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Melatonin Supplementation Decreases Aerobic Exercise Training Induced-Lipid Peroxidation and Malondialdehyde in Sedentary Young Women

Fatemeh Ziaadini[1], Mohsen Aminae[2], Mahsa Rastegar M.M.[3], Sadegh Abbasian[4], Amir Hossein Memari[5]*

[1]Faculty of Physical Education and Sport Sciences, Shahid Bahonar University of Kerman, Kerman, Iran
[2]Faculty of Physical Education and Sport Sciences, Shahid Bahonar University of Kerman, Kerman, Iran
[3]Faculty of Physical Education and Sport Sciences, Hakim Sabzevari University, Sabzevar, Iran
[4]Faculty of Physical Education and Sport Sciences, University of Tehran, Tehran, Iran
[5]Social Neuroscience Group, Department of Sports Medicine, Tehran University of Medical Sciences, Iran

Key words: malondialdehyde, aerobic exercise, lipid peroxidation, melatonin

Five percent of consumed oxygen produces a number of reactive oxygen species (ROS) including free radicals and other chemical products such as malondialdehyde (MDA). MDA increases lipid peroxidation such as low density lipoproteins cholesterol (LDL-c). Melatonin can decrease MDA and lipid peroxidation, but there are limited data about melatonin supplementation on MDA and lipid peroxidation of women. So the aim of this study was to evaluate the effects of melatonin supplementation on exercise-induced MDA and lipid peroxidation of sedentary young women. Twenty sedentary young (20–25 years old) women were selected and randomly divided into two exercise training-supplement (n=10) and exercise training (n=10) groups. Pretest/posttest body mass, BMI, rest heart rate (RHR), body fat percent, menstrual cycle, blood sampling for MDA and lipid profile were collected. Aerobic exercise training was performed for 8 weeks, triple weekly. Melatonin supplementation was ingested at 3 mg/day for exercise training-supplement. Results showed that the long term exercise training increased MDA concentrations, and melatonin supplementation significantly suppressed MDA surge (-25.2±2.87; 95% CI=-30.91 to -19.49). Moreover, post-exercise training LDL-c levels significantly declined due to melatonin supplementation in sedentary young women (19.5±2.41; 95% CI=12.272 to 25.728). We concluded that 3 mg melatonin supplementation following aerobic exercise training would attenuate ROS and improve lipid profile of young sedentary women.

INTRODUCTION

Exercise training *via* an increase in both metabolism and oxygen consumption leads to advance in free radicals or reactive oxygen species (ROS) [Crespo *et al*., 1999; Powers & Jackson, 2008]. Low to moderate levels of free radicals have multidimensional regulatory roles in cells, in contrast high levels of free radicals induce cellular damage [Bailey et al., 2007]. While the free radicals concentrations rise more than the capability of body immune system, oxidative stress is produced [El-Sokkary *et al*., 2006; Finaud *et al*., 2006]. By increasing the oxidative stress, the levels of sensitivity and impairment of lipid membrane function rise up [Acuna-Castroviejo *et al*., 2007; Finaud *et al*., 2006]. Furthermore, LDL-c as one of the blood lipids, is not safe from the free radical species penetration into its bilayer membrane structure, which is called lipid peroxidation [Finaud *et al*., 2006]. Malondialdehyde (MDA) as lipid peroxidation most frequent marker, produces more active radicals, which rapidly interact with oxygen and drive peroxyl radicals up [Spirlandeli *et al*., 2014]. Peroxyl radicals, also stimulate lipid peroxidation and lipid peroxidation is high in phospholipid polyunsaturated fatty acids. While oxidation expands, polyunsaturated fatty acids, cholesterols, cholesteryl esters, and apolipoprotein B alter and encourage MDA and 4-hydroxynonenal [Bailey *et al*., 2007; Finaud *et al*., 2006].

