Prosthetics and Orthotics International

Thermoregulatory responses in persons with lower-limb amputation during upper-limb endurance exercise in a hot and humid environment

Kouki Fukuhara[1,2], Yukio Mikami[3], Hiroshi Hasegawa[4], Daigo Nakashima[2], Yasunari Ikuta[2], Fumihiro Tajima[3], Hiroaki Kimura[5] and Nobuo Adachi[2,6]

Abstract

Background: Persons with an amputation may have an increased heat strain due to reduced surface area. However, there is limited evidence on the thermoregulatory responses in persons with lower-limb amputation (LLA). Although a previous study reported no difference in their rectal temperatures (Tres) in a hot environment, suggesting compensatory sweating of the intact limb, we examined the thermoregulatory responses of such persons in a hot and humid environment.

Objective: To compare the thermoregulatory responses–through changes in Tre, sweat, and oxygen uptake ($\dot{V}O_2$)–between persons with LLA and able-bodied (AB) individuals, in hot and humid environments.

Study design: A nonrandomized control trial.

Methods: Nine AB men (AB group) and nine persons with LLA group performed the arm ergometer exercise at 60% peak power output intensity for 60 min in a hot and humid environment, and they were tested before and after performing. The $\dot{V}O_2$, Tre and skin temperature, and total body sweating, and local sweating during exercise were measured and compared between the groups.

Results: The changes in $\dot{V}O_2$ and Tre after the endurance exercise did not differ between the groups (ΔTre: AB group, 1.1°C ± 0.5°C; LLA group, 1.2°C ±0.3 °C; $P = 0.65$), whereas the amount of local sweating of the chest (group effect, $P < 0.01$ by two-way analysis of variance [group × time], the group effect size was medium, $\eta^2 = 0.10$) and dehydration rate (AB group, 1.5% ± 0.5%; LLA group, 2.1% ± 0.5%; $P = 0.03$) were higher in the LLA than in the AB group.

Conclusions: We compared the thermoregulatory responses of persons with LLA with those of AB individuals in hot and humid environments. Core body temperatures of persons with LLAs during endurance exercise were not different from those of AB men even in hot and humid environments. We found compensatory increases in the sweat rate of the chest and increased dehydration rate in persons with LLA. More sweat potentially means that athletes with LLA need to drink more fulids.

Keywords

persons with lower-limb amputation, thermoregulatory responses, heat-related illness, hot and humid environment, upper-limb endurance exercise

Date received: 19 March 2021; accepted 27 July 2021.

Background

The body temperature of humans changes in accordance with the balance between heat production and heat dissipation but is maintained at approximately 37°C by regulatory functions.[1] When persons engage in exercise or physical activity, they generate more heat but their temperature is regulated by the heat loss reactions of increased cutaneous perfusion and sweating.[2] However, when heat production exceeds these heat loss responses, the body temperature begins to rise.[3] The factors that affect this rise in body temperature include exercise intensity[4] and duration and ambient temperature.[2] Higher exercise intensity increases oxygen uptake ($\dot{V}O_2$), which provides an indirect measure of heat production.[1] When investigating exercise-induced changes in body temperature under two conditions, it is recommended that exercise intensity ($\dot{V}O_2$) be kept constant.[5,6]

Encouraging persons with disabilities to participate in sports and to engage in physical activity brings about improvements in both physical function and psychological state, thereby maintaining and improving their state of health and increasing their quality of life.[7] By contrast, the risks associated with sports for persons with disabilities include sports-related injury[8,9] and heatstroke.[10] Guidelines for exercise in hot environments have been formulated to prevent heatstroke in the able-bodied (AB),[11] although as yet there is no consensus on measures to deal with heat and thermoregulation for persons with disabilities.[12] However, a number of studies have addressed thermoregulation in persons with spinal cord damage. Price[13] found that persons with injuries at a high spinal level produced less overall sweat, and their body and skin temperatures (Tsk) in the paralyzed area were both

[1]Department of Rehabilitation Medicine, Graduate School of Biomedical and Health Sciences, Hiroshima University, Japan

[2]Sports Medical Center, Hiroshima University Hospital, Japan

[3]Department of Rehabilitation Medicine, Wakayama Medical University, Japan

[4]Graduate School of Humanities and Social Sciences, Hiroshima University, Japan

[5]Department of Rehabilitation Medicine, Hiroshima University Hospital, Japan

[6]Department of Orthopaedic Surgery, Graduate School of Biomedical and Health Sciences, Hiroshima University, Japan

Corresponding author:

Kouki Fukuhara, Kasumi 1-2-3, Minamiku, Hiroshima, 7348551, Japan. Email: kouki@hiroshima-u.ac.jp

Associate Editor: *Christopher Wong*

DOI: 10.1097/PXR.0000000000000030

higher.[14] Griggs et al[15] reported that patients with injuries at a high spinal level required longer time for their body temperature to recover after exercise mainly due to reduced heat dissipation rather than to heat production. These studies suggested that the capacity to sweat decreased in the paralyzed region of these tetraplegic individuals; thus, their skin and body temperatures also tend to increase. It is believed that the compensatory heat dissipation mechanism does not function in individuals with spinal cord injuries because the information cannot be transmitted in both directions between the peripheral effector organs and central nervous system due to impaired neurotransmission.[16]

Persons with lower-limb amputation (LLA) have a lower body surface area (BSA) for heat dissipation, and their body temperatures may therefore tend to increase.[12,17–19] Although there have been some studies on the changes in the body temperature of persons with LLA during exercise, such as body temperature changes during a marathon[20] and during upper-limb endurance exercises in a climate-controlled room,[21] almost no studies have addressed the thermoregulation mechanisms of persons with LLA.[12] Hasegawa et al[21] reported that the core temperature of persons with LLA did not rise above that of AB people during exercise, suggesting that a mechanism to compensate for the leg(s) was in operation. The study discussed the possibility of compensatory sweating not only of the lower limbs but also of the upper body (with high volume of sweating sites); however, the details are unclear. Besides, the study focused solely on heat dissipation and did not sufficiently discuss heat production. The evidence on thermoregulatory responses in persons with LLA thus remains scant, and further research on persons with LLA is needed to improve our understanding of their thermoregulatory responses.

