



www.aassjournal.com

ISSN (Online): 2322 – 4479

Original Article

Received: 31/07/2013

Accepted: 05/11/2013

Effect of Pyramidal Training on Plasma Lipid Profile and Fibrinogen, and Blood Viscosity of Untrained Young Men

¹Abbass Ghanbari –Niaki, ²Mostafa Behzad Khameslo*,
³Seyed Morteza Tayebi

1- Exercise Biochemistry Division, Faculty of Physical Education and Sport Sciences, Mazandaran University, Babolsar, Iran.

2- Sama Technical and Vocational Training college, Islamic Azad University, Sari Branch, Sari, Iran.

3- Faculty of Physical Education and Sport Science, Allameh Tabataba'i University, Tehran, Iran.

ABSTRACT

The present study examined the effects of progressive exercise (pyramidal) short-term program on plasma fibrinogen, lipid profile and blood viscosity in untrained young men. Changes and imbalances in homeostasis lead to cause of heart attacks. There is conflicting information about the effect of exercise on these factors. 19 young healthy untrained men were randomly assigned to the exercise group (n = 10) and controls (n = 9) groups. Exercise training group with increasing severity of heart maximum 25 to 100 percent began to run the practice (pyramid) in 42-minute sessions, 3 times a week, for 4 weeks. There was no significant difference between the parameters of body composition, control, and training groups. Levels of total cholesterol and low density lipoprotein density and viscosity of the blood significantly decreased in the training group compared with the control group (p value, respectively is 0.001, 0.001 and 0.035). The changes in the concentration of fibrinogen, high-density lipoprotein and triglycerides in both groups are not significant in both groups (p value, respectively is 0.645, 0.993 and 0.421). The present results show that it is possible that progressive training (pyramidal) short-term program changed the levels of cardiovascular risk factors by reducing blood viscosity.

Keywords: progressive exercise pyramid, fibrinogen, blood viscosity, lipid profile.

Corresponding Author:

Mostafa Behzad Khameslo

E-mail: mostafabehzadkh@yahoo.com

INTRODUCTION

Cardiovascular is a hazardous and lethal problem in the industrialized world today. One of the most important heart diseases - coronary common is atherosclerosis (1). Studies have shown that inflammation plays a pivotal role in the progression of atherosclerosis. It can also lead to increased inflammation, coagulation factors including fibrinogen levels, which stimulate prothrombin and causing the development and progression of atherosclerosis (2). The research suggest that there are some fibrinogens as a cofactor for platelet aggregation and thrombin specific substances in blood coagulation, and it plays a central role in the initiation and progression of cardiovascular disease (3). Fibrinogen is not only considered as an indicator of inflammation (3), but as a part of the process depending on homeostasis and thrombosis which has also been noted (4). Homeostasis body system imbalance can lead to a form of sudden and often dying of blood clots (5). Several factors such as hypercholesterolemia, hyperlipidemia, and increased lipoprotein can stimulate or increase deformed inflammation (2). One of the main mechanisms triggering inflammation, low density lipoprotein levels may change as the main cause of damage to the endothelium and vascular smooth muscle is known (6). Also low density lipoprotein in the presence of fibrinogen deposition appears to be related to the vascular wall (7). On the other hand, it is believed to improve cardiovascular index of high-density lipoprotein. Lipoprotein lipase activity, fibrinogen, homocysteine, and blood viscosity prevent heart disease, which are directly related to cardiovascular disease (8, 9). Clotting factors in the blood stream and spread cardio - vascular problems associated with it are important (10). Blood viscosity depends on blood viscosity and thickness. One can say hematocrit, viscosity and blood

viscosity are directly correlated with each other and plasma volumes are inversely correlated with plasma volume (11). Increased hematocrit, blood viscosity increased and thus the amount of blood flow velocity decreases and thus reduces the oxygen supply to the tissues (11, 12). In addition to the increase in blood hematocrit, increased friction between adjacent layers, as a result, leads to the progress of heart-vascular disease (11).

