

IMPACT OF 6 WEEKS OF INTENSIVE INTERMITTENT TRAINING WITH TAKING VITAMIN E ON P53 CHANGES IN BLOOD SERUM LEVELS AND VISCERAL ADIPOSE TISSUE IN SPRAGUE–DAWLEY RATS

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Abstract

There is little information about how to modify the P53 protein in response to exercise activity in different tissues. The aim of this study is to evaluate the impact of 6 weeks of intensive intermittent training with and without vitamin E supplement on serum P53 and visceral adipose tissue changes in Sprague-Dawley male rats. For this purpose, 32 Sprague-Dawley male rats at 8 - 10 weeks of age were randomly divided into 5 groups: 1) intensive intermittent training (IIT, n = 7), 2) intensive intermittent training + vitamin E (IIT + VE, n = 7), 3) Vitamin E (VE, n = 6), 4) control (CON, n = 6), 5) Sham (S, n = 6). Training protocol of this study was implemented for 6 days per week for 6 weeks, with observing the overload principle on the treadmill. Biochemical analysis of P53 protein was performed by ELISA. For data analysis, one way analysis of variance was used. The results showed that there is a significant difference between the P53 protein in the blood serum of rat that underwent only intensive intermittent training compared with blood serum in the control group ($P < 0.002$). In contrast, no significant difference was observed in the adipose tissue P53 protein research groups. Intensive intermittent training can increase protein P53 level in blood serum, but does not have a significant effect on visceral adipose tissue. Also, taking too much vitamin E in normal diet has no significant effect on changes in levels of P53.

Key words: P53 protein, intensive intermittent training, vitamin E, blood serum, visceral adipose tissue

Introduction

Scientific evidence now suggests that maintaining a *healthy diet pattern* with regular physical activity can be an important factor in the prevention of many diseases (Warburton, Nicol & Bredin, 2006). In this regard, there is considerable evidence that physical activity reduces the risk of several types of cancer (Peel et al., 2009; Laukkanen et al., 2010). Genes-activation associated with disease prevention is one of the important factors in disease prevention. A tumor suppressor gene, or anti-oncogene, is a gene that protects a cell from one step on the path to cancer. Tumor protein p53, also known as p53, is responsible for encoding the P53 protein in cells. Regardless of its isoforms, this protein is expressed in almost all tissues of the body that its elevation is associated with increased levels of blood transfusion (Lutz & Nowakowska-Svwrta, 2002; Bourdon et al., 2005). It has been found that P53 protein expression plays an important role in the development of insulin resistance in adipose tissue that can cause cardiovascular, metabolic diseases and consequently aging (Minamino et al., 2009).

In fact, by regulating the expression of many genes, such as TIGAR, G6PD, TFAM and P21, P53 protein plays a role in the inhibition of glycolysis and reduced anabolic activity, enhanced aerobic metabolism and mitochondrial biogenesis, increased apoptosis and cell genome stability.

Results of the studies showed that the biochemical pathways that are stimulated by P53 are the most important factors that will activate anti-cancer biochemical pathways (Wang et al., 2012). Scientific evidence has shown that change in normal cell homeostasis increases p53 gene expression and more activation of the protein. Change in cellular redox is one of the most important of these changes, which are affected by the increase or decrease oxidative stress. In fact, physical exercise with increased oxidative stress in muscle cells can activate different biochemical waterfalls including P38-MAPK pathway as well as a group of protein kinase such as ATM that activates p53 protein (Wang et al., 2012). On the other hand, it has been found that the intensity and duration of aerobic exercise have an important role in determining the level of oxidative stress (Yu, 2016; Minyi et al., 2007). In *increased oxidative stress response*, P53 as a transcription factor enhances expression of various antioxidant genes such as glutathione peroxidase-1 (GPX-1) and in fact *regulates cellular redox homeostasis* (Wang et al., 2012; Han et al., 2008). Moreover, in addition to antioxidant enzymes, body can decrease oxidative stress by antioxidant molecules, like vitamin E (Ham & Liebler, 1995). Apart from the antioxidant properties of vitamin E, the evidence show that this vitamin has other positive effects in regulating gene expression and thus the production of specific