Previous body temperature studies on persons with LLA have been conducted in hot conditions,[21] but not in hot and humid conditions. Tropical climatic zones and summers in temperate climatic zones are frequently hot and humid, and as international parasports competitions may be held under these climatic conditions, measures to deal with heat and humidity are regarded as important.[22] It is, therefore, necessary to conduct experience-based studies during disabled sporting competitions to provide insights on the effects of hot and humid conditions. Comparative studies on persons with LLA and AB individuals of the same age under these conditions are thus required. In summary, there have been few studies on the changes in the body temperatures of persons with an LLA but consensus has yet to be reached. The details on studies undertaken during parasports competitions held in hot and humid conditions are unclear, as are the compensatory mechanisms involved.

Our objective was to measure the core temperature, local sweating, and $\dot{V}O_2$ of persons with LLA in a hot and humid environment, and to compare these with the thermoregulatory responses of AB individuals. In light of the study by Hasegawa et al,[21] we hypothesized that given the existence of a compensatory mechanism whereby sweating in areas other than the legs increases, there would be no increase in core temperature even in hot and humid conditions. This study aimed to establish evidence on thermoregulation in persons with LLA. Knowledge of the characteristics of thermoregulation in persons with LLA under hot and humid conditions will be useful in the formulation of proposals for measures to reduce heatstroke during both international parasports competitions and daily life. Making progress in the risk management of heat-related issues may also raise awareness and participation of persons with LLA in sports and improve their competitiveness.

Methods

Study design

This was a nonrandomized control trial.

Study setting

The study was performed in an artificial climate chamber (FCC-5000S, Fuji Medical Science Co Ltd, Japan) installed in Hiroshima University Graduate School of Humanities and Social Sciences.

Research ethics and patient consent

This study was approved by Hiroshima University Hospital Comprehensive Medical Research Promotion Center (approval number, C-230), and the participants provided written informed consent. The clinical trial has been registered online (registry name, University hospital Medical Information Network; trial name, prevention of heatstroke for athletes with LLA; trial accession URL, https://upload.umin.ac.jp/cgi-open-bin/ctr_e/ctr_view.cgi?recptno=R000035898; trial registration number, UMIN000031441).

Participants

We recruited participants by attaching posters to the sports center for persons with disabilities and representatives from each sports organization. Nine AB men group (AB group) and nine persons with LLA (LLA group) participated as volunteers. The inclusion criteria were 6 months of sports or parasports history (in any amputee football, triathlon, or wheelchair basketball, tennis, or marathon) and at least 6 months of athletic history. The exclusion criteria were severe upper limb joint disease, mental illness, seizures, and heart disease.

Study protocol

All participants underwent a cardiopulmonary exercise test as a preliminary study. Based on the results of the preliminary study, 60% peak power output (PPO) intensity exercise was performed for 60 minutes using an arm ergometer (Monarch 881E, Monarch, Sweden). To control the metabolic heat production, arm ergometer exercise instead of prosthetic walking was selected as the form of exercise. This intensity was chosen to enable continuous exercising in a hot and humid environment for 1 hour. The environmental conditions were set at 33°C and 70% relative humidity (RH).[22] Participants were required to maintain a normal lifestyle, including normal physical activity and nutrition throughout the study. They were asked to avoid alcohol and caffeine intake for 24 hours before the experiment. All experiments were initiated at 10 AM to eliminate circadian rhythm effects.

Preliminary study

When participants arrived at Hiroshima University laboratory, they were asked an open-ended question about their age, period of

Table 1. Participant demographics.

	AB group (n = 9)	LLA group (n = 9)	*P* value
Age (y)	39.3 ± 7.5	41.3± 7.6	0.58
Height (cm)	169.4 ± 3.6	171.9 ± 6.6	0.19
Body weight (kg)	63.9 ± 9.5	63.1 ± 7.6	0.50
Subcutaneous fat thickness (mm)	47.3 ± 21.3	57.9 ± 10.2	0.14
BSA (m^2)	1.7 ± 0.1	1.5 ± 0.2	0.01
$\dot{V}O_2$ peak (mL/kg/min)	31.3 ± 8.9	34.1 ± 5.9	0.26
PPO (W)	66.7 ± 16.2	81.1 ± 13.4	0.06
Period of playing sports (y)	11.7 ± 7.9	8.7 ± 5.9	0.38
Exercise habits (min/wk)	136.7 ± 128.3	176.7 ± 96.6	0.46

AB group, able-bodied group; BSA, body surface area; LLA group, lower-limb amputation group; PPO, peak power output; $\dot{V}O_2$, oxygen uptake.
Data are expressed as means ± SDs.
Independent t-test, P < 0.05/9, a Bonferroni-adjustment for repeated measures, AB group vs LLA group.

playing sports, and exercise habits. Then, the height and bodyweight of the participants were mesured using a digital scale (UC-300, A & D Co Ltd, Japan). The following skinfolds were measured: triceps, subscapular, and abdominal. These three points were defined as the subcutaneous fat thickness. Skinfold caliper (Holtain, UK) was used and calibrated according to the manufacturers' instructions. Anthropometric measurements were performed according to International Society for the Advancement of Kinanthropometry standards.[23] Then, they performed a cardiopulmonary exercise test using an arm ergometer. The arm ergometer was positioned at the midsternal level using a ruler, with the participant's elbow flexed slightly with a maximum extension of the upper limb. The persons with LLA sat on a plastic chair without prostheses, and the intact leg was positioned on the floor. Meanwhile, the AB men sat with both legs on the floor. All participants wore face masks connected to a mobile aero monitor (Aero Monitor AE-310S, Minato, Japan), and $\dot{V}O_2$ was measured for each breath. Participants performed the incremental exercise test described by Ohtsuki.[24] After resting for 3 minutes, a warm-up was performed at 0 Watt (W) for 3 minutes, and then the exercise was performed from 25 W to exhaustion with a 5-W increase every minute. Participants were asked to perform at a cadence of 50 revolutions per minute (rpm). The cardiopulmonary exercise test was terminated when a cadence of 50 r/min could not be maintained, and the PPO and peak oxygen uptake ($\dot{V}O_2$ peak) of each participant were measured.

