

Influence of Neuromuscular Fatigue and Metabolic Acidosis on the 400-Meter Race



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ABSTRACT

Unusual exercises can lead to muscle damage that persists for a few days reducing performance ability due to fatigue onset. Moreover, intramuscular acidity increase can limit the cell metabolism in the process of producing work. Therefore, the objective of this research was to analyze the influence of neuromuscular fatigue and metabolic acidity in the 400 m race. The selected sample consisted of 20 sedentary individuals, aged between 18 and 35 years. They were submitted to the following protocols: treadmill incremental test for determination of O_{2max} ; aerobic and anaerobic threshold; 400m race test (400/R); plyometric activity with active/passive rest followed by 400m race immediately after (400/Post) and 24 hours after the plyometric activity (400/24h). The obtained results show that when the active and passive groups are compared, they do not show significant difference in 400/Post performance, but this time was longer for both groups when compared with the 400/R. Nevertheless, the 400/24h was not significantly different when compared with the 400/R to both groups. It was concluded that regardless of the kind of recovery, active or passive, the performance reduction in a 400-meter race after plyometric activity seems to occur by neuromuscular mechanisms that lead to fatigue and not to metabolic limitations.

Keywords: neuromuscular fatigue, anaerobic performance, eccentric activity.

INTRODUCTION

It is known that the muscle tissue is able to produce strength when activated ⁽¹⁾. However, the incapacity of producing strength repeatedly is characterized as neuromuscular fatigue, a symptom which can last for many days or weeks ⁽²⁾. The causes of neuromuscular fatigue during exercise can be of central origin (cortical and subcortical regions of the brain) or of peripheral origin (skeletal muscle tissue)⁽³⁾.

Plyometric exercises are defined as those which activate the eccentric- concentric cycle of the skeletal muscle, causing its mechanical, elastic and reflex boosting. This cycle refers to the concentric activities subsequent to an eccentric action, whose aim is to increase the explosive strength of the muscle through the storage of elastic energy in the pre-stretching phase and its reuse during the concentric contraction, besides the activation of the myotatic reflex ⁽⁴⁾.

The skeletal muscles have the function to dislocate the segments, stabilize the articulations and absorb the external forces. An example of external force is the landing after a vertical jump, in which the ankle, knee and hip joints are flexed and the extensor muscles of these joints produce negative work; that is to say, the direction of the movement is contrary to the produced strength, in which an eccentric action where the muscle is stretched is characterized, with concomitant generation of tension, showing hence that the mechanism of this kind of muscle action as well as the control mechanisms of strength production are different from the ones used in concentric and isometric muscle actions and characterizing the eccentric actions as a contraction able to generate greater tension by muscle unit ⁽⁵⁾.

Therefore, it is known that the higher the muscle work intensity performed; the higher the concentrations of metabolites produced by the recruited musculature, among these, lactate. It is known that the lactate production rate after exercise is intensity-dependent; that is, the higher the exertion intensity, the higher the production and build up of this metabolite ⁽⁶⁾.

Concerning the lactate build up rate in the blood, it is a result from the this metabolite production and removal processes, being determined by the combination of many factors, among these, the type of fiber, the muscle respiratory capacity, the mobilization of energetic substrates as well as the biochemical characteristics of the skeletal muscle cells ⁽⁷⁾.

However, regardless of the tissue oxidation, the lactate build up occurs due to the increase of the

fast contraction fibers which are recruited according to the increase of the exercise intensity⁽⁸⁾.

Nevertheless, it is known that after a high intensity exercise the type of recovery used may be an important factor to the higher removal rates. A priori, active recovery presents a lactate removal capacity higher than the passive recovery⁽⁶⁾. The oxidation of this metabolite during the active recovery occurs mainly in the active skeletal muscles and in lower level in the non-active skeletal muscles during recovery, as well as by the myocardium⁽⁹⁾.

Thus, generally assessing the interaction of different metabolic ways and different types of muscle contraction which can occur in a single exercise session, the main aim of this study was to verify the influence of neuromuscular fatigue and metabolic acidosis on the subsequent performance in a 400-meter race.

CAUSES AND METHODS

Environmental conditions

The present study was carried out in the Laboratory of Physical Effort Evaluation (LAEF) and on the athletics track of the Auxilium Salesian Catholic University Center (Unisaesiano – Lins). The experimental procedures were held from 11h to 19h and indoor environmental temperature (laboratory) was controlled by an air conditioner and the times for each volunteer during this period were respected in order to minimize any influence concerning the circadian cycle as well as daily habits.

