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Original article

Improved sweat gland function during active heating in tennis athletes

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Abstract

Background: Relatively few studies on the peripheral sweating mechanisms of trained tennis athletes have been conducted. The purpose of this study was to compare the sweating capacities of tennis athletes against untrained subjects (controls).
Methods: Thirty-five healthy male volunteers participated including 15 untrained subjects and 20 trained tennis athletes (nationally ranked). Active heat generation was performed for 30 min (running at 60%VO_{2max}) in a climate chamber (temperature, 25.0°C ± 0.5°C; relative humidity, 60% ± 3%, termed active heating). Sweating data (local sweat onset time, local sweat volume, activated sweat glands, sweat output per gland, whole body sweat loss volume) were measured by the capacitance hygrometer-ventilated capsule method and starch-iodide paper. Mean body temperature was calculated from tympanic and skin temperatures.
Results: Local sweat onset time was shorter for tennis athletes ($p < 0.001$). Local sweat volume, activated sweat glands of the torso and limbs, sweat output per gland, and whole body sweat loss volume were significantly higher for tennis athletes than control subjects after active heating ($p < 0.001$). Tympanic and mean body temperatures were lower among tennis athletes than controls ($p < 0.05$).
Conclusion: These results indicate that tennis athletes had increased regulatory capacity of their sweat gland function.

Keywords: Activated sweat glands; Active heating; Sweat onset time; Sweat output; Sweating function; Tennis athletes

1. Introduction

For a given temperature, endurance-trained subjects generally display greater sweat output than their untrained counterparts through an elevated responsiveness of their sweat mechanism.[1,2] It is clear that endurance exercise training improves the sweating response to heat generation. In contrast, the sweating function was not improved in sprinters who had trained for at least 3 years as compared with untrained men, although the maximal oxygen uptake (VO_{2max}) was 33% greater in sprinters.[3] It is possible that sweat gland activity in sprinters during daily training is insufficient to improve their sweating capacity relative to distance runners, regardless of the enhanced VO_{2max}.[3]

Sprinters train at a high intensity over a relatively short duration, and the volume of training is relatively low in comparison to middle and long distance runners. Therefore, the oxygen cost of sprinting and related training may not appear to be high.[4]

A tennis player's metabolism during play in a hot environment generates an abundance of heat, which is primarily eliminated from the body by the evaporation of sweat.[5] The physical activity of playing tennis is characterized by quick starts and stops, repetitive overhead motions, and the involvement of several muscle groups during different strokes, which fluctuate randomly from brief periods of maximal or near maximal work to longer periods of moderate and low intensity activity.[6] In a previous study, the mean VO_{2max} values of tennis players were found to be 55 mL/kg/min in male players, and this level of aerobic metabolism was higher than the values reported for untrained middle aged subjects.[7]

However, it is not known whether daily training in tennis athletes can induce adaptations in sweating. Our hypothesis is that tennis athletes have an upregulation of their sweating mechanisms. Therefore, the present study compared the sweating responses and changes in sweat gland function during active heat generation between tennis athletes and untrained men.

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Table 1
Physical characteristics of the subjects (mean ± SD).

	Control ($n = 15$)	TA ($n = 20$)
Age (year)	23.6 ± 3.4	24.1 ± 3.7
Height (cm)	173.9 ± 5.2	175.0 ± 3.4
Weight (kg)	70.8 ± 6.1	67.2 ± 4.3
BSA (m^2)	1.9 ± 1.6	1.8 ± 1.4
%BF	22.5 ± 4.8	16.3 ± 2.9*
VO_{2max} (mL/kg/min)	43.2 ± 5.1	59.4 ± 3.7*

* $p < 0.001$, compared with control.
Abbreviations: %BF = percent body fat; BSA = body surface area; Control = untrained subjects; TA = tennis athletes; VO_{2max} = maximal oxygen uptake.

