



Effective of aerobic and anaerobic exercise training on the inflammatory markers and lipid plasma levels

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Abstract

The purpose of the present study was to investigate the effective of aerobic and anaerobic exercise training in some inflammatory markers affecting cardiovascular disease. 45 young healthy men randomly were selected among the students of Hamadan Payame Noor University and were divided into 3 groups including: 1. The experimental group 1 receiving aerobic exercise training, 2. The experimental group 2 receiving anaerobic exercise training, and 3. The control group receiving no training. The experimental groups received the relevant exercise training for 8 weeks. The control group did not receive any exercise training in this period. The levels of C-Reactive Protein (CRP), inter leukin-6, High-Density Lipoprotein (HDL), low-density lipoprotein, and fibrinogen were assessed and compared before and after the training period. The levels of Fibrinogen in the experimental group 1 decreased significantly in the post-test than the pre-test. Also, the results showed that the levels of CRP in the experimental group 1 decreased significantly than the control group. The levels of fibrinogen in the experimental group 1 decreased significantly than the control group in the post-test. In the experimental group 2, the levels of HDL increased significantly than the control group in the post-test. Other variables did not change between the groups. The effective of both aerobic and anaerobic exercise training was proven in reducing some cardiovascular risk factors, although the aerobic exercise was more effective than the anaerobic exercise.

Keywords: Acute Proteins, Atherosclerosis, Inflammation

Introduction

Cardiovascular disease is one of the main causes of death in the world [1]. Various mechanisms and factors are involved in the development of cardiovascular disease and inflammatory mechanism is one of the most

effective mechanisms in the development of this disease [2,3]. In the past decade, paying attention to the role of inflammation in atherosclerosis increased and it was found that inflammatory mechanism plays an important

role in the pathogenesis of several chronic disease processes such as cardiovascular disease [3]. In addition, it has been shown that sensitive and specific markers of inflammation are able to predict cardiovascular disease [4]. Cardiovascular disease is the main cause of death in Iran. According to the Ministry of Health, 38.5 percent of deaths in Iran occur due to the cardiovascular disease [5]. Also, more than 64 million people in the United States suffer cardiovascular disease; in addition, annually about 931,000 deaths in the United States are due to cardiovascular disease which spends more than 365 million Dollars a year [6]. Inflammation is a response of tissue to damage or infection. Low-grade of coronary inflammation has been shown by the increased levels of some cytokines such as interleukin-6 and the reduced levels of C-reaction protein and fibrinogen [7]. It has been suggested that indices like fibrinogen, coagulation factors of VIII and IX, haptoglobin, adhesion molecules, serum amyloid-A, cytokines [particularly interleukin-1 and interleukin-6], and C-Reaction Protein (CRP) are able to predict the risk of cardiovascular disease with high sensitivity and accuracy [8]. Elevated reactants of acute phase such as fibrinogen and CRP may reflect the burden of atherosclerosis or extra-vascular inflammation that can exacerbate atherosclerosis and its complications [9]. CRP is an Acute Phase Protein and a sensitive marker of cardiovascular disease which is produced in the liver and its production will increase at inflammation [10]. The increased levels of CRP are associated with the high risk of heart disease, stroke, and vascular diseases. It is assumed that the change in the concentration of inflammatory cytokines such as Inter Leukin-1, Inter Leukin-6 (IL-6), and tumor necrosis factor-alpha leads to the changes in CRP production. Stewart and colleagues showed that changes in IL-6 production levels occur through intensive training exercise which could be effective in CRP production [7]. In fact, IL-6 is a cytokine that plays an essential role in the induction of CRP production in the liver [4]. The production and release of IL-6 from myofibrils can also

