Effects of different strengths in the judo fights, muscular electrical activity and biomechanical parameters in elite athletes

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ABSTRACT

The sportive training causes neuromuscular adaptations and metabolic alterations aiming the competition performance. In judo competitions, the number of fights to what athletes are submitted, as well as their respective endurance and intervals are randomized, and these factors may influence the aimed training performance. This study investigated the hypothesis that different fight endurances, 90s, 180s, and 300s could influence the enzymatic and muscular electrical activity, as well as the torque peak production. Before and after each fighting, a blood sampling was collected from each athlete. After and before each fight, they performed five dynamic contractions (90º/s) using an isokinetic Dynamometer (Biodex System 3). Simultaneously, it was recorded the electromyographic signal of the agonist, antagonist and synergistic muscles of the movement assessed. It was observed no alterations in the torque. The AST and ALT enzymes presented an increasing activity in the 90 sec. ($p = 0.0033/p = 0.00059$), 180 sec. ($p = 0.0044/p = 0.0033$), and in the 300 sec. ($p = 0.0044/p = 0.0033$) fights. It was verified an increase ($p = 0.0180$) in the CK activity after the 300 sec. fight. LDH decreased after the 90 sec. fight ($p = 0.0392$). Upon the intermuscular analysis, it was observed an increase in the electromyographic signal of the agonist muscle after the 90 sec. fight ($p = 0.005$), an increase of the antagonist muscle in the 180 sec. fight ($p = 0.0129$), and a decrease ($p = 0.0137$) in the activity of the agonist muscle in the 300 sec. fight. It was observed that the strength in the 300 sec. fight might reduced the injuries in the muscular tissue characterized by a raise in the plasmatic CK, although the injury was not sufficient to detect the fatigue through the isokinetic dynamometry. It can be concluded that the proposed protocol was sufficient to the enzymatic and electromyographic alteration, suggesting metabolic and neural adaptations from stress caused by the judo fights.

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INTRODUCTION

The physical training has as main target to maintain the sportive performance in competitive periods. The plasticity of the muscular skeletal tissue allows its adaptation to several states upon functional demands[1] that along with the training corresponds to the increase in the tolerance to the exercise, provoking adaptation processes in the mechanical, metabolic and electrophysiological features[2], according to the specificities of each modality. The fatigue is characterized by the inability to keep the torque, and it is a limiting factor to the performance in competitions.

The measurement of the variables that indicates the fatigue in sports has been strongly investigated, and among them, the plasmatic serum levels of the muscular enzymes used as an indicator for the injury state and/or damage in the muscular tissue after training periods[3,4], or indicators of the exercise's intensities[5,6].

The alterations in the electrical activity of the muscular fibers submitted to isometric contractions in the fatigue protocols have been investigated[7-9]. But dynamic contractions protocols, mainly in the sportive area, need to be further studied[10-12] when the aim is to investigate electromyographic adaptations of the sportive gesture to specific trainings[13-17], the performance[18], and the quantitative analysis of the muscular fatigue[17].

Despite the fatigue is a shared experience in the sportive area, the process involved in such mechanism still demands further studies due to the divergent results found mainly as to the analysis of the electromyographic signals[13,19].

Before anything, the performance of the sportive gesture depends on the strength training. A strength optimization aims the activation of the agonist muscles with or without low antagonist co-activations[14,20,21], as to spare unnecessary energetic expenditures. Thus, the muscular co-activation suggests debility in the muscular activation performance to accomplish the motor action[7] that suggests damages in the functional performance[17].

In a judo competition, the number of fights to what athletes are submitted and their respective endurances and intervals are randomized factors that may influence the aimed performance in every fight[22].

It is generally accepted that the increase in the strength production is due to neural factors, and this supports the hypothesis that it occurs adaptations in the electrical muscular activity upon physical strengths. In the literature, it is found that these alterations in the fatigue status appear in divergent ways. On the other hand, the relationship between the increases in the activity of the plasmatic muscular enzyme related to the increase in the strength caused by the exercises or to the muscular injury status has long been established.

