

The Effects of Eight Weeks Aerobic Interval Exercise with Variable Volume on the Cardiovascular Risk Factors and Liver Enzymes of Women with Dyslipidemia

Reyhaneh Zolfaghari¹, Amirhossein Haghighi^{*2}, Roya Askari³, Keyvan Hejazi³

MSc Student of Exercise Physiology, Faculty of Sport Sciences, Hakim Sabzevari University, Sabzevar, Iran.
 Associate Professor of Exercise Physiology, Faculty of Sport Sciences, Hakim Sabzevari University, Sabzevar, Iran.

3. Assistant Professor of Exercise Physiology, Faculty of Sport Sciences, Hakim Sabzevari University, Sabzevar, Iran.

ARTICLEINFO	ABSTRACT			
<i>Article type:</i> Research Paper	Introduction: Metabolic dyslipidemia could lead to non-alcoholic fatty liver disease, and its secondary consequence is the development of metabolic syndrome and diabetes. The present study aimed to investigate the effects of eight weeks of aerobic interval exercise with variable volumes on			
<i>Article History:</i> Received: 08 Feb 2020 Accepted: 13 Apr 2020 Published: 07 Jun 2020	 the cardiovascular risk factors and liver enzymes of the women with dyslipidemia. Methods: This quasi-experimental study was conducted on 30 middle-aged women with high blood lipids. The patients were selected and divided into three groups of low-volume training (three sessions per week; n=10; LVT), high-volume training (four sessions per week; n=10; HVT), and control (n=10; C). The exercise program was implemented in eight weeks 3-4 sessions per week for 			
<i>Keywords:</i> Dyslipidemia Liver Enzymes	45-60 minutes with the intensity of 65-75% of the maximal heart rate. The inter-group and intra- group comparison were performed using student's t-test, and one-way analysis of variance (ANOVA) was used to assess the differences between the groups.			
Lipid Profile	Results: In the training groups, a significant reduction was observed in weight (LVT: 72.01 vs. 67.26, HVT: 72.80 vs. 68.06), body mass index (LVT: 28.19 vs. 26.31, HVT:27.85 vs. 26.04), body fat (LVT: 26.86 vs. 25.69, HVT:27.21 vs. 25.91), waist-to-hip ratio (LVT: 1.05 vs. 1.03, HVT:1.07 vs. 1.05), alanine transaminase(LVT: 46.60 vs. 39.60, HVT: 43.80 vs. 38.50), aspartate transaminase(LVT: 36.50 vs. 31.00, HVT: 33.50 vs. 29.40), and triglyceride (LVT: 171.80 vs. 163.60, HVT:176.90 vs. 161.40). However, the maximum oxygen uptake increased significantly after the intervention in both the training groups (LVT: 32.17 vs. 35.93, HVT:30.93 vs. 35.98). The levels of total cholesterol (211.20 vs. 204.90) and low-density lipoprotein cholesterol (134.13 vs. 126.68) significantly decreased only in the LVT group, while no such changes were observed in the HVT group. In addition, the systolic blood pressure (LVT: 135.40 vs. 128.60, HVT: 137.00 vs. 129.60) decreased significantly in both groups, while no significant change was observed in the diastolic blood pressure.			
	Conclusion: According to the results, eight weeks of aerobic interval exercise could improve the cardiovascular risk factors, liver enzymes, and body composition of the women with dyslipidemia. Therefore, it is recommended that some cardiovascular risk factors and liver enzymes of women with dyslipidemia be used for the improvement of these patients.			

Please cite this paper as:

Zolfaghari R, Haghighi A, Askari R, Hejazi K. The Effects of Eight Weeks of Aerobic Interval Exercise with Variable Volume on the Cardiovascular Risk Factors and Liver Enzymes of Women with Dyslipidemia. J Nutrition Fasting Health. 2020; 8(2): 105-113. DOI: 10.22038/jnfh.2020.46400.1249.

Introduction

Dyslipidemia is a disorder associated with metabolic syndrome, which is characterized by disorders at the levels of three lipids, including the increased levels of very-low-density lipoprotein (VLDL) and low-density lipoprotein and decreased high-density lipoprotein [1]. High blood lipids are considered to be the atherogenic factors associated with metabolic syndrome, such as obesity, diabetes [2], and cardiovascular diseases[3]. Most of the studies in this regard have indicated that dyslipidemia is more prevalent in women compared to men[4, 5]. Blood lipids are an important risk factor associated with significant complications, such as coronary artery disease and fatty liver disease [6]. Cardiovascular diseases are known as the leading cause of mortality in most countries (especially the United States), and more than half of the patients with cardiovascular diseases are diagnosed with blood lipid disorders [2, 7],

* Corresponding author: Amirhossein Haghighi, Associate Professor of Exercise Physiology, Faculty of Sport Sciences, Hakim Sabzevari University, Sabzevar, Iran. Email: ah.haghighi@hsu.ac.ir. Tel: +985144012765 © 2020 mums.ac.ir All rights reserved.

