Effects of Short-term Exercise in the Heat on Thermoregulation, Blood Parameters, Sweat Secretion and Sweat Composition of Tropic-dwelling Subjects

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Abstract This study investigates the effects of a short-term aerobic training program in a hot environment on thermoregulation, blood parameters, sweat secretion and composition in tropic-dwellers who have been exposed to passive heat. Sixteen healthy Malaysian-Malay male volunteers underwent heat acclimation (HA) by exercising on a bicycle ergometer at 60% of $\dot{V}O_2_{\text{max}}$ for 60 min each day in a hot environment (Ta: 31.1±0.1°C, rh: 70.0±4.4%) for 14 days. All parameters mentioned above were recorded on Day 1 and at the end of HA (Day 16). On these two days, subjects rested for 10 min, then cycled at 60% of $\dot{V}O_2_{\text{max}}$ for 60 min and rested again for 20 min (recovery) in an improvised heat chamber. Rectal temperature (Tre), mean skin temperature (Tsk), heart rate (HR), ratings of perceived exertion (RPE), thermal sensation (TS), local sweat rate and percent dehydration were recorded during the test. Sweat concentration was analysed for sodium [Na$^{+}$]sweat and potassium. Blood samples were analysed for biochemical changes, electrolytes and hematologic indices. Urine samples were collected before and after each test and analysed for electrolytes.

After the period of acclimation the percent dehydration during exercise significantly increased from 1.77±0.09% (Day 1) to 2.14±0.07% (Day 16). Resting levels of hemoglobin, hematocrit and red blood cells decreased significantly while [Na$^{+}$]sweat increased significantly. For Tre and Tsk there were no differences at rest. Tre, HR, RPE, TS, plasma lactate concentration, hemoglobin and hematocrit at the 40th min of exercise were significantly lower after the period of acclimation but mean corpuscular hemoglobin and serum osmolality were significantly higher while no difference was seen in [Na$^{+}$]sweat and Tsk. It can be concluded that tropic-dwelling subjects, although exposed to prolonged passive heat exposure, were not fully heat acclimatized. To achieve further HA, they should gradually expose themselves to exercise-heat stress in a hot environment.

Keywords: heat-acclimation, tropic-dwelling, thermoregulation, sweat secretion, sweat composition

Introduction

Heat acclimation (HA) refers to an increase in heat tolerance level while physically working under stress conditions in the natural environment (Armstrong and Maresh, 1991; Nielsen, 1994). The physiological adaptations of HA include improved cardiac output with lowered heart rate (HR), together with increased stroke volume (SV), sweat rate and blood plasma volume, decreased core temperature and mean skin temperature (Tsk) (Wyndham et al., 1976), rectal temperature at rest (Buono et al., 1998) and oxygen consumption at a given work rate, earlier onset of sweating during exercise and decreased sodium chloride losses in sweat and urine (Armstrong and Maresh, 1991). The culmination of these adaptations facilitates more efficient heat transfer from exercising muscles to the skin, to be evaporated away into the external environment.

Physiological adaptations such as decreased HR and expansion of blood plasma volume take place as early as 3 to 6 days after continuous heat exposure, whereas improvement in sweat rate takes place at 8 to 14 days of continuous exposures to heat-stress (Armstrong and Maresh, 1991). In attempting to optimize HA, athletes should exercise at intensities greater than 50% $\dot{V}O_2_{\text{max}}$ for 10 to 14 days (Armstrong and Maresh, 1991). Although there are many studies on HA responses with short-term physical and stress conditioning, those mentioning HA responses with prolonged passive heat exposure are scarce (Strydom et al., 1966; Shapiro et al., 1981). In Malaysians residing in Japan, the sweat onset time tended to decrease with longer duration of residence in Japan. This might indicate that long-term HA acquired in tropical subjects, born and raised in a tropical area decays after immigration to a temperate area (Saat et al., 1999). Because there is no formal definition for passive heat exposure, this study interprets it as living in a hot
climate without performing intense exercise on a regular basis. Are individuals who experience prolonged passive heat exposure acclimatized to perform physical work under similar conditions? Past research (Piwonka et al., 1965; Strydom et al., 1966; Gisolfi et al., 1969; Strydom et al., 1969; Taylor, 1986; Armstrong et al., 1987) has shown that some form of physical conditioning is required for HA to take place. This, however, does not necessarily imply that passive heat exposure is irrelevant, because the subjects in these studies were living in temperate countries which do not permit continuous and prolonged heat exposure. The summer climate was suggested by Strydom et al. (1966) to have contributed to some degree to HA in subjects who exercised in a cool environment. Shapiro et al. (1981) observed that 10 sessions of HA at the end of the summer season did not alter the rectal temperature ($T_{re}$) and mean sweat rate ($M_{sw}$) of male recruits. They concluded that in terms of thermoregulation and sweating mechanisms, HA to the conditions of the experiment (Ta: 40°C, rh: 30%, wind speed of 1 ms$^{-1}$) was “completed naturally” by being exposed to the summer season. These studies suggested that some degree of HA was possible through passive heat exposure.

