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INTERACTION OF ACUTE SWIMMING EXERCISE AND EFFECT ON LIPID PEROXIDATION AND ANTIOXIDANT DEFENSE SYSTEM IN PLASMA AND BLOOD OF 10-12 AGES SWIMMERS

ABSTRACT

Aim of our investigation was to assess whether bout of exercise induced adaptations to reduce the extent of oxidative damage, and change antioxidant system. Also we investigated in short distance (100-m) adolescence swimmers, change the anaerobic metabolisms. For this study, swimmers aged between 12-14 swam 100 m (n=19). Venous blood samples were taken before acute swimming exercise and 20 min. after acute swimming exercise. We concluded that short-distance (100-m) swimming reducing oxidative stress in female adolescence swimmers are due to proper adaptive mechanisms. This is possibly due to different mechanisms or different activities of specific antioxidant and repair system.

Keywords: Sport, Exercise, Swimming Exercise, Lipid Peroxidation Antioxidant Defense, Swimmer

10-12 YAŞ GRUBU YÜZÜCÜLERİN AKUT YÜZME EGZERSİZLERİNİN KAN VE PLAZMADA LİPİD PEROKSİDASYONU VE ANTİOKSİDAN SİSTEM ÜZERİNE ETKİSİ

ÖZET

Bu araştırmada, egzersizin oksidatif hasarı azaltmadaki etkisi ve antioksidan sistemde meydan gelen değişiklikler ele alınmış ve kısa mesafe yüzmede (100m) yüzücülerin anaerobik metabolizmasında meydan gelen değişikliklerin incelenmesi amaçlanmıştır. Çalışmaya 10-12 yaşları arasında (n= 19) yüzücü katılmıştır. Yüzücülerin kan alınımı yarıştan 20 dakika önce ve yarışı tamamlandıktan sonra yapılmıştır. Araştırmada akut yüzme egzersizi sonrası değerler öncesi değerlerle karşılaştırıldığında; serum KAT KAT, CK, LDH aktivitesi istatistiksel düzeyde yüksek bulunurken (0.01, $p<0.05$), MDA, eritrosit MDA düzeyleri ile GPX ve SOD aktivitelerinde ise istatistiksel düzeyde bir farklılık bulunmamıştır. Ancak bayan deneklerde ise GPx ve GSH aktivitesinde istatistiksel olarak anlamlı şekilde daha düşük olduğu bulunmuştur. Sonuç olarak 100 m kısa mesafe yüzmenin bayan yüzücülerde oksidatif hasarı azalttığı dönük bir adaptasyonun olduğu belirlenirken, bunun özel antioksidan ve korunma sistemlerinin farklı mekanizmaları ya da farklı aktiviteleri tarafından sağlandığı düşünülmektedir.

Anahtar Kelimeler: Spor, Egzersiz, Yüzme Egzersizi, Lipitperoksidasyon, Yüzücü

1. INTRODUCTION (GİRİŞ)

The cellular responses to oxidative stress vary from cell death, growth arrest to cell proliferation or transformation. These responses are dependent on the types of stress stimuli, the dose or the exposure time to stress, cell types and the surrounding cell environment [1]. Reactive oxygen species (ROS) are formed during normal physiologic processes by nonenzymatic and enzymatic sources and continuously cause damage to lipids, proteins and nucleic acid [2].

Peroxidation of unsaturated fatty acid residues of phospholipids in cell membranes might result in significant loss in membrane integrity, that is one of the most striking effects of oxidative damage leading to generation of potentially harmful aldehydes and alkanes [3]. Unfortunately, if 4-hydroxynonenal (4-HNE) is excluded, the detection of membrane adducts is not directly measurable. Therefore, the nonspecific test of thiobarbituric acid reactive substances (TBARS) is very often applied to assess the pressure of oxidative stress [4]. In addition, oxidative modification of amino acids has significant effects on cell functions as oxidatively modified proteins lose their physiological activity and tend to be very sensitive to proteolytic degradation [5]. All living organisms contain several enzymatic superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPX), glutathione reductase (GR) and S transferases (GST) and nonenzymatic (α-tocopherol, ascorbic and uric acids, glutathione (GSH) and other thiol protein groups) antioxidants with specific purpose of protecting the functional and structural integrity of biologically fundamental macromolecules (nucleic acids, proteins, phospholipids). Cells undergo oxidative stress when reactive oxygen species (ROS) exceed counter-regulatory antioxidant capacity. The phenomenon is generated either from elevated production and accumulation of ROS or from a diminution in the cellular antioxidant defence system [6 and 7].

