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RESEARCH ARTICLE

The effect of seasonal acclimatization on whole body heat loss response during exercise in a hot humid environment with different air velocity

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Abstract

Seasonal acclimatization from winter to summer is known to enhance thermoeffector responses in hot-dry environments during exercise whereas its impact on sweat evaporation and core temperature (T_{core}) responses in hot-humid environments remains unknown. We, therefore, sought to determine whether seasonal acclimatization is able to modulate whole body sweat rate (WBSR), evaporated sweat rate, sweating efficiency, and thermoregulatory function during cycling exercise in a hot-humid environment (32°C, 75% RH). We also determined whether the increase in air velocity could enhance evaporated sweat rate and sweating efficiency before and after seasonal acclimatization. Twelve males cycled for 1 h at 40% $\dot{V}O_{2max}$ in winter (preacclimatization) and repeated the trial again in summer (after acclimatization). For the last 20 min of cycling at a steady-state of T_{core}, air velocity increased from 0.2 (0.04) m/s to 1.1 (0.02) m/s by using an electric fan located in front of the participant. Seasonal acclimatization enhanced WBSR, unevaporated sweat rate, local sweat rate and mean skin temperature compared with preacclimatization state (all $P < 0.05$) whereas sweating efficiency was lower ($P < 0.01$) until 55 min of exercise. T_{core} and evaporated sweat rate were unaltered by acclimatization status (all $P > 0.70$). In conclusion, seasonal acclimatization enhances thermoeffector responses but does not attenuate T_{core} during exercise in a hot-humid environment. Furthermore, increasing air velocity enhances evaporated sweat rate and sweating efficiency irrespective of acclimated state.

NEW & NOTEWORTHY Seasonal acclimatization to humid heat enhances eccrine sweat gland function and thus results in a higher local and whole body sweat rate but does not attenuate T_{core} during exercise in a hot-humid environment. Sweating efficiency is lower after seasonal acclimatization to humid heat compared with preacclimatization with and without the increase of air velocity. However, having a lower sweating efficiency does not mitigate the T_{core} response during exercise in a hot-humid environment.

INTRODUCTION

Several studies (1–4) have documented that thermoregulatory strain can be significantly reduced following repeated passive exposure to a high ambient temperature during the summer period, commonly referred to as seasonal acclimatization. The reduction of thermoregulatory strain following seasonal acclimatization can be partly explained by the sudomotor adaptations such as an earlier onset of sweating and a greater sensitivity to increasing body temperature (1, 5–7). This enhanced sudomotor function promotes whole body heat loss and could potentially mitigate T_{core} during passive exposure in an environment which permits evaporation of sweat at a greater magnitude (i.e., hot-dry or thermoneutral) (3, 6, 7). However, whether seasonal acclimatization can lower thermoregulatory strain during exercise in an environment where sweat evaporation is limited (i.e., hot humid) remains unknown. Furthermore, current studies have not differentiated local to the whole body sweat loss (1–3, 5–8) during exercise in a hot humid environment before and after seasonal acclimatization to humid heat.

It is well known that evaporated sweat rate is greatly impaired in a hot-humid environment owing to the reduced vapor pressure gradient between the skin and the environment (9).Therefore, any increase in sweating would result in greater unevaporated sweat, thus a lower sweating efficiency ensues (10). Therefore, the increase in whole body sweat rate (WBSR) following seasonal acclimatization, as commonly observed by the previous studies (1–3, 5–7), may not be able to reduce thermoregulatory strain during exercise in a high vapor

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pressure environment, as evaporated sweat rate is greatly impaired by the reduced vapor pressure gradient between the skin and the environment. However, this issue has not been investigated as previous studies did not examine the evaporated and unevaporated sweat rate and sweating efficiency following seasonal acclimatization in a hot-humid environment.

Second, although the effect of air velocity on the change of sweating efficiency before and after seasonal acclimatization during exercise in a hot-humid environment has been investigated (11), the findings are counterintuitive to the established principles of heat exchange. In particular, Candas et al. (11) revealed that sweating efficiency was either unchanged or reduced with increasing air velocity before acclimation but it was higher after heat acclimation. This observation is in fact against the established principle of heat exchange where increasing air velocity in humid heat should theoretically enhance sweating efficiency due to a greater increase of evaporated sweat rate (12–14). This issue may have been confounded by a very small sample size (n = 4) as well as measurement apparatus such as using the less accurate weight scales, which was conducted using the old Potter beam balance system. This issue could be addressed by using high precision weight scales along with a greater number of participants to enhance its accuracy of evaporated and unevaporated sweat rate as well as allowing for proper statistical comparison. Using this technique, before and after seasonal acclimatization to humid heat with increasing air velocity in the latter half of the exercise would allow us to verify the findings of Candas et al. (11).

The aim of this study was first to systemically determine whether the increase of sudomotor function [local sweat rate (LSR) and WBSR], following seasonal acclimatization from winter to summer, could enhance evaporated sweat loss in a hot-humid environment. The second objective of this study was to determine whether the increase of air velocity could reduce unevaporated sweat rate and increase evaporated sweat rate before and after seasonal acclimatization, thereby enhancing sweating efficiency irrespective of acclimated status. As seasonal acclimatization has only been shown to minimally enhance LSR and WBSR (5, 15, 16) during passive exposure to dry heat and given the fact that even two weeks of active humid heat acclimation was unable to enhance evaporated sweat rate during exercise in a hot-humid environment (10), we therefore hypothesized that the increase in LSR and WBSR following seasonal acclimatization from winter to summer would not be able to enhance evaporated sweat rate and mitigate T_{core} during exercise in a hot-humid environment. As increasing air velocity has been shown to increase maximal evaporative cooling capacity in humid heat (14), we therefore hypothesized that the increase of air velocity would be able to enhance evaporated sweat rate and thus result in a higher sweating efficiency during prolonged exercise in a hot-humid environment before and after seasonal acclimatization to humid heat.

METHODS

Participants

Twelve healthy males were recruited for this study. All participants were not taking any medication and were familiar with the instrumentation and the protocol of this study. This study was approved by the Kobe University Human Ethics committee (No. 435) and performed in accordance with the latest version of the Declaration of Helsinki. Each participant provided verbal and written informed consent. Participants physical characteristics are summarized in Table 1. It was deemed appropriate to recruit only men as we have previously reported that during humid heat stress, core body temperature (17, 18) is affected by the menstrual cycle and oral contraceptive use, respectively, therefore, we did not want to introduce an additional confounding factor.

Experimental Overview

In each season (winter and summer), participants were asked to report to the laboratory on two occasions: *1*) pilocarpine iontophoresis and maximal oxygen consumption ($\dot{V}O_{2max}$) test, and *2*) exercise heat stress test. Each occasion was separated by at least 48 h and was conducted in the morning between 8 AM and 11 AM to minimize the effect of circadian rhythm on body temperature fluctuation, and performed 2 h postprandial. Furthermore, all participants were told to abstain from coffee and alcohol intake, as well as avoiding strenuous exercise 48 h before each testing. In both seasons, to avoid the effect of exercise training on thermoregulatory adaptations, all participants were told to minimize their outdoor physical activity or training less than or equal to three times per week, 1 mo before the testing. During the heat stress test, exercise was performed on an electronically braked cycle ergometer with a participant-specific set up for the seat, handle bars and pedals, which was maintained constant for each trial, within a participant. All trials were conducted in the beginning of January to the end of February (winter) and in August (summer), 2019. The average ambient temperature and humidity in Kobe city between winter and summer seasons were 7 (2.8)°C, 66 (10.3)% RH and 30 (2)°C, 71 (8)% RH respectively.