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

obesity [8], and dyslipidemia [9], which in turn contribute to fatty liver diseases.

According to the literature, 35% of the individuals with morbid obesity progress toward non-alcoholic fatty liver disease (NAFLD) [10]. NAFLD is the liver inflammation caused by the excessive accumulation of fats in the liver tissues, which occasionally interferes with the normal function of the liver tissues and maybe progressive, causing liver damage or cirrhosis [11]. Furthermore, the disease has been reported to alter liver enzyme levels, such as aminotransferases, which are among the most sensitive diagnostic liver enzymes (e.g., aspartate aminotransferase and alanine transferase)[12].

On the other hand, previous findings have indicated that hyperlipidemia control is essential to the prevention of cardiovascular events [7], which highlights the utmost importance of preventive and therapeutic strategies for this disease, especially in the female population. Energy consumption, exercise, and physical activity have been reported to be correlated. Exercise is a beneficial and cost-efficient approach to the prevention and treatment of hepatic issues. Moreover, previous studies have indicated that lifestyle changes, physical activity, and exercise could reduce weight and improve liver enzymes [13, 14].

According to the literature, acute exercise elevated leads activity to alanine aminotransferase (ALT) and aspartate aminotransferase (AST) [15]. On the other hand, participation in long-term aerobic activities decreases these enzymes [16-18]. In a study in this regard, Zeinvand et al. (2018) investigated the impact of eight weeks of aerobic and resistance training on the blood lipid profile of elderly women, claiming that aerobic training was more effective than resistance training in improving the blood lipid profile in the elderly with NAFLD [19]. In contrast, Park et al. (2019) reported that eight weeks of training (five times per week) with 50-60% of the heart rate reserved for 1-2 weeks and at 60-80% of the heart rate reserved for 3-8 weeks in eight women caused no significant changes, and only alkaline phosphatase increased significantly [20].

The number of the training sessions per week is an exercise variable that could affect metabolic changes[21, 22]. However, limited and contradictory findings have been reported regarding the effects of exercise training on cardiovascular risk factors and liver enzymes [22, 23]. The sample populations of these studies have mainly been diabetic patients and children with intellectual disability, and the exercise protocols have encompassed aerobic exercises. Nevertheless, evidence is scarce regarding the effects of the weekly volume of aerobic interval exercises on cardiovascular risk factors and liver enzymes in women with hyperlipidemia.

The present study aimed to investigate the effects of eight weeks of aerobic interval exercise with variable volumes on the cardiovascular risk factors and liver enzymes of women with dyslipidemia.

Materials and Methods

Subjects

This quasi-experimental study was conducted with three experimental groups, which were compared with each other. In total, 30 middleaged women with high blood lipids (age: 35-50 years) were enrolled in the study. The patients had the total cholesterol level of 200-240 mg/dl and body mass index (BMI) of 25-30 kg/m²and were selected voluntarily and objectively. At the first stage of the research, the patients were introduced to the concept and approaches of cooperation. Important notes were also provided to the subjects regarding nutrition, diseases, drug consumption, supplements, drug abuse, no smoking habits, and lack of participation in other studies for a minimum of six months prior to the research schedule.

Participation in the study was voluntary, and written informed consent was obtained from the patients. Afterwards, the subjects were randomly divided into three groups of low-volume training (LVT; three sessions per week; n=10), high-volume training (HVT; four sessions per week; n=10), and control (n=10).

The study protocol was approved by the Ethics Committee of Hakim Sabzevari University, Iran (code: IR-MEDSAB.REC.1396.941). The following equation was used to determine the sample size:

$$n = \frac{2\sigma^2 (Z_{1-\alpha/2} + Z_{1-\beta})^2}{d^2} = \frac{2(2.5)^2 (2+1.28)^2}{3.5^2} = 10.97 \cong 11$$

Body Composition

At the second stage of the research, the height of the patients was measured in centimeters using stadiometer (Seca, Germany) with the sensitivity of five millimeters, and their weight was calculated using a digital scale (model: PS07-PS06, Beurer, Germany). Following that, the waist-to-hip ratio (WHR) was determined, and BMI was calculated by dividing the body weight by the squared height in meters (kg/m²). In order to measure the waist of the participants, a tape measure was used (MABIS, Japan) at the midway between the lowest rib and top of hips (above the navel). The hips were measured at the widest point around the buttocks; to do so, the tape was held snugly without pulling and leveled around, and the value was divided for the WHR.

