Sodium ion concentration vs. sweat rate relationship in humans

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A number of studies (1, 9, 12, 17, 30) have reported that heat acclimation results in a significant reduction in the sodium ion concentration ([Na⁺]) of human sweat. For example, the classic study by Kirby and Convertino (12) found that 10 days of exercise in the heat (40°C, 45% relative humidity) caused a significant reduction in sweat [Na⁺] from ~88 to 42 mmol/l. However, whole body sweat rate significantly increased by 14% during the same time period. More recently, Nielsen et al. (17) found that humid heat acclimation caused sweat [Na⁺] to fall from 107 to 70 mM, whereas sweat rate significantly increased by 26%. However, all of the above studies (1, 9, 12, 17, 30) did not attempt to control for the effect that changes in sweat rate can have on sweat [Na⁺]. This poses a concern because it is well known that the [Na⁺] in sweat is influenced by sweat rate (21, 29, 33). Specifically, the amount of sodium ions that escape reabsorption increases linearly with increases in sweat rate (1, 6, 26, 28). The exact mechanism responsible for this is unknown; however, it has been suggested that above a certain sweat rate threshold there is insufficient time for complete sodium ion reabsorption from the duct (28). It is believed that this threshold reflects the capacity for ion reabsorption of the sweat duct (11, 28). In light of the above, it is clear that changes in sweat [Na⁺] following heat acclimation can be misleading if they are not presented in relation to sweat rate. The preferred method for doing this is to examine the sweat [Na⁺] vs. sweat rate relationship across a range of sweat rates (11, 14).

METHODS

The subjects for the study were eight healthy male volunteers. They had a mean (±SD) age, height, and weight of 26 ± 7 yr, 174 ± 5 cm, and 69.8 ± 11.2 kg, respectively. The San Diego State University institutional review board approved the study, and written informed consent was obtained from each subject before participation. The subjects were asked to refrain from initiating new exercise programs during the duration of the study. No restrictions were placed on their diet because it has been reported that electrolyte supplementation is not necessary during heat acclimation (24). Furthermore, unless the subjects are on very low-sodium-intake diets, manipulations of dietary sodium do not appear to greatly affect sweat sodium ion secretion during heat acclimation (2, 3).

Each subject completed a 10-day heat acclimation protocol. On a separate day, before heat acclimation, each subject performed a graded treadmill test to volitional exhaustion to determine maximal oxygen uptake (V˙O₂max). During the treadmill test oxygen uptake was
measured continuously using a calibrated metabolic cart (TrueMax 2400, Parvomedics, Sandy, UT). All subjects achieved a respiratory exchange ratio of >1.1 during the test. The mean (± SD) \( \dot{V}O_2_{max} \) for the group was 55.6 ± 12.7 ml·kg\(^{-1}\)·min\(^{-1}\).

Within 7 days of completing the treadmill test to exhaustion, each subject began 10 consecutive days of exercise in the heat. Each day consisted of three 30-min exercise bouts of walking on a motorized treadmill in an environmental chamber. The chamber temperature and relative humidity for the three exercise bouts were 36, 40, and 42°C and 40, 40, and 60%, respectively. Ten minutes of seated rest outside the chamber (23°C and 50% relative humidity) occurred between the three exercise bouts. During this time, water was given to completely replace the fluids lost via sweating, thus preventing dehydration during each exercise session. Furthermore, before the first day of the heat acclimation period, the subjects were asked to consume 1 liter of water before going to bed and on arising in the morning. Preexercise nude body weight was measured to the nearest 0.1 kg on the first day before the start of the first exercise bout. This was considered the baseline, euhydrated nude body weight, and all successive days were compared with it. Thus, if the subject reported to the laboratory and was administered until rehydration occurred and baseline weight was reestablished. Ensuring euhydration, both before and throughout the heat acclimation period was critical, because dehydration has been previously shown (16) to significantly effect sweat [Na\(^+\)]. This was achieved as evidence by the fact that preexercise nude body weight for each of the 10 heat acclimation days fluctuated by <1% (Table 1).

The intensity for the three exercise bouts was calculated to approximate 30, 40, and 50% of the previously determined \( \dot{V}O_2_{max} \), respectively. This was achieved by altering the treadmill speed and grade for each subject. On the first day of heat acclimation, expired air samples were collected from the 8th to the 10th min of each exercise bout, and oxygen uptake was determined to ensure that the proper intensity was obtained. The treadmill speed and grade for each of the three exercise bouts remained constant for each individual subject throughout the 10-day heat acclimation protocol. The oxygen uptake data showed that as a group the subjects worked at 28 ± 3, 38 ± 2, and 47 ± 3% of their preacclimation \( \dot{V}O_2_{max} \) during the three exercise bouts. The increases in temperature, relative humidity, and exercise intensity for the three exercise bouts were purposely used to ensure that three different sweat rates would be produced.

