

Research Article

Sweat Rate Monitoring During Maximal Exercise in Healthy Soccer Players: A Close Relationship with Anaerobic Threshold

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- Ergospirometry
- Galvanic skin response
- Soccer players

Abstract

Purpose: Sweating is a homeostatic phenomenon regulated by both thermal and non-thermal factors during exercise. There are no evidences whether anaerobic metabolism induced during isotonic maximal exercise can modify sweating rate. Aim of the study was to investigate the relationship between sweating and the anaerobic threshold (AT).

Methods: The sweat rate in thirteen soccer players was measured by a sensor providing a continuous monitoring of sweating, whereas the anaerobic threshold was assessed with ergospirometry during maximal isotonic stress test. During stress test, cardio respiratory, metabolic and galvanic skin response (GSR) were also monitored.

Results: At AT, stroke volume, heart rate and systolic blood pressure significantly increased ($p < 0.001$), as well as GSR ($p = 0.04$). Sweat rate abruptly increased at AT compared with rest ($p < 0.001$). AT-to-rest changes in sweating rate were associated with concomitant changes in VO_2 max ($r = 0.82$, $p < 0.001$), heart rate ($r = 0.73$, $p = 0.04$) and GSR ($r = 0.79$, $p = 0.001$).

Conclusion: We suggest that aerobic-to-anaerobic switch is associated with a sudden increase in sweating likely induced by sympathetic activation. Considering the role of hydration in preserving the health status and optimizing the physical performance, we believe that this finding may have relevant practical implication in particular in soccer, which is characterized by an alternation of aerobic and anaerobic phases.

ABBREVIATIONS

AT: Anaerobic Threshold; VO_2 : Oxygen Uptake; VCO_2 : Carbon Dioxide Output; VO_2/Kg : VO_2 Normalized For The Body Surface Area; VE/VCO_2 : Equivalent Ventilators For Carbon Dioxide; VE/VO_2 : Equivalent Ventilators For Oxygen; VE: Ventilation; VO_2 Peak: Peak Oxygen Uptake; VO_2/HR : Oxygen Uptake And Its Relationship With Heart Rate; BF: Breathing Frequency; BR: Breathing Respiratory Reserve; RER: Respiratory Exchange

Ratio; CO: Cardiac Output; SV: Stroke Volume; Sat.O₂%: Oxygen Saturation; REST: Before Starting Exercise; INT: Intermediate Point; PEAK: Corresponding to the Peak VO_2 ; REC: Recovery Phase; GSR: Galvanic Skin Responses; ANOVA: Analysis of Variance; SD: Standard Deviation; IQR: Median With Interquartile Range; HR: Heart Rate; HRR: Heart Rate Reserve; O₂/HR: Oxygen Pulse; DBP: Diastolic Blood Pressure; SBP: Systolic Blood Pressure; RH: Relative Humidity

INTRODUCTION

During physical exercise, the maintenance of body temperature in a physiological range is a crucial factor [1]. In fact, the rise in body core temperature and the subsequent dehydration causes potential negative effects on the perception of fatigue, the efficiency of exercise performance and brain's motor control centers, with adverse consequences for athlete's health [2,3]. In particular, athletes have a peculiar ability to regulate their internal and skin temperatures by sweating to optimize physical performance during dynamic exercise.

Sweating is a homeostatic phenomenon occurring before a measurable change in internal temperature during exercise, and it is characterized by complex and integrated mechanisms of regulation [4]. This suggests that rapid variations in sweating rate during exercise are independent from changes in internal, muscle, or skin temperatures and are therefore modulated by both thermal and non-thermal factors [5-7]. In general, the non-thermal sweating response during dynamic exercise is a feed-forward mechanism of thermoregulation, because this effect precedes changes in the thermal factors.

Although there are several experimental evidences on sweating during recovery from exercise [8] and during isometric exercise [9], the integrated control mechanism of sweating at dynamic exercise is still partly unknown. Currently, to our knowledge, no studies have examined the sweating response during the different metabolic phases of isotonic exercise. The knowledge and the understanding of the involvement of various metabolic components is a crucial aspect to design training programs, develop personalized assessment protocols and maximize training and competition performance.

In the context of the aerobic-to-anaerobic metabolism and sweat secretion, the autonomic nervous system is likely involved, since the metabolic switch is associated with a gradual vagal withdrawal and a sympathetic activation [5, 10]. Similarly, sweat secretion is also under the autonomic control, and is activated by nerve impulses from the sympathetic nervous system through several cholinergic and few adrenergic terminals [11].

Anaerobic threshold (AT), usually measured with ergospirometry and defined as the highest sustained intensity of exercise for which measurement of oxygen uptake can account for the entire energy requirement, identifies the metabolic switch between the aerobic and anaerobic phase observed during exercise [12]. The purpose of this study was to test the hypothesis that the aerobic-to-anaerobic metabolic switch influences sweat rate, with particular reference to the possible involvement of non-thermal factors in modulating sweating response. For this purpose, by means of a sweat rate sensor [13] providing continuous sweat rate data during exercise, we quantified sweating responses in healthy soccer players at different metabolic phases of maximal exercise stress test (ergospirometry), including aerobic and anaerobic phases (before and during AT), maximal exercise and recovery.

