Physiol. Res. 68: 981-989, 2019 *https://doi.org/10.33549/physiolres.934114*

Relationship Between the Skin Surface Temperature Changes During Sprint Interval Testing Protocol and the Aerobic Capacity in Well-Trained Cyclists

R. HEBISZ[1], P. HEBISZ[1], J. BORKOWSKI[1], I. WIERZBICKA-DAMSKA[1], M. ZATOŃ[1]

[1]Department of Physiology and Biochemistry, University School of Physical Education in Wroclaw, Poland

Received January 7, 2019
Accepted June 20, 2019
Epub Ahead of Print October 25, 2019

Summary

The study investigated whether changes in body surface temperature in a sprint interval testing protocol (SITP) correlated with aerobic capacity in cyclists. The study involved 21 well-trained cyclists. Maximal aerobic power and maximal oxygen uptake relative to lean body mass (LBM-P_{max} and LBM-VO_{2max}, respectively) were determined by incremental exercise testing on a cycle ergometer. SITP was administered 48 hours later and involved four 30-s maximal sprints interspersed with 90-s active recovery. Body surface temperature was recorded at the temple and arm and the delta difference between baseline temperature and temperature measured immediately after the first sprint (ΔTt_1 and ΔTa_1, respectively) and 80 seconds after the fourth sprint (ΔTt_4 and ΔTa_4, respectively) was calculated. Significant correlations were found between ΔTt_4 and LBM-P_{max} and LBM-VO_{2max} (r=0.63 and r=0.75, respectively) with no significant change in ΔTa_1 or ΔTa_4. Body surface temperature, measured at the temple region, can be used to indirectly assess aerobic capacity during maximal sprint exercise.

Key words

Body surface temperature • Thermoregulation • Maximal oxygen uptake • Maximal sprint exercise

Corresponding author

Paulina Hebisz, University School of Physical Education, Department of Physiology and Biochemistry, 35 J.I. Paderewski Avenue, 51-612 Wroclaw, Poland. E-mail: paulinahebisz@interia.pl

Introduction

Maximal oxygen uptake (VO_{2max}) is one of several physiological factors determining the endurance performance in athletes. (Levine 2008, Lucia *et al.* 2001, Mujika and Padilla 2001a). VO_{2max} is also important in efforts that involve single and repeated sprints due to its relationship with the rate of phosphocreatine resynthesis, phosphate concentration, and energy production via aerobic glycolysis (Girard *et al.* 2011, Glaister 2005). VO_{2max} is treated as a reliable and valid measure of training-induced changes in exercise capacity (Coquart *et al.* 2014) and has been used as a criterion in the athlete selection process (Faria *et al.* 2005). Among non-athletes, VO_{2max} has been reported to reflect the efficiency of the cardiovascular (De Lorenzo *et al.* 2018) and respiratory system (Lopes *et al.* 2018) or muscular content of energy metabolism proteins (Vigelsø *et al.* 2016).

Another important indicator of performance among endurance athletes is the body's thermoregulatory response to exercise, which ensures that the core temperature is maintained within a temperature range which will conserve the physiological functions (Nybo and Nielsen 2001). There exists an inter-relationship between VO_{2max} and exercise-induced changes in internal temperature, in which temperature is dependent on relative exercise load, which can be expressed as a percentage of individual VO_{2max} (Yoshida *et al.* 1997). Hence, individuals with different levels of aerobic capacity show a similar increase in temperature during exercise at a similar relative load despite significant

Fax +420 241 062 164, e-mail: physres@fgu.cas.cz, www.biomed.cas.cz/physiolres

variation in power output. The body's thermoregulatory response to exercise is thought to be dependent, among several other factors, on the distribution of blood and several hemodynamic mechanisms. At the onset of dynamic exercise of large muscle groups, the body signals a vasoconstrictor response in non-exercising muscle (Johnson 1992, Kamon and Belding 1969, Simmons *et al.* 2011). This reduction in blood flow is greatest during maximal-intensity exercise (Taylor *et al.* 1990). Concomitantly, blood flow to working skeletal muscle is enhanced by vasodilatation in order to meet the metabolic demands of contracting muscles (Kenney and Johnson 1992). Previous research has found cutaneous blood flow to be significantly higher in trained subjects compared with untrained cohorts (Fritzsche and Coyle 2000; Johnson 1998). The changes in blood flow and distribution are easily observable by the decrease in body surface temperature in the first minutes of exercise (Kamon and Belding 1969, Simmons *et al.* 2011) followed by an increase in the body surface temperature with continued exercise (Kamon and Belding 1969, Simmons *et al.* 2011) as cutaneous blood flow increases (Johnson and Rowell 1975). The onset of perspiration and further temperature modulation is then dependent on several internal (aerobic capacity and cutaneous blood circulation) and external (ambient temperature and other environmental conditions) factors (Tankersley *et al.* 1991, Wingo *et al.* 2010).