2. Materials and methods

2.1. Subjects

The physical characteristics of the subjects are summarized in Table 1. Thirty-five healthy male volunteers participated; 15 untrained subjects (had not performed regular physical activity in the previous 1 year, control) and 20 trained tennis athletes (nationally ranked) who had 7–12 years of training and who had been training an average of 25 h per week (an average of 5 days each week, self-reported). Percent body fat (%BF) and VO_{2max} were significantly higher for tennis athletes than control subjects. All of the subjects had lived their whole life in the city of Cheonan, Republic of Korea, which is located in the southwest part of Korea (126°52′N, 33.38′E) and extends northeast (130°4′N, 43.0′E). No differences in mean age, height, weight, or body surface area were observed between the groups. Each subject returned a informed written consent to participate in the study after being thoroughly acquainted with the purpose and the experimental procedures, as well as any potential risks. The subjects were fasted for 8 h and instructed to refrain from alcohol consumption or smoking 24 h before the test. The volunteers also refrained from medications during the testing period. All experimental protocols were approved by the Soonchunhyang University Research Committee, and the procedures complied with the 2000 Declaration of Helsinki of the World Medical Association.

2.2. Measurement and experimental procedure

All experiments were conducted in the city of Cheonan. The tests were performed in a climate chamber from 2:00 p.m. to 5:00 p.m., and the environmental conditions were maintained at 25.0°C ± 0.5°C, 60.0% ± 3.0% relative humidity, and 1 m/s air velocity. Subtle interpersonal variability result in human body temperatures being their lowest at 4:00 a.m., and at their highest from 4:00 p.m. to 6:00 p.m.[8] Thus, we conducted this experiment during 2:00 p.m. and 5:00 p.m. to control for the influence of the body temperature circadian rhythm, as described previously.[9–14] After the subjects arrived in the laboratory, urine specific gravity was tested with a urine strip (Uriscan, Seoul, Korea) to confirm hydration equilibrium. These test results were confirmed by visually inspecting the color change of the strip. The measurements were delayed until recovery to a normal range in cases in which the test strip color change exceeded the reference range of 1.010–1.025. The subjects were given ~5–7 mL/kg of tap water at 4 h before test on the day of the study, in order to maintain a sufficient level of hydration throughout the experiment. However, no subjects consumed water during the test.

2.3. Setting of physical loading and testing

To precisely determine the exercise intensity, a physical loading test was conducted 1 week before the experiment for all subjects. In the physical loading test, the VO_{2max} of each individual was measured. The VO_{2max} was obtained by applying the Bruce protocol with a Quinton Medtrack SR 60 treadmill (Quinton, Bothell, WA, USA) and a Quark Pulmonary Function Testing Lung Volumes Module 2 metabolic test system (COSMED, Rome, Italy). The average physiological responses to tennis match play have been reported to be rather modest, with mean exercise intensities generally less than 60%–70% of VO_{2max}.[7] Therefore, the physical loading test was terminated by subject declaration (until the subject became exhausted), and the 60%VO_{2max} was calculated.

2.4. Tympanic temperature (T_{ty}) measurements

After 60 min of rest, the T_{ty} was recorded during active heating (running on a treadmill for 30 min at 60%VO_{2max}). The T_{ty} was assessed by inserting a model TSK7 + 1 thermistor probe (Songkitopia, Inchen, Korea) with a small spring into the left ear canal (K923, Takara, Yokohama, Japan). The probe was connected to a model CF-T1 personal computer (Panasonic, Tokyo, Japan) and a model K-720 data logger (Technol Seven, Yokohama, Japan). As the thermistor probe contacted the tympanic membrane, the subject felt slight discomfort and could hear a scratching noise. The inner pinna was then filled with small cotton balls in order to secure the probe in place.[10]

2.5. Mean body temperature measurements

The skin temperatures (T) on the chest (T_{chest}), upper arm (T_{arm}), thigh (T_{thigh}), and leg (T_{leg}) were measured using model PXK-67 thermistor thermometers (Technol Seven) connected to a model K-720 data logger (Technol Seven).[10] The mean skin temperature ($\bar{T}_{sk}$) was calculated as $0.3 \times (T_{chest} + T_{arm}) + 0.2 \times (T_{thigh} + T_{leg})$.[1,7] The mean body temperature ($\bar{T}_b$) was calculated from the T_{ty} and $\bar{T}_{sk}$ using the formula by Ramanathan[15] as cited by Sugenoya and Ogawa:[16]
$\bar{T}_b = (0.9 \times T_{ty} + 0.1 \times \bar{T}_{sk})$.