Antioxidants such as melatonin are widely used to reverse the ROS-related harmful consequences [Maldonado *et al*., 2010; Viitala *et al*., 2004]. Melatonin with its chemical name as N-acetyl-5-methoxy tryptamine is a hormone that is secreted by the pineal gland and is a strong free radical scavenger [Maldonado *et al*., 2010]. It impacts toxic radicals and breaks them down, eliminates ROS-induced H_2O_2 (one of the most important ROS), acts on uncoupling proteins (UCPs), and decreases body heat production [Acuna-Castroviejo *et al*., 2007; Nishida 2005]. Moreover, melatonin leads to mitochondria function development and ATP production, and increases electron transportation as well [Acuna-Castroviejo *et al*., 2001; Brown 1992].

Regular exercise training promote an elevation in the free radicals. There are different studies, which examined the effects of exercise training on the MDA and lipid profiles. Early studies showed that exercise training produced a 2-fold increase in muscle active oxygen species and increased expiration-pentane, lipid peroxidation, and MDA levels [Dillard

* Corresponding Author: Tel: +989120620717; Fax: +985138830826; E-mail: rastegar.moghadam.mansouri@gmail.com

et al., 1978; Hara *et al*., 1997]. Additionally, 30-min pre-exercise melatonin administration (6 mg) resulted in a significant decrease in blood lipids triglyceride and MDA concentration of male football players [Maldonado *et al*., 2012]. Excess exercise could lead to oxidative challenge and ROS generation and melatonin can scavenge free radicals and ROS generation [Tutkun 2013]. Also, it has been shown that only 10-day melatonin supplementation followed by strenuous exercise [Borges Lda *et al*., 2015] and 4-week melatonin administration (3 mg/kg/day) followed by a single swimming exercise (lasting for 30 min) could decrease the oxidative stress and MDA levels [Tutkun, 2013]. Moreover, melatonin administration (3 mg) attenuated triglyceride, ameliorated HDL-c, and improved lipid profile of rats [Agil *et al*., 2011]. In contrast, the administration of melatonin reported no significant effects on exercise-induced adaptations [Atkinson *et al*., 2005a,b]. Generally, the effects of melatonin supplementation on exercise-related free radicals of women are not yet fully explored. Documented literatures about the beneficial outcomes of the antioxidant on exercise training-induced lipid peroxidation and MDA are even more controversial and because of the lack of data on women, we aimed to examine the long-term exercise training and melatonin supplementation effects on lipid peroxidation and malondialdehyde (MDA) of sedentary young women.

MATERIALS AND METHODS

Ethical approval

The study protocol was performed according to existing guidelines of the Helsinki Declaration and it was done according to the guidance and approval of the Ethics Committee of the Zarand School of Nursing and Midwifery of the Kerman Medical Science University before the study begin.

Participants

After overall calling, 200 sedentary young women (age 24.2±1.03 years, BMI 24.48±1.52 kg/m^2) were participated for study inclusion criteria, that consisted of medical history (PAR-Q, menstrual cycle questionnaire, special disability such as cardiovascular disease, musculoskeletal injury) [De Sanctis *et al*., 2014; Shephard, 2014], drug usage and food supplementation. The following 30 subjects were chosen. In the next step, the Rockport test (VO_2max evaluating test), rest heart rate (RHR), and body fat percentage (evaluated with 3-point caliper) were calculated and 10 individuals out of 30 were removed from the study. Twenty subjects were split into the exercise training-supplement (n=10) and exercise training (n=10) groups, randomly. Prior to the interventions, the specific purposes of the study were detailed and informed consent was gotten from all subjects. The primary data (mean±standard deviation) of both exercise training-supplement and exercise training groups were collected as; age 24.2±1.03 and 23.4±1.83 years, body mass 67.7±6.05 and 65.9±5.36 kg, BMI 24.48±1.52 and 23.45±3.07 kg/m^2, rest heart rate 71.9±1.52 and 70.7±3.02 HR/min, body fat percent 25.64±1.2 and 23.7±2.4%, VO_2max 33.6±2.52 and 34.54±3.65 mL/kg/min, and menstrual cycle 28.5±1.2 and 29.5±1.3 days, respectively. There were no meaningful differences between groups in primary data and data distributions were normal.

