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Application of A Physiological Strain Index in Evaluating Responses to Exercise Stress – A Comparison Between Endurance and High Intensity Intermittent Trained Athletes

by
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The study evaluated differences in response to exercise stress between endurance and high-intensity intermittent trained athletes in a thermoneutral environment using a physiological strain index (PSI). Thirty-two subjects participated in a running exercise under normal (23°C, 50% RH) conditions. The group included nine endurance trained athletes (middle-distance runners - MD), twelve high-intensity intermittent trained athletes (soccer players - HIIT) and eleven students who constituted a control group. The exercise started at a speed of 4 km·h^{-1} which was increased every 3 min by 2 km·h^{-1} to volitional exhaustion. The heart rate was recorded with a heart rate monitor and aural canal temperature was measured using an aural canal temperature probe. The physiological strain index (PSI) and the contribution of the circulatory and thermal components to the overall physiological strain were calculated from the heart rate and aural canal temperature. The physiological strain index differed between the study and control participants, but not between the MD and HIIT groups. The physiological strain in response to exercise stress in a thermoneutral environment was mainly determined based on the circulatory strain (MD group - 73%, HIIT group – 70%). The contribution of the circulatory and thermal components to the physiological strain did not differ significantly between the trained groups (MD and HIIT) despite important differences in morphological characteristics and training-induced systemic cardiovascular and thermoregulatory adaptations.

Key words: *physiological strain, exercise, adaptation, thermoneutral conditions, athletes.*

Introduction

Exercise is physiologically demanding due to the metabolic heat strain accompanying muscular activity. In order to maintain tolerable body temperature during exercise, the metabolic heat load must be balanced by an equal transfer of heat from the body to the environment. Exercise heat production coupled with insufficient heat loss results in an increase in physiological strain (including the thermoregulatory and cardiovasculatory system). Numerous indices have been proposed to determine physiological strain depending on the organism itself and environmental conditions. These indices can be divided into two main categories, ie., empirical and rational (Epstein and Moran, 2006). Moran et al. (1998a) developed a rational index referred to as the Physiological Strain Index (PSI) based upon heart rate and body temperature measurements, allowing the instantaneous assessment of overall physiological strain on a scale of 0-10. This index reflects the combined load on the thermoregulatory and cardiovascular systems. Thermoregulatory strain depends on the efficiency of heat dissipation mechanisms and metabolic heat production whereas the heart rate (HR) corresponds to demands placed on the circulatory system. Predictions of physiological strain are important in determining physiological endurance and protecting athletes against thermal stressors.

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The strain of the physiological mechanisms during exercise is influenced by many factors such as morphological including body mass (Anderson, 1999; Cheung et al., 2000; Havenith et al., 1998; Havenith, 2001) and body composition (Jay and Kenney, 2007), age (Moran et al., 2002; Pandolf, 1997), gender (Moran et al., 1999), body hydration (Merry et al., 2010; Moran et al., 1998b; Maughan and Shireffs, 2010), aerobic capacity (Merry et al., 2010; Mora-Rodrigez et al., 2010; Tikuisis et al., 2002), heat acclimation (Aoyagi et al., 1997; Kondo et al., 2009; Nadel et al., 1974; Periard et al., 2015), a type of exercise, environmental conditions and protective clothing (Borg et al., 2015; Epstein and Moran, 2006; Gonzalez-Alonso, 2012; Pilch et al., 2014; Pokora et al., 2014).

Following training, the cardiovascular and thermoregulatory systems along with their components go through various adaptive changes. The nature of these systemic and organ adaptations is highly specific to a particular sport with respect to the type of conditioning and apparent stimuli to which the athlete is subjected (Dudley and Djamil, 1985).