Heart rate and core temperature were continuously measured each day during the three exercise bouts. Heart rate was measured using a Polar monitor while core temperature was measured using a rectal thermistor (YSI series 400) inserted by the subject to 10 cm past the anal sphincter.

Chest sweat samples were also collected during each of the three 30-min exercise bouts on day 1 and day 10 of the heat acclimation protocol using separate Macroduct sweat collectors (Wescor, Logan, UT). The collectors were held in place by elastic straps placed around the torso, which prevented leakage and sample contamination. The Macroduct collectors had a surface area of 5.2 cm\(^2\) and were fitted with an additional airtight reservoir to increase their sweat collection capacity to ~1.5 ml. A new Macroduct was applied to the same chest site immediately before each of the 3 exercise bouts and sweat was collected for the complete 30 min of each exercise bout. Whole body sweat rate was calculated on days 1 and 10 using pre- and postexercise dry nude body weights, which were corrected for the amount of fluids consumed.

The skin was cleaned with deionized water and dried immediately before securing each collector. Chest sweat rate was calculated by the change in volume within the sample collection tubing and expressed in milligrams per square centimeter per minute. The chest sweat samples were analyzed in duplicate for osmolality (mosmol/kgH\(_2\)O) using a vapor pressure osmometer (Wescor) and for [Na\(^+\)] using a flame photometer (Cole-Palmer, Chicago, IL).

Regression equations were developed for each subject, and the mean slope and y-intercept for the group were calculated. Pre- and postacclimation data were analyzed using paired t-tests. The sweat osmolality vs. sweat rate and sweat [Na\(^+\)] vs. sweat rate relationships obtained before and after heat acclimation were compared using a two × three repeated-measures ANOVA. Significance was set at the \( P < 0.05 \) level.

**RESULTS**

The results of the present study showed that 10 days of heat acclimation significantly reduced the heart rate and rectal temperature during exercise in the heat. The mean ending heart rate and core temperature for each of the three different exercise bouts on each of the 10 heat acclimation days are presented in Table 1. As can be seen, there were significant reductions in core temperature and heart rate as a result of heat acclimation. For example, on day 1, the group had a mean (± SD) heart rate following the third exercise bout of 158 ± 18 beats/min. On day 10, the heart rate at the same time point was significantly reduced to 140 ± 17 beats/min. Similarly, the mean ending rectal temperature was significantly reduced from 39.0 ± 0.4°C on day 1 to 38.4 ± 0.4°C on day 10. The magnitude of the decreases in rectal temperature and heart rate are consistent with the results of previous studies (5, 10, 18). Such reductions strongly suggest that the 10-day protocol used in the present study was successful in conferring heat acclimation in the subjects. There were no significant changes in chest sweat rate over the course of the 10-day heat acclimation period. Specifically, the mean chest sweat rate for the three exercise bouts on day 1 were 0.26 ± 0.06, 0.59 ± 0.17 and 0.95 ± 0.26 mg ·cm\(^{-2}\)·min\(^{-1}\), respectively. On day 10 the

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**Table 1. Physiological responses during 10 days of heat acclimation**