The efficacy of the body's thermoregulatory mechanisms for eliminating excess heat, particularly those which protect brain tissue from overheating, is an important determinant of exercise performance. Body temperature among endurance athletes can reach 41.5 °C during intense exercise (Racinais *et al.* 2019). For this reason, the inclusion of a heat acclimatization component in athlete training has been suggested in order to improve work output and increase VO_{2max} (Lorenzo *et al.* 2010). These physiological adaptations are explained by an increase in plasma volume and have been observed after a heat acclimation program (Lorenzo *et al.* 2010) or after interval (Abderrahman *et al.* 2013) or endurance training (Martino *et al.* 2002, Warburton *et al.* 2004). The exercise-induced expansion in plasma volume is considered by many to be beneficial as it can also enhance performance during heavy exercise by facilitating thermoregulation (Coles and Luetkemeier 2005, Nelson *et al.* 2008) or reducing blood viscosity (Abderrahman *et al.* 2013).

The above cited works suggest a relationship between thermoregulatory efficacy, VO_{2max}, and exercise performance. However, little is known about the effects of maximal exercise on changes in body surface temperature among athletes with high levels of VO_2max and maximal aerobic power. A correlation between aerobic capacity and exercise-induced changes in body surface temperature could provide a regular estimate of VO_{2max} during the training cycle. The aim of this study was to investigate this association in well-trained cyclists during a single sprint interval training session. It was assumed that the high maximal oxygen uptake allows more efficient blood flow through the cutaneous vessels, influencing the elimination of increasing temperature during sprint interval testing protocol.

Methods

Participants

Twenty-one well-trained mountain bike (MTB) cyclists aged 17-22 years participated in the study. All had been regularly training MTB for at least 3 years. Table 1 presents the basic anthropometric and the aerobic capacity characteristics of the cyclists. The study was approved by a local ethics committee and all procedures were designed to be in accordance with the Declaration of Helsinki. The participants were familiarized with the research protocol and provided their written consent to participate in the study.

Table 1. Anthropometric and physiological characteristics of the studied group.

Age [years]	Mass [kg]	Height [cm]	Fat tissue [%]	LBM [kg]	VO_{2max} [$ml \cdot min^{-1} \cdot kg^{-1}$]	P_{max} [$W \cdot kg^{-1}$]	$T_{plateau}$ [s]
18.9 (2.2)	67.3 (6.6)	178.1 (5.6)	13.2 (4.1)	58.3 (7.3)	66.3 (4.6)	5.6 (0.3)	117.6 (51.8)

LBM - lean body mass, VO_{2max} - maximal oxygen uptake; P_{max} - maximal aerobic power; $T_{plateau}$ - time of plateau VO_2max; data are presented as arithmetic mean (standard deviation).

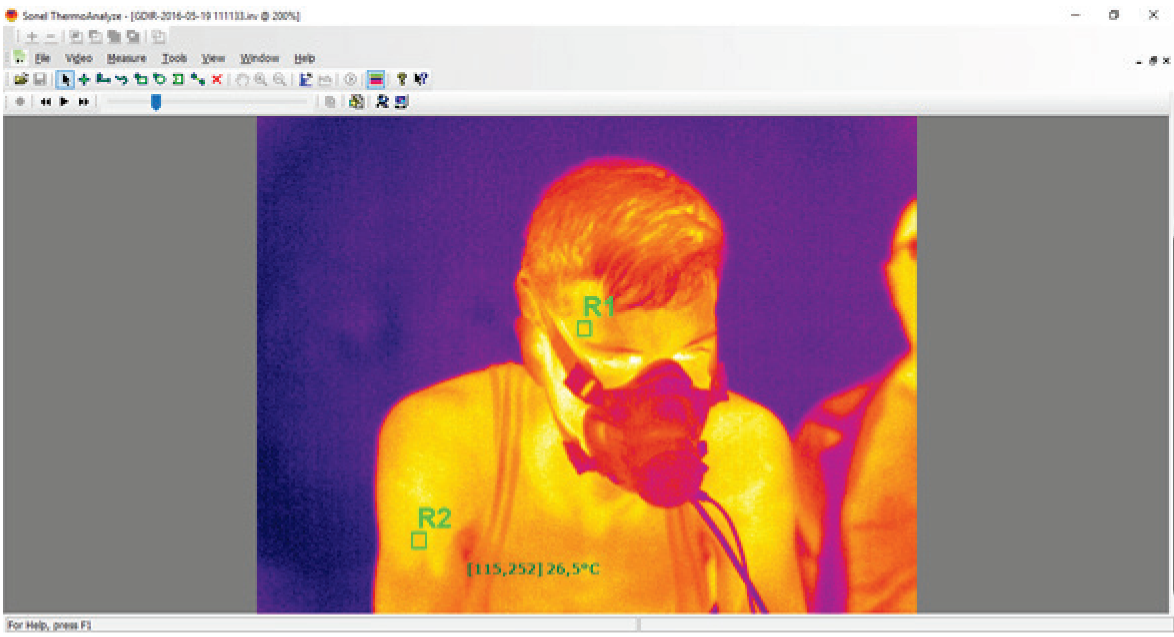

Fig. 1. A single frame of a film recorded during SITP with marked fields for the temple surface temperature (R1) and arm surface temperature (R2) analysis.