The stress in different judo fight endurances could be sufficient to cause enzymatic alterations that would be an indicator of the increasing strength caused by the exercises and/or the status of

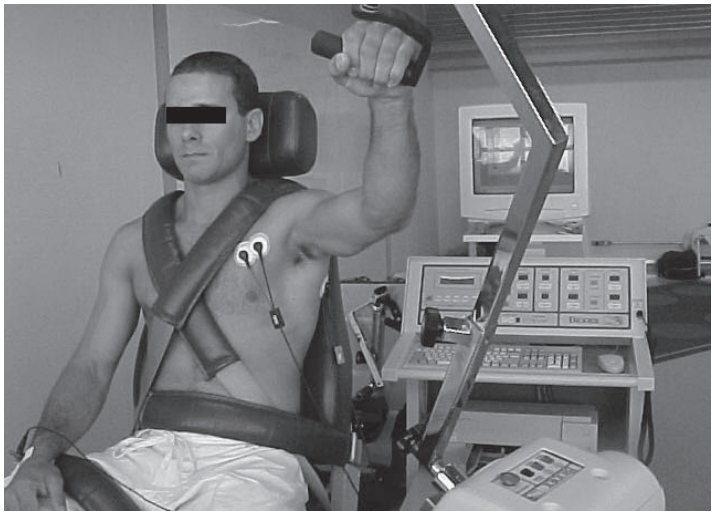

Fig. 1 *– Simultaneous collection of the electromyographic signals using an EMG System do Brazil electromyography as well as the torque peak using the Biodex System 3 isokinetic dynamometer*

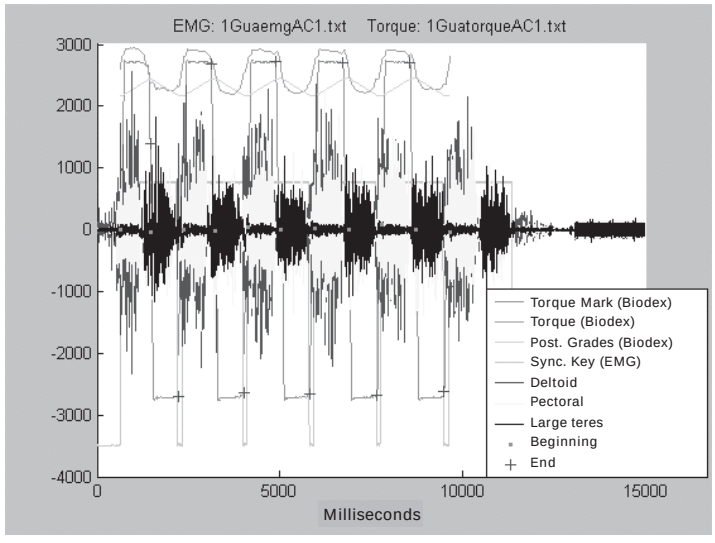

Fig. 2 *– Recording of a simultaneous collection of the electromyographic signals and the torque of the anterior deltoid, large teres, and the upper pectoral muscles synchronized in the time domain through a key placed on the dynamometer arm and activated on the beginning of the movement*

muscular injury. Those same strengths could change the electromyographic activity of the assessed muscles, and this alteration is characterized by a muscular co-activation that would compromise the production of the strength in the upper limbs of the athletes.

The increase in the enzymatic levels into the plasma could change the electrical activity of the muscle, and maybe those biochemical parameters would have a correlation to the indicators of the electrical signals of the muscular fatigue, and this could characterize a deficient status of the athletes' fitness for the competition due to a lack of or excessive training.

The aim of this study was to verify if different judo fighting endurances, respectively of 90s, 180s, and 300s could change the enzymatic metabolism as well as the athletes' electromyographic activity, thus damaging the torque maintenance, which is an indicator of the muscular fatigue.

MATERIALS AND METHODS

Subjects

Twelve 22 ± 4.5 years male elite judo athletes who voluntarily signed the consent term to the research. This study was approved by the UNIVAP Research Ethics Committee under the protocol no. A017/2003/CEP.