Main experiment

Tests were performed before and after upper-limb endurance exercise. The main experiment was conducted on a different day from the preliminary study. Participants entered the laboratory 90 minutes after drinking 500 mL of water. Upon entering the laboratory, a small sample of urine was collected, of which 1 mL was used for urine specific gravity (USG) measurements, and then the naked weight was measured. When an individual's body temperature starts to rise during exercise, they begin to sweat, although as the sweating response varies depending on their preexercise hydration, USG must be measured as an indicator of hydration.[25] Before initiating this experiment, we ensured that all participants had a preexercise USG value of <1.030 to confirm that they were sufficiently hydrated. The rectal temperature (Tre) was defined as the core body temperature.[26] To measure the Tre, a rectal thermistor (LT-ST08-21; Nikkiso Therm Co Ltd, Japan) covered with a disposable rubber sheet (11Y24; Nikkiso Therm Co Ltd, Japan) was inserted over 12 cm below the anal sphincter. Then, a skin thermistor was attached halfway between the sternum notch and xiphoid process in the chest and midpoint between the inguinal fold and superior border of the patella in the intact thigh. A heart rate monitor was wrapped as a belt over the sternum notch. Participants then wore a polyester T-shirt and short pants and entered the artificial climate chamber (33°C, 70% RH) without prostheses and were kept at rest for 20 minutes, and the baseline data were measured. The exercise intensity was then set to 60% PPO, and the participants exercised on an arm ergometer for 60 minutes without rest. Subsequently, they sat for a 20-minute recovery period. The probe was then removed, and naked weight and USG were remeasured to estimate the sweat loss. Participants did not drink any fluids during exercise to eliminate the effects of fluid intake on the thermoregulatory responses. When a participant could not maintain a cadence of 50 r/min or when the Tre reached 39.5°C, the situation was judged as exhaustion. This was because the torque increased if the participant could not maintain 50 r/min, and subjects incurred exhaustion at core temperatures of 39.5°C.[27] The exercise was thereby stopped. During the experiment, the participants were accustomed to the procedure, and a habituation test (familiarization) was performed to minimize the effects of potential learning or anxiety.

Outcome measures

The BSA of an AB person was calculated using the Du Bois equation[28]: AB group BSA [m^2] = (body height [cm]$^{0.725}$) × (bodyweight [kg]$^{0.425}$) × (0.007184). The BSA of a person with LLA was calculated after considering the percentage of amputated limbs.[20] LLA group BSA [m^2] = (body height [cm]$^{0.725}$) × (estimated bodyweight [kg]$^{0.425}$) × (0.007184) × (1 – (% BSA), where the estimated bodyweight [kg] = (current weight [kg])/(1 – p), in which p is the ratio of the amputated limb to the total body weight; % *P* was 12.8% for transfemoral amputation (TFA) and 4.4% for transtibial amputation (TTA),[29] and the estimated BSA

Table 2. Disability and parasports characteristics of persons with lower-limb amputation.

	Level of amputation	Cause of amputation	Period after amputation (y)	Parasports
1	TFA	Osteosarcoma	11	Amputee football
2	TFA	Traffic accident	12	Amputee football
3	TFA	Traffic accident	5	Amputee football
4	TFA	Traffic accident	12	Amputee football
5	TTA	Traffic accident	14	Track and field
6	TTA	Osteosarcoma	50	Jogging
7	TTA	Traffic accident	38	Amputee football and tennis
8	TTA	Buerger disease	3	Amputee football
9	TTA	Traffic accident	17	Baseball and wheelchair basketball

TFA, transfemoral amputation; TTA, transtibial amputation.

ratio (% BSA) presented by Colangelo et al[30] was 21.04% for TFA and 9.15% for TTA.

We measured $\dot{V}O_2$, Tre, Tsk, heart rate (HR), local sweating rate (LSR), naked body weight, and USG as the physiological outcome measures. Tsk was measured at two sites (chest and intact thigh), and the thermistor was fixed with a micropore tape. All thermistors were connected to a data acquisition device (LT-8A; Gram Corporation, Japan), and Tre, Tsk, and HR were recorded every 5 minutes. Before and after the experiment, the naked weight was measured using a digital scale (UC-300; A & D Co Ltd, Japan), and the USG was measured using a digital USG scale (UG-D; Atago Co Ltd, Japan). Whole-body sweating and dehydration rates were calculated using the following equations: total body sweating (kg) = (naked body weight before experiment [kg] − naked body weight after experiment [kg] and dehydration rate (%) = (total body sweating [kg]/naked weight before the experiment [kg]) × 100. LSR was measured by the ventilation method using a probe with a detection area of 1 cm^2 and a local perspiration meter (POS-02; Skinos Technical Co Ltd, Japan). LSR was measured continuously at two sites (chest and intact thigh), and the capsules were fixed with double-sided tape.

Statistical analysis

All data were expressed as means ± SDs. The normal distribution of the data was determined using the Shapiro–Wilk test. Age, height, weight, corrected BSA, $\dot{V}O_2$ peak, PPO, sports experience, exercise habits, total body sweating, dehydration rate, and postexercise USG were compared using an independent test between the AB and LLA groups. The subcutaneous fat thickness and preexercise USG were compared using the Mann–Whitney U test between the AB and LLA groups, with a Bonferroni adjustment for repeated measures. Differences in Tre, Tsk, and LSR were analyzed using a two-way (group × time) repeated-measures analysis of variance. If a significant difference was found, a post hoc test was performed, with a Bonferroni adjustment for repeated measures. Besides, when a significant difference was found, it was calculated based on the effect size. Data analysis was performed using JMP Pro (version 14.2.0; SAS Institute Inc, Japan), and $P < 0.05$ was regarded as significant for all outcome measures. The guidelines for the effect size were as follows[31]: small when η^2 = 0.01–0.06, medium when η^2 = 0.06–0.14, and large when $\eta^2 \geq 0.14$.

Results

Table 1 shows the participant demographics. The BSA of the LLA group was smaller than that of the AB group. Table 2 shows the disability and parasports characteristics of persons with LLA. There were four persons with TFA and five with TTA. The causes for amputation were traffic accidents, tumors, or Buerger disease. The average period after amputation was approximately 18 years. The parasports played by persons with LLA were mainly amputee football and track and field, jogging, tennis, baseball, and wheelchair basketball.

All participants had preexercise USG values of <1.030, and they were not dehydrated (Table 3). Table 4 shows the results of the two-way analysis of variance of $\dot{V}O_2$, Tre, Tsk, and LSR during upper-limb endurance exercises. The change in $\dot{V}O_2$ during upper-limb endurance exercise did not differ between both groups (Table 4, Figure 1), and the metabolic heat production was equivalent in both groups. The rectal core temperature also increased similarly in both groups, and there were no differences between the groups (Figure 2). The chest and intact thigh skin temperatures increased in both groups during exercise, although the chest skin temperature was higher in the LLA than in the AB group (main effect of group factors, $P < 0.01$; Figure 3(a)); the effect size was small ($\eta^2 = 0.01$). By contrast, the intact thigh skin temperature was not different between both groups (Figure 3(b)).

Table 3. Urine specific gravity mesuarments.