Experimental design

The participants were instructed to refrain from any physical exercise 48 hours prior to the experiment. First the participants performed an incremental testing protocol (ITP) to determine aerobic capacity and then 48 h later a sprint interval testing protocol (SITP). Testing was performed in controlled laboratory conditions (ambient temperature 20.5 °C at 55-60 % humidity) in the Exercise Laboratory of the University School of Physical Education (PN-EN ISO 9001:2001 certified).

Incremental testing protocol

Body composition was determined by ultrasound prior to testing using a Bodymetrix analyzer (Intelametrix, Brentwood, USA). Body mass (BM) was recorded in kg and body fat percentage (BF %) was determined at nine measurements sites (biceps, triceps, chest, scapula, lower back, waist, hip, thigh, and calf). Lean body mass (LBM) in kg was then calculated using the formula LBM = BM - (BM - %BF). The ITP was performed on a Lode Excalibur Sport cycle ergometer (Lode B.V. Groningen, Netherlands) calibrated before each trial. Starting workload was set at 50 W and increased by 50 W every 3 min until volitional exhaustion was reached. If the participant was unable to complete an entire 3 min stage, 0.28 W was subtracted for each missing second from the work rate at that stage. The highest power output determined in the ITP was recorded as maximal aerobic power (P_{max}).

Respiratory function was measured throughout the test by a Quark gas analyzer (Cosmed, Milan, Italy). The gas analyzer was calibrated before each trial with a reference gas mixture of carbon dioxide (5 %), oxygen (16 %), and nitrogen (79 %). The participant wore a face mask and tidal air was analyzed on a breath-by-breath basis to determine oxygen uptake (VO_2), carbon dioxide excretion (VCO_2), and minute pulmonary ventilation (VE). Maximal oxygen uptake (VO_{2max}) was calculated based on the composition of expired air and minute ventilation. All measures were averaged over 30-s intervals. Maximal oxygen uptake and maximal aerobic power were calculated relative to body mass (VO_{2max} and P_{max}, respeticely) and lean body mass (LBM-VO_{2max} and LBM-P_{max}, respectively).

Achievement of VO_{2max} was verified based on the incidence of the plateau phase in VO_{2max} (Dempsey and Wagner 1999). The plateau response was determined using previously established methods in which VO_2 was averaged over 15-s intervals and designated as the period when VO_2 did not fluctuate $\leq$1.5 ml.kg^{-1}.min^{-1} from the VO_{2max} (Doherty *et al.* 2003, Lucia *et al.* 2006). Plateau VO_{2max} duration was calculated in seconds ($T_{plateau}$) and is presented in Table 1.

Sprint interval testing protocol

The SITP was performed on the same Lode Excalibur Sport cycle ergometer. The test was preceded by a warm-up begun at a workload corresponding to

40 % Pmax (determined in ITP) for 5 min and then at 50 % P_{max} for 15 min. The participant then performed an active cool-down at 10 % P_{max} for 10 min followed by a 5-min rest. The test proper involved a set of four 30-s maximal cycling sprints with the participant motivated to attain peak power as quickly as possible and maintain maximum cycling cadence. Workload was individually determined at a fixed-mean crank torque of 0.8 N·m per kg of BM. Each repetition was interspersed with 90 seconds of active recovery in which workload was decreased to 30 W. Upon concluding the last repetition, the participant performed an active cool-down at 30 W for 2 min.

Power output was recorded in each sprint repetition to determine the highest absolute measure of peak power in the set (P_{peak}) and average power for the entire set (P_{av}). Respiratory function was evaluated using the same procedures and equipment as in the ITP with data collected from the beginning of the first sprint until 2 min after set termination. Total oxygen uptake (VO_{2tot}) was calculated as the sum of oxygen uptake across this interval and peak oxygen uptake (VO_{2peak}) was determined as the highest absolute measure of VO_2 (Hebisz *et al.* 2017). The power and oxygen measures were then calculated in relation to body mass (P_{av}, P_{peak}, VO_{2tot}, VO_{2peak}) and lean body mass (LBM-P_{av}, LBM-P_{peak}, LBM-VO_{2tot}, LBM-VO_{2peak}).

Body surface temperature during the SITP was measured using a Sonel KT384 thermal imaging camera (Sonel S.A., Świdnica, Poland). The camera has a 384x288 infrared resolution at a spectral range of 8-14 µm and thermal sensitivity of 0.08 °C. Data was processed using prepackaged software (Sonel ThermoAnalyzer) to extrapolate skin temperature into °C. During playback, single frames were analyzed and the average temperature across 10-pixel square targets on the temple and arm were recorded (Fig. 1). The temple site was chosen due to the superficial network of blood vessels local to the temporal artery (Marano *et al.* 1985) whereas the forearm measurement site served as a comparative measurement. Temperature was measured while stationary after the warm-up as a baseline immediately prior to SITP (T_b), immediately after the first sprint (T_1), 80 seconds after the first sprint (T_2), immediately after the fourth sprint (T_3), and 80 seconds after the fourth sprint (T_4).

Table 2. Temple and arm temperature in subsequent measurements during sprint interval testing protocol.

