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Original research article

NITRIC OXIDE AND LACTATE RESPONSES TO MAXIMAL INTERMITTENT ACUTE EXERCISE

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Abstract. *Nitric oxide (NO) is a gas with vasodilator and metabolic regulator properties. The present study aimed to examine the relation between NO and lactic acid, and NO difference in acute anaerobic and aerobic loads in aerobic and anaerobic exercise groups. Three groups participated in this study; each group consisted of 11 men with similar physical characteristics. The groups consisted of swimmers as the aerobic group (AeG), volleyball players as the anaerobic group (AnG), and control group (CG). Participants were given 3 acute exercise phases in the scope of lactate minimum speed test (LMSt). NO and lactic acid measurements were taken at particular phases in LMSt. Lactate minimum speed values of AeG (11.5±1.1 km·h-1) were significantly higher than those of the CG ($p<0.05$). Significant decrease (25.6%) was found in the NO levels in AeG after the Wingate test ($p<0.05$). The difference between NO values after Wingate test and recovery NO (ΔWNO) in AeG was significantly greater than that of AnG and CG ($p<0.05$). AeG NO value following the reloading phase was greater ($p>0.05$; 9.2%) than the base NO. Additionally, contrary to the AnG, an increase was observed in ΔNOlevel during active recovery and in NO level after the reloading phase in AeG. Also, lactate elimination level of the aerobic group was higher than the other groups. As a result; these findings show the role of a more active lactate elimination capacity since NO levels in the aerobic group are higher than the other groups following a maximal intermittent exercise.*

Key words: *Nitric oxide, lactate minimum, performance, aerobic, anaerobic.*

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INTRODUCTION

Nitric oxide (NO), which is produced and segregated by many tissues such as vascular endothelium and muscles, has vasodilator, antioxidant, antiatherosclerotic and metabolic regulator properties (Kingwell, 2000). NO is a small and reactive free radical molecule functioning as a regulator both inside and outside the cell with a half-life of 2-5 seconds (Gruetter, Barry & McNamara, 1979). The biological properties of NO are the formation of the vascular dilator tone, the regulation of local cell growth and maintenance of vascular homeostasis (Kingwell, 2000; Stamler & Meissner, 2001).

In the adaptations that occur after training, NO mediated vasodilatation is observed in the short term while metabolic enzyme change and vascular reformation is seen in the long term. Vascular endothelial function has been reported to improve after short-term training (a few days). It has been reported that the increase in shear stress, which occurs and causes NO production during exercise, has been stated to help recovery due to increased blood flow as a result of these trainings (Kingwell, 2000; Tietz, 1986). If adaptations are also present in coronary circulation as well, the increased dilator reserve capacity in athletes can be stimulated physiologically by endothelial mechanisms (Haskell et al., 1993; Kingwell, 2000).

Shear stress during physical exercise is an important factor in NO production. Exercise increases intracoronary blood flow and this increased blood flow raises shear stress in the endothelium of epicardial vessels and causes venous vasodilatation. The metabolic control of NO in exercise is effective along with several mechanisms, which be listed as the increase in skeletal muscles and blood flow with exercise, increase in substrates, regulating hormones and oxygen transportation, protecting energy storages of intracellular skeletal muscles by promoting glucose uptake independently from insulin (Kingwell, 2000).

Considering all of these, the above-mentioned effects of NO on blood flow, substrate use and contraction function are directly intended for protection from ischemia. Transportation of blood and substrates to working muscles including myocardium will contribute to the increased exercise performance (Kingwell, 2000). The use of L-arginine, the nitric oxide precursor, has been reported to decrease lactate levels in athletes during submaximal exercise and improve working capacity with the increase in NO production (Burtscher et al., 2005; Maxwell et al., 2001; Schaefer et al., 2002).