Urine specific gravity	AB group (n = 9)	LLA group (n= 9)	*P* value
Pre-exercise	1.009 ± 0.010	1.012 ± 0.007	0.37
Post-exercise	1.012 ± 0.007	1.015 ± 0.008	0.43

AB group, able-bodied group; LLA group, lower-limb amputation group.
Data are expressed as means ± SDs.Independent t-test, P < 0.05/2, with a Bonferroni-adjustment for repeated measures, AB group vs LLA group.

Table 4. Results of two-way analysis of variance.

	Group effect				Time effect				Group × time			
	Freedom	F-ratio	*P* value	η^2	Freedom	F-ratio	*P* value	η^2	Freedom	F-ratio	*P* value	η^2
$\dot{V}O_2$	1	1.1	0.29	N/A	19	32.1	<0.01	0.73	19	0.1	1.00	N/A
Tre	1	2.0	0.16	N/A	20	29.0	<0.01	0.63	20	0.04	1.00	N/A
Tchest	1	12.8	<0.01	0.01	20	31.7	<0.01	0.64	20	1.7	0.04	0.03
Tintact thigh	1	1.9	0.17	N/A	20	30.0	<0.01	0.65	20	0.5	0.96	N/A
LSRchest	1	84.6	<0.01	0.10	20	21.8	<0.01	0.52	20	0.8	0.72	N/A
LSRintact thigh	1	54.3	<0.01	0.08	20	15.5	<0.01	0.44	20	0.1	1.00	N/A

LSRchest, chest local sweating rate; LSRintact thigh, intact thigh local sweating rate; N/A, not applicable; Tre, rectal temperature, Tchest, chest skin temperature, Tintact thigh, intact thigh skin temperature; $\dot{V}O_2$, oxygen uptake.

The amounts of local sweating increased in both groups during exercise, although the amounts of the chest and thigh were higher in the LLA than in the AB group (main effect group factors, $P < 0.01$ and $P < 0.01$, respectively; Figure 4) and the effect sizes were medium ($\eta^2 = 0.10$ and $\eta^2 = 0.08$, respectively). The amount of total body sweating (0.96 ± 0.28 kg in the AB group, 1.40 ± 0.36 kg in the LLA group, $P = 0.01$) and dehydration rate (1.51% ± 0.48% in the AB group, 2.07% ± 0.47% in the LLA group, $P = 0.03$) were both higher in the LLA than in the AB group.

Discussion

We aimed to compare the core body temperature, and heat production and dissipation responses in persons with LLA with those of AB individuals during endurance upper-limb ergometer exercise with constant intensity in a hot and humid environment. A previous study had reported that compensatory sweating occurs,[21] and we hypothesized that compensatory sweating would occur on the chest and the core temperature of persons with LLA would not rise above that of AB individuals even under more realistic hot and humid conditions compared with those used in that previous study. Our results substantiated this hypothesis, showing that although the core temperature of persons with LLA rose during exercise, there was no difference between this rise of that in AB individuals even in hot and humid conditions. Besides, persons with LLA produced more sweat on their chests in compensatory sweating than the AB individuals. We also found that the total amount of sweat and rate of dehydration in persons with LLA were both higher than those of AB individuals, with a dehydration rate of approximately 2%.

Excessive rises in body temperature are prevented by two different heat dissipation responses. One comprises nonevaporative heat dissipation via increased cutaneous perfusion causing a rise in and heat loss due to the difference between the ambient and

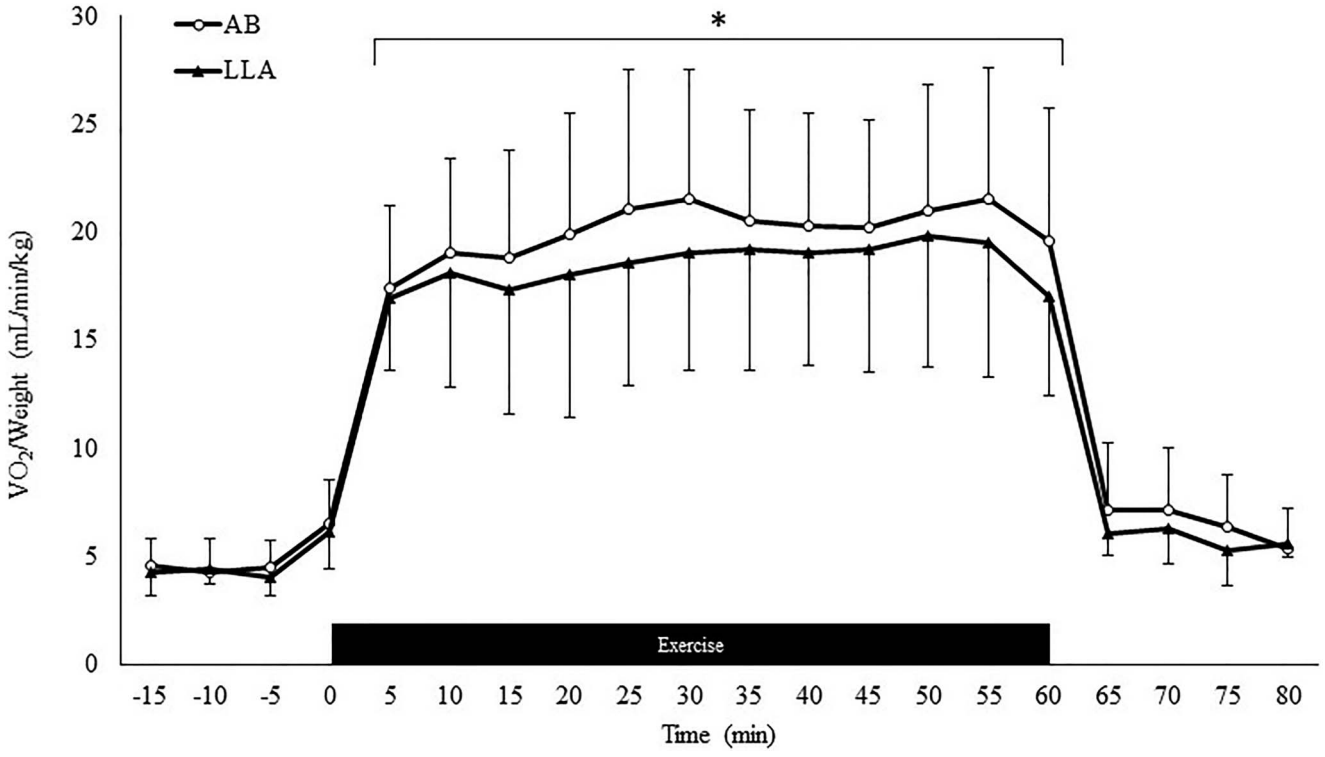

Figure 1. Change in oxygen uptake. AB group, able-bodied group; LLA group, lower-limb amputation group. *$P < 0.01$ vs 0 minutes.