At the next step, the body fat percentage was measured based on two points (triceps, superficial and abdominal sections) using Lafite type calipers. These measurements were obtained from the right side of the patients, so that the thickness of the subcutaneous fat would be recorded in the three arms of the middle skinfold in the posterior section in the thigh, as well as the thickest section of the tibia. The measured values were applied to the Luhmann-Slater formula, and the body fat percentage was obtained using Equation 1. In this equation, the body density of the subjects is represented by *BdF*, the sum of the three points of the skinfold is shown by Σ 3, and the age of the patients is indicated by age. Based on the satiation equation, the body fat percentage was obtained (Equation 2).

Equation 1: BdF = 1.099421- (0.0009929 × Σ3sits) + (0.0000023 × (Σ3sits)2) - (0.0001392× age)

Equation 2: % F (siri, 1956) = [(4.95÷ BdF) – 4.5] × 100

The blood pressure of each patient was measured before physical activity using the Maximed Exipres TD-3018 machine and converted into the mean blood pressure using the formula of the mean arterial blood pressure, as shown in Equation 3.

Equation3: Mean Blood Pressure – (2× Diastolic Blood Pressure + Systolic Blood Pressure) /3

In order to homogenize the nutritional status of the patients due to its impact on the study parameters, the patients were required to write down a checklist report on a three-day diet. After collecting all the nutrition information, the amount of the received calorie and supplements was determined. In addition, the patients were asked to consume common foods with the same calorie content prior to the two periods of blood testing.

Blood Sample Collection

Blood samples were collected 48 hours before the training and 48 hours after the training sessions. Sampling was performed between 8:0-10:00 AM. After 10-12 hours of fasting in the laboratory, blood sample were collected from the left vein of each subject in the sitting position and at rest. Serum liver enzymes were also determined using the photometric method and a bionic acid kit with the sensitivity of 1 unit per liter. Serum biochemical concentrations were determined using an autoanalyzer spectrophotometer and various kits at different wavelengths. Moreover, serum triglyceride concentration was determined (mg/dl) using the Man kits and GPO-PAP enzymatic method at the wavelength of 546 nanometers. High-density lipoprotein (HDL) and low-density lipoprotein (LDL) were also measured using the enzymatic method (PishtazTeb kit, Tehran, Iran).

Maximum Oxygen Uptake (VO_{2мах})

The maximum oxygen uptake was estimated based on the Rockport walking protocol, which functions when walking at the maximum speed of one mile (1609 m), recording the heart rate immediately for 15 seconds, and multiplying the obtained number by four. The maximum oxygen consumption in the Rockport walking protocol was calculated using Equation 4, in which the body weight is measured in pounds, and age is determined in years, and other parameters such as the sex factor (men=1, women=0), time to complete one mile per minute, and heart rate after performing the test (beats per minute) are estimated [24].

Equation 4: VO2_{MAX}=132.853-(Weight×0.0769)-(0.3877×age)+(6.315×sex)-(3.2649×time)-0.1565×Heart Rate

Exercise Protocol

The training protocol consisted of eight weeks of aerobic interval training with two different volumes (3 and 4 days per week), and the duration of each session was 45-60 minutes with the intensity of 65-75% of the maximal heart rate reserve. The training duration progressively increased from 40 minutes at baseline to 45 minutes at the end of the study period. The training protocol encompassed a general warmup for 10 minutes (walking, moderate running, stretching, and mobility), and the training intensity was controlled using a pulse meter (model: POLAR, Finland).At the end of each

Zolfaghari R et al

session, the exercise protocol was performed for 10 minutes to return to baseline and cool down (slow running, walking, and stretching). After the implementation of the protocol (6 weeks), all the measurements were repeated in order to obtain the posttest data. In addition, the control group had no activity during the study period, and the patients inactive similar to their lifestyle before the study.

Statistical Analysis

Data analysis was performed in SPSS version 16. The normality of the theoretical distribution of the data was confirmed using the Shapiro-Wilk test, and variance homogeneity was confirmed using the Levene's test. Moreover, paired sample t-test and repeated measures ANOVA were applied for the inter-group and intra-group comparison of the variance changes. In all the statistical analyses, the significance level was considered to be less than 0.05.

