African Journal of Pharmacy and Pharmacology Vol. 5(9), pp. 1218-1222, 8 September, 2011
Available online http://www.academicjournals.org/ajpp
DOI: 10.5897/AJPP11.263
ISSN 1996-0816 ©2011 Academic Journals

Full Length Research Paper

Effects of acute twelve minute run test on oxidative stress and antioxidant enzyme activities

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Accepted 1 June, 2011

The aim of this study was to investigate the effects of 12 min run test on oxidative stress, some antioxidant enzyme activities, and muscle and DNA damage. Twenty two healthy and well trained male athletes were recruited to this study. The12 min run test, cooper test, was applied to the participating athletes. The blood samples were taken before, immediately after and 24 h after the run test, and sera and plasma were separated and then stored at –80°C until the analysis. Serum MDA and NO levels as the indicators of oxidative damage; serum SOD and CAT, plasma GPx activities as the indicators of antioxidant defense system; serum 8-OHdG as the indicator of DNA damage, CK and LHD enzyme activities as the indicators of muscle damage, and glucose level were measured. Serum SOD, NO, CAT, and 8–OHdG levels showed no significant changes before, immediately after and 24 h after the run rest. There was a significant decrease in serum MDA level immediately after exercise, which is returned to baseline level after 24 h–rest period ($p<0.05$). Serum LHD ($p<0.001$) and CK activities ($p<0.05$) and glucose level ($p<0.001$) increased immediately after exercise but these increments returned to pre-exercise level after 24 h–rest period. Acute twelve minute endurance exercise increased CK, LDH and glucose decreased oxidative stress and whereas has no effect on antioxidant capacity and DNA damage in trained young men.

Key words: Antioxidant enzymes, exercise, oxidative stress, twelve minute run test.

INTRODUCTION

Reactive oxygen species (ROS) are continuously generated in all cells of aerobic organisms as part of their normal metabolism. The presence of low concentrations of free radicals and other related species is important for normal cellular redox status, tissue function and intracellular signaling processes (Vincent et al., 2007; Sachdev and Davies, 2008). However, excessive free radicals and ROS damage lipids, proteins and DNA, and compromise cell function (Vincent et al., 2007). The generation of ROS and nitrogen species (RNOS) occurs regularly as part of normal cellular metabolism, and is increased under conditions of physical stress (Sachdev and Davies, 2008).

Exercise has been known to increase oxygen consumption. The 95 to 98% of the oxygen consumed is reduced to water during aerobic metabolism, but the remaining fraction may be converted to oxidative by-products like ROS (Sen and Packer, 2000; Lekhi et al., 2007). During exercise, when volume of oxygen consumption (VO_2) is elevated to 10 to 15 folds above rest, it is very likely that free radicals are produced to a greater extent, compared to the rest (Atalay and Laaksonen, 2002; Lekhi et al., 2007). Reactive oxygen species may damage body tissues if their production is not controlled precisely and adequately. Irreversible oxidative damage to certain vulnerable molecules is thought to contribute to the degenerative process associated with cell breakdown and aging (Diaz et al., 2010; Vincent et al., 2007).

In order to confirm the production or clarify the function of ROS, one often has to search for end products or by–products of radical–induced reactions, examining the reaction "path" of the radicals. The quantitative

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Table 1. Anthropometric characteristics and physical status of the subjects.

Variables	Mean ± SEM
Age (years)	21.45 ± 0.43
Height (cm)	174.18 ± 1.44
Body weight (kg)	67.82 ± 1.65
Body mass index ($kg.m^{-2}$)	22.31 ± 0.41
Waist to hip ratio	0.84 ± 0.01
Body fat (%)	13.39 ± 0.80
VO_{2max} ($mL.kg^{-1}.min^{-1}$)	55.44 ± 2.15

Data are presented as mean ± standard error of mean, n=22.

measurement of minor products of lipid peroxidation, such as malonyldialdehyde (MDA) in tissues, or pentane in exhaled breath, is an attempt to estimate the overall extent of lipid peroxidation (Sachdev and Davies, 2008).

