was the difference in body mass measured be-fore and immediately after exercise.

The changes in plasma volume (APV) induced by HDBR were calculated from hematocrit (Htc) and blood hemoglobin (Hb) concentration:

\[
\Delta PV (\%) = 100 \left(\frac{(\text{Hb}1/\text{Hb}2) \cdot (100 - \text{Htc}2 \cdot 0.874)/(100 - \text{Htc}1 \cdot 0.874) - 1\right)
\]
The physiological strain index (PSI) was calculated according to Moran et al. (18) using Ttymp instead of rectal temperature:

$$\text{PSI} = 5 \cdot \Delta \text{Ttymp} \cdot (39.5 - \text{Ttym})^{-1} + 5 \cdot \Delta \text{HR} \cdot (180 - \text{HR})^{-1}$$

where: $\Delta \text{Ttymp}$ is the difference between Ttymp at the 45th min of exercise minus pre-exercise Ttymp ($\text{Ttym}_0$); $\Delta \text{HR}$ is the difference between HR at the 45th min of exercise minus pre-exercise HR ($\text{HR}_0$).

**Statistics.** Data are presented as means ± SE, unless otherwise stated. The effects of HDBR and training on thermoregulatory responses to exercise were tested by two-way ANOVA for repeated measures. Subsequent post-hoc pairwise comparisons were performed using the Student t-test. The null hypothesis was rejected when $p<0.05$. For calculations the Statistica (2001) version 6. (Statsoft Inc., Tulsa, OK., USA) was used.

**RESULTS**

Plasma volume decreased significantly ($p<0.01$) during both HDBR periods (by 10.0±1.5% and 9.7±1.0% after HDBR1 and HDBR2, respectively).

Training increased VO$_{\text{max}}$ by 12.1% (from 3.39 ± 0.11 to 3.80 ± 0.11 L/min, $p<0.001$). During all exercise-temperature tests the mean exercise load was 147 ± 4 W which corresponded to 63.3 ± 1% and 52.0 ± 0.7% of VO$_{\text{max}}$ determined before and after training, respectively. Analysis of variance did not reveal significant differences between the tests in VO$_2$, respiratory exchange ratio and heat production, while exercise HR decreased ($p<0.001$) following training and increased ($p<0.001$) after both HDBR periods. The values of these variables in the last 10 min of exercise are given in Table 1.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before HDBR1</th>
<th>After HDBR1</th>
<th>Before HDBR2</th>
<th>After HDBR2</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (min$^{-1}$)</td>
<td>156 ± 2.5</td>
<td>168 ± 1.6***</td>
<td>141 ± 2.5***</td>
<td>153 ± 1.5††</td>
</tr>
<tr>
<td>VO$_2$ (L·min$^{-1}$)</td>
<td>2.04 ± 0.07</td>
<td>2.04 ± 0.08</td>
<td>1.92 ± 0.07</td>
<td>1.93 ± 0.06</td>
</tr>
<tr>
<td>RER</td>
<td>0.90 ± 0.01</td>
<td>0.95 ± 0.01</td>
<td>0.90 ± 0.01</td>
<td>0.93 ± 0.01</td>
</tr>
<tr>
<td>Heat production (kJ·min$^{-1}$·kg$^{-1}$)</td>
<td>0.57 ± 0.02</td>
<td>0.57 ± 0.02</td>
<td>0.53 ± 0.01</td>
<td>0.54 ± 0.02</td>
</tr>
<tr>
<td>$\Delta$ Ttymp ($^\circ$C)</td>
<td>0.73 ± 0.11</td>
<td>0.78 ± 0.10</td>
<td>0.58± 0.07</td>
<td>0.86 ± 0.10††</td>
</tr>
<tr>
<td>$\Delta$ meanTsk ($^\circ$C)</td>
<td>0.01± 0.30</td>
<td>-0.63± 0.25*</td>
<td>-0.20± 0.41</td>
<td>-1.24± 0.30†</td>
</tr>
<tr>
<td>$\Delta$ body mass (g)</td>
<td>673 ± 51</td>
<td>616 ± 41</td>
<td>578 ± 32</td>
<td>576 ± 30</td>
</tr>
<tr>
<td>Hsk (kJ·m$^{-2}$·h$^{-1}$·°C$^{-1}$)</td>
<td>214 ± 14</td>
<td>179 ± 13*</td>
<td>164 ± 12*</td>
<td>152 ± 12</td>
</tr>
<tr>
<td>PSI</td>
<td>5.3 ± 0.2</td>
<td>6.1 ± 0.2***</td>
<td>4.5 ± 0.2***</td>
<td>5.4 ± 0.2††</td>
</tr>
</tbody>
</table>