Fight protocol

All subjects were divided in pairs, according to their body weight (kg) measured on a Fillizola scale. Three 90s, 180s, and 300s endurance fights were performed between the pairs at the following hours: 8am, 1pm, and 6pm in alternate days, with a 72 hours interval. Athletes were orally encouraged by the coach to fight at maximal intensity. All subjects were assessed immediately before and after each fight.

EMG protocol

An 8-channel EMG System do Brasil electromyography was used, and the EMG signals were collected from the following muscles: anterior deltoid, large pectoral, and large teres muscle, respectively: agonist, antagonist, and synergistic of the movement. A local (trichotomia) and the local asepsis on the skin made with cotton, alcohol, and gel. A ground electrode was placed on the non-dominant arm, and bipolar surface electrodes were fixed in the medium portion of muscles. The interval between the signal takes was 30sec. from the order of execution of the movement.

Isokinetic dynamometer protocol

In order to measure the torque peak, it was used a Biodex System 3 isokinetic dynamometer. All individuals remained seat on the chair with the rotation angle aligned to the glenohumeral region. The individuals performed five flexion movements and of shoulder extension at a 90º/sec. velocity, thus optimizing the collection of the electromyographic signal for dynamic protocols (see Gerdle[18]).

The movement started with the dominant arm extended anterior to the body in a 90º angle from the fundamental anatomic positioning, followed by a shoulder flexion up to reach a 70º. Prior to the trial, an adaptation to the equipment was performed along three days. The motor action was based on the judo's sleeve grip frequently performed by an athlete. As to the equipments' synchronization, a key connected to the dynamometer arm was used as a timer device to the beginning of the movement and this allowed the simultaneous collection of the electromyographic signals and of the dynamometer (figure 1).

TABLE 1
Determination of the torque peak (TP/) before (AL) and after (DL) the 90sec., 180sec. and 300sec. fights, performed in the isokinetic test performed at 90ºs

n = 12	AL	DL	AL	DL	AL	DL
PT ($N.m^{-1}$)	70.54 ± 26.8	70.94 ± 29.11	65.7 ± 24.57	71.77 ± 27.10	66.55 ± 25.33	64,36 ± 28.32

T he minimum significance level set was $\alpha = 0,05$. $N.m^{-1}$ = newtons per meter.

Signal processing protocol

Signals were processed using MATLAB Math Works 6.1 software. The rough data were collected at a 15sec. interval, and at 2 KHz sampling frequency, which was filtered through a 20 Hz high-pass filter and a 250 Hz low-pass filter, and rectified by the F.F.T. – Fast Fourier Transformation. In order to attain the same time resolution in the EMG (0,05 ms) signals as well as in the torque (10 ms), the torque signal was interpolated using the Cubic Spline at 0,05 ms intervals. The first phase of each concentric contraction was analyzed from the beginning of the movement.

The EMG signals were normalized by the basic line, and they were analyzed in the time domain through the total calculation of the absolute EMG signal value (iEMG) for each contraction using the below showed equation, where T is the endurance of the contraction, and f(t) is the electromyographic signal.

$$iEMG = 1 / T \int_0^T I f(t)\, dt$$

Protocol to the blood sampling collection and biochemical dosage

It was collected 10 ml of the individual's blood using a disposable syringe and needle. All sampling were immediately centrifuged in a 4ºC JOUAN CR3I centrifuge. The activity of the LDH, CK, AST, and ALT enzymes were verified using a HITACHI UV-2001 spectrophotometer and the Analisa Diagnostica kit through the colorimetric method.

Statistical analysis of data

To the statistical analysis of the data, the non-parametric WILCOXON test for paired data was used with a significance level of $\alpha = 0.05$.

RESULTS

No difference was found in the torque peak (TP) before and after different fight endurances, as it can be seen in the values shown on table 1.

Upon the AST and ALT activities, it was verified an increase related to the increase in the 90sec, 180sec, and 300sec endurance fights, as it can be observed in the values shown on table 2 and figure 3.

It was observed a decrease in the LDH only after the 90sec fight, as it can be observe on table 2, and an increase in the CK activity after the 300sec fight, according to figure 3c.