2.6. Measurements of local sweating rate and sweat onset time

During heat loading, the sweating rate at the chest, abdomen, back, and thigh were continuously recorded by the capacitance hygrometer-ventilated capsule method. In brief, dry nitrogen gas was flowed at a constant flow rate of 500 mL/min into a capsule (9.621 cm^2 in area) attached to the skin at the point to be measured.[10] The humidity of the effluent gas was evaluated with a model H211 hygrometer (Technol Seven). The sweating

rates (local sweat volume) were recorded every 30 s with a personal computer (model CF-T1; Panasonic), and the results are expressed in mg/cm^2/min.[10]

2.7. Measurements of activated sweat glands (ASGs) and whole body sweat loss volume

The ASGs were measured by the starch-iodide paper to evaluate qualitative glandular activity. Strips of starch-iodide paper were attached to the chest, upper back, lower back, abdomen, upper arm, thigh, forearm, and calf-to-leg (all on the right side), and then scrubbed to obtain blue-black colored marks at the 90 s time point, after 10, 15, and 20 min, and at the end of the running. To obtain average ASGs (count/cm^2), 3 sectors of 0.5 × 0.5 cm were marked on the paper strip, and the number of sweat glands was counted.[11] Counting was performed by a single experienced researcher who counted each sector in 10–15 s. The average ASGs (count/cm^2) represented the sum of 3 sectors/(3 × 4).[11] Weight loss after one-off physical activity is caused by sweating, and therefore we measured the body weight to obtain the whole body sweat loss volume.

2.8. Statistical analysis

Values are presented as means ± SD. Analysis of covariance (ANCOVA) was performed to remove the possible confounding effects of the variable (%BF), and to explore group differences. *Post hoc* paired sample *t* tests were applied where appropriate, and were evaluated with a Bonferroni adjustment. Statistical significance was accepted at $p < 0.05$.

3. Results

3.1. T_{ty} and $\bar{T}_b$

The T_{ty} after active heating increased significantly in both control and tennis athletes groups ($p < 0.001$) (Table 2). It was increased by 0.42°C in tennis athletes, which was significantly lower than the controls (0.64°C) during the same period ($p < 0.05$). The $\bar{T}_b$ was increased significantly in control (0.74°C) and tennis athletes (0.49°C) ($p < 0.001$) and tennis athletes was significantly lower than the controls during the same period ($p < 0.05$).

Table 2
Comparison of the $\bar{T}_b$ between groups (mean ± SD).

	Group	Before active heating	After active heating	△ value
T_{ty}	Control	36.65 ± 0.14	37.29 ± 0.16#	0.64
	TA	36.73 ± 0.12	37.15 ± 0.13#	0.42*
$\bar{T}_b$	Control	36.12 ± 0.13	36.86 ± 0.14#	0.74
	TA	36.23 ± 0.11	36.72 ± 0.12#	0.49*

* $p < 0.05$, comapred with control; # $p < 0.001$, compared with before active heating.
Abbreviations: Active heating = running at 60%VO_{2max}; Control = untrained subject; TA = tennis athletes; $\bar{T}_b$ = mean body temperature; T_{ty} = tympanic temperature.

3.2. ASGs

ASGs measurements from 8 local regions are depicted in Table 3. In general, tennis athletes displayed a higher level of sweat gland activation as compared to controls. Tennis athletes displayed a significantly higher level of sweat gland activation as compared to control in the early stage (10–15 min). This trend was more distinctive in the limbs (upper arm, forearm, leg) than in the torso (chest, abdomen, upper back, lower back).

3.3. Local sweat onset time and local sweat volume

The sweat onset time was shorter by an average of 2.00 ± 0.33 min in the chest (1.91 ± 0.35 min, 24%), abdomen (2.06 ± 0.27 min, 25%), back (2.11 ± 0.30 min, 26%), and thigh (2.05 ± 0.38, 25%) in the tennis athletes than in controls (Table 4). The differences between the 2 groups were

Table 3
Comparison of activated sweat glands between groups (mean ± SD).