Table 1.	Characteristic of Patients
----------	----------------------------

Results

Table 1 shows the characteristics of the patients. According to the information in Table 2, a significant reduction was observed in the body weight (LVT: 72.01 vs. 67.26; HVT: 72.80 vs. 68.06; P=0.001), BMI (LVT: 28.19 vs. 26.31; HVT:27.85 vs. 26.04; P=0.001), body fat(LVT: 26.86 vs. 25.69; HVT:27.21 vs. 25.91; P=0.001), and WHR (LVT: 1.05 vs. 1.03; HVT:1.07 vs. 1.05; P=0.001), in the training groups. On the other hand, a significant increase was denoted in the oxygen consumption maximal after the intervention in both the training groups(LVT: 32.17 vs. 35.93; HVT:30.93 vs. 35.98; P=0.001).In addition, significant differences were observed in intergroup mean changes between the training groups in terms of the body weight, BMI, and maximal oxygen consumption (P<0.05).

Groups	Variations (Mean±SD)			
	Age	Height	Weight	BMI (kg/m²)
	(year)	(cm)	(kg)	
Low-volume Training (LVT)	40.30±3.74	159.85±7.48	72.01±6.14	28.19±1.76
High-volume Training (HVT)	41.20±3.88	162.04±6.66	72.80±4.72	27.85±2.80
Control	42.90±4.20	160.70±6.11	73.28±5.13	28.36±0.93
	P=0.65	P=0.84	P=0.41	P=0.25

Table 2. Changes in Bo	dy Composition i	n Study Groups
------------------------	------------------	----------------

		Stages	Variations			
Variables	Groups	Posttest Mean±SD*	Pretest Mean±SD*	P-value**	P-value***	
	-			P-value	F	P-value
Weight	LVT	72.01±6.14	67.26±5.72	0.001†	5.50	0.001†
(kg)	HVT	72.80±4.72	68.06±4.29	0.001†		
	Control	73.28±5.13	74.30±5.46	0.206		
BMI	LVT	28.19±1.76	26.31±0.92	0.001†	6.56	0.005†
(kg/m²)	HVT	27.85±2.80	26.04±2.72	0.001†		
	Control	28.36±0.93	28.77±1.46	0.231		
Body Fat	LVT	26.86±1.28	25.69±1.40	0.002†	1.46	0.25
Percentage	HVT	27.21±1.26	25.91±1.06	0.001†		
(%)	Control	26.45±1.17	26.57±1.11	0.633		
WHR	LVT	1.05 ± 0.07	1.03±0.07	0.009†	1.58	0.224
(cm)	HVT	1.07 ± 0.06	1.05 ± 0.05	0.003†		
	Control	1.08 ± 0.07	1.08±0.07	0.558		
VO2 _{max}	LVT	32.17±5.48	35.93±5.13	0.001†	9.35	0.001†
(ml/kg/min)	HVT	30.93±3.30	35.98±2.57	0.001†		
	Control	29.76±2.92	29.75±2.87	0.633		

+Significance level P<0.05 **P-value within groups

*Data presented as mean±standard deviation ***P-value between groups

According to the information in Table 3, the levels of ALT (LVT: 46.60 vs. 39.60; HVT: 43.80 vs. 38.50; P=0.001) and serum AST decreased in

both the training groups (LVT: 36.50 vs. 31.00; HVT: 33.50 vs. 29.40; P=0.001).Moreover, serum triglyceride levels decreased significantly in both

groups (LVT: 171.80 vs. 163.60; HVT:176.90 vs. 161.40; P=0.001), while the total cholesterol levels decreased only in the LVT group(211.20 vs. 204.90), and no significant change was observed in the HVT group in this regard. On the other hand, HDL significantly increased in both the training groups (LVT: 42.70 vs. 45.50; HVT: 42.80 vs. 46.10; P=0.001), while a significant reduction was observed in the control group. According to the findings, LDL significantly reduced only in the LVT group (134.13 vs.

126.68; P=0.002), while no significant changes were observed in the HVT and control groups in this regard. Moreover, the systolic blood pressure decreased significantly in both groups after the intervention (LVT: 135.40 vs. 128.60; HVT: 137.00 vs. 129.60; P=0.001), while no significant changes were observed in the diastolic blood pressure. A significant difference was denoted in the intergroup mean changes in both the training groups in terms of LDL and the systolic blood pressure.