Study protocol

Study design illustrated in Figure 1 consisted of individualized running exercise training, performed thrice weekly for 8 weeks [Bas *et al*., 2011]. Exercise training group was designated as a control to explain the effects of melatonin supplementation during exercise training. The Karvonen for-

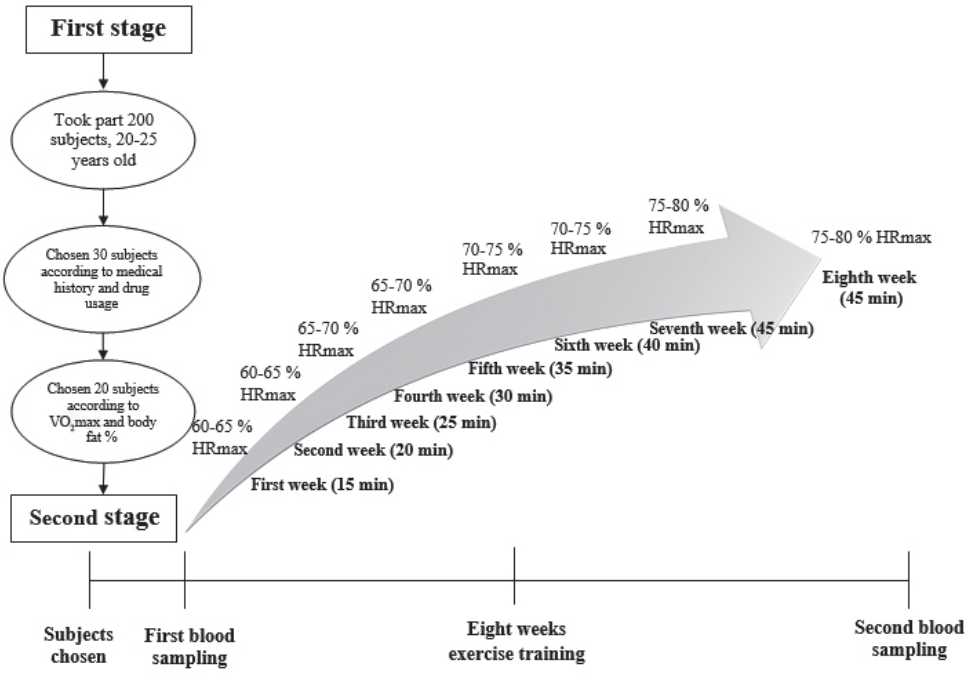

FIGURE 1. Study design divided into first and second stage. First stage consisted of subjects selection according to the given criteria. Second stage consisted of study protocol, which included 8-week, 3 days/week exercise training with volume and intensity that rose regularly from 15 to 45 min and 60 to 80 HRmax percent.

mula was used to calculate the exercise heart rate for counting the training intensity. It is written as; 1) Hrmax = 208–0.7 × age (year), 2) Reserve heart rate (RHR) = [(HRmax – HRrest) × 60 to 80 percent] + HRrest [Camarda *et al*., 2008]. The experimental protocol started after warm-up with 30–40 percent of maximum heart rate. To prevent high stress and apply progressive overload, exercise training volume was progressively increased from 15 to 45 min and exercise training intensity was gradually increased from 60 to 80 HRmax percent. To control the effects of training protocol, all subjects did not take part in any other exercise and their food habits were controlled once a day. Exercise training-supplement group consumed a commercial melatonin supplement (Vitane Pharmaceuticals Inc. USA) at a dose of 3 mg/day [Maldonado *et al*., 2012]. The study protocol was completed in spring season (Figure 1).

Biochemical analysis

Blood samples were collected after a 12-hour overnight fasting and at 7 – 8 a.m., in pre- and post- interventions. In each phase, 10 mL (5 mL for MDA assessment and 5 mL for lipid profile assessment) of blood samples were collected from antecubital vein. Samples were centrifuged for 10 min at 3600 rpm (centrifuge sys, Behdad Co, Ir), then isolated serums were frozen at -70°C (Scien.Temp., Compact ultra-low freezer, USA).