Local region	Group	Active heating (min)			
		10	15	20	30
Chest	Control	8.81 ± 5.98	51.50 ± 12.33#	67.88 ± 10.54	64.75 ± 11.02
	TA	16.38 ± 12.08*	60.38 ± 10.19#	67.50 ± 12.31	63.88 ± 11.46
Abdomen	Control	22.63 ± 14.53	67.88 ± 26.54#	79.88 ± 8.25	76.00 ± 12.65
	TA	40.38 ± 22.89*	79.38 ± 11.67#	84.50 ± 16.65	79.88 ± 19.53
Upper back	Control	13.63 ± 11.60	61.50 ± 17.49#	67.75 ± 10.39	69.88 ± 8.78
	TA	24.25 ± 14.75*	65.50 ± 10.94#	68.25 ± 9.74	65.88 ± 13.21
Lower back	Control	30.25 ± 12.55	78.75 ± 9.40#	85.50 ± 6.87	75.88 ± 10.93
	TA	60.00 ± 17.89*	88.88 ± 11.72#	83.50 ± 12.64	80.38 ± 17.83
Upper arm	Control	9.50 ± 5.08	36.88 ± 23.18#	74.25 ± 18.25	70.00 ± 12.29
	TA	15.50 ± 15.20*	62.25 ± 10.76**,#	76.13 ± 14.75	74.63 ± 17.46
Forearm	Control	13.38 ± 8.64	44.88 ± 19.25	74.38 ± 9.04	70.75 ± 9.91
	TA	21.75 ± 19.82*	68.13 ± 6.77**,#	78.88 ± 9.96	80.00 ± 10.28
Thigh	Control	24.50 ± 13.10	49.00 ± 7.63	50.75 ± 8.54	41.88 ± 7.85
	TA	43.38 ± 16.88**	48.50 ± 9.00#	48.50 ± 7.30	49.13 ± 13.40
Leg	Control	18.43 ± 10.53	25.78 ± 8.41#	32.65 ± 7.85	35.45 ± 6.87
	TA	37.26 ± 13.64**	45.21 ± 8.75**,#	46.70 ± 8.43*	43.85 ± 12.51

* $p < 0.05$, ** $p < 0.01$, compared with control; # $p < 0.001$ compared with previous level.
Abbreviations: Active heating = running at 60%VO_{2max}; Control = untrained subject; TA = tennis athletes.

Table 4
Comparison of local sweat onset time, local sweat volume, and sweat output per gland between groups during active heating (mean ± SD).

	Chest		Abdomen		Back		Thigh	
	Control	TA	Control	TA	Control	TA	Control	TA
Local sweat onset time (min)	7.86 ± 2.17	5.95 ± 2.34*	8.11 ± 2.08	6.05 ± 2.18*	8.04 ± 2.16	5.93 ± 2.54*	8.06 ± 2.16	6.01 ± 2.16*
Local sweat volume (mg/cm^2)	17.05 ± 6.41	24.89 ± 7.23*	9.85 ± 5.71	15.67 ± 8.05*	19.51 ± 7.85	31.02 ± 9.79*	16.04 ± 4.54	25.03 ± 8.13*
Sweat output per gland (μg/min)	9.97 ± 2.31	12.98 ± 2.04*	5.06 ± 2.07	6.52 ± 1.58*	9.58 ± 2.65	13.85 ± 2.42*	12.77 ± 2.32	16.95 ± 1.96*

* $p < 0.001$, compared with control.
Abbreviations: Active heating = running at 60%VO_{2max}; Control = untrained subject; TA = tennis athletes.

statistically significant ($p < 0.001$). The local sweat volume was greater, by 8.50 ± 2.58 mg/cm^2 on average in the chest (46%), abdomen (59%), back (59%), and thigh (56%) in the tennis athletes than in controls, and the difference in each measurement location was also significant ($p < 0.001$) (Table 4).

3.4. Sweat output per gland (SGO) and whole body sweat loss volume

The results of the SGO measurements are depicted in Table 4. The difference in every measurement between the groups was significant ($p < 0.001$). The biggest differences were evident for the back and thigh, and the smallest difference was noted for the abdomen (approximately one-third of the back and thigh). The whole body sweat loss volume was significantly higher in tennis athletes than in controls (481 ± 101 mL *vs.* 325 ± 87 mL) ($p < 0.001$).

4. Discussion

Many previous studies have reported adaptive responses in sweat gland function in runners (long distance runner or sprinter) as compared with untrained subjects. However, these adaptations have not been investigated in tennis athletes.

The present study demonstrated that sweat gland functions differ significantly between tennis athletes and control subjects. Local sweat volume, ASGs, SGO, and whole body sweat loss volume were greater in tennis athletes than in control subjects. Tennis is characterized by short intervals of maximal or near maximal work interspersed throughout longer periods of moderate and low intensity activity. Thus, these results suggest that long-term tennis training for players not only increases the VO_{2max}, but is sufficient to enhance sweating gland function to improve heat loss responses.