Volunteers

20 male individuals, apparently healthy, sedentary, aged between 18 and 35 years participated in this project. These individuals after having received verbal and written information through a consent form on all the study's procedures, consented the participation and release of the collected data. All procedures were sent, analysed and approved by the Ethics in Research Committee of the Auxilium Salesian Catholic University Center (Unisaesiano – Lins – Protocol 116/2008).

Experimental Outline

The tests were conducted in four different days. On the first week of experimental procedures, the volunteers were assessed concerning the anthropometrical measurements (total body mass, stature and body fat percentage) and power and aerobic capacities (O_{2max} , aerobic and anaerobic thresholds). After an interval of three to seven days, the individuals were taken to the athletics track for the 400-meter test performance. Once again, after an interval of three to seven days, they were submitted to an activity of plyometric jumps, and at the end of these jumps, they were randomly designated to an active recovery (G1) or passive recovery (G2) of 30 minutes. After this recovery, they were once submitted to the 400-meter test again, immediately after the plyometric activity and 24 hours after the performance of this exercise.

TESTS

Determination of the fat percentage

Fat percentage was determined with a scientific adipometer (Cescorf), which measures the subcutaneous adipose tissue thickness. The following skinfold measurements were collected:

tricipital, suprailiac and abdominal. The fat percentage was determined from these measurements with a specific table⁽¹⁰⁾.

Blood collection and analysis and heart rate monitoring

25µl of blood from the earlobe were collected for determination of lactate concentrations, which was transferred to an Ependorf tube containing 50µl of sodium fluoride (1%) and stored in a freezer (-20°C) for later analysis (Yellow Springs 1500 – Sport). The collections were performed in the tests for determination of O_{2max} , anaerobic and aerobic thresholds, in the 400-meter test, in the active rest and passive rest. Likewise, heart rate (HR) was monitored with a cardiac frequency meter (RS200sd – POLAR) during all the study's procedures, being observed mainly at the same moments that the blood collection occurred.

VO_{2max} , anaerobic threshold and aerobic threshold determination

The anaerobic (LAn) and aerobic thresholds (Laer) and O_{2max} were assessed in the same test. Such test was performed in a progressive and intermittent manner on a treadmill (Imbramed – 10200 ATL), and initial velocity was of seven or eight kilometers per hour, being increased in one kilometer per hour at every three minutes, until voluntary exhaustion of the participants. At the end of each stage, 25µl of blood were collected for blood lactate analysis. Thus, the velocity concerning the thresholds was determined by linear interpolation, in which the steady concentrations of 2mM and 3.5mM were adopted for Laer and LAn, respectively. The O_{2max} was determined with a gas analyser (Metalyser 3B – CORTEX), in which through the assessment of the gas exchanges during the exercise, the O_{2max} was determined through the highest value of oxygen consumption during the test.

400-meter race

The maximum 400-meter race was performed on an athletics track of 233 meters, where, the volunteers were verbally encouraged to run the 400 meters distance previously marked at the fastest time possible. At the end of this effort, at moments one, three and five minutes after it, 25µl of blood were collected for determination of peak lactate concentration. It was performed at three situations: control (400/C), at which the volunteers should arrive at the test site with at least 48 hours of absence of any kind of extenuating physical activity; after the plyometric jumps and the application of the type of recovery (active and passive) (400/Post) and 24 hours after the performance of the plyometric jumps (400/24h).

Plyometric activity

It was composed of 10 sets of 10 jumps at depth, having a one-minute interval between each set. The participants started from a raised plane (0.6m). After landing on the ground, they were asked to perform a vertical jump as strong and fast as possible, with the least of contact time with the ground, landing on another raised plane placed forward and one meter away from the first one. The action went on like this, until all the jumps and sets were performed⁽¹¹⁾.

Active and passive recovery

The active and passive recovery was observed in a 30-minute

period at the Laer velocity (2mM) and at total rest, respectively, and at each five minutes 25µl of blood of the earlobe were collected for subsequent analysis. At this moment of the experimental procedures the volunteers were randomly named in the two treatments active recovery group (G1) and passive recovery group (G2).