proteins in the cell. In this regard, Schwartz et al. (1993), have shown that vitamin E can cause over expression of P53 protein and reduce the risk of cancer (Shwartz et al., 1993). In another study by Beilby et al. (2010), no relationship was observed between serum concentration of vitamin E and P53 changes (Beilby et al., 2010). On the other hand, it seems that aerobic exercise after a long period of 10 years can increase the amount of P53 protein in lymph nodes cancer cells of prostate (LNCaP) by decreased amount of IGF-1 and increased IGFBP-1 levels (Leung et al., 2004). In relation with the effect of resistance training on *blood-serum p53* concentrations, Sharafi and Rahimi (2012), have demonstrated that serum level of P53 protein immediately after resistance training in people who have underwent one year of resistance training, is significantly less compared to those who are not compatible with this type of training. However, no significant difference was found in serum levels of P53 protein between the two groups several hours after resistance exercise. Their results showed that resistance training can change biomarkers of apoptosis particularly intrinsic pathway of apoptosis, which is affected by the P53 protein (Sharafi & Rahimi, 2012). Thus, according to the different results of past studies and the interaction between P53 protein and oxidative stress as well as the effect of aerobic exercise on oxidative stress and the amount of IGF-1 in blood circulation and also the importance of the antioxidant vitamin E against oxidative stress, it seems that the current study is noticeable regarding the limited literature in this field. Thus, the aim of this study is to evaluate the impact of 6 weeks of intensive intermittent training with and without vitamin E supplement on serum P53 and visceral adipose tissue changes in Sprague-Dawley male rats.

Methods

Subjects

In this study, 32 Sprague-Dawley male rats at 8 - 10 weeks of age were selected and after transferring to the research setting and familiarity with the treadmill, were randomly assigned into five groups: 1) intensive intermittent training (IIT, n = 7), 2) intensive intermittent training + vitamin E (IIT + VE, n = 7), 3) Vitamin E (VE, n = 6), 4) control group (CON, n = 6), 5) Sham (S, n = 6). These animals were purchased from *laboratory animal breeding center, Shiraz University of Medical Sciences* and were kept in polycarbonate cages (4 rats per cage) in controlled environmental conditions with an average temperature of 22 ± 3 °C, relative humidity 30 to 70%, 12/12-h light/dark cycle with free access to food and water for laboratory animals. This study was approved by the ethics Committee of the School of Medicine Sciences, Shiraz University.

Vitamin E Supplementation

In this study, 25-gram succinate package of Sigma Company ((+) - α - Tocopherol acid succinate, Sigma-Aldrich) was used. Six days per week and three hours before the implementation, 60 mg of

vitamin E per kg body weight was given to rats of VE, IIT + VE groups by gavage (Metin et al., 2002; Valenca et al., 2008). Sesame oil was used to prepare vitamin E (60 mg in 1 ml of sesame oil) (Malafa et al., 2002). Additionally, 1 ml sesame oil was given for sham group rats per kg of body weight by gavage. It should be noted that the rats were weighed on the first day of every week and were supplemented with vitamin E based on weight during the week. Also, in order to control the effect of sesame oil, subjects of sham group received sesame oil without vitamin E similar to subjects of supplement groups.

Exercise training protocol

After transferring subjects in the research setting, they began to train with a speed of 16 meters per minute for 10 minutes on the treadmill for 10 days to adapt to the new environment and familiarity with the treadmill. The intensive intermittent program was implemented for 6 weeks in 3 even and odd days on the treadmill for animals (manufactured by *PISHRO Industry Company*). Training protocol for even days consisted of 3 minutes at 40 m/min followed by intervals of running at 16 m/min for 1 minute. Initially two repetitions were performed and increased to six repetitions by the 4th week. These intensities were subsequently maintained for 6 weeks. Training protocol for odd days consisted of 30 seconds at 54 m/min followed by intervals of running at 16 m/min for 1 minutes. Initially three repetitions were performed and increased to 20 repetitions by the 4th week. These intensities were subsequently maintained for 6 weeks.

Sampling and laboratory evaluation

After the training period, 1 rat was removed from each of the training groups for a variety of reasons, including injury and death. 48 hours after the last training session at 8 am, rats were anesthetized by intraperitoneal injection of a mixture of ketamine (30 to 50 milligrams per kilogram of body weight) and xylazine (3 to 5 milligrams per kilogram of body weight) and blood samples were taken from the heart and then *after centrifugation* (13 mins, 3000rpm), serum was separated from blood samples and were stored at -80 °C for subsequent analysis. In addition, using ELISA kits for rats (manufactured in *Germany's Zell BioCompany*), serum concentrations of P53 ml with sensitivity 1.04 pg/ml was read by Elisa Reader device and according to the manufacturer's instructions of manufactured by HUISONG company in China.

