

Genomic and non-genomic effects of aldosterone on the individual variation of the sweat Na^+ concentration during exercise in trained athletes

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Abstract To clarify whether the resting background effects (genomic) or exercise-related action (non-genomic) of aldosterone (ALD) is primarily affected to an individual variation in the sweat Na^+ concentration ($[\text{Na}^+]_{\text{sweat}}$), we analyzed the cross-sectional relationship between $[\text{Na}^+]_{\text{sweat}}$ and the plasma ALD concentration during rest and exercise in a hot environment. Eleven college-aged male subjects with a mean maximal oxygen uptake of 48 (range 42–59) $\text{ml kg}^{-1} \text{min}^{-1}$ performed three sessions of 20-min cycle exercise at two levels of intensity (40 or 60% $\dot{V}O_{2\text{max}}$) in a room maintained at 31°C. The chest sweat rate (SR_{ch}) and its containing Na^+ were higher and individual differences in SR_{ch} and $[\text{Na}^+]_{\text{sweat}}$ were greater at 60% exercise than at 40% exercise. In each individual, the $[\text{Na}^+]_{\text{sweat}}$ increased significantly ($P < 0.05$) with the increase in the SR_{ch} . In all subjects, the mean $[\text{Na}^+]_{\text{sweat}}$ during exercise corre-

lated negatively with the resting plasma ALD level at either percentage, but it did not correlate with the exercising ALD. These results suggest that individual variations in the increase of the $[\text{Na}^+]_{\text{sweat}}$ in response to a rise in the SR_{ch} may thus be more closely related to the resting ALD than to the exercising ALD. As a result, the genomic action of ALD may be affected more by the sweat Na^+ variation than by the rapidly non-genomic action during exercise in humans.

Keywords Chest sweat rate · Individual difference · Atomic absorption spectrometry · Filter paper method · Radioimmunoassay

Introduction

The increase in the sweat Na^+ concentration ($[\text{Na}^+]_{\text{sweat}}$) in response to an increase in sweat secretion has been observed (Allan and Wilson 1971; Sato 1977; Sato and Dobson 1970), and there are individual variations in the $[\text{Na}^+]_{\text{sweat}}$ due to heat acclimation, salt deficit, and other factors (Allan and Wilson 1971; Allsopp et al. 1998; Kirby and Convertino 1986; Verde et al. 1982). Effects of aldosterone (ALD) concentration on sweat Na^+ reabsorption has been documented (Collins 1966; Sato and Dobson 1970). In the subcutaneous injection of ALD, the sweat Na^+ reabsorption did not begin to increase until about 6 h after the first injection of ALD (Collins 1966), which is a genomic action of steroids that are thought to primarily affect the transcription of mRNA and, subsequently, protein synthesis (Wehling 1997). On the other hand, it

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has been reported that ALD at physiological levels induces rapid (<5 min) increase in the intracellular protein kinase C (PKC) activity and an increase in the calcium level and pH in such mineralocorticoid hormone target epithelia, as the distal colon (Maguire et al. 1999) and sweat gland (Hegarty and Harvey 1998, 1999). The end targets of these rapid responses in epithelia are Na^+/H^+ exchange and K^+ channels, and these responses have been suggested to be a non-genomic PKC-dependent pathway (Harvey and Higgins 2000). This rapid ALD effect could theoretically increase the Na^+ excretion in the secretory coil (Morgan et al. 2004). Because of these two possible mechanisms that may be responsible for the ALD effect on sweat Na^+ regulation, it remains to be elucidated whether the genomic or non-genomic action of ALD is primarily affected by the $[\text{Na}^+]_{\text{sweat}}$ in humans.