In addition, high amounts of blood lactic acid can be accumulated during aerobic and anaerobic training and competitions. Lactic acid may impede glycolysis rate by inhibiting the activity of glycolytic enzymes (Ahmaidi et al., 1996). Moreover, high levels of lactate suppress fatty acid oxidation (Shephard, 1984). Therefore, it is important to remove lactate from blood following exercise. During exercise, lactate is eliminated by the heart, liver and working and resting muscles (Costill et al., 1997). Studies have shown that L-arginine, the nitric oxide precursor, support, decreases lactate levels during submaximal exercise in humans and improves working capacity with the increase in NO production (Burtscher et al., 2005; Maxwell et al., 2001). Muscles, the liver, heart and kidneys may influence recovery by allowing for the transportation of more substrates and hormones due to the increased blood flow via NO (Costill et al., 1997). Therefore, endurance athletes, who have greater endothelium dependent vasodilator reserves, are expected to metabolize lactate faster than athletes trained in anaerobic exercise. Therefore, a relationship between NO and lactate elimination is possible especially in aerobic athletes during exercise. Knowing these relationships can provide information about the appropriate training loads to be used in intermittent training frequently and especially about

quality of active recovery phases. To this end, we planned an acute maximal intermittent exercise and aimed to examine NO and lactate responses to these exercises.

THE METHOD

Participants

The study was conducted with the voluntary participation of 33 male individuals. The anaerobic group (AnG) consisted of 11 volleyball players, (age 21.7±1.1 years, body height 185.3±5.6 cm, body mass 79.1±6.7 kg), the aerobic group (AeG) of 11 swimmers (age 21±1.4 years, body height 178.4±7 cm, body mass 75.5±9.8 kg) and, the control group (CG) of 11 men who had not exercised regularly for at least three months (age 21.7±1 years, body height 182.2±6.5 cm, body mass 77.2±6.2 kg). The volleyball group has 10 years and the swimming group had 12 years of sporting experience. The participants of the study were informed about the aim, benefits, necessary tests and possible risks of the study. Ethics committee approval and written informed consent form were obtained (*Approval no: 07/7-3*). The participants were instructed not to change their diets in the week prior to the measurings and not to do strenous exercise for at least two days.

Measures and procedures

Lactate minimum speed (*LMS*) *determination*

Each participant was given 3 acute exercise phases in the scope of the Lactate minimum speed test as required by the protocol (Simoes et al., 2003). The first one of these was the maximal 30-second Wingate anaerobic power and capacity test (75 g·kg-1 load for body weight) performed on the bicycle ergometer (Monark 834; Monark Exercise, Varberg, Sweden) (Bar-Or, 1987). This exercise phase was named the Wingate Phase (WP). The point after the 5-minute passive resting following this exercise phase was called (W) and the lactate and NO values seen in the blood taken from finger tips at this point were named (WLA and WNO) respectively. In order to buffer the metabolic acidosis produced as a result of this WP and to eliminate the increased lactate value, an Active Recovery Phase (ARP) was held on the motorized treadmill (Star Track 4000, Unisen, Inc.) (the second exercise phase). ARP was in the form of a running exercise with 3 different submaximal intervals, each step of which lasted 4 minutes at increasing intensity. This phase continued until the Lactate minimum point (LM) where a balance occurred between the production and elimination of lactate. Blood lactate and NO values at this point were called (LMLA and LMNO). After this point, a reloading phase (RP) was held which continued until maximum values were determined (the third exercise phase). RP consisted of an exercise with 3 different 4-minute maximal intervals at increasing intensity. This point at the end of the lactate minimum test was called (E) and LA and NO values at this point were named as (ELA and ENO). For each participant, the load increase continued until exhaustion, and the participants mostly left the test at step 6. The participants' heart rates at the end of the test reached or exceeded the age predicted maximum heart rates and LA values were ~8mM. At the end of these three exercise phases, exercise load where lactate was minimum was called lactate minimum speed (Simoes et al., 2003). The initial speed for the ARP of the LMS test was taken in the present study as 70% of the time each athlete runs 3000 meters.

The speed increase for each step was set to 0.5-1 km·h-1. Polar RS 400 heart rate monitor (Polar RS 400, Polar Electro, Sweden) was used for heart rate measurements during testing.