Typically, regular endurance-training programs use low-resistance, high-repetition aerobic exercises to stimulate physiological and psychological adaptations that enable athletes to better tolerate exercise stress and result in significant improvements in maximal oxygen uptake (Bessett and Howley, 2000; Pollock, 1977), thermoregulatory capacity (Bessett and Howley, 2000; Smorawiński and Grucza, 1994) and cardiorespiratory endurance (Fritzsche and Coyle, 2000; Smorawinski and Grucza, 1994). Elite endurance athletes typically record high VO_{2max} values, primarily due to a high Q (cardiac output), blood volume expansion (Convertino, 1991). Sports such as soccer are examples of combined endurance and speed exercise and have been classified as high-intensity intermittent exercise team sports (Bansbo, 1994) due to their acyclical nature and high intensity. Team sports require athletes to combine maximal effort and low-intensity exercise. Maximal O_2 uptake of elite soccer players is similar to that found in other team sports, but substantially lower than that of elite endurance performers (Drust et al., 2000; Fowkes Godek et al., 2004, 2005; McMillan et al., 2005). In either case, physiological adaptations in response to physical training are highly specific to the nature of the training activity (Nelson et al., 1990) and differ in endurance and soccer-trained men.

We hypothesized that the type of training could significantly modify the character of the load placed on physiological mechanisms during exercise and influence the magnitude and contribution of thermal and cardiovascular strain to the overall physiological strain during exercise. We also suspected that the physiological strain in response to a running exercise test to exhaustion might be smaller in athletes practicing middle-distance running than those involved in high-intensity intermittent training.

Therefore, the purpose of this study was to examine whether the type of training-induced adaptation differentiates the magnitude and contribution of thermal and circulatory strain to the overall physiological strain induced by exercise in a thermoneutral environment.

Material and Methods

Subjects

A total of 32 healthy men participated in the study. Eleven of them were students of the Academy of Physical Education (a control group). The athletes group included: nine middle distance runners (MD) and twelve speed-endurance trained soccer players (HIIT). During the preliminary trial, total body mass, height, body fat (%) and maximal oxygen uptake were measured. Body surface area (BSA; m^2, DuBois and DuBois, 1916) and BSA/BM ($m^2 \cdot kg^{-1}$) were calculated for each subject.

The study subjects' physical characteristics are presented in Table 2**.**

Procedures

Prior to the onset of this study, the research methodology was approved by the Research Ethics Committee of the Academy of Physical Education in Katowice and was performed in accordance with the Declaration of Helsinki.

All exercise tests were performed in a laboratory. Laboratory conditions were determined using wet-bulb dry temperature (WBDT) calculated according to the formula of Moran et al. (2001). Upon arrival in the laboratory, the subjects were weighed (body composition was determined; Tanita BC-418, Korea) and provided

a urine sample for the assessment of their hydration status (euhydration state corresponds to urine specific gravity (<1.025 g/cm^3)). To assure proper hydration in the evening prior to each exercise session, the participants were instructed to refrain from alcohol and caffeine and to drink approximately 600 ml of water the night before the scheduled session.

All exercise sessions were conducted at the same time of day. The sessions involved a run-to-exhaustion performance test on a motor-driven treadmill (HP/Cosmos–Pulsar, Germany). During exercise (at a 1% treadmill grade), the running speed was set at 4 km·h^{-1} for all participants. Every 3 min the speed was increased by 2 km·h^{-1}. Exercise stopped when the participant reached volitional exhaustion or achieved an oxygen uptake plateau.

Physiological measures

Oxygen uptake (VO_2), minute ventilation (VE), a respiratory exchange ratio (RER) and a heart rate (HR) were measured at rest (baseline measurement) and in the last minute of exercise (endpoint measurement) using an open-circuit respiratory analyzer (Matalyzer 3B, Cortex, Germany).

The heart rate was monitored by a telemetric heart rate monitor (PE 3000E, Polar Electro, Finland). Before and after exercise, the subjects were weighed and body mass loss was calculated (ΔBM; kg, and %). Aural canal temperature (Tty) was measured with a ThermoScan thermometer (Type 6201, Braun, Germany). The thermometer was inserted fully into the aural canal and held in position for 5 s while the measurement took place. Sweat loss was estimated by change in body mass corrected for urine loss. The sweating rate was divided by total exercise time and BSA, (SR), [g· (m^2)$^{-1}$ · h^{-1}].