Pilocarpine Iontophoresis and $\dot{V}O_{2max}$ Test

Pilocarpine-induced sweating test was determined using the iontophoresis method (19) as an indication of the cholinergic sweat gland function, as well as an index of seasonal acclimatization from winter to summer (20). Before the data collection, subjects were seated quietly for 30 min in an environmental chamber (Model: FLC 2700 s, Fuji Medical Science, Japan) at an ambient temperature of 25°C and relative humidity of 50%, respectively. During this period, 1% (equivalent to 0.05 molar) pilocarpine (Tokyo Chemical

Table 1. *Physical characteristics of 12 participants across both winter and summer seasons*

	Winter	Summer	*P* Value
Age, yr	23 (3)	24 (3)*	<0.01
Height, cm	172.7 (6.9)	172.6 (6.9)	0.7
Weight, kg	64.5 (7.3)	64.2 (7.4)	0.7
BSA, m^2	1.76 (0.1)	1.76 (0.1)	0.7
Body fat, %	13.9 (2.6)	14.4 (3.8)	0.3
$\dot{V}O_{2max}$, $mL \cdot kg^{-1} \cdot min^{-1}$	44.4 (7.8)	39.9 (4.6)*	0.02

Values were expressed as means ± SD for 12 participants. *Significantly different from winter season ($P < 0.05$). BSA, body surface area.

Industry Co., Ltd., Japan) was iontophoretically applied onto the distal ventral forearm via a capsule (5.31 cm^2) dissolved in distilled water via gauze. As according to previous literature, this molar concentration was sufficient to elicit nearly maximal cholinergic sweat output (21). A 1.5-mA iontophoresis current was applied for 5 min between an electrode on the pilocarpine capsule (anode) and a flexible plate-electrode (cathode, HV-LLPD; Omron Healthcare, Japan) was attached to the wrist. Immediately after iontophoresis, the iontophoresis capsule was removed, the skin surface was then wiped with clean gauze, and another sweat capsule (5.31 cm^2) was attached at the same location for sweat rate measurement using the ventilated capsule method with an airflow of 0.6 $L \cdot min^{-1}$ (15). The duration of this procedure was 12 min in total but only the last 10 min was averaged for data analysis. At the end of the procedure, the capsule was removed and an activated sweat gland (ASG) test was performed using the starch-iodine technique as according to the method from Gagnon et al. (22). Sweat gland output (SGO) on the forearm was calculated as the ratio between the averaged forearm sweat rate and the ASG on the forearm region.

After the activated sweat gland measurement, all participants completed a $\dot{V}O_{2max}$ test on the cycle ergometer (Aerobike, 75XLIII, Konami, Japan) with workload starting at 20 W for the first 2 min and then increased 20 W every minute until volitional exhaustion. $\dot{V}O_{2max}$ was determined by the plateaued of oxygen consumption ($\dot{V}O_2$) with an increasing workload. The linear relation between power output and $\dot{V}O_2$ was subsequently used to calculate workload for the heat stress test, as 40% $\dot{V}O_{2max}$.

Exercise Heat Stress Test

Before the exercise heat stress test, participants voided their bladder and provided a urine sample to confirm euhydration status with no participants exceeding the value of 1.02 (16). Immediately after this procedure, a nude body weight was measured and participants were instrumented in a thermoneutral environment (25°C, 50% RH, model: SR3000, Nagano Science, Japan) for ~35 min before entering the heat chamber (Model: FLC 2700 s, Fuji Medical Science, Japan) set at 32°C, 75% RH and with an air velocity of 0.2 (0.04) m/s, respectively. After entering the heat chamber, participants mounted the cycle ergometer connected to the modified Potter beam balance system (see *Whole body sweat rate measurement* below for detail) for 30 min to allow for further instrumentation as well as allowing 5-min baseline measurements to take place. Participants then performed 1 h cycling at the same 40% of their $\dot{V}O_{2max}$ calculated from the winter period. The frontal windspeed for the first 40 min of the exercise was 0.2 (0.04) m/s (lower air velocity) and was subsequently increased to 1.1 (0.02) m/s (higher air velocity) from 40 min onward by two small sized electric fans (IRIS OHYAMA, Japan), using a 30-cm blade diameter located 0.5 meter directly in front of the participants. Throughout the entire heat stress trial, T_{core}, mean skin temperature (T_{sk}), skin blood flow on the forearm and back ($SkBF_{Forearm}$, $SkBF_{Back}$), LSR on the forearm and back ($LSR_{Forearm}$, LSR_{Back}), WBSR, and unevaporated sweat rate were recorded continuously. Sweat ion concentration was measured at the following time intervals: 20, 40, and 60 min during exercise. ASG on the forearm ($ASG_{Forearm}$) and on the back (ASG_{Back}) were collected during the interval between 35 and 40 min (lower air velocity) and again between 50 and 55 min (higher air velocity), in duplicate. SGO was calculated as the ratio between LSR and ASG at each anatomical site (forearm and on the upper back) and expressed as SGO_{Back} and $SGO_{Forearm}$. $\dot{V}O_2$ was measured at 55 min during exercise to ensure the same metabolic heat production across both winter and summer season.

Justification of the Exercise Protocol

Pilot testing indicated that a thermoregulatory steady-state was obtained after 40 min of cycling. Therefore, an additional 20 min at the air velocity of 1.1 (0.02) m/s (higher air velocity) was sufficient to significantly increase evaporated and unevaporated sweat rate and sweating efficiency compared to 0.2 (0.04) m/s (lower air velocity). The mean value and the standard deviation of the air velocity inside the chamber was 0.2 (0.04) m/s and this was increased to 1.1 (0.02) m/s as this velocity did not result in an increase in the electrical noise of our esophageal temperature measurement as well as the electrical signal of our WBSR measurement.

Measurements

Anthropometric.

Participant height and mass were measured using a stadiometer (Seca, Germany; accurate to 0.1 cm) and scale (Mettler-Toledo, Germany; accurate to 10 g), from which the body surface area (BSA) was estimated (23). Percent of body fat was determined by skin fold measurement according to the guideline from Jackson et al. (24) at three anatomical sites (Abdomen, Triceps and Suprailiac).

Core and skin temperature.

Esophageal or rectal temperature were used as an indicator of T_{core}. Ten participants self-inserted an esophageal thermocouple to the distance of quarter of their standing height. Two participants were unable to tolerate the insertion of the esophageal thermometer and they self-inserted a rectal thermometer 12 cm past the anal sphincter. The methods of core temperature within participants was consistent between seasons. T_{sk} was computed by measuring four different local skin temperatures on the calf, thigh, chest and forearm area by thermocouples and secured using surgical tape (3 M Micropore). Area-weighted T_{sk} was calculated according to the equation of Ramanathan (25). An additional thermocouple was secured on the back in order to quantify the effect of local skin temperature on LSR_{Back}. T_{core} and skin temperatures were connected to a data logger with a sampling rate of 1 Hz and displayed continuously by MX100 software (Yokogawa, Japan). Data were then expressed as a 5-min average until the end of the trial. To account for the relative influence of T_{core} and T_{sk} on the activation of heat loss responses (26), mean body temperature (T_B) was calculated as: $0.8\,T_{core} + 0.2\,T_{sk}$ (27).

Respiratory.

Expired respiratory gases were collected and analyzed for $\dot{V}O_2$ and carbon dioxide elimination (CO_2), minute ventilation ($\dot{V}E$), and respiratory exchange ratio (RER), using an online, breath-by-breath system (AE-300S; Minato Medical

Science, Japan) using a 30-s average. The system was calibrated before each trial using a precision gas mixture.

Cardiovascular.