Collection, preservation and analyses of blood samples

Blood samples were drawn from the finger of the hand, at certain steps of the LMS test into 4 heparinized capillary tubes and two of them were put and stirred in lactate preservative tubes and kept refrigerated until lactate analysis. Other capillary tubes of blood samples were centrifuged for 1500 g (Nüve NF 200, Ankara/Turkiye) at 15 min and acquired plasmas were stored in the freezer -80 ºC until NO analysis. Nitric oxide (total nitrite) measurements were taken from these plasma samples in 10-15 days.

Lactic acid analysis

Lactate samples were analyzed with YSI 1500 lactate analyzer (YSI 1500 Sport, YSI, Yellow Springs, OH) on the same day with an immobilized enzyme electrode technology method. Before obtaining each participant's measurements, analyzer calibration was done with 5 mM standard calibration solution and daily calibration with 5 and 15 mM.

Nitric oxide analysis

With a few seconds of half-life, NO is oxidized into nitrite, which is a vasoinactive and stable metabolite in the blood after a while. Nitrite is converted into nitrate in all of the blood. Therefore, measurements of nitrite and nitrate, which are stable metabolites of NO, were used for blood NO analysis (Kingwell, 2000; Turgay, 2004). For this reason, in the present study, blood total nitrite levels were taken as the criteria for blood NO levels. Analyses of plasma nitric oxide levels were carried out with NO kits (Oxis International Inc. USA). The method is based on the principle of spectrophotometric determination of the absorbance of the pink azo dye generated by 'Griess reactive' and the nitrite produced as a result of reducing nitrate (NO3), the main metabolite of nitric oxide, to nitrite (NO2) with cadmium (Cd+2). With this method, it is possible to measure the total levels of nitrite which are produced by reduction from the nitrite and nitrate present in the sample.

Statistical analysis

The data were presented as means and standard deviations. Normal distribution was determined with the Kolmogorov-Smirnov test while homogeneity was determined using the Levene test. As a result of the normal distribution and homogeneity tests, the data were found to be normally distributed, the groups were determined to be homogenous, and parametric analysis techniques were employed. The 'Pearson rho' correlation analysis was used to reveal the relationships between blood NO levels and LMS, peak power, mean power, the fatigue index and the relationships between delta NO (ΔNO) and delta LA (ΔLA). The one-way analysis of variance (One-way ANOVA) was used to compare the mean differences between groups and LSD was applied as the 'post hoc' test. The data were analyzed using SPSS version 11.0 (SPSS Inc, Chicago, IL). The value of significance was taken as $p<0.05$.

RESULTS

Lactate minimum speed test

Significant differences were found in peak power between three groups ($p<0.0001$). The highest peak power was observed in the AnG group (13.11±0.96 W·kg-1). AnG was found to be 24.94% higher than AeG and %11.36 higher than CG. Mean power of the AnG (8.55±0.43 W·kg-1) was significantly higher than the AeG (7.36±0.84 W·kg-1, $p=0.001$, 13.91%), whereas mean power of the CG was 4.77% lower than the AnG, and no significant difference was found ($p=0.219$).

No significant difference was found between the heart rates of groups as a result of the Wingate test (AeG=177.7±7.1, AnG=179.3±5.1, CG=180.2±8.4 beats·min-1). AeG lactate minimum speed (11.59±1.17 km·h-1) was 5.52% higher than the AnG (10.95±0.96 km·h-1, $p=0.382$), and 11.38% higher than the CG value (10.27±0.68 km·h-1, $p=0.003$). No significant difference was found between groups in heart rates at lactate minimum point (AeG=171.2±7.2, AnG=169±5.2, CG=170.9±5.2 beats·min-1) and at the end of the reloading phase (AeG=190±6.4, AnG=187.1±3.8, CG=190.4±6.6 beats·min-1).

Blood lactate values of groups on the LMS test

Blood lactate values of groups on the LMS test are presented in Figure 1. Lactate minimum values of the groups obtained at different lactate measurement points were determined as 4.38±0.78 for AeG, 4.66±0.67 for AnG and 4.63±0.62 mM for CG. WLA value of AnG was significantly higher than that of AeG ($p<0.05$). In addition, the ELA value of CG was significantly higher than that of AeG ($p<0.05$).