be effective in reducing subcutaneous fat; so the interleukin-6 may lead to the significant increase in rates of lipolysis and fat oxidation. Thus, IL-6 have introduced as a new lipolytic factor [10,11]. Fibrinogen as an inflammatory factor is also associated with cardiovascular disease [3,12]. The theory of the relationship between fibrinogen and cardiovascular disease was first presented in 1950. At that time, fibrinogen levels in patients with ischemic heart disease were measured and it was found that its level was higher than the normal [12]. Nowadays, it is absolutely clear that a high level of fibrinogen has a direct association with the risk of cardiovascular disease and some evidences show that it acts independently of lipoproteins [3]. The increased fibrinogen levels could lead to the increased risk of thrombosis. Increasing the amount of fibrinogen and plasminogen activator inhibitor-1 (PAI-1) is one of the risk factors of atherosclerosis which is related to blood coagulation system and causes fibrinolysis inhibition [13,14]. Fibrinogen is also capable to displacement and proliferation of smooth muscle cells and probably, such as IL-6, it is effective in the formation of atherosclerotic plaques [15]. Studies have shown that individuals, who regularly exercise, have lower fibrinogen levels than sedentary people [12]. Generally, offering a good exercise program that has high efficacy in reducing cardiovascular risk factors may have other consequences including promoted public health, reduced costs of health, reduced mortality and morbidity rate, and also extended life span, therefore it has a great importance. However, the high rate of mortality due to cardiovascular disease is so high, and also the mechanisms and factors involved in this disease have not been discovered by the studies conducted so far, therefore in order to achieve the training program which has the highest efficacy in this regard, it is necessary that the effects of different training methods on various factors involved in the onset of atherosclerosis are scrutinized. For this purpose, according to

the general classification of exercise training into two categories of aerobic and anaerobic, we investigated the effective of both aerobic and anaerobic exercise training in some inflammatory markers including CRPs, IL-6, High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), and fibrinogen to offer some important and practical suggestions to prevent cardiovascular disease.

Method

This was a quasi-experimental and a practical study, conducted to evaluate and compare the effective of aerobic and anaerobic exercise training in lipid and inflammatory markers level in young people. All participants had to meet the following requirements before enrollment: no smoking, no specific disease, no family history of heart disease, no taking specific drugs, and the age range of 18-24 years. The sample size by Cochran relation was estimated as follows [16]. The size of population was 149 participants; therefore by using the formula, the sample size was estimated 45 participants. Totally, based on the inclusion criteria and taking into account the possibility of drop-out, 48 participants were selected from all the participating volunteers and 45 participants remained in the study to the end. The target population of the present study was healthy men, so regarding the inclusion and exclusion criteria, 48 healthy young men randomly were selected among the students of Payam-Noor University of Hamadan as participants. According to Beck Health Questionnaire and their history of sport activities, the participants were selected randomly among all the volunteers who did not have any regular exercise in the last year. All the participants completed a consent form. The participants were trained as the protocol and the proper implementation of the motion in a gathering and their weight and height were measured by using a digital scale manufactured by Sartorius Company of Germany. The participants were randomly divided into three groups (N=15), including the experimental group-1 (EG-1), the experimental group-2

(EG-2), and the control group. Then, both experimental groups carried out their planned exercise training for 8 weeks with frequency of 3 times a week. The training program of the experimental group-1 comprised endurance training, so that they investigated 25 min running at 60% of maximal heart rate (HR_{max}) per session in the first four weeks, and 30 min running at 60% of maximum heart rate (HR_{max}) per session in the second four weeks. Polar watches were provided for the participants to measure heart rate and intensity of exercise. The maximum heart rate was calculated based on the equation of [$HR_{max} = (220 - \text{age}) \pm 10$] to control the exercise intensity. Then, the exercise heart rate equal to 60% of HRmax was calculated for each subject, so that each participant was running at his desired intensity and also was able to control the exercise intensity by his polar watch. The experimental group 2 investigated resistance trainings in the first four weeks with an intensity of 65-70% of one maximum repetition (1RM) for 3 sets, 6-8 reps per set and in order to comply the principle of overload in the next four weeks with an intensity of 75-85% of one maximum repetition (1RM) for 3 sets, 6-8 reps per set. The resistance training program used in the present study was based on the Keiser, Fresno, CA protocol. Before starting the training program, one repetition maximum (1RM) of participants was measured according to the formula of [$1RM = \text{Displaced weight (kg)} \div [1.0278 - (0.027 \times \text{maximum number of repetitions to fatigue})]$], at eight workouts including bench press barbell, barbell biceps, barbell triceps, leg press machine, leg extension machine, lying leg curls, lunge and military press. The 1RM data were used to plan the resistance training for each subject. The rest time was considered as 3 minutes between any two stations and 1 minute between every set [7]. The control group did not investigate any regular sport or physical activity during this period. The subject's regular diet was not changed and the participants did not use any drug during