Values are means ± SE. Asterisks denote significant differences in comparison with the test before HDBR1; * $p<0.05$, ** $p<0.01$, ***$p<0.001$; Crosses denote significant differences in comparison with the test before HDBR2: † $p<0.05$, †† $p<0.01$.
After the initial steep increase Ttymp tended to plateau within 20 – 45 min of exercise in all four tests (Fig.1). After HDBR1, Ttymp was increased at rest (p<0.05) and during exercise (p<0.01) in comparison with the control test; while after HDBR2 only the exercise Ttymp values were elevated (p<0.05). Comparison of Ttymp in the pre-HDBR tests before and after training did not show significant differences. The exercise-induced ΔTtymp was greater (p < 0.01) after than before HDBR2 (Table 1).

The mean Tsk decreased during the first 10-15 min of exercise and then increased slightly (ns) in all four exercise-temperature tests (Fig. 1). At the end of exercise the mean Tsk was lower in comparison with the resting values in both post-HDBR tests (p<0.05 and p<0.01 after HDBR1 and HDBR2, respectively). Analysis of variance did not reveal significant effects of training and HDBR on the mean Tsk during exercise; however, a tendency towards lower values was noted after both HDBR periods in comparison with the respective pre-HDBR tests. Within the last 25 min of exercise the mean Tsk was lower (p<0.05) after HDBR2 than after HDBR1. The exercise-induced change in mean Tsk (ΔTsk) differed significantly between the pre- and post HDBR tests (Table 1).

![Fig.1. Time-course of changes in tympanic and mean skin temperatures during the four exercise tests. Asterisks denote significant differences between values obtained before and after HDBR; *p<0.05, **p<0.01.](image)
Analysis of changes of Tsk in the individual points did not reveal significant differences between the tests. However, temperatures on the trunk and thigh showed lower values after than before both HDBR periods; an opposite tendency was observed on the forehead, while on the arm there were no uniform changes (data not presented).

Skin heat conductance (Hsk), calculated from the data obtained in the last 10 min of exercise, was diminished (p<0.05) by training. Before training HDBR attenuated Hsk (p<0.05), while after training the pre- and post HDBR Hsk values were not different. High correlation (r=0.853, p<0.001) was ascertained between Hsk and the mean Tsk in the last 10 min of exercise.

Total sweat loss during exercise was not different in all four exercise temperature tests (Table 1), and the time-course of the local sweating response was not affected either by HDBR or by training (Table 2, Fig.2 left panel). However, the sweating threshold in relation to Ttymp was slightly but significantly higher (p<0.05) after than before HDBR1, while the slopes of the relationship between sweating rate and Ttymp were not different (Fig.2 right panel). Training or HDBR2 did not affect this relationship.

The physiological strain index (PSI), calculated from HR and Ttymp, was increased (p<0.001) by both HDBR periods, and there was also a significant effect of training on this index (Table1).

Table 2. Local sweating response: time of sweating onset (time delay), time constant of the sweating response (τ), and sweating threshold related to tympanic temperature.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before HDBR1</th>
<th>After HDBR1</th>
<th>Before HDBR2</th>
<th>After HDBR2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sweating onset (s)</td>
<td>281 ± 29</td>
<td>318 ± 33</td>
<td>287 ± 23</td>
<td>315 ± 35</td>
</tr>
<tr>
<td>τ (s)</td>
<td>267 ± 37</td>
<td>244 ± 34</td>
<td>232 ± 21</td>
<td>262 ± 24</td>
</tr>
<tr>
<td>Sweating threshold (°C)</td>
<td>37.08 ± 0.08</td>
<td>37.23 ± 0.06*</td>
<td>37.1 ± 0.08</td>
<td>37.0 ± 0.08</td>
</tr>
</tbody>
</table>

Values are means ± SE. Asterisk denotes significant difference in comparison with the test before HDBR1: * p<0.05

Fig.2. Time-course of local (chest) sweat rate during exercise (left panel), and relationship between sweat rate and tympanic temperature (right panel) for the four tests. Asterisk denotes significant difference in sweating threshold between the pre- and post-HDBR1 tests; *p<0.05.
Effects of HDBR on thermoregulatory responses to exercise before training.

After 3 days of HDBR, Ttymp was elevated at rest by about 0.20°C. This effect is similar to that found in rectal temperature after 24 hours of HDBR by Eartl et al. (10) and only slightly lower than the increase in intestinal temperature reported by Lee et al. (12) after 13 days of HDBR. The latter authors suggested that the HDBR-induced increase in core temperature measured in the morning might result from the decreased amplitude of the circadian rhythm described by Lkhagva (19). However, decreased amplitude of the circadian rhythm cannot be the reason of increased Ttymp in our study because the subjects were examined in the afternoon when body temperature should have been higher than in the morning. Moreover, recent data did not show any changes in amplitude of the circadian rhythm of Ttymp measured weekly during 5 weeks of bed rest (20). One plausible explanation for the effect of HDBR on resting internal temperature is that HDBR induces elevation of the body temperature set point.