Chin and Kenneth (1997) claimed that there were no significant differences in the mean sweat rate and total sweat loss in their subjects living in a tropical country, after going through basic military training for 16 weeks. Hence, they suggest that sweating mechanisms in these subjects were acclimatized before the study and were not influenced by the training. The hypothesis that prolonged passive heat exposure contributed to HA was therefore supported. However to our knowledge there are no recent data available for subjects in the tropics undergoing athletic training.

The primary purpose of this study is to answer the question of whether HA occurs in subjects from the tropics when engaged in a series of exercise programs in a hot environment as assessed by thermoregulatory and blood parameters, sweat secretion and sweat composition.

Methods

Subjects

Sixteen healthy Malaysian-Malay male volunteers underwent HA by exercising on a bicycle ergometer for 60 min each day at 60% of maximal oxygen uptake ($\dot{V}O_{2,\text{max}}$) in a hot environment (Ta: 31.1±0.1°C, rh: 70.0±4.4%) for 14 days. The subjects were not previously trained in a hot environment but were physically active with basic recreational activities ranging from “very, very light” at 7 to “very, very hard” at 19. The Helsinki Declaration of 1975.

Procedure

Subjects were tested at the start (Day 1) and at the end of HA (Day 16). They were not allowed caffeinated drinks, alcoholic beverages, or any drugs and they were confined to bed before 23:30 on the nights prior to the test days. On the test days, subjects reported to the laboratory two hours before testing. A standard breakfast consisting of two slices of white bread and 500 mL of plain water were given to subjects within 15 min of arrival to ensure a normal hydration state (ACSM, 1996). The subjects then sat in a thermoneutral environment (Ta: 24.0±0.2°C, rh: 67.7±1.5%) and remained in a comfortable sitting position for 15 minutes before a Teflon venous catheter was inserted into a forearm vein and fitted with a three-way stopcock for blood sampling: this remained in place for the remainder of the test. Five minutes before entering the hot experimental chamber each subject voided his bladder as completely as possible and the entire volume was collected. Nude body weight was then recorded using an electronic scale with an accuracy of ±0.01 kg. The subjects were then asked to enter the hot experimental chamber (Ta: 31.1±0.1°C, rh: 70.0±4.4%) and spent 90 min in the hot environment: an initial 10 min at rest, 60 min cycling and 20 min recovering. During the first ten minutes and the last twenty minutes, the subjects rested on an electrically-braked bicycle ergometer. During the 60 min of cycling, the subject pedaled the bicycle ergometer at a constant rate of 60 RPM for 60 min, with workload controlled to achieve an intensity of 60% $\dot{V}O_{2,\text{max}}$. All tests were carried out between 10:00 and 12:00 noon to avoid the influence of circadian rhythms.

Measurements and analysis of blood, urine and sweat composition

Rectal temperature ($T_{re}$) was monitored continuously by a thermistor probe (Gram Cooperation Thermistor Sensor, Japan; accuracy, ±0.01°C), which was inserted 12 cm beyond the anal sphincter. Skin temperature was monitored throughout the test with thermistors attached at seven sites (forehead, abdomen, forearm, hand, thigh, calf and foot) with surgical tape. Mean skin temperature ($T_{sk}$) was calculated by using Hardy and Dubois’ equation (1937). The HR was recorded every 10 min using a polar HR detector (Sport Tester S170, Polar, Finland).

Reports of thermal sensation (TS) and ratings of perceived exertion (RPE) were elicited at the same time intervals as the HR. TS was rated using a 17-point scale as modified by Pandolf (2001). The scale consisted of numbers from 0.0 to 8.0 in half-number increments (i.e., 0, 0.5, 1.0, 1.5, 2.0, 2.5...). Descriptive words were attached to the whole numbers and ranged from “unbearably cold” at 0.0 to “unbearably hot” at 8.0. RPE was measured utilizing the scale developed by Borg (1970). This bimodal scale had numbers from 6 to 20; with every odd number having an attached verbal expression ranging from “very, very light” at 7 to “very, very hard” at 19.
Explicit written instructions regarding the use of these scales were read to each subject prior to experimentation.