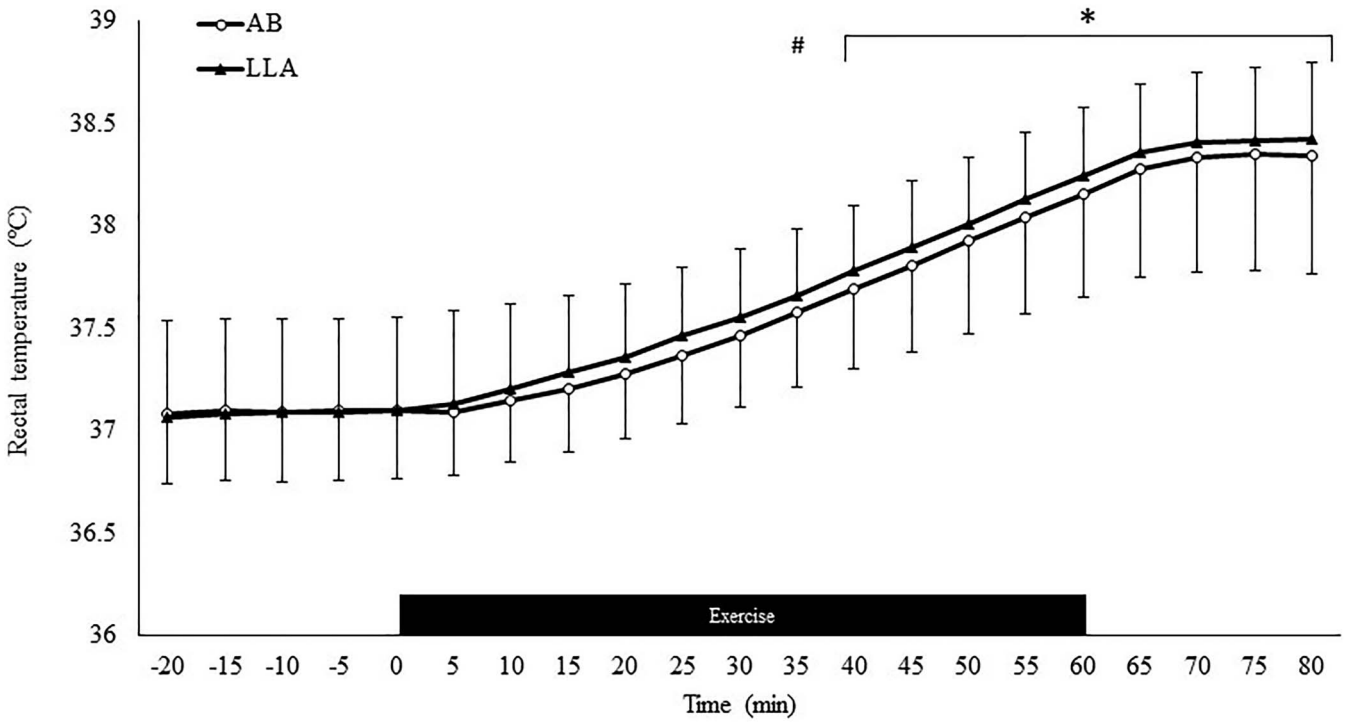

Figure 2. Change in rectal temperature. AB group, able-bodied group; LLA group, lower-limb amputation group. #$P = 0.02$ vs 0 minutes. *$P < 0.01$ vs 0 minutes.

body temperatures, and the other is evaporative heat loss due to the evaporation of sweat.[1] In very hot environments, the thermal gradient between the air and skin is smaller; therefore, evaporative heat loss becomes the main means of dissipating heat.[1] High humidity also makes it harder for sweat to evaporate; therefore, people sweat more.[1] The potential for heat loss depends on BSA,[1] and the loss of effective body surface results in increased sweating of the available areas.[32] A good example of this is compensatory sweating as a complication after treatment for hyperhidrosis.[32] Hyperhidrosis is a condition in which the amount of sweat from all or certain parts of the body increases. Sympathectomy that governs the region of excessive sweating reduces the amount of sweat in that region but increases the amount of sweat elsewhere. This is known as compensatory sweating and is explained by the long-term activation of the sweat glands.[32]

Another mechanism by which AB individuals accustom themselves and deal with the heat is heat adaptation. Heat adaptation consists of exercising under hot conditions, increasing the body temperature by approximately 1°C, and maintaining this heat stress for around seven consecutive days to bring about physiological adaptive reactions, such as a heightened sweating response by activating the sweat glands.[33] A heightened sweating response means that the body temperature rises more gradually than would otherwise be the case.[33] People with LLA are subject daily to heat stress in the form of elevated body temperatures and sweating due to everyday physical activities or participation in sports,[34] and it is thus possible that their sweat glands have been activated to deal with this heat stress.[18] They may thus have compensatory sweating via repeated heat stress stimuli and activation of sweat glands in their remaining skin. Compensatory sweating may also contribute to increased Tsk. Hirata et al[35] reported that placing a pressure cuff on one ankle to cut off the flow of blood lowered the Tsk and reduced the amount of sweating in that leg, whereas the Tsk and amount of sweat on the trunk and in other areas increased as a compensatory response to make up for the limited heat loss. This can be explained in terms of the function of numerous arteriovenous anastomoses in the sole of the foot.[35] Although the situation of persons with LLA differs from the use of a pressure cuff, they have also lost the function of the arteriovenous anastomoses in the sole of the foot by amputation, and this may increase Tsk and compensatory sweating. These compensatory responses may exist in people with LLA precisely because the transmission of information between the temperature control center and peripheral effector organs is not impaired. Previous studies of compensatory sweating have only looked at the legs,[21] but differences in sweating between different parts of the body are well known, and more sweating occurs on the chest than on the legs.[36] In this study, we found that compensatory sweating was evident on the chest, where more sweating occurred, and this may be one reason why the core temperature of lower-limb amputees did not increase to a greater extent than that of AB individuals.