Exercise is now well accepted to increase oxidative stress due to free radical production, as oxygen consumption (VO_2) becomes elevated 10- to 15-fold above the resting condition [8 and 9]. Recently, many authors and their collaborators have published data from a series of experimentations addressing the very issue of the paradoxical nature of exercise and oxidative stress [10 and 11]. These researchers clearly document that in the absence of any adaptive or compensatory responses, exercise causes widespread oxidative damage, as they detected in DNA [12], proteins [13], and lipids [14]. On the other hand, exercise training - both endurance and interval type - appears to induce antioxidant protection and decrease oxidative insult. Thus regular physical exercise protects against exercise induced oxidative stress [15]. Numerous studies on humans have suggested that life long regular exercise reduces the incidence of cardiovascular diseases and certain types of cancer, and thereby prolongs life-span [16]. One consensus on the effect of exercise among researchers is single bouts of exercise of moderate intensity and long duration or of relatively short duration and high intensity might lead to increases in the content of TBARS, RCD, some antioxidant enzymes (i.e. GSH peroxidase and catalase) and decrease antioxidant systems (i.e. GSH) [17]. Although the data from most studies seem to support the notion of exercise-induced oxidative damage, the biochemical mechanisms of bout of exercise are not well understood.

2. RESEARCH SIGNIFICATION (ÇALIŞMANIN ÖNEMİ)

Free radical formation due to acute and chronic exercise, the resulting lipid peroxidation and antioxidant systems in blood and muscle, and their effect on immune system have been widely documented

in experimental animals and old humans. However, there is limited information on the changing biochemical parameters due to oxidative stress after acute and chronic exercise in growing children. The relationship between the style, distance, exercise characteristics, swimming interval and the oxidative stress is another point that needs clarification. The aim of our study was to investigate the blood and plasm free radical levels and their effect on aerobic and anaerobic metabolism in growing children after short distance (100 meters) swimming exercise.

3. METHODS (YÖNTEM)

This investigation 10 boy swimmers (12.7±0.4 years old) and 9 girl swimmers (12.1±0.3 years old) from the reserve team of a club with an age average of 12.4±0.3 volunteered for the study. While choosing the volunteers, at least 3 years of swimming experience, their practicing program and the conditions in which they practiced were considered.

The field experiments performed on the subjects were done in the 25 m Bursa indoor swimming pool with an air temperature of 25±2°C and water temperature of 24±2°C. Three Casio 2000 chronometers were used for the calculations. Official race rules were applied in figuring out the swimming time of the subjects. Swimmers prepared for the races with a 6 weeks training program. The swimmers went through the race after general training period, 4 weeks of aerobic endurance (Endurance-I), 4 weeks of anaerobic capacity training, 3 weeks pre-race period and 1 week race (peak) period.

Through the biochemical experiments, subjects were asked to keep up their usual diets, to sleep enough at nights, not to drink coffee or tea or do any body pushing activities before the experiments, and not to use any medication. Blood samples were taken from the swimmers 20 minutes before the race and in 3-5 minutes after the race in the indoors swimming pool. Blood samples were taken from the vein on the forearm antekübital area and kept in one dry tube and one tube with heparin. The obtained samples were analyzed in the Department of Biochemistry laboratories of Uludağ University, Faculty of Medicine.