Local sweat samples were collected every 10 min using pieces of filter paper (12.4 cm²) attached to the lower part of the sub scapular region and covered with a sheet of vinyl to prevent evaporation of the sweat. The local sweat rates on the back ($M_{\text{sw-back}}$) were calculated from the mass gained by the filter paper with the use of an electric balance accurate to 1 mg. The sweat collected on the paper was eluted with 10 mL of distilled water for 48 hours and stored in refrigerators (−80°C) before analysis for sodium ([Na⁺]_{sweat}) and potassium ([K⁺]_{sweat}) with an Atomic Absorption Spectrophotometer (5100 Perkin Elmer).

Blood samples were collected 5 min before exercise and at 5, 20, 40 and 60 min during exercise and 10 and 20 min post exercise. Ten milliliters (10 mL) of venous blood were drawn, of which three milliliters (3 mL) were transferred to an EDTA tube for estimation of hematologic indices using a hematology analyzer (Sysmex KX-21N). Hemoglobin (Hb) concentration was determined using a the cyanmethemoglobin method (3.24 mmol · L⁻¹ serum or urine and potassium [K⁺]_{sweat} at rest was shown in Table 2. [Na⁺]_{sweat} and [K⁺]_{sweat} at rest were significantly higher after HA. $M_{\text{sw-back}}$, [Na⁺]_{sweat} and [K⁺]_{sweat} at the 40th min of exercise were not significantly different after HA.

Compared to Day 1 no changes in resting levels of [Lac]_{plasma}, [Gluc]_{plasma}, [Na⁺]_{serum}, [K⁺]_{serum}, [Cl⁻]_{serum} and serum osmolality (Osm_{serum}) were evident on Day 16 (Table 3). After HA, a pronounced reduction ($p<0.01$) in [Lac]_{plasma} (3.24 mmol · L⁻¹ or 39%) and an increase in Osm_{serum}

### Table 1 Changes in $T_{\text{re}}, T_{\text{sk}},$ HR, RPE and TS at rest and the 40th min of exercise

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Day 1</th>
<th>Day 16</th>
<th>Day 1</th>
<th>Day 16</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_{\text{re}}$ (°C)</td>
<td>37.35 ± 0.34</td>
<td>37.14 ± 0.32</td>
<td>38.85 ± 0.45</td>
<td>38.30 ± 0.29**</td>
</tr>
<tr>
<td>$T_{\text{sk}}$ (°C)</td>
<td>34.20 ± 0.70</td>
<td>34.42 ± 1.02</td>
<td>36.09 ± 1.09</td>
<td>36.11 ± 1.35</td>
</tr>
<tr>
<td>HR (beats · min⁻¹)</td>
<td>79 ± 13</td>
<td>82 ± 19</td>
<td>179 ± 11</td>
<td>159 ± 11**</td>
</tr>
<tr>
<td>RPE</td>
<td>16.67 ± 2.26</td>
<td>14.44 ± 2.13**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TS</td>
<td>4.31 ± 0.63</td>
<td>4.00 ± 0.00</td>
<td>6.50 ± 1.00</td>
<td>5.56 ± 0.77**</td>
</tr>
</tbody>
</table>

$T_{\text{re}},$ mean rectal temperature; $T_{\text{sk}},$ mean skin temperature; HR, heart rate; RPE, ratings of perceived exertion; TS, thermal sensation.

***, significantly different from Day 1 at the 40th min of exercise $p<0.01$. 

#### Statistical analysis

Statistical analyses were performed using paired t-tests, the level of significance being set at $p<0.05$. For RPE and TS, a non-parametric Wilcoxon test was used. Results are presented as mean±Standard Deviation (SD).

### Results

On Day 1 of the test, only 31% (5 out of 16 subjects) completed 60 min of exercise at 60% of $\dot{V}O_{2\text{max}}$. However, since all the subjects completed at least 40 min of exercise on day 1, this time was then used as a comparison of our study for HA during exercise for all parameters. After acclimation, the percent of dehydration increased significantly from 1.77±0.09% (Day 1, n=5) to 2.14±0.07% (Day 16, n=5).