MATERIALS AND METHODS

Participants

Thirteen soccer players with a mean age \pm SD of 25.9 \pm 4.34

years, height 178 \pm 6.3 cm and weight 74 \pm 6.88 Kg were recruited for this study. Athletes were examined according to a standardized protocol consisting of a clinical and functional assessment. All participants were free from cardiovascular or other systemic diseases and none was taking any medications. The local Ethics Committee (Pisa, Italy, protocol number for study acceptance 2805) approved the experimental protocol. After receiving a description of the procedures and potential risks, each study participant gave his written informed consent prior to testing.

Spirometry and Ergospirometry

Ergospirometry has been performed in an environment with a temperature of 25° C and 35% relative humidity. The "breath by breath" analysis of flows and concentrations of inhaled and exhaled respiratory gases (VO₂ and VCO₂) obtained via mass flow and fast-responding gas analyzers (paramagnetic oximeter and infrared analyzers), allowed the determination of the following parameters: peak oxygen uptake (VO₂peak) and its relationship with heart rate (pulse oxygen or VO₂/HR), derived ventilation parameters (total ventilation minute, VE, and breathing respiratory reserve, BR), equivalent gas ventilators (VE/VO₂, VE/VCO₂). The AT for a respiratory ratio (RER) greater than 1 was investigated using indirect measurements of respiratory variables derived from the increase in slope of VO₂/VCO₂ and sudden increase in the ventilatory response to the VE/VO₂ exercise [14]. The prediction equations of Jones were used as reference values of the individual measurements of VO₂, according to the guidelines proposed for normal adult subjects [15]. The quantification of maximum exercise tolerance was expressed as peak oxygen uptake normalized to the body surface area (peak VO₂/kg) on the modified Weber and Janicki scale, taking into account the athlete's anthropometric data [16]. Peak VO₂/kg values lower than 30 ml/min/Kg were considered indicators of physical deconditioning. All measurements were performed according to the standardized criteria of the American Thoracic Society [17]. Cardiac output (CO) was non-invasively estimated from oxygen uptake (VO₂) during exercise according to the following formula: CO=VO₂/C(a-vD_{O₂}) where C(a-vD_{O₂}) corresponds to the arteriovenous oxygen difference.¹⁷ Stroke volume was calculated as CO/HR[18].

For the electrocardiogram, we used a 10-lead electrocardiograph (CAM 14 care fusion) with six precordial and four peripheral leads. For the ergospirometric test, a continuous "ramp" incremental type of protocol was chosen on a cycle ergometer (Sensormedics V_{max} system), aimed at the attainment of a functional exhaustion for each athlete (maximal exercise). The protocol consisted in 25 watts increases every 60 seconds from an initial workload of 0 watts. The arterial oxygen saturation (Sat.O₂%) was measured by pulse oximetry.

According to the ergospirometry examination, the following steps were identified: 1) rest (REST), before starting exercise, 2) intermediate point (INT), corresponding to the middle point between rest and AT step, 3) anaerobic threshold (AT); 4) exercise stress test peak, corresponding to the peak VO₂ (PEAK), and 5) recovery phase (REC).

Sweating Rate Quantification

The sweat rate sensor consisted of an open capsule equipped

with two humidity and temperature sensors (SHT25, Sensirion AG) placed at two different heights from the skin (0.2 cm and 1 cm)[13,19]. A dedicated read-out electronic retrieved the relative humidity (RH) values and calculated the corresponding partial pressures, P (mmHg), of water vapour. The density of water vapour flow, J (g/m²·h), emitted from the skin was calculated using Fick's first law of diffusion, which states that J is proportional to $\Delta P / \Delta x$, where ΔP is the pressure difference and Δx is 0.8 cm. The proportionality constant can be found with the calibration methods described in Imhof [20]. The sweat rate sensor was placed on the volunteers' forearm and kept in the same position until the end of each cycling test. The sampling time was 2s and data were saved on a removable SD card.

Temperature and Galvanic Skin Response

The participants' skin temperatures and galvanic skin responses (GSRs, uS) were monitored by a SenseWear™ Armband (Body media) [21]. Skin temperature was measured by a thermistor situated on the Armband backside. The Armband has two hypoallergenic stainless metal pads on its backside in contact with the skin. One pad is thermally connected to the thermistor and allows temperature measurements. The two pads are also used to measure GSR, which is the value of the skin resistance between the pads. A low level electric voltage is applied to the skin and, measuring the current, the conductance can be found by Ohm's law. When the activity of sweat glands increases, the skin conductivity also increases, due to the presence in sweat of electrolytes such as Na⁺, Cl⁻ and K⁺.

Statistical Analysis

The Kolmogorov-Smirnov test was used to assess the normality

of data. Repeated-measure analysis of variance (ANOVA) was employed to detect the overall changes throughout the phases of the ergospirometric test. Logarithmic transformations were applied to skewed variables having a non-Gaussian distribution according to the Kolmogorov-Smirnov test. Post-hoc comparison between baseline and subsequent phases was carried out using the Dunnett's test for the correction of significance.

For each parameter, changes (Δ) were calculated as the difference between values at each phase and baseline measurements (REST phase), and evaluated by paired Student's t-test for difference in mean values and Wilcoxon test for skewed variables. The Pearson's (R) and Spearman's (ρ) correlation coefficients were employed to assess association between changes for Gaussian and skewed variables, respectively.

A p-value less than 0.05 were considered statistically significant. Data are presented as mean \pm standard deviation (SD) or median with interquartile range (IQR). Analyses were performed using SPSS (version 21, IBM Corp, Armonk, NY, USA).