It was observed an increase in the iEMG (mV) signal after every muscle in the 90sec fight. But such increase was significant (p = 0.005) in the agonist muscle as it can be seen in table 3.

After the 180sec fight, the agonist muscle did not present any alteration, keeping the resting values, but it was observed an increase (p = 0.0129) in the iEMG (mV) signal in the antagonist muscle, and an increase, although non-significant, in the synergistic muscle.

In the 300sec fight, it was verified a decrease in the activity of the agonist muscle (p = 0.0137), and a decrease (p = 0,05), although non-significant, in the activation of the antagonist muscle, and the synergistic muscle kept its mean resting values.

It can be observed a decrease in the intensity of the electrical activity of the muscles analyzed from the endurance of the fights, as it can be observed in figure 4.

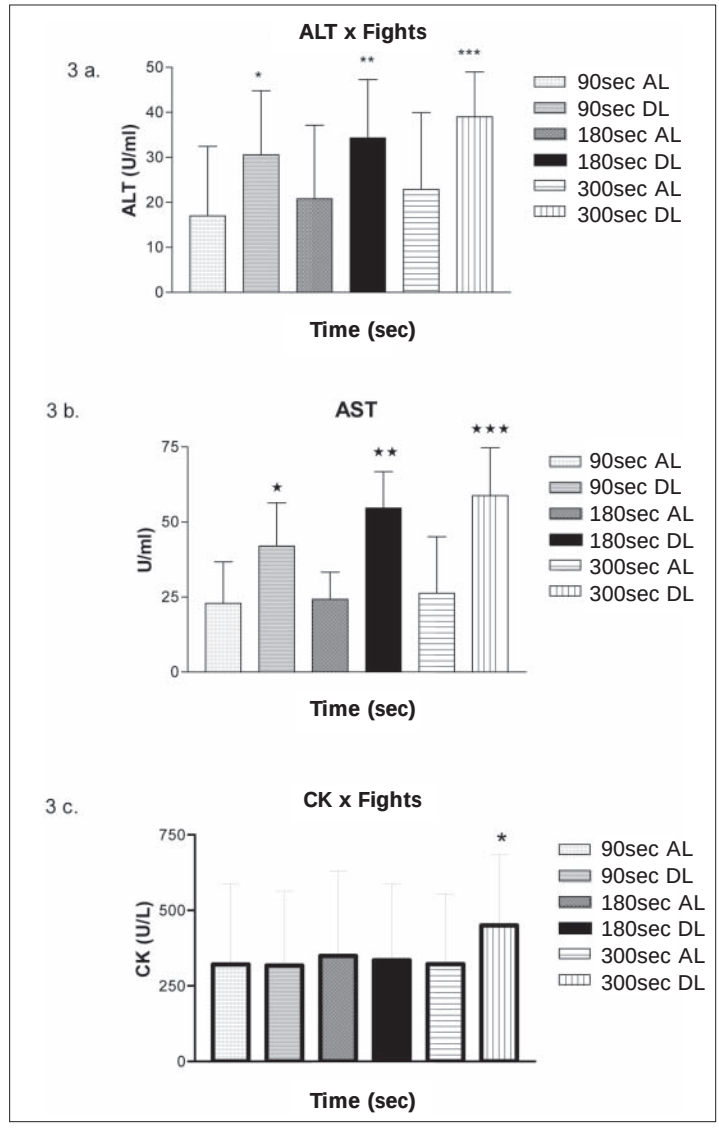

Fig. 3 *– **3a)** Activity of the transferase alanine enzyme (ALT), and **3b)** transaminase aspartate (ALT) and creatinine kinase (CK) before and after the 90sec. 180sec., and 300sec. fight*