Changes in $T_{\text{re}}, T_{\text{sk}},$ HR, RPE and TS are provided in Table 1. At rest, no significant differences were noted in these parameters but at the 40th min of exercise, $T_{\text{re}},$ HR, RPE and TS were significantly lower after HA while there was no significant change in $T_{\text{sk}}$.

Percent dehydration during the test was calculated using the following formula:

Percent dehydration (%) = \[
\frac{(BW \text{ before test}) - (BW \text{ after whole test (kg)})}{(BW \text{ before test})} \times 100
\]

\(BW=\text{nude body weight}\)

Percent dehydration (%)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Day 1</th>
<th>Day 16</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_{\text{re}}$ (°C)</td>
<td>37.35 ± 0.34</td>
<td>37.14 ± 0.32</td>
</tr>
<tr>
<td>$T_{\text{sk}}$ (°C)</td>
<td>34.20 ± 0.70</td>
<td>34.42 ± 1.02</td>
</tr>
<tr>
<td>HR (beats · min⁻¹)</td>
<td>79 ± 13</td>
<td>82 ± 19</td>
</tr>
<tr>
<td>RPE</td>
<td>16.67 ± 2.26</td>
<td>14.44 ± 2.13**</td>
</tr>
<tr>
<td>TS</td>
<td>4.31 ± 0.63</td>
<td>4.00 ± 0.00</td>
</tr>
</tbody>
</table>

BW = nude body weight.
Table 2  Changes in local sweat rate on the back ($M_{\text{sw-back}}$), [Na$^{+}$]$_{\text{sweat}}$ and [K$^{+}$]$_{\text{sweat}}$ at rest and the 40th min of exercise

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Rest</th>
<th>At the 40th min of exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 1</td>
<td>Day 16</td>
</tr>
<tr>
<td>$M_{\text{sw-back}}$ (mg·cm$^{-2}$·min$^{-1}$)</td>
<td>0.02±0.03</td>
<td>0.04±0.06</td>
</tr>
<tr>
<td>[Na$^{+}$]$_{\text{sweat}}$ (mmol·L$^{-1}$)</td>
<td>0.62±0.28</td>
<td>2.26±1.60**</td>
</tr>
<tr>
<td>[K$^{+}$]$_{\text{sweat}}$ (mmol·L$^{-1}$)</td>
<td>0.27±0.67</td>
<td>0.26±0.36</td>
</tr>
</tbody>
</table>

**, significantly different from Day 1 at rest $p<0.01$.

Table 3  Changes in [Lac]$_{\text{plasma}}$, [Gluc]$_{\text{plasma}}$, [Na$^{+}$]$_{\text{serum}}$, [K$^{+}$]$_{\text{serum}}$, [Cl$^{-}$]$_{\text{serum}}$ and Osm$_{\text{serum}}$ at rest and the 40th min of exercise

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Rest</th>
<th>At the 40th min of exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 1</td>
<td>Day 16</td>
</tr>
<tr>
<td>[Lac]$_{\text{plasma}}$ (mmol·L$^{-1}$)</td>
<td>1.87±0.91</td>
<td>1.64±0.32</td>
</tr>
<tr>
<td>[Gluc]$_{\text{plasma}}$ (mmol·L$^{-1}$)</td>
<td>4.78±0.72</td>
<td>5.08±0.55</td>
</tr>
<tr>
<td>[Na$^{+}$]$_{\text{serum}}$ (mmol·L$^{-1}$)</td>
<td>138±7</td>
<td>136±5</td>
</tr>
<tr>
<td>[K$^{+}$]$_{\text{serum}}$ (mmol·L$^{-1}$)</td>
<td>3.4±0.3</td>
<td>3.9±0.2</td>
</tr>
<tr>
<td>[Cl$^{-}$]$_{\text{serum}}$ (mmol·L$^{-1}$)</td>
<td>95±11</td>
<td>91±13</td>
</tr>
<tr>
<td>Osm$_{\text{serum}}$ (mOsm·kg$^{-1}$)</td>
<td>300±16</td>
<td>302±12</td>
</tr>
</tbody>
</table>

**, significantly different from Day 1 at the 40th min of exercise $p<0.01$.

Table 4  Changes in selected hematologic indices and $\Delta$PV (%) at rest and the 40th min of exercise