	T_b[°C]	T_1 [°C]	T_2 [°C]	T_3 [°C]	T_4 [°C]
Temple	30.9 (2.8)	30.3 (2.9)	30.1 (2.7)	31.2 (3.1)	32.1 (2.9)^{*T2}
Arm	29.4 (3.5)	29.7 (3.6)	30 (3.8)	30.9 (4.5)	31.5 (4.4)

T_b- the baseline temperature of the body surface, T_1 - body surface temperature immediately after the first sprint; T_2 - body surface temperature 80 seconds after the first sprint; T_3 - body surface temperature immediately after the fourth sprint; T_4 - body surface temperature 80 seconds after the fourth sprint; ^{*T2} - P<0.05 significant difference between T_4 vs. T_2; data are presented as arithmetic mean (standard deviation)

Table 3. Power output and oxygen uptake values.

Incremental testing protocol		**Sprint interval testing protocol**	
P_{max} [$W \cdot kg^{-1}$]	5.6 (0.3)	P_{peak} [$W \cdot kg^{-1}$]	19.9 (3.0)
LBM-P_{max} [$W \cdot kg^{-1}$]	6.6 (0.5)	LBM-P_{peak} [$W \cdot kg^{-1}$]	23.1 (3.4)
VO_{2max} [$ml \cdot min^{-1} \cdot kg^{-1}$]	66.3 (4.6)	P_{av} [$W \cdot kg^{-1}$]	9.4 (0.5)
LBM-VO_{2max} [$ml \cdot min^{-1} \cdot kg^{-1}$]	76.6 (5.5)	LBM-P_{av} [$W \cdot kg^{-1}$]	10.9 (0.7)
		VO_{2peak} [$ml \cdot min^{-1} \cdot kg^{-1}$]	53.3 (4.1)
		LBM-VO_{2peak} [$ml \cdot min^{-1} \cdot kg^{-1}$]	69.0 (7.0)
		VO_{2tot} [$ml \cdot kg^{-1}$]	315.6 (20.8)
		LBM-VO_{2tot} [$ml \cdot kg^{-1}$]	366 (33.5)

P_{max} - maximal aerobic power; VO_{2max} - maximal oxygen uptake; P_{peak} - peak power; P_{av} - average power; VO_{2peak} - peak oxygen uptake; VO_{2tot} - sum of total oxygen uptake; LBM - relative to lean body mass; data are presented as arithmetic mean (standard deviation)

The measurement of temperature during actual sprint execution was not possible as the participants stood on the pedals and introduced significant motion blur. The delta difference between the temperature measures and baselines was then calculated separately for the temple (ΔTt_1, ΔTt_2, ΔTt_3, ΔTt_4) and arm (ΔTa_1 ΔTa_2, ΔTa_3, ΔTa_4).

Statistical analysis

Data were processed using the Statistica 13.1 software package (Statsoft, USA). The distribution of the data set was checked using the Shapiro-Wilk test with normal distributions found for all measures except ΔTa_2. Means and standard deviations were calculated and one-

Table 4. Correlations between selected power output and oxygen uptake variables and the difference in temple surface temperature measured during sprint interval testing protocol.

	ΔTt_1 [°C]	**ΔTt_2 [°C]**	**ΔTt_3 [°C]**	**ΔTt_4 [°C]**
Incremental testing protocol				
P_{max} [$W \cdot kg^{-1}$]	0.13	0.16	0.41	0.47*
LBM-P_{max} [$W \cdot kg^{-1}$]	0.16	0.39	0.55*	0.63*
VO_{2max} [$ml \cdot min^{-1} \cdot kg^{-1}$]	0.27	0.28	0.47*	0.52*
LBM-VO_{2max} [$ml \cdot min^{-1} \cdot kg^{-1}$]	0.4	0.54*	0.72*	0.75*
Sprint interval testing protocol				
P_{peak} [$W \cdot kg^{-1}$]	-0.16	-0.15	-0.28	-0.28
LBM-P_{peak} [$W \cdot kg^{-1}$]	-0.07	0.00	-0.09	-0.1
P_{av} [$W \cdot kg^{-1}$]	-0.24	-0.15	-0.03	-0.06
LBM-P_{av} [$W \cdot kg^{-1}$]	0.07	0.19	0.43	0.37
VO_{2peak} [$ml \cdot min^{-1} \cdot kg^{-1}$]	-0.08	-0.04	0.56*	0.51*
LBM-VO_{2peak} [$ml \cdot min^{-1} \cdot kg^{-1}$]	0.08	0.17	0.66*	0.61*
VO_{2tot} [$ml \cdot kg^{-1}$]	-0.23	-0.18	0.34	0.26
LBM-VO_{2tot} [$ml \cdot kg^{-1}$]	0.00	0.1	0.52*	0.45*