		Stages Variations					
Variables	Groups	Posttest	Pretest	P-value**	P-value***		
		Mean±SD*	Mean±SD*	P-value	F	P-value	
ALT	LVT	46.60±4.95	39.60±5.13	0.001†	2.06	0.146	
(IU/I)	HVT	43.80±5.87	38.50±3.92	0.004†			
	Control	43.60±4.88	42.80±5.55	0.619			
AST	LVT	36.50±4.60	31.00±4.85	0.001†	0.67	0.520	
(IU/I)	HVT	33.50±6.65	29.40±9.06	0.037†			
	Control	32.80±4.66	33.00±6.33	0.897			
TG	LVT	171.80±12.81	163.60±8.78	0.029†	0.492	0.057	
(mg/dl)	HVT	176.90±9.72	161.40±9.88	0.001†			
	Control	168.80±7.45	170.80±7.24	0.427			
ТС	LVT	211.20±7.22	204.90±8.89	0.004†	0.483	0.622	
(mg/dl)	HVT	206.10±12.28	201.90±11.17	0.166			
	Control	204.50±11.02	200.90±8.08	0.229			
HDL-C	LVT	42.70±3.19	45.50±2.63	0.004†	17.79	0.001†	
(mg/dl)	HVT	42.80±3.39	46.10±2.84	0.006†			
	Control	42.40±3.40	39.60±2.59	0.016†			
LDL-C	LVT	134.14±8.36	126.68±9.11	0.002†	0.386	0.684	
(mg/dl)	HVT	127.92±13.24	123.52±12.60	0.116			
	Control	128.34±9.34	127.14±7.75	0.635			
Systolic Bloo	d LVT	135.40±5.25	128.60±3.95	0.004†	6.30	0.006†	
Pressure	HVT	137.00±2.75	129.60±2.72	0.001†			
(mm/mg)	Control	132.50±8.36	136.00±7.33	0.265			
Diastolic Bloo	d LVT	85.50±4.74	83.60±4.24	0.25	0.559	0.578	
Pressure	HVT	85.10±3.14	84.50±3.70	0.61			
(mm/mg)	Control	86.60±3.13	85.80±3.36	0.49			

*†Significance level P<0.05 **P-value within groups*

*Data presented as mean±standard deviation ***P-value between groups

Discussion

According to the results of the present study, body weight, BMI, body fat, and WHR significantly decreased in the LVT and HVT groups. Furthermore, the exercise protocol increased the energy consumption, and if energy intake was low, it could lead to weight loss in the exercise groups. Hormonal changes such as decreased insulin and increased catecholamines have been reported to stimulate beta-adrenergic receptors and activate hormone-sensitive lipase, which activate adipocyte lipolysis [25]. Through lipolysis, the fatty acids in the body (triglycerides) are broken down and released into the bloodstream as free fatty acids and a substrate for energy metabolism[26]. In the current research, the body composition of the patients improved, which was represented by the decreased weight, fat percentage, and WHR. On the other hand, decreased body fat percentage and increased lean body mass cause the basal metabolism to increase[27], which in turn could increase insulin sensitivity and reduce inflammation [28, 29]. This is considered to be a major clinical goal in the treatment of high blood lipids.

According to the results of the present study, the maximal oxygen consumption increased significantly in both the training groups. The increased $VO2_{max}$ compared to the pre-exercise

activity could be attributed to cardiovascular and metabolic adaptation to the exercises, such as the increased muscle oxidative capacity, total hemoglobin, and fat, decreased glycolysis, increased end-diastolic volume (cardiac load), decreased end-systolic volume, and increased volume impact, arterial-venous blood oxygen difference, activity of Krebs cycle enzymes, number and size of mitochondria, and muscle tissues and their efficiency [30].Therefore, great emphasis has been placed on physical activity and cardiorespiratory fitness in order to prevent cardiovascular diseases[30, 31].

According to the findings of the current research, the ALT and AST levels decreased in both the training groups, which might be due to the improved body composition or positive changes in the lipid profile following aerobic interval exercise. In the present study, a significant decrease was observed in anthropometric indices such as weight, body fat percentage, and lipid profile improvement after the exercise protocol with two volumes of three and four training sessions per week. Considering the associations between these variables (lipid profile and body composition) with the changes in the liver enzyme, better weight control is required, which could be attained through proper diets and exercise periods.

Insulin resistance is another cause of fatty liver [32, 33]. Exercise increases insulin sensitivity by increasing the glucose carriers in the muscle cell membrane and improving insulin messaging [32, 33]. A significant association has also been reported between insulin resistance and fatty liver [34]. Therefore, it could be stated that aerobic interval exercise could reduce insulin sensitivity and insulin resistance, which in turn reduced the transfer of free fatty acids to the liver and fat accumulation in the liver [35].It is also expected that the increased lipid metabolism in the cells involved in the activity (muscle cells) may contribute to the decreased liver fat content. Considering the association between obesity and inflammation [36, 37] and role of inflammation in the incidence of fatty liver[38], it is possible that aerobic interval exercise decrease the levels of inflammatory cytokines by improving the body composition and reducing plasma lipid oxidation, thereby decreasing ALT and AST in the exercise groups after aerobic training for three and four sessions per week. However, these changes made no significant difference between the training groups and the control group.