Statistical analysis

The obtained results were assessed through descriptive statistics, in which data were expressed in mean and standard deviation. However, prior to any analysis, data were assessed concerning distribution normality through normality test by Shapiro-Wilk. Once data normality was confirmed, parameter statistical methods were adopted for their treatment. Paired Student's t test for independent data was used for analysis of the initial characteristics of the volunteers as well as comparison between groups. Analysis of variance (one-way ANOVA) with Tukey post hoc test was adopted for comparison of results of the anaerobic performance and peak lactate concentration of the performance tests. In order to correlate the blood samples to the individual characteristics and performance data Pearson correlation test was used. Statistical significance was identified with significance level of $p \leq 0.05$ for all procedures.

RESULTS

Table 1 presents the initial characteristics of the volunteers of the study, distributed in the groups G1 and G2. The groups presented variance equality and did not present significant differences concerning means of the presented variables. Such fact demonstrates homogeneity between the studied groups, avoiding any problem concerning heterogeneity.

Regarding the time spent to complete 400 meters on the athletics track as fast as possible in the three situations, namely control, immediately after the plyometric activity (400/Post) and in the situation 24 hours after the plyometric activity (400/24h) for G1 and G2, it can be verified through a two-way analysis of variance that the performance behavior in the 400-meter race was similar when the groups are compared and no significant differences are presented in the three situations mentioned above. Concerning intragroup analysis, it was verified that the plyometric activity caused statistically significant decrease of performance in the 400/Post; however, this performance was recovered after 24 hours of rest, when the same 400-meter exercise was repeated (table 2).

Tables 3 and 4 present the lactate concentration values in the pre and post recovery situations and lactate concentrations in the pre and post 400-meter situations in the control and after active or passive recovery periods. Table 3 shows that for the two examples of recovery the lactate removal occurred at the same magnitude at first; that is to say, the removal process occurred for both, but with no difference between the means.

Table 4 presents that for all situations control (G1-400/C and G2-400/C) and after recovery period (G1-400/Post and G2-400/Post), the lactate concentrations increased subsequently to the performance of the 400-meter race when compared to the moment before it. Nonetheless, in an intragroup analysis, that is, control and after recovery period, it was observed that the behavior was almost similar for both groups, where the lactate

Table 1. Initial characteristics of the active (G1, n = 10) and pswasive groups (G2, n = 10).

	G1	G2
Height (cm)	175.0 ± 4.1	172.7 ± 6.14
Total body mass (kg)	70.6 ± 8.6	73.38 ± 11.82
Fat percentage (%)	16.2 ± 8.0	16.27 ± 8.32
O _{2max} (ml/kg/min)	44.0 ± 9.2	47.88 ± 10.16
O _{2max} (km/h)	14.1 ± 1.7	13.5 ± 1.7
Laer (km/h)	10.2 ± 1.4	10.3 ± 2.1
Laer x O _{2max} (%)	73.0 ± 8.4	75.9 ± 8.0

$p \leq 0.05$.

Table 2. Times in the 400m in the control situations (C), after plyometrics (400/Post) and 24 hours after plyometrics (400/24h).

	Control (seg.)	400/Post (seg.)	400/24h (sec.)
G1	73.49 ± 8.27	83.06 ± 10.53 ^a	74.96 ± 8.90 ^b
G2	75.45 ± 8.87	83.03 ± 14.02 ^a	76.67 ± 9.26 ^b

a – significant differences concerning the Control;

b – significant differences concerning the 400/Post; $p \leq 0.05$.

Table 3. Lactate concentrations in the pre and post situations after 30 minutes of active (G1) and passive recovery (G2).

	Pre (mM)	Post (mM)
G1	4.27 ± 1.90	2.66 ± 1.33 ^a
G2	4.94 ± 3.68	1.85 ± 1.71 ^a

a – significant difference concerning the pre situation; $p \leq 0.05$

Table 4. Lactate concentrations in the pre and post 400 meter situations for the active groups in the control situation (G1-400/C) and after plyometrics (G1-400/Post) and passive in the control situation (G2-400/C) and after plyometrics (G2-400/Post).