Heart rate (HR) was recorded from detection of R-R intervals (Polar Vantage XL, Finland). SkBF was measured by using laser Doppler flowmetry (ALF21; Advance, Japan) and expressed as arbitrary unit (au). This procedure began by affixing two laser Doppler probes on the forearm and at the back using an adhesive ring tape (3M, Transpore, USA) and taking care to avoid veins. To prevent movement artefact on the laser Doppler sites, both hands were relaxed on the handle bar of the cycle ergometer throughout the entire trial.

Local sweat rates and sweat ion measurement.

LSR was measured using a ventilated capsule method (15). To account for regional variation in sudomotor responses during exercise (28), two custom made capsules (3.14 cm^2) were tightly sealed on the forearm and on the upper back by collodion glue and ventilated with dry air at 0.5 $L \cdot min^{-1}$. The effluent gas was sensed for humidity and temperature (Vaisala, Finland) and being recorded continuously by MX100 software (Yokogawa, Japan). Sweat ion concentration (NaCl, mmol/L) on the back was measured by the sweat patch technique in duplicate and analyzed immediately after the exercise by a sweat conductivity analyzer (Wescor, India) as previously described by Buono et al. (29).

Whole body sweat rate measurement.

WBSR and unevaporated sweat rate were measured by the modified Potter beam balance system (Takei Scientific Instruments S-17206, Japan) as according to the method from Alber-Wallerström and Holmer (30). The graphical illustration of this apparatus is outlined in Fig. 1. The cycle ergometer was suspended over the aluminum platform with the legs of the platform being attached to the high precision weight scale (Mettler-Toledo, cc150, Germany; accurate to 5 g, readability to 0.2 g) for the detection of the body weight changes from the participants during the exercise. Another high precision weight scale (Mettler-Toledo, B60, Germany; accurate to 2 g, readability to 0.1 g), was attached to a tray filled with paraffin oil and water, which was placed directly below the cycle ergometer but above the first weight scale with no physical contact in between. As unevaporated sweat drips of the skin and falls into the pan, the sweat cannot evaporate from the oil-filled pan. The tray pan was big enough to successfully capture most of the sweat drippage from the participants without any evaporation. To further reduce the movement artifact on our recording of WBSR and unevaporated sweat rate, all participants were required to stop cycling and remained absolutely still during the last 30 s of every 5-min interval. The output signal of those high precision weight scales was connected to a data acquisition system (Biopac MP100) with a sampling rate of 200 Hz and displayed in real time (Biopac Acknowledge Software, Goleta, CA).

WBSR, evaporated and unevaporated sweat rate, and sweating efficiency were calculated as according to the equations of Alber-Wallerström and Holmer (30). WBSR (g/m^2/min) at every 5 min was calculated as follows:

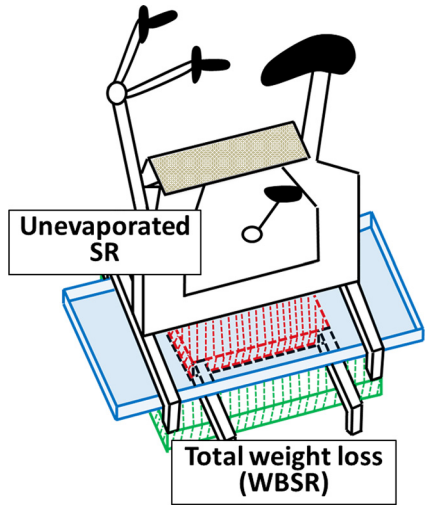

Figure 1. A graphical illustration of the measurement apparatus to accurately record whole body sweat rate (WBSR) and unevaporated sweat rate continuously during exercise in warm humid environment. The cycle ergometer was mounted on the aluminum platform with its leg being attached to the green color weight scale. The change of body weight from baseline measurement period was used as an index of WBSR. Another weight scale marked in red color with the attachment of a paraffin oil and water filled tray was placed directly below the cycle ergometer but above the first weight scale with no physical contact in between. The sweat dripped from the body was then captured by this paraffin oil and water filled tray and was used as an index of unevaporated sweat rate.

$$WBSR = \frac{\left[\frac{Body\ Weight_{post} - Body\ Weight_{pre}}{BSA}\right]}{Time}, \quad (1)$$

where Body $Weight_{post}$ (kg) was the average body weight at the end of the 5-min stage, Body $Weight_{pre}$ (kg) was the average body weight from the previous stage, Time (min) was 5 min and BSA (m^2) was the body surface area of each subject.

Unevaporated sweat rate (g/m^2/min) at every 5 min was calculated as follows:

$$Unevaporated\ sweat\ rate = \frac{\left[\frac{Dripped\ Sweat_{post} - Dripped\ Sweat_{pre}}{BSA}\right]}{Time}, \quad (2)$$

where Dripped $Sweat_{post}$ was the average dripping sweat at the end of the 5-min stage, Dripped $Sweat_{pre}$ (g) was the average dripping sweat from the previous stage, Time (min) was 5 min, and BSA (m^2) was the body surface area of each subject.

Evaporated sweat rate (g/m^2/min) at every 5 min was calculated as follows:

$$\text{Evaporated sweat rate} = \text{WBSR} - \text{Unevaporated sweat rate}, \quad (3)$$

where WBSR is the average WBSR at each 5 min stage and Unevaporated sweat rate is the average 5 min unevaporated sweat rate at the end of the 5 min stage.

Sweating efficiency (%) at every 5-min was calculated as follows:

$$\frac{Evaporated\ sweat\ rate}{WBSR} \times 100. \quad (4)$$

Partitional Calorimetry

All of the partitional calorimetry analysis was calculated using the equations from Cramer and Jay (31).

Metabolic heat production (H_{Prod}) ($W \cdot m^{-2}$) was calculated as the following: $H_{Prod} = M - W$, where M is the metabolic rate ($W \cdot m^{-2}$) and W was the external load in $W \cdot m^{-2}$. M was calculated as the following:

$$M = \dot{V}O_2 \times \frac{\left\{\left[\left(\frac{RER-0.7}{0.3}\right) \times 21.13\right] + \left[\left(\frac{1.0-RER}{0.3}\right) \times 19.62\right]\right\}}{60} \times 1,000, \quad (5)$$

where $\dot{V}O_2$ is the volume of oxygen consumption in L/min and RER is the respiratory exchange ratio.

Dry heat exchange ($C_{skin} + R_{skin}$) from the skin was calculated from the following:

$$C_{skin} + R_{skin} = \frac{\left\{\frac{(t_{sk}-t_0)}{\left(R_{cl}+\frac{1}{h_r f_{cl}}\right)}\right\}}{BSA} (W \cdot m^{-2}), \quad (6)$$

where t_{sk} is the mean skin temperature (°C) and t_0 (°C) is the ambient temperature. R_{cl} is the dry heat transfer resistance of clothing ($m^2 \cdot °C \cdot W^{-1}$) and h is the combined dry heat transfer coefficient from convective heat transfer coefficient (h_c) and radiant heat transfer coefficient (h_r). To account for different air velocity (0.2 m/s vs. 1.1 m/s), h_c was calculated as the following: $8.3 \times (0.07 + 0.0043.f_{Ped} + v_{air})^{0.86}$. h_r was calculated according to the standard equation (31) after accounting for nondimensional emissivity of the body surface (ε = 0.98) (exposed skin) and fraction of the body surface participating in radiant heat transfer (A_r/A_D) after adjusting for posture (A_r/A_D = 0.70).

