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ORIGINAL ARTICLE

Correlation between acute muscle damage and oxidative protection enzymes during different aerobic exercises

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Abstract

Different types of aerobic exercise can cause different disorders of homeostasis. This cross-over experiment aimed to determine the muscle fatigue and the antioxidative protection of female basketball players following a load caused by three different aerobic-type exercises (low-intensity continuous, high-intensity continuous, and high-intensity interval training). Twelve female basketball players (age 17.7±4.3 years; weight 67.3±9.8 kg; height 178.0±7.4 cm) voluntarily participated in the study. A wash-out period of 7 days between single sessions of different training was provided. Venous blood was drawn right before and immediately after each exercise session. The parameters that were analyzed are markers of muscle damage and enzymes of antioxidant protection. As a marker of muscle damage, myoglobin ($F=2.884$; $p=0.065$) and lactates ($F=5.254$; $p=0.008$) have higher values and statistically significant differences between training types. Creatinine shows higher values after each training session ($F=4.053$; $p=0.022$). Results of enzyme activity for oxidative protection show statistically significant differences between groups for catalase ($F=5.811$; $p=0.005$) with different types of training intervention. At the beginning of the preparatory period, parameters of acute muscle damage values are high. During the season, in response to different types of training, those parameters decrease in response to the body's adaptation to exercise-induced stress. Training leads to maintenance of physiological balance in the body and oxidative stress is not a necessary phenomenon of high aerobic training load. The inclusion of antioxidant protection enzymes decreases as the body adapts to a certain type of exercise.

Keywords: aerobic exercise · muscle damage · antioxidant protection

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Introduction

Professionalization in sports carries the risk of disrupting vital functions in the body. Without adequate training planning that contains the basic principles of the training process (frequency, intensity, and duration), in combination with inadequate preparation of athletes and their objective capabilities, pathophysiological changes in the body may occur. To avoid chronic fatigue, overtraining, or injury, there is a whole series of physiological and biochemical markers that can be monitored, which facilitate and enable the planning and programming of the training process. However, different types of aerobic exercise (low-intensity continuous, high-intensity continuous, and high-intensity interval training) can cause different disorders of homeostasis and can lead to temporary muscle fatigue. Serum enzyme activities such as creatine kinase, troponin, and myoglobin are used as indirect markers of exercise-induced muscle damage (Paulsen et al., 2012, Brancaccio, Lippi, & Mafulli, 2010). The more markers we take into consideration, the more precise the data will be (Marić, 2018). When it comes to markers of cellular bioenergetics, in addition to lactate, which is used to assess physical fitness as a key energy-facilitating compound in cellular bioenergetics (Ostojić, 2019), the literature often contains creatine, creatinine, and guanidino-acetic acid, as a natural precursor of creatine in conditions when creatine is in deficit (Ostojić, 2017).

In the oxidation process, free radicals are created that have positive physiological functions. However, when the state of the organism is such that there is an increased production of free radicals with a reduced possibility of their removal and neutralization, it is a state of oxidative stress that can lead to unwanted changes in the human body (Chaudhary at al., 2023). Oxidative stress is defined as the imbalance between reactive oxygen species, reactive nitrogen species, and antioxidative stress (Marić, 2020). The occurrence of oxidative stress, as a result of physical activity, leads to the activation of the antioxidant system, providing protection for the body against free radicals during repeated physical activities. In this way, the physiological capacity of the body will expand or adapt, which will ultimately lead to the improvement of the athlete's health. Increased production of reactive oxygen species during and after training is a sign of increased activity of antioxidant protective mechanisms (Fisher-Wellman & Bloomer, 2009). Different types of physical activity differ from each other in terms of energy needs, the level of oxygen consumption, as well as mechanical tissue damage during physical activity depending on the free radicals created (Fisher-Wellman & Bloomer, 2009). Increased production of reactive oxygen and nitrogen species, as well as oxidative stress, occur even among top athletes due to maximum loads, regardless of the type of energy demand of the sport itself (Stanković & Radovanović, 2012). In general, it can be said that most studies indicate an increase in oxidative stress in response to physical exercise (Urso & Clarkson, 2003). Antioxidant protection is provided by compounds that have the role of protecting the body from oxidative stress by preventing the formation of free radicals, that is, by eliminating them if they already occur. To avoid cell damage, most biological systems have developed a battery of antioxidants, enzymes such as superoxide dismutase, catalase, and reduced glutathione, which represent the basic defense of the organism against oxidative stress during physical activity (Dekany et al., 2006) and are mostly used when examining oxidative stress after exercise (Urso & Clarkson, 2003). Determining the antioxidant status in the body is especially recommended for people who are exposed to increased mental and physical stress, i.e. athletes, precisely because training causes increased consumption of oxygen, especially from skeletal muscles, and thus increased generation of free radicals (Pešić et al., 2009). This cross-over experiment aimed to determine muscle fatigue and the antioxidative protection of female basketball players following a load caused by three different aerobic-type exercises. It is expected that different episodes of aerobic training will lead to an increase in markers of muscle damage and to an increased activity of enzymes and antioxidant protection in relation to the level of exercise.