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Rest</th>
<th>At the 40th min of exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 1</td>
<td>Day 16</td>
</tr>
<tr>
<td>Hb, g·dL$^{-1}$</td>
<td>14.74±0.62</td>
<td>14.19±0.64**</td>
</tr>
<tr>
<td>Hct, %</td>
<td>43.18±1.49</td>
<td>41.48±2.23**</td>
</tr>
<tr>
<td>RBC, (X10$^9$ L$^{-1}$)</td>
<td>4.94±0.35</td>
<td>4.72±0.41**</td>
</tr>
<tr>
<td>MCV, fL</td>
<td>87.69±4.82</td>
<td>88.18±4.18</td>
</tr>
<tr>
<td>MCH, pg</td>
<td>29.94±1.65</td>
<td>30.19±1.81</td>
</tr>
<tr>
<td>MCHC, g·dL$^{-1}$</td>
<td>34.10±0.94</td>
<td>34.24±1.08</td>
</tr>
<tr>
<td>RDW (%)</td>
<td>12.67±0.96</td>
<td>13.17±1.39</td>
</tr>
<tr>
<td>WBC, (X10$^9$ L$^{-1}$)</td>
<td>7.25±2.23</td>
<td>6.54±1.64</td>
</tr>
<tr>
<td>PLT, (X10$^9$ L$^{-1}$)</td>
<td>254±65</td>
<td>261±63</td>
</tr>
<tr>
<td>$\Delta$PV (%)</td>
<td>0.00</td>
<td>0.00</td>
</tr>
</tbody>
</table>

Hb, haemoglobin concentration; Hct, Hematocrit; RBC, red blood cells; MCV, mean corpuscular volume; MCH, mean corpuscular Hb; MCHC, mean corpuscular Hb concentration; RDW, red cell window width. WBC, white blood cells; PLT, platelets; $\Delta$PV (%), percentage change of plasma volume.

**, significantly different from Day 1 at rest $p<0.01$. +, ++ significantly different from Day 1 at the 40th min of exercise at $p<0.05$ and $p<0.01$ respectively.
(6 mOsm·kg⁻¹ or 2%) \((p<0.01)\) was seen at the 40th min of exercise.

Changes in hematologic indices (Hb, Hct, RBC, MCV, MCH, MCHC, RDW, WBC and PLT) and the percentage difference in plasma volume \((\text{MCH, MCHC, RDW, WBC and PLT)}\) and the percentage changes of core temperature could be an indication of HA with an ecologically helpful for the reduction in the amount of sweating (Nguyen and Tokura, 2002). The higher core temperature in people who are living in a tropical climate and who are repeatedly exposed to high ambient temperatures may be shifted toward a higher level (Nguyen and Tokura, 2002). These results led to the suggestion that the set-point of core temperature in people who are living in a tropical climate may be shifted toward a higher level (Nguyen and Tokura, 2002). These results led to the suggestion that the set-point of core temperature in people who are living in a tropical climate and who are repeatedly exposed to high ambient temperatures may be shifted toward a higher level (Nguyen and Tokura, 2002). The higher core temperature in people who are living in a tropical climate may be shifted toward a higher level (Nguyen and Tokura, 2002). These results led to the suggestion that the set-point of core temperature in people who are living in a tropical climate and who are repeatedly exposed to high ambient temperatures may be shifted toward a higher level (Nguyen and Tokura, 2002). The higher core temperature, which is actively regulated in warm temperatures, seemed of adaptive significance. The higher set-point value of core temperature could be an indication of HA with a

### Discussion

In the current study, the 14-day HA protocol was successful as evidenced by a significant decrease in T\(_\text{re}\) at the 40th min of exercise from 38.85°C to 38.30°C and in HR from 179 to 159 beats·min⁻¹. The time course and magnitude of decreases in T\(_\text{re}\) and HR were consistent with those in previous studies (Garden et al., 1966; Wyndham et al., 1973; Shvartz et al., 1979; Pandolf et al., 1988). For instance, Buono et al. (1998) and Shvartz et al. (1979) reported a 0.6°C decrease following 7 days of HA. Hence the above findings suggest that our subjects were successfully acclimated to the heat.

According to a previous study by Buono et al. (1998), there was a significant decrease of 0.3°C (37.0°C to 36.7°C) in resting T\(_\text{re}\) following HA. Although this finding agreed with several older studies (Ladell et al., 1951; Wyndham et al., 1973; Shvartz et al., 1979) that have anecdotally reported a reduction in resting T\(_\text{re}\) with HA, they claimed that their study was the first to report the significant effects of HA on resting T\(_\text{re}\) using a controlled study design. Specifically, in order to obtain a stable resting T\(_\text{re}\) during the HA period, some controls are imposed, such as on hydration level, 12-h exercise abstinence, gender of subjects, time of day for data collection and room temperature.