	Pre (mM)	Post (mM)
G1-400/C	0.96 ± 1.01	9.74 ± 1.35 ^a
G1-400/Post	2.66 ± 1.33 ^b	6.87 ± 1.32 ^{ab}
G2-400/C	0.93 ± 0.59 ^c	10.01 ± 2.23 ^{ac}
G2-400/Post	1.85 ± 1.71	7.89 ± 1.18 ^{abd}

a – significant differences concerning the pre situation;

b – significant difference concerning the G1-400/C group;

c – significant difference concerning the G1-400/Post group;

d – significant difference concerning the G2-400/C group;

$p \leq 0.05$.

concentrations in the situation immediately after the 400meters followed by recovery was significantly lower than the lactate concentrations after the 400m in the control situation. The only difference occurred in the pre 400 meters after active recovery situation, where the lactate concentration was significantly higher than in the control situation of the same. Regarding the intergroup analysis, we highlight the significant difference occurred in the lactate concentrations immediately after the 400 meters followed by the active and passive recovery. The lactate concentrations in this situation for the group which performed passive recovery was significantly higher when compared to the group which performed active recovery. In addition to the analyses mentioned above, the Pearson correlation test was applied in order to determine the associations between the lactate concentrations prior to the 400-meter races and the time of the 400 meters, both in the control and after plyometric activity situations. However, due to the similar behavior between both groups concerning the 400-meter time at the three moments, they were grouped in a single factor for this analysis. Thus, it was possible to observe that the lactate concentrations positively correlated with

the 400-meter time after the plyometric activity ($r = 0.70$); that is to say, the higher the lactate concentrations after plyometrics, the longer the 400-meter time.

DISCUSSION

One of the aims of the present study was to analyse the anaerobic performance of sedentary individuals after a recovery period, both active and passive, subsequent to a session of plyometric exercises and verify whether it was dependent on the metabolic acidosis or muscle stress.

It is known that for a given strength production there is lower recruiting of motor units for the eccentric contraction compared to the concentric one⁽¹²⁾. This higher strength/activation ratio provides high stress on the involved tissues, being considered the main factor in the structural damage of the muscle fibers⁽¹³⁾.

Muscle damage and delayed onset muscle soreness can occur at different magnitude depending on the type of muscle contraction (concentric and eccentric), type of exercise (plyometric jumps, resistance exercises, slope races, etc.), movement velocity (high or low angle velocity), time of interval between sets and level of training of the individual (sedentary, physically active or trained)⁽¹³⁾.

The individuals submitted to the present study had been in hypokinesia status for at least six months before its beginning. One of the ways of checking training levels and hypokinesia status can be established through the O_{2max} values. In a study using sedentary individuals⁽¹⁴⁾, mean O_{2max} values of 47.67ml/kg/min were found, corroborating the one found in the present study, in which the G1 and G2 volunteers presented mean values of 44.05ml/kg/min and 47.88 ml/kg/min, respectively.

Concerning anaerobic performance measured through races at maximum velocity in the 400-meter distance, it was observed that they significantly decreased after performance of the plyometric activity, returning to the '24 hours after performance of plyometry control' values, regardless of the type of removal, either active or passive.

Regarding the types of recovery, active or passive, the highest effectiveness of active recovery compared to the passive on the decrease of lactate concentration after high-intensity exercise is reported in the literature⁽⁹⁾. This superiority can be explained by the increase in the blood flow, and consequently, by the increase in lactate transportation to the heart and the skeletal muscles, sites which are mentioned as the main ones for uptake of this metabolite. Lactate oxidation mainly occurs in the active skeletal muscles, and in a lower level, in the non-active skeletal muscles during exercise, as well as by the myocardium. Such phenomenon was not found in the present study, regardless of the type of rest performed after the plyometric exercise session. It is known that not habitual eccentric contractions performed at high intensity decrease in about 20% the capacity of the lactate transporters (MCTs)⁽¹⁵⁾. Thus, as the concentrations of this metabolite in the blood depend on its removal capacity from the muscle to the blood, it could explain the same lactate concentrations after plyometrics for both groups (G1 and G2) and, as its removal from the blood to other muscle groups and to the myocardium to be oxidized is dependent on this transportation capacity, it could explain the same behavior for the lactate removal for both groups, since the active recovery exercise intensity used in this study is in agreement with the literature. In a classical study⁽¹⁶⁾,

the authors set intensities between 90 and 100% of anaerobic threshold as being optimum intensities of active recovery, that is, the same intensity used in the present study.