Measurement of malondialdehyde and blood lipids

MDA is the most frequent marker of lipid peroxidation [Spirlandeli *et al*., 2014]. Colorimetry and 2-thiobarbituric acid (TBA) were used to assess its level [Nielsen *et al*., 1997]. MDA standards were prepared using 1, 3, 3- tetramethoxypropane (2.5, 10, 20, 30, 40, and 50 mmol/L). Hundred μL of samples and stock standards with 300 μL Aqua-Pura and 100 μL sodium dodecyl sulfate (SDS) were poured in bonnet vials before being mixed with 750 μL of 20% acetic acid fluid and 750 μL of 86% TBA. The resulting MDA standard was heated for one hour (95°C) in the Bon Mari. After cooling, vials were centrifuged at 6700×g for 10 min and were analyzed spectrophotometrically at 532 nanometer wavelength (spectrophotometer, Pharmacia Biotech Model, UK).

Blood lipids were evaluated by Clinical Biochemistry Analyzer (Auto Analyzer Alpha Classic, Azma Co, Ir) using assay kits (Parsazmun Co, Ir). Triglyceride was evaluated by an enzymatic method (sensitivity assay of human kit was 5–700 mg/dL and intra- and inter-assay CVs were <5 and <10, respectively). Total serum cholesterol was evaluated by an enzymatic method (sensitivity assay of human kit was 5–500 mg/dL and intra- and inter-assay CVs were <5 and <14, respectively). HDL-c was evaluated by an enzymatic method (sensitivity assay of human kit was 1–300 mg/dL and intra- and inter-assay CVs were <5 and <10, respectively) and LDL-c was evaluated by an enzymatic method (sensitivity assay of human kit was 1–150 mg/dL and intra- and inter-assay CVs were <5 and <13, respectively).

Anthropometric measurement

Body mass (kg) and height (m) were measured three times using digital scale and meter, respectively (Sahand Co, Ir). Their average scores were recorded for each body mass and height, separately. Body mass index (BMI) was calculated according to its formulae as follows [Lunt *et al*., 2013]:

$$\text{BMI} = \frac{\text{Body mass (kg)}}{\text{Height (m)}^2} \quad (1)$$

Body fat percentage was calculated based on a 3-site caliper method according to the previous study, which could be read in details elsewhere [Selkow *et al*., 2011]. All skinfolds were calculated from the right part of the body (Yagami, Jap). Briefly, triceps, iliac, and thigh were three given sites. The middle distance between inferior-process of acromion in lateral end of right shoulder to olecranon process in the same hand was marked and vertically examined as the triceps skinfold point. In the iliac, skinfold was assessed from up of the iliac crest and caliper put 1 centimeter below the finger of examiner and estimated. In thigh, measurement was done vertically from the middle of thigh in standing position. All parts of estimation were repeated three times separately for each part and their averages were presented. Body fat percentage was recorded for each participant [Selkow *et al*., 2011]. This method was used to determine the women's body density (BD), body fat (BF percent), body fat mass (kg), and lean body mass (LBM kg), as follows:

$$\text{BD} = 1.099421 - 0.000929\ (\text{STP}) + 0.0000023\ (\text{STP})^2 - 0.001392\ (\text{Age}) \quad (2)$$

$$\text{BF percent} = \frac{457}{\text{BD} - 414.2} \quad (3)$$

$$\text{Fat Mass} = \text{Body Mass} \times \text{BF percent} \quad (4)$$

$$\text{LBM} = \text{Fat Mass} - \text{Body Mass} \quad (5)$$

STP: sum of three skin folds.

Rest heart rate and its assessment

Early morning wake up in lying position is defined situation for rest heart rate recording [Camarda *et al*., 2008]. Subjects' rest heart rate and exercise training-induced heart rate were calculated by Polar heart rate monitors (Polar heart rate monitor AXN 300, Fin).

Rockport walking test

The Rockport walking test was planned to appraise the maximum oxygen consumption (VO2max). The Rockport walking test was most appropriate to monitor the sedentary individuals' maximum oxygen consumption due to their walking instead of running [Lunt *et al*., 2013]. The test consisted of one-mile walking time (min), sex (men = 1, woman = 0), age (year), body mass (kg), and final (post-walking) heart rate (bpm). Consequently, the Rockport test is valid for submaximal VO_2max as subsequent formula [Lunt *et al*., 2013]:

6) 132.853 – (0.0769 × body mass) – (0.3877 × age) + (6.315 × sex) – (3.2649 × time) – (0.1565 × final heart rate).

