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Full Length Research Paper

Effects of acute exercise and aerobic exercise training on oxidative stress in young men and women

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This research investigated the effect of acute exhaustive exercise and exercise training on thiobarbituric acid-reactive substances (TBARS), protein carbonyl (PC), total glutathione (GSH) levels and total superoxide dismutase (SOD) activities. Thirty-two healthy young men and women, 19 to 27 years old, who volunteered to participant in this study, underwent two months of endurance training. The training program consisted of cycling exercises for 60 min, four times a week. Before and after the training course, they performed acute exhaustive exercise, and blood samples were collected immediately before, immediately after and 30 min after this exercise. The training significantly increased maximal oxygen uptake in the training groups. Also, body weight did not change in any of the groups. TBARS, PC and GSH levels were significantly affected by acute exercise in both women and men. However, acute exercise increased the activities of total SOD in women. Also, in men, the changes in SOD activity during acute exercise were significantly different between the control and training groups. There was no significant interaction effect among time, acute exercise and group for the TBARS, PC, GSH levels and SOD activities in either the women or men. These results indicate that the lipid and protein damage in response to acute exercise was not altered by aerobic exercise-training.

Key words: Thiobarbituric acid-reactive substances, protein carbonyl, superoxide dismutase, glutathione, exercise training, acute exercise.

INTRODUCTION

Oxidative stress is the status of deterioration of the balance between oxidant formation and antioxidant defense, which leads to an increase in the amount of antioxidants (Urso and Clarkson, 2003). Free radicals and reactive oxygen species (ROS) in particular are produced by enzymatic and non-enzymatic sources in normal physiological processes and cause permanent damage to lipids, proteins and DNA (Finaud et al., 2006). Depending on the increased metabolic activity during exercise, the use of oxygen and electron leakage from the mitochondrial electron transport chain is increased, resulting in an increase in many reactive oxygen species including superoxide, hydrogen peroxide and hydroxyl radicals. The consumption and flow of oxygen in mitochondria is increased with physical exercise, which can lead to oxidative stress after a certain intensity and/or duration (Ji, 1999; Alessio, 1993; Leeuwenburgh and Heinecke, 2001; Vollaard et al., 2005). For this reason, it has been suggested in many studies that physical activities, such as running, cycling and swimming cause free radical production in both human and experimental animals (Alessio et al., 2000, Liu et al., 2000; Aguilo et al., 2005; Nikolaidis et al., 2007; Balci et al., 2010). However, there are also studies reporting that markers of oxidative damage decrease (Groussard et al., 2003; Kretzschmar et al., 1991) or remain unchanged (Margaritis et al., 1997; Vasankari et al., 1997; Revan et al., 2010) after intensive and/or long-term acute exercises.

The severity of exercise plays an important role in acute exercise-induced oxidative damage (Quindry et al., 2003; Muñoz Marín et al., 2010). The unusual intensity and duration of acute exhaustive exercises in particular cause oxidative stress (Viña et al., 2000). In a large number of different tissues, it has been reported that regular aerobic exercises can reduce oxidative damage caused by acute exhaustive exercises (Oh-ishi et al., 1997; Miyazaki et al., 2001; Oztasan et al., 2004; Revan and Erol, 2011). However, it has not been understood yet, according to the results of the study, whether the

reduction in the formation of oxidative stress after acute exercise is due to the decline in the formation of free radicals or an increase in the effectiveness of antioxidant capacity (Finaud et al., 2006). Many anti-oxidant enzymes help to reduce oxidative stress during exercise. Superoxide dismutase (SOD) and GSH form the first line of the defense against the ROS that occurs during exercise. Antioxidant enzyme activities have been indicated to be increased in response to exercise in both human and animal studies (Ji, 1999).

ROS plays important roles in many physiological processes at rest; however, the physiological functions of ROS during exercise have not been fully elucidated (Vollaard et al., 2005). Therefore, in this study, the effects of acute exhaustive exercises and aerobic training on oxidative stress parameters in young men and women were investigated.