Laboratory evaluation of adipose tissue

After sampling of visceral adipose tissue, the samples were rinsed with physiological serum and stored at -80 °C. Then, a certain amount of tissue was homogenized using PBS solution (PH: 7.4, 100mM) and homogenizer machine according to manufacturer's instructions. After centrifuge (20 min, 4000 rpm), *serum samples were separated* and were again stored at a temperature of -80 degrees Celsius. P53 serum concentrations were then measured using an ELISA kit for rats made in *Germany's Zell BioCompany*.

Statistical analysis of data

In this study, descriptive statistic was used to calculate the mean and standard deviation. one way analysis of variance was used to determine significant difference of P53 levels among the groups, and Bonferroni post hoc test was utilized if it was meaningful. SPSS software (version 23) was used for data analysis, and GrafPad Prism software (version 6) was also used for creating the graphs. The study used a significant level of $P < 0.05$.

Results

Table 1. Bonferroni post hoc test

Variable	Group	Mean	Standard deviation	F	df	SIG
blood-serum p53 (Pg/ml)	Control	116.15	30.52	9.29	9 and 50	0.000
	Intermittent training	159.60	20.33			
	Vitamin E	137.40	14.vlj			
	Intermittent training+ E	135.60	ruj.74			
	Sham	127.73	16.65			
P53 in visceral adipose tissue (Pg/ml)	Control	112.28	13.27	9.29	9 and 50	0.000
	Intermittent training	94.98	stu.94			
	Vitamin E	101.98	14.24			
	Intermittent training+ E	100.20	13.77			
	Sham	102.80	pro.37			

The results showed that the highest average levels of P53 in adipose tissue is related to the control group (112.28 pg/ml) and the lowest levels of P53 in adipose tissue is related to the intensive intermittent training group (94.98 pg/ml). The highest average levels of P53 in the blood sample is related to the intensive intermittent training group (159.60 pg/ml) and the lowest average levels of P53 in blood sample is related to the control group (116.15 pg/ml). Based on the obtained F (9.29) with degrees of freedom 9 and 50, there is a significant difference between the groups in mean levels of P53 ($p < 0.000$) (Table 1). Accordingly, blood sample of intensive intermittent training group (B-IIT) is significantly higher than the blood sample of control group (B-CON) ($p < 0.002$). In addition, P53 level in blood sample of intensive intermittent training group (B-IIT) is significantly higher than all adipose tissue sample groups ($p < 0.000$). On the other hand, P53 level in blood sample of intensive intermittent training group + vitamin E group (B-IIT + VE) is significantly higher than the adipose sample of intensive intermittent training group (A-IIT), Adipose sample of intensive intermittent training + Vitamin E group (A-IIT + VE) and adipose sample of Vitamin E group (A -VE) ($p < 0.005$, $p < 0.026$, $p < 0.045$, respectively). The P53 level in blood sample of Vitamin E group (A-VE) is significantly higher than the adipose sample of intensive intermittent training group (A-IIT), adipose sample of intensive intermittent training group + E Vitamin (A-IIT + VE), adipose sample of Vitamin E group (A -VE) and adipose sample of Sham group (A -S) ($p < 0.002$, $p < 0.014$, $p < 0.026$, $p < 0.033$, respectively) (Fig. 1).

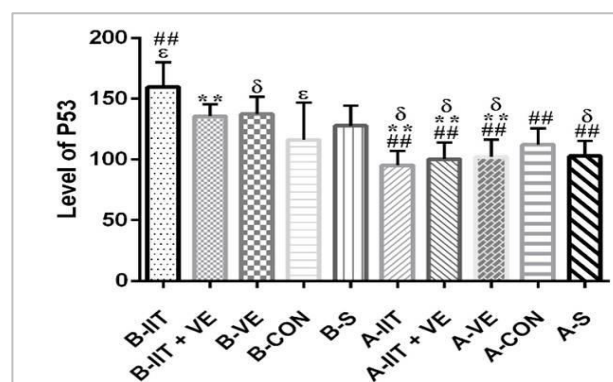


Figure 1. P53 levels

Figure 1 shows the P53 level in the different study groups. After the training protocol, P53 level in blood sample of intensive intermittent training group (B-IIT) was increased significantly compared to blood serum of control group (B-CON). In addition, P53 level in blood sample of intensive intermittent training group (B-IIT) increased significantly compared to all adipose sample groups. On the other hand, P53 level in blood sample of intensive intermittent training group + vitamin E (B-IIT + VE) was increased significantly compared to the groups of adipose samples of intensive intermittent training + vitamin E (A-IIT), Adipose sample of intensive intermittent training + Vitamin E group (A-IIT + VE) and adipose sample of Vitamin E (A-VE) (**). P53 level in blood sample of Vitamin E (B-VE) was increased significantly compared to the adipose sample of intensive intermittent training (A-IIT), adipose sample of intensive intermittent training + Vitamin E (A-IIT + VE), adipose sample of Vitamin E (A-VE) and adipose sample of Sham group (A-S) (δ).

