Effects of exercise and vitamin E on lipid profile and hematological parameters in young sportsmen*

Genç sporcularda lipit profili ve hematolojik değerler üzerinde egzersiz ve E vitamini etkisi*

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Abstract

Background: Exercise may increase the generation of free oxygen radicals and lipid peroxidation. In investigation, it has been shown that strenuous exercise induces oxidative damage of lipid and lipoproteins and leads to enhanced muscle injury. Vitamin E is one of an important antioxidant and dietary vitamin E may decrease the exercise-induced increase in the rate of lipid peroxidation. Therefore, this investigation was carried out to investigate possibly effects of exercise and vitamin E on markers of oxidative stres, the values of lipids, some hematological and biochemical parameters in young sportsmen.

Methods: This study was carried out on twenty two male volunteer sportsmen. Young sportsmen were divided to control, exercise, exercise plus vitamin E (100 mg/day) groups and they trained an hours in one day for three days in a week. Biochemical parameters were analysed by authoanalyser and blood cells were determined in cell-counter. Plasma lipid peroxides (TBARS) were determined spectrophotometrically.

Results: While the levels of high-density lipoprotein-cholesterol were increased ($p<0.05$), the values of cholesterol and low-density lipoprotein-cholesterol were significantly decreased ($p<0.05$) with exercise and vitamin E treatment according to the control group. The ratios of high density lipoprotein cholesterol/cholesterol and high-density lipoprotein-cholesterol/low density lipoprotein-cholesterol were significantly increased ($p<0.05$ to $p<0.01$, respectively) after exercise and vitamin E. While TBARS was increased ($p<0.05$) with exercise, it was decreased ($p<0.05$) with vitamin E supplementation. In addition, the counts of erytrocytes and platelets, and hematocrit values (%) were significantly decreased ($p<0.01$, $p<0,01$, $p<0.05$, respectively), whereas the values of mean corpuscular volume were significantly increased ($p<0.05$, respectively) after exercise and the ingestion of vitamin E.

Conclusions: We have showed that exercise and vitamin E affected total cholesterol, high-density lipoprotein-cholesterol, low-density lipoprotein-cholesterol, the lipid peroxidation and some hematological parametrs. In light of these results, exercise and vitamin E may play a role in the increasing of high-density lipoprotein-cholesterol and decreasing of low-density lipoprotein-cholesterol. Lipid peroxids were increased with exercise, they were decreased with vitamin E supplementation. Therefore, vitamin E may play an important role in the prevention of oxidative damage produced by free radicals.

Key words: exercise, vitamin E, lipids, hematological parameters

Özet

Amaç: Egzersiz serbest oksijen radikalleri ve lipit peorksidasyonunu artırabilir. Araştırmalarda, aşırı egzersizin lipit ve lipoproteinlerde oksidatif hasarı artırarak kas hasarında ilerlemeye neden olduğu ileri sürülmektedir. Vitamin E önemli bir antioksidandır ve diyete ilavesi, egzersiz ile indüklenen lipid peroxidasyon hızını azaltabilir. Bu çalışma, genç sporcularda egzersiz ve vitamin E'nin oksidatif stres belirteçleri, lipit değerleri ile bazı hematolojik ve biyokimyasal parametreler üzerine etkilerini araştırmak amacı ile yapıldı.

Materyal ve metod: Bu çalışma yirmiki erkek gönüllü sporcu üzerinde yürütüldü. Genç sporcular kontrol, egzersiz ve egzersiz+vitamin E (100 mg/gün) gruplarına ayrıldı ve çalışma haftada üç gün, günde bir saat egzersiz yaparak gerçekleştirildi. Biyokimyasal değerler otoanalizörde, kan hücreleri ise cell-counterda belirlendi. Lipit peroksidasyonu spektrofotometrik yöntemle analiz edildi.