In the current research, triglyceride decreased significantly in both the training groups, while the total cholesterol levels decreased only in the LVT group, and no significant changes were observed in the HVT group in this regard. HDL significantly increased in both the training groups, while it significantly decreased in the control group. On the other hand, LDL significantly reduced only in the LVT group, while no significant changes were observed in this variable in the HVT and control groups.

One of the most effective adaptations following aerobic activities is the increased mitochondrial volume and the subsequent activity of lipolysis enzymes, which increase the catabolic ability of fats during exercise [39]. Exercise may also exert beneficial effects on plasma HDL levels through affecting the muscle and liver lipoprotein lipase (LPL) activity[40]. Changes in the LPL activity are associated with increased VLDL entry from the liver to the bloodstream and its clearance from the bloodstream [41].On the other hand, lecithin cholesterol acyltransferase (LCAT) converts cholesterol into HDL particles, as well as LDL. The increment of this enzyme may be responsible for the increased HDL induced by physical exercise. Furthermore, previous findings have demonstrated that LCAT increases significantly in some sports activities. In this context, the other mechanisms (e.g., decreased insulin sensitivity) that may alter the levels of lipids and blood lipoproteins may be effective as well [42]. Other possible mechanisms (e.g., increased cholesterol transmission) may also be important in this regard.

Some of the effects of exercise on fats may be indirect and associated with abdominal fat reduction. The movement of free fatty acids from the abdominal fat to the liver also decreases, and production decreases VLDL as well [43].Continuous and prolonged physical activity leads to a decrease in liver lipase[44].Overall, these changes could improve metabolic adaptation and lipid metabolism (e.g., lipid profile).In the present study, no significant difference was observed in this regard between the exercise groups with different volumes, which could be due to the lack of a significant difference between three and four sessions of calorie intake or other confounding factors, such as nutritional quality, excess calories, and level of daily activity. Increasing the number of the training sessions or length of the intervention period may cause a significant difference in this regard.

Conclusion

According to the results, exercise training in with the two volumes of three and four sessions per week could reduce cardiovascular risk factors, liver enzymes, and body composition, while increasing the maximal oxygen consumption. However, no significant difference was observed between these two weekly training volumes in these measured variables. There seems to be no significant difference between three and four training sessions regarding cardiovascular variables and the measured liver enzymes. For further changes, coaches are advised to modify other exercise variables, such as increasing the number of the training sessions per week, adjusting other variables (e.g., exercise intensity, training time per training session) or other interventions (e.g., calorie restriction). The main limitation of the present study was the lack of diet control in the women with dyslipidemia. It is hoped that the research scope and observations would be expanded in the future.

Conflicts of interest

None declared.

Acknowledgements

Hereby, we extend our gratitude to Hakim Sabzevari University for the financial support of this study. We would also like to thank the volunteers for assisting us in this research project.

References

1. Srikanth S, Deedwania P. Management of dyslipidemia in patients with hypertension, diabetes, and metabolic syndrome. Curr Hypertens Rep. 2016; 18(10): 76.

2. Georgiadis AN, Papavasiliou EC, Lourida ES, Alamanos Y, Kostara C, Tselepis AD, et al. Atherogenic lipid profile is a feature characteristic of patients with early rheumatoid arthritis: effect of early treatment–a prospective, controlled study. Arthritis Res Ther. 2006; 8(3): R82.

3. Kannel WB. Lipids, diabetes, and coronary heart disease: insights from the Framingham Study. Am Heart J. 1985; 110(5): 1100-7.

4. Legro RS, Kunselman AR, Dodson WC, Dunaif A. Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome: a prospective, controlled study in 254 affected women. J Clin Endocrinol Metab. 1999; 84(1): 165-9.

5. Onat A, Can G, Hergenç G, Yazici M, Karabulut A, Albayrak S. Serum apolipoprotein B predicts dyslipidemia, metabolic syndrome and, in women, hypertension and diabetes, independent of markers of central obesity and inflammation. Int J Obes. 2007; 31(7): 1119-25.

6. Gaggini M, Morelli M, Buzzigoli E, DeFronzo RA, Bugianesi E, Gastaldelli A. Non-alcoholic fatty liver disease (NAFLD) and its connection with insulin resistance, dyslipidemia, atherosclerosis and coronary heart disease. Nutrients. 2013; 5(5): 1544-60.

7. Wilmot KA, O'Flaherty M, Capewell S, Ford ES, Vaccarino V. Coronary heart disease mortality declines in the United States from 1979 through 2011: evidence for stagnation in young adults, especially women. Circulation. 2015; 132(11): 997-1002.