The wet-bulb globe temperature has been proposed as a helpful index for preventing heatstroke,[37] and the heat index, calculated from temperature and RH, has been widely used to measure heat exposure.[38] The National Weather Service operates a website for calculating the heat index,[39] and uses it to categorize the risk of heatstroke into four levels as follows: 80–90, caution; 91–103, extreme caution; 104–124, danger; and ≥125, extreme danger.[40] The climatic conditions of 32°C and 50% RH used in a previous study corresponded to a heat index of 94, requiring extreme caution for the risk of heatstroke, and exertional changes in the body temperature of persons with LLA under these conditions

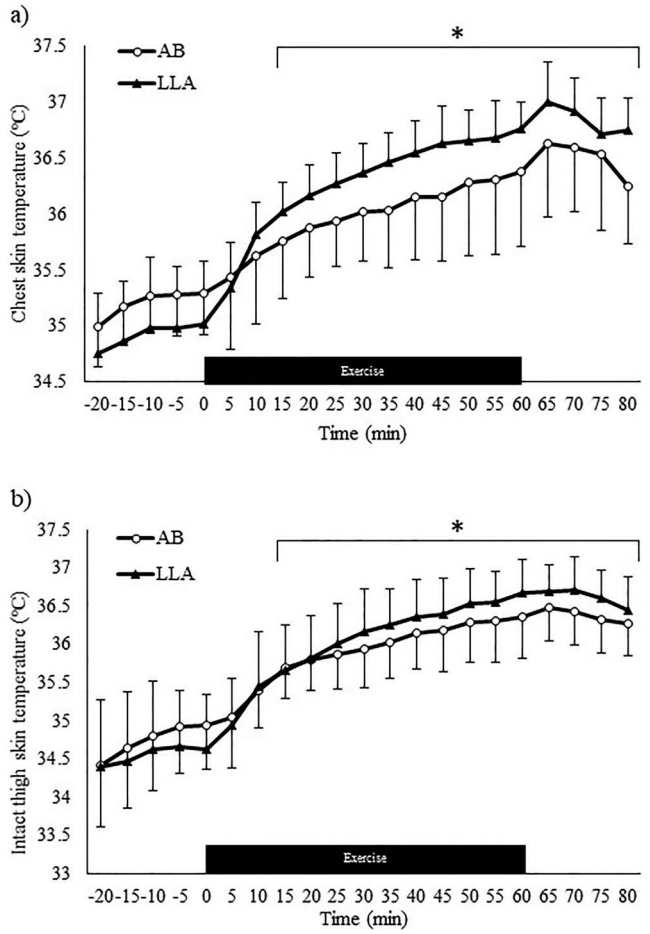

Figure 3. Change in skin temperature: (a) chest and (b) intact thigh. AB group, able-bodied group; LLA group, lower-limb amputation group. *$P <$ 0.01 vs 0 minutes.

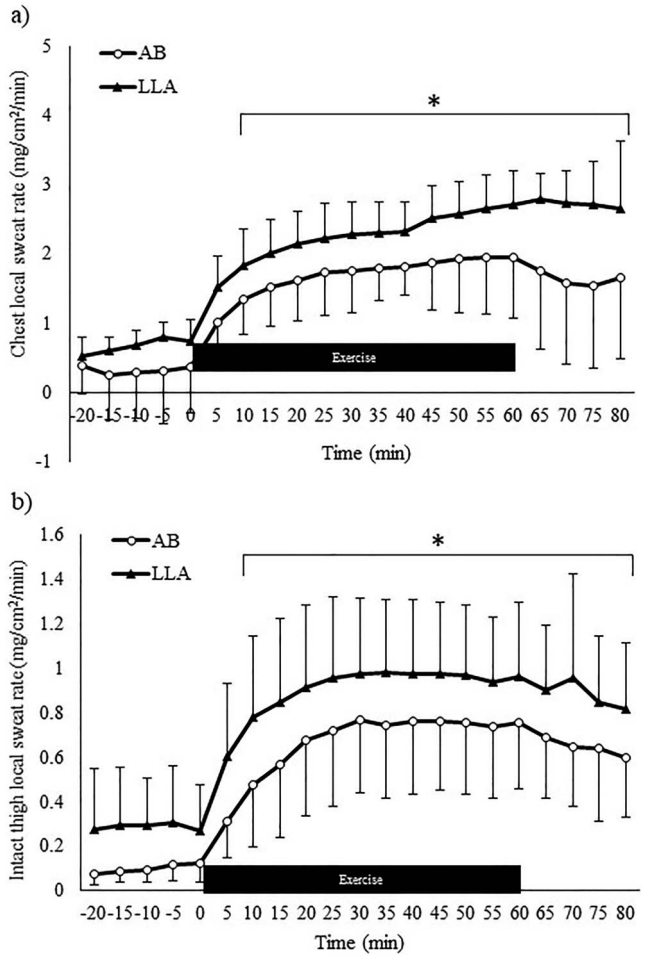

Figure 4. Change in the amount of local sweating: (a) chest and (b) intact thigh. AB group, able-bodied group; LLA group, lower-limb amputation group. *$P <$ 0.01 vs 0 minutes.

have already been reported.[21] The conditions of 33°C and 70% RH used in this study correspond to a heat index of 110, 16 points higher than that of the previous study; this is classified as danger, one risk level higher for heatstroke. However, the exercise intensity was the same as that used in the previous study.[21] Under these environmental conditions, heat production ($\dot{V}O_2$) due to exercise will also cause an increase in body temperature.[3] In our experiment, $\dot{V}O_2$ increased during exercise in both groups, with no significant difference between them; therefore, there was no difference in exercise intensity or heat production. Although both groups generated the same amount of heat, compensatory sweating of the chest as described above suggested that the core temperature of persons with LLA was not higher than that of AB individuals even under hot and humid conditions in the heatstroke risk danger zone. It is conventionally believed that the smaller BSA of persons with LLA makes their core temperature more likely to rise, although our experimental verification of changes in the body temperature of persons with LLA at the laboratory level showed that there was no difference between their core temperatures and those of AB individuals, irrespective of the ambient temperature.