In studying the hormonal responses to various intensities of exercise, Freund et al. (1991) reported the plasma ALD level tended to increase progressively with increases in the workload. During intensive exercise in a hot environment, an enhanced body temperature elevation induced greater sweat secretion containing greater Na^+ , and plasma ALD markedly increased. Furthermore, acute dehydration increases the $[\text{Na}^+]_{\text{sweat}}$ and plasma ALD concentration during 2 h exercise in heat (Morgan et al. 2004). As a result, during exercise the non-genomic action of ALD may be a primary factor for an increase in sweat Na^+ secretion.

In humans, however, Francesconi et al. (1983) demonstrated that heat-acclimation increases the resting ALD, and reduced $[\text{Na}^+]_{\text{sweat}}$ have also been observed in heat-acclimated subjects (Allan and Wilson 1971; Robinson and Robinson 1954). Heat-acclimation increases the volume of total body water, and much of the increase is accounted for by an expansion of the resting plasma volume (Senay et al. 1976; Shapiro et al. 1981). This increase in the volume of body water can be explained in part by the increased ALD secretion and/or renal sensitivity to a given plasma concentration (Sawka et al. 1996). Accordingly, we hypothesized that during intensive exercise, the individual variation in the $[\text{Na}^+]_{\text{sweat}}$ may therefore be more closely related to the genomic ALD action affecting a chronic individual physiological condition than to a non-genomic ALD, which has increased due to exercise.

To clarify the effect of ALD on the $[\text{Na}^+]_{\text{sweat}}$ during exercise in a hot environment, we analyzed the cross-sectional relationship between the sweat rate, $[\text{Na}^+]_{\text{sweat}}$, and the plasma ALD concentration during rest and exercise in trained athletes.

Methods

Subjects

Eleven college-aged male track and field athletes (four long-distance runners, five sprinters, and two jumpers; mean \pm SE of age, 20 ± 1 years; height, 173 ± 1 cm; body mass, 60.6 ± 1.0 kg) participated in this study with the approval of the Institutional Human Subjects Committee, and after providing their written informed consent. These athletes had been training four times a week over a period of 2 years. We selected subjects who had similar body surface areas (1.73 ± 0.02 m²), body fat content ($9.73 \pm 0.28\%$), and fat free mass (54.7 ± 0.8 kg), in which the coefficients of variation for these subjects were within 10%, to minimize the differences in body size and body composition. The body fat content was estimated using the sum of two skinfold thickness (triceps and subscapular sites) measured with calipers (Takei, Tokyo) using the equations of Brožek et al. (1963) and Nagamine and Suzuki (1964). The body surface area was calculated according to the equation for Japanese by Fujimoto and Watanabe (1969).

At least 1 week before the exercise experiments, the maximal oxygen uptake ($\dot{V}O_{2\text{max}}$) was determined using a graded cycle-ergometer protocol [50 rpm, up to 1 kp (10 N) every 4 min until 3 kp, and then gradually up to 0.25 kp (2.5 N) every 1 min until exhaustion] in a room maintained at 25°C. The $\dot{V}O_{2\text{max}}$ of the subjects determined in this protocol was 48.7 ± 1.4 (range 42–59) ml kg⁻¹.

Protocol

On the day of the experiment, each subject reported to the laboratory without having eaten breakfast and then drank 200–300 ml of water between 8 and 9 a.m. They had refrained from heavy exercise for 24 h and also from the intake of salty food, alcohol, and caffeine for 17 h before arriving at the laboratory. The exercise experiments were started between 9 and 10 a.m. and then were performed in the sitting position on the counter chair of the cycle-ergometer in an environmental chamber maintained at 31°C (50% relative humidity) with the subjects wearing only swimming trunks. The schedule of the exercise experiment is shown in Fig. 1. After 20 min of rest, the subjects repeated three sessions of 20-min cycle exercise with a 5-min rest period. The exercise experiment was performed twice, with an interval of at least 1 week, with exercise intensities of 40 or 60% $\dot{V}O_{2\text{max}}$, presented random order. To minimize the effect of heat acclimatization on the thermoregulatory