Calculations

The physiological strain index (PSI) was calculated according to the Moran et al.'s (1998a) equation. The PSI reflects combined cardiovascular and thermoregulatory strain on a universal scale of 0 to 10. The PSI was calculated as follows:

$$PSI = 5(Tty_T - Tty_o) \cdot (39.5 - Tty_o)^{-1} + 5(HR_T - HR_o) \cdot (180 - HR_o)^{-1}$$

where Tty_o and HR_o are the baseline aural canal temperature and heart rate measurements, whereas Tty_T and HR_T are the endpoint aural canal temperature and heart rate measurements.

The fractional cardiovascular system contribution to the physiological strain, *f*HR, was calculated according to Tikuisis et al. (2002) using the following formula:

$$fHR=[5/PSI] \cdot [(HR_T - HR_o)/(180 - HR_o)]$$

The baseline heart rate (HR_o) was obtained in the standing position after a rest of >30 min at a comfortable room temperature before the beginning of exercise.

The endpoint heart rate HR_T was taken at the maximal workload. Heart rate reserve, HRR, was calculated as the difference between HR_T and HR_o.

The fractional thermoregulatory system contribution to the physiological strain *f*Tty was calculated according to Tikuisis et al. (2002) using the following formula:

$$fTty=[5/PSI] \cdot [(Tty - Tty_o)/(39.5 - Tty_o)]$$

The metabolic rate (M) was assessed via open circuit spirometry using an automated gas analyser indirect calorimetry system. All metabolic variables are expressed in watts (W). The metabolic rate was calculated using a validated formula (Nishi, 1981).

Statistical analysis

The data are presented as means and SEM (x; SEM). One-way repeated measures ANOVA was used to examine group differences (groups MD, HIIT and C) in physiological variables and changes (Δ) of thermophysiological and cardiovascular responses to exercise. When significant main effect was found, a post-hoc Tukey's test was used. Pearson correlation coefficients were used to identify relationships between the PSI and HRR, VO_{2max} and ΔTty. For all statistical analyses $p<0.05$ was considered significant.

Results

Laboratory conditions determined as WBDT did not significantly differ between experimental sessions ($p>0.05$), average 23.17 (0.22)°C.

Participant characteristics

The study groups were heterogeneous. Significant differences were found between MD and HIIT participants regarding body height, body mass, body surface area and BSA/BM, fat free mass, BMI and VO_{2max} (ml· kg^{-1} ·min^{-1}). Both groups of trained athletes differed significantly

from the control group with respect to BF%, BMI and VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) (Tables 1, 2).

Exercise responses

Longer time to exercise termination, greater maximal running speed and VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) were observed in the endurance group compared to the HIIT and C participants. Time to exhaustion was significantly longer in the MD compared to the HIIT group. The maximal power output, expressed in relative ($W \cdot kg^{-1}$) and absolute (W) values, was significantly higher in athletes (Table 2).

There was no significant difference in baseline Tty_o ($p>0.05$) between the groups tested whereas baseline HR_o was significantly lower in the MD compared to the HIIT group (Table 3).

Aural canal temperature increased during exercise in all subjects, but did not reach the 39.5 ^oC limit in any of the groups. The mean Tty increased over time ($p<0.001$) but no group effect was observed ($F=2.59$; $p>0.05$).

An analysis of Tty changes (ΔTty ^oC) did not reveal significant differences between the study groups although the rate of aural canal temperature increase ($\Delta Tty \cdot min^{-1}$ ($\Delta^oC \cdot min^{-1}$) was significantly lower in the MD compared to HIIT and C groups ($p<0.01$) (Table 3).

Metabolic rate and Power output

The average levels of the metabolic rate (at rest and at the end of exercise) and maximal power are presented in Table 2.

The baseline metabolic rate M_O [$W \cdot (m^2)^{-1}$] and M_O [$W \cdot kg^{-1}$] were significantly higher in the HIIT compared to the control group while the endpoint net metabolic heat production (M_T), expressed as [$W \cdot (m^2)^{-1}$] and [$W \cdot kg^{-1}$], was highest in the MD group. There was no significant difference in maximal power output (W) expressed in absolute values and in relation to body mass [$W \cdot kg^{-1}$] between the HIIT and MD groups. Both groups exhibited significantly higher values of maximal power output when compared to the control group.