Data analysis

Data normality was checked by the Shapiro-Francia test (W=0.966, Z=-0.206, p=0.582 for body mass) and equal-

TABLE 1. Comparison of the pre- and post-differences and time×group interaction of blood lipids and body mass of exercise training-supplement group (n=10) and exercise training group (n=10).

Variables	Groups	Pre-test (M±SD)	Post-test (M±SD)	95% CI for Difference *			Time × Group interactions **		
				Mean difference	Lower bound	Upper bound	Mean difference	Lower bound	Upper bound
Triglyceride (mg/dL)	Exerc-Supp	85.8±7.95	78.7±6.71	7.1±2.716	-0.484	14.684	6±2.716	-1.58	13.28
	Exerc	78.4±4.5	72.7±4.34	5.7± 2.716	-1.884	13.284			
HDL-c (mg/dL)	Exerc-Supp	49.4±4.42	51.3±4.08	-1.9±1.876	-7.138	3.338	1.4±1.876	-3.838	6.638
	Exerc	48.7±4.05	49.9±4.2	-1.2±1.876	-6.438	4.038			
Total cholesterol (mg/dL)	Exerc-Supp	170.5±7.85	152.28±5.3	18.26±2.717 †	10.673	25.847	-1.36±2.72	-8.947	6.227
	Exerc	169.32±5.26	153.64±5.5	15.68±2.717 †	8.093	23.267			
LDL-c to HDL-c ratio	Exerc-Supp	2.10±0.28	1.65±0.21	0.453±0.093 †	0.194	0.713	-0.139±0.03	-0.399	0.121
	Exerc	1.157±0.17	1.79±0.147	0.366±0.093 †	0.106	0.626			
Total cholesterol to HDL-c ratio	Exerc-Supp	3.47±0.311	2.98±0.23	0.49±104 †	0.199	0.781	0.49±0.104	-0.398	0.183
	Exerc	3.49±0.194	3.09±0.169	0.49±104 †	0.109	0.691			
Body mass (kg)	Exerc-Supp	67.7±6.05	64.7±6.15	3.0±2.56	-4.149	10.149	0.7±2.56	-6.449	7.849
	Exerc	65.9±5.36	64±52.27	1.9±2.56	-5.249	9.049			

Abbreviations: Exerc-Supp, exercise training-supplement; Exerc, exercise training; CI, confidence interval. † Denote significant different from pre-test, p<0.05, * 95% Confidence Interval (CI) for pre-test and post-test Differences (M±SEM), ** 95% Confidence Interval (CI) for Time × Group interactions (M±SEM).

ity of variances was assessed by Levene's test (F=0.072 and P=0.792 for pre-intervention body mass). Two-way ANOVA was used to compare the differences between two times (pre and post exercise training) in each group and also to determine the differences between two groups (2 times × 2 groups). All data were analyzed by Stata 12 (Stata Institute, USA) and the level $p < 0.05$ was considered significant, also Graph Pad prism-6 (GraphPad Software, Inc.USA) was utilized for graphs. Data were exemplified as mean±standard deviation (SD).

RESULTS

Lipid profile and body mass

Table 1 shows the lipid profile and body mass (mean±SD). Eight-week exercise training had no significant effects on serum triglyceride in exercise training-supplement group compared with exercise training group (6±2.7; 95% CI=-1.58 to 13.58). Also, within group changes showed similar, non-significant, decreases in triglyceride (p=0.078 *vs*. p=0.258 for exercise training-supplement and training groups, respectively). With respect to exact 1-β value of both groups, it seemed that exercise training led to greater reduction in triglycerides compared with combination of exercise training and supplementation (1-β=0.822 for training group *vs*. 1-β=0.579 for exercise training-supplement group) (Table 1).

There was a non-significant increase in HDL-c of exercise training-supplement group compared with exercise training group (1.4±1.87; 95% CI=-3.83 to 13.58). Moreover, within group statistical analysis represented no rise in the HDL-c levels in exercise training-supplement and exercise training groups after intervention ($p > 0.05$). From a statistical viewpoint, the exact value of 1-β for HDL-c was 0.9 (Table 1).