the research. In order to determine the levels of CRP, IL-6, fibrinogen, HDL, and LDL in the pre and post-test, blood samples (10 cc) were collected at 8 to 10 A.M. from left anterior veins of the participants. In order to control the life cycle effects, the participants fasted for 12 hours. HDL and LDL levels were measured by enzymatic (colorimetric) method using kits Rosch companies and Cobas Integra auto-analyzer manufactured in the United States. Fibrinogen was measured by biuret method. In this method, for quantitative measurement of fibrinogen, we used plasma derived from citrated blood samples by centrifugation in 1500-2000 rpm and finally when the sample was prepared, by adding reagents and solutions (saline, biuret solution, serum control) it was measured against a blank using a spectrophotometer at wavelength of 560 nanometers. CRP levels were measured by using the Markit-M Kit manufactured in Japan and immunoturbidometric method. Also, the levels of IL-6 were measured by Enzyme-Linked Immuno Sorbant Assay (ELISA) method using Bender Med Systems Company

kit manufactured in Austria. Due to the random distribution of the participants in the three groups as well as the normality of data (confirmed in Kolmogorov-Smirnov test), we used one-way ANOVA and Tukey test to evaluate the variations in the pre and post-test. In addition, we used paired sample T-test to investigate the variations within the groups. The level of significance was set at $\alpha \leq 0.05$ and the data were analyzed using SPSS-18 software.

Results

The age distribution and demographic characteristics of participants have been shown in Table 1 and 2, respectively. The participants were randomly divided into three groups (N=15), the experimental group 1 (age: 20.87 ± 1.59 years, height: 175.46 ± 5.51 cm, weight: 67.51 ± 7.79 kg), the experimental group 2 (age: 21.60 ± 2.06 years, height: 178.93 ± 5.09 cm, weight: 66.88 ± 8.58 kg), and the control group (age: 21.53 ± 1.88 years, height: 176.33 ± 5.44 cm, weight: 65.61 ± 9.98 kg).

Table 1 The distribution of participants by age

	Frequency	Percent of frequency	Percent of cumulative frequency
18-20 years	24	24%	24%
20-22 years	45	45%	69%
22-24 years	31	31%	100%
	100	100	

Table 2 Demographic characteristics of participants

Variable	M \pm SD	Min	Max
Weight (Kg)	4.5 \pm 66.5	62	71
Height (cm)	4.88 \pm 176.7	171	179
Age (years)	2.2 \pm 21.62	18	24

According to the results of one-way ANOVA, in the pre-test there was not any significant difference between variables in three groups, so the groups were homogeneous. As shown in Table 3, fibrinogen levels of experimental group 1 significantly decreased in the post-test compared to the pre-test ($p \leq 0.05$). Also, the results of one-way ANOVA and Tukey post-hoc test showed that in the post-test, the CRP levels significantly decreased in the experimental group 1 compared with the control group ($p \leq 0.029$, $F=0.037$), and the

levels of fibrinogen significantly decreased in the experimental group 1 compared with the control group. ($p \leq 0.037$, $F=3.579$). The HDL levels in the experimental group 2 significantly increased compared with the control group ($p \leq 0.026$, $F=4.007$). Other variables did not significantly change between the groups. In Table 3, the levels of CRP, fibrinogen, IL-6, HDL, and LDL in the experimental group 1 and 2 and the control group in the pre and post-test have been shown.

Table 3 *The levels of risk factors for cardiovascular disease in participants*

	Group	M±SD (Pre-Test)	M±SD (Post-Test)	P
CRP (Kg)	EG-1	0.59±0.9	0.52±0.4	0.27
	EG-2	0.80±0.9	0.67±0.75	0.34
	Control	1.08±1.0	1.17±0.77	0.59
Fibrinogen (cm)	EG-1	207.6±27.8	177.5±13.1	*<0.01
	EG-2	206.1±24.2	187.7±13.9	0.18
	Control	199.1±37.8	197.5±29.9	0.26
IL-6 (years)	EG-1	1.73±2.15	2.31±2.46	0.50
	EG-2	2.93±3.24	3.07±2.57	0.38
	Control	1.63±2.06	1.53±1.62	0.25
HDL (cm)	EG-1	54.13±8.7	55.87±8.4	0.93
	EG-2	54.60±6.2	60.93±5.8	0.96
	Control	55.07±7.4	53.47±7.7	0.97
LDL (years)	EG-1	88.07±26.35	88.20±24.85	0.93
	EG-2	104.53±25.06	93.33±25.59	0.81
	Control	106.67±27.89	104.33±28.49	0.87