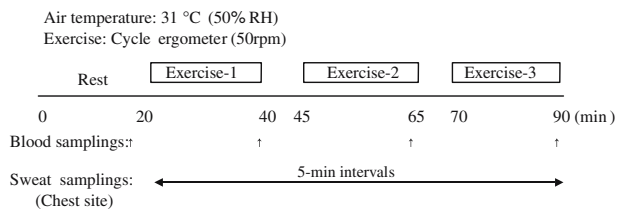


Fig. 1 Schedule of the experiment

responses, all exercise experiments were performed between August and September when the subjects had trained outdoors continuously for 5 months throughout the hot summer days.

Measurements

The total sweat loss ($m_{sw, tot}$) was determined from the nude body mass obtained before and after the exercise experiment, using a scale with an accuracy of 1 g (Chou, Kyoto, Japan). Local sweat samples were collected by capsules (7 cm²) on the chest at 5-min intervals using the filter paper method (Ohara 1966) during the exercise period. The chest sweat rate (SR_{ch} , mg cm⁻² min⁻¹) was determined based on changes in filter paper mass during a 5-min period, using a scale with an accuracy of 0.1 mg (Sartorius, BJ-60S). The $[Na^+]_{sweat}$ in filter paper was measured by atomic absorption spectrometry. Blood samplings of 10 ml were taken from the antecubital vein in the sitting position at rest and at the end of each exercise session. A 0.5-ml aliquot of blood was used to determine the hematocrit (Hct, microcentrifuge), plasma protein (PPC, refractometry), and hemoglobin concentrations (Hb, cyanometry), and 1.5-ml aliquot was centrifuged at 4°C and plasma was used to measure the plasma electrolyte concentrations (Na^+ and K^+ , flame photometry) and the plasma osmolality (Posmo, freezing-point depression, Vogel, Federal Republic of Germany). The remaining 8 ml of blood were centrifuged at 4°C, and the aliquots of plasma were frozen and stored at -90°C until the plasma ALD concentration was measured (radioimmunoassay). The percent changes in the plasma volume (ΔPV) during exercise were calculated based on changes in the Hct and Hb concentrations (Elkinton et al. 1946). In addition, the esophageal temperature (Tes) was measured every 30 s with a thermistor probe.

Statistics

The two-way repeated measures of ANOVA were used to analyze the difference between rest and exercise, and between the exercise intensities (40 and 60%

$\dot{V}O_{2max}$). Fisher LSD was used to locate differences when ANOVA revealed a significant interaction. Correlations between the variables were evaluated by means of a standard linear regression analysis. In all cases, significance was accepted at $P < 0.05$

Results

Body temperature responses

The Tes in 40% exercise increased gradually during first exercise session (Ex-1), and this variable reached a plateau at second (Ex-2) and third (Ex-3) exercise session. At the end of Ex-3 in 40% exercise, the Tes reached at $37.9 \pm 0.1^\circ C$. With 60% exercise, the Tes increased continuously during all the three exercise sessions, and this parameter did not reach plateau levels. At the end of Ex-3 in 60% exercise, the Tes reached at $39.0 \pm 0.1^\circ C$, and this parameter was significantly ($P < 0.01$) higher than that found in the 40% exercise group.

Sweating responses during exercise

The time courses of SR_{ch} and $[Na^+]_{sweat}$ during exercise increased gradually according to the increase of exercise sessions at either level, and these parameters were significantly ($P < 0.05$) higher and individual variations to be greater at 60% than at 40% exercise for each exercise session. The $m_{sw, tot}$ (percentage of the body mass) at 40 and 60% exercise were 1.32 ± 0.05 and $2.32 \pm 0.11\%$, respectively.