Concerning the performance in the 400/24h, it was seen that it was not affected by the muscle damage caused by the plyometric jumps previously performed. The delayed onset muscle soreness is characterized as discomfort and/or pain in the skeletal musculature which, together with decrease in capacity of generating work, can occur after exercise, being intensified from 24 to 72 hours, and can persist for up to seven days, with its progressive decline after this period^(17,18), which could explain the decline in performance in the 400 meters after the plyometric exercises session. Moreover, the eccentric exercise performed before anaerobic high intensity exercises seems to affect mainly the strength capacity in a delayed manner, while the capacity to generate power is affected more specifically, that is, only at moments after performance of eccentric exercise⁽¹⁹⁾, which could explain the time recovery of the 400/24h not having been influenced by neuromuscular fatigue and by previous lactate concentrations, since these are totally removed from the skeletal musculature between 60 and 120 minutes after exercise⁽⁶⁾.

In the eccentric actions, the amplitude of the electromyographic signal is lower when compared to the concentric and isometric actions concerning the levels of absolute and relative strength, indicating hence lower muscular recruiting in the eccentric actions. It was suggested that this lower activation can be related to some mechanism of neural inhibition; for example, the Golgi tendon organs (GTO)⁽²⁰⁾. When we speak about neural inhibition, the existence of sensorial feedback is evidenced which inhibits the motoneurons release rate during fatigue, justifying that the central mechanisms are important to the maintenance of a given strength level. As mentioned before, this inhibition is originated from the neuromuscular fuses and/or GTOs, or even from type II and IV nervous endings, which are apparently sensitive to the build up of some metabolites at muscular level during exercise⁽²¹⁾. Therefore, neuromuscular fatigue can be a result from alterations in the neural signal which arrives at the muscle, being a translation of a progressive velocity and frequency of voluntary impulses conduction reduction to the motoneurons during the activity⁽²²⁾. Therefore, the intramuscular acidosis is seen as one of the neural signals which induce the neuromuscular fatigue process⁽²³⁾. The decline in motor activities similar to the one performed in the present study can be explained by a cyclical and permanent system of muscular recruiting and muscular metabolites. In other words, the higher the acidosis, the lower the recruiting of motor units and the higher the selective recruiting of type II fibers, making the muscle less resistant to fatigue⁽²³⁾. Thus, especially through the results of positive and significant correlation between lactate concentration after plyometric activity and the 400 meters subsequently performed, we can partly explain the performance decrease in the 400/Post. However, performance decrease in 400/Post cannot be associated only with the metabolic acidosis. In an interesting study where the nature of the alterations in the capacity of producing strength in the impulse for hurdle trespassing, induced by processes intrinsic to the 400 meters with hurdles, demonstrate that, at fatigue conditions, there is significant increase in the loss of horizontal velocity of the gravity center associated with significant increase of the time of contact with the ground,

both in the landing and propulsion phases ⁽²⁴⁾. These phases can be explained by the strong eccentric contractions caused by the hurdle trespassing, which stimulate the stretching reflex triggered by the muscular fuses, increasing the muscle capacity of resistance to stretching. Thus, the reduced stretching is related to a pre-activation, resulting from a pre-setting of the central nervous system, preparing the muscle to resist the impact load ⁽²⁴⁾.

The results of the study mentioned above can be associated with the results of the present study. Although the 400 meters performed in this study are with no hurdling, which minimizes the eccentric actions during the race, the plyometric jumps performed prior to the 400 meters can lead to the same mechanisms previously mentioned, leading to performance decrease in the 400/Post regardless of the blood lactate concentrations found in both groups.

Thus, it is suggested that the decline in the 400/Post com-

pared to the 400/C is due to neuromuscular factors which can be related to alterations of some electromyographic parameters, acting as a protective device against possible deleterious effects of the muscle fiber integrity ⁽¹⁾.

It can be concluded from the results presented that, after the active or passive recovery the performance behavior in the 400 meters after the plyometric exercises session was similar when the two recovery methods are compared. Therefore, it can be suggested that the decline found in the time of the 400/Post is related to the neuromuscular fatigue associated to the intracellular acidosis. Further studies are suggested in order to investigate the capacity of the lactate transporters after eccentric exercises associated with active recovery.

All authors have declared there is not any potential conflict of interests concerning this article.