TABLE 2
Activity of the enzymes: aminotransferase aspartate (AST), aminotransferase alanine (ALT), creatinekinase (CK), and dehydrogenase lactate (LDH) before (AL) and after (DL) the 90sec 180sec, and 300sec fights

n = 12 (x ± D)	90sec fight		180sec fight		300sec fight	
Enzymes	AL	DL	AL	DL	AL	DL
AST (U/ml)	22.9 ± 13.8	42 ± 14.4*	24.2 ± 9.0	54.6 ± 12.1*	26.3 ± 12.1	58.8 ± 15.9*
ALT (U/ml)	17 ± 15.4	30.6 ± 14.2*	20.8 ± 16.3	34.3 ± 12.9*	22.91 ± 17.03	39 ± 9.9*
CK (U/L)	320.9 ± 266.6	317 ± 247.2	349.2 ± 281.4	334.7 ± 254	322 ± 233.3	450.5 ± 233.3*
LDH (U/ml)	184.7 ± 70	165.6 ± 66.1*	186.3 ± 93.9	175.9 ± 94.6	189.3 ± 77.9	177.1 ± 60.5

* Minimum significance level set was $\alpha = 0.05$. After the 90sec fight, it was verified an increase in the AST (p = 0.0059) and in the ALT (p = 0.0033), and a decrease in the LDH (p = 0.0392). After the 180sec fight, it was observed an increase in the AST (p = 0.0033) and in the ALT (p = 0.0044). After the 300sec fight it was verified an increase in the AST (p = 0.0033), in the ALT (p = 0.0044), and in the CK (p = 0.0180).

TABLE 3
Integral activity of the electromyographic signal (iEMG) in the agonist, antagonist, and synergistic muscles of the muscular (puxada de manga) action in the judo, simulated in the isokinetic dynamometer before (AL) and after (DL) the 90sec, 180sec, and 300sec fight

n = 12 (X ± DP)	90s fight		180sec fight		300sec fight	
iEMG (mV)	AL	DL	AL	DL	AL	DL
Agonist	4.04 ± 2.42	9.7 ± 8.02*	4.46 ± 2.61	4.45 ± 2.72	5.34 ± 2.73	3.16 ± 1.23
Antagonist	3.35 ± 1.77	4.89 ± 5.45	2.79 ± 1.75	4.72 ± 2.64**	3.14 ± 1.45	2.10 ± 1.03***
Synergistic	7.14 ± 3.8	8.74 ± 5	7.1 ± 4.85	7.72 ± 6.61	5.55 ± 1.96	5.38 ± 2.44

* The minimum significance level established was $\alpha = 0.05$. After the 90sec fight, it was verified an increase in the iEMG signal of the agonist muscle ($p = 0.005$); after the 180sec fight, it was observed an increase in the iEMG signal of the antagonist muscle ($p = 0.0129$), and after the 300sec fight it was verified a decrease in the iEMG signal in the agonist muscle ($p = 0.0137$).