Blood parameters

The changes in the blood parameters during the experiment are shown in Table 1. The Hb, Hct, PPC, plasma K^+ and Na^+ , and Posmo significantly ($P < 0.05$) increased during the exercise sessions at either level. With the exception of the plasma K^+ and Na^+ , these blood parameters and ΔPV were significantly ($P < 0.05$) higher at 60% than at 40% exercise during each exercise session. The ALD significantly ($P < 0.05$) increased at 60% exercise, but it did not increase at 40% exercise during experiment, and it was significantly ($P < 0.05$) higher at 60% exercise than at 40% exercise during each exercise session.

Correlations between parameters

Figure 2 shows the relationship between SR_{ch} and $[Na^+]_{sweat}$ in each subject during exercise. In 10 of

Table 1 Changes in blood parameters during rest and exercise at two levels of intensity

	Rest	Exercise-1	Exercise-2	Exercise-3
Hb (g/dl)				
40%	14.3 ± 0.1	15.0 ± 0.2*	15.0 ± 0.2*	15.2 ± 0.2*
60%	14.5 ± 0.2	15.2 ± 0.2*,**	15.2 ± 0.2*,**	15.9 ± 0.2*,**
Hct (%)				
40%	42.4 ± 0.4	43.5 ± 0.5*	43.2 ± 0.5*	43.5 ± 0.7*
60%	42.7 ± 0.6	44.4 ± 0.6*,**	44.6 ± 0.6*,**	44.8 ± 0.7*,**
ΔPV (% changes)				
40%		-5.9 ± 1.4	-5.4 ± 1.3	-7.3 ± 1.7
60%		-9.5 ± 1.1**,	-9.9 ± 1.2**,	-12.3 ± 0.9**,
PPC (g/dl)				
40%	6.4 ± 0.1	6.7 ± 0.1*	6.8 ± 0.1*	6.9 ± 0.1*
60%	6.5 ± 0.1	7.1 ± 0.1*,**	7.1 ± 0.1*,**	7.3 ± 0.1*,**
Plasma Na ⁺ (meq/l)				
40%	142 ± 1	145 ± 1*	146 ± 1*	147 ± 1*
60%	142 ± 1	146 ± 1*	145 ± 1*	146 ± 1*
Plasma K ⁺ (meq/l)				
40%	4.2 ± 0.1	4.9 ± 0.1*	4.9 ± 0.1*	4.8 ± 0.1*
60%	4.2 ± 0.1	4.9 ± 0.1*	4.9 ± 0.2*	4.9 ± 0.2*
Posmo (mosmol/kg)				
40%	287 ± 1	294 ± 1*	294 ± 1*	295 ± 2*
60%	289 ± 2	296 ± 1*	298 ± 1*,**	300 ± 1*,**
Plasma aldosterone (pg/ml)				
40%	86 ± 13	116 ± 17	106 ± 12	83 ± 7
60%	98 ± 18	167 ± 29*,**	203 ± 39*,**	218 ± 40*,**

Values are mean ± SE for 11 subjects

Hb hemoglobin, Hct hematocrit, PPC plasma protein concentration, ΔPV changes of plasma volume, Posmo plasma osmolality

*Significant difference from rest values ($P < 0.05$); **significant difference between 40 and 60% exercise ($P < 0.05$)

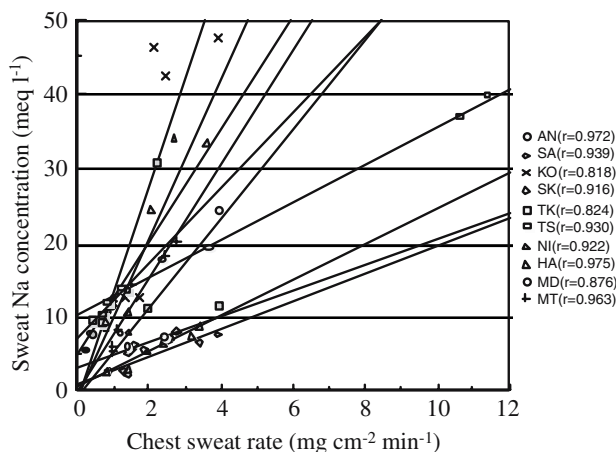


Fig. 2 Individual variations in the relationship between the chest sweat rate and the sweat Na⁺ concentration during exercise. Each data point indicates the values obtained during three exercise sessions at 40 and 60% exercise for each subject