Table 1

Physical characteristics and peak exercise responses for each tested group (measured and calculated)

Variable	C n=11	MD n=9	HIIT n=12	*Effect of group*
Age (years)	21.40(0.79)	21.56()	23.15(0.60)	
Body height (cm)	176.85(1.86)	170.78(1.55) #	185.79(1.42) *	F=7.08 $p<0.05$
BM (kg)	76.30(2.22)	69.22(1.86) ##	80.75(1.65)	F=8.74 $p<0.005$
BSA (m^2)	1.94(0.04)	1.85(0.03) ##	2.02(0.03)	F=7.50 $p<0.005$
BSA/BM ($cm^2 \cdot kg^{-1}$)	2.55(0.03)	2.68(0.02) ##	2.52(0.02) ***	F=9.18 $p<0.005$
Body Fat (%)	13.06(0.92)	6.78(0.77) **	7.54(0.70) **	F=8.94 $p<0.005$
FFM (kg)	66.03(2.14)	62.72(1.64) ##	72.53(1.79)	F=8.86 $p<0.005$
BMI ($kg \cdot (m^2)^{-1}$)	24.36(0.44)	21.63(0.37) *#	23.28(0.34) *	F=9.79 $p<0.005$
VO_{2max} ($l \cdot mim^{-1}$)	3.77(0.12)	4.36(0.14)	4.41(0.12)	F=3.61 $p=0.052$
HR_T ($beats \cdot min^{-1}$)	192.91(2.17)	193.77(2.38)	192.92(2.09)	F=0.20 $p>0.05$

Significantly different from control (p<0.05), ** (p<0.01); * (p<0.005)*
 # Significant difference between endurance and high –intermittent intensity trained athletes (p<0.05; ## p<0.01); ### (p<0.005)
Values are means (SEM). BM - Body mass; BSA - Body surface area; BMI - Body mass index; VO_{2max} - Maximal oxygen uptake; HR_T - Endpoint heart rate;
FFM (Fat Free Mass in kg) = nude BM - [nude BM x % body fat];
C - control group; MD - endurance trained athletes;
HIIT - high –intermittent intensity trained athletes

Table 2

Exercise responses (maximal power output, maximal velocity and time of exercise) for each tested group

Variable	C n=11	MD n=9	HIIT n=12	*Effect of group*
VO_{2max} (ml·kg^{-1}· min^{-1})	48.51(1.19)	63.06(1.36) **##	54.76(1.19) **	F=17.43 p<0.005
V_T (km·h^{-1})	14.07(0.6)	21.25(0.4) *#	18.0(0.4) *	F=79.47 p<0.005
W_{max} (Watts)	303(15)	392(11) **	385(10) **	F=19.54 p<0.005
W_{max}/BM (Watts·kg^{-1})	3.90(0.11)	5.68(0.12) **	4.79(0.11)**	F=79.47 p<0.005
Time of exercise (min)	22.91(0.62)	25.88(0.71) #	21.00(0.64)	F=10.82 p<0.005

Significantly different from control (p<0.05); ** (p<0.01); * (p<0.005)*
 # Significant difference between endurance and high –intermittent intensity trained athletes (p<0.05); ## (p<0.01); ### (p<0.005)
Values are means (SEM). W - power output (Watts); V_T - maximal velocity; W_{max} - maximal power output; C - control group; MD - endurance trained athletes; HIIT - high –intermittent intensity trained athletes

Table 3

Physiological (cardiac, thermal and metabolic) responses to exercise for each tested group