ΔTt_1 - difference between the temple temperature measured immediately after the first sprint and the baseline temperature, ΔTt_2 - difference between the temple temperature measured 80 seconds after the first sprint and the baseline temperature; ΔTt_3 - difference between the temple temperature measured immediately after the fourth sprint and the baseline temperature; ΔTt_4 - difference between the temple temperature measured 80 seconds after the fourth sprint and the baseline temperature; LBM - parameter converted into lean body mass; P_{max} - maximal aerobic power; VO_{2max} - maximal oxygen uptake; P_{peak} - peak power; P_{av} - average power; VO_{2peak} - peak oxygen uptake; VO_{2tot} - the sum of oxygen uptake; * - P<0.05

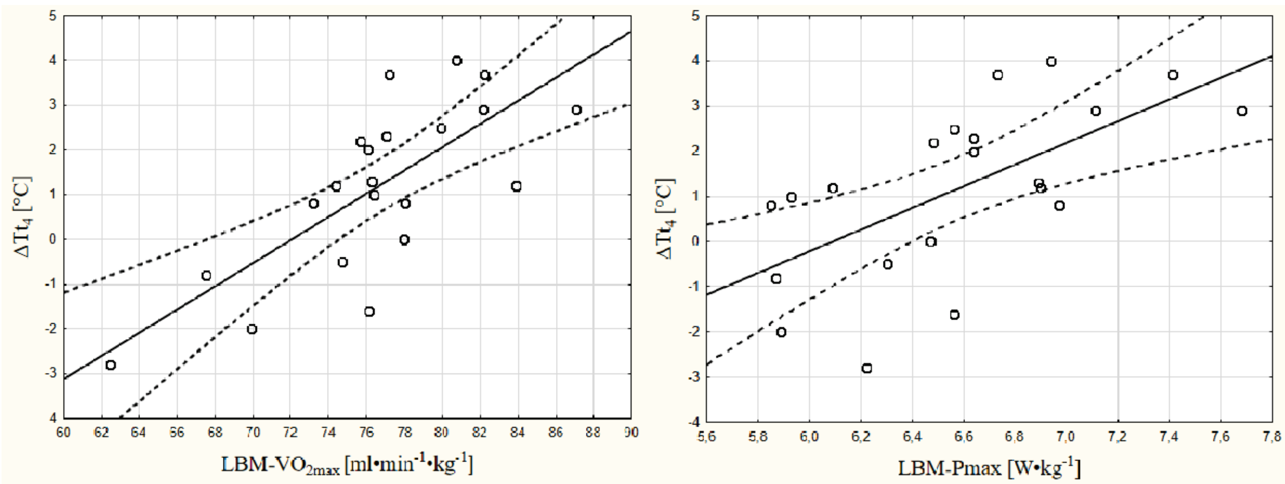

Fig. 2. Plots showing the correlation between aerobic capacity, after taking the lean body mass into account, and the change in the temple surface temperature 80 seconds after the fourth sprint in relation to the baseline temperature. The aerobic capacity is represented by maximal oxygen uptake (left plot) and maximal aerobic power (right plot).

way analysis of variance with the Duncan test administered post hoc was used to compare the differences for statistical significance. Pearson's correlation coefficients were calculated between the power and oxygen uptake measures in the ITP and SITP and ΔTt_1, ΔTa_1, ΔTt_2, ΔTa_2, ΔTt_3, ΔTa_3, ΔTt_4, and ΔTa_4. Linear regression analyses were performed for the variables that showed the strongest correlations. The significance level for all statistical procedures was set at $p<0.05$.

Results

Temple temperature significantly increased 80 seconds after the fourth sprint (Tt_4) in comparison with temperature 80 seconds after the first sprint (Tt_2). There were no significant changes in arm temperature (Table 2). The values of power output and oxygen uptake in the ITP and SITP as absolute measures and relative to lean body mass are presented in Table 3. The difference between the temple temperature measured 80 seconds after the first sprint and baseline (ΔTt_2) correlated positively only with relative maximal oxygen uptake measured in the ITP (LBM-VO_{2max}). The differences between temple temperature measured immediately after the fourth sprint (ΔTt_3) and 80 seconds after the fourth sprint (ΔTt_4) and baseline correlated positively with P_{max}, LBM-P_{max}, VO_{2max} and LBM-VO_{2max} obtained in ITP (Table 4 and Fig. 2) as well as VO_{2peak}, LBM-VO_{2peak}, and LBM-VO_{2tot} obtained in the SITP (Table 4). Regression equations were determined for LBM-P_{max} (LBM-P_{max} = $6.38+0.17 \cdot \Delta Tt_4$), LBM-$VO_{2max}$ (LBM-VO_{2max}=$74.08 + 2.15 \cdot \Delta Tt_4$), and LBM-$VO_{2peak}$ (LBM-VO_{2peak} = $68.46 + 2.52 \cdot \Delta Tt_3$). There were no statistically significant correlations between the arm temperature changes and oxygen uptake and power variables in either the ITP or SITP.