Plasma malondialdehyde (MDA) level was measured according to the procedure established by Kamal and collegues [18]. The sensitivity of erythrocyte membrane lipids was measured according to the procedure established by Stocks and collegues [19]. Glutathione perperoxidase (GPx) activity was measured by using the kit (Randox-England). Superoxide dismutase (SOD) activity was measured by using the kit (Ransod- England). Erythrocyte glutathione (GSH) concentration was prepared by using the method determined by Beutler and collegues [20]. Creatine kinase (CK) activity was measured by using Thermo; TR 14103 kit which measures the total CK. CK enzyme was analyzed by using Poct Multilayer analyzer. Catalase (CAT) enzyme level was measured by using the method determined by Aebi and collegues [21]. Lactate dehidrogenase (LDH) enzyme levels were measured by the method determined by Levine and collegues [22].

Results were analyzed by "SPSS 10.0" statistics programme. For the in-group statistical comparison of data obtained from the participants' tests before and after the competition season t-test and wilcoxon test were used while Mann-Witney U test was used for comparison between the groups.

4. RESULTS (BULGULAR)

The physical data of the subjects are shown in Table 1, before and after acute swimming exercise.

Table 1. Physical data of the subjects
(Tablo 1. Deneklerin bazı fiziksel özellikleri)

Variables	Boys (n= 10)	Girls (n= 9)
Age (y)	12.7 ± 0.4	12.1 ± 0.3
Height (cm)	162.7 ± 8.7	156 ± 10.9
Weight (kg)	55.9 ± 11.2	40.5 ± 13.6
Body fat (%)	11.4 ± 3.4	10.2 ± 1

The comparison of the subjects before and after acute swimming exercise physical data showed that, Cerum KAT, CK, LDH activities and E-RCD level were significantly higher but no statistically significant difference was determined at MDA, erythrocyte MDA level, GPX, SOD activities and GSH levels (Tablo 2).

Table 2. Biochemical values before and after acute swimming
(Tablo 2. Deneklerin akut yüzme egzersizi öncesi ve sonrası biyokimyasal ölçümleri)

Variables	Before Acute Swim Exercise	After Acute Swim Exercise	p
MDA (nmol/ml)	8.7 ± 1.9	8 ± 2.3	0.064
E-MDA (nmolMDA/gr Hb)	106.5 ± 11.4	104.6 ± 10.3	0.740
GSH (mg/dL erythrocyte)	67.3 ± 5.7	68.6 ± 10.4	0.658
SOD (U/gr Hb)	1330 ± 180	1339 ± 315	0.629
GP x (U/gr Hb)	26.8 ± 10.7	31.2 ± 7.4	0.064
KAT (U/L)	4.8 ± 1.2	7.4 ± 1.5	0.000
CK (U/L)	112 ± 49	169 ± 79	0.000
LDH (U/L)	95.2 ± 29	221 ± 60	0.000
Hct (%)	40.2 ± 2.4	41.2 ± 3.9	0.355
T (sn)	78.9 ± 8.9	75.2 ± 8.2	0.001

It was found that cerum KAT, CK and LDH activities are significantly higher in the comparison of the biochemical tests values before and after acute swimming exercise at two groups of swimmers. The comparison of the biochemical tests before and after acute swimming exercise showed that While the level of GPx activities and GSH are significantly lower at female swimmers but the changes at other measurements are not statistically significant (Tablo 3).

Table 3. Boys and girls biochemical values, before and after acute swimming. Data are means ± SD
(Tablo 3. Erkek ve bayan yüzücülerin akut yüzme egzersizi öncesi ve sonrası uygulanan biyokimyasal testlerin karşılaştırılması)