RESULTS AND DISCUSSION

Cardiovascular and respiratory response

The values of each parameter at every stage of the experimental protocol are shown in Table 1. All subjects performed a maximal exercise for metabolic stress, with a ventilatory threshold between 60-80% of maximum oxygen uptake, and for maximum heart rate corresponding, on average, to 97% of the theoretical value predicted), in the absence of pathological changes on ventilatory and cardiovascular responses. On average, athletes had a moderate degree of conditioning to physical exercise ($VO_{2peak} = 36.5 \pm 6.1$ mL/min/kg). At the onset of the anaerobic

Table 1: Mean values of cardio respiratory parameters during the study steps.

	REST	INT	AT	PEAK	REC
Load (Watts)	0 \pm 0	75 \pm 19.6	144.6 \pm 36.9	233.9 \pm 23.2	0 \pm 0
BF (min ⁻¹)	18.4 \pm 2.9	20.2 \pm 4.3	23.9 \pm 9.8	30.6 \pm 8.4	27.7 \pm 4.6
VE (L/min)	12 \pm 1.8	23.8 \pm 5.8	49.3 \pm 17.7	76 \pm 20.4	60 \pm 11.6
BR (%)	93.9 \pm 1.5	88 \pm 4.1	76.4 \pm 9.1	56.2 \pm 8.9	71.1 \pm 4.8
VCO ₂ (mL/min)	297.9 \pm 51.9	744.3 \pm 169.9	1853.3 \pm 663.6	2769.8 \pm 515.9	1975.1 \pm 264.7
VO ₂ (mL/min)	363.3 \pm 68.7	902.8 \pm 226.7	1832.6 \pm 639	2334 \pm 423.9	1390.9 \pm 174.5
VO ₂ /kg (mL/min/kg)	4.8 \pm 1	12.2 \pm 3.6	23.1 \pm 6.5	36.5 \pm 6.1	17.7 \pm 1.8
VE/VCO ₂	35 \pm 6.6	28.8 \pm 2.6	25.4 \pm 2.8	26.4 \pm 4.9	29.4 \pm 3.9
VE/VO ₂	29.5 \pm 5	24.1 \pm 3	25.2 \pm 2.8	31.6 \pm 8.6	44.5 \pm 7
RER	0.8 \pm 0.1	0.8 \pm 0.1	1.1 \pm 0	1.2 \pm 0.1	1.5 \pm 0.1
HR (min ⁻¹)	79.1 \pm 15.6	94.4 \pm 18.2	125.4 \pm 21.5	159.1 \pm 13.8	136.6 \pm 12.4
HRR (min ⁻¹)	114.8 \pm 16	98.8 \pm 17.1	67.9 \pm 19	34.1 \pm 12.4	56.6 \pm 11.1
O ₂ /HR (mL/beat)	4.6 \pm 1	9.6 \pm 2.6	13.7 \pm 3	17.3 \pm 3.4	9.5 \pm 1.5
DBP (mmHg)	78.2 \pm 9.1	78.2 \pm 10.7	81.1 \pm 8.4	84.3 \pm 9.6	80 \pm 9.8
SBP (mmHg)	122.9 \pm 12.7	128.2 \pm 16	147.5 \pm 16.3	180.4 \pm 23.3	167.9 \pm 21.5
CO (L/min)	5.2 \pm 0.9	10.3 \pm 1.7	15.5 \pm 3	17.6 \pm 3.1	13.1 \pm 1.8
SV (mL)	67.9 \pm 12.9	112.1 \pm 25.8	125.2 \pm 24.2	111.1 \pm 19.3	94.8 \pm 12.6

Abbreviations: BF : VE : BR : Breathing Respiratory Reserve; VCO₂ : carbon dioxide output; VO₂ : oxygen uptake; VO₂/kg : VE/VCO₂ : VE/VO₂ : RER : Respiratory exchange ratio; HR : Heart Rate; HRR : Heart Rate Reserve; O₂/HR : DBP : Diastolic Blood Pressure; SBP : Systolic Blood Pressure; CO : Cardiac Output; SV : Stroke Volume. Data are reported as mean \pm SD.

phase, Qtc doubled as compared to the basal values whereas there was a three-fold increase in VO₂. Stroke volume, heart rate and systolic blood pressure increased on average at AT about 85%, 46% and 25% respectively (all $p < 0.001$), whereas the diastolic blood pressure was unchanged ($p = 0.18$).

Sweating Rate Response

The values of sweat rate at every protocol stage are shown in Figure 1, panel a. As compared to baseline, sweating rate was unchanged in the intermediate stage ($p = 0.36$), whereas it abruptly increased four times with respect to rest at AT ($p < 0.001$), and continued to increase up to approximately 7-fold at the maximal test peak ($p < 0.001$) (Figure 1a). In addition, for each subject, experimental data of sweating rate was interpolated to match a standard template having the following phase durations: baseline

(210 sec), intermediate point (180 sec), anaerobic threshold (210 sec); exercise stress test peak (30 sec) and recovery phase (120 sec) (Figure 2a).

The AT-to-Rest variation in the sweat rate was strongly associated with the VO₂ max AT-to-Rest change ($r = 0.82$, $p = 0.001$) (Figure 3a). Results were still significant after adjustment for the time of AT onset (partial $r = 0.69$, $p = 0.02$). In addition, the sweat rate increment at AT was positively correlated with the AT-to-Rest variation in heart rate ($r = 0.73$, $p = 0.004$) (Figure 3b) and also showed a moderate correlation with the systolic blood pressure ($r = 0.48$, $p = 0.09$). Also, sweating rate was positively correlated to the AT-to-Rest changes in GSR (Spearman's $\rho = 0.79$, $p = 0.001$) (Figure 3c), even after adjustment for AT onset time (partial $r = 0.61$, $p = 0.04$).