Bulgular: Kontrol grubuna göre, egzersiz ve vitamin E grubunda yüksek dansiteli lipoprotein kolesterol artarken ($p<0,05$), düşük dansiteli lipoprotein kolesterol değerleri azaldı ($p<0,05$). Egzersiz ve E vitamini alımı ile yüksek dansiteli lipoprotein kolesterol/kolesterol ve yüksek dansiteli lipoprotein kolesterol/düşük dansiteli lipoprotein kolesterol oranlarının anlamlı (sırası ile $p<0,05$, $p<0,01$) bir biçimde arttığı belirlendi. Lipit peroksidasyonu egzersiz ile artarken ($p<0,05$), E vitamini alınması ile anlamlı olarak ($p<0,05$) azaldı. Ayrıca, egzersiz ve E vitamini alınması ile alyuvar sayıları, % hematokrit değerleri ve trombosit sayıları anlamlı olarak azalırken ($p<0,01$, $p<0,01$, $p<0,05$), ortalama alyuvar hacminin arttığı ($p<0,05$) gözlemlendi.

Sonuç: Egzersiz ve E vitamini alınması ile toplam kolesterol, yüksek dansiteli lipoprotein kolesterol, düşük dansiteli lipoprotein kolesterol, lipit peroksidasyonu ve bazı hematolojik değerlerin etkilendiği belirlendi. Bu verilere göre, egzersiz ve E vitamini uygulanması ile yüksek dansiteli lipoprotein kolesterol değerlerinin arttığı, düşük dansiteli lipoprotein kolesterol değerlerinin azaldığı belirlenmiştir. Egzersizin lipit peroksitleri artırdığı, vitamin E'nin ise azalttığı saptanmış, bu nedenle vitamin E'nin serbest radikallerin neden olduğu oksidatif hasarı önlemede önemli bir rol oynayabileceği düşünülmüştür.

Anahtar kelimeler: egzersiz, vitamin E, lipitler, hematolojik değerler.

Introduction

Physical activity increases the production of reactive oxygen species and plasma total peroxide levels during and after the exercise (1,2). In both trained and untrained individuals, it has been investigated the reletionships between antioxidant supplements and exercise-related oxidative stress (3-7). Exercise training is known to induce an increase in free radical production potentially leading to enhanced oxidative injury. Some investigations have reported that supplementation with vitamin E or antioxidant mixtures can reduce the indicators of oxidative stress as a result of exercise (4-7). Oxidative damage of cellular components such as enzymes, nucleic acids and proteins may be an important factor in cell injury (8). Vitamin E is a chain-breaking antioxidant in cellular membranes (9-11).

Numerous studies have been reported relationships between plasma lipids and the different exercise trainings (12-17). Vitamins E is known as an antioxidant that has beneficial effects in the prevention of muscle cell damage produced with oxidative stres (4,5). The indications of vitamin E has been investigated and several procedures were used to determine some effects of exercise and this vitamin on the levels of high-density lipoprotein-cholesterol (HDL-C) and low-density lipoprotein-cholesterol (LDL-C) and other some hematological parameters (10, 18-21). Low levels of HDL-C are one of the important risk factors for cardiovascular diseases (22-24). In addition, it has been reported that endurance training affected the levels of HDL-C and LDL-C (12,13) and minerals (3,25,26).

It is not fully known whether the body's natural antioxidant defense system is sufficient to counteract the increase in free radicals with exercise or whether additional supplements are needed. Therefore, the purpose of this study was to investigate the effects of oral administration of vitamin E and exercise on lipid profiles, some hematological and biochemical parameters in amateur sportsmen.

Methods

Subjects, exercise and treatment: This study was carried out on twenty two male volunteer young sportsmen of the School of Pysical Education and Sports, Harran University, Şanlıurfa. None of the students had obvious health problems and all were non-smokers. All students informed about the aim the study prior to giving their consent. The volunteers had body weights of 67.66±1.52 kg, 23.50±0.59 years old and 1.74±0.03 m heights. The first group (n=7) was examined as a control. The second group (n=7) was only exposed to exercise. The third group (n=8; Exercise+vit. E) was exposed to exercise and vitamin E (100 mg/day) was administered per oral. The sportsmen trained an hours in one day for three days in a week. Local Medical committee responded for using of vitamins and general condition of the sportsmen.