Nevertheless several older reports also showed decreases in T\(_\text{re}\) at resting level after HA but with no statistically significant differences. For example, Ladell et al. (1951) reported that in HA, involving 17 men for 9 days in a hot, humid (Ta: 38°C, rh: 80%) environment, the mean resting T\(_\text{re}\) decreased 0.3°C over the course of the study. Wyndham et al. (1973) heat acclimated men for nine successive days in warm air (32°C) that was fully saturated with water vapour (~100% rh). Although the point was not specifically addressed in their original manuscript, tabular data showed that mean resting T\(_\text{re}\) fell from 37.4°C to 37.0°C. More recently, Shvartz et al. (1979) anecdotally reported that mean resting T\(_\text{re}\) decreased 0.4°C following 8 days of HA.

The combined past findings strongly suggest that HA has the potential to significantly reduce T\(_\text{re}\) \((-0.3–0.5°C)\) at rest. The effect of a decrease in T\(_\text{re}\) at rest after HA, would be to reduce T\(_\text{m}\) at any given point during exercise in the heat, if the same amount of heat was generated. Several researchers have suggested that a reduction in resting metabolic rate is responsible for reducing resting T\(_\text{re}\) with HA (Shvartz et al., 1973; Buget et al., 1988). Thyroid function has been observed to decrease with HA (Buget et al., 1988; Wenger, 1988). According to Aoyagi et al. (1997) physical training alone decreases T\(_\text{re}\) during exercise but not at rest while HA lowers T\(_\text{re}\) both at rest and exercise. Buono et al. (1998) reached the conclusion that a reduction in resting T\(_\text{re}\) is partially responsible for the attenuation in ending T\(_\text{re}\) during heat exposure following acclimation to humid heat.

Our findings showed a decrease of 0.21°C in T\(_\text{re}\) at rest (37.35°C to 37.14°C) after HA (Table 1). The smaller reduction in T\(_\text{re}\) after HA may be due to higher T\(_\text{re}\) at the resting level of tropical subjects compared to temperate subjects. Many studies indicate that average T\(_\text{re}\) at rest was higher in tropical natives when compared to temperate natives. As reported by Nguyen and Tokura (2002), the average T\(_\text{re}\) during the daytime was significantly higher in Vietnamese than Japanese. The results led to the suggestion that the set-point of core temperature in people who are living in a tropical climate and who are repeatedly exposed to high ambient temperatures may be shifted toward a higher level (Nguyen and Tokura, 2003). The raised level of core temperature might be ecologically helpful for the reduction in the amount of sweating (Nguyen and Tokura, 2002). The higher core temperature, which is actively regulated in warm temperatures, seemed of adaptive significance. The higher set-point value of core temperature could be an indication of HA with a

### Table 5

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Before test</th>
<th>At the end of test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 1</td>
<td>Day 16</td>
</tr>
<tr>
<td>Osmo(_\text{urine}) (mOsm·kg(^{-1}))</td>
<td>294±276</td>
<td>284±200</td>
</tr>
<tr>
<td>[Na(^+)](_\text{urine}) (mmol·L(^{-1}))</td>
<td>71±53</td>
<td>77±50</td>
</tr>
<tr>
<td>[K(^+)](_\text{urine}) (mmol·L(^{-1}))</td>
<td>13±16</td>
<td>10±7</td>
</tr>
<tr>
<td>[Cl(^-)](_\text{urine}) (mmol·L(^{-1}))</td>
<td>51±63</td>
<td>56±50</td>
</tr>
</tbody>
</table>
reduction in the amount of sweating, resulting in the reduction of water consumption. The physiological mechanisms for establishing a higher set-point in core temperature remain to be studied (Nguyen and Tokura, 2003). Residents in Singapore who were acclimated to the tropical climate have been found to have a higher body temperature than people in temperate climates (Adam and Ferres, 1954). The absence of a significant difference in $T_{re}$ at rest after HA in this study is most likely due to the subjects’ long residence in a tropical zone.