	Boys			Girls		
Variables	Before Acute Swimming Exercise	After Acute Swimming Exercise	p	Before Acute Swimming Exercise	After Acute Swimming Exercise	p
MDA (nmol/ml)	9.8 ± 1.9	9.4 ± 2.4	0.262	7.50 ±1.05	6.58 ± 0.98	0.110
E-MDA (nmolMDA/gr Hb)	105.4 ± 11	105 ±12.5	0.096	108 ±12.1	104.2± 7.6	0.575
GSH (mg/dL Eritrosit)	66.1 ± 4.3	72.6 ± 12.1	0.241	69 ± 7	64 ± 5.68	0.021
SOD (U/gr Hb)	1263 ±168	1246 ± 205	0.721	1405 ±170	1443.6 ± 390	0.859
GP x (U/gr Hb)	32.2 ± 11.3	30.6 ± 4.3	0.721	20.8 ± 6.1	31.9 ± 10.1	0.008
KAT (U/L)	4.60 ± 1.1	7.07 ± 1.1	0.005	5.12 ± 1.3	7.73 ± 1.9	0.008
CK (U/L)	142.2 ± 39.8	220 ± 70.1	0.005	77.7 ± 33.1	111.3 ± 39	0.008
LDH (U/L)	101 ± 33.5	224.5 ± 74	0.005	88.7 ± 24.7	216.1 ± 45.1	0.008
Hct (%)	40.3 ± 2.8	41.5 ± 5.1	0.575	40.1 ± 1.8	40.9 ± 2.1	0.441
T (sn)	77.61±10.8	72.6 ± 9.35	0.017	80.33± 6.47	78.13 ± 6	0.015

The comparison of biochemical tests value*s* before and after acute swimming exercise showed that MDA level before the acute swimming exercise 9.8±1.9 nmol/ml at male swimmers and 7.55±1.05 nmol/ml, $p<0.01$ at female swimmers. The values after the acute swimming exercise were found as significantly lower at male (9.4±2.4 nmol/) and female (6.58 ± 0.9 nmol/ml, $p<0.01$) swimmers

While CK activity before the acute swimming exercise was found as 142.2±39.8 U/L, $p<0.01$ at male and 77.7±33.1 U/L, $p<0.01$ at female swimmers after the acute swimming exercise this value was found as significantly higher at male and female swimmers respectively (220±70.1 U/L and 111.3±39 U/L, $p<0.01$)

The GPx activity was found as significantly lower (32.2+_11.3 U/gr Hb and 20.8±6.1 U/gr Hb, $p<0.05$) at male and female swimmers respectively) before the acute swimming exercise and after the acute swimmimg exercise the value of GPx level was not found as statistically significant. No significant difference was observed in the other variables (Table 4).

Table 4. Before and after acute swimming (BASE-AASE). Data are means ±SD
(Tablo 4. Akut yüzme öncesi ve sonrası BASE-AASE değerleri)

Variables	Before - After Acute Swimming Exercise (BASE- AASE)	Boys (n= 10)	Girls (n= 9)
MDA (nmol/ml)	BASE	9.8±1.9	7.50±1.05
MDA (nmol/ml)	AASE	9.4±2.4	6.58±0.98
E-MDA (nmolMDA/gr Hb)	BASE	105.4±11	108±12.1
E-MDA (nmolMDA/gr Hb)	AASE	105±12.5	104.2±7.6
GSH (mg/dL Eritrosit)	BASE	66.1±4.3	69±6.9
GSH (mg/dL Eritrosit)	AASE	72.6±12.1	64±5.68
SOD (U/gr Hb)	BASE	1263±168	1405±170
SOD (U/gr Hb)	AASE	1246±205	1443.6±390
GP x (U/gr Hb)	BASE	32.2±11.3	20.8±6.1
GP x (U/gr Hb)	AASE	30.6±4.3	31.9±10.1
KAT (U/L)	BASE	4.60±1.1	5.12±1.3
KAT (U/L)	AASE	7.07±1.1	7.73±1.9
CK (U/L)	BASE	142.2±39.8	77.7±33.1
CK (U/L)	AASE	220±70.1	111.3±39
LDH (U/L)	BASE	101±33.5	88.7±24.7
LDH (U/L)	AASE	224.5±74	216.1±45.1
Hct (%)	BASE	40.3±2.8	40.1±1.8
Hct (%)	AASE	41.5±5.1	40.9±2.1
T (sn)	BASE	77.6±10.8	80.3±6.4
T (sn)	AASE	72.6±9.3	78. ±6

5. DISCUSSION AND CONCLUSSION (TARTIŞMA VE SONUÇ)

To examine acute oxidative stress in response to exercise, most researchers have assessed various stress markers in blood and urine. Few studies have examined oxidative stress in muscle tissue, plasma and blood of humans in response to bout of exercise for training [23 and 24].