Characteristics	C n=11	MD n=9	HIIT n=12	*Effect of group*
HR_O (beats·min^{-1})	72.78(2.89)	66.22(3.17) #	79.83(2.78)	F=4.46 p<0.05
HR_T (beats·min^{-1})	192.91(2.17)	193.77(2.38)	192.92(2.09)	F=0.20 p>0.05
HRR (beats · min^{-1})	120.13(2.98)	124.40(3.12)	113.08(2.92)	F=3.02 p>0.05
ΔHR/time of exercise (beats·min^{-1})	5.50(0.18)	5.00(0.19)	5.39(0.17)	F=2.03 p>0.05
Tty_o (^{o}C)	36.63(0.06)	36.51(0.06)	36.43(0.05)	F=3.15 p=0.057
Tty_T (^{o}C)	37.96(0.10)	37.54(0.12)	37.83(0.09)	F=1.39 p>0.05
ΔTty (^{o}C)	1.36(0.10)	1.11(0.09)	1.39(0.07)	F=2.59 p>0.05
ΔTty(Δ^{o}C ·min^{-1})	0.06(0.004)	0.04(0.004) **##	0.06(0.004)	F=9.73 p<0.005
M_0/BSA (Watts ·(m^2)$^{-1}$)	62.37(3.63)	68.90(3.24)	77.34(2.84) **	F=8.16 p<0.005
M_T/BSA (Watts ·(m^2)$^{-1}$)	400.06(20.38)	510.99(18.22) **#	443.44(15.99)	F=8.59 p<0.005
M_0/BM (Watts · kg^{-1})	1.38(0.08)	1.61(0.07)	1.70(0.06)*	F=7.36 p<0.005
M_T/BM (Watts · kg^{-1})	8.38(0.48)	9.85(0.42) #	8.33(0.38)	F=4.14 p<0.05
PSI	8.89(0.20)	7.34(0.22) *	8.14(0.20)	F=14.72 p<0.005
fHR	0.63(.017)	0.75(0.018) ***	0.70(0.015) *	F=10.62 p<0.005
fTty	0.34(0.02)	0.28(0.02)	0.35(0.02)	F=1.20 p>0.05

Significantly different from control (p<0.05); ** (p<0.01); * (p<0.005)*
 # Significant difference between endurance and high –intermittent intensity trained athletes (p<0.05); ## (p<0.01); ### (p<0.005)
M-metabolic rate (metabolic heat production ; in Watts); HR_o – resting heart rate; HR_T - endpoint heart rate; Tty_o - resting aural canal temperature; Tty_T – endpoint aural canal temperature, M_o- resting metabolic rate; $M_T = (M_{max}-M_{rest})-W_{max}$ - net maximal metabolic heat production; ΔTty – change in body temperature; ΔTty/time-the ratio of increased aural canal temperature (Δ^{o}C ·min^{-1}); PSI - physiological strain index-; ƒHR - cardiovascular fraction of the physiological strain; ƒTty - thermal fraction of the physiological strain; HRR - cardiac reserve; C - control group; MD - endurance trained athletes; HIIT - high –intermittent intensity trained athletes

Table 4

The sweating response to exercise for each tested group

Characteristics	C n=11	MD n=9	HIIT n=12	*Effect of group*
ΔBM (kg)	-0.65(0.09)	-0.87(0.09)	-0.61(0.10)	F=2.39 $p>0.05$
BM loss (%)	-0.86(0.12)	-1.24(0.12) #	-0.76(0.10)	F=4.31 $p<0.05$
ΔBM ($g \cdot h^{-1}$)	-1.77(0.22)	-2.03(0.24)	-1.73)0.21)	F=0.46 $p>0.05$
SR_L ($g \cdot (m^2)^{-1}$)	0.34(0.04)	0.45(0.04)	0.31(0.04)	F=3.47 $p=0.06$
SR ($g \cdot (m^2)^{-1} \cdot h^{-1}$)	0.92(0.10)	1.08(0.11)	0.86(0.11)	F=0.93 $p>0.05$
SR/ΔTty ($g \cdot {}^0C^{-1}$)	0.49(0.11)	0.82(0.05) #	0.46(0.07)	F=4.97 $p<0.05$

Significantly different from control (p<0.05); ** (p<0.01); * (p<0.005)*
 # Significant difference between endurance and high –intermittent intensity trained athletes (p<0.05); ## p<0.01); ### (p<0.005)
SR- sweating rate; SR/ΔTty - sweating sensitivity; SR_L - sweat loss;
C - control group; MD - endurance trained athletes;
HIIT - high –intermittent intensity trained athletes

Table 5

Correlations between selected variables (ΔTty (°C), HRR, VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) and the PSI

Relationships	C	MD	HIIT
PSI vs HRR	n.s.	r= -0.84 $p<0.005$	n.s.
PSI vs ΔTty (°C)	r= 0.74 $p<0.05$	n.s.	r=0.89 $p<0.005$
PSI vs VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)	n.s.	n.s.	n.s.