Discussion

The results obtained in the present study did not show any significant correlations between the delta change in body surface temperature measured immediately after the first sprint compared with baseline (ΔTt_1) were found with any of the measures of aerobic capacity. The lack of a correlation may be related to the relatively low stroke volume induced at this phase of the SITP due to delayed cardiac output kinetics in response to high-intensity exercise (Davies *et al.* 1972, Zakynthinaki 2015). In addition, this finding may also be explained by the delayed increase in cutaneous blood circulation at the onset of exercise (Johnson 1992, Simmons *et al.* 2011). The growing demand for oxygen by exercising skeletal muscle at the beginning of such high-intensity efforts induces the secretion of norepinephrine stimulating the post-synaptic receptors $alpha_1$ and $alpha_2$ to cause strong vasoconstriction of the cutaneous blood vessels (Johnson 1992, Johnson 1998).

Further comparisons in temple temperature showed significant correlations with both the ITP-obtained measures of aerobic capacity and SITP-based aerobic metabolism. Particularly strong correlations were obtained when comparing the delta temperatures with VO_{2max} relative to lean body mass (LBM-VO_{2max}), which may be explained by the propensity of individuals with high aerobic capacity to show enhanced metabolic function and thermoregulatory adaptations. Thermal output by contracting skeletal muscle is dependent on the metabolic response to exercise and the involvement of the aerobic and anaerobic energy pathways (Gonzalez-Alonso 2012). Sprint interval training is a particularly high energy demanding activity that severely taxes both pathways as evidenced by high levels of oxygen uptake and post-exercise lactate concentrations (Buchheit *et al.* 2012, Hebisz *et al.* 2017). The dissipation of heat generated by such intensive exercise is then modulated by several physiological mechanisms including stroke volume (Johnson 1998), blood volume (Morimoto 1990, Warburton *et al.* 1999), and the convective transport of heat via blood flow (Gonzalez-Alonso 2012). The above mechanisms are also strongly interrelated with VO_{2max} (Gledhill *et al.* 1992, Martino *et al.* 2002, Mujika and Padilla 2001b, Mujika and Padilla 2000, O'Neill *et al.* 2016), which further explains the correlations that were observed with post-exercise changes in temple surface temperature. The present results are also in concurrence with the literature on the high level of cutaneous blood flow during exercise in trained individuals (Fritzsche and Coyle 2000, Johnson 1998, Tankersley *et al.* 1991) and higher rectal temperature of individuals with high VO_{2max} during exercise in a warm environment (Mora-Rodriguez 2010).

The strongest correlation was observed between the delta change in temple temperature and VO_{2max} relative to lean body mass (LBM-VO_{2max}). This may be explained by the significant redistribution of blood flow in response to the heat generated by working skeletal muscle (Johnson 1992). This results in a significant increase in external carotid artery blood flow with a concomitant decrease in internal carotid artery and vertebral artery blood flow (Ogoh *et al.* 2013). Wilson *et al.* (2006) observed markedly increased cerebral vascular resistance and reduced blood velocity in response to thermal stress. As a result, blood distribution is modified with increased extracranial blood supply to enhance the dissipation of heat via cutaneous circulation (Ogoh *et al.* 2013). The above adaptations are observed with a concurrent reduction in cutaneous vascular resistance due to the decrease in adrenergic innervation and, as exercise continues, increased non-adrenergic vasodilatation (Hogan 2009, Johnson 1998). Johnson (1998) had previously suggested that cutaneous blood circulation is regulated by an internal temperature threshold that modulates blood flow and the body's sensitivity to internal temperature changes particularly when exercise is performed. These mechanisms best explain the increase in temple surface temperature post-SITP in comparison with the temperature after the first sprint and the strong correlation that was observed between VO_{2max} and the changes in the temperature upon concluding the SITP. This finding suggests that VO_{2max} may be predicted via changes in temple temperature during maximal sprint exercise as the higher the maximal oxygen uptake, the greater increase in skin surface temperature in order to enhance heat dissipation during exercise.

A strong correlation was also found between the delta change in temple temperature and VO_{2peak} relative to lean body mass (LBM-VO_{2peak}). This result suggests that temple temperature can also serve as an indicator of aerobic metabolic rate during repeated sprint exercise. However, in contrast to the results of the ITP, no statistically significant relationship was observed between the delta change in temple temperature and P_{peak} or P_{av} in the SITP. The lack of any correlation may reside in the fact that repeated sprinting performance is determined to a larger extent by the anaerobic metabolism of phosphocreatine and glucose and the accumulation of metabolites in muscles than an incremental exercise test (Girard *et al.* 2011). Therefore, it appears that the role of cardiovascular fitness in the delivery of oxygen to working muscle during repeated sprint exercise is less important than when performing incremental exercise (Buchheit *et al.* 2012, Girard *et al.* 2011, Hebisz *et al.* 2017).

Based on the present results, it can be concluded that body surface temperature during and after a single bout of sprint interval exercise can serve as an indirect method for predicting aerobic capacity, as higher VO_{2max} is associated with a greater increase in temple surface temperature during such intensive exercise. Presently, the strongest correlations were observed between VO_{2max} and the delta change in temple temperature immediately and 80 seconds after the fourth (and last) sprint repetition when compared with baseline values. Future research is needed to confirm the changes in body surface temperature in a wider range of measurement sites during and after additional exercise modalities in populations differentiated by VO_{2max}, age, and physical activity level.

Conflict of Interest

There is no conflict of interest.