Although the acute exercise have a no effect (13) or results in a blood coagulation increase (14, 15); exercise training, particularly aerobic exercises by reducing lipid levels, high-density lipoprotein levels, reduce oxidative stress and improve clotting factors, which can enhance the performance of cardio-vascular causes (15-17). However, studies have shown that improving the physical fitness of exercise, especially after aerobic exercise has a significant positive effect on blood clotting time leaves (17, 18). Hence, aerobic exercises can be as a tool for treatment and prevention of heart-vascular disease (17, 18). Borer et al. reported that after 15 weeks of endurance training fibrinogen levels significantly increased in postmenopausal women (19). While, the 12-week walking exercises (20 to 30 minutes of fast walking, three times a week, on with moderate intensity during daily activity) were not associated with a significant decrease in fibrinogen levels in women (20). Fibrinogen levels in adolescent boys are off, but after an exhaustive session up and down stairs, (The alternating rhythmic activity of one minute and 30 seconds of rest) they mean no significant change (21). The research results indicate the effects of different variables such as subject number, type, severity, duration, acute and chronic exercise on inflammatory protein response. Although some of the benefits of physical activity may be due to its effects on homeostasis system; but increased

cardiovascular events and sudden death occur during or immediately after exercise, to further investigate the relationships between physical activity and the demands of homeostasis (22). Very limited research on the effect of increasing aerobic exercise (pyramidal) on plasma levels of fibrinogen, especially in young people is done. Therefore, this study was conducted. The present study investigated the short-term effects of 4 weeks (pyramidal) on fibrinogen, blood viscosity and lipid profile in untrained young men.

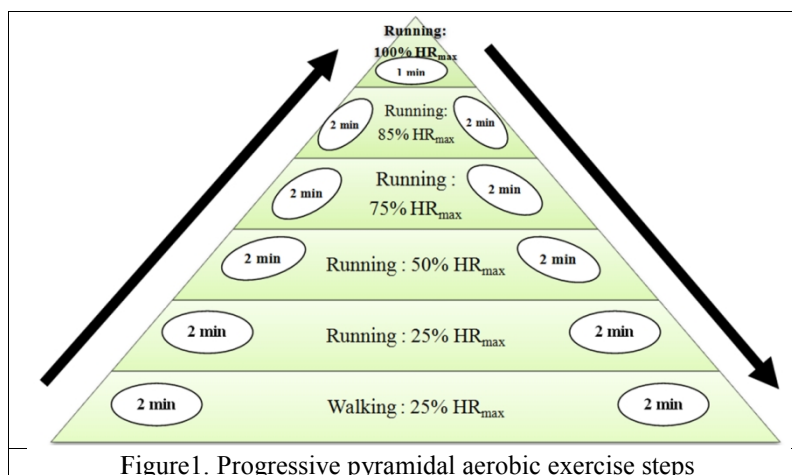
MATERIALS AND METHODS

This quasi-experimental study was performed with pre-test and post-test model.

Subject. 19 inactive male students from Maziar University of city of Mahmud Abad, who hadn't heart and cardiovascular disease, renal and liver dysfunction, and tobacco and alcohol consumption, and also were not treated with steroid drugs, special diets, and regular exercise, participate in the study

voluntarily. Written consent was obtained from participants after preliminary investigation and primary selection. Then Eligible subjects were randomly divided into two groups, the practice ($n = 10$) and the control ($n = 9$).

Procedure. Height, weight, percent body fat, blood pressure of subjects and body fat percentage were measured using a body composition analyzer. Training program includes four weeks (three sessions per week) of extension training (pyramidal) in two 21-minute cardio, with 10 minutes rest, between exercises, 42 minutes in total at an intensity between 25 and 100 percent maximum heart rate of subjects (Figure 1), the maximum heart rate of each subject was controlled using a polar heart rate monitor (Model AXN300 made in Finland) based on Karvonen Heart Rate formula calculation. Each training session contains a warm-up (10 min), main exercise (figure 1), and cool-down (10 min) with light running and stretching (23).