MATERIALS AND METHODS

Participants

Sixteen healthy young women (n = 8 control, 22.63 ± 0.84 years old; n = 8 training, 20.38 ± 0.38 years old) and sixteen healthy young men (n = 8 control, 22.88 ± 0.83 years old; n = 8 training, 20.63 ± 0.26 years old) volunteered to participant in this study. None of the students were involved in any regular training program prior to the study. All of the participants were nonsmokers, and for at least two months prior to the study, they had not taken any vitamins, minerals or medications that may affect oxidative stress markers. The procedures and risks were thoroughly explained to the participants, and their written and informed consent was obtained. The study was approved by the Local Ethical Committee of the School of Physical Education and Sports of Selçuk University.

Experimental design

Assessment of physical characteristics and acute exercise tests

Body weight was measured with a SECA scale (SECA, Hamburg, Germany), and body height with a stadiometer that was incorporated into the scale. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters.

The subjects were required to cycle to volitional exhaustion and eliciting maximal oxygen uptake (VO_{2max}), on a calibrated cycle ergometer (834 E, Monark, Stockholm, Sweden). Subjects were habituated to the exercise test in the week prior to commencement and were instructed to refrain from exercise for 24 h before the test while maintaining their usual dietary pattern. Breath-by-breath oxygen uptake was continuously recorded using a computerized on-line portable gas analysis system (Cosmed K4b^2, Italy). Heart rate was continuously monitored and recorded during the test using a portable heart rate telemetry device (Polar Electro, Finland). The subjects warmed up for 3 min at 25 W prior to starting the test. The test was incremental and progressive; all subjects commenced at a 50 W workload and were required to maintain a constant cadence of 60 rpm. Workload was increased by 25 W every 3 min until exhaustion. Tests were carried out in the morning after an overnight fast. Blood samples were taken from each of the participants immediately before, immediately after and 30 min after the acute exercise tests.

Exercise training

Sixteen participants participated in an exercise training program during the follow-up period of eight weeks. The subjects in the control group were instructed not to change their habitual activity pattern over this period. After the first acute exercise experimental trial was completed, all participants in the training groups participated in an endurance training program, which consisted of cycling on a bike for 60 min, four days a week for eight weeks. Exercise intensity was based on the percentage of each subject's maximal heart rate (HR_{max}) determined during the initial aerobic capacity test. Heart rate was monitored continuously during the training sessions (Polar Electro, Finland). Exercise intensity was increased progressively from 50 to 75% HR_{max} during the first four weeks of the training period. All training sessions took place at the laboratory under the supervision of a professional instructor. After the exercise training program, the second acute exercise tests were performed.

Biochemical analysis

Blood samples were taken from the subjects after an overnight fast. Blood was drawn from the antecubital vein into a 10 ml Vacutainer tube (with Ethylene Diamine Tetra-acetic Acid [EDTA]). Plasma was obtained by centrifugation of blood at 2,500 rpm for 10 min at +4°C. The plasma was then stored at -80°C until analysis.

The thiobarbituric acid-reactive substances (TBARS) levels (catalog no. 10009055), PC levels (catalog no. 10005020), GSH levels (catalog no. 703002) and SOD activities (catalog no. 706002) in the plasma were determined by commercial kits (Cayman Chemical Company, Ann Arbor, Michigan) according to the manufacturer's instructions.

Statistical analysis

Statistical analysis was performed using SPSS for Windows (Chicago, SPSS Inc.). A two-way (time × group) repeated measures analysis of variance (ANOVA) was used to analyze changes in physical characteristics from pre-training to post-training. Unpaired t-tests were used to compare mean values between groups at both the beginning and end of the eight weeks of training. A two (training and control group) × two (pre-training and post-training) × three (pre-exercise, post-exercise and 30 min recovery) repeated measures ANOVA was used to analyze changes in TBARS, protein carbonyl (PC), total glutathione (GSH) levels and SOD activities. If a significant P value was identified for the main effect of time (pre-training and post-training, pre-exercise, post-exercise and 30 min), multiple pairwise comparisons were made using Bonferroni confidence interval adjustment. Results were reported as the mean ± SEM of all observations, and the level of significance was set at $P < 0.05$.