Acknowledgements

This work was supported by the Ministry of Science and Higher Education under grant number NRSA300253 titled "Development of Academic Sport". The authors wish to thank Michael Antkowiak for his translation and assistance in preparation of this manuscript.

References

ABDERRAHMAN AB, PRIOUX J, CHAMARI K, OUNIS OB, TABKA Z, ZOUHAL H: Running interval training and estimated plasma-volume variation. *Int J Sports Physiol Perform* **8**: 358-365, 2013.

BUCHHEIT M, ABBISS CR, PEIFFER JJ, LAURSEN PB: Performance and physiological responses during a sprint interval training session: relationships with muscle oxygenation and pulmonary oxygen uptake kinetics. *Eur J Appl Physiol* **112**: 767-779, 2012.

COLES MG, LUETKEMEIER MJ: Sodium-facilitated hypervolemia, endurance performance and thermoregulation. *Int J Sports Med* **26**: 182-187, 2005.

COQUART JB, GARCIN M, PARFITT G, TOURNY-CHOLLET C, ESTON RG: Prediction of maximal or peak oxygen uptake from ratings of perceived exertion. *Sports Med* **44**: 563-578, 2014.

DAVIES CTM, DI PRAMPERO PE, CERRETELLI P: Kinetics of cardiac output and respiratory gas exchange during exercise and recovery. *J Appl Physiol* **32**: 618-625, 1972.

DE LORENZO A, DA SILVA CL, CASTRO SOUZA FC, DE SOUZA LEÃO LIMA R: Value of the oxygen pulse curve for the diagnosis of coronary artery disease. *Physiol Res* **67**: 679-686, 2018.

DEMPSEY JA, WAGNER PD: Exercise-induced arterial hypoxemia. *J Appl Physiol* **87**: 1997-2006, 1999.

DOHERTY M, NOBBS L, NOAKES T: Low frequency of the "plateau phenomenon" during maximal exercise in elite British athletes. *Eur J Appl Physiol* **89**: 619-623, 2003.

FARIA EW, PARKER DL, FARIA IE: The science of cycling: physiology and training - part 1. *Sports Med* **35**: 285-312, 2005.

FRITZSCHE RG, COYLE EF: Cutaneous blood flow during exercise is higher in endurance-trained humans. *J Appl Physiol* **88**: 738-744, 2000.

GIRARD O, MENDEZ-VILLANUEVA A, BISHOP D: Repeated-sprint ability - Part I. Factors contributing to fatigue. *Sports Med* **41**: 673-694, 2011.

GLAISTER M: Multiple sprint work. Physiological responses, mechanisms of fatigue, and the influence of aerobic fitness. *Sports Med* **35**: 757-777, 2005.

GLEDHILL N, WARBURTON D, JAMNIK V: Hemoglobin, blood volume, cardiac function, and aerobic power. *Can J Appl Physiol* **24**: 54-65, 1992.

GONZALEZ-ALONSO J: Human thermoregulation and the cardiovascular system. *Exp Physiol* **97**: 340-346, 2012.

HEBISZ R, HEBISZ P, ZATOŃ M, MICHALIK K: Peak oxygen uptake in a sprint interval testing protocol vs. maximal oxygen uptake in an incremental testing protocol and their relationship with cross-country mountain biking performance. *Appl Physiol Nutr Metab* **42**: 371-376, 2017.

HOGAN TS: Exercise-induced reduction in systemic vascular resistance: a covert killer and an unrecognised resuscitation challenge? *Med Hypotheses* **73**: 479-484, 2009.

JOHNSON JM: Exercise and the cutaneous circulation. *Exerc Sport Sci Rev* **20**: 59-97, 1992.

JOHNSON JM: Physical training and the control of skin blood flow. *Med Sci Sports Exerc* **30**: 382-386, 1998.

JOHNSON JM, ROWELL LB: Forearm skin and muscle vascular responses to prolonged leg exercise in man. *J Appl Physiol* **39**: 920-924, 1975.

KAMON E, BELDING HS: Dermal blood flow in the resting arm during prolonged leg exercise. *J Appl Physiol* **26**: 317-320, 1969.

KENNEY WL, JOHNSON JM: Control of skin blood flow during exercise. *Med Sci Sports Exerc* **24**: 303-312, 1992.

LEVINE BD: VO_2max: what do we know, and what do we still need to know? *J Physiol* **586**: 25-34, 2008.

LOPES AJ, VIGÁRIO PS, HORA AL, DEUS CAL, SOARES MS, GUIMARÃES FS, FERREIRA AS: Ventilation distribution, pulmonary diffusion and peripheral muscle endurance as determinants of exercise intolerance in elderly patients with chronic obstructive pulmonary disease. *Physiol Res* **67**: 863-874, 2018.

LORENZO S, HALLIWILL JR, SAWKA MN, MINSON CT: Heat acclimation improves exercise performance. *J Appl Physiol* **109**: 1140-1147, 2010.

LUCIA A, HOYOS J, CHICHARRO JL: Physiology of professional road cycling. *Sports Med* **31**: 325-337, 2001.