Blood sampling was conducted 48 hours before the first training session and 48 hours after the last training session. Subjects were asked to refrain from consuming food 12 hours before blood sampling. In order to maintain circadian rhythm and control sampling time, samples were assessed before

and after 8 am. Right brachial vein blood samples was maintained from the subjects was 10 ml, the material in tubes containing anticoagulant (EDTA) (Ethylene diamine Tetra Acetic Acid) is quickly gathering speed centrifugation and collected by centrifugation (2000 rpm speed for 10

minutes) and plasma samples were obtained until the freezer at - 80 ° C. Fibrinogen level measured by the analyzer were made in Germany. Plasma viscosity was calculated by the following equation (24):

$$\text{Plasma viscosity} = 1.352 + 0.0167 \bullet \text{TC (mmol)} + 0.0285 \bullet \text{fibrinogen (g/l)} + 0.0054 \bullet \text{TG (mmol)} + 0.00318 \bullet \text{hematocrit} - 0.03 \bullet \text{HDL-C (mmol)}$$

Concentrations in plasma lipid levels (TC, TG, and HDL-C) were measured by a device (Cobas Integra 400 plus Analyzer) made in Germany. For LDL-c was calculated by the following equation (25):

$$\text{LDL-c (mg/dL)} = \text{TC} - (\text{HDL-c} + \text{TG} / 5)$$

Statistical Analysis. Normal distribution confirmed using Kolmogorov - Smirnov test, ANOCOVA was used to statistical analysis and comparison between groups, and Pearson's correlation coefficient was used to

determine the relationship between the variables. All data as mean and standard deviation are presented in calculations using SPSS16 and the significance level of the tests was $p \leq 0.05$.

RESULTS

Physical and functional factors.

Physical and functional characteristics of experimental and control groups are listed in Table 1. Within the group of subjects, a mean showed weight training 19/1%, and body mass index of 14/1 were decreased, but the changes were not statistically significant ($p=0.407$) and ($p=0.283$). ANCOVA test results also show that 4 weeks cumulative pyramid in the experimental group than in the control group had no effect on body fat and lean body mass, the value of p is respectively equal to ($p=0.467$) and ($p=0.320$).

Table 1. physical characteristics of subjects

Variables	Group	Pre-Training (M ± SD)	Post-Training (M ± SD)
Age (Years)	Experimental	18.70 ± 3.97	-
	Control	18.56 ± 1.33	-
Length (CM)	Experimental	176.80 ± 4.88	-
	Control	175.33 ± 3.02	-
Weight (Kg)	Experimental	67.10 ± 9.43	66.30 ± 10.13
	Control	63.39 ± 13.09	62.50 ± 13.11
BMI (M)	Experimental	21.43 ± 2.45	21.19 ± 2.82
	Control	22.18 ± 3.84	22.32 ± 3.66
Percent body fat (%)	Experimental	15.90 ± 6.26	15.06 ± 6.76
	Control	17.07 ± 7.50	16.68 ± 7.18
The net mass of the body (Kg)	Experimental	78.55 ± 4.94	80.02 ± 6.18
	Control	78.20 ± 6.95	78.53 ± 6.64

Coagulation and cardiovascular factors. The results from the concentrations of measured variables are shown in Table 2. Fibrinogen concentration after training in the experimental group is 5.55% and in the control group is 8.81%, however, the changes between the two groups was not statistically significant ($p=0.246$). Intergroup

study shows that the average level of blood viscosity in the experimental group is 0.125, which decreased, and there was no change in the control group, but in between the two groups were observed that the effect of exercise on blood viscosity levels were statistically significant ($p=0.035$). The serum lipid profiles of measured variables are

shown in Table 2. after 4 weeks of exercise training increased the levels of triglycerides and high-density lipoprotein pyramid was not statistically significant, The p value respectively was (p=0.421) and (p=0.993),

while no significant difference were observed in levels of low-density lipoprotein cholesterol in the experimental group than the control, respectively, with the p value (p=0.001) and (p=0.001).