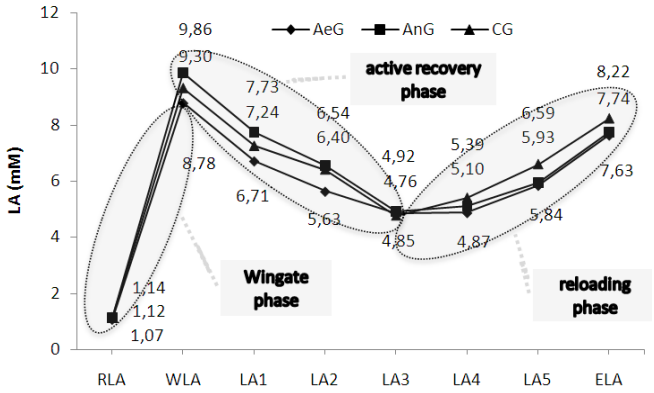

Fig. 1 Blood lactate values of groups in the LMS test
RLA: resting lactate, WLA: Wingate test lactate (after 5 min.), LA1: lactate at first stage, LA2: lactate at second stage, LA3: lactate at third stage, LA4: lactate at fourth stage, LA5: lactate at fifth stage, ELA: lactate at end of the test, AeG: aerobic group, AnG: anaerobic group, CG: control group.

NO values of groups during rest and three acute exercise phases

NO values of groups during rest and three acute exercise phases are presented in figure 2. In the repeated NO measurements of AeG, WNO value was found to be significantly lower than the RNO value ($p<0.05$).

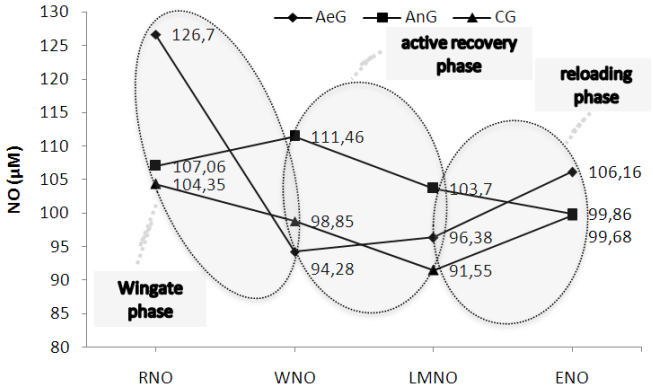

Fig. 2 NO (µM) values of groups at rest and the lactate minimum test
RNO: resting nitric oxide, WNO: Wingate test nitric oxide (after 5 min.), LMNO: nitric oxide at lactate minimum point, ENO: nitric oxide at end of the test, AeG: aerobic group, AnG: anaerobic group, CG: control group.

ΔNO and ΔLA values of groups during rest and at the end of three acute exercise phases

ΔNO values of groups during rest and at the end of three acute exercise phases are presented in figure 3. The ΔWNO value of AeG was significantly higher than that of AnG and CG ($p<0.05$). ΔLA values of the male groups during rest and at the end of three acute exercise phases are given in Table 1. The ΔWLA value of AnG was found to be significantly higher than the AeG value ($p<0.05$).