Current study data revealed a non-significant decline in the total cholesterol of exercise training-supplement compared with exercise training group (-1.36±2.17; 95% CI= -8.95 to 6.23). In contrast, a significant decrease was detected in the total cholesterol of both groups in two times (within group comparison) (p=0.0001 for each group). The exact value of 1-β for the total cholesterol was 0.9 (Table 1).

Body mass illustrated negligible change after exercise training (p>0.05). Also, intragroup comparison showed no significant decline in the body mass of women from both groups (p>0.05).

Lipid profile ratios

Time × group LDL-c/HDL-c ratio revealed no statistical differences in exercise training-supplement and exercise training groups (-0.139±0.03; 95% CI=-0.399 to 0.121). Although, LDL-c/HDL-c ratio significantly decreased in both groups compared with their pre-exercise training (p=0.0001 *vs*. p=0.002 for exercise training-supplement and exercise training groups, respectively). The exact value of 1-β for LDL-c/HDL-c ratio was 0.9 (Table 1).

Time × group evaluation of total cholesterol/HDL-c ratio showed a non-significant decrease in both groups (0.49±0.104; 95% CI=-0.398 to 0.183). While, intergroup comparison of total cholesterol/HDL-c ratio illustrated a significant decrease in both groups (p=0.0001 *vs*. p=0.005 for exercise training-supplement and exercise training groups, respectively). The exact value of 1-β for total cholesterol/HDL-c ratio was 0.9 (Table 1).

Lipid peroxidation

Following exercise training LDL-c concentration showed a non-significant change between groups (-4.8±2.41; 95% CI=-11.53 to 1.93). The exact value of 1-β for LDL-c between groups was 0.617. In contrast, intragroup LDL-c comparison presented a significant decline in both groups (19.5±2.41; 95% CI=12.272 to 25.728 *vs*. 15.6±2.41; 95% CI=8.872 to 22.328 for exercise training-supplement and exercise training groups, respectively) (Figure 2).

According to time×group data, melatonin supplementation significantly attenuated MDA elevation in exercise training-supplement group compared with exercise training group (-25.2±2.87045; 95% CI=-30.91 to -19.49). Besides, MDA concentration significantly increased in both groups compared with their pre-intervention (-21.5±2.045; 95% CI=-27.21 to -15.79 and -47.35±1.87; 95% CI=-53.06 to -41.64 in exercise training-supplement and exercise training groups, respectively; Figure 3).

Also, there was a significant correlation between decreased MDA and LDL-c levels, particularly following exercise training-supplement (r=0.499, p=0.025).