Table 2. concentration of coagulation and cardiovascular factors

Variables	Group	Pre-Training (M ± SD)	Post-Training (M ± SD)
Blood viscosity (mm)	Experimental	1.4648 ± 0.0201	1.4523 ± 0.01709*
	Control	1.4462 ± 0.0984	1.4468 ± 0.0096
Fibrinogen (mg/dl)	Experimental	246.06 ± 61.42	232.40 ± 63.01
	Control	201.46 ± 11.64	183.70 ± 23.51
TG (mg/dl)	Experimental	107.30 ± 35.33	97.40 ± 37.64
	Control	87.44 ± 35.39	81.44 ± 27.69
TC (mg/dl)	Experimental	166.10 ± 28.39	147.40 ± 24.29**
	Control	143.88 ± 59.88	159.88 ± 24.31
HDL-c (mg/dl)	Experimental	47.80 ± 16.65	47.60 ± 14.33
	Control	41.55 ± 8.27	42.66 ± 8.48
LDL-c (mg/dl)	Experimental	96.50 ± 17.31	79.90 ± 16.22**
	Control	84.33 ± 17.88	100.44 ± 23.11

TG: Triglyceride. **TC:** Total Cholesterol. **HDL-c:** Cholesterol High Density Lipoprotein. **LDL-c:** Cholesterol Low Density Lipoprotein. *: Significant difference between the groups at p < 0.05. **: Significant difference between the groups at p < 0.01

Relationship of coagulation and cardiovascular factors. To determine the correlation between blood viscosity and fibrinogen levels with other cardiovascular parameters measured variables was used the Pearson correlation analysis. As it can be seen in Table 3, the blood viscosity changes with LDL and total cholesterol levels were

significantly related to changes there. While the viscosity of the blood, there was no significant correlation between changes in HDL and triglycerides. There is no correlation between fibrinogen changes with changes in cardiovascular parameters as well.

Table3. Pearson correlation of coagulation and cardiovascular factors

Variables	TG	TC	HDL-c	LDL-c
Fibrinogen	0.048	-0.291	-0.213	-0.232
Blood Viscosity	0.445	0.611*	-0.206	0.536**

TG: Triglyceride. **TC:** Total Cholesterol. **HDL-c:** Cholesterol High Density Lipoprotein. **LDL-c:** Cholesterol Low Density Lipoprotein. *: Significant correlation at p < 0.05. **: Significant correlation at p < 0.01

DISCUSSION

The study found that 4 weeks of growing pyramidal practice made no significant changes in fibrinogen levels, triglycerides, high-density lipoprotein; however, they led to a significant reduction

in blood viscosity levels, total cholesterol and low-density lipoprotein. There is a disagreement about the effects of exercise on plasma fibrinogen, blood viscosity and its relationship with other metabolic parameters. Some researchers have reported

an increase or decrease in plasma fibrinogen, which is contrary to the results of the present study (19, 20). Increased levels of fibrinogen reported by Borer et al. in postmenopausal women after endurance training (19). While Furukawa and colleagues observed decline in middle-aged women that is contrary to the results of this study (20). However, some other researchers such as Hilberg et al. (26), El-Sayed et al. (27) observed no significant changes in plasma levels of fibrinogen, which are consistent with the results of the present study. Since a clear mechanism of fibrinogen functions and its relationship to other metabolic factors have not specified yet, it is not possible to properly explain the conflicting results of research. However, studies have shown that among the coagulation parameters, fibrinogen clotting system is the best indicator of the final substrate that is converted to fibrin by thrombin and this process depends on the amount of plasma fibrinogen. The findings showed that the physical activity can be influenced by human haemostatic system. In some studies, the duration and intensity of activities have stated as impacting factor on fibrinogen (28-30). This slight decrease in fibrinogen can be attributed to two factors: the severity and duration. Since the duration of action is relatively low, it is likely that insignificant decrease in fibrinogen levels is due to practice time. Other mechanisms for changes in fibrinogen is increased by activity of the sympathetic nervous system and changes in lipid profiles of subjects (29, 30). In the present study, for subjects in the aerobic exercise group, there is a slight reduction in lipid profiles. Some slight decreases fibrinogen can also be attributed to the decrease in lipid profiles. Fibrinogen levels associated with stress, obesity, low density lipoprotein and high-density lipoprotein has an inverse relation. . So, high-density lipoprotein and low-density lipoprotein and fat reduction is achieved as a result of aerobic exercise, which can reduce