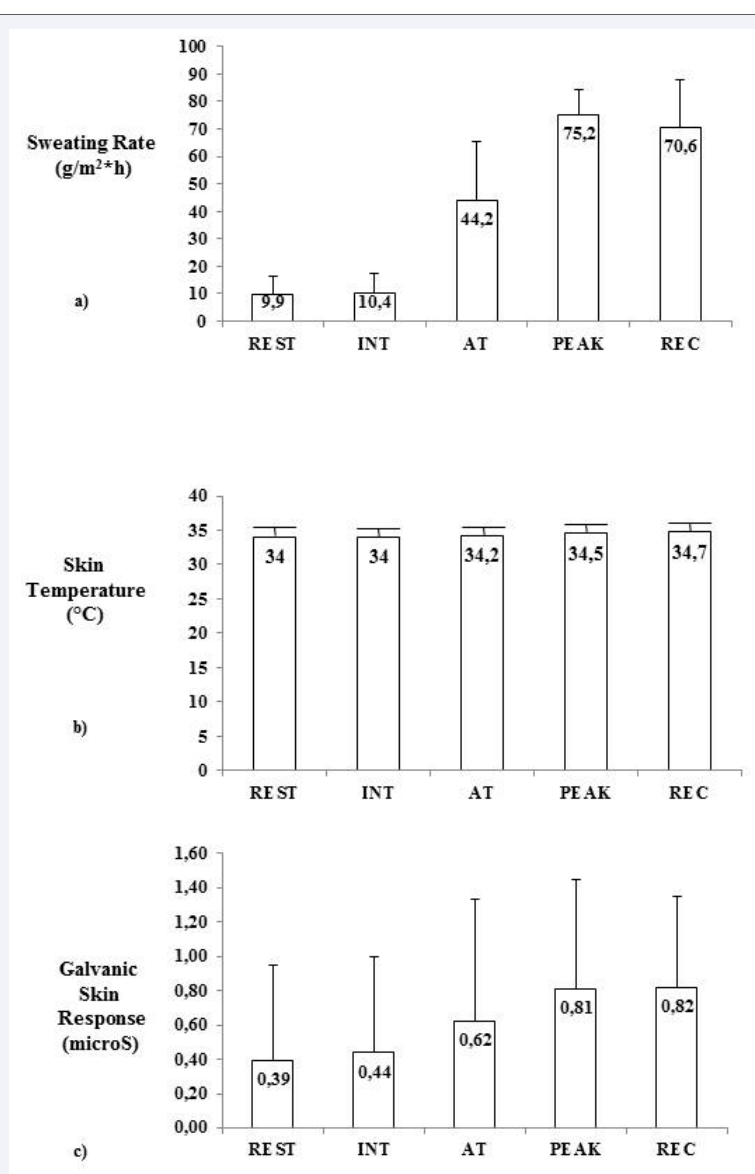


Figure 1 The histograms show the mean and SD values of sweat rate (a), mean skin temperature (b), and galvanic skin response (GSR) (c) in the different steps of the study: REST, INT (intermediate point, corresponding to the middle point between rest and AT step), AT (anaerobic threshold); PEAK (exercise stress test peak, corresponding to the VO₂peak), REC (recovery phase).