RESULTS

Table 1 shows the physical characteristics of the four groups. All baseline variables in the pre-training stage were not different between the control and training groups. No significant time, group, or interaction effects were observed for body weight and BMI in women. In young men, time and group interaction was significant for body weight and BMI; however, there were no significant differences between the control and training groups. There was a significant time and interaction effect ($P <$

Table 1. Physical characteristics of the participants.

	Gender	Group	Pre-training	Post-training	Time	Group	Time × Group
Body weight (kg)	W	Co	59.01 ± 1.9	59.05 ± 2.19	1.69	3.41	1.41
		Tr	54.30 ± 0.8	55.11 ± 1.31			
	M	Co	70.93 ± 2.86	69.55 ± 2.95	0.98	0.01	19.78*
		Tr	69.64 ± 2.31	70.51 ± 2.38			
BMI (kg/m^2)	W	Co	21.96 ± 0.63	21.98 ± 0.72	0.20	0.49	0.06
		Tr	21.43 ± 0.21	21.51 ± 0.31			
	M	Co	22.58 ± 0.81	22.16 ± 0.81	2.35	0.15	5.62*
		Tr	21.9 ± 0.74	21.99 ± 0.77			
VO2max (ml/kg/min)	W	Co	35.49 ± 2.1	35.11 ± 2.2‡	32.24*	1.76	37.24*
		Tr	33.46 ± 1.36	43.64 ± 1.4#			
	M	Co	48.24 ± 2.46	48.75 ± 2.0‡	28.66*	1.98	22.86*
		Tr	47.92 ± 1.36	56.95 ± 2.2#			

Values are mean ± SEM. W, women, M, men, Co, control group, Tr, training group, BMI; body mass index, VO_{2max}, maximal oxygen consumption. *P < 0.05 significant main or interaction effect (2-way repeated measures ANOVA). #P < 0.05 significantly different from pre-training. ‡P < 0.05, significantly different between control and training groups.

0.05) for VO_{2max}, but no significant group effect. The training group exhibited a significantly greater VO_{2max} in comparison to the control group after training in both genders (P < 0.05). No variables in the control group differed between pre-training and post-training in young women and men.

The plasma levels of TBARS were significantly affected by acute exercise in women and men (F = 4.87, F = 9.45, respectively, P < 0.05). Also, there was a significant interaction effect between time and acute exercise for the TBARS levels in both genders. Acute exercise decreased TBARS levels after a 30 min recovery in the training groups for women and men. There were significant main effects of time, acute exercise and the interaction effect between time and acute exercise for PC levels in women and men (P < 0.05). The PC levels increased after acute exercise in the control groups (P < 0.05), but the levels did not change in the training groups in the post-training (Tables 2 and 3).

Acute exercise increased the activities of total SOD in women (acute exercise effect; F = 6.03, P < 0.05). In men, there was a significant group effect (F = 6.03, P < 0.05) and a significant interaction between acute exercise and group (F = 3.82, P < 0.05) for SOD activities. Changes in SOD activity during acute exercise were significantly different between the control and training groups. The total GSH levels were significantly affected by acute exercise in women and men (F = 4.89, F = 52.67, respectively, P < 0.05). Also, there was a significant time effect (F = 58.18, P < 0.05) in women and a significant interaction between time and acute exercise in men (F = 30.71, P < 0.05) for GSH levels. In training women, levels of GSH at 30 min recovery after acute exercise were significantly higher than at pre-exercise levels (P < 0.05). In men, the GSH levels at post-exercise levels were significantly higher than at pre-exercise and at 30 min recovery levels for both the control and training groups (P < 0.05). The changes in GSH levels during acute exercise and 30 min recovery were not significantly different between the control and training groups in women and men (Tables 2 and 3).