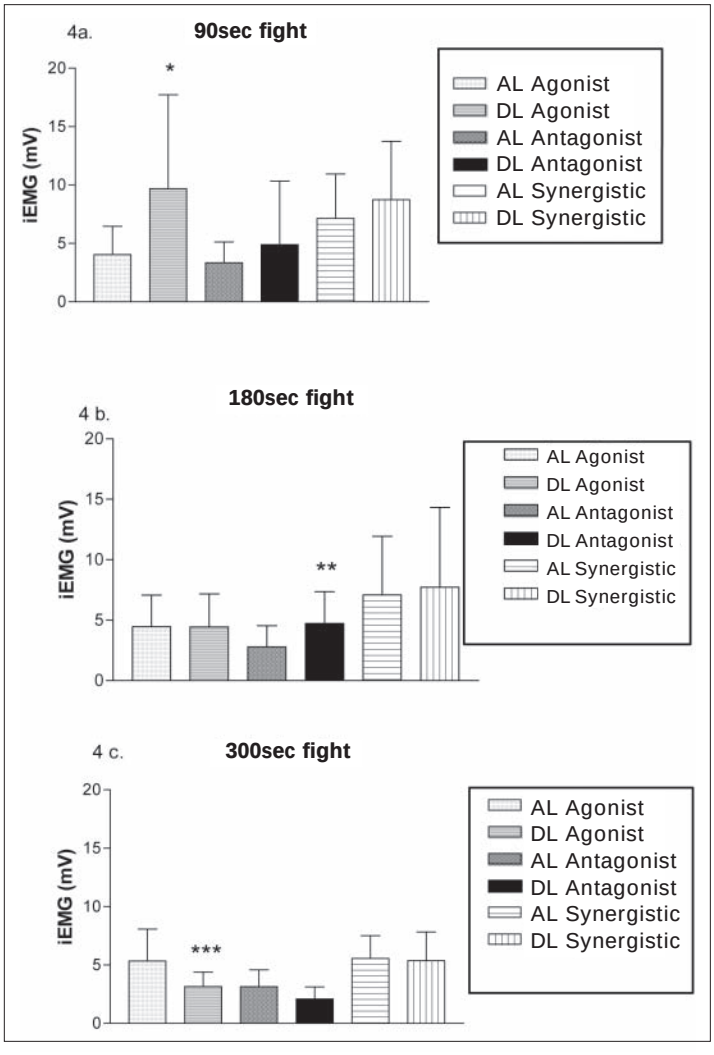

Fig. 4 – ***4a)*** *iEMG (mV) activity of the agonist, antagonist, and synergistic muscles of the movement assessed before and after the 90sec fight.* ***4b)*** *iEMG (mV) activity of the agonist, antagonist and synergistic muscles of the movement assessed before and after the 180sec fight.* ***4c)*** *iEMG (mV) activity of the agonist, antagonist, and synergistic muscles of the movement assessed before and after the 300sec fight.*

DISCUSSION

The mean values seen on table 1 suggest that the stressing fights to which individuals were submitted was not enough to change the torque peak of muscles, and/or the resting time between the strength and the signal collection posed a sufficient recovering time to the ability to generate strength in the analyzed muscles.

The main biomechanical phenomena were verified in the simultaneous analysis of the muscles involved in the movement, which was already observed by other authors[23].

Despite de 90sec fight was not sufficient to decrease the torque, it caused neural adaptations observed in the increasing iEMG signals of the agonist muscle, and this may represent an adaptation dynamics observed in other studies using fatigue protocols[18].

Opposed to Kellis[17], it could be verified in the 180sec fight an increase in the amplitude of the iEMG signal of the antagonist muscle, suggesting the muscle has an important role for the stability of the joint as a motor controlling of the regulation, and to the protection of the joints in a fatigue process[24]. An explanation for such increase can be attributed to a central nervous system attempt to compensate the muscular fatigue through the increase in the number of motor units available[25].

If the antagonist muscle is able to increase the strength upon the accomplishment of repetitive strengths, it could represent an additional load to attend the agonist muscle and keeping the strength, contributing to the resulting decrease in the involved joint and in protecting it, and this must be a parameter to be considered when it is aimed the training performance in sportive gestures.

On the other hand, such co-activation suggests debility in the activation performance of the muscles to the accomplishment of the motor action, and this could pose a damage to the functional performance[24]. The optimization of the strength training aiming the athlete's performance has as purpose the activation of the agonist muscles with or without low antagonist co-activation[16,22].

The neuromuscular adaptations related to the endurance of fights suggest that it was necessary a higher energetic demand to perform the same strength observed with the AST and ALT activity regulated by the energetic demand necessary to the strengths.

Even with a predominance of the glycolytic way of the judo fights, the muscular proteins, the alanine and the glutamine were recruited to the ATP synthesis, suggesting in this study that the AST and ALT were biomarkers to the intensity level of the exercise[6].

The increasing iEMG (mV) of the agonist muscle after the first fight, as well as the co-activation after the second fight can both be explained by the dynamics of the neuromuscular adaptation to perform the work following a gradual increase in the energetic demand characterized by the ASL and ALT activities, as it can be seen in figures 3a and 3b.