During exercise Ttymp reached higher values after than before HDBR although the exercise-induced increases in Ttymp (ΔTtymp) were similar, which is in agreement with the previous studies (10, 12). Hypovolemia may be considered as a factor diminishing effectiveness of heat dissipation. In the present study, in spite of the short duration of HDBR, plasma volume decreased by approx. 10% which confirms previous data indicating that the greatest loss of plasma volume occurs during the first few days of BR (15, 21). However, the role of hypovolemia in the BR-induced "impairment" of thermo-regulation is still uncertain. Fortney (22) demonstrated that PV in women treated with estrogen remained unchanged during 12 days of BR but their pre-exercise and exercise core temperatures were elevated significantly. Thus, it seems likely that factors other than hypovolemia are also involved. Reduced heat conductance from the core to skin during exercise, found in the present and previous (9) studies, suggests alterations in vasomotor tone. There are few data showing lowered baseline forearm skin blood flow after HDBR (13, 23) and attenuated skin blood flow increase during local (3) or whole body heat exposure (23, 24). These changes are caused by reduced vasodilation rather than increased vasoconstriction because the forearm and leg cutaneous vasoconstriction induced by norepinephrine was unchanged after HDBR, while vasoconstriction induced by the venoarteriolar reflex in the leg was diminished (25).

In the present study the pre-exercise mean Tsk was unchanged by HDBR, however, within the last 15 min of exercise a clear tendency toward lower values were noted after than before HDBR. Lowered Tsk during exercise after BR was also observed in previous studies (10,9) and probably reflected decreased skin blood flow. Some local changes in vasodilation induced by HDBR might have occurred since decreased exercise Tsk was found on the thigh and trunk, whilst on the forehead there was an opposite response.
In spite of higher Ttymp during exercise after HDBR, total sweat loss was not different from that before HDBR. Similar results were previously obtained in men (9,12) after two weeks of bed rest, while Fortney (22) reported significantly increased sweat loss in women. In our study and in that by Lee et al. (12) the only effect of HDBR on the local sweating response to exercise was elevation of the sweating threshold when related to core temperature. This may result from the elevated core temperature already before exercise. Because the time-course of sweat secretion during exercise and the slope of the relationship between the sweating rate and Ttymp were not changed by HDBR, it seems likely that the sweating response to exercise depends also on nonthermal factors, such as central command, baroreceptors, mechanoreceptors and metaboreceptors (26, 27, 28).

Influence of endurance training on the effects of HDBR on thermoregulatory responses to exercise. Six weeks of endurance training, in this study, increased VO\textsubscript{2}\text{max} by 12.1%. As a result the relative load of the submaximal exercise during which thermoregulatory responses were measured was smaller than before training. It was reflected by the decreased exercise HR but the exercise-induced increases in Ttymp and total sweat loss showed only a tendency towards lower values. Mean Tsk in the last 20 min of exercise tended to be lowered by training. Local sweating response did not differ between the control (before HDBR1) and post-training tests. It seems, therefore, that duration or intensity of the training was insufficient to evoke significant differences in thermoregulatory functions. Decreased heat conductance during exercise was the only significant effect of training, suggesting attenuation of skin vasodilation which may reflect the smaller relative workload. A decrease in skin heat conductance with training is consistent with a tendency to lower mean Tsk in the last period of exercise. It should be noted that the high correlation between heat conductance and mean Tsk was calculated for all four exercise-temperature tests. Similar correlation was described previously by Greenleaf and Reese (9).

In contrast to the effect of HDBR1 (before training), the pre-exercise Ttymp after HDBR2 was not elevated but the exercise-induced increase of this temperature was significantly greater. As a result, the Ttymp reached similar levels during exercise after HDBR1 and HDBR2. After training the decrease in mean Tsk during exercise induced by HDBR was even greater than before training suggesting that the training preceding HDBR did not prevent impairment of skin vasodilation during exercise.

The shift of sweating threshold related to Ttymp, which occurred after HDBR1, was not found after HDBR2. This is consistent with the unchanged pre-exercise core temperature after HDBR2.

In summary: the present data showed that even a relatively short (3-days) period of HDBR results in increased tympanic temperature both at rest and during exercise. This increase contributed to the markedly higher value of the physiological strain index which may be of importance for patients undergoing exercise therapy after bed rest and perhaps for astronauts subjected to even short
term microgravity. The new finding is that moderate exercise training applied before HDBR attenuates its effect on resting but not on exercise core temperature.

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