these 11 subjects, the [Na⁺]_{sweat} significantly ($P < 0.05$) correlated with the increase in SR_{ch}, and there were greater variations in the [Na⁺]_{sweat} with a range from 5 to 45 meq l⁻¹ in approximately 2 mg cm⁻² min⁻¹ of SR_{ch}. The mean correlation between the parameters in all subjects was: [Na⁺]_{sweat} = 4.43SR_{ch} + 3.34 ($r = 0.926$, $P < 0.01$). Figure 3 shows the relationship

between the resting or mean exercising ALD and the mean [Na⁺]_{sweat} during exercise sessions at both 40 and 60% exercise. Since no significant correlation between the increase in SR_{ch} and [Na⁺]_{sweat} was observed in one of these 11 subjects, a symbol of this subject was deleted in Fig. 3 at 60% exercise. A significant negative correlation between the resting ALD and the [Na⁺]_{sweat} was observed at either level ($P < 0.05$). However, the mean ALD during exercise did not correlate with mean [Na⁺]_{sweat} while the tendency of a negative correlation between parameters was observed at either level (40%, $r = 0.511$; 60%, $r = 0.563$).

Discussion

In the present investigation, the mean [Na⁺]_{sweat} during exercise at either level significantly correlated with the resting ALD, but not with the exercising ALD. These results suggest that the level of the resting ALD concentration, which is a genomic action of ALD indicating chronic physiological conditions of the subjects, may be at least partially responsible for the individual variations in [Na⁺]_{sweat} during exercise in heat-acclimated athletes. In

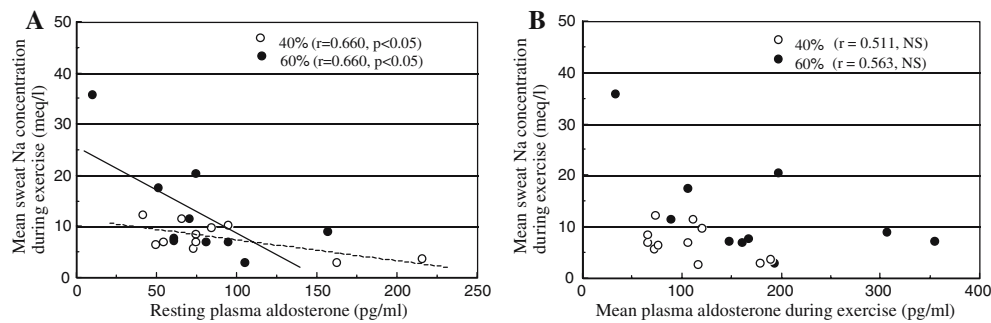


Fig. 3 The relationship between the resting plasma aldosterone (a) or the mean exercising plasma aldosterone concentration (b) and the mean sweat Na^+ concentration during exercise sessions

the present environmental conditions at 31°C (50% relative humidity), some sweating would be expected to have already occurred during the 20-min rest period, and sweat Na^+ reabsorption may be activated by a genomic action of the resting ALD level during the exercising period. Although many factors causing the sweat rate and sweat Na^+ variation during exercise, such as sweat gland responsiveness to ALD (Kirby and Convertino 1986), other endogenous steroids with mineral corticoid action, adrenal cortex (Sawka et al. 1996), and the activity of the sympathetic nervous system (Morgan et al. 2004) have been discussed, the genomic action of ALD is thus considered to primarily affect the sweat Na^+ variation during exercise in humans.