*p<0.05

Discussion

According to the findings of present study, it is clear that aerobic exercise had favorable effects on inflammatory markers such as CRP and fibrinogen, and anaerobic exercise had remarkable effects on HDL levels, therefore it can be cited that exercise training can reduce the risk of cardiovascular disease. In relation to the CRP levels, the results of one-way ANOVA and Tukey test showed that the CRP levels significantly decreased in the experimental group 1 compared with the control group in the post-test. Prospective studies have shown that inflammation and endothelial dysfunction are involved in the pathogenesis of cardiovascular disease [17]. The increased levels of adhesion molecules as an indicator of endothelial dysfunction and inflammatory markers are associated with the incidence of atherosclerosis, insulin resistance, and type-2 diabetes [18-20]. CRP is an acute phase protein and its increase causes the enhanced risk of coronary artery disease by 2-5 folds. CRP increases in people with high fat and it has an inverse relation to the insulin sensitivity and direct relation to the risk of type II diabetes [21]. Gaeini stated that among the inflammatory markers of atherosclerosis development, CRP is known as the most sensitive indicator of inflammation and an independent predictor of this disease.

He investigated the effects of 12-week aerobic exercise on CRP levels of old obese female rats. The results showed that regular aerobic exercise significantly decreased the CRP levels and atherogenesis process [22]. Ryan observed that 6-month resistant-aerobic combined exercise training reduced the CRP levels and IL-6 in obese postmenopausal women [10]. King and O'Donovan showed that the intensity and duration of workout has an effect on changes of inflammatory markers [2,23]. In another study, Nicklas found out that 18 months resistant-aerobic combined exercise training has not any significant effect on serum CRP levels in elderly obese men and women with knee osteoarthritis symptoms [24]. Therefore, our results are consistent with the results of Ryan, King and O'Donovan, but are inconsistent with the results of Nicklas. The reason for this inconsistency may be due to the participants studied, because the participants in the present study were young healthy inactive participants with normal weight, while in the study conducted by Nicklas participants were elderly obese men and women with osteoarthritis knee arthritis symptoms. In relation to the fibrinogen levels, it has been shown that the aerobic exercise has a significant effect on reducing the amount of

cardiovascular risk factors. The paired T-test results showed that the fibrinogen levels of experimental group 1 significantly decreased in the post-test compared to the pre-test ($p \leq 0.05$). Also, the results of one-way ANOVA and Tukey test showed that in the post-test, the fibrinogen levels of experimental group 1 significantly decreased compared with the control group. In a review study, Siscovick surveyed the studies during the years of 1989-1990 in relation to the effects of intensity and duration of exercises on reducing cardiovascular risk factors and it was found that the fibrinogen levels decreases by investigating high intensity exercises [25]. Also, Ernst in a study observed that the fibrinogen levels decreased by investigating exercises training [12]. Therefore, according to the findings of present study, our findings in relation to the decreased levels of fibrinogen are consistent with the findings of Ernst and Siscovick. In relation to the levels of IL-6, although there was no statistically significant change in the IL-6 levels of participants, IL-6 concentration increased remarkably in the post-test compared with the pre-test. This shows that if workout programs continued for a longer period, it might cause the more remarkable changes in the participants. Bodell showed that although IL-6 is a pre-inflammatory marker, not only do not make disorders, but also it can have favorable effects on the growth of children [26]. Keller stated that the expression of IL-6mRNA and IL-6 levels were significantly increased compared to the pre-test after 10 weeks exercise training [27]. In a study, Drenth showed that significant changes in plasma cytokines occurred due to the exercise training. He cited that the levels of IL-1ra and IL-6 significantly increased after the training period [11]. Ronsen showed that increasing the levels of IL-6 and IL-1ra is more remarkable by continuous short-term exercise training (3 hours) compared with the long-term exercises (6 hours) [28]. The results of one-way ANOVA and Tukey post-hoc test showed that the HDL levels in the experimental group 2 significantly increased compared with the control group in the post-test. These results indicated that