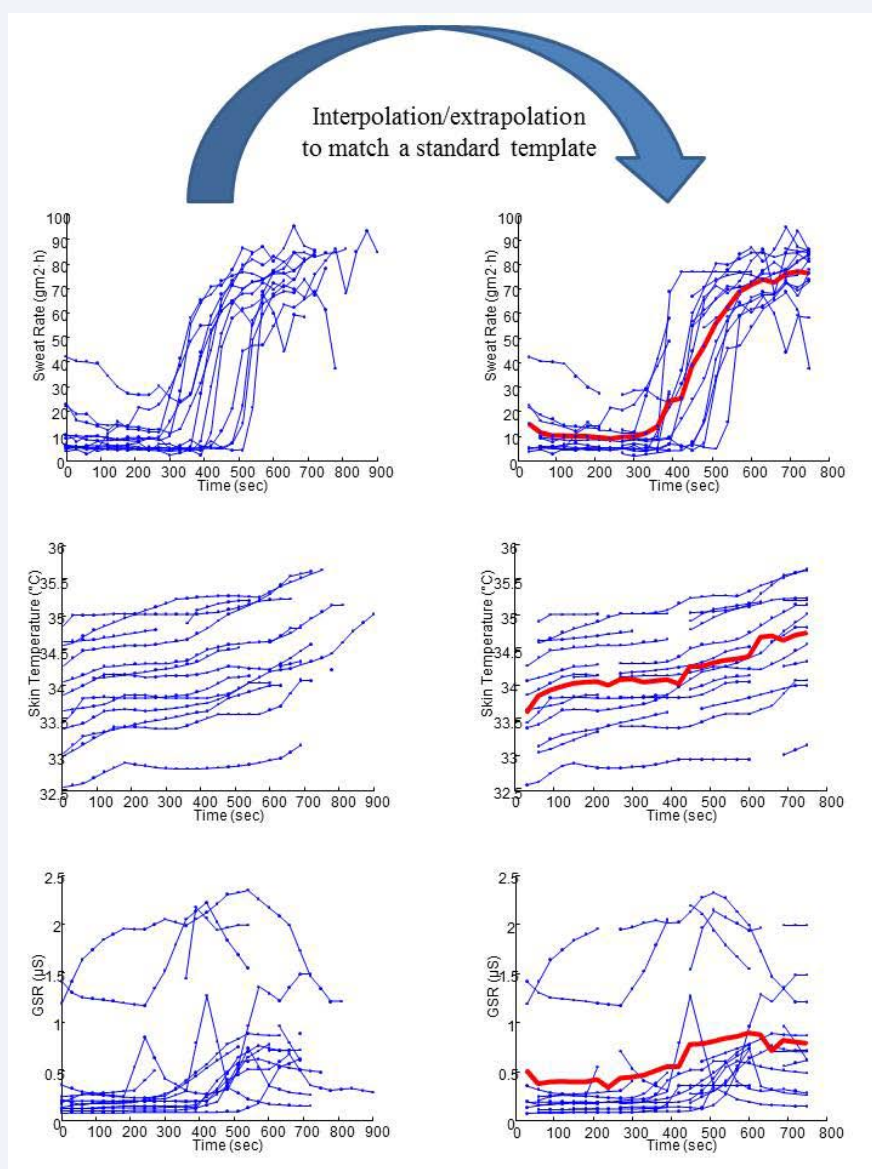


Figure 2 Interpolated data of each subject regarding sweating rate (a), skin temperature (b), and galvanic skin response (GSR) (c) in the different phases of the protocol study: REST, INT, AT, PEAK, REC.

Temperature and galvanic skin response

The values of skin temperature and galvanic skin response at every stages of the experiment are shown in Figure 1b,c. Interpolated data of skin temperature and GSR were displayed in Figure 2b, c. Importantly, body temperature was unchanged during all the steps of the study. GSR was unchanged in the intermediate phase as compared to rest ($p=0.36$), and increased significantly at onset of AT ($p=0.04$), continuing to rise up to the maximal stress test ($p=0.007$) (Figure 1c).

Sweating is a physiological response finely tuned by intermingled mechanisms of thermal and non-thermal factors, whose complexity can explain the remarkable capacity of humans to run and adapt to different environments. The present study, performed on healthy non-professional soccer players, shows that the aerobic-to-anaerobic shift induced by maximal

exercise is characterized by an abrupt increase in sweating rate, independently from the mean skin temperature changes and the associated increased stroke volume, heart rate, systolic blood pressure and galvanic skin response. During our exercise stress tests, sweating rate was unchanged at the intermediate point (i.e. between rest and anaerobic threshold) and suddenly increased at AT. Conversely, skin temperature linearly increased, although not significantly, during the entire duration of the test. The absence of a relationship between sweating rate and body temperature is not surprising, if the crucial role of non-thermal factors in modulating the sweating response during exercise is considered.

As described for the first time by van Beaumont and Bullard [22], many factors unrelated to the rise of the internal temperature are involved during exercise and can modulate the immediate sweating response during a dynamic exercise.

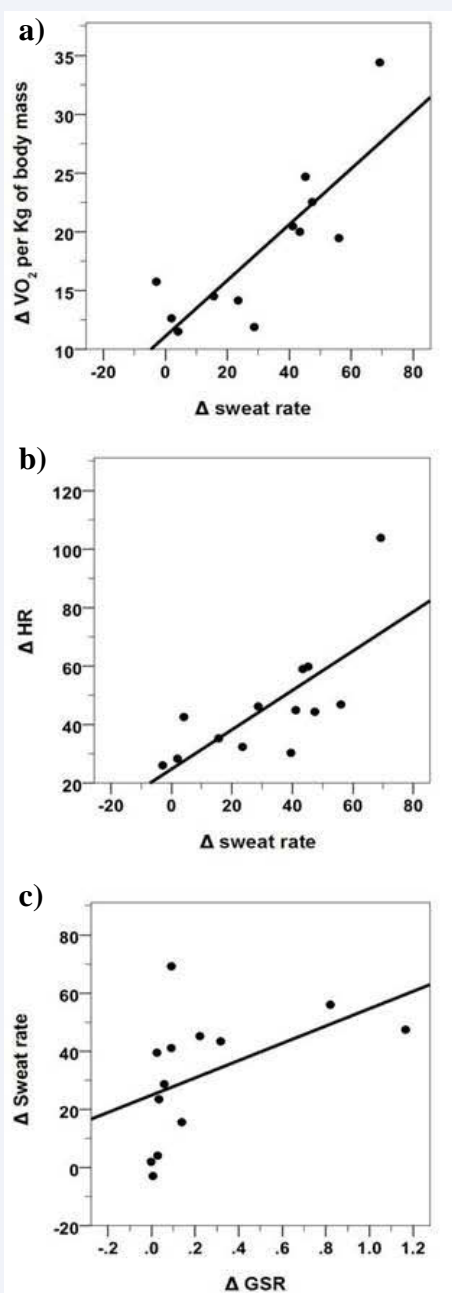


Figure 3 Significant correlations between Δ sweat rate and ΔVO_2 (a) Δ HR (heart rate) (b) and ΔGSR (galvanic skin response) (c). Δ represent the changes of these variables between REST and AT.