Body temperature studies in the field of persons with disabilities have long focused on patients with spinal cord injuries, and in the 1980s, it was concluded that such patients are unable to maintain a constant core temperature because of vasomotor disorders and the lack of an effective sweating system due to neural paralysis.[41] The thermoregulatory mechanisms in patients with spinal cord injury were subsequently investigated in detail, and it was found that those with complete paralysis due to a high-level spinal cord injury (tetraplegia) were particularly susceptible to core temperature elevation during exercise.[14] This is because of their diminished ability to dissipate heat despite generating equivalent amounts of heat, causing their body temperatures to rise.[15] Recent studies have further led to the discovery that patients with spinal cord injury exhibit reduced sweating in the paralyzed parts of their body. Besides, there is no feedback of sensory information, such as increased Tsk to the temperature control center, meaning that there are no functioning compensatory mechanisms for heat dissipation as a result of their elevated body temperatures.[16] However, no studies have been conducted on the thermoregulatory mechanisms in persons with other disabilities, such as persons with LLA; hence, these remain unclear.[12] In light of the diminished heat dissipation capacity of patients with spinal cord injury, the decreased BSA of persons with LLA was expected to decrease their capacities for heat dissipation, and correspondingly, their body temperatures were expected to rise.[12] However, rather than their capacities for heat dissipation being diminished by the loss of BSA, persons with LLA exhibited an adaptive response that enabled them to withstand

increased body temperatures by compensatory sweating. We should understand these differences between the exertional body temperature changes and physiological responses of patients with spinal cord injury and persons with LLA as disability-specific thermoregulation characteristics.

A number of cooling strategies to improve the exercise performance of patients with spinal cord injury and prevention strategies for heatstroke have been reported. Because patients with spinal cord injury sweat less, mist sprays of artificial sweat are considered to be effective in reducing the body temperature.[42] Persons with LLA, on the other hand, sweat more, both locally and in total, and as their dehydration rate is also high, they are at higher risk of progressing to dehydration than are AB people. In this study, conducted under hot and humid conditions, the dehydration rate in the LLA group was approximately 2%. This may reduce the performance in endurance exercises and affect competitiveness.[43] In addition, if dehydration progresses, it increases the risk of heatstroke.[44] Although compensatory sweating is highly effective in cooling the body, the increased risk of heatstroke due to the progression of dehydration must be paid attention. This suggests that in hot and humid conditions, persons with LLA may need to drink more fluids to offset their increased sweating and to mitigate any dehydration-related drop in exercise performance and the risk of heatstrokes. A previous study reported that persons with LLA were less sensitive to warmth than were AB individuals,[21] and this may make them less likely to notice that they were becoming dehydrated and take necessary action to drink more fluids, thereby increasing their risk of heatstrokes. Based on these findings, researchers should educate persons with LLA, their coaches, and competition staff on this issue. Knowledge of disability-related compensatory sweating will be useful in the formulation of proposals for measures to reduce heatstroke and will help with the risk management of heat-related issues and in improving competitiveness.

This study has some limitations. First, these experiments were performed with moderate and constant load exercises for 60 minutes. Under these conditions, there was no difference in core body temperature between the groups. However, the thermoregulatory responses may change after prolonged periods (>60 minutes) or intermittent high-intensity exercises. Further studies are needed to elucidate thermoregulatory responses in parasports. Second, the residual limb could not be supported on a floor because some devices were attached to the residual limb in the LLA group, which might have influenced the results. Third, the number of samples was too small to confirm the differences in the amputation site. Recruitment of healthy persons with LLA was difficult, and we were only able to recruit nine participants (four TFA and five TTA). Shorter remaining limbs might increase compensated sweating; this should be verified by further studies. Whether persons with LLAs acquire heat tolerance as sensitivity in sweat glands and heat acclimation should be verified in future research.

Conclusions

We asked persons with LLA to perform upper-limb endurance exercises under hot and humid conditions and compared the changes in their core body temperature and amount of sweating with those of AB individuals. We discovered that as a result of compensatory sweating on the chest and intact thigh, there was no difference between the amounts by which the core temperatures increased in persons with LLA and AB individuals. Heat dissipation via compensatory sweating is effective in cooling the body but increases the risk of dehydration. This implies that for athletes with LLA, sweating more may require them to drink more fluids. It is also important to discuss the fact that the thermoregulatory responses in persons with LLA differ from those with spinal cord injuries, and to understand the differences in thermoregulation between different disabilities. Understanding the thermoregulatory mechanisms of persons with LLA may lead to the formulation of logical heatstroke prevention measures, enable safer participation in parasports, and improve competitiveness.

Declaration of conflicting interest

The authors disclosed no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Data accessibility statement

This research will be published in the institutional repository (URL, https://ir.lib.hiroshima-u.ac.jp/en).

Acknowledgments

The authors would like to thank Editage (URL, www.editage.com) for English language editing. The first author received support for the Ministry of Education, Culture, Sports, Science and Technology, Japan Society for the Promotion of Science, KAKENHI Grant-in-Aid for Scientific Research (C), Grant Number JP 17K01723.

ORCID iDs

K. Fukuhara: https://orcid.org/0000-0003-1681-0035
Y. Mikami: https://orcid.org/0000-0001-6855-8439
H. Hasegawa: https://orcid.org/0000-0003-1732-4207
D. Nakashima: https://orcid.org/0000-0002-5818-2850
Y. Ikuta: https://orcid.org/0000-0002-8419-9154
F. Tajima: https://orcid.org/0000-0003-2091-7636
H. Kimura: https://orcid.org/0000-0003-0005-5805
N. Adachi: https://orcid.org/0000-0002-5570-3729

Supplemental material

There is no supplemental material in this article.

References

1. Cramer MN and Jay O. Biophysical aspects of human thermoregulation during heat stress *Auton Neurosci* 2016; 196: 3–13.
2. Kenny GP and McGinn R. Restoration of thermoregulation after exercise. *J Appl Physiol* 2017; 122: 933–944.
3. Kenny GP and Jay O. Thermometry, calorimetry, and mean body temperature during heat stress. *Compr Physiol* 2013; 3: 1689–1719.
4. Stapleton JM, Poirier MP, Flouris AD, et al. At what level of heat load are age-related impairments in the ability to dissipate heat evident in females? *PLoS One* 2015; 10: e0119079.