REFERENCES

1. Ascensão A, *et al.* Fisiologia da fadiga muscular. Delimitação conceptual, modelos de estudo e mecanismos de fadiga de origem central e periférica. *Revista Portuguesa de Ciências do Desporto.* 2003;3:108-23.
2. Rowlands AV, Eston RG, Tilzey C. Effect of stride length manipulation on symptoms of exercise-induced muscle damage and the repeated bout effect. *J Sports Sci.* 2001;19:333-40.
3. Gibson H, Edwards RH. Muscular exercise and fatigue. *Sports Med.* 1985;2:120-32.
4. Rossi LP, Brandalize M. Pliometria aplicada à reabilitação de atletas. *Revista Salus-Guarapuava-PR.* 2007;1:77-85.
5. Barroso R, Tricoli V, Ugrinowitsch C. Adaptações neurais e morfológicas ao treinamento de força com ações excêntricas. *Rev Bras Ci e Mov.* 2005;13:111-22.
6. Higino WP, Denadai BS. Efeito do período de recuperação sobre a validade do teste de lactato mínimo para determinar a máxima fase estável de lactato em corredores de fundo. *Revista Paulista Educação Física.* 2002;16:5-15.
7. Donovan CM, Pagliassotti MJ. Quantitative assessment of pathways for lactate disposal in skeletal muscle fiber types. *Med Sci Sports Exerc.* 2000;32:772-7.
8. Beneke R, *et al.* Effect of test interruptions on blood lactate during consent workload testing. *Med Sci Sports Exerc.* 2003;35:1626-30.
9. Franchini E, *et al.* Nível competitivo, tipo de recuperação e remoção do lactato após uma luta de judô. *Revista Brasileira de Cineantropometria & Desempenho Humano.* 2004;6:7-16.
10. Guedes DP, Guedes JERP. Manual prático para avaliação em educação física. Barueri: Manole, 2006.
11. Miyama M, Nosaka K. Protection against muscle damage following fifty drop jumps conferred by ten drops jumps. *J Strength Cond Res.* 2007;4:1087-92.
12. Eston RG, Lemmey AB, McHugh P, Byrne C, Walsh SE. Effect of stride length on symptoms of exercise-induced muscle damage during a repeated bout of downhill running. *Scand J Med Sci Sports.* 2000;10:199-204.
13. Foschini D, Prestes J, Charro MA. Relação entre exercício físico, dano muscular e dor muscular de início tardio. *Revista Brasileira de Cineantropometria e Desempenho Humano.* 2007, v. 9, n. 1, p. 101-6.
14. Souza, *et al.* Comparação do perfil antropométrico e funcional de praticantes de caminhada da cidade de Maringá, nos anos de 1999 e 2000. *Revista de Educação Física/UEM.* 2002;13:81-8.
15. Pilegaard H, Asp S. Effect of prior eccentric contractions on lactate/H⁺ transport in rat skeletal muscle. *Am J Physiol.* 1998;274:E554-9.
16. McLellan TM, Skinner JS. Blood lactate removal during active recovery related to the aerobic threshold. *Int J Sports Med.* 1982;3:224-9.
17. Tricoli V. Mecanismos envolvidos na etiologia da dor muscular. *Rev Bras Ci e Mov.* 2001;9:39-44.
18. Clarkson PM, Hubal MJ. Exercise-induced muscle damage in humans. *Am J Phys Med Rehabil.* 2002;81:552-69.
19. Nottle C, Nosaka K. Changes in power assessed by the Wingate Anaerobic Test following downhill running. *J Strength Cond Res.* 2007;21:145-50.
20. Aagaard P, Simonsen EB, Andersen JL, Magnusson SP, Halkjaer-Kristensen J, Dyhre-Poulsen P. Neural inhibition during maximal eccentric and concentric quadriceps contraction: effects of resistance training. *J Appl Physiol.* 2000;89:2249-57.
21. Gandevia SC. Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev.* 2001;81:1725-89.
22. Davis M, Bailey S. Possible mechanisms of central nervous system fatigue during exercise. *Med Sci Sports Exerc.* 1997;29:45-57.
23. Matsuura R, Ogata H, Yunoki T, Arimitsu T, Yano T. Effect of blood lactate concentration and the level of oxygen uptake immediately before a cycling sprint on neuromuscular activation during repeated cycling sprints. *J Physiol Anthropol.* 2006;25:267-73.
24. Valamatos, *et al.* Impulsão dinâmica da transposição da barreira. Alterações na capacidade de produção mecânica do complexo músculo-tendinoso provocadas pela instalação da fadiga. *Revista Portuguesa de Ciência do Desporto.* 2005;5:15-30.