Enzymatic and non-enzymatic defenses such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) and vitamin C, vitamin E, flavonoids, ubiquinone and reduced glutathione are endogenous protective mechanisms of the body in order to combat the deleterious effects of ROS and other free radicals (Diaz et al., 2010). It is now accepted that free-radical generation is enhanced during exhaustion exercise (Lekhi et al., 2007). However, some studies have indicated that lipid peroxidation markers do not change after acute exercise (Pepe et al., 2009; Revan et al., 2010). On the other hand, an acute bout of exercise at sufficient intensity has been shown to stimulate activities of antioxidant enzymes. Training seems to reduce the oxidative stress of exercise, such that trained athletes show less evidence of lipid peroxidation for a given bout of exercise and an enhanced defense system in relation to untrained subjects (Clarkson and Thompson, 2000). This could be considered as a defensive mechanism of the cell under oxidative stress. However, the influence of exercise on free–radical chemistry is not well understood. It is yet to be confirmed whether an adequate biochemical defense system exists in the human body to provide protection from oxy–centered radicals generated by exercise (Lekhi et al., 2007).

Serum creatine kinase (CK) and lactate dehydrogenase (LDH) activities have been used as markers of muscle cell damage because they are released into blood when a disruption occurs in the sarcomere (Clarkson et al., 2006; Neubauer et al., 2008). In a study, in which the effect of vitamin E re-enforcement on the exercise which composes oxidative stress has been searched, in the measurements after the exercise, a significant rise was found in CK value (Keong et al., 2006). Schröder et al. (2001) expressed a significant increase in the LDH activity in basketballers after 24 h of the exercise.

The aim of this study was to investigate the effects of a 12 min run test on MDA and nitric oxide (NO) as the index of lipid peroxidation, and 8–OHdG level which is the indicator of DNA damage, and CK and LDH activities and glucose concentration as the indicators of exercise stress as well as some antioxidant enzyme activities such as SOD, CAT, GPx in trained male athletes.

MATERIALS AND METHODS

Subjects

Twenty-two healthy and trained male athletes, who were selected from University of Erciyes–Kayseri, participated in the present study. All subjects were non–smokers and did not take any supplements including vitamins and medications. Informed consent was obtained from all of the subjects prior to the study. All experimental procedures were performed in accordance with the Helsinki Declaration of 1975. The study protocol and the procedures were approved by Erciyes University Ethical Committee.

Study design

Age, weight, height, body mass index, body fat percentage, waist to hip ratio and maximal oxygen uptake (VO_{2max}) were recorded (Table 1). Body fat percentage was assessed using a body fat analyzer (Tanita BC-418 MA, Tanita Corp, Tokyo, Japan) according to the manufacturer's protocol. The athletes performed a 12 min run test after 10 to 15 min warming up. At the end of the test, the distance covered by the athletes was recorded. Twelve minute walk/run tests were executed outdoors on the field of the residential centre. VO_{2max} values of athletes were calculated according to the following formula;

VO_{2max}: (Distance covered in meters – 504.9) ÷ 44.7 ml/kg/min (Noonan and Dean, 2000; Mackenzie, 2005).

Blood samples

The blood samples were collected into two vacutainers, either containing EDTA or no additives at resting, before and immediately after exercise and 24 h after exercise. Plasma and sera were separated and then stored in multiple aliquots at –80°C until the analysis. All assays were performed duplicated on first thaw.

Biochemical analysis

Serum 8–OHdG (Northwest Life Science Specialties, Catalogue: NWK-8-OHdG02), NO (Nitrate/Nitrite Colorimetric Assay Kit, Cayman Chemical, and Ann Arbor, Catalogue: 780001 USA), MDA (Cayman Chemical, and Ann Arbor, USA, Catalogue; 10009055). CAT activity was measured using ELISA kit (Cayman Chemical, and Ann Arbor, USA, Catalogue: 707002) and SOD activity (Cayman Chemical, and Ann Arbor, USA, Catalogue; 706002,) were measured with ELISA kits. Plasma GPx activity was measured with ELISA Kit (Northwest Life Science Specialties, Catalogue; NWK-GPx01).

Serum glucose level (Biolabo, France), CK and LDH activities (Teco Diagnostics, USA) were measured with Shimadzu UV/Vis 1208 spectrophotometer using commercially available kits.

Statistical analysis

The Statistical Package for the Social Sciences (SPSS Inc, version

Table 2. Effects of acute exercise on oxidative stress, antioxidant enzymes and some serum variables in male athletes.