5. Gass GC, McLellan TM and Gass EM. Effects of prolonged exercise at a similar percentage of maximal oxygen consumption in trained and untrained subjects. *Eur J Appl Physiol Occup Physiol* 1991; 63: 430–435.
6. Mora-Rodriguez R, Del Coso J, Hamouti N, et al. Aerobically trained individuals have greater increases in rectal temperature than untrained ones during exercise in the heat at similar relative intensities. *Eur J Appl Physiol* 2010; 109: 973–981.
7. Lertwanich P. The disabled athletes and related medical conditions. *Siriraj Med J* 2009; 61: 100–103.
8. Fagher K and Lexell J. Sports-related injuries in athletes with disabilities. *Scand J Med Sci Sports* 2014; 24: e320–331.
9. Tuakli-Wosornu YA, Mashkovskiy E, Ottesen T, et al. Acute and chronic musculoskeletal injury in para sport: a critical review. *Phys Med Rehabil Clin N Am* 2018; 29: 205–243.
10. Grobler L, Derman W, Racinais S, Ngai ASH, et al. Illness at a para athletics track and field world championships under hot and humid ambient conditions. *PM R* 2019; 11: 919–925.
11. Gibson OR, James CA, Mee JA, et al. Heat alleviation strategies for athletic performance: a review and practitioner guidelines. *Temperature (Austin)* 2019; 7: 3–36.
12. Griggs KE, Stephenson BT, Price MJ, et al. Heat-related issues and practical applications for paralympic athletes at Tokyo 2020. *Temperature* 2019; 27: 37–57.
13. Price MJ. Thermoregulation during exercise in individuals with spinal cord injuries. *Sports Med* 2006; 36: 863–879.
14. Price MJ and Campbell IG. Effects of spinal cord lesion level upon thermoregulation during exercise in the heat. *Med Sci Sports Exerc* 2003; 35: 1100–1107.
15. Griggs KE, Leicht CA, Price MJ, et al. Thermoregulation during intermittent exercise in athletes with a spinal-cord injury. *Int J Sports Physiol Perform* 2015; 10: 469–475.
16. Forsyth P, Miller J, Pumpa K, et al . Independent influence of spinal cord injury level on thermoregulation during exercise. *Med Sci Sports Exerc* 2019; 51: 1710–1719.
17. Wendt D, van Loon LJ and Lichtenbelt WD. Thermoregulation during exercise in the heat: strategies for maintaining health and performance. *Sports Med* 2007; 37: 669–682.
18. Ghoseiri K and Safari MR. Prevalence of heat and perspiration discomfort inside prostheses: literature review. *J Rehabil Res Dev* 2014; 51: 855–868.
19. Klute GK, Rowe GI, Mamishev AV, et al. The thermal conductivity of prosthetic sockets and liners. *Prosthet Orthot Int* 2007; 31: 292–299.
20. Andrews AM, Deehl C, Rogers RL, et al. Core temperature in service members with and without traumatic amputations during a prolonged endurance event. *Mil Med* 2016; 181: 61–65.
21. Hasegawa H, Makino H, Fukuhara K, et al. Thermoregulatory responses of lower limb amputees during exercise in a hot environment. *J Therm Biol* 2020; 91: 102609.
22. Gerrett N, Kingma BRM, Sluijter R, et al. Ambient conditions prior to Tokyo 2020 Olympic and Paralympic Games: considerations for acclimation or acclimatization strategies. *Front Physiol* 2019; 10: 414.
23. Marfell-Jones M; Olds T, Stewart A, et al. *International Standards for Anthropometric Assessment*. Potchefstroom, South Africa: ISAK; 2006.
24. Ohtsuki K. Significance of evaluation of exercise capacity in healthy male subjects during upper limb exercise. *J Phys Ther Sci* 2006; 21: 209–213.
25. Zubac D, Reale R, Karnincic H, et al. Urine specific gravity as an indicator of dehydration in olympic combat sport athletes; considerations for research and practice. *Eur J Sport Sci* 2018; 18: 920–929.
26. Moran DS and Mendal L. Core temperature measurement: methods and current insights. *Sports Med* 2002; 32: 879–885.
27. Sawka MN, Latzka WA, Montain SJ, et al. Physiologic tolerance to uncompensable heat: intermittent exercise, field vs laboratory. *Med Sci Sports Exerc* 2001; 33: 422–430.
28. DuBois D and DuBois EF. A formula to estimate the approximate surface area if height and weight be known. *Arch Intern Med* 1916; 17: 863–871.
29. Osterkamp LK. Current perspective on assessment of human body proportions of relevance to persons with amputations. *J Am Diet Assoc* 1995; 95: 215–218.
30. Colangelo PM, Welch DW, Rich DS, et al. Two methods for estimating body surface area in adult persons with amputations. *Am J Hosp Pharm* 1984; 41: 2650–2655.
31. Cohen J. A power primer. *Psychol Bull* 1992; 112: 155–159.
32. Cramer MN and Jay O. Compensatory hyperhidrosis following thoracic sympathectomy: a biophysical rationale. *Am J Physiol Regul Integr Comp Physiol* 2015; 302: 352–356.
33. Périard JD, Racinais S and Sawka MN. Adaptations and mechanisms of human heat acclimation: applications for competitive athletes and sports. *Scand J Med Sci Sports* 2015; 25: 20–38.
34. Hansen C, Godfrey B, Wixom J, et al. Incidence, severity, and impact of hyperhidrosis in people with lower-limb amputation. *J Rehabil Res Dev* 2015; 52: 31–40.
35. Hirata K, Nagasaka T and Noda Y. Venous return from distal regions affects heat loss from the arms and legs during exercise-induced thermal loads. *Eur J Appl Physiol Occup Physiol* 1989; 58: 865–872.
36. Smith CJ and Havenith G. Body mapping of sweating patterns in male athletes in mild exercise-induced hyperthermia. *Eur J Appl Physiol* 2011; 111: 1391–1404.
37. Budd GM. Wet-bulb globe temperature (WBGT)—its history and its limitations. *J Sci Med Sport* 2008; 11: 20–32.
38. Anderson GB, Bell ML and Peng RD. Methods to calculate the heat index as an exposure metric in environmental health research. *Environ Health Perspect* 2013; 121: 1111–1119.
39. NWS (National Weather Service). *Meteorological Conversions and Calculations: Heat Index Calculator*. National Oceanic and Atmospheric Administration (NOAA), National Weather Service (NWS), National Centers for Environmental Prediction, Weather Prediction Center 5830 University Research Court, College Park, Maryland 20740 2020. https://www.wpc.ncep.noaa.gov/html/heatindex.shtml.
40. NWS (National Weather Service). *Heat Index*. National Oceanic and Atmospheric Administration (NOAA), National Weather Service (NWS) Amarillo, TX, 1900 English Road, Amarillo, TX 79108 2020. https://www.weather.gov/safety/heat-index.
41. Attia M and Engel P. Thermoregulatory set point in patients with spinal cord injuries (spinal man). *Paraplegia* 1983; 21: 233–248.
42. Trbovich M, Koek W and Ortega C. Efficacy of water spray for evaporative cooling in athletes with spinal cord injury. *Spinal Cord Ser Cases* 2019; 5: 51.
43. Cheuvront SN and Kenefick RW. Dehydration: physiology, assessment, and performance effects. *Compr Physiol* 2014; 4: 257–285.
44. O'Connor FG. Sports medicine: exertional heat illness. *FP Essent* 2019; 482: 15–19.