LUCIA A, RABADAN M, HOYOS J, HERNANDEZ-CAPILLA M, PEREZ M, SAN JAUN AF, EARNEST CF, CHICHARRO JL: Frequency of the VO2max plateau phenomenon in world class cyclists. *Int J Sports Med* **27**: 984-992, 2006.

MARANO SR, FISCHER DW, GAINES C, SONNTAG VK: Anatomical study of the superficial temporal artery. *Neurosurgery* **16**: 786-790, 1985.

MARTINO M, GLEDHILL N, JAMNIK V: High VO2max with no history of training is primarily due to high blood volume. *Med Sci Sports Exerc* **34**: 966-971, 2002.

MORA-RODRIGUEZ R, DEL COSO J, HAMOUTI N, ESTEVEZ E, ORTEGA JF: 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* **109**: 973-981, 2010.

MORIMOTO T: Thermoregulation and body fluids: role of blood volume and central venous pressure. *Jpn J Physiol* **40**: 165-179, 1990.

MUJIKA I, PADILLA S: Detraining: loss of training-induced physiological and performance adaptations. Part I: short term insufficient training stimulus. *Sports Med* **30**: 79-87, 2000.

MUJIKA I, PADILLA S: Physiological and performance characteristics of male professional road cyclists. *Sports Med* **31**: 479-487, 2001a.

MUJIKA I, PADILLA S: Muscular characteristics of detraining in humans. *Med Sci Sports Exerc* **33**: 1297-1303, 2001b.

NELSON MD, STUART-HILL LA, SLEIVERT GG: Hypervolemia and blood alkalinity: effect on physiological strain in a warm environment. *Int J Sports Physiol Perfor* **3**: 501-515, 2008.

NYBO L, NIELSEN B: Hyperthermia and central fatigue during prolonged exercise in humans. *J Appl Physiol* **91**: 1055-1060, 2001.

OGOH S, SATO K, OKAZAKI K, MIYAMOTO T, HIRASAWA A, MORIMOTO K, SHIBASAKI M: Blood flow distribution during heat stress: cerebral and systemic blood flow. *J Cereb Blood Flow Metab* **33**: 1915-1920, 2013.

O'NEILL CD, KIMMERLY DS, DOGRA S: Central and peripheral response to incremental cycling exercise in older untrained active men: A comparison of those in-between. *Physiol Res* **65**: 303-309, 2016.

RACINAIS S, MOUSSAY S, NICHOLS D, TRAVERS G, BELFEKIH T, SCHUMACHER YO, PERIARD JD: Core temperature up to 41.5ºC during the UCI Road Cycling World Championships in the heat. *Br J Sports Med* **53**: 426-429, 2019.

SIMMONS GH, PADILLA J, YOUNG CN, WONG BJ, LANG JA, DAVIS MJ, LAUGHLIN MH, FADEL PJ: Increased brachial artery retrograde shear rate at exercise onset is abolished during prolonged cycling: role of thermoregulatory vasodilation. *J Appl Physiol* **110**: 389-397, 2011.

TANKERSLEY CG, SMOLANDER J, KENNEY WL, FORTNEY SM: Sweating and skin blood flow during exercise: effects of age and maximal oxygen uptake. *J Appl Physiol* **71**: 236-242, 1991.

TAYLOR WF, JOHNSON JM, KOSIBA WA: Roles of absolute and relative load in skin vasoconstrictor responses to exercise. *J Appl Physiol* **69**: 1131-1136, 1990.

VIGELSØ A, PRATS C, PLOUG T, DELA F, HELGE JW: Higher muscle content of perilipin 5 and endothelial lipase protein in trained than untrained middle-aged men. *Physiol Res* **65**: 293-302, 2016.

WARBURTON DE, GLEDHILL N, JAMNIK VK, KRIP B, CARD N: Induced hypervolemia, cardiac function, VO2max, and performance of elite cyclists. *Med Sci Sports Exerc* **31**: 800-808, 1999.

WARBURTON DE, HAYKOWSKY MJ, QUINNEY HA, BLACKMORE D, TEO KK, TAYLOR DA: Blood volume expansion and cardiorespiratory function: effects of training modality. *Med Sci Sport Exerc* **36**: 991-1000, 2004.

WILSON TE, CUI J, ZHANG R, CRANDALL CG: Heat stress reduces cerebral blood velocity and markedly impairs orthostatic tolerance in humans. *Am J Physiol Regul Integr Comp Physiol* **291**: 1443-1448, 2006.

WINGO JE, LOW DA, KELLER DM, BROTHERS RM, SHIBASAKI M, CRANDALL CG: Skin blood flow and local temperature independently modify sweat rate during passive heat stress in humans. *J Appl Physiol* **109**: 1301-1306, 2010.

YOSHIDA T, NAGASHIMA K, NOSE H, KAWABATA T, NAKAI S, YORIMOTO A, MORIMOTO T: Relationship between aerobic power, blood volume, and thermoregulatory responses to exercise-heat stress. *Med Sci Sports Exerc* **29**: 867-873, 1997.

ZAKYNTHINAKI MS: Modelling heart rate kinetics. *PLoS One* **10**: 1-26, 2015.