Maximal evaporative cooling capacity of the environment (E_{max}) was calculated as the following:

$$E_{max} = \frac{\frac{(p_{skin,sat}-P_a)}{\left(R_{e,cl}+\frac{1}{h_e f_{cl}}\right)}}{BSA} \quad (7)$$

($W \cdot m^{-2}$), where P_{sk} is the vapor pressure at the skin surface with the assumption of 100% relative humidity at the skin surface of the participants and P_a is the partial vapor pressure of the ambient air in kPa. $R_{e,cl}$ is the evaporative resistance of clothing in $m^2 \cdot kPa \cdot W^{-1}$ (32) and h_e is the evaporative heat transfer coefficient.

Hemological Variables

In each season before and after exercise, capillary blood samples were taken in duplicate. Whole blood was used to measure hemoglobin concentration (HemoCue Hb+ 201 System, Ängelholm, Sweden). The capillary blood samples were subsequently centrifuged at 805 *g* for 10 min to determine hematocrit and plasma volume changes according to Dill and Costill (33).

Data and Statistical Analyses

We used 1-min averages from $LSR_{Forearm}$, LSR_{Back} to determine the onset of sweating by plotting the values against T_{core} and T_B. Subsequently, the onset of sweating was determined by a substantial deviation from the baseline values using segmental regression as according to Cheuvront et al. (34). To determine the onset and thermal sensitivity of the WBSR, we used the linear regression analysis by plotting the WBSR against T_{core} and T_B at every 5 min interval until the end of the 40 min (lower air velocity) and the *y*-intercept was used as the onset and the slope of the regression line was used as an index of thermal sensitivity of WBSR. The decision to use linear regression was because we could not determine both of the onset and the sensitivity of WBSR by using segmented regression.

Descriptive values were obtained and reported as means and standard deviation (SD). Homogeneity of variance was examined by Levene's test and the normality of the data was examined by the Kolmogorov–Smirnov test. Sphericity was assessed and where the assumption of sphericity could not be assumed, adjustments to the degrees of freedom were made ($\varepsilon > 0.75$= Huynh–Feldt; $\varepsilon < 0.75$ = Greenhouse–Geisser). The thermoregulatory variables (T_{core} and T_{sk}, $LSR_{Forearm}$, LSR_{Back}, LSR_{Mean}) and cardiovascular data at rest and the data of the pilocarpine induced sweating test were analyzed by paired samples *t* tests. However, during exercise and comparing the effect of air velocity, all variables were analyzed by two-way repeated measure ANOVA (seasons × time points); in the cases where main or interaction effects occurred, post hoc pairwise analyses were performed, using paired samples *t* tests with Bonferroni corrections, if appropriate. The comparison of different air velocities (lower and higher air velocities) for most variables were made on the interval between 35 and 40 min (lower air velocity) and 55 and 60 min (higher air velocity) except for LSR, ASG, and SGO were made on 50–55 min (higher airflow). Statistical significance was set at $P \leq 0.05$. All data were analyzed by GraphPad Prism software (Prism, v. 8, San Diego, CA).

RESULTS

Physical Characteristics

Following seasonal acclimatization, subjects had a lower $\dot{V}O_{2max}$ ($P < 0.01$, Table 1) compared with the winter season whereas percent of body fat and BSA were similar between seasons (all $P > 0.1$).

Pilocarpine-Induced Sweating

Pilocarpine-induced sweat rate and sweat gland output per gland on forearm were significantly higher in summer than in winter [summer vs. winter; $LSR_{Forearm}$: 0.80 (0.16) vs. 0.60 (0.15) $mg/cm^2/min$; $SGO_{forearm}$: 5.31 (0.84) vs. 4.12 (0.95) μg/gland/min, all $P < 0.01$], but $ASG_{forearm}$ was not different between summer and winter seasons [151.4 (29.3) vs. 147.4 (27.8) glands/cm^2, $P = 0.65$]. Average forearm skin temperature in the pilocarpine-induced sweating test was significantly higher in summer than in winter [33.1 (0.4)°C vs. 31.5 (1.2)°C, $P < 0.01$].

Body Temperature

T_{core} at baseline did not differ between summer and winter ($P = 0.40$, Fig. 2). T_{core} increased with time during exercise ($P < 0.01$) and reached steady-state (plateau in T_{core}) from 40 min (lower air velocity) onward until the end of the exercise (higher air velocity). T_{core} was not different between summer and winter ($P = 0.77$) and showed no interactional effect between seasons and timepoints ($P = 0.60$, Fig. 2).

Mean T_{sk} (mean skin temperature) at baseline did not differ between summer and winter seasons ($P = 0.18$, Fig. 2). In both seasons, T_{sk} increased with time during exercise ($P < 0.01$)

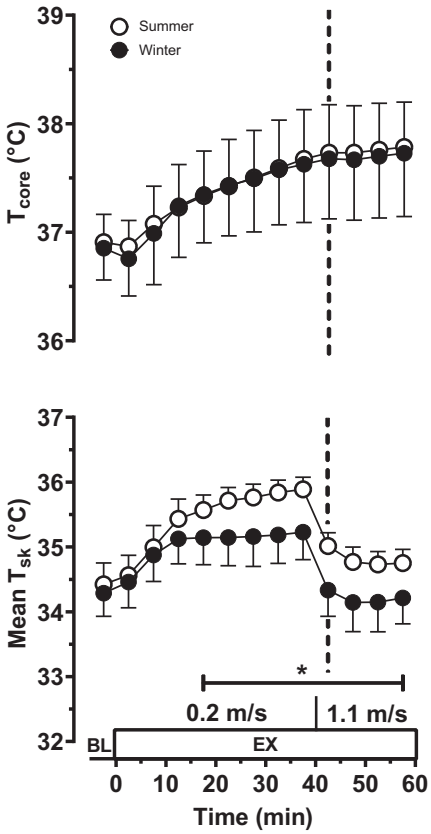

Figure 2. Mean (SD) core (T_{core}, $n = 12$) and mean skin temperature (T_{sk}, $n = 12$) during exercise with the air velocity of 0.2 m/s and 1.1 m/s in both winter and summer seasons. *Significantly different from winter season ($P < 0.05$).

until the end of the 15 min and subsequently remained unchanged until the end of the 40 min (Low air velocity, $P > 0.05$). At higher air velocity, T_{sk} decreased in a stepwise manner (>0.8°C, $P < 0.01$) from the end of the 40 min to the end of the 50 min and remained unchanged until the end of the exercise ($P = 1.00$). Furthermore, the rise of T_{sk} ($P < 0.01$) from baseline was different between seasons and showed an interactional effect ($P < 0.01$). Specifically, T_{sk} was higher in summer than in winter season from 10 min of the exercise until the end of exercise (all $P < 0.01$). Regional skin temperatures responded differently before and after seasonal acclimatization. For example, forearm skin temperature at baseline was higher in summer than in winter [34.4 (0.3)°C vs. 33.6 (0.6)°C, $P < 0.01$] whereas back skin temperature at baseline was lower in summer than in winter [34.2 (0.6)°C vs. 34.9 (0.4)°C, $P < 0.01$]. During exercise, forearm skin temperature increased with time ($P < 0.01$) and plateaued at the end of the 40 min (lower air velocity). At a higher air velocity of 1.1 m/s, forearm skin temperature showed a 0.8°C decrease compared with lower air velocity of 0.2 m/s ($P < 0.01$). Seasonal acclimatization had a significant effect on forearm skin temperature such that it was higher in summer than in winter [35.4 (0.1)°C vs. 34.5(0.2)°C, $P < 0.01$] at every time point during exercise and showed no interactional effect between seasons and time points ($P = 0.90$). Back skin temperature during exercise was not different between seasons ($P = 0.40$) but an interaction effect (season × time) was observed ($P < 0.01$), with a lower back skin temperature in summer than in winter, during the first 15 min of the exercise but was not significantly different thereafter. Furthermore, back skin temperature was not different between lower and higher air velocities ($P = 0.10$).