C - control group; MD - endurance trained athletes;
HIIT - high –intermittent intensity trained athletes;
ΔTty – change in body temperature;
PSI - physiological strain index; HRR - cardiac reserve;
VO_{2max} - maximal oxygen uptake

Physiological strain

Physiological responses and the respective PSIs in the control, endurance and high-intensity intermittent trained athletes are presented in Table 3.

The physiological strain induced by the exercise with progressively increased intensity was significantly lower in the MD group. PSI values increased in all tested groups, but reached significantly lower values in the MD than in the control. No significant PSI differences were found for the matched experimental model between the MD and HIIT groups. The PSI was primarily governed by the rise in the HR and less by the Tty in all tested groups.

There were no significant differences between groups regarding the contribution of the thermal fraction to the PSI (fTty) (Table 3).

The contribution of the cardiac fraction to the total physiological strain during exercise was significantly greater in the MD than in the control group (Table 3).

Prior to exercising, the heart rate (HR_0) was significantly lower in the MD than in the HIIT group ($p<0.05$). For all tested groups, the maximal heart rate measured during the last exercise workload was comparable (Table 3). Hence, the cardiac reserve (HRR) (calculated as HR_T-HR_0) of the MD group was significantly greater than in the HIIT group. There were no

significant differences in HRR between the C and HIIT groups (Table 3).

Sweating rate

There were no significant differences between groups regarding SR [$g \cdot (m^2)^{-1}$] and SR [$g \cdot (m^2)^{-1} \cdot h^{-1}$]. However, when normalized to body surface area, SR [$g \cdot (m^2)^{-1}$] was greater in the MD compared to control and HIIT groups (p=0.06), while the overall sweat rate per ΔTty (SR/ΔTty-sweating sensitivity; g/Δ°C) was higher in the MD group (Table 4).

The PSI was negatively and significantly correlated (r= -0.64; p<0.01) with VO_{2max} (in $ml \cdot kg^{-1} \cdot min^{-1}$) in all study subjects, but not in any of the study groups considered separately. The PSI was positively and significantly correlated (p<0.05) with changes in aural canal temperature (ΔTty °C) in control and HIIT groups, whereas it was negatively and significantly correlated with HRR in the MD group. Correlations between selected variables (ΔTty (°C), HRR and VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) and the PSI are presented in Table 5.

Discussion

Metabolic response to exercise

The change in the response of particular physiological variables to exercise-induced physiological strain in a thermoneutral environment may be determined based on the physiological responses to metabolic cost (of thermal load) associated with exercise as heat production arising from muscular activity is usually the main component of exercise heat stress. On the other hand, the capacity of the cardiovascular system to meet the oxygen demand of the working muscles and skin perfusion during exercise determines the cardiovascular strain.

Heat is held in the body as a function of its mass, mean temperature and mean specific heat of body tissues (Jay and Kenney, 2007). Therefore, body mass determines the metabolic heat production and represents the capacity of the body to store heat at rest; hence, individuals with greater body mass typically have smaller increases in core temperature during heat stress (Havenith et al., 1998; Havenith, 2001). Thus, greater metabolic heat production at rest was the product of larger body mass and greater FFM in the HIIT group (Table 3).

During exercise such as treadmill running, metabolic energy production is proportional to body mass, intensity (velocity) of treadmill running, and a treadmill incline (Johnson et al., 2002). Consequently, treadmill running at a faster velocity (a higher external workload) and/or greater body mass elicits a greater rate of metabolic heat production. At the maximum power output (W), the net metabolic heat production (M_T/BM) was significantly higher in the MD group. Despite the greater heat production of fitter individuals (MD) at a 100%VO_2 [W], their heat loss mechanisms were probably proportionally better, resulting in similar body temperature changes (ΔTty) as in their less fit counterparts, under conditions where heat loss was not limited by environmental conditions (Havenith et al., 1998). According to Fritzsche and Coyle (2000) and Periard et al. (2015), such results seem to indicate that endurance athletes are able to dissipate more heat and have slower body heating (ΔTty/time ($\Delta^oC \cdot min^{-1}$; Table 3). These results support the findings that endurance athletes exhibit the same characteristics of heat–acclimated individuals (Kenefick et al., 2007; Taylor, 2014).