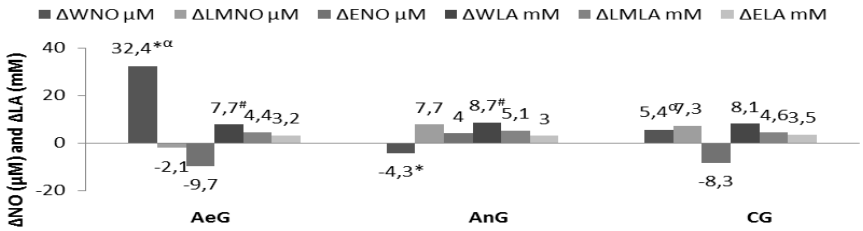

Fig. 3 ΔNO (µM) and ΔLA (mM) values of groups during rest and at the end of three acute exercise phases
$^{\alpha}p<0.05$; between AeG and AnG at ΔWNO,*$p<0.05$; between AeG and KG at ΔWNO, $^{\#}p<0.05$; between AnG and AeG at ΔWLA. ΔWNO; difference between resting nitric oxide (RNO) and Wingate test nitric oxide (WNO), ΔLMNO; difference between Wingate test nitric oxide (WNO) and nitric oxide at lactate minimum point (LMNO), ΔENO; difference between nitric oxide at lactate minimum point (LMNO) and nitric oxide at end of the test (ENO), ΔWLA; difference between resting lactate (RLA) and Wingate test lactate (WLA), ΔLMLA; difference between Wingate test lactate (WLA) and lactate at lactate minimum point (LMNO), ΔELA; difference between lactate at lactate minimum point (LMLA) and lactate at end of the test (ELA), AeG: aerobic group, AnG: anaerobic group, CG: control group.

Results of the ΔNO and ΔLA correlation analysis of the groups

A positive correlation was found between ΔLMNO and ΔLMLA values in AeG (r=0.628, p<0.05). The non-significant correlations were obtained between ΔLA and ΔNO values in AnG and CG (Table 1).

Table 1 Results of the ΔNO and ΔLA correlation analysis.

	Variable	ΔWLA	ΔLMLA	ΔELA
AeG	ΔWNO	-0.380	-	-
	ΔLMNO	-	0.628*	-
	ΔENO	-	-	-0.264
AnG	ΔWNO	0.025	-	-
	ΔLMNO	-	-0.151	-
	ΔENO	-	-	0.022
CG	ΔWNO	-0.414	-	-
	ΔLMNO	-	0.355	-
	ΔENO	-	-	0.082

*p<0.05, ΔWNO; difference between resting nitric oxide (RNO) and Wingate test nitric oxide (WNO), ΔLMNO; difference between Wingate test nitric oxide (WNO) and nitric oxide at lactate minimum point (LMNO), ΔENO; difference between nitric oxide at lactate minimum point (LMNO) and nitric oxide at end of the test (ENO), ΔWLA; difference between resting lactate (RLA) and Wingate test lactate (WLA), ΔLMLA; difference between Wingate test lactate (WLA) and lactate at lactate minimum point (LMNO), ΔELA; difference between lactate at lactate minimum point (LMLA) and lactate at end of the test (ELA), AeG: aerobic group, AnG: anaerobic group, CG: control group.

Results of the nitric oxide and LMS test correlation analysis of the groups

No significant correlations were found between the groups' nitric oxide values belonging to different phases of the lactate minimum test and the lactate minimum speed test performance data (Table 2).

Table 2 Results of the NO and LMS test correlation analysis

	Variable	LMS ($km \cdot s^{-1}$)	Peak power ($W \cdot kg^{-1}$)	Mean power ($W \cdot kg^{-1}$)	FI (%)
AeG	RNO	0.015	0.159	-0.335	0.521
	WNO	-0.361	0.386	-0.164	0.334
	LMNO	-0.088	0.164	-0.373	0.298
	ENO	-0.329	0.126	-0.354	0.075
AnG	RNO	0.319	-0.060	0.201	-0.585
	WNO	0.040	-0.290	-0.192	-0.321
	LMNO	-0.159	0.174	0.045	-0.185
	ENO	0.093	-0.247	-0.140	-0.367
CG	RNO	0.463	-0.152	-0.084	-0.063
	WNO	0.532	-0.163	-0.134	-0.175
	LMNO	0.229	-0.242	-0.441	0.377
	ENO	0.387	-0.378	-0.387	-0.059

For all r values, p>0.05, LMS: lactate minimum speed, FI: fatigue index, RNO: resting nitric oxide, WNO: Wingate test nitric oxide (after 5 min.), LMNO: nitric oxide at lactate minimum point, ENO: nitric oxide at end of the test, AeG: aerobic group, AnG: anaerobic group, CG: control group.