Method

Twelve female basketball players (age 17.7±4.3 years; weight 67.3±9.8kg; height 178.0±7.4cm) voluntarily participated in this cross-over experiment. All of the subjects had at least 5 years of professional training experience and they were also part of the national team for at least a year. The study was planned following the ethical standards of the Helsinki Declaration. Venous blood was drawn right before and immediately after each of the three exercise sessions. A wash-out period of 7 days between single sessions of different training was provided to prevent the residual effect of interventions across study periods. The type of each training episode is detailed in Table 1.

Table 1. Content of each training episode

AEROBIC EXERCISE	Warm-up	Target	Cool down
1st training - low-intensity continuous (LIC)	10 minutes of running at an easy pace 10 minutes of dynamic stretching 10 minutes of athletic running exercise	30 min of running at a moderate pace with 60-70 % VO_{2max}	10 minutes of stretching and relaxation exercises
2nd training – high-intensity continuous (HIC)		20 min of high-intensity running at 85 – 95 % VO_{2max}	
3rd training – high-intensity interval training (HIIT)		Running 3x3 min at 90-95% VO2max, rest between sets is 3 min at 50-60% VO_{2max}	

Training intensity was controlled with a pulse monitor (polar r810, polar electro oy, Kempele, Finland). Overall, 7 parameters of muscle damage markers were taken: creatine kinase, creatine, creatinine, guanidinoacetic acid (GAA), myoglobin, troponin, and lactate. The activity of protection enzymes against oxidative damage is determined through 3 enzymes: superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH). The venous blood was drawn and centrifuged within the next 10 minutes at 3000×g, with serum separated and analyzed for serum creatine kinase, creatine, creatinine, GAA, myoglobin, troponin, and lactate using a modified LC-MS/MS (1200 Series LC System, Agilent Technologies Inc., Santa Clara, CA, USA), while SOD, CAT, and GSH were analyzed with UV/vis-spectrophotometer (Specord S 600, Analytik Jena AG, Jena, Germany).

The data were processed in the statistical program package - Statistical Package for Social Science (IBM SPSS 20.0, SPSS ID: 729225). The differences between the obtained sampling results were calculated using the multivariate analysis of variance MANOVA and the Post Hoc test between training sessions. The minimum condition for the existence of a statistically significant difference is p (significance level) less than or equal to 0.05.

Results

The anthropometric characteristics of the respondents are shown in Table 2.

Table 2. Anthropometric characteristics of participants

Variable	M±SD [MIN-MAX]
Age (decimal years)	17.75±4.07 [14 - 27]
Body height (cm)	177.96±7.01 [165 - 189]
Body weight (kg)	67.32±10.43 [49 - 80]
Body mass index BMI (kg/m^2)	21.68±2.59 [18 - 25]
Relative fat mass (%)	27.43 ±8.07 [13 - 42]
Relative muscle mass (%)	31.74 ±4.32 [22 - 38]

As a marker of muscle damage, myoglobin ($F=2.884$; $p=0.065$) and lactates ($F=5.254$; $p=0.008$) have statistically significant differences between training types. Creatinine, as a surrogate marker of creatine utilization and kidney function, shows statistical significance at the 0.05 level ($F=4.053$; $p=0.022$). Troponin did not show sensitivity to any type of training, it remained unchanged. The creatine kinase marker showed a difference after the first and third training, however, when the interaction of the factors of training type and sampling time was observed, it did not show a statistically significant difference. Creatine showed similar movements in all three types of training and remained almost unchanged, as is the case with guanidinoacetic acid. The enzyme activity for oxidative protection shows statistically significant differences between groups only for catalase ($F=5.811$; $p=0.005$) with different types of training intervention, while superoxide dismutase and

reduced glutathione did not show a statistically significant difference. The results are shown in Table 3.