Physiological strain

Comparable physiological strain can be obtained in different subjects although the strategy of obtaining the same level of strain indicated by the PSI could be different in trained men and in the control group. Under the conditions of this study, the PSI was similar for both groups of athletes, ie., MD and HIIT and lower in comparison to the control group (Table 3).

Our findings indicated that the level of physical fitness (VO_{2max}) significantly determined physiological strain (PSI) during exercise performed to exhaustion in a thermoneutral environment in all tested men (r=-0.47; p<0.05). These results are in accordance with those reported in literature (Merry et al., 2010; Tikuisis et al., 2002).

Thermoregulatory strain

The ambient temperature was not modified in our study. All exercise tests were performed in a temperature compensable environment; the study did not focus on the physiological strain resulting from the environment, but from exercise stress. Thus, the rate of heat storage and, consequently, the rise in

core temperature was primarily determined by balance between the metabolic of heat production and net heat dissipation from the body.

At the end of the exercise tests, aural canal temperatures ranged from 36.54 to 37.90 ^{o}C, and were lower than those reported by Moran et al. (1998a) (37.1-38.7^{o}C) in an experiment performed under thermally stressful conditions. Changes in core temperature are likely best assessed with exercise intensities administered to generate the metabolic heat production per unit mass, whereas changes in thermoregulatory sweating are potentially best assessed with exercise intensities administered to generate metabolic heat production per unit surface area (Cramer and Jay, 2014; Gonzalez-Alonso, 2012).

It was observed that a large body mass (BM), small body surface area (BSA), and low BSA-to-mass ratio predisposed to higher levels of heat strain (body heat storage) if workloads were equal or of the same relative level (same % VO_{2max}) (Havenith et al., 1998; 2001; Mora-Rodriguez et al., 2010; Mortensen et al., 2005). Both the whole-body and local sweating are determined largely by the athlete's metabolic heat production/ per unit BSA (in W $\cdot(m^2)^{-1}$) irrespective of relative exercise intensity (i.e. percentage of VO_{2max}) and core temperature (Jay et al., 2011). The Jay et al.'s (2011) study indicated that large differences in VO_{2max} (>20 ml $\cdot$ kg^{-1} $\cdot$min^{-1}) did not influence changes in core temperature and sweating during exercise in a neutral climate when the subject's body mass, BSA and a relative intensity (same % VO_{2max}) of exercise were similar. Our data suggests that MD athletes have an increased potential for heat dissipation independently of any difference in metabolic heat production.

We assumed that MD athletes would be characterized by a slower increase in aural canal temperature ΔTty (^{o}C/min) due to adaptive changes in thermoregulatory functions developed through endurance training, an increase in evaporative cooling, a greater sweating rate (Aoyogi et al., 1997; Smorawiński and Grucza, 1994) and sweating sensitivity. In our study performed in a temperature compensable environment, thermal adaptation developed by the MD group was expressed via slower body heating (a significantly slower increase of body temperature during exercise ΔTty/min), greater sweating sensitivity and a total sweat rate (expressed as BM loss %) in comparison with the other study groups (Table 4). Hence, thermoregulatory contribution to overall physiological strain in the MD group was 28% (*f*Tty: 0.28; Table 3). Greater whole body and local sweat rates as well as body heat accumulation are regularly reported in men of an increased fitness level in neutral and warm climates (Jay et al., 2011; Mora-Rodriguez et al., 2010). Higher VO_{2max} does not affect the endpoint body temperature during moderate exercise in a thermoneutral environment (Fritzsche and Coyle, 2000). However, the fitness level (VO_{2max}) may affect the body temperature of trained subjects during intense exercise in the heat (Gotshall et al., 2001; Mora-Rodriguez et al., 2010).