8. Ahmed A, Wong RJ, Harrison SA. Nonalcoholic fatty liver disease review: diagnosis, treatment, and outcomes. Clin Gastroenterol Hepatol. 2015; 13(12): 2062-70.

9. Manimmanakorn A, Hamlin MJ, Ross JJ, Taylor R, Manimmanakorn N. Effects of low-load resistance training combined with blood flow restriction or hypoxia on muscle function and performance in netball athletes. J Sci Med Sport. 2013; 16(4): 337-342. 10. Davoodi M, Moosavi H, Nikbakht M. The effect of eight weeks selected aerobic exercise on liver parenchyma and liver enzymes (AST, ALT) of fat liver patients. Journal of Shahrekord Uuniversity of Medical Sciences. 2012; 14(1): 84-90. (Persian)

11. Shamsoddini A, Sobhani V, Ghamar Chehreh MH, Alavian SM, Zaree A. Effect of aerobic and resistance exercise training on liver enzymes and hepatic fat in Iranian men with nonalcoholic fatty liver disease. Hepat mon. 2015; 15(10): e31434.

12. Fathei M, Khairabadi S, Ramezani F, Hejazi K. The effects of eight weeks aerobic training, green tea supplementation and compound of them on serum liver enzymes and apolipoproteins in inactive overweight women. Medical journal of mashhad university of medical sciences. 2016; 59(2): 114-23.

13. Omagari K, Kadokawa Y, Masuda J, Egawa I, Sawa T, Hazama H, et al. Fatty liver in non-alcoholic nonoverweight Japanese adults: incidence and clinical characteristics. J Gastroenterol Hepatol. 2002; 17(10): 1098-105.

14. De Piano A, do Prado WL, Caranti DA, Siqueira KO, Stella SG, Lofrano-Prado MC, et al. Metabolic and nutritional profile of obese adolescents with nonalcoholic fatty liver disease. J Pediatr Gastroenterol Nutr. 2007; 44(4): 446-52.

15. Valizadeh A, Yousefi Bilehsavar O, Abdi H, Jahanshahi Z, Madadi B. The Effect of acute exercise on changes Liver enzymes (ALT ‹ALP ·AST) in Non-athletes middle-aged women with hypertension. Int J Adv Biotechnol Res. 2016; 7(3): 365-71.

16. Khaoshbaten M, Gholami N, Sokhtehzari S, Monazami AH, Rostami Nejad M. The effect of an aerobic exercise on serum level of liver enzymes and

liver echogenicity in patients with non alcoholic fatty liver disease. Gastroenterol Hepatol Bed Bench. 2013; 6(Suppl 1): S112-6.

17. Valizadeh R. Nikbakht M, Davodi M, Khodadoost M. The effect of eight weeks elected aerobic exercise on the levels of (AST, ALT) enzymes of men patients with have fat liver. Procedia Soc Behav Sci. 2011; 15: 3362-5.

18. Yao J, Meng M, Yang S, Li F, Anderson RM, Liu C, et al. Effect of aerobic and resistance exercise on liver enzyme and blood lipids in Chinese patients with nonalcoholic fatty liver disease: a randomized controlled trial. Int J Clin Exp Med. 2018; 11(5): 4867-74.

19. Zeinvand lorestani A, Mirnasouri R, Rahmati M. The effect of eight weeks of aerobic training on the levels of enzymes associated with non-alcoholic fatty liver in obese children. Yafte. 2018; 20(2): 53-61. (Persian)

20. Park JH. Kim HJ, Han A, Kang DM, Park S. Effects of aerobic exercise training on the risk factors for liver diseases in elderly women with obesity and impaired fasting glucose: A pilot study. J Exerc Nutrition Biochem. 2019; 23(1): 21-7.

21. Braith RW, Graves JE, Pollock ML, Leggett SL, Carpenter DM, Colvin AB. Comparison of 2 vs 3 days/week of variable resistance training during 10and 18-week programs. Int J Sports Med. 1989; 10(6): 450-4.

22. Balducci S, Cardelli P, Pugliese L, D'Errico V, Haxhi J, Alessi E, et al. Volume-dependent effect of supervised exercise training on fatty liver and visceral adiposity index in subjects with type 2 diabetes The Italian Diabetes Exercise Study (IDES). Diabetes Res Clin Pract. 2015; 109(2): 355-63.

23. Haghighi AH, Gelardi N, Hamedinia MR. The Effect of an Aerobic Exercise Program with Two Different Volumes on Some Risk Factors of Cardiovascular Diseases in Mentally Retarded Girls. Journal of Sport Biosciences. 2015; 7(2): 241-59.