DISCUSSION

In particular, exhaustive exercises have been indicated to cause oxidative damage (Gomez-Cabrera et al., 2008, 2009). However, studies have shown that exercise-induced oxidative stress may show some differences between the sexes (Yamamoto et al., 2002; Borrás et al., 2003; Pepe et al., 2009). For these reasons, inthis study, lipid and protein oxidation and antioxidant capacity changes caused by acute exercise and the effects of the training on these changes were separately evaluated in young men and women. The VO_{2max} values of young women and men in the training group were increased significantly, whereas no significant change was observed in both control groups. One of the most important findings of this study is comparable changes of lipid and protein oxidation levels due to acute exhaustive exercises between the training and control groups in both young women and men. Another important finding of the study is that, although, there was an increase in SOD enzyme activity and GSH levels after acute exercise, endurance training had no significant effect on these parameters.

It has been suggested that acute and irregular exercises have negative effects, whereas regular physical activity creates an advanced anti-oxidant system

Table 2. Changes in TBARS, PC and GSH levels, and SOD activities during the acute exercise in pre-training and post training in young women.

	Groups	Pre-and post-training	Pre-exercise	Post-exercise	30 min recovery	ANOVA						
						T	A	G	TxG	AxG	TxA	TxAxG
TBARS (µM)	Co	Pre	1.35 ± 0.07	1.68 ± 0.19	1.56 ± 0.16	7.86*	4.87*	0.17	0.01	3.67	12.08*	0.71
		Post	1.54 ± 0.12	1.39 ± 0.16	1.15 ± 0.02							
	Tr	Pre	1.59 ± 0.16	1.54 ± 0.1	1.34 ± 0.07							
		Post	1.78 ± 0.08	1.10 ± 0.03$^{\#}$	1.10 ± 0.06$^{\#}$							
PC (nmol/ml)	Co	Pre	5.14 ± 0.46	4.89 ± 0.37	4.89 ± 0.35	104.25*	7.98*	1.28	0.20	1.30	6.90*	3.54
		Post	6.41 ± 0.32	6.77 ± 0.49	9.30 ± 0.38$^{\#\ddagger}$							
	Tr	Pre	4.74 ± 0.47	5.49 ± 0.53	5.35 ± 0.39							
		Post	7.69 ± 0.34	7.59 ± 0.55	8.54 ± 0.26							
SOD (U/ml)	Co	Pre	0.08 ± 0.00	0.08 ± 0.00	0.09 ± 0.00	1.34	6.03*	0.40	3.08	0.31	0.57	1.96
		Post	0.08 ± 0.00	0.09 ± 0.00	0.10 ± 0.01							
	Tr	Pre	0.08 ± 0.00	0.09 ± 0.00	0.09 ± 0.00							
		Post	0.08 ± 0.00	0.09 ± 0.00	0.09 ± 0.00							
GSH (µM)	Co	Pre	5.76 ± 0.13	6.00 ± 0.10	6.45 ± 0.19	58.18*	4.89*	1.74	1.53	0.41	0.99	1.37
		Post	6.79 ± 0.29	6.92 ± 0.22	7.45 ± 0.39							
	Tr	Pre	5.74 ± 0.09	5.59 ± 0.19	5.73 ± 0.15							
		Post	6.54 ± 0.27	7.18 ± 0.26	7.42 ± 0.42$^{\#}$							