resistance training cause significant changes in the concentration of HDL. Slentz showed an increase in the HDL levels of all three experimental groups with different exercise intensity but he cited that high-intensity exercise trainings showed the more significant increase in HDL concentrations [29]. In a study by Duncan an increase in the HDL levels observed only in high-intensity exercise [30]. According to what was mentioned, our results are consistent with the results of Slentz and Duncan. In a study by Weise, the HDL levels decreased, so their results are inconsistent with our findings [31]. Reduction in the HDL levels was probably due to the reason that a single session of aerobic exercise cannot increase the HDL levels and long-term training is needed to create favorable variations in the HDL levels. Meanwhile, Banz stated that resistance training does not induce changes in the HDL levels, so his findings are inconsistent with the results obtained in the present study [32]. Probably no changing HDL levels were due to the reason that their workout protocol included sub-maximal resistance training. The results of the present study showed that the LDL levels did not change significantly in all the three groups, so our results are consistent with the findings of Swain, Duncan and Banz that cited exercise training cannot significantly reduce the LDL levels [1,30,32]. Slentz stated that the reduction in LDL levels observed in the high intensity, low volume training group [29]. Therefore, it can be said that to favorable changes in LDL levels, workouts should be investigated as prolonged high intensity and volume exercises.

Conclusion

In conclusion, we can say that both aerobic and anaerobic exercise training have favorable effects on reducing the risk of cardiovascular disease. Aerobic exercise has desirable effects on inflammatory markers such as CRP and fibrinogen, and anaerobic exercise affects the HDL levels. However, more studies are needed to control other mechanisms in various exercise training. In

the present study, the control of motivation and the rest of the participants were not possible and also we were not able to investigate all the factors affecting cardiovascular disease. So, researchers in future studies can evaluate the effective of exercise training in other factors of this disease.

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Contribution

Study design: KAD

Data collection and analysis: KAD

Manuscript preparation: KAD

Conflict of Interest

"The author declares that they have no competing interests."

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References

- 1- Swain DP, Franklin BA. Comparison of cardioprotective benefits of vigorous versus moderate intensity aerobic exercise. *Am J Cardiol*2006; 97(1): 141-7.
- 2- King DE, Carek P, Mainous A, et al. Inflammatory markers and exercise: differences related to exercise type. *Med Sci Sports Exerc*2003; 35(4): 575-81.
- 3- Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. *N Engl J Med*2005; 352(16): 1685-95.
- 4- Tuomisto K, Jousilahti P, Sundvall J, et al. C-reactive protein, interleukin-6 and tumor necrosis factor alpha as predictors of incident coronary and cardiovascular events and total mortality a population-based, prospective study. *Thromb Haemost*2006; 95(3): 511-8.
- 5- Sheikholeslami Vatani D, Ahmadi S, Mojtahedi H, et al. Effect of moderate and high intensity resistant exercises on cardiovascular risk factors in non-athlete university students. *Kowsar Medical Journal*2011; 16(2): 115-21.
- 6- Braith RW, Stewart KJ. Resistance exercise training its role in the prevention of cardiovascular disease. *Circulation*2006; 113(22): 2642-50.
- 7- Stewart LK, Flynn MG, Campbell WW, et al.

The influence of exercise training on inflammatory cytokines and C-reactive protein. *Med Sci Sports Exerc*2007; 39(10): 1714.

8- Wilund K. Is the anti-inflammatory effect of regular exercise responsible for reduced cardiovascular disease? *Clin Sci*2007; 112: 543-55.

9- Dixon NC, Hurst TL, Talbot DC, et al. Active middle-aged men have lower fasting inflammatory markers but the postprandial inflammatory response is minimal and unaffected by physical activity status. *J Appl Physiol*2009; 107(1): 63-8.

10- Ryan AS, Nicklas BJ. Reductions in plasma cytokine levels with weight loss improve insulin sensitivity in overweight and obese postmenopausal women. *Diabetes Care*2004; 27(7): 1699-705.

11- Drenth J, Van Uum S, Van Deuren M, et al. Endurance run increases circulating IL-6 and IL-1ra but downregulates ex vivo TNF-alpha and IL-1 beta production. *J Appl Physiol*1995; 79(5): 1497-503.