Sudomotor Responses

$LSR_{Forearm}$, LSR_{Back} at baseline were higher in summer than in winter (all $P < 0.01$, Fig. 3). In both seasons, $LSR_{Forearm}$, LSR_{Back} increased during exercise (all $P < 0.01$) but differed between seasons (all $P < 0.01$) and showed an interactional effect between seasons and time points (all $P < 0.01$). Specifically, LSR_{Back} was higher in summer than in winter season at the onset of the exercise and remained higher throughout irrespective of air velocity. Although $LSR_{Forearm}$ in summer season only became higher than winter season from 5 min onward. In line with this observation, summer season had a higher SGO_{Back} and $SGO_{Forearm}$ than winter season (all $P < 0.05$) but $ASG_{Forearm}$ and ASG_{Back} did not differ between seasons (all $P > 0.60$). No interactional effects were observed between seasons and time points (all $P > 0.10$). The T_{core} onset of sweating at all skin sites (forearm and back) was not different between seasons (all $P > 0.70$, Table 2) but the sensitivity of LSR_{Back} with changing T_{core} was greater in summer than in winter season (all $P < 0.05$, Table 2). However, when expressing the LSR_{Back} and $LSR_{Forearm}$ using T_B, neither the T_B onset nor the sensitivity with changing T_B showed any differences between summer and winter seasons (all $P > 0.08$, Table 2). Similarly, the T_{core} and T_B onset for WBSR were not different between seasons (all

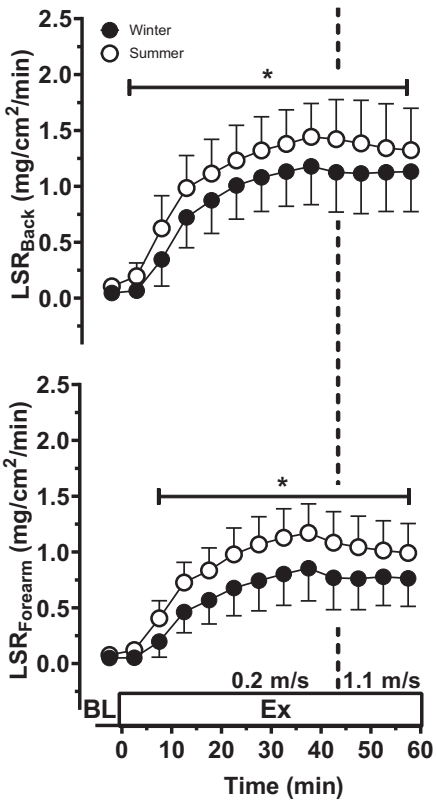

Figure 3. Means (SD) of local sweat rate (LSR_{Back}, $LSR_{Forearm}$, $n = 12$) during exercise with the air velocity of 0.2 m/s and 1.1 m/s in both winter and summer seasons. *Significantly different from winter season ($P < 0.05$).

Table 2. *Sweating onset and thermal sensitivity during exercise for LSR and WBSR across both winter and summer seasons*

		LSR			WBSR	
		Winter	Summer		Winter	Summer
Back						
T_{core}	Threshold, °C	36.9 (0.40)	36.9 (0.30)	Threshold, °C	36.7 (0.30)	36.8 (0.30)
	Slope, mg/cm^2/min/°C	3.0 (4.80)	4.3 (5.50)*	Slope, g/m^2/min/°C	7.6 (3.10)	12.9 (6.40)*
T_B	Threshold, °C	36.5 (0.30)	36.4 (0.30)	Threshold, °C	36.3 (0.30)	36.4 (0.20)
	Slope, mg/cm^2/min/°C	2.7 (3.00)	3.6 (5.40)	Slope, g/m^2/min/°C	8.0 (3.00)	10.8 (4.60)*
Forearm						
T_{core}	Threshold, °C	37.0 (0.40)	36.9 (0.30)			
	Slope, mg/cm^2/min/°C	1.5 (0.70)	3.8 (5.80)			
T_B	Threshold, °C	36.6 (0.30)	36.5 (0.30)			
	Slope, mg/cm^2/min/°C	1.6 (0.80)	2.7 (3.30)			

Values were expressed as means ± SD for 12 participants. *Significantly different from winter season ($P < 0.05$). LSR, local sweat rate; T_{core}, core temperature; Mean, mean value of back and forearm; T_B, mean body temperature; WBSR, whole body sweat rate.

$P > 0.50$), whereas the sensitivity of the WBSR with changing T_{core} and T_B were higher in summer than in winter season (all $P < 0.01$). Air velocity had a significant effect on $LSR_{Forearm}$ and LSR_{Back} such that they were all lower ($P < 0.01$, Table 3) at higher air velocity (between 50 and 55 min) than at lower air velocity (from 35 to 40 min) in both winter and summer seasons. However, $ASG_{Forearm}$ was only lowered in summer season (season by air velocity interaction: $P < 0.01$).

WBSR and unevaporated sweat rate increased with time during exercise (all $P < 0.01$, Fig. 4). Evaporated sweat rate increased during exercise ($P < 0.01$) whereas sweating efficiency decreased during the first 40 min of the exercise ($P < 0.01$) and increased significantly at the higher air velocity (1.1 m/s) ($P < 0.01$). All variables (WBSR, evaporated sweat rate, unevaporated sweat rate, and sweating efficiency) were different between seasons (Fig. 5) (all $P < 0.01$) and interactions between seasons and time points were observed (all $P < 0.05$). Specifically, WBSR during summer was higher than in winter, from 15 min onward until the end of the exercise (Fig. 4). Similarly, unevaporated sweat rate was higher ($P < 0.01$) in summer than in winter from 15 min to the 55 min of the exercise, whereas evaporated sweat rate was only higher (all $P < 0.05$) in summer than in winter at time point 5 and 10 min, but was not significant thereafter (all $P > 0.08$). Sweating efficiency on the other hand showed an opposite result; sweating efficiency in summer was lower ($P < 0.01$) than in winter from the 15 min of the exercise to the end of 55 min. air velocity had a significant effect (all $P < 0.05$, Table 3) on WBSR, unevaporated and evaporated sweat rate, and sweating efficiency. In particular, increasing air velocity progressively lowered WBSR in summer whereas it only lowered WBSR at 50 min in winter (seasons by air velocity interaction: $P < 0.01$). However, increasing air velocity was effective to lower unevaporated sweat rate ($P = 0.01$) in both seasons (winter and summer). Furthermore, increasing air velocity was effective to enhance both evaporated sweat rate and sweating efficiency (all $P < 0.05$) in both seasons.