Obtaining of blood and plasma samples: The whole blood of all students was drawn into vacutiner tubes (Beckon Dickinson System, France) containing disodium salt of ethylenediaminetetraacetic acid (EDTA) as anticoagulant. The blood samples from ante-cubital veins (0.5 ml) in the tubes were used for analysis of hematological parameters, and the other portion of the blood was centrifuged (1500 g, 15 min; Heraus Inst., Mega Fuge 1.0), and their plasma was removed using disposable pipettes.

Biochemical and hematological parameters: Levels of cholesterol (CHOL), high density lipoprotein-cholesterol (HDL-C), low density lipoprotein-cholesterol (LDL-C), triglyceride (TG), glucose (GLU), urea (URE), creatinin (CRE), total bilirubin (T-BIL), direct bilirubin (D-BIL), indirect bilirubin (I-BIL), total protein (TP), albumin (ALB), globulin (GLOB), aspartate transaminase (AST), alanin transaminase (ALT), gamma gluthamile transaminase (GGT), alkaline phosphotase (ALP), amylase (ALZ), creatine phosphokinase (CPK) and lactate dehydrogenase (LDH), asit-pH, iron (Fe), iron percent saturation (Fe%), transferrin, calcium (Ca), sodium (Na), potassium (K), chloride (Cl) and magnesium (Mg) were determined by automated chemistry analyser (Boehringer Mannheim) using commercially available kits in all plasma samples. Plasma lipid peroxides (TBARS) was determined spectrophotometrically as described previously (27). In addition, ratios of CHOL/HDL-C, HDL-C/CHOL and HDL-C/LDL-C were calculated for the values of CHOL, TG, HDL-C and LDL-C.

Erythrocytes counts (RBC), hemoglobin concentration (HGB), hematocrit values (HCT%), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentrations (MCHC), platelets (PLT), leukocyte counts (WBC), lymphocyte counts (LYM), neutrophil (NEU), and their lymphocyte and neutrophil rates (%) were determined using cell counter (Cell-Dyn 3500R) in the blood samples.

Statistical analysis: Statistical analysis was carried out using the SPSS 11.5 statistical program (SPSS Inc., Chicago, IL, USA). For the analysis of hematological and biochemical parameters, Kruskal–Wallis test and Mann–Whitney U tests were used for analysis of variance and post hoc test, respectively. The data were expressed as means ± standard errors (SE), and differences on the statistical analysis of results were

considered to be significant at $p<0.05$.

Results

Biochemical and hematological parameters and their comparisons among all groups are presented in Tables 1-3. No significant changes were observed in the all volunteers' height and loss weight over the four-week period ($p>0.05$). While the levels of HDL-C were increased ($p<0.05$), the values of CHOL and LDL-C were significantly decreased ($p<0.05$) in exercise and vitamins E treatments groups than the control group. Administration of vitamin E was decreased the CHOL and LDL-C. While the ratios of CHOL/HDL-C and LDL-C/HDL-C were significantly decreased ($p<0.05$, respectively), the ratios of HDL-C/CHOL and HDL-C/LDL-C were significantly increased ($p<0.05$) after exercise and vitamin E treatments. While plasma TBARS contentration as lipid peroxidation was increased ($p<0.05$) with exercise, it was decreased ($p<0.05$) by vitamin E supplementation (Table 1).