Discussion

The results of the study showed that serum level of p53 protein in group of people with intensive intermittent training was 37.4% higher than the serum level of control group which did not undergo any training. On the other hand, the serum level of p53 protein in blood sample of intensive intermittent training + Vitamin E group (B-IIT + VE) was only 16.74% higher than serum level of p53 protein in the control group (B-CON). This difference in increasing the amount of protein P53 between the two groups of blood sample of intensive intermittent training (B-IIT) and blood sample of intensive intermittent training + Vitamin E group (B-IIT + VE) could possibly be the result of loss adjustment resulting from the use of antioxidant supplements (Yfanti et al., 2011; Paulsen et al., 2014). Although it was shown that the use of antioxidant vitamin E supplements could possibly increase the amount of P53 proteins in specific tissues (Shwartz et al., 1993), according to the results of this study, it appears that P53 protein changes in serum level and visceral adipose tissue as a result of vitamin E or sesame oil can't be dramatic or remarkable. The result of the study regarding the impact of intensive intermittent training on serum level.

Results of protein P53 is consistent with findings of the research by Leung et al. (2004) and is inconsistent with research conducted by Sharafi and Rahimi (2012), Qi et al. (2011) and Schwartz et al. (1993). Due to lack of adequate studies regarding the impact of intensive intermittent training on serum level of P53 protein and adipose tissue, we inevitably referred to the findings of other tissues, as well as other trainings such as continuous workouts for interpreting and analyzing the results. Although we did not measure the serum level of IGF-1, it seems that the IGF-1 level in the circulation is probably an important factor in the regulation of cellular P53 protein (Leung et al., 2004). IGF-1 is produced in many tissues of the body, including cancer cells, but 75 to 80% of circulating IGF-1 is secreted by the liver in the bloodstream. In a study, the researchers reported that IGF-1 can suppress apoptosis via the activation pathway of p38MAPK in cells that their DNA has been damaged. They demonstrated that the suppression of apoptosis by IGF-1 is associated with a decrease in cellular P53 protein, without a change in the *p53 mRNA* (Héron-Milhavet & LeRoith, 2002). It seems that aerobic exercise with moderate to severe intensity can reduce circulating levels of IGF-1 and impacts on the P53 protein in a cell (Leung et al., 2004; Barnard et al., 2003). In their study, Leung et al. (2004), selected a group of healthy *men of the same age* (57 to 64) for the control group who were susceptible to prostate cancer due to sedentary lifestyle and poor eating habits, and selected subjects of training group from among those who were at least 10 years of experience participating in a training program for adults of University of Nevada Las Vegas. The researchers found that intensive continuous training may inhibit prostate cancer cells by changing the components of the IGF axis to increase the *cellular p53 protein levels* (Leung, 2004). Among men who had been participating in a regular training program for more than 10 years, reduced IGF-1 and increased IGFBP-1 levels were observed as compared to men without regular exercise. Both of these changes (reduced serum IGF-1 and increased IGFBP-1) increased P53 protein level to 100% in *prostate carcinoma cells* in *lymphnode* (LNCaP) in vitro. This increase led to a significant increase in apoptosis and decreased the growth of prostate cancer cells. Since all subjects of past and present research were healthy men, the comparison of their results revealed that intensive intermittent training has significant effects on increased serum P53 protein in the short term. However, it seems that observing the impact of continuous workout on *p53 protein* in *prostate tissue* requires a long-term experience of participating in sports programs of continuous intensity. As mentioned in the recent study, an increased level of P53 protein in prostate tissue was observed in those who had 10 years of intensive continuous training. Sharafi and Rahimi (2012) showed in their study that immediately after one *strength training program* session (consisting of 4 sets of 6 exercise at 80% of 1 repetition maximum until failure and 2 minutes' rest between sets and