DISCUSSION

Responses to maximal exercises (Wingate and reloading phase)

Mean values of LA (9.31 mM) obtained with the Wingate test (WLA) pertaining to three groups were higher than the mean values of LA (7.86 mM) obtained in the reloading phase (ELA). While mean heart rate was found to be around 179.06 beats·min-1 in all groups following the Wingate test, and 189.16 beats·min-1 following the reloading phase, it has a maximal quality. Considering the data pertaining to lactate and heart rate, it can be said that the reloading phase possesses a maximal quality and creates an anaerobic environment.

In aerobic and control groups, unlike anaerobic group, an expected negative but non-significant relationship was found between ΔWNO and ΔWLA after the Wingate phase. These relations show that individuals who produce much lactate during the Wingate phase may produce less NO. Briefly stated, these relations indicate that the anaerobic condition created after supramaximal exercise like the Wingate phase may suppress NO levels or decrease the survival time (bioefficacy) of NO. But unlike these relationships, anerobic group's NO levels increased in contrary to the other groups. This could result from the fact that the anaerobic group has a more developed lactate tolerance (an improved acid-base buffer system) than the others. Moreover, the fact that peak lactate and Wingate performance values of the anaerobic group following the Wingate test were higher than the others also support our opinion in this respect. These findings may mean that in the anaerobic group, even under the anaerobic conditions of a Wingate type maximal exercise, NO production could be allowed without being impeded by high lactate, unlike in the case of the other groups.

Responses to active recovery exercise

A positive relationship was found between ΔLMNO and ΔLMLA in the aerobic group. This finding shows that individuals who have a high capacity of lactate elimination during the active recovery phase may also have a high capacity of NO production in the meantime. In the aerobic group, this significant relationship found between lactate recovery capacity and NO increase may indicate the mutual role of lactate and NO in the adaptations created by aerobic training. It can be concluded that in the aerobic group that has a high endurance level, as lactate elimination increases and metabolic acidosis is buffered, a suitable environment is created for blood NO levels to increase again or to decrease NO elimination at the LM point where a balance is set up between the production and elimination of lactate. At LM, NO only increased in AeG while LA decreases, in addition to, LM speed of AeG was higher than those of the other groups, this findings support our view. Following a maximal effort, while NO fell in the aerobic group, after active recovery phase the NO increase in this group supports the argument that aerobic exercises increase NO production; whereas oxidative stress conditions created with metabolic acidosis inhibited NO production (Kingwell, 2000).

Moreover, the fact that the positive relationship found between ΔLMNO and ΔLMLA at LM of the aerobic group was contrary to the one in the anaerobic group may result from the inability of this group's aerobic endurance level to eliminate high lactic acid levels in the environment as effectively as the aerobic group, or the rapid formation of some factors (as oxidative stress) suppressing NO existence, in the formation of an additional aerobic environment again and that the anaerobic group has not immediately adapted to this environment.

Lactic acid may impede muscle glycolysis speed by inhibiting glycolytic enzyme activity (Ahmaidi et al., 1996), high levels of lactate suppress fatty acid oxidation (Shephard, 1984). Active recovery has been reported to remove lactate from blood in a much quicker way than the passive (Ahmaidi et al., 1996). Significant relations have been found between endurance levels and lactate elimination; and between physical fitness (maxVO2) and basal blood NO levels (Jungersten, Ambring, Wall, & Wennmalm, 1997; Turgay, 2004). Therefore, similar relations are expected to exist between LMS and blood NO levels and ΔLMLA. In the present study; however, no significant relation was found between these parameters in any of the groups. Another study, on the other hand, has shown that L-arginine, the nitric oxide precursor, supports decreases in lactate levels during a submaximal exercise in humans (Schaefer et al., 2002) and improves working capacity with the increase in NO production (Burtscher et al., 2005; Maxwell et al., 2001).

In a study carried out on trained endurance athletes, it was found that as a result of the NO precursor L-Arginine infusion during a bicycle exercise, plasma concentration remained the same while glucose destruction increased, the increase in plasma fatty acid levels fell and lactate levels went up (McConell, et al., 2006). These findings reveal that this type of exercise increases both glucose and fat oxidation. In this respect, it could be asserted that unlike the Wingate phase, NO produced during an intermittent maximal reloading phase increases muscle glucose uptake and use; however, it increases lactate levels in maximal exercise phases by restricting oxygen use (Kingwell, 2000). The use of more aerobic energy may have played a role in the finding that peak lactate values after the active recovery phase were lower than those in the Wingate phase.