The values of Ca, Na, Cl, Mg and Fe in exercise and vitamin E groups significantly decreased ($p<0.05$ to $p<0.01$) than the control groups. In addition, the values of RBC, HCT and PLT were significantly decreased ($p<0.01$, $p<0.01$ and $p<0.05$, respectively), whereas the values of MCV were significantly increased ($p<0.05$) by exercise and the treatments of vitamin E (Table 2). However, levels of TG, GLU, URE, CRE, T-BIL, D-BIL, I-BIL, TP, ALB, GLOB, AST, ALT, GGT, ALP, ALZ, CPK, LDH, ASIT-PH, Fe, Fe%, transferrin in plasma of all the footballers were not statistically influenced ($p>0.05$) (Table 2 and 3).

Discussion

Lipids and lipoproteins are particularly susceptible to free radical-mediated peroxidation, and oxidative damage of these components may play an important role in early period of pathogenesis in different stresses (1,2,8,23). Several factors, such as obesity, dietary lipids, carbohydrates, alcohol, smoking, hormones, various drugs and physical activity may alter HDL-C levels in humans. LDL-C carries the major component of plasma cholesterol responsible for transportation of cholesterol to peripheral tissues and also HDL-C carries the blood borne cholesterol responsible for transportation of extra hepatic cholesterol to LDL-C, or to the liver for degradation (22). Some investigations have been performed to explain the possible mechanism of the relations in serum lipids with exercise (12-14,16,17) and using of antioxidant vitamins (3-6,11,18,20,21). One of these mechanisms may be an increase in the transfer of cholesterol from lipoproteins and tissues to HDL-C as due to increase in lecithin cholesterol-acyltransferase activity induced by exercise. The other hand, exercise may decrease the serum levels of LDL-C and total triglyceride. Decrease of these biochemical substances may be due to the increase of triglyceride uptake by the tissues because of the increase in lipoprotein lipase (LPL)-activity after exercise in men and animals (22,24).

Training of longitudinal physical activity has elevated HDL-C and reduced LDL-C in serum. Low levels of HDL-C and high levels of LDL-C are two important risk factors for cardiovascular diseases. The intensity, period of the sample collections and of the exercise training is important factors that are positively associated with increases in serum levels of HDL-C and decrease of LDL-C. It has been reported that ratios of total cholesterol/HDL-C and LDL-C/HDL-C may be an important factor for coronary artery diseases. Decrease of the two ratios is considered as lower risk for cardiovascular disease than that of total cholesterol and only HDL-C. This effect of long time on lipids may minimize by analyzing all samples after a few hours from exercise (12,13).

Lipid peroxidation in plasma was increased ($p<0.05$) with exercise, but it was decreased ($p<0.05$) with vitamin E treatment (Table 1). While the levels of HDL-C were significantly increased ($p<0.05$), the values of CHOL and LDL-C were statistically decreased ($p<0.05$ to $p<0.01$, respectively) after vitamin E treatment according to the control group (Table 1). The increase in plasma HDL-C levels in footballers with the exercise performed is confirmed with the results of the studies in this subject (1,2). Therefore, exercise training may diminish the risk of coronary artery diseases. These results are in agreement with the results of the studies performed with exercise (13,15-17) and vitamin E (4,5,9,21).

Vitamin E is an important antioxidant that diminishes the peroxidation of unsaturated lipids by chain-breaking free radicals; thus it contributes stability to cellular membranes and creates the phospholipid stability in cellular membrane (11,20, 22,24). The values of CHOL, HDL-C and LDL-C (Table 1) were corroborated by the results of some references (9,11,19)) reported to relation with the effects of exercise training, treatments of vitamins C and E. Muscular exercise promotes the production of reactive oxygen species in the working muscle. Growing evidence indicates that reactive oxygen species are responsible for exercise-induced protein oxidation and contribute to muscle fatigue. To protect against exercise-induced oxidative injury, muscle cells contain complex endogenous cellular defence mechanisms (enzymatic and non-enzymatic antioxidants) to eliminate reactive oxygen species. Exogenous dietary antioxidants interact with endogenous antioxidants to form a defence by cellular antioxidants (11,20). In light of these considerations,

vitamin E may play an important role in the prevention of oxidation of lipids and lipoproteins, and in prophylaxis of cardiovascular diseases produced by different stresses. Corrective and profilactic treatments can be made with received vitamin E for increasing of physical performance (4,7). These considerations are in accordance with the results of the studies performed to investigate the effects of some mineral and antioxidant supplementations on exercise performance (3-7,28,29). In addition, the values of some hematological parameters are in general resembles with the results of studies performed by exercise and biochemical parameters (26). Indeed, vitamin E is known for its antioxidant quality in the inhibition of the oxidative processes of lipids and lipoproteins in cell membranes in beginning of some tissue injury (11,20,23).