Variables	Mean ± SEM			F	p
	Immediately before exercise	Immediately after exercise	24 h after exercise		
MDA (µM)	10.52 ± 1.26[a]	7.36 ± 0.99[b]	11.41 ± 1.62[a]	8.469	0.014
SOD (U/ml)	0.97 ± 0.11[a]	1.26 ± 0.13[a]	0.94 ± 0.09[a]	2.807	0.072
NO (µM)	3.91 ± 0.36[a]	4.61 ± 0.48[a]	4.22 ± 0.33[a]	1.173	0.319
GPx (mU/ml)	7.56 ± 0.87[a]	7.03 ± 0.66[a]	6.62 ± 0.56[a]	0.393	0.677
CAT (mU/ml)	7.58 ± 0.28[a]	8.02 ± 0.47[a]	8.02 ± 0.45[a]	0.503	0.608
8-OHdG (ng/ml)	0.92 ± 0.23[a]	1.22 ± 0.41[a]	1.40 ± 0.34[a]	0.624	0.541
LDH (IU/L)	133.46 ± 4.32[a]	175.08 ± 6.56[b]	139.88 ± 4.56[a]	45.893	0.000
CK (IU/L)	342.17 ± 84.56[a]	384.42 ± 95.67[b]	362.17 ± 83.74[ab]	5.277	0.013
Glucose (mg/dl)	96.34 ± 1.20[a]	110.25 ± 2.34[b]	98.31 ± 1.44[a]	21.582	0.000

a, b, c : Values on the same row with different superscripts are significantly different, n=22.

15.0) was used for all of the analysis. Means, standard error of means (SEM) were calculated for all variables. A one–way repeated measures ANOVA test was performed followed by post–hoc analysis with the Bonferroni test. The significance level was set at 5%.

RESULTS

Anthropometric characteristics of subjects are shown in Table 1. A significant decrease was determined in MDA values immediately after the exercise that returned to normal level after 24 h–rest period ($p<0.05$). The NO, SOD, GPx, CAT and 8–OHdG levels were not significantly different between the time periods (before exercise, immediately after exercise and 24 h after the exercise) ($p>0.05$). The LDH ($p<0.001$) and CK ($p<0.05$) activities significantly increased immediately after exercise compared to the pre–exercise levels. However, LDH activity decreased after 24 h of rest and returned to the baseline levels whereas there was no significant difference between the sampling times of immediately after exercise–after 24 hours and before exercise–24 hours after the exercise in CK activity.

Glucose level significantly increased immediately after exercise compared to the pre-exercise level. However, glucose level decreased after 24 hours of rest and returned to the baseline level ($p<0.001$) (Table 2).

DISCUSSION

Energy consumption and oxygen need increase in physical activity. Free radicals are formed as by–products of the normal metabolism while it is suggested that production of reactive oxygen species would increase as the result of consuming more oxygen by working muscle (Sjödin et al., 1990). In the present study, serum MDA levels of the trained young men showed significant decreases immediately after the exercise and then returned to baseline levels after a 24 h rest. Similarly, serum TBARS concentrations were significantly low in trained individuals who had high anaerobic threshold in a study (Falone et al., 2010), whereas in another research, training had no significant impact on resting MDA levels but acute exercise significantly increased the MDA levels even after the training period (Ookawara et al., 2003). In a number of studies, it has been shown that lipid peroxidation increased in untrained individuals doing acute exercise in unusual intensity (Allesio et al., 1997; Balci et al., 2010; Branth et al., 2009; Bailey et al., 2001). In a study conducted in sedentaries, sedentary group had an increased lipid peroxidation (MDA) level in their liver and skeleton muscle after acute exercise while no increment was seen in training group (Allesio and Goldfarb, 1988). Acute exhaustion exercise performed after eight week endurance training was found to significantly increase erythrocyte MDA levels in sedentary group whereas important changes were not seen in training group in a study (Öztaşan et al., 2004). The contradictory results may be caused by variables such as sample characteristics, type and intensity of exercise. Increase in MDA may be dependent on oxygen intake (Bloomer et al., 2005). The short duration of the exercise in our study could have lead to a decrease in TBARS level. Although long term acute exercise significantly increases NO synthetase levels in trained subjects (Marfe et al. 2010), in the present study, no significant change was seen in serum NO levels immediately and 24 h after the exercise in trained young men. Similarly, Fadıllıoğlu et al. (2001) could not demonstrate a difference between baseline and final serum NO levels.