Table 3. Biochemical parameters and parameters of oxidative protection

Variable	LIC		HIC		HIIT		F	p
	before	after	before	after	before	after		
Troponin (ng/ml)	0.2±0	0.2±0	0.2±0	0.2±0	0.2±0	0.2±0	0.000	-
Myoglobin(ng/ml)	25±5	47±20*	39±14	83±31*	32±10	52±20*	2.844	0.065
Lactates(mg/dl)	15±7	30±10*	16±6	14±5	19±12	29±13	5.254	0.008
Creatine kinase (U/I)	145±71	190±109*	380±167	279±187	279±187	356±219*	1.992	0.145
Creatine(μmol/l)	22±5	24±6	22±4	22±4	22±4	22±4	0.344	0.710
Creatinine(μmol/l)	69.8±10	86.8±12*	67±8	69.2±9	69.3±4	76.3±10	4.053	0.022
GAA(μmol/l)	2	1.9	1.9	1.8	1.9	1.8	0.119	0.888
SOD(nmol/l)	20	21	19	14	15	18	0.786	0.460
CAT(nmol/l)	6.4	3.3*	3.7	7.1*	4.2	6.4	5.811	0.005
GSH(μmol/l)	83246 ±8921	88557 ±7900	82517 ±7043	84355 ±6970	91416 ±5224	90804 ±8052	0.960	0.388

Discussion

Muscle fatigue due to physical exercise has become one of the most important topics in sports science (Paulsen et al., 2012). Exercise parameters such as intensity, duration, or average energy expenditure increase typical markers of skeletal muscle damage (Park et al., 2014). During the examination of the first training, when continuous low-intensity training, the difference between the arithmetic means of the results before and after the training of all analyzed variables, except for troponin, creatine, and GAA, was determined. Based on the increased values of myoglobin, lactate, creatine kinase, and creatinine after training, we can state that this type of training caused muscle fatigue. These results indicate basketball players who are in the process of basic preparations. Analyzing the results before and after high-intensity training, we determined that the difference between the arithmetic means of the results before and after training of all analyzed variables, except for myoglobin, is not statistically significant. These results indicate a gentle introduction of the body into the training process, given that the second sampling was done seven days apart. Interestingly, the troponin values were unchanged in both types of training, while the biggest differences in the mean values were observed in myoglobin. Also, creatine kinase values are two and a half times higher at the beginning of the second training, which is above the reference values for this marker because increased values of creatine kinase activity in the serum are considered an indirect marker of muscle fiber damage (Bloomer, 2007). For interval training, the difference between the arithmetic means of the results before and after the training of all analyzed variables was determined, except for troponin in favor of the second measurement, that is, for GAA in favor of the first measurement in all three training sessions, but it is statistically significant only for the myoglobin and creatine kinase variables. The initial values of myoglobin and creatine kinase measured before the third training session were lower compared to the initial sampling at seven days, therefore statistically significant differences were obtained after treatment, and the highest values for myoglobin and creatine kinase, as well as for lactate.

By analyzing the interaction of the factors type of training and sampling time (before and after each training), it is observed different results compared to the markers we analyzed. The troponin value was unchanged in all three cases and before and after each training session. Previous research on troponin values shows different results with regard to physical activity. Research has mainly focused on long-distance runners, marathoners, and ultramarathoners, as well as on triathletes and cyclists (Bauer et al., 2016), where, according to the results, troponin values are generally elevated. Sports games and team sports, where there are intermittent work sessions during the game, such as basketball, do not show differences in troponin values (Carranza-García et al., 2011). In our research, there was no difference in troponin values. The main drawback of our study may be the sampling time, whether it was four or six hours (Sorichter et al., 1997) per activity, which according

to some authors is most important for testing troponin levels (George et al., 2004).

The presence of myoglobin shows a clear difference between all samples. According to Sorichter et al. (1997), the best time for myoglobin sampling is two hours after activity, Park et al. (2014) believe that it is immediately after and two hours after activity, as well as that the complete recovery of myoglobin is seven days later. It also emphasizes that myoglobin is released from injured tissue faster than creatine kinase, reaching an earlier peak plasma concentration and returning to baseline. In our research, the myoglobin level increased above the reference values during each training session, which indicates that muscle fatigue is visible with each training session, even after the first one, due to the initial phase of preparation, and in the other two training sessions, due to the intensity of exercise and the cumulative effect of previous training sessions.

Lactates are not the main metabolic cause of fatigue or muscle pain, but other metabolites (salts), but since they are easily measurable, they can be used very successfully as a parameter (Fratrić & Nićin, 2006). In our research, observing the overall influence of the interaction of factors, we conclude that there is a statistically significant difference with regard to the type of aerobic training and the sampling time and that there is an increase in lactate concentration after high-intensity training, as has been the case so far (Cipryan, Tschakert, & Hofmann, 2017).