Analyzing the LDH, it was verified a decrease in every fight protocol, but it was significant only after the 90sec fight, and this can be explained by the potentialized capability of the aerobic resistance that training athletes are submitted to. It is generally accepted that the endurance training and the reduced intensity cause an accentuated reduction in the LDH activity[26,27]. Thus, the regulating factor to the increasing LDH is the pyruvate and NADH offering. Maybe the individual's training condition made the level of the substrates did not exceed the PDH capability to metabolize the pyruvate and/or the alpha-glycerophosphate system to supply the energetic demands[28]. As the LDH is a regulating enzyme, maybe a negative modulator that could reduce the cytosolic NADH would cause such inhibition.

In the 180sec. and 300sec. fights, the potentialized increase of the lactate due to the intermitting feature of the judo fight maybe has been enough to not allow a significant decrease in the LDH activity[29].

In the 300sec. fight, the iEMG (mV) signals presented a decrease in the agonist and antagonist activity, and at the same time, it kept the synergistic activation as the resting values.

The decreased activation of the agonist muscle may suggest a deficient motor action, combined with the synergistic actuation that should be stabilizing the movement instead of acting as primary motor.

These data show the importance of studies aiming the synergistic activity of the movement, as to the effective strength training, besides the activation of the agonist muscle and the reduction in the co-activation, an optimization in the synergistic muscle would be able to exert an important role in the increase of the torque system produced on the joints[12].

It was verified that the 300sec fight protocol caused physiological adaptations with the increasing CK, and this damaged the motor units and/or the spreading of the muscular electric signal.

The lack of alteration in the CK activity in the first two fights can be explained by the inter-individual variations expressed by the high values of the standard deviation, making difficult to analyze the alterations caused by the exercise.

The increase in the CK after the 300sec fights can be a result of the intermittent submaximal strength[6] to what individuals were submitted, with an increase in the temperature and in the metabolic rate and a predominant eccentric contraction, thus increasing the release of that enzyme[30]. The intensity of the exercise and its endurance are directly related variables to the alterations in the plasmatic CK concentrations[30]. While the strength increases, the sarcolemma's porosity and/or the rupture of the membrane provokes the passage of such cellular proteins to the plasma, and this is a phenomenon observed in this study after the 300sec fight.

The decrease in the iEMG of the primary motor muscle of the movement associated to the increase in the plasmatic CK during the 300sec fight suggests that the strength was sufficient to cause an increase in the permeability and/or micro-injuries in the sarcolemma, and this provoked an alteration in the polarity, damaging the activation and spreading of the electrical signals, resulting in a decrease in the electromyographic signal amplitude.

Such phenomenon can result from the presence of metabolic by-products caused by the exercise, provoking an alteration in the membrane's potential through the decrease in the pH caused by the acidification resulting from the injured muscular fiber, what could lead to damages to the cellular activation[31].

The fights athletes were submitted to in this study constitute a limiting factor, because despite the stimuli given by the coach and every official rules of the competition were followed, they were simulated to the accomplishment of this trial. So, athletes would present different and individual physiological responses in an official competition.

Despite of the increase which was verified in the strength intensities through the AST and ALT activity, the mechanics of the muscular actions used in different fighting endurances is another aspect to be considered, once the fights are different as to their actions, limiting the functional assessment of the motor actions used by athletes in the fights.

The adaptations in the EMG signals observed in this study evidence the need of a simultaneous analysis as to the intermuscular coordination. The results of the AST and ALT activity related to the respective fight endurances confirm the utilization of such enzymes as strength intensity biomarkers, indicating a dynamics in the metabolic adaptation to the gradually increased strengths.

The alterations in the electrical and biochemical parameters in the proposed protocol suggest that the intensity of the strength is related to the alteration in the electrical activity of the skeletal muscular tissue, and the effects of the 300sec fight provoked an increase in the CK activity, and a decrease in the electrical activity of the agonist muscle. This suggests that the decrease in the amplitude of the primary motor muscle signal is associated to possible micro-injuries in the muscular membrane that damage the activation and spreading of the electromyographic signal.

CONCLUSION

It can be concluded that the stress of the 90sec, 180sec, and 300sec fights, despite of being unable to change the torque capability was stimuli sufficient to cause alterations in the enzymatic and muscular electrical activity that can be damaging to the athletes' performance.

All the authors declared there is not any potential conflict of interests regarding this article.

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