Mitchell et al. (1976) showed that the reduction in $T_{sk}$ during acclimatization occurs with a decrease in $T_{sk}$, the gradient $T_{re}$-$T_{sk}$ remaining constant throughout the period of acclimatization. The fall in $T_{sk}$ during acclimatization was shown to be due to improved evaporation from the skin surface which, in turn, was associated with an increase in the rate of sweating. An important factor in the reduction of $T_{sk}$ was the more rapid achievement of thermal balance after the commencement of exercise in heat (Wyndham et al., 1976). The decreases in core and skin temperature during exercise in the heat following HA have been attributed to an increased rate of sweating and associated increases in evaporative cooling. There is no difference between the effect of physical training and that of HA on $T_{sk}$ (Aoyagi et al., 1997).

In our study, no significant changes occurred in $T_{sk}$ at rest (34.20°C to 34.42°C) and at the 40th min of exercise (36.09°C to 36.11°C) (Table 1). This could be due to vasodilation during passive heat exposure. Tropical inhabitants possessed heat-tolerance due to enhanced dry heat loss such as radiation, convection and conduction, which was convenient for maintaining body fluid and osmolarity (Matsumoto et al., 1993, 1997). According to Hori (1995) the higher $T_{sk}$ for subjects born in Okinawa (subtropical) may be due to the result of an increased blood volume with improvement in the cutaneous circulation and reduction in the subcutaneous fat level induced by long-term HA. Residents of temperate zones have been reported to show a decrease in $T_{sk}$ after HA (Aoyagi et al., 1997). In the current study no such fall was demonstrated. This may have been due to the acclimatization of Malaysians by passive heat exposure over a prolonged period. As they were acclimatized, at least where $T_{sk}$ was concerned, no further lowering of $T_{sk}$ was seen after HA.

HR at the 40th min of exercise dropped significantly (20 beats·min⁻¹) following HA (Table 1). This suggests that, in terms of cardiovascular adjustment, the subjects were not fully acclimated although they were from tropical areas. According to Wyndham et al. (1976) their results indicated that the causes of the changes in HR during acclimatization are complex. HR is significantly correlated with both SV and $T_{re}$ suggesting that both an increase in SV and a fall in $T_{re}$ are independently associated with the decrease in HR.

RPE and TS were significantly lower during exercise after HA (Table 1). According to Armstrong and Maresh (1991) the reduction of cardiovascular strain reduces ratings of perceived exertion, which is proportional to central cardiorespiratory stress, which decreases during the first five days of exercise.

Chin and Kenneth (1997) claimed that there were no significant differences in the mean sweat rate and total sweat loss of their subjects after going through basic military training (BMT) for 16 weeks. In contrast, our study showed occurrence of HA in which there was a significant increase in percent dehydration from 1.77±0.09% (Day 1) to 2.14±0.07% (Day 16). This might suggest that the BMT did not cause HA. According to Armstrong and Maresh (1991) in attempting to optimize HA, athletes should exercise at intensities greater than 50% $V_{O2\text{max}}$ for 10 to 14 days. Chin and Kenneth (1997) suggested that sweating mechanisms in these subjects were acclimatized before the study and were not influenced by BMT.

Sweat sodium concentration ([Na⁺]_{sweat}) at rest was significantly higher after HA. [Na⁺]_{sweat} at the 40th min of exercise was lower, but not significantly, after HA (Table 2). Why the [Na⁺]_{sweat} at rest is increased after HA is not clear. To our knowledge this has not been reported previously. The sweat rate at rest is higher after HA, although not significantly. It is possible that this higher sweat rate diminished the reabsorption of Na⁺ in the ductules resulting in a higher [Na⁺]_{sweat}. In previous studies, subjects from temperate countries showed a significantly lower [Na⁺]_{sweat} during exercise after HA (Armstrong, 2000). The [Na⁺]_{sweat} is greatly influenced by sweat rate because the capacity of ductal reabsorption is limited and the amount of the electrolytes that escapes from reabsorption increases with an increase of sweat flow in the duct. After HA, there is an enhanced ability of sweat glands to reabsorb Na⁺, possibly by an increased secretion of adrenal corticoids (Ladell et al., 1961) and/or the altered responsiveness to aldosterone (Kirby et al., 1986). The [Na⁺]_{sweat} at any given rate of sweating and the slope of the relationship between Na⁺ concentration and sweat rate are reduced by HA (Allan et al., 1971; Kirby et al., 1986). Unaltered [K⁺]_{sweat} observed in this study (Table 2) supported earlier findings that HA does not in any way alter the [K⁺]_{sweat} (Methotra et al., 1981).