DISCUSSION

Based on the current study results, long-term exercise training-supplement and exercise training had the same beneficial effects on lipid profile of sedentary young women. Moreover, melatonin supplementation can decrease exercise-induced MDA elevation. Totally, current study represented the beneficial effects of aerobic exercise training with melatonin ingestion on the improvement of the body antioxidant-defense system and decline of lipid peroxidation in sedentary young women. Aerobic exercise leads to lipid peroxidation *via* free radicals production [Moflehi *et al*., 2012]. Maldonado and colleague studied the effects of melatonin supplementation following intense exercise on immunological defense and lipid metabolism of football players [Maldonado *et al*., 2012]. The participants were divided into two experimental (exercise and melatonin) and control (exercise) groups, the former group ingested 6 mg of melatonin 30 min before the high-intensity continuous exercise. Lipid peroxidation and MDA levels of both groups increased due to exercise but MDA elevation was decreased and lipid profile was improved in the experimental group. Particularly, melatonin ingestion increased antioxidant capacity of male football players [Maldonado *et al*., 2012]. Also, different aerobic intensities revealed different effects on MDA concentration. Moflehi and colleague evaluated the effects of different aerobic intensities on lipid peroxidation (MDA) and muscle damage (creatine kinase; CK) markers of sedentary young (21.76±1.89 year-old) men [Moflehi *et al*., 2012]. The effects of acute aerobic exercise in three intensities (low, moderate, and high) on MDA and CK levels were assessed. According to their results, by increasing the aerobic exercise intensity, the amount of MDA and CK was also elevated. The low intensity aerobic training was more beneficial for better adaptation and prevention of lipid peroxidation in sedentary individuals [Moflehi *et al*., 2012]. Agil and colleague examined the effects of melatonin and training on obesity and lipid profile of lean and obese rat [Agil *et al*., 2011]. Melatonin supplementation (10 mg/kg/day) for 6 weeks led to improved dyslipidemia, decreased triglyceride and LDL levels of obese rat and increased HDL of both obese and lean rats. Melatonin could probably amend lipid metabolism in obese humans and prevent the cardiovascular disease [Agil *et al*., 2011]. In contrast, other antioxidants did not attenuate the MDA levels. Viitala and colleague studied the effect of antioxidant supplementation (vitamin E, 885 mg/d) on lipid peroxidation of young trained and untrained participants following resistance exercise [Viitala *et al*., 2004]. Before the study, all participants had normal range of vitamin E, and were divided into four groups as; untrained placebo, untrained supplement, trained placebo, and trained supplement. Soy bean oil capsule was used as placebo. Resistance exercise training led to a significant elevation of MDA in all groups and vitamin E as antioxidant did not have any effect on reducing the oxidative harms and did not quench negative health consequences of oxidative stress, maybe due to its slow transference in muscle tissue to combat oxidative stress [Viitala *et al*., 2004]. It is noteworthy, that in spite of all mentioned differences such as study protocol and participants, MDA responses to melatonin supplementation in the current study were similar to previous studies [Agil *et al*., 2011; Maldonado *et al*., 2012].

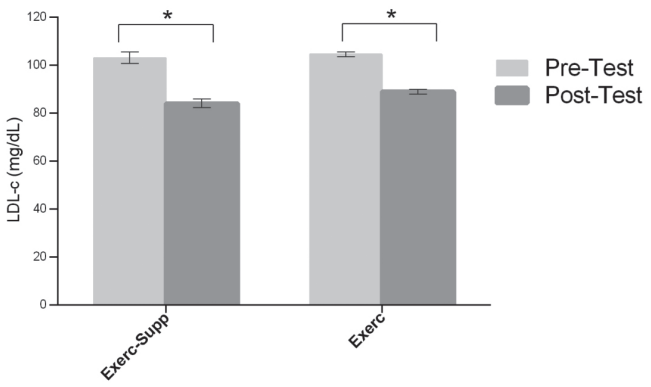

FIGURE 2. LDL-c changes before and after interventions in exercise training-supplement group (n=10) and exercise training group (n=10). * Significant different from pre-test results.

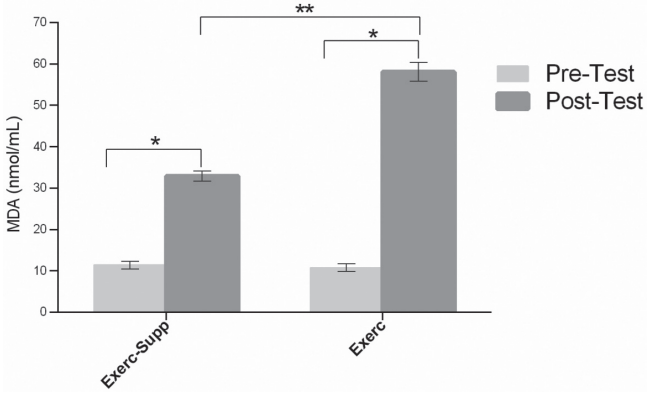

FIGURE 3. MDA changes before and after interventions in exercise training-supplement group (n=10) and exercise training group (n=10). * Significant different from pre-test results. ** Significant differences between post-test of two groups.