Values are mean ± SEM. Co; control group (n = 8), Tr; training group (n = 8), TBARS; thiobarbituric acid reactive substances, PC; protein carbonyl, SOD; superoxide dismutase, GSH; total glutathione. T; time main effect, A; acute exercise main effect, G; group main effect, TxG; time and group interaction effect, AxG; acute exercise and group interaction effect, TxA; time and acute exercise interaction effect, TxAxG; time, acute exercise and group interaction effect. *P < 0.05; significant main or interaction effect (3-way repeated measures ANOVA). $^{\#}$P < 0.05; significantly different from pre-exercise levels. ‡P < 0.05; significantly different from post-exercise levels. $^{\&}$P < 0.05; significantly different from 30 min recovery levels.

and decreases oxidative damage that trained individuals have a higher antioxidant capacity and lower levels of oxidative damage than sedentary individuals in resting conditions (Ortenblad et al., 1997; Selamoglu et al., 2000), and that moreover, sedentary individuals have higher levels of oxidative damage caused by acute exhaustive exercise than athletes (Metin et al., 2003; Caimi et al., 2009). In contrast, there are studies indicating that athletes have higher lipid peroxidation levels at rest or after exhaustive exercise than sedentary individuals in spite of regular exercise programs (Lekhi et al., 2007; Teixeira et al., 2009). In this study, the change in the levels of TBARS, which is a marker of lipid peroxidation after acute exhaustive exercises in young women and men, did not differ between exercise training and control groups. TBARS levels during the recovery period, 30 min after the exhaustive exercise were found to be significantly lower in proportion to the resting level, in the training group in particular. After the training period, while the PC levels of men and women in the control group were significantly increased after the exhaustive exercise, it remained unchanged in the training group. The lack of difference in the PC levels between the groups after aerobic exercise training shows that these increases in the control group are due to seasonal changes. However, young men and women in the training group were not affected by these changes. Miyazaki et al. (2001) reported that endurance training applied for a period of 12 weeks reduced TBARS levels

Table 3. Change in TBARS, PC and GSH levels, and SOD activities during the acute exercise in pre-training and post training in young men.

	Groups	Pre-and post-training	Pre-exercise	Post-exercise	30 min recovery	ANOVA						
						T	A	G	TxG	AxG	TxA	TxAxG
TBARS (μM)	Co	Pre	1.74 ± 0.14	1.78 ± 0.12	1.77 ± 0.21	3.96	9.45*	2.74	0.39	0.24	5.06*	2.13
		Post	1.88 ± 0.26	2 ± 0.15	1.06 ± 0.03$^{\#\ddagger}$							
	Tr	Pre	1.68 ± 0.11	1.73 ± 0.24	1.48 ± 0.11							
		Post	1.53 ± 0.07	1.57 ± 0.15	1.13 ± 0.06$^{\#}$							
PC (nmol/ml)	Co	Pre	4.92 ± 0.25	5.7 ± 0.27	5.13 ± 0.39	47.46*	7.82*	1.84	0.33	0.81	3.59*	1.07
		Post	5.27 ± 0.42	7.5 ± 0.48$^{\#}$	7.82 ± 0.81$^{\#}$							
	Tr	Pre	5.33 ± 0.32	5.48 ± 0.35	5.70 ± 0.52							
		Post	6.60 ± 0.20	8.00 ± 0.44	7.64 ± 0.65							
SOD (U/ml)	Co	Pre	0.09 ± 0.00	0.08 ± 0.00	0.09 ± 0.00	0.22	1.43	4.95*	1.42	3.82*	0.85	1.07
		Post	0.09 ± 0.00	0.08 ± 0.00	0.09 ± 0.00							
	Tr	Pre	0.09 ± 0.00	0.10 ± 0.01	0.09 ± 0.00							
		Post	0.09 ± 0.00	0.10 ± 0.01	0.10 ± 0.01							
GSH (μM)	Co	Pre	6.17 ± 0.19	6.14 ± 0.23	5.98 ± 0.16	0.01	52.67*	0.90	0.68	3.56	30.71*	2.89
		Post	5.45 ± 0.16	7.4 ± 0.29$^{\#\&}$	5.19 ± 0.10							
	Tr	Pre	5.78 ± 0.09	5.89 ± 0.12	5.91 ± 0.14							
		Post	5.83 ± 0.34	6.77 ± 0.10$^{\#\&}$	5.31 ± 0.07							