Our data clearly demonstrate that differences in absolute sweat production (SR; g $\cdot$ h^{-1}; g $\cdot$ (m^2)$^{-1}$ $\cdot$ h^{-1}) and body heating (a slower body temperature increase during exercise (ΔTty ^{o}C/min)) cannot be solely attributed to differences in the PSI between groups exercising in thermoneutral conditions. In our study, the control and HIIT groups exhibited a positive and significant correlation between the PSI and ΔTty (^{o}C). The PSI was negatively and significantly correlated with HRR only in the MD participants. These results may confirm the occurrence of subtle differences in the nature of adaptive changes achieved as a result of the endurance and heavy intermittent intensity type of training (Fowkes Godek et al., 2004; McMillan et al., 2005). The alternative possibility is that endurance athletes might be better prepared to tolerate thermal strain due to a better defensive adaptation to heat stress (Periard et al., 2015; Tikuisis et al., 2002).

Circulatory strain

The heart rate was selected as the principal variable for detection of physiological strain as it provides an integrated response to energy requirement, thermal stress and posture demand (Johnson and Park, 1981; Kellogg et al., 1993; Periard et al., 2010; Taylor, 2014), moreover, it also reflects interindividual differences.

In all our subjects, circulatory strain (*f*HR) was the major component (0.63-0.75) determining the overall physiological strain during the exercise test performed to exhaustion in a thermoneutral environment. The contribution of the circulatory

component to the overall physiological strain was significantly greater in the MD athletes compared to the control group. These results are contrary to heavy work in a hot and humid environment, where the fraction of thermoregulatory strain is the major component of the overall physiological strain (PSI) (Frank et al., 2001; Moran et al., 1998a). Optimal thermoregulatory responses are observed in trained male runners who are heat acclimated (Kenefick et al., 2007). It seems that during exercise performed in a thermoneutral environment and involving optimal thermoregulatory reactions, the overall physiological strain (PSI) resulting from exercise stress in the MD athletes is mainly determined by the cardiovascular load.

It has been established that the blood flow to an active muscle (and the myocardium) is required to meet the energetic demand for muscular activity (principally the demand for oxygen), while the blood flow to the skin is required to meet the demand for temperature regulation (Fritzsche and Coyle, 2000; Gonzalez-Alonso et al., 2008; Kellogg et al., 1993). These combined demands for the blood flow can result in a competition for the available cardiac output (Rowell, 1974) which has, as a bottom line, a limit to the ability to meet the dual demands of exercise *per se* and of temperature regulation, particularly during intense dynamic exercise in the heat (Gonzalez-Alonso et al., 2008; Gonzalez-Alonso, 2012; Johnson, 2010). In thermoneutral conditions, the thermoregulatory demand of the skin blood flow is not as high as in a hot environment; therefore, the muscle blood flow is not limited (Mortensen et al., 2005). Furthermore, the strategy of obtaining the same level of strain as indicated by the PSI could be different in hot and thermoneutral environments. Therefore, the demand for increasing the blood flow to an active muscle resulting from the metabolic demand during exercise in a thermoneutral environment did not induce strong competition between an increased blood flow to the active muscle and the skin. The greater cardiac output allows the athlete to achieve greater fractional muscle recruitment, peak aerobic power production and VO_{2max} than it has been observed in sedentary individuals (Mortensen et al., 2005). The heart rate reserve in the MD subjects was approximately 9 bpm higher than the HRR of the HIIT group. However, the maintenance of cardiovascular function and the intracellular metabolism during an exercise test is important both for endurance and high-intermittent intensity trained athletes.

Conclusions

The physiological strain during an exercise test performed to exhaustion was lower in athletes compared to untrained subjects. Variables of circulatory strain might be more important than thermoregulatory strain in predicting the overall physiological strain during exercise in a thermoneutral environment. However, the physiological strain itself and the contribution of its circulatory and thermal components did not differ significantly in response to exercise stress between trained (MD and HIIT) groups despite important differences in their morphological characteristics and (the type of training-induced systemic) circulatory and thermoregulatory adaptive changes.

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