Exercise results in free radicals production, which could be defined by several mechanisms. Highly intense aerobic exercise training through high oxygen uptake and oxygen flux into active muscles lead to hyperoxic injury and increase lipid peroxidation by enhancing oxygen flux into mitochondria electron transport chain [Child *et al.*, 1998; Moflehi *et al*., 2012]. Moreover, exercise training causes the increase of energy expenditure and melatonin as a non-toxic molecule [Maldonado *et al*., 2012] supplies the energy demands which are required during the exercise [Kozirog *et al*., 2011]. Melatonin by both central way and direct way can affect brown adipose tissue and increase energy expenditure in exercise. In the central way, melatonin is under the nervous system run, meanwhile exercise training as a direct way, stimulates the sympathetic system and noradrenaline secretion [Coskun *et al*., 2006]. Noradrenaline turnover has a key role in increasing tryptophan-secretion and L-tryptophan is a melatonin precursor [Zouhal *et al*., 2008]. Also, in a direct way, protein kinase C (PKC) pathway in brown adipose tissue results in an increase of growth factor and mitochondria biogenesis [Agil *et al*., 2011].

Lipid peroxidation increases membrane permeability, which results in cytosolic enzymes and proteins loss and also destruction of sarcoplasmic reticulum structure. Such changes cause oxidative damage and MDA elevation [Bulbuller *et al*., 2005; Col *et al*., 2010]. Melatonin suppresses visceral fat and ameliorates insulin sensitivity, that leads to enhanced activity of lipoprotein lipase, reduces the lipolysis in visceral adipose tissue [Agil *et al*., 2011] and improves the lipid profile [Maldonado *et al*., 2012]. Also, HDL increases *via* melatonin supplementation by cholesterol esterification and activity of lecithin-cholesterol acyltransferase [Agil *et al*., 2011; Tamura *et al*., 2008]. Melatonin protects LDL from oxidation by inhibiting the activity of LDL receptor, prevents the cholesterol synthesis, and increases the cholesterol metabolism to bile acids [Agil *et al*., 2011; Chan&Tang 1995].

Conferring to several studies that examined aerobic training [Agil *et al*., 2011; Casella-Filho *et al*., 2011; Greene *et al*., 2012] and melatonin supplementation [Bhattacharyya *et al*., 2006; Kozirog *et al*., 2011; Maldonado *et al*., 2012; Tamura *et al*., 2008], all coordinately reported lipid profile improvement and lipid peroxidation, and oxidative stress decline [Borges Lda *et al*., 2015]. Almost all exercises result in MDA elevation and melatonin as a non-toxic antioxidant [Maldonado *et al*., 2012] can attenuate MDA elevation irrespective of the physical activity level of participants and their gender.

It was assumed that 8 weeks of exercise training were not adequate time to generate satisfactory adaptations during exercise, and that exercise training led to increased mitochondrial activity and consequently elevated MDA [Alessio *et al*., 1988; Finaud *et al*., 2006]. Nevertheless, MDA in the current study was more decreased in exercise training-supplement group. Also, there are various important parameters, which induce oxidative stress and were not assessed in the present study [Finaud *et al*., 2006; Gul *et al*., 2006].

CONCLUSION

In conclusion, 8-week exercise training with and without melatonin supplementation had significant effects on MDA and blood lipids profile of sedentary young women, as melatonin attenuated ROS and improved their lipid profile. In addition, 3 mg dosages of melatonin in sedentary young women were suitable to prevent exercise induced--MDA elevation.

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CONFLICT OF INTEREST

The authors affirm no conflict of interest.

AUTHOR INFO

Fatemeh Ziadin is MSc of Exercise Physiology at the Faculty of Physical Education and Sport Sciences, Shahid Bahonar University of Kerman, Kerman, Iran. **Mohsen Aminae** is Assistant Professor of Exercise Physiology at the Faculty of Physical Education and Sport Sciences, Shahid Bahonar University of Kerman, Kerman, Iran. **Mahsa Rastegar M.M.** is PhD of Exercise Physiology at the Faculty of Physical Education and Sport Sciences, Hakim Sabzevari University, Sabzevar, Iran. **Sadegh Abbasian** is PhD of Exercise Physiology at the Faculty of Physical Education and Sport Sciences, University of Tehran, Tehran, Iran. **Amir Hossein Memari** is MD, Director of Social Neuroscience Group, Department of Sports Medicine, Tehran University of Medical Sciences.

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