In this study we investigated the biochemical responses of adolescent swimmers to acute exercise. Adolescent swimmers were tested after 16 weeks of training and enzyme levels and times were recorded (Table 2).

Lipid peroxidation is a complex phenomenon [25], involving the generation of many products. However, the content of malondialdehyde (MDA), one of most important end-products of lipid peroxidation, in the tissues is usually accepted as an index of lipid peroxidation intensity.

In this study when the pre- and post-race values were compared plasma MDA levels were found significantly lower in females (Table 3). The rate of lipid peroxidation did not change significantly that suggests a balance between ROS-induced damage to lipids and antioxidant-repair systems. The fact that the changes in the measured oxidative damage indices of proteins and lipids are not consistent. Glutathione is generally considered to be a multifunctional antioxidant which links the different scavenger systems (lipophilic/hydrophilic, intracellular/extracellular). As a consequence, glutathione content in the blood may be used as one of the indices of the total antioxidant capacity of the body [26 and 27]. The glutathione content (Table 3) in the blood confirms the above because it shows a trend similar to that of MDA. After an oxidative stress, glutathione is released in the blood; the oxidized form (GSSG) is transferred from the cells to the liver to be reduced and the

reduced form (GSH) is then released by the liver to support increased requirement of cells for this substrate which is necessary for the activity of glutathione peroxidase [12].

One consensus on the effect of exercise among researchers is the observation of increased tolerance to oxidative stress by training. Lower levels of lipid peroxidation are found in trained versus untrained subjects. One possibility for this outcome may be related to the boosting of defense systems against oxidative damage by inducing antioxidative, scavenging enzymes. Recent data on the antioxidant system in rat brain regions in its response to exercise training showed increased GSH peroxidase and catalase produced in the hippocampus [28] (Devi and Kiran, 2004). These investigators also showed that although training exercise had little impact on hepatic and myocardial antioxidant defenses, it induced adaptive responses in skeletal muscle, particularly increasing glutathione peroxidase [29].

In our study, when the GPx enzyme activity was examined, a non-significant increase in males and a significant increase in females were observed. In both groups, no change was observed in SOD enzyme activity. Interestingly KAT enzyme activity was found significantly different in both groups. We surmise that this increased may be an adaptive response to keep the increased hydrogen peroxide (H_2O_2) levels low after short term swimming exercise. One reason for females to better cope with oxidative stress after 16 weeks of training programme can be the oestrogen hormone. Female rats have been shown to better tolerate exercise induced oxidative injury than male rats in cases of vitamin E deficiency [30 and 31]. Furthermore, the muscle enzyme CK and LDH levels were remarkably increased in post training group as expected [32]. We believe that this increase may be due to the decreased cystein levels in blood, although no measurements were done in our study [33] Kinnaert et al., 2004 have reported that cys play important role reaching within the cells resulting in a cellular defense against oxidative stress. Furthermore, GSH is endogenously synthesized from cys in the liver and is exported to blood and other tissues as a source of antioxidant protection. Depletion of GSH resulting in decraesed cys levels. Depletion of GSH resulting in increased susceptibility of the cell to oxidative stress. The increased production of oxygen species, as represented by the low antioxidant system levels in the players during training, is formed in vivo as byproducts and intermediate of aerobic metabolism and during oxidative stress [34 and 35].

As a conclusion, we suggest that males and females give different responses to oxidative stress after training for swimming races. We can say that tolerance develops to oxidative stress induced by 100 m free style swimming as indicated by the biochemical responses observed in the blood and plasma biochemical values. This is possibly due to different mechanisms or different activities of specific antioxidant and repair systems.

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