<table>
<thead>
<tr>
<th>Day</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ending exercise core temperature, °C</td>
<td>37.6 (0.3)</td>
<td>37.4 (0.4)</td>
<td>37.5 (0.4)</td>
<td>37.5 (0.3)</td>
<td>37.4 (0.3)</td>
<td>37.4 (0.4)</td>
<td>37.4 (0.4)</td>
<td>37.4 (0.3)</td>
<td>37.3 (0.4)</td>
</tr>
<tr>
<td><em>Bout 1</em></td>
<td></td>
<td>38.2 (0.3)</td>
<td>38.0 (0.4)</td>
<td>38.0 (0.2)</td>
<td>38.0 (0.3)</td>
<td>38.0 (0.2)</td>
<td>37.8 (0.3)</td>
<td>37.9 (0.3)</td>
<td>37.9 (0.3)</td>
<td>37.9* (0.3)</td>
</tr>
<tr>
<td><em>Bout 2</em></td>
<td></td>
<td>39.0 (0.4)</td>
<td>38.8 (0.4)</td>
<td>38.7 (0.5)</td>
<td>38.6 (0.5)</td>
<td>38.6 (0.4)</td>
<td>38.6 (0.3)</td>
<td>38.5 (0.4)</td>
<td>38.4 (0.3)</td>
<td>38.4* (0.4)</td>
</tr>
<tr>
<td></td>
<td>Ending exercise heart rate, beats/min</td>
<td>108 (12)</td>
<td>108 (9)</td>
<td>106 (11)</td>
<td>104 (11)</td>
<td>105 (9)</td>
<td>104 (11)</td>
<td>100 (8)</td>
<td>103 (11)</td>
<td>101 (10)</td>
</tr>
<tr>
<td><em>Bout 1</em></td>
<td></td>
<td>129 (15)</td>
<td>127 (14)</td>
<td>122 (12)</td>
<td>122 (13)</td>
<td>120 (10)</td>
<td>115 (11)</td>
<td>114 (12)</td>
<td>116 (14)</td>
<td>115 (11)</td>
</tr>
<tr>
<td><em>Bout 2</em></td>
<td></td>
<td>158 (18)</td>
<td>154 (17)</td>
<td>149 (16)</td>
<td>146 (17)</td>
<td>142 (17)</td>
<td>143 (15)</td>
<td>138 (17)</td>
<td>141 (17)</td>
<td>140 (17)</td>
</tr>
<tr>
<td>Preexercise body weight, kg</td>
<td>69.8 (11.2)</td>
<td>69.5 (11.3)</td>
<td>69.7 (11.5)</td>
<td>69.4 (11.3)</td>
<td>69.6 (11.6)</td>
<td>69.8 (11.7)</td>
<td>69.7 (11.8)</td>
<td>69.4 (11.8)</td>
<td>69.5 (10.8)</td>
<td>69.4 (11.1)</td>
</tr>
</tbody>
</table>

*Day 10 value was significantly less than day 1, \( P < 0.05 \).
mean values were 0.31 ± 0.14, 0.66 ± 0.22, and 1.03 ± 0.34 mg·cm⁻²·min⁻¹, respectively. In addition, the mean (± SD) whole body sweat rate was 0.92 ± 0.46 l/h on day 1 and 1.08 ± 0.30 l/h on day 10. The 17% increase is consistent in magnitude with past heat acclimation studies (12, 17); however, it was not significant.

The relationship between sweat osmolality and chest sweat rate obtained on day 1 and day 10 of heat acclimation is presented in Fig. 1. As can be seen, the relationship was shifted to the right following heat acclimation, thus sweat osmolality was reduced for a given sweat rate as a result of heat acclimation. The interaction, however, was not significant, which suggests that the slope of the relationship was not affected by heat acclimation. Rather, heat acclimation significantly reduced the mean y-intercept from 83 ± 31 to 55 ± 34 mosmol/kgH₂O.

The relationship between sweat [Na⁺] and chest sweat rate obtained on days 1 and 10 of heat acclimation is presented in Fig. 2. Similarly, the relationship was shifted to the right following heat acclimation; thus sweat [Na⁺] was reduced for a given sweat rate. The slope of the relationship was not changed by heat acclimation, as suggested by a nonsignificant interaction. Heat acclimation, however, significantly reduced the mean y-intercept from 22 ± 11 to 7 ± 16 mmol/l. It was not surprising that the two figures were similar because it has previously been shown that sweat osmolality and [Na⁺] are highly correlated, with sodium usually accounting for approximately half of the sweat osmolality (7).

**DISCUSSION**

The most important finding of the present study was that 10 days of heat acclimation shifted the sweat osmolality and sweat [Na⁺] vs. sweat rate relationship to the right; thus both variables were reduced for a given sweat rate, up to ~1 mg·cm⁻²·min⁻¹. More specifically, the slopes of the sweat osmolality and sweat [Na⁺] vs. sweat rate relationships were not significantly changed by heat acclimation. However, there were significant reductions in the mean y-intercept of both relationships following heat acclimation.

Sweating has the potential to cause significant amounts of sodium and chloride ions to be lost; therefore, they must be conserved. Structurally, the human eccrine sweat gland consists of two distinct regions. These are the secretory coil, which produces an isosmotic precursor sweat by the electrogenic movement of sodium and chloride ions and the coupled movement of water via osmosis, and the reabsorptive duct, which actively reabsorbs salt from the precursor sweat (19, 29). The reabsorption depends on Na⁺ being actively transported into the interstitial fluid via an ouabain-sensitive Na⁺-K⁺-ATPase localized on the basolateral membrane of the ductal cells. Sodium ions enter the duct cell via an amiloride-sensitive channel expressed in the apical membrane. Chloride ions follow down their electrochemical gradient during reabsorption using the cystic fibrosis transmembrane conductance regulator located in the luminal membrane (19, 29).