The serum creatine kinase level is different between low and high-intensity continuous training and between high-intensity and interval training and is statistically significant, which is most likely due to either a cumulative effect of training during the preparatory period (Bauer et al., 2016) or an elevated value caused by interval training. training. Park et al. (2014) state that during exhausting training, the peak concentration of creatine kinase is eight hours after the activity, and in eccentric-type activities, the elevation is between the second and seventh day after physical exercise. The release time of creatine kinase from plasma depends on the type, duration, and degree of training (Yamin et al., 2010). Scientists state that the ideal time for sampling serum creatine kinase levels are one day after activity (Neubauer, König, & Wagner, 2008), therefore the main drawback of our results may be that sampling immediately after training is an insufficient indicator of the state of the organism and may it is not a consequence of the applied training, but a reaction to the training before it.

Analyzing the obtained results of testing markers of cellular bioenergetics: guanidinoacetic acid, creatine, and creatinine, we can conclude that the results of our study match the latest research. One of the future markers, which is in the process of research and discovery, and which could replace the other, so far known markers of muscle damage, is guanidinoacetic acid. As a natural precursor of creatine, when the availability of creatine is unhindered, its role as a substrate is negligible, while, on the other hand, in case of creatine deficit, it can completely saturate creatine kinase and act as a replacement phosphagen (Ostojić, 2015). The results of a study that investigated the effect of endurance training (treadmill running "to failure") and repetitive strength training (bench press "to failure") on the concentration level of guanidino-acetic acid, creatine, and creatinine (Štajer et al., 2016) indicate that a decrease in GAA levels was observed after exercise "to failure" with lower values after running training, in both men and women. Creatine levels increased with running, while strength training had no effect. Creatinine level was elevated after both trainings, a significantly greater difference was noted after running training. A negative linear correlation was observed after exercise between guanidino-acetic acid and creatinine and after strength training and running training. The authors emphasize that longer exercise, which includes large muscle groups, leads to a greater reduction of guanidino-acetic acid immediately after exercise due to greater involvement of skeletal muscles, which are the main organ of creatine utilization. The conclusion of this study is precisely that in the future the serum level of guanidinoacetic acid can be used as a new biomarker of fatigue in a physically active population. In our case, elevated creatinine values and decreased guanidino-acetic acid values were recorded in all three training sessions, with a significant difference between training sessions when a greater amount of energy is required. Another study by Semeredi et al. (2018) showed that supplementation with a mixture of creatine and GAA was superior to creatine supplementation alone and that it increased creatine levels and improved upper body strength. GAA appears to be a new supplement that is more effective than creatine in increasing brain and muscle creatine concentrations (Ostojić et al., 2016).

By analyzing the activities of the antioxidant enzymes, superoxide dismutase, catalase, and reduced glutathione are the primary defense against reactive oxygen species generated during exercise

and increase the response to exercise (Steinbacher & Eckl, 2015).

In this study, the changes in the form of a decrease in catalase activity after low-intensity and high-intensity were statistically significant, which coincides with the findings of Fisher et al. (2011). After the first training session, catalase values decreased, probably due to the initial stage of the basketball players' preparations, and after the second and third training sessions, with the improvement of aerobic abilities, catalase activity increased. The irreversible change in catalase activity indicates that in the examined group of basketball players, compensatory mechanisms developed after possible oxidative damage caused by physical exercise.

Reduced glutathione makes no difference between training sessions, except that after interval training, a reduction is noted, that is, it clearly shows that its antioxidant effect is actively involved in the fight against free radicals during the training of this sport.

Small differences in superoxide dismutase activity are consistent with the findings of other studies (Tong et al., 2012), and 30 minutes after interval training, no changes in activity are observed (Zwetsloot et al., 2014). Namely, superoxide dismutase activity changes significantly only after high production of reactive oxygen species, which was not the case here (Marić, 2018).

The limitations of this experiment would be in the sampling time. Namely, some of the markers show sensitivity several hours after the activity, and some can remain elevated for days. Therefore, it may happen that the results that are elevated before training itself, which was the case with creatine kinase, are the result of a previous cumulative effect of training.

Conclusion

The parameters of acute muscle damage are high and the inclusion of antioxidant protection enzymes can change during the season due to different types of aerobic training and adaptation of the body to exercise-induced stress. Training leads to the maintenance of physiological balance in the body and oxidative stress is not a necessary phenomenon of a high aerobic training load.

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