Sweat ion concentration (NaCl, mmol/L) increased with time ($P < 0.01$) during exercise and was different between seasons ($P < 0.01$). Furthermore, an interactional effect was observed between seasons and time points ($P = 0.04$). Specifically, sweat ion concentration was lower during

Table 3. *The value of the thermoregulatory markers at baseline and at the lower (0.2 m/s) and higher velocities (1.1 m/s) in acute humid heat exposure as well as after seasonal acclimatization*

	Winter			Summer		
	BL	0.2 m/s	1.1 m/s	BL	0.2 m/s	1.1 m/s
T_{core}, °C	36.8 (0.30)	37.6 (0.50)	37.7 (0.60)#	36.9 (0.30)	37.7 (0.50)	37.8 (0.40)#
T_{sk}, °C	34.3 (0.30)	35.2 (0.40)	34.2 (0.40)#	34.4 (0.30)	35.9 (0.20)*	34.8 (0.20)#*
T_B	36.3 (0.30)	36.9 (0.4)	37.0 (0.50)#	36.4 (0.20)	37.0 (0.30)	37.2 (0.40)#
WBSR, g/m^2/min		7.4 (2.60)	6.7 (1.60)#		9.9 (3.40)*	7.9 (2.30)#*
Evaporated sweat rate, g/m^2/min		4.2 (0.60)	5.2 (0.80)#		4.6 (0.60)	5.6 (0.80)#
Unevaporated sweat rate, g/m^2/min		3.2 (2.30)	1.5 (1.00)#		5.3 (3.00)*	2.2 (1.50)#*
Sweating efficiency, %		63.3 (21.20)	79.8 (12.10)#		51.2 (16.40)*	75.1 (13.40)#*
LSR_{back}, mg/cm^2/min	0.1 (0.02)	1.2 (0.30)	1.1 (0.40)#	0.1 (0.10)	1.4 (0.30)*	1.3 (0.4)#*
$LSR_{forearm}$, mg/cm^2/min	0.1 (0.01)	0.9 (0.30)	0.8 (0.30)#	0.1 (0.02)	1.2 (0.30)*	1.0 (0.30)#*
ASG_{back}, glands/cm^2		115.3 (21.10)	111.3 (24.40)		115.1 (17.30)	107.8 (14.70)
$ASG_{forearm}$, glands/cm^2		149.9 (26.4)	143.8 (25.90)		157.3 (21.30)	140.8 (18.60)#
SGO_{back}, µg/gland/min		10. 5 (3.20)	10.4 (3.00)		12.6 (2.40)*	12.6 (4.10)*
$SGO_{forearm}$, µg/gland/min		5.8 (2.20)	5.6 (2.170)		7.4 (1.30)*	7.1 (1.60)*
$SkBF_{back}$, au	0.3 (0.11)	1.1 (0.40)	1.1 (0.30)	0.3 (0.10)	1.4 (0.60)	1.2 (0.50)#
$SkBF_{forearm}$, au	0.1 (0.10)	0.8 (0.40)	0.8 (0.40)	0.1 (0.04)	0.9 (0.20)	0.8 (0.30)#

Despite BL value, all markers for the lower velocity (0.2 m/s) were taken between 35 and 40 min. All markers for the higher airflow (1.1 m/s) were taken between 55 and 60 min, except LSR, ASG, and SGO were taken between 50 and 55 min. #Significantly different from lower airflow (0.2 m/s) ($P < 0.05$); *Significantly different from winter season. ASG, activated sweat gland; BL, baseline; LSR, local sweat rate; SGO, sweat gland output; SkBF, skin blood flow; T_{core}, core temperature; T_{sk}, skin temperature; T_B, mean body temperature; WBSR, whole body sweat rate.

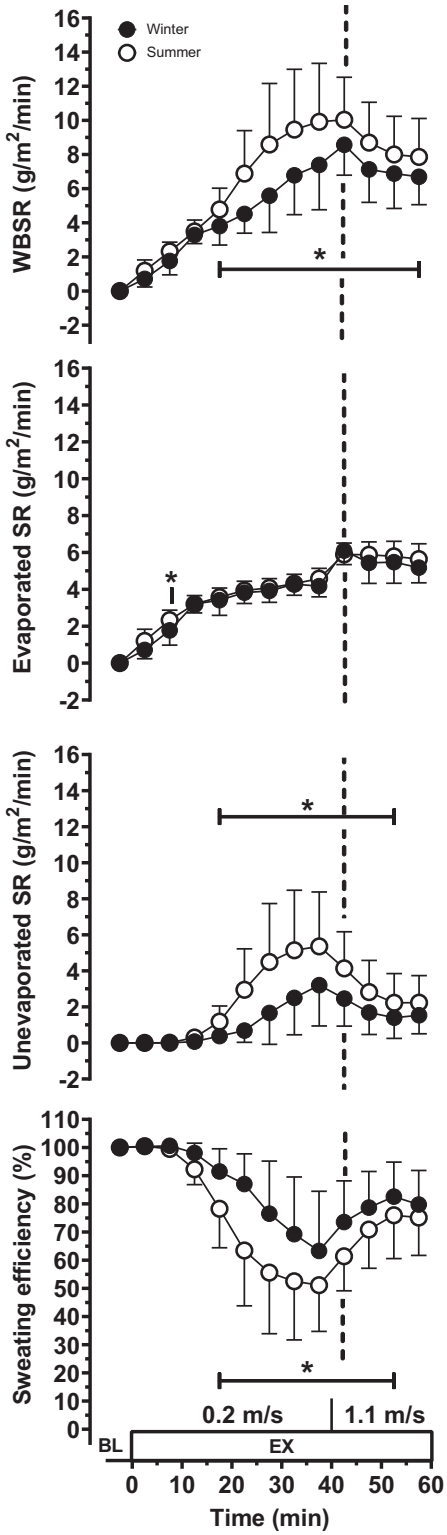

Figure 4. Means (SD) of whole body sweat rate (WBSR, $n = 12$), evaporated ($n = 12$) and unevaporated sweat rate ($n = 12$), and sweating efficiency (SE, $n = 12$) during exercise with the air velocity of 0.2 m/s and 1.1 m/s in both winter and summer seasons. *Significantly different from winter season ($P < 0.05$). SR, sweat rate.

summer season than winter season at all three time points [summer vs. winter: 20 min: 51.1 (22.4) vs. 74.5 (14.4) mmol·L^{-1}; 40 min: 56.6 (21.5) vs. 87.5 (19.4) mmol·L^{-1}; 60 min: 56.6 (23.5) vs. 87.5 (19.5) mmol·L^{-1}]. Sweat ion concentration was not different between different air velocities (0.2 m/s vs. 1.1 m/s) [71.5 (5.4) vs. 71.8 (5.6), $P = 0.83$].

Skin blood flow and heart rate response.

SkBF at baseline across all skin sites ($SkBF_{Forearm}$, $SkBF_{Back}$) did not differ between seasons (all $P > 0.20$, Fig. 6). During exercise, SkBF at all skin sites increased with time (all $P < 0.01$) but did not differ between seasons (all $P > 0.10$). However, an interactional effect was observed between seasons and time points (all $P < 0.05$). Specifically, $SkBF_{Back}$ was higher during summer compared with winter, from 10 min of

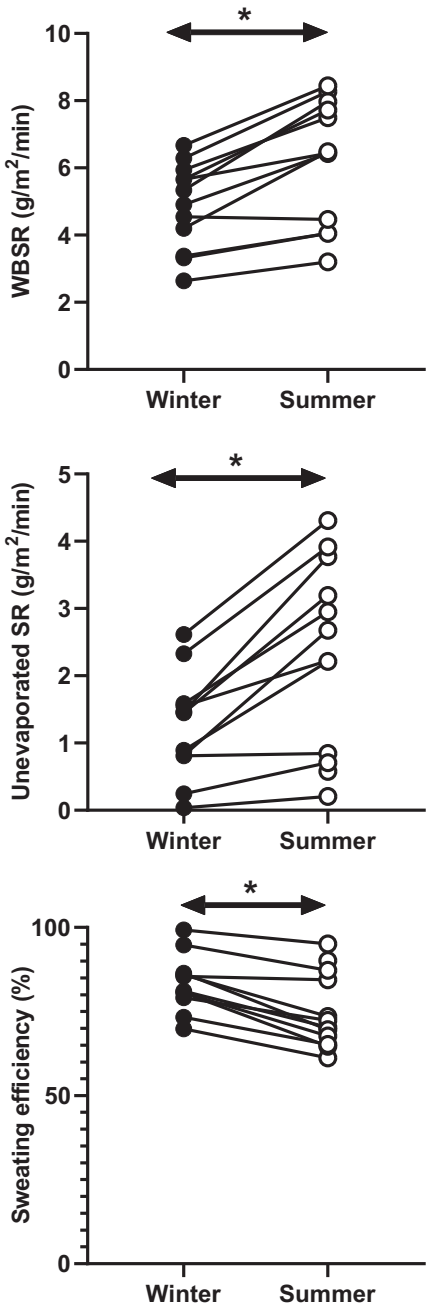

Figure 5. Individual value of whole body sweat rate (WBSR; $n = 12$), evaporated sweat rate ($n = 12$) and unevaporated sweat rate ($n = 12$) of the entire trial before and after seasonal acclimatization to humid heat. *Significantly different from winter season ($P < 0.05$). SR, sweat rate.