A slight but not significant increase was determined in 8–OHdG levels after the exercise and this slight increase continued at the last sampling time after a 24 h rest. In a study conducted by Bloomer and Fisher-Wellman, (2008), the relation between blood oxidative stress markers and gender, training status and dietary content were examined and no significant differences in 8–OHdG levels

were determined. On the contrary of this study and the results of theour study, it is mentioned that exercise causes a significant increase in 8–OHdG activity (Radak et al., 2002; Orhan et al., 2004). Statistically significant increases in CK and LDH activities, which are the indicators of muscle damage, were seen immediately after the exercise and there has been slow return to baseline levels in CK activity while LDH activity returned to basal levels after a 24 h rest. In a study, LDH and CK activities immediately after and 24 h after the exercise were similar to the present study results (Schneider et al., 2005). Creatine kinase levels increased significantly one and 24 h after acute exercise (Keong et al., 2006). Additionally, CK levels were found to be elevated after a 7 h cycle race (Branth et al., 2009). Creatine kinase activity was reported to reach the highest level at 72 h after an exercise program which was performed in different periods for two weeks (Jubeau et al., 2008). Creatine kinase and LDH activities were significantly higher in athletes than in sedentary individuals (Evelson et al., 2002). A significantly higher LDH activity was determined in athletes compare to control group including sedentary subjects (Brites et al., 2006).

Superoxide dismutase function in the cell means an advanced resistance against oxidative stress as one of the basic antioxidative defense mechanisms towards superoxide radicals (Powers and Lennon, 1999). The effect of acute exercise on serum SOD activity was not significant in the present study. There has not been found a significant difference in the SOD activity levels immediately and 24 h after the exercise in trained young men. Endurance exercise performed 30 min a day for 6 weeks could not have an impact on SOD activities in men and women (Tonkonogi et al., 2000). However, a five week exercise program resulted in a significant increment in SOD enzyme activity immediately and 24 h after acute exercise (Fauzi et al., 2007).

In a study evaluating the effect of 12 week aerobic exercise program on G6PD activity, glutathione peroxidase and glutathione S transferase levels in basketball players, glutathione peroxidase levels were found to be increased after the exercise program (Kaldırımcı, 2010). In contrast, in the present study, there has been a slight decrease in GPx activity after the exercise which continued by decreasing after 24 h rest. Similarly, it was found that endurance training performed 30 min a day for 6 weeks did not affect GPx activity in men and women (Tonkonogi et al., 2000).

After a 21 km run, oxidant and antioxidant status in early and later resting periods of trained adolescent runners were investigated and CAT value was found to be increased at 2 and 24 h after the run (Tian et al. 2010). Higher serum MDA and lower CAT activities were found in elite cyclers compared with sedentary subjects after exhaustion exercise (Lekhi et al., 2007). It was reported that in CAT activity, there was a difference before and 24 h after exercise while CAT activity was significantly different after and 24 h after the exercise (Brown et al., 1992). In another study, in which exercise duration was limited to 20 to 25 min, regular exercise had an activating effect on antioxidant system (Fadıllıoğlu et al., 2000).

In trained and untrained groups, catalase activities were measured in the blood before and after three different intensities of exercise on the treadmill. There were no changes in catalase activity (Schneider et al., 2005). Similarly, in our study, CAT activity was not significantly different between the time periods. In our study, glucose levels significantly increased immediately after the exercise and returned to baseline levels of a 24 h rest. Glucose level was found to be higher after exercise with an increase in catecholamine levels (Coker and Kjaer, 2005). Similarly, glucose levels were found high after exercise compare to rest values. Blood glucose levels are maintained by the liver through glycogenolysis and glyconeogenesis during exercise (Gaitanos et al., 1993; Sawka et al., 2007). This increase may be explained by hormonal factors which may be effective on glucose usage during exercise. This study has some limitations: Firstly, the participants were informed about dietary measures but were not controlled, and diet was not recorded the day before the exhaustive exercise tests. Secondly, the exercise protocols were performed on highly physically trained individuals. Therefore, the responses observed may not be representative of sedentary individuals.

In conclusion, our results indicate that acute 12 min endurance exercise on trained young men had no effect on antioxidant capacity and DNA damage marker. However, we have found that short-term endurance training decreased the level of MDA. But we have also demonstrated that CK and LDH activities are the markers of muscle cell damage, and blood glucose increases immediately after acute exercise, returning to baseline level after a 24 h rest.

ACKNOWLEDGEMENTS

Erciyes University, Research Foundation has supported this research (contract grant number BSYO–07–01).

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