From this perspective, Kondo et al. [9] showed that the sweating response to a sustained exercise is primarily related to the magnitude of thermal loading when the internal temperature is high. Conversely, when the increase in the core or skin temperature is mild or moderate as in our study (the mean temperature value reaches 34.5 °C at the maximal stress peak), the sweating response is mainly due to changes of non-thermal factors.

Our results are in line with previous studies showing that both dynamic exercise and brief isometric exercise can increase sweating without significant changes in skin temperature [23-25].

However, there are substantial differences due to the different experimental protocol. In particular, we used an isotonic exercise, whereas isometric exercises were used in the Kondo's [9, 25] and in other studies. Furthermore, the above-mentioned studies were performed at higher ambient temperatures compared to our mean environmental temperature of about 25°C. In addition, body temperature increased slightly during the stress test in our study, whereas it was completely unchanged in the Kondo's study.

These discrepancies could be related to the different stress test we used. In fact, our protocol was characterized by separate anaerobic and aerobic phases, whereas the stress test (handgrip) used in the other studies was shorter and defined by a rapid occurrence of an anaerobic phase.

The non-thermal factors that may affect sweating rate during exercise include the training status, the heat acclimation, the environmental conditions, the host factors and the exercise intensity [6, 26]. In particular, Yanagimoto et al. showed that sweating rate linearly raised with the intensity the isotonic exercise from 30% to 70% VO_2 max without marked changes in the skin or esophageal temperature [27]. Similarly, Kondo et al. demonstrated in a isometric handgrip exercise that the sweating response increased linearly with the maximal voluntary contraction from 30 to 60% [25]. During exercise, other non-thermal factors affecting sweating include central command and peripheral stimulation of mechanosensitive and metabosensitive factors in exercising muscle, baroreflex, osmoreflex and chemoreflex [5,6,24,28,29]. Also, changes in arterial blood pressure [30], the level of hydration [31] and increased plasma osmolality [32], independent on plasma volume, can modulate sweating response during exercise. In particular, the sweating rate on the forearm increased without modifications of the thermal factors at the onset of dynamic exercise in conditions where sweating was ongoing before the exercise [22], suggesting an involvement of the sympathetic activation in the modulation of the sweating rate.

From our point of view this is a relevant element, considering that we observed a widespread increase in sweating during isotonic cycling exercise only at the anaerobic threshold. In particular, the aerobic-to-anaerobic metabolic switch is a cornerstone characteristic of strenuous exercise, in particular in soccer, which is characterized by alternating aerobic-anaerobic metabolic exertions [33]. This switch is associated to a marked change in the autonomic function, with a gradual vagal withdrawal followed by a sympathetic activation [10], as well as to a more efficient energetic profile with reduced heat dissipation [34].

So far, experimental studies have provided inconsistent results with respect to sweating response during dynamic exercise and possible triggered physiological mechanisms. To the best of our knowledge, this is the first study that documents the sweating response during a dynamic exercise, with particular focus to the shift from aerobic to anaerobic phase. Nevertheless, further studies are needed to elucidate the role of non-thermal factors during dynamic exercise.

Importantly, in the present study we assessed sweating rate in a non glabrous region (forearm) innervated by sympathetic

fferent nerve, as demonstrated by the micro neurography technique [28]. In this regard, during exercise or heat stress, the sympathetic nerve activity is linked with galvanic skin response (an index of sweating) and sweating rate [30,35]. Accordingly, we observed a progressive decrease of skin response (galvanic skin response, GSR) during the different stages of the exercise. These changes were more pronounced at the AT compared to baseline or intermediary point, in which increases in HR and SBP were more evident. All together these results obtained in normothermic subjects suggest that the rise in sweating rate at AT may be possibly due to the central command, which has a greater influence on muscle metaboreceptors. In fact, according to Shibasaki's results [36], central command is capable of modulating the sweating rate, but its effect is reduced when sweating rate is high as in our study. In this condition, the increase in sweating rate is mainly related to metaboreceptor stimulation [23,37], and is also generally associated with a rise in arterial blood pressure accompanied by a reduction in cutaneous vascular conductance [23]. Importantly, the role of metaboreceptor is further strengthened when sweating increase occurs without an arterial blood pressure rise, and especially in absence of the baroreceptor modulation [24]. Further, we identified a positive correlation between GSR, HR and SBP variations and sweating rate, suggesting that the sweating response during AT may start from the metaboreceptor stimulus with the following activation of the sympathetic pathway. In this frame, also the increased cardiac performance we observed, consisting in raised cardiac output and stroke volume at the AT level, is in agreement with the evidence that muscle metaboreceptor is one of the physiological mechanisms modulating central hemodynamics [38]. The evidence that anaerobic metabolism increases sweating has relevant practical implications considering water loss and thus dehydration, in particular in the mixed aerobic-anaerobic sports such as soccer, characterized by brief but intensive and repetitive anaerobic efforts. In particular, although the aerobic metabolism influences the energy delivery during a soccer game, the key actions, such as short sprints and jumps, are covered by the anaerobic metabolism, which is crucial for the physical performance and the match outcome [39]. In this context, dehydration and the consequent hyperthermia, together with lactate accumulation, is the main cause of reduced performance, although muscle strength appears to be relatively unaffected [40]. During exercise, fluid loss due to sweating is distributed in different proportions among the various body districts: plasma, extracellular and intracellular water. In particular, the dehydration may induce a reduction in the blood flow to the exercising muscles and to the skin, and the falling cardiac output is one of the signals responsible for the weakness during prolonged exercise [41]. In addition, the decline in plasma volume that accompanies dehydration may be of particular importance in influencing the performance. The American College of Sports Medicine states that an adequate fluid replacement helps to maintain hydration and, therefore, promotes the health, safety, and optimal physical performance of individuals having during regular physical activity. This position statement is based on the scientific evidence concerning the influence of fluid replacement on exercise performance and the risk of thermal injury associated with dehydration and hyperthermia [42]. In fact, although the physiological consequences of dehydration due to sweat loss