In the present investigation, the ALD and $[\text{Na}^+]_{\text{sweat}}$ were significantly ($P < 0.05$) higher at 60% exercise than at 40% exercise for each exercise session. Since a rapid ALD effect could theoretically increase Na^+ excretion at the secretory coil (Morgan et al. 2004), increases in the $[\text{Na}^+]_{\text{sweat}}$ during exercise observed in the present investigation might be attributable to an increase in the non-genomic action of ALD. However, the duct is thought to be the major site for the action of ALD triggering Na^+ reabsorption (Sato and Dobson 1970), which may take 1–6 h before the first phase of genomic action. In our present study, it is clear that some reabsorption of Na^+ occurred which was considered to be related to $[\text{Na}^+]_{\text{sweat}}$ during 1 h of exercise at both levels. Whether $[\text{Na}^+]_{\text{sweat}}$ during exercise can be mainly attributed to the rapid non-genomic or genomic action of ALD remains to be established. If the individual variation in $[\text{Na}^+]_{\text{sweat}}$ during exercise can be entirely induced by the non-genomic action of ALD, then a significant positive correlation between the mean $[\text{Na}^+]_{\text{sweat}}$ and the exercising ALD would be observed. In our present study, the mean $[\text{Na}^+]_{\text{sweat}}$ during exercise at either level did not significantly

at both 40% (open symbols and a dotted line) and 60% (closed symbols and a solid line). NS indicates no significant correlation

correlate with the exercising ALD and a tendency of a negative correlation between the parameters was observed in Fig. 3 (40%, $r = 0.511$; 60%, $r = 0.563$), thus suggesting that the rapid non-genomic action of ALD might be ineffective for the individual variations of $[\text{Na}^+]_{\text{sweat}}$ during 1 h of exercise. During the high intensity and short duration (less than 1 h) of exercise with greater sweat secretion containing greater Na^+ in a hot environment, however, the exercising ALD may be positively correlated with $[\text{Na}^+]_{\text{sweat}}$ because of the rapid non-genomic action of ALD.

In our present study, the $[\text{Na}^+]_{\text{sweat}}$ values, ranged from 5 to 45 meq l^{-1} in approximately $2 \text{ mg cm}^{-2} \text{ min}^{-1}$ of SR_{ch} as shown in Fig. 2, was lower than those values from previous studies (Allan and Wilson 1971; Verde et al. 1982). Since the experiments were performed between August and September, the lower $[\text{Na}^+]_{\text{sweat}}$ values observed in this study might therefore be attributable to heat-acclimation (Allan and Wilson 1971; Robinson and Robinson 1954). In addition, changes in the blood parameters following exercise shown in Table 1 were similar to several previous studies (Freund et al. 1991; Nose et al. 1991). Although the plasma Na^+ and K^+ levels at 60% exercise did not differ substantially from those at 40% exercise, both the SR_{ch} and $[\text{Na}^+]_{\text{sweat}}$ levels were significantly higher during 60% exercise than during 40% exercise. These results thus suggest the blood parameters to only have a slight influence over the SR_{ch} and $[\text{Na}^+]_{\text{sweat}}$ levels during the 60 min of exercising period. Furthermore, endurance exercise training has been observed to have different physiological and hormonal stimuli compared with sedentary control during exercise (Kirby and Convertino 1986; Hanane et al. 1977). In four long-distance runners in our study, however, there was no tendency for marked increase in blood parameters and for decrease in $[\text{Na}^+]_{\text{sweat}}$ at a given SR_{ch} during exercise.

In summary, the present study demonstrated that individual variations in the $[\text{Na}^+]_{\text{sweat}}$ in response to an increase in the SR_{ch} during exercise are related to the resting ALD, but not to the exercising ALD. As a result, the genomic action of ALD may, at least partially, be more strongly affected by the sweat Na^+ variation than by the rapid non-genomic action of ALD during exercise in humans.

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