Sweating occurs via stimulation of the sweat glands upon neurotransmitter release from sympathetic cholinergic neurons. Thus, we hypothesized that the elevated peripheral sensitivity by long-term tennis training was due to heightened cholinergic sensitivity and glandular hypertrophy.[17,18]

In 2 previous studies, the potentiation of sweating after training was not related to central mechanisms, but rather to peripheral mechanisms.[17,19] However, increased sweating capacity from training has also been attributed to central mechanisms.[20] The latter study reported a shift to the left of the regression lines of the sweating rate to the $\bar{T}_b$ and sweat expulsions to the $\bar{T}_b$ relationships in individuals after training. Thermoregulatory sweating is initiated primarily by increased core and skin temperatures, with associated afferent neural signals integrated at the hypothalamus.[21] The central sudomotor mechanisms, which integrate the thermal information from the core and skin, generate the signals for activating sweat gland function.[19] Aerobic training improves endurance capacity and enhances heat dissipation by lowering the core temperature threshold for skin vasodilation and sweating.[22]

In this study, the local sweat onset time was significantly shorter in tennis athletes than in control subjects. The T_{ty} and $\bar{T}_b$ were significantly lower in tennis athletes as compared with control subjects after active heat generation, but the result was a negligible change (below 0.3°C). Furthermore, the T_{ty} remained below 38°C in both groups after 30 min of active heat generation. Therefore, the result of a significantly shorter local sweat onset time in tennis athletes than in control subjects is considered to be due to adaptive changes of the central sudomotor mechanism of perspiration.

Increases in skin blood flow and sweating are the primary heat dissipation mechanisms in humans. In a previous study, internal body temperatures reached the upper safe limit within 10 min of moderate exercise in multiple sclerosis patients[23] because their sudomotor functions are limited by the disease state. Greater increases in core temperature during intermittent exercise have been attributed to either a lower rate of evaporative heat loss[24] or reduced skin blood flow.[25,26]

Under the experimental conditions of this study, we presumed that tennis athletes had a higher metabolic rate and, as such, would generate more heat as compared to control subjects. Despite their relatively higher rates of heat production, the tennis athletes displayed lower T_{ty} and $\bar{T}_b$ after active heating, indicating that they were able to dissipate more heat through an efficient redistribution of blood flow and a higher sweat rate.

A previous study reported that skin blood flow was significantly higher in endurance-trained subjects in comparison to untrained subjects.[27] Hence, it was logical to hypothesize that the skin blood flow was improved by long-term training in tennis athletes who had participated in the study. Furthermore, the whole body sweat loss volume after active heating was greater in the tennis athletes as compared with control subjects in this study. It is likely that a higher sweat rate at a given relative intensity contributed to the better heat dissipation in

tennis athletes. Therefore, the efficient evaporative cooling caused by increased sweat gland function had resulted in lower T_{ty} and $\overline{T}_b$ in tennis athletes.

The discrepancy in body temperature and sweat responses of trained athletes between previous studies and the present one may reflect differences in the work load and living environments of study locales. In a similar investigation in which active heat generation through relative intensity to maximal aerobic capacity was used, trained athletes were found to have accelerated sweat responses.[28] In another study, active heat generation through absolute exercise intensity was associated with suppressed sweating in trained athletes.[29] In our study, the control subjects had body surface areas similar to the tennis athletes counterparts (1.9 ± 1.6 m^2 *vs.* 1.8 ± 1.4 m^2), but had a higher body mass and %BF (Table 1). The greater adiposity (%BF) could have partially limited heat dissipation, counteracting the heat sink effect.[27] Thus, in the present study, no influence of body morphology was identified.

5. Conclusion

The major finding of this study was that long-term tennis training improves the regulation of sweat gland function. This result was not due to a change in the central sudomotor mechanism, but rather peripheral sudomotor mechanisms.

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Authors' contributions

JBL conceived of the study, and participated in its design and coordination and helped to draft the manuscript; SBN performed the statistical analysis and drafted the manuscript. TWK has contributed to the data analysis and manuscript completed. All authors have read and approved the final version of the manuscript, and agree with the order of presentation of the authors.

Competing interests

None of the authors declare competing financial interests.

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