In conclusion, exercise and vitamin E may play a role in the increasing of high-density lipoprotein-cholesterol and decreasing of low-density lipoprotein-cholesterol and they may play an important role in the prophylactic indication for oxidative injury produced in lipids and lipoproteins by reactive oxygen species (O2-, OH and HOCl) and other free radicals like cytokines. Thus, vitamin E can modulate the increasing of lipid peroxidation produced by the different oxidative stresses. However, there is a need for more detailed studies in order to assess possible relationships between exercise and antioxidants. Therefore, we are presently ongoing such our studies investigating possible protective roles of antixidant vitamins against different stresses affecting lipids and lipoproteins.

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Table 1. Mean values and comparison of the lipid profile in all groups[1].

Parameters	Control (n=7)	Exercise (n=7)	Exercise+Vit. E (n=8)
CHOL, $mg.dl^{-1}$	157,28 ± 4.10	136,30 ± 4.65[a]	143.46 ± 5.49[a]
HDL-C, $mg.dl^{-1}$	39.02 ± 2.24	45.85 ± 2.37[a]	45.14 ± 1.65[a]
LDL-C, $mg.dl^{-1}$	110.75 ± 2.51	92.71 ± 2.46[a]	92.57 ± 2.10[a]
TG, $mg.dl^{-1}$	87.00 ± 2.58	94.72 ± 8.33	97.75 ± 5.80
TBARS, µmol/L	1.21 ± 0.08	1.34 ± 0.21[a]	1.16 ± 0.09[†]
CHOL/HDL-C	4.10 ± 0.22	3.01 ± 0.18[a]	3.29 ± 0.23[a]
HDL-C/CHOL	0.25 ± 0.01	0.32 ± 0.02[a]	0.32 ± 0.02[†]
HDL-C/LDL-C	0.37 ± 0.02	0.50 ± 0.07[†]	0.52 ± 0.01[†]
LDL-C/HDL-C	2.62 ± 0.11	2.02 ± 0.10[a]	2.05 ± 0.07[a]

[1]Data are expressed as means and standard errors.

Statistical significant according to control group, [a]p<0.05.

Statistical significant according to exercise and vitamin E groups, [†]p<0.05.

Table 2. Mean values and comparison of the hematological parameters in all groups[1].

Parameters	*Control* (n=7)	*Exercise* (n=7)	*Exercise+Vit.E* (n=8)
RBC, $x10^6.\mu l^{-1}$	5.12± 0.85	4.80± 0.19[b]	4.52± 0.11[b]
HGB, $g.dl^{-1}$	14.69± 0.26	15.05± 0.42	14.15± 0.38
HCT, %	44.44± 0.75	41.20±1.01[b]	41.04± 0.13[b]
MCV, μm^3	86.81± 0.55	91.08±1.61[a]	90.57± 0.46[a]
MCH, pg	28.96± 0.34	31.50± 0.65	31.27± 0.13
MCHC, $g.dl^{-1}$	33.02± 0.32	34.58± 0.35	34.48± 0.26
PLT, $10^3/mm^3$	206.31± 5.21	179.00± 4.29[b]	194.28± 5.20[a]
WBC, $x10^3.\mu l^{-1}$	6.19± 0.29	6.31± 0.29	5.90± 0.47
LYM, $x10^3.\mu l^{-1}$	2.05± 0.11	2.14± 0.25	1.98± 0.24
NEU, $x10^3.\mu l^{-1}$	3.91± 0.27	3.78± 0.43	3.12± 0.37
LYMP, %	33.21± 1.67	34.47± 4.42	35.78± 0.06
NEUTR, %	62.75± 1.91	58.61± 4.30	55.37± 0.80

[1]Data are expressed as means and standard errors.