during exercise have been thoroughly addressed, relatively little scientific evidence is available concerning the effects of a fluid deficit incurred prior to and during exercise, especially in study groups practicing sports in warm environments. However, it has been shown that moderate levels of hypohydration lead to an impairment of the cognitive function and decrease the subjective perception of alertness and the ability to concentrate, impairing the performance capacity [4]. A limitation of the study was that we measured skin instead of core temperature. However, the main objective of our study was to assess the relationship between AT and sweating. In this context, previous studies showed that skin and deep-body (esophageal) temperature did not change during handgrip [25].

CONCLUSION

The results of this study showed that the aerobic-to-anaerobic shift during exercise is associated with sudden increase in sweating likely induced by sympathetic activation. If confirmed in a larger set of subjects, these data may have relevant practical implication due to the role of hydration in preserving health status and optimizing physical performance. Understanding the involvement of various metabolic components during sport activity is a crucial aspect to prescribe personalized training programs and maximize training and competition performance.

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Salvo P. was involved in the data collection, in final writing, and approval of the paper submitted.

Mastorci F was involved in the data collection, in final writing, and approval of the paper submitted.

Catapano G. was involved in the study design, in the data collection; in the drafting, and final writing and approval of the paper.

Sordi L. was involved in the data collection and approval of the paper.

Piaggi P. was involved in the statistical analysis, in final writing and approval of the paper submitted.

Di Francesco F. was involved in the study design, in the data collection; in the drafting, and final writing and approval of the paper.

REFERENCES

1. Ozgüven KT, Kurdak SS, Maughan RJ, Zeren C, Korkmaz S, Yazici Z, et al. Effect of hot environmental conditions on physical activity patterns and temperature response of football players. *Scand J Med Sci Sports*. 2010; 20: 140-147.
2. González-Alonso J, Teller C, Andersen SL, Jensen FB, Hyldig T, Nielsen B. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol* (1985). 1999; 86: 1032-1039.
3. Nybo L, Secher NH. Cerebral perturbations provoked by prolonged exercise. *Prog Neurobiol*. 2004; 72: 223-261.