exercises), serum level of P53 protein was significantly increased in healthy resistance trained men who had at least 1 year of resistance training than healthy untrained men who had a regular resistance training for at least 1 year (Sharafi & Rahimi, 2012). However, no significant change was found in serum level of P53 protein 24 hours after a resistance training session. This result could be due to the fact that plasma level of IGF-1 in the group who had regular resistance training is significantly higher than those who were adaptable with resistance training. Qi et al. (2011), have shown that 30 to 60 minutes of aerobic exercise on a treadmill at a speed of 20 meters per minute 6 days a week for 8 weeks decrease oxidative stress via increasing FSH, COX, COXII and mtDNA markers (Qi et al., 2011). This adaptation is associated with a significant decrease in the amount of P53 protein in the skeletal muscle of type 2 diabetic rats. The results of this study show that signalling related to the reduction of P53 protein through physical activity in skeletal muscle of type 2 diabetic rats, are a type of compatibility which probably contributed with preventing oxidative stress in insulin resistance.

Although the training protocol used in the study by Qi et al. (2011), consisted of 8 weeks of aerobic exercise with a maximum speed of 20 meters per minute for 60 minutes and the training protocol used in our study consisted of continuous training of 6 weeks with a maximum speed of 27 meters per minute for 60 minutes, it seems that the main reason for different results are unhealthy subjects with type 2 diabetes. Because, as it was mentioned, the hormone insulin has an important indirect contribution in the regulation of P53 protein and consequently blood serum. The loss of insulin sensitivity in type 2 diabetes can have negative effects on miscellaneous growth factors including *slight increase* in the *levels of IGF-I* and significant increase in IGF-II and IGFBP-3 as well as a significant decrease in IGFBP-2 (Frystyk et al., 1999). The disruption of normal homeostasis in these hormones and other hormones and most importantly metabolic status of the cells may also be the most important factors that will reduce the P53 protein in muscle tissue. On the other hand, it should be noted that the study by Qi et al. (2011), was performed on muscle tissue, whereas the current study P53 protein was measured in *different tissues*. Thus, the effect of exercise on the level of P53 protein in different tissues could be the focus of future researches.

Conclusion

The results of the study indicated that changes in the blood serum levels of P53 protein are affected by the training protocol. So, it can be concluded that intensive intermittent training is likely to have an impact on P53 protein levels in specific tissues. It was also found that taking too much vitamin E with a normal diet and without malnutrition has no significant effect on P53 protein levels in *visceral adipose tissue* and blood serum.

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UTJECAJ 6-TJEDNOG INTERMITENTNOG TRENINGA S UZIMANJEM VITAMINA E NA PROMJENE U RAZINI P53 U KRVNOM SERUMU I VISCERALNOG ADIPOZNOG TKIVA KOD SPRAGUE-DAWLEY ŠTAKORA

Abstract

Malo je informacija o tome kako modificirati P53 protein kao odgovor na aktivnost vježbanja u različitim tkivima. Cilj ove studije je procijeniti utjecaj 6 tjedana intermitentnog treninga sa i bez vitamina E na serum P53 i promjene visceralnog masnog tkiva u mužjaka Sprague-Dawley štakora. U tu svrhu 32 štakora Sprague-Dawley uzrasta 8 do 10 tjedana po slučajnom ključu je podijeljeno u 5 skupina: 1) intenzivni intermitentni trening (IIT, $n = 7$), 2) intenzivni intermitentni trening + vitamin E (IIT + VE, $n = 7$), 3) Vitamin E (VE, $n = 6$), 4) kontrolna (CON, $n = 6$), 5) Sham (S, $n = 6$). Protokol treniranja ove studije proveden je 6 dana tjedno 6 tjedana, uz poštivanje principa preopterećenja na traci za trčanje. Biokemijska analiza P53 proteina provedena je pomoću ELISA. Za analizu podataka korištena je jednosmjerna analiza varijance. Rezultati su pokazali da postoji značajna razlika između P53 proteina u krvnom serumu štakora koji je podvrgnut samo intenzivnom intermitentnom treningu u usporedbi s krvnim serumom u kontrolnoj skupini ($P < 0,002$). Nasuprot tome, nije zabilježena značajna razlika u istraživačkim skupinama P53 proteina u adipoznom tkivu. Intenzivni intermitentni trening može povećati razinu proteina P53 u krvnom serumu, ali nema značajan utjecaj na visceralno masno tkivo. Također, uzimanje previše vitamina E u normalnoj prehrani nema značajnog utjecaja na promjene razine P53.

Ključne riječi: P53 protein, intenzivni intermitentni trening, vitamin E, krvni serum, visceralno masno tkivo

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