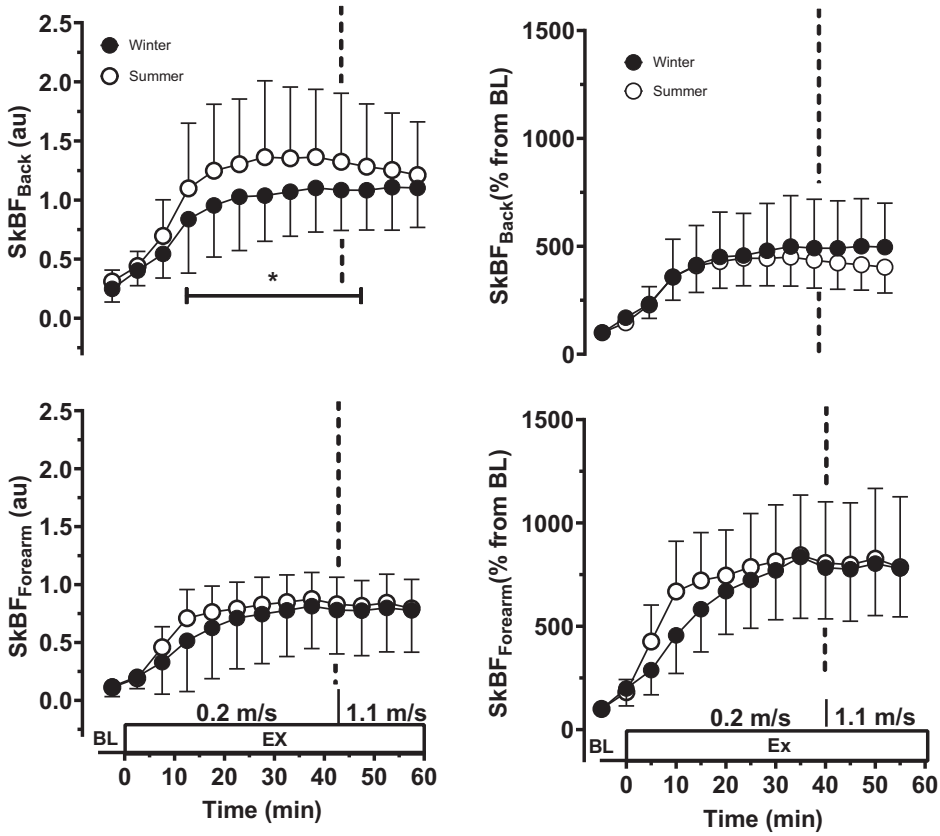

Figure 6. Means (SD) of unnormalized and normalized SkBF ($SkBF_{Back}$ and $SkBF_{Forearm}$, $n = 12$) relative to baseline value during exercise with the air velocity of 0.2 m/s and 1.1 m/s in both winter and summer seasons. *Significantly different from winter season ($P < 0.05$). SkBF, skin blood flow.

the exercise to 50 min. $SkBF_{Forearm}$ and $SkBF_{Back}$ were lower at the higher airflow of 1.1 m/s in summer only (season by air velocity interaction: $P < 0.01$, Table 3). When normalizing $SkBF_{Forearm}$ and $SkBF_{Back}$ from baseline, $SkBF_{Forearm}$ and $SkBF_{Back}$ increased with time (all $P < 0.01$) but were not different between summer and winter seasons (all $P > 0.40$) and no interactions were observed (all $P > 0.06$). HR at rest was ~5 beats/min lower in summer than in winter season [79.5 (8) vs. 74 (8) beats/min, $P < 0.01$]. During exercise, HR increased with time ($P < 0.01$) and showed an interactional effect between seasons and time point ($P < 0.01$). In particular, HR was lower in summer than in winter at the 55 min. Increasing in air velocity was only effective to reduce HR in winter season (season by wind speed interaction: $P < 0.01$).

Plasma Volume Changes and Percent of Body Mass Loss

Percent of body mass loss was greater in summer compared with winter [1.1 (0.09)% vs. 0.9 (0.06)%, $P < 0.01$]. However, the plasma volume changes were not different between summer and winter seasons [−3.5 (6.3)% vs. −7.5 (5.3)%, $P = 0.09$].

Partitional Calorimetry

H_{Prod} during exercise in a hot-humid environment was not different between summer and winter [211.2 (9.6) $W \cdot m^{-2}$ vs. 216.4 (9.7) $W \cdot m^{-2}$, $P = 0.24$].

$C_{skin} + R_{skin}$ during exercise increased with air velocity [23.0 (0.54) $W \cdot m^{-2}$ vs. 28.92 (0.84) $W \cdot m^{-2}$, $P < 0.01$] and was different between summer ($P < 0.01$) and winter with an interactional effect ($P < 0.01$). In particular, $C_{skin} + R_{skin}$ in summer was higher than in winter at the air velocity of 0.2 m/s [21.7 (2.6) $W \cdot m^{-2}$ vs. 24.4 (1.3) $W \cdot m^{-2}$, $P < 0.01$] and also in 1.1 m/s [25.3(4.8) $W \cdot m^{-2}$ vs. 32.5 (2.4) $W \cdot m^{-2}$, $P < 0.01$].

E_{max} during exercise increased with air velocity [113.2 (1.3) $W \cdot m^{-2}$ vs. 286.6 (2.8) $W \cdot m^{-2}$, $P < 0.01$] and was different between summer ($P < 0.01$) and winter with an interactional effect ($P < 0.01$). In particular, E_{max} was higher in summer than in winter at the air velocity of 0.2 m/s [116.6 (3.6) $W \cdot m^{-2}$ vs. 109.8 (6.2) $W \cdot m^{-2}$, $P < 0.01$] and also in 1.1 m/s [301.9 (11.3) $W \cdot m^{-2}$ vs. 271.3 (18.4) $W \cdot m^{-2}$, $P < 0.01$].

DISCUSSION

We systematically examined the thermoregulatory adaptations of seasonal acclimatization from the glandular level to the whole body level. By conducting this integrative approach, we have identified two novel findings. First, we have identified that seasonal acclimatization is able to enhance peripheral thermoeffector responses, but is unable to increase whole body evaporated sweat rate and reduce T_{core} during exercise in a hot-humid environment. Second, we have observed that the increase of air velocity is able to increase evaporated sweat rate and thus leads to a higher sweating efficiency irrespective of acclimatization status. These findings agree with our study hypothesis. Collectively, these observations indicate that despite of the typical adaptive responses in sweating following seasonal acclimatization, we found that seasonal acclimatization from winter to summer does not mitigate thermoregulatory strain during

exercise in a hot-humid environment and having a low or high sweating efficiency has no real impact on regulating steady state T_{core} during exercise in humid heat.