CONCLUSION

In the aerobic group, the increase in NO despite the increased lactate concentration after the reloading phase, although NO levels fell after the Wingate phase, may be caused by the aerobic active recovery phase performed between these two maximal phases. Finding a significant positive relationship between ΔLMLA and ΔLMNO after an active recovery phase in the aerobic group only supports this opinion. Relations found between ΔLA and ΔNO after two different maximal phases moved in a similar direction and in a negative one in the aerobic group, whereas the relations between these two parameters moved in a positive direction after the Wingate phase in the anaerobic group, but it was in the opposite direction after the active recovery phase. It is interesting that contrary to the aerobic group, NO level decreased in the control group in the active recovery phase, following the reloading phase, NO level increased similar with the aerobic group. This could be claimed to be due to the fact that the control group created a physiological environment in which it can benefit from active recovery more than the aerobic group and increased NO levels. On the other hand, for the decrease in NO levels after the active recovery phase in the anaerobic group, it could be considered that they may have been inadequate in creating an environment that can reproduce NO or eliminate a possible NOS inhibition in the present environment after active recovery as a result of the chronic anaerobic training done by this group. These findings show the role of a more active lactate elimination capacity in the fact that NO levels in the aerobic group are higher than other groups after a maximal intermittent exercise.

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UTICAJ INTERVALNOG TRENINGA MAKSIMALNOG INTENZITETA NA NIVOE AZOT MONOKSIDA I LAKTATA

Azot monoksid (NO) je vazodilatatorni gas sa svojstvima regulatora metabolizma. Cilj ovog istraživanja je da istraži odnos između azot monoksida i mlečne kiseline, i razlike u nivou azot monoksida pri anaerobnom i aerobnom opterećenju kod osoba uključenih u aerobni i anaerobni trening. Tri grupe ispitanika učestvovale su u istraživanju; svaku grupu činilo je 11 muškaraca sa sličnim fizičkim karakteristikama. Jednu grupu ispitanika činili su plivači, koji su predstavljali aerobnu grupu (AeG), drugu odbojkaši koji su predstavljali anaerobnu grupu (AnG), dok je treća grupa bila kontrolna (CG). Učesnici su u toku treninga imali tri faze inenzivnih vežbi u okviru takozvanogtesta minimalne brzine za određivanje vrednosti laktata (lactate minimum speed test, LMSt). Vrednosti NO i mlečne kiseline merene su tokom određenih faza LMSt. Vrednosti laktata pri minimalnoj brzini kod AeG grupe (11.5±1.1 km·h-1) bile su značajno veće od onih kod CG grupe ($p<0.05$). Značajno smanjenje (25.6%) nivoa NO uočeno je kod AeG grupe nakonVingejt testa ($p<0.05$). Razlika izmeđuvrednosti NO nakon Vingejt testa i oporavka (ΔWNO) kod grupe AeG bila je značajno veća od vrednostigrupa AnG i CG ($p<0.05$). AeG vrednosti NO nakon faze dodavanja opterećenja bile su veće ($p>0.05$; 9.2%) od osnovnih vrednosti NO. Pored toga, za razliku od AnG grupe, povećanje je uočeno na ΔNOlevel nivou tokom aktivnog oporavka, i kod nivoa NO nakon faze povećanja opterećenja kod AeG grupe. Takođe, nivo eliminacije laktata kod članova aerobne grupe bio je viši nego kod ostalih grupa. Kao posledica toga, ovi podaci ukazuju na veću sposobnost eliminacije laktata, s obzirom na to da su NO nivoi kod aeerobne grupe veći nego kod ostalih grupa nakon intervalnog treninga maksimalnog intenziteta.

Ključne reči: *azot monoksid, minimalne vrednosti laktata, izvođenje vežbi, aerobno, anaerobno.*