4. Maughan RJ. Impact of mild dehydration on wellness and on exercise performance. *Eur J Clin Nutr.* 2003; 57 Suppl 2: S19-23.
5. Shibasaki M, Wilson TE, Crandall CG. Neural control and mechanisms of eccrine sweating during heat stress and exercise. *J Appl Physiol* (1985). 2006; 100: 1692-1701.
6. Kondo N, Nishiyasu T, Inoue Y, Koga S. Non-thermal modification of heat-loss responses during exercise in humans. *Eur J Appl Physiol.* 2010; 110: 447-458.
7. Todd G, Gordon CJ, Groeller H, Taylor NA. Does intramuscular thermal feedback modulate eccrine sweating in exercising humans? *Acta Physiol (Oxf).* 2014; 212: 86-96.
8. Kenny GP, Periard J, Journeay WS, Sigal RJ, Reardon FD. Effect of exercise intensity on the postexercise sweating threshold. *J Appl Physiol* (1985). 2003; 95: 2355-2360.
9. Kondo N, Horikawa N, Aoki K, Shibasaki M, Inoue Y, Nishiyasu T, et al. Sweating responses to a sustained static exercise is dependent on thermal load in humans. *Acta Physiol Scand.* 2002; 175: 289-295.
10. Simões RP, Castello-Simões V, Mendes RG, Archiza B, Santos DA, Machado HG, et al. Lactate and heart rate variability threshold during resistance exercise in the young and elderly. *Int J Sports Med.* 2013; 34: 991-996.
11. Vetrugno R, Liguori R, Cortelli P, Montagna P. Sympathetic skin response: basic mechanisms and clinical applications. *Clin Auton Res.* 2003; 13: 256-270.
12. Svedahl K, MacIntosh BR. Anaerobic threshold: the concept and methods of measurement. *Can J Appl Physiol.* 2003; 28: 299-323.
13. Salvo P, Di Francesco F, Costanzo D, Ferrari C, Trivella MG, De Rossi D. A wearable sensor for measuring sweat rate. *IEEE Sensors.* 2010; 10: 1557-1558.
14. Wasserman K, Hansen JE, Sue DY, Stringer WW, Whipp BJ. Principles of Exercise Testing and Interpretation. Fourth Edition; Lippincott Williams and Wilkins, 2005.
15. Jones NL, Summers E, Killian KJ. Influence of age and stature on exercise capacity during incremental cycle ergometry in men and women. *Am Rev Respir Dis.* 1989; 140: 1373-1380.
16. Weber KT, Janicki JS. Cardiopulmonary exercise testing for evaluation of chronic cardiac failure. *Am J Cardiol.* 1985; 55: 22A-31A.
17. Miller MR, Crapo R, Hankinson J, Brusasco V, Burgos F, Casaburi R, et al. General considerations for lung function testing. *Eur Respir J.* 2005; 26: 153-161.
18. Stringer WW, Hansen JE, Wasserman K. Cardiac output estimated noninvasively from oxygen uptake during exercise. *J Appl Physiol* (1985). 1997; 82: 908-912.
19. Nilsson GE. Measurement of water exchange through skin. *Med Biol Eng Comput.* 1977; 15: 209-218.
20. Imhof RE, De Jesus ME, Xiao P, Ciortea LI, Berg EP. Closed-chamber transepidermal water loss measurement: microclimate, calibration and performance. *Int J Cosmet Sci.* 2009; 31: 97-118.
21. Liden CB, Wolowicz M, Stivorc J, Teller A, Kasabach C, Vishnubhatla S, et al. Characterization and Implications of the Sensors Incorporated into the SenseWear Armband for Energy Expenditure and Activity Detection, 2012.
22. Van Beaumont W, Bullard RW. Sweating exercise stimulation during circulatory arrest. *Science.* 1966; 152: 1521-1523.
23. Crandall CG, Stephens DP, Johnson JM. Muscle metaboreceptor modulation of cutaneous active vasodilation. *Med Sci Sports Exerc.* 1998; 30: 490-496.
24. Shibasaki M, Kondo N, Crandall CG. Evidence for metaboreceptor stimulation of sweating in normothermic and heat-stressed humans. *J Physiol.* 2001; 534.
25. Kondo N, Tominaga H, Shibasaki M, Aoki K, Okada S, Nishiyasu T. Effects of exercise intensity on the sweating response to a sustained static exercise. *J Appl Physiol* (1985). 2000; 88: 1590-1596.
26. Thomas C, Sirvent P, Perrey S, Raynaud E, Mercier J. Relationships between maximal muscle oxidative capacity and blood lactate removal after supramaximal exercise and fatigue indexes in humans. *J Appl Physiol* (1985). 2004; 97: 2132-2138.
27. Yanagimoto S, Kuwahara T, Zhang Y, Koga S, Inoue Y, Kondo N. Intensity-dependent thermoregulatory responses at the onset of dynamic exercise in mildly heated humans. *Am J Physiol Regul Integr Comp Physiol.* 2003; 285: R200-207.
28. Vissing SF, Secher NH, Victor RG. Mechanisms of cutaneous vasoconstriction during upright posture. *Acta Physiol Scand.* 1997; 159: 131-138.
29. Ogawa T. Thermal influence on palmar sweating and mental influence on generalized sweating in man. *Jpn J Physiol.* 1975; 25: 525-536.
30. Bini G, Hagbarth KE, Hynninen P, Wallin BG. Regional similarities and differences in thermoregulatory vaso- and sudomotor tone. *J Physiol.* 1980; 306: 553-565.
31. Montain SJ, Latzka WA, Sawka MN. Control of thermoregulatory sweating is altered by hydration level and exercise intensity. *J Appl Physiol* (1985). 1995; 79: 1434-1439.
32. Shibasaki M, Aoki K, Morimoto K, Johnson JM, Takamata A. Plasma hyperosmolality elevates the internal temperature threshold for active thermoregulatory vasodilation during heat stress in humans. *Am J Physiol Regul Integr Comp Physiol.* 2009; 297: 1706-1712.
33. Stølen T, Chamari K, Castagna C, Wisløff U. Physiology of soccer: an update. *Sports Med.* 2005; 35: 501-536.
34. Craig NP, Norton KI, Bourdon PC, Woolford SM, Stanef T, Squires B, et al. Aerobic and anaerobic indices contributing to track endurance cycling performance. *Eur J Appl Physiol Occup Physiol.* 1993; 67: 150-158.
35. Sugenoja J, Iwase S, Mano T, Ogawa T. Identification of sudomotor activity in cutaneous sympathetic nerves using sweat expulsion as the effector response. *Eur J Appl Physiol Occup Physiol.* 1990; 61: 302-308.
36. Shibasaki M, Secher NH, Selmer C, Kondo N, Crandall CG. Central command is capable of modulating sweating from non-glabrous human skin. *J Physiol.* 2003; 553: 999-1004.
37. Kondo N, Takano S, Aoki K, Shibasaki M, Tominaga H, Inoue Y. Regional differences in the effect of exercise intensity on thermoregulatory sweating and cutaneous vasodilation. *Acta Physiol Scand.* 1998; 164: 71-78.
38. Crisafulli A, Milia R, Lobina A, Caddeo M, Tocco F, Concu A, et al. Haemodynamic effect of metaboreflex activation in men after running above and below the velocity of the anaerobic threshold. *Exp Physiol.* 2008; 93: 447-457.

39. Wragg CB, Maxwell NS, Doust JH. Evaluation of the reliability and validity of a soccer-specific field test of repeated sprintability. *Eur J Appl Physiol.* 2000; 83: 77-83.
40. Coyle EF. Physiological determinants of endurance exercise performance. *J Sci Med Sport.* 1999; 2: 181-189.
41. González-Alonso J, Calbet JA, Nielsen B. Muscle blood flow is reduced with dehydration during prolonged exercise in humans. *J Physiol.* 1998; 513: 895-905.
42. Convertino VA, Armstrong LE, Coyle EF, Mack GW, Sawka MN, Senay LC Jr, et al. American College of Sports Medicine position stand. Exercise and fluid replacement. *Med Sci Sports Exerc.* 1996; 28.

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