12- Ernst E. The role of fibrinogen as a cardiovascular risk factor. *Atherosclerosis*1993; 100(1): 1-12.

13- Green DJ, O'Driscoll G, Joyner MJ, et al. Exercise and cardiovascular risk reduction: time to update the rationale for exercise? *J Appl Physiol*2008; 105(2): 766-8.

14- Szymanski LM, Kessler CM, Fernhall B. Relationship of physical fitness, hormone replacement therapy, and hemostatic risk factors in postmenopausal women. *J Appl Physiol*2005; 98(4): 1341-8.

15- Black S, Kushner I, Samols D. C-reactive protein. *J Biol Chem*2004; 279(47): 48487-90.

16- Fleiss J. The design and analysis of clinical experiments. London: Taylor & francis publishing; 1986.

17- Vanhoutte PM. Endothelial dysfunction the first step toward coronary arteriosclerosis. *Circ J*2009; 73(4): 595-601.

18- Sahakyan K, Klein BE, Lee KE, et al. Inflammatory and endothelial dysfunction markers and proteinuria in persons with type 1 diabetes mellitus. *Eur J Endocrinol*2010; 162(6): 1101-5.

19- Bertoni AG, Burke GL, Owusu JA, et al. Inflammation and the incidence of type 2 diabetes the multi-ethnic study of atherosclerosis (MESA). *Diabetes Care*2010; 33(4): 804-10.

20- Rodondi N, Marques-Vidal P, Butler J, et al. Markers of atherosclerosis and inflammation for prediction of coronary heart disease in older adults. *Am J Epidemiol*2010; 171(5): 540-9.

21- Hamedinia M, Haghghi A, Ravasi A. The effect of aerobic training on inflammatory markers of cardiovascular disease risk in obese men. *World J Sport Sci*2009; 2(1): 7-12.

22- Gaeini A, Dabidi RV, Ravasi A, et al. The effect of

the period of the intermittent aerobic training on hs-crp in the old rats. *Research on Sport Sciences*2008; 19(6): 39-54.

23- O'Donovan G, Owen A, Bird SR, et al. Changes in cardiorespiratory fitness and coronary heart disease risk factors following 24 wk of moderate-or high-intensity exercise of equal energy cost. *J Appl Physiol*2005; 98(5): 1619-25.

24- Nicklas BJ, Ambrosius W, Messier SP, et al. Diet-induced weight loss, exercise, and chronic inflammation in older, obese adults: a randomized controlled clinical trial. *Am J Clin Nutr*2004; 79(4): 544-51.

25- Siscovick DS, Fried L, Mittelmark M, et al. Exercise intensity and subclinical cardiovascular disease in the elderly The cardiovascular health study. *Am J Epidemiol*1997; 145(11): 977-86.

26- Bodell PW, Kodesh E, Haddad F, et al. Skeletal muscle growth in young rats is inhibited by chronic exposure to IL-6 but preserved by concurrent voluntary endurance exercise. *J Appl Physiol*2009; 106(2): 443-53.

27- Keller C, Steensberg A, Hansen AK, et al. Effect of exercise, training, and glycogen availability on IL-6 receptor expression in human skeletal muscle. *J Appl Physiol*2005; 99(6): 2075-9.

28- Ronsen O, Lea T, Bahr R, et al. Enhanced plasma IL-6 and IL-1ra responses to repeated vs. single bouts of prolonged cycling in elite athletes. *J Appl Physiol*2002; 92(6): 2547-53.

29- Slentz CA, Houmard JA, Johnson JL, et al. Inactivity, exercise training and detraining, and plasma lipoproteins. STRRIDE: a randomized, controlled study of exercise intensity and amount. *J Appl Physiol*2007; 103(2): 432-42.

30- Duncan GE, Anton SD, Sydeman SJ, et al. Prescribing exercise at varied levels of intensity and frequency: a randomized trial. *Arch Intern Med*2005; 165(20): 2362-9.

31- Weise SD, Grandjean PW, Rohack JJ, et al. Acute changes in blood lipids and enzymes in postmenopausal women after exercise. *J Appl Physiol*2005; 99(2): 609-15.

32- Banz WJ, Maher MA, Thompson WG, et al. Effects of resistance versus aerobic training on coronary artery disease risk factors. *Exp Biol Med*2003; 228(4): 434-40.