After HA, percent dehydration is significantly higher due to greater loss of water so as to regulate the body temperature. As a result, serum osmolality at the 40th min of exercise was significantly higher than the value before acclimation (Day 1) (Table 3). This indicated that after HA, free water loss from the body increased. It appears that after HA the higher serum osmolality may be due to increased capacity of sweat glands for sodium reabsorption resulting in a reduction in [Na⁺]_{sweat}. Similarly, [Na⁺]_{urine} increased from 92 mmol·L⁻¹ (Day 1) to 103 mmol·L⁻¹ (Day 16) after HA. When there is a shortage of water in the body the kidney absorbs more water, resulting in a lower urine output with higher concentration. This was supported by the finding that urine collected at the end of the test on day 16 showed a higher osmolality (from 438 mOsm·kg⁻¹ on day 1 to 485 mOsm·kg⁻¹ on day 16) (Table 5).

Previous studies have observed lactate accumulation (Young
et al., 1985) after exercise in heat and reduced muscle glycogen utilization (King et al., 1985; Kirwan et al., 1987) and muscle lactate accumulation (Young et al., 1985) after HA. The lower accumulation of plasma lactate during exercise is caused by a decrease in the amount of lactate produced by working muscle tissue due to an improvement of circulation accompanied by a lower core temperature (Banister et al., 1983). Our results support the previous findings in that the plasma lactate concentration at the 40th min of exercise showed a significant decrease (39%) after HA (Table 3).

The haemodilution in HA is associated with a net gain in the total quantity of intravascular protein, so that the protein concentration remains virtually unchanged (Senay et al., 1976; Senay and Kok, 1977; Sawka et al., 1987). Several hypotheses exist with regard to the physiological mechanism(s) responsible for haemodilution during acclimation. Senay (1975) attributed the expansion of the plasma volume to an influx of protein into the vascular volume, although others (Finberg and Berlyne, 1977) attributed it to an electrolyte shift. Senay and Kok (1976) have also noted that a lack of plasma volume expansions was related to heat intolerance. Although the mechanisms involved remain speculative, it appears that an expansion of the plasma volume is a crucial hemodynamic change associated with cardiovascular adjustment to heat. Unacclimated individuals are seen to haemoconcentrate, whereas heat-acclimated individuals haemodilute (Senay and Pivarnik, 1985). Our results support the previous finding in that the percent change in plasma volume was nearly positive after HA at the 40th min of exercise when compared to Day 1 (Table 4). Similar changes occur in plasma volume in response to both physical training and HA (Aoyagi et al., 1997).

Hemodilution during exercise may assist in the maintenance of cardiac output and offset any effect of fluid loss through sweating on plasma volume. It is not clear, however, whether this transient hemodilution during exercise-heat exposure following acclimatization is large enough to compensate for the displacement of blood volume into the cutaneous veins and thus provide an explanation for the reduced HR component. The proposition that a reduced HR and increased SV is the result of expansion in plasma volume during the early stages of exercise-HA has been challenged by Wyndham et al. (1976). They observed that SV increases as plasma volume increased during the first six days of a ten-day acclimatization study. However, during the final days, SV decreased while plasma volume continued to increase. These results questioned the suggestion that there is a close functional link between plasma volume and changes in HR and SV.

As was expected from the previously published literature (Senay, 1978, 1979; Diaz et al., 1979; Paolone et al., 1980), the differences between the pre and post exposure values in all the hematologic parameters were found to be significant. Resting levels of Hb, Hct and RBC were significantly lower after HA (Table 4). Hb, Hct, RBC and WBC, after the 40th min of exercise (Table 4) were significantly lower after HA. However, MCH at the 40th min of exercise, was significantly higher after HA. These results indicate that the cellular compartment of the blood shrank relative to the plasma component during the period of HA.

Serum osmolality values were significantly higher (Table 3) during exercise after HA. These findings are consistent with previous reports on hyperosmotic plasma during exercise (Costill et al., 1974; Greenleaf et al., 1979; Van Beaumont et al., 1981; Kolka et al., 1982; Miles et al., 1983).

Athletes must always be prepared to participate in events throughout the world, regardless of different climatic conditions. Thus, environmental adaptation must be considered in every event. To reduce the adverse effect that changes in temperature and environment may have on the performance of athletes, it would be useful for them to reacclimatize before competition.

In conclusion, tropic-dwelling subjects although exposed to prolonged passive heat exposure, were not fully heat acclimatized. To achieve further HA, they should gradually expose themselves to exercise-heat stress in a hot environment.

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