Values are mean ± SEM. Co, control group (n = 8), Tr, training group (n = 8), TBARS, thiobarbituric acid reactive substances, PC, protein carbonyl, SOD, superoxide dismutase, GSH; total glutathione. T; time main effect, A, acute exercise main effect, G, group main effect, TxG; time and group interaction effect; AxG, acute exercise and group interaction effect, T×A, time and acute exercise interaction effect, T×A×G, time, acute exercise and group interaction effect. *$P < 0.05$, significant main or interaction effect (3-way repeated measures ANOVA). $^{\#}P < 0.05$; significantly different from pre-exercise levels. $^{\ddagger}P < 0.05$, significantly different from post-exercise levels. $^{\&}P < 0.05$, significantly different from 30 min recovery levels.

that had been increased in response to endurance exercise training when compared with the pre-training levels. In this study, although similar results were obtained in the training groups, comparable results in the control groups have shown that this reduction was not only due to aerobic workout. In another study with a similar research model to this study, eight-week endurance workouts were reported to have no effect on the levels of lipid peroxidation and protein damage caused by acute exercise in young men (Rahnama et al., 2007). In another study, it has been reported that an increased antioxidant defense capacity may be insufficient to prevent protein damage even in very well-trained athletes (Tauler et al., 2006).

Although, changes in antioxidant capacity after acute exercise and training can show differences between the tissues, antioxidant parameters have been observed to be decreased in response to acute exercise and be increased after exercise training (Liu et al., 2000). However, in this study, young women and men were determined to have significant increases in plasma SOD activity and GSH levels after acute exercise. However, aerobic exercise training was found to have no effect on the changes in the plasma SOD activity and GSH levels during and after acute exercise. ROS is not only toxic, but also plays an important role in cell signaling in the regulation of gene expression. Cellular signals lead to the increase of powerful anti-oxidant enzymes during exercise, so the exercise can be characterized as an anti-oxidant

on its own. The role of free radical metabolism in cell signaling and regulation of gene expression may also interfere with useful adaptations of exercise training (Gomez-Cabrera et al., 2008). There are studies reporting that SOD activity and other antioxidant parameters in resting conditions and after acute exercise are higher in trained individuals than non-trained individuals (Balakrishnan and Anuradha, 1998; Dékány et al., 2006; Lekhi et al., 2007), as well as studies reporting that SOD activity and GSH levels decrease (Duthie et al., 1990) or remain unchanged (Alessio et al., 1998; Miyazaki et al., 2001; Ookawara et al., 2003; Elosua et al., 2003; Oztasan et al., 2004; Pittaluga et al., 2006) after acute exercise in trained individuals. The results of studies investigating the effects of training on changes in oxidative stress parameters caused by acute exercise are contradictory. These contradictory results may have resulted from factors including which methods were used to measure oxidative stress parameters, how late the study sample was collected after the exercise and how much later the sample was studied after its collection (Jenkins, 2000).

However, seasonal changes (Balog et al., 2006), differences in the physical fitness levels of individuals who took part in the studies (Dékány et al., 2006) or differences in the study designs might also have affected the study results.

This study has some limitations, such as the eight-week training period. In fact, different results can be achieved in longer-term studies. Secondly, participants were informed about the diet they would adhere to the day before the tests when the blood samples were taken, but this was not controlled. Finally, the small number of samples in the study groups may have reduced the statistical power of the study. Therefore, these results should be confirmed with greater number of samples in future studies.

Conclusion

Conclusively, it can be said that the changes in TBARS, PC, GSH levels and SOD activities caused by acute exhaustive exercises in young women and men are not affected by an eight-week aerobic training program.

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