During low levels of sweat production significant amounts of sodium and chloride ions can be reabsorbed from sweat during its passage along the duct, resulting in a very hypomotic sweat appearing at the skin surface. However, the rate of sodium and chloride ion reabsorption has a finite capacity; thus, at high sweat rates, there is insufficient time for complete ion reabsorption. This is evidenced by the fact that the concentration of sodium ions in the sweat increases linearly with increases in sweat rate (1, 6, 11). The results of the present study agree with the above findings. As can be seen in Fig. 2, sweat [Na⁺] increased linearly with increases in sweat rate both before and after heat acclimation. Heat acclimation, however, shifted the relationship to the right, significantly reducing the y-intercept, without changing the slope. These findings suggest that heat acclimation increases the reabsorptive ability of the eccrine sweat duct. Shamsuddin et al. (28) have previously shown that a sweat rate threshold exists above which sweat [Na⁺] increases rapidly. Furthermore, this threshold may reflect the ion reabsorptive capacity of the sweat gland duct and be the result of an imbalance between sodium accumulation in the precursor sweat and sodium reabsorption from the proximal duct (28). The reduction in the y-intercept of the sweat [Na⁺] vs. sweat rate relationship found in the present study is consistent with an increase the sodium ion reabsorption capacity. This would delay the imbalance between sodium accumulation in the precursor sweat and sodium reabsorption to a greater sweat rate following heat acclimation. The exact mechanism responsible for the increased sodium reabsorption following heat acclimation is currently unknown. Most likely, it involves aldosterone-induced increases (8) in the Na⁺-K⁺-ATPase and the sodium channels in the reabsorption duct (22, 23). This is
HEAT ACCLIMATION REDUCES SWEAT SODIUM CONCENTRATION 993
evidenced by the fact that administration of the aldosterone antagonist, spironolactone, has been shown to increase sweat [Na⁺] during heat acclimation (13). This finding is in contrast to the results of Allan and Wilson (1), who reported that heat acclimation reduced the slope of the sweat sodium concentration vs. sweat rate relationship. However, as discussed earlier, that study had several methodological concerns, including a small sample size and an unorthodox heat acclimation protocol.

The unchanged slope, coupled with increased electrolyte reabsorption, found in the present study would be consistent with the data of Shamsuddin et al. (27). They altered sodium reabsorption of the eccrine sweat gland by changing skin temperature. Increases in temperature are known to increase sodium channel excitability (20). Thus they reported that sweat ion reabsorption capacity was significantly increased during exercise in a 25 vs. a 15°C environment. However, the slope of ion reabsorption was unchanged. Rather the increased sweat ion reabsorption was the result of a lower \( y \)-intercept on the sweat ion concentration vs. sweat rate relationship in the 25°C condition.

Furthermore, a recent study (14) measured the sweat [Na⁺] vs. sweat rate relationship before and after 21 days of heat acclimation in horses. The results clearly showed that the slope of the relationship was unchanged with heat acclimation, whereas the \( y \)-intercept was reduced \( \sim 15 \) mmol/l for the six animals studied. Such findings are in agreement with the present results (Fig. 2).

The primary advantages of the reduced sweat [Na⁺] seen following heat acclimation appear to be threefold. First, by virtue of having more dilute sweat, heat-acclimated individuals have additional solutes (primarily sodium and chloride) remaining within the extracellular space to exert an osmotic pressure to redistribute fluid from the intracellular space (24). This is evidence by the fact that following heat acclimation, subjects have a significantly smaller reduction in plasma volume (5 vs. 12%) for a given level of dehydration (25). Second, an increased sweat [Na⁺] has been one of the factors associated with the development of hypohydration during prolonged exercise (15). For example, cystic fibrosis patients, who secrete high levels of sodium and chloride in their sweat, are predisposed to the development of hypohydration when exposed to heat (24, 31). Thus heat acclimation has the potential to attenuate the development of hypohydration by reducing the sodium lost via sweating (15). Lastly, it has recently been shown (32) that subjects with lower sweat [Na⁺] had attenuated osmotic inhibition of various thermoregulatory responses to heat stress. Specifically, the hyperosmotic-induced increases in the core temperature threshold for sweating and cutaneous vasodilatation were smaller in those subjects with lower sweat [Na⁺]. It was hypothesized that such results would be beneficial in maintaining thermoregulatory sweating and skin blood flow during prolonged heat stress, thus preventing hyperthermia (32).

In conclusion, the results of the present study are the first in the literature to show that active heat acclimation significantly reduces both the sweat osmolality and sweat [Na⁺] for a given sweat rate up to \( \sim 1 \) mg·cm⁻²·min⁻¹ in humans. Specifically heat acclimation reduces the \( y \)-intercept of both the sweat osmolality and sweat [Na⁺] vs. sweat rate relationships, without changing their slopes. Such a finding suggests that heat acclimation increases ion reabsorption in the sweat gland duct.

REFERENCES