Statistical significant according to control group, [a]p<0.05, [b]p<0.01.

No significant according to exercise plus vitamins E.

Table 3. Mean values and comparison of the biochemical parameters in all groups[1].

Parameters	*Control* (n=7)	*Exercise* (n=7)	*Exercise +Vit. E* (n=8)
AST, U.l^{-1}	33.62 ± 4.73	31.57 ± 2.75	31.85 ± 3.23
ALT, U.l^{-1}	26.37 ± 3.17	24.28 ± 2.15	31.00 ± 4.00
GGT, U.l^{-1}	17.44 ± 1.63	15.57 ± 3.23	19.71 ± 3.56
ALP, U.l^{-1}	200.56 ± 11.82	198.42 ± 19.26	227.14 ± 21.16
ALZ, U.l^{-1}	178.37 ± 11.16	180.71 ± 33.71	201.28 ± 31.04
CPK, U.l^{-1}	546.37 ± 104.2	314.28 ± 88.86[b]	276.85 ± 76.79[b]
LDH, U.l^{-1}	358.12 ± 0.18	335.14 ± 2.30	345.00 ± 16.33
GLU, mg.dl^{-1}	94.50 ± 3.27	87.28 ± 2.02	90.28 ± 1.86
ÜRE, mg.dl^{-1}	31.87 ± 2.02	30.42 ± 2.10	31.28 ± 2.03
CRE, mg.dl^{-1}	1.09 ± 0.27	1.20 ± 0.34	1.02 ± 0.22
T.BIL, mg.dl^{-1}	1.14 ± 0.12	0.86 ± 0.69[a]	0.55 ± 0.26[a]
D.BIL, mg.dl^{-1}	0.59 ± 0.49	0.29 ± 0.11[a]	0.14 ± 0.22[a]
I.BIL, mg.dl^{-1}	0.54 ± 0.25	0.57 ± 0.13	0.51 ± 0.24
TP, mg.dl^{-1}	7.70 ± 0.12	7.70 ± 0.11	7.74 ± 0.14
ALB, mg.dl^{-1}	5.49 ± 0.80	5.54 ± 0.25	5.42 ± 0.28
GLOB, mg.dl^{-1}	2.19 ± 0.77	2.20 ± 0.11	2.31 ± 0.27
ASIT-PH	3.93 ± 0.21	3.17 ± 0.32	3.16 ± 0.21
Fe, µg.dl^{-1}	150.31 ± 10.66	98.42 ± 15.51[b]	97.28 ± 9.13[b]
Fe%	39.25 ± 3.08	23.57 ±1.90[b]	23.14 ± 2.29[b]
Transferrin mg.dl^{-1}	287.87 ± 10.26	296.57 ± 43.67	320.00 ± 18.30
Ca, mg.dl^{-1}	9.59 ± 0.78	8.87 ± 0.13[a]	8.80 ± 0.17[a]
Na, meq.L^{-1}	144.87 ± 0.57	140.57 ± 0.57[a]	141.00 ± 1.36
K, meq.L^{-1}	4.50 ± 0.99	4.47 ± 0.84	4.44 ± 0.12
Cl, meq.L^{-1}	109.75 ± 0.42	106.57 ± 0.89[a]	107.42 ± 0.84
Mg, mg.dl^{-1}	1.96 ± 0.52	1.88 ± 0.75[a]	1.55 ± 0.25[b]

[1]*Data are expressed as means and standard errors.*

Statistical significant according to control group, [a]p<0.05, [b]p<0.01.

No significant according to exercise plus vitamin E.

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