24. Swain DP, Abernathy KS, Smith CS, Lee SJ, Bunn SA. Target heart rates for the development of cardiorespiratory fitness. Med Sci Sports Exerc. 1994; 26(1): 112-6.

25. Mottillo EP, Shen XJ, Granneman JG. Role of hormone-sensitive lipase in β -adrenergic remodeling of white adipose tissue. Am J Physiol Endocrinol Metab. 2007; 293(5): E1188-97.

26. DeLany JP, Kelley DE, Hames KC, Jakicic JM, Goodpaster BH. Effect of physical activity on weight loss, energy expenditure, and energy intake during diet induced weight loss. Obesity (Silver Spring). 2014; 22(2): 363-70.

27. Blundell JE, Finlayson G, Gibbons C, Caudwell P, Hopkins M. The biology of appetite control: do resting metabolic rate and fat-free mass drive energy intake? Physiol Behav. 2015; 152(Pt B): 473-8.

28. McLaughlin T, Lamendola C, Coghlan N, Liu TC, Lerner K, Sherman A, et al. Subcutaneous adipose cell size and distribution: relationship to insulin resistance and body fat. Obesity (Silver Spring). 2014; 22(3): 673-80.

29. Hsieh CJ, Wang PW, Chen TY. The relationship between regional abdominal fat distribution and both insulin resistance and subclinical chronic inflammation in non-diabetic adults. Diabetol Metab Syndr. 2014; 6(1): 49.

30. Yari Z, Rahimlu M, Poustchi H, Ebrahimi Daryani N, Hekmatdoost A. Effect of Faxseed Supplementation on Liver enzymes, Hepatic Fibrosis and Steatosis in Nonalcoholic Fatty Liver Disease: A Randomizedcontrolled Clinical Trial. Iranian Journal of Nutrition Sciences & Food Technology. 2016. 10(4): 1-12.

31. Baldi JC, Wilson GA, Wilson LC, Wilkins GT, Lamberts RR. The type 2 diabetic heart: its role in exercise intolerance and the challenge to find effective exercise interventions. Sports Med. 2016; 46(11): 1605-17.

32. Henriksen EJ. Invited review: Effects of acute exercise and exercise training on insulin resistance. J Appl Physiol (1985). 2002; 93(2): 788-96.

33. Borghouts LB, Keizer HA. Exercise and insulin sensitivity: a review. Int J Sports Med. 2000; 21(1): 1-12.

34. Sullivan S, Kirk EP, Mittendorfer B, Patterson BW, Klein S. Randomized trial of exercise effect on intrahepatic triglyceride content and lipid kinetics in nonalcoholic fatty liver disease. Hepatology. 2012; 55(6): 1738-45.

35. Angulo P. Nonalcoholic fatty liver disease. N Engl J Med. 2002; 346(16): 1221-31.

36. Arkan MC, Hevener AL, Greten FR, Maeda S, Li ZW, Long JM, et al. IKK- β links inflammation to obesity-induced insulin resistance. Nat Med. 2005; 11(2): 191-8.

37. Recasens M, Ricart W, Fernández-Real JM. Obesity and inflammation. Rev Med Univ Navarra. 2004; 48(2): 49-54.

38. McManaman JL, Bales ES, Orlicky DJ, Jackman M, MacLean PS, Cain S, et al. Perilipin-2-null mice are protected against diet-induced obesity, adipose inflammation, and fatty liver disease. J Lipid Res. 2013; 54(5): 1346-59.

39. Plotnikoff RC, Courneya KS, Trinh L, Karunamuni N, Sigal RJ. Aerobic physical activity and resistance training: an application of the theory of planned behavior among adults with type 2 diabetes in a random, national sample of Canadians. Int J Behav Nutr Phys Act. 2008; 5(1): 61.

40. Durstine JL. Action plan for high cholesterol. Human Kinetics; 2006.

41. Takahashi S. Triglyceride rich lipoprotein-LPL-VLDL receptor and Lp (a)-VLDL receptor pathways for macrophage foam cell formation. J Atheroscler Thromb. 2017; 24(6): 552-9.

42. Ahn N, Kim K. High-density lipoprotein cholesterol (HDL-C) in cardiovascular disease: effect of exercise training. Integr Med Res. 2016; 5(3): 212-5.

43. Mittendorfer B, Yoshino M, Patterson BW, Klein S. VLDL triglyceride kinetics in lean, overweight, and

obese men and women. J Clin Endocrinol Metab. 2016; 101(11): 4151-60.

44. Despres JP, Moorjani S, Tremblay A, Poehlman ET, Lupien PJ, Nadeau A, et al. Heredity and changes in

plasma lipids and lipoproteins after short-term exercise training in men. Arteriosclerosis. 1988; 8(4): 402-9.