We have found that seasonal acclimatization from winter to summer is able to enhance both LSR ($LSR_{Forearm}$, LSR_{Back}, Fig. 3) and WBSR irrespective of air velocity, but is unable to enhance evaporated sweat rate in a hot-humid environment. Furthermore, sweating efficiency was significantly lower after seasonal acclimatization, which is consistent with previous heat acclimation studies conducted in humid heat (10, 35). The plausible physiological mechanism for such sudomotor adaptations is the combination of greater SGO, an elevated T_{sk} and, to a lesser extent, the release of neurotransmitter following seasonal acclimatization (36). An elevated T_{sk} enhances the central drive of sweating (37) as well as causing a greater reactivity of the muscarinic receptor response to cholinergic stimulus (20, 36). These adaptations together significantly enhance LSR and hence WBSR. Most of our data support this proposed mechanism, as our pilocarpine-induced sweating test revealed that $LSR_{Forearm}$, forearm skin temperature, and $SGO_{Forearm}$ were all higher following seasonal acclimatization. Thus, demonstrating the presence of a greater SGO and its reactivity to cholinergic drug stimulus. This observation continues during exercise as we observed a significant increase of T_{sk}, forearm skin temperature, $LSR_{Forearm}$, LSR_{Back} (Fig. 3), WBSR (Figs. 4 and 5), $SGO_{Forearm}$, SGO_{Back}, and greater sensitivity of WBSR with increasing T_{core} (Table 2). However, skin temperature at the back was higher in winter than in summer season and so we believe that the effect of local skin temperature on the regulation of LSR is site specific and cannot override the dominant role of the central drive in controlling the sensitivity of LSR with changing T_B during prolonged exercise in a hot-humid environment.

Unlike previous studies (1, 4, 5, 7) showing that seasonal acclimatization in summer is effective to reduce T_{core} during passive exposure to dry heat or thermally neutral conditions where the environment permits partial-to-full evaporation of sweat, we observed no net change of T_{core} from baseline to the end of the exercise following seasonal acclimatization to humid heat (Fig. 2). This contradictory finding may be due to the effect of our environment being hot and humid. It is well known that sweating becomes relatively inefficient under hot-humid environment as sweat evaporation is greatly inhibited by the reduced vapor pressure gradient between the skin and the environment and so any increase of sweating would subsequently result in a greater unevaporated sweat rate (9, 10, 35). Therefore, it is necessary to simultaneously elevate T_{sk} to promote greater dry heat loss (35). Our data partially support this previous notion as we observed a higher T_{sk} (Fig. 2) following seasonal acclimatization to humid heat. However, this small increase of dry heat loss had no effect on overall heat loss response following seasonal acclimatization as the change of T_{core} remained similar between summer and winter seasons (38).

We also showed that increase in air velocity was able to reduce unevaporated sweat rate and increased both evaporated sweat rate and sweating efficiency at the latter half of the exercise acutely, as well as after seasonal acclimatization to humid heat. These findings agree with the established principle of biophysics, where increase in air velocity leads to greater evaporative sweat rate and thus greater sweating efficiency ensues. This observation is against the finding of Candas et al. (11), where increase in air velocity before acclimation led to lower sweating efficiency. Such discrepancy may be due to our sweating efficiency was lower than Candas et al. (11) [63.3 (23.2)% vs. 89.1 (9.0)%], due to greater WBSR from our exercise approach, and so the increase in air velocity was able to enhance evaporated sweat rate and hence sweating efficiency. Also, as our weight scales can allow for continuous recording of the dripping sweat rate with greater accuracy compared to Candas et al.(11) using pre- and postdripping sweat differences, greater accuracy can be expected since it can prevent the dripping sweat being evaporated into the environment and thus results in a greater accuracy of evaporated sweat rate and hence sweating efficiency. However, having a low or a high sweating efficiency because of acclimated status or the increase in air velocity has no real impact on T_{core} response during exercise in humid heat. This may be due to the fact that a thermoregulatory steady state had been reached and thus increasing air velocity in the last 20 min of the exercise had no additional benefit on reducing T_{core} response.

Finally, we also observed a significant reduction of cardiovascular strain with a lower sweat ion concentration following seasonal acclimatization. In particular, the percent change of our $SkBF_{Forearm}$ and $SkBF_{Back}$ (Fig. 6) were similar before and after seasonal acclimatization whereas HR was significantly lower at rest and during the last stage of the exercise. This reduction in cardiovascular strain may be due to the greater increase of whole body blood volume due to greater electrolyte retention, which leads to similar change in plasma volume despite greater WBSR being observed (39). However, this increase in cutaneous vasodilation along with a lower sweat ion concentration was unable to further increase evaporated sweat and hence T_{core} (38).

Consideration of This Study

Although we have successfully demonstrated the sudomotor adaptations from the glandular level to the whole body level following seasonal acclimatization, however there are numerous limitations which warrant further considerations. First, we did not increase the SkBF to its maximal value and therefore could not fully explain the variation of skin blood flow response across different skin sites ($SkBF_{Back}$ and $SkBF_{Forearm}$) as well as the microvascular adaptation following seasonal acclimatization. Second, we acknowledge that we did not record the total outdoor exposure time of our participants in both seasons and therefore there may be different level of adaptations across different participants. However, this would not affect our primary markers such as LSR and WBSR as our thermoeffector responses demonstrated a typical pattern of heat acclimatization. We also did not randomize the order of different seasons and this, therefore, could result in potential bias. However, as the main purpose of this study was to observe the physiological/thermoregulatory adaptations from winter to summer but not from summer to winter, which would indicate decay. Indeed, this would have been interesting to observe as the decay effect of seasonal acclimatization remains unknown and thus warrants further investigation. Last, our participants demonstrated a 10% reduction of their $\dot{V}O_{2max}$

following seasonal acclimatization. This reduction of $\dot{V}O_{2max}$ may be explained by the fact that we tend to reduce the training intensity during summertime to prevent physical fatigue in the heat. However, this reduction of $\dot{V}O_{2max}$ would not affect the thermoeffector response as all of our participants exercised at the same metabolic heat production [273.9 (41.6) $W \cdot m^{-2}$ vs. 281.8 (42.3) $W \cdot m^{-2}$] in both winter and summer seasons.

Importance and Implication of This Work

This study provides further mechanistic insight on the sudomotor adaptations following seasonal acclimatization to humid heat from the glandular level to the whole body level. Furthermore, this study also demonstrates that having a low sweating efficiency or high sweating efficiency, due to acclimated status or the increase of air velocity in the last 20 min (attainment of a thermoregulatory steady state) has no real effect on regulating T_{core} during exercise in humid heat.

Conclusions

Seasonal acclimatization from winter to summer is able to enhance local thermoeffector responses and WBSR, but is unable to enhance evaporated sweat rate and to reduce T_{core} during exercise in a hot-humid environment. Furthermore, the increase of air velocity leads to greater evaporated sweat rate and sweating efficiency with a simultaneously reduction of unevaporated sweat rate. However, the increased air velocity during the last 20 min of exercise does not affect T_{core}.

GRANTS

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DISCLAIMER

T.-H. Lei is the postdoctoral research fellow from the Japan Society for the Promotion of Science.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

T.-H.L., M.F., T.M., Y.I., D.O., T.N., and N.K. conceived and designed research; T.-H.L., M.F., D.O., and N.K. performed experiments; T.-H.L., M.F., T.A., T.M., and N.K. analyzed data; T.-H.L., M.F., N.G., T.A., T.M., Y.I., and N.K. interpreted results of experiments; T.-H.L., M.F., T.A., T.M., Y.I., and N.K. prepared figures; T.-H.L., M.F., N.G., T.A., T.M., Y.I., and N.K. drafted manuscript; T.-H.L., M.F., N.G., T.A., T.M., Y.I., T.N., and N.K. edited and revised manuscript; T.-H.L., M.F., N.G., T.A., T.M., Y.I., D.O., T.N., and N.K. approved final version of manuscript.

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