fibrinogen (31). One reason for the general increase in fibrinogen levels in the body is too much fat, because fat can cause inflammation in the body. Thereby, reducing the body fat can reduce inflammatory processes and fibrinogen concentration in the blood (32). Also, it is likely to reduce the effect of aerobic exercise on cytokine activity, while fibrinogen is decreased. Fibrinogen synthesis is associated with decreased risk of liver cells resulting in muscle adaptation to aerobic training including such as IL-1 cytokine activity which may be reduced (33). Therefore, it is likely that the exercise intensities of 50 and 75% heart rate Byshnh cytokine such as IL-1 for 4 weeks, which in turn can decrease the hepatic synthesis of fibrinogen is also impressive. Some researchers have suggested a direct relationship between changes in BMI with fibrinogen changes. In the present study the possibility that BMI is decreased as a result of aerobic exercise followed by a decrease of high fibrinogen. Plasma fibrinogen is a major determinant of blood viscosity and flow. Based on epidemiological studies, high levels of plasma fibrinogen is not any increased risk of blood clots, cardiovascular and other diseases associated with vascular occlusion. . Regular aerobic exercise, catecholamine stimulation by increasing blood flow to the muscles and increased blood volume decreased fibrinogen levels and general well-being. Decrease in plasma fibrinogen has been insignificant in the experimental group and the control group. One of the possible reasons could be the low basal levels of fibrinogen insignificance with the control group compared to the experimental one. In general, factors such as age, malnutrition, decreased body weight, physical conditions, daily activities, seasonal changes, and hormonal status on the plasma fibrinogen affect blood coagulation (34).

The most common lipid abnormalities including elevated TG and low HDL, which

is the last parameter of risk factors for heart disease is a vessel. Associated with increased triglycerides while increasing, LDL particles are known as atherogenic factors. Research suggests that the effect of exercise on lipoprotein levels is not clear. It also suggests that a relatively high amount of regular exercise which can significantly improve the overall lipoprotein profile is created (35). Studies have shown that triglyceride levels are inversely associated with HDL levels. Overview of research on the effects of aerobic exercise on lipid profile shows some researchers rarely exercise on Tc and LDL influence levels, unless it can be combined with diet and weight loss (36). Results of this study showed a significant decrease of total cholesterol reduction. Fat tissues are autonomic with numerous capillaries and nerves. Hence, all the metabolic actions of thyroid hormones by sex and neurological control and many of which are influenced by physical activity that could be one reason for the lack of change in cholesterol levels (36). Insignificant reduction in triglycerides in the present study can be answered by lipoprotein lipase (LPL) which is attributed to exercise. Lipoprotein lipase enzymes regulate the breakdown of triglycerides in lipoproteins, triglyceride, rich lipoproteins, and triglycerides. However, studies show that regular exercise is then inhibited decreased hepatic lipase enzyme. Thus reduced triglyceride content in LDL, and appeared to be greater than the duration of the exercise, more reduction of triglycerides (36). Also in this study, there was no difference between groups in HDL levels. Researchers believe that HDL and LDL are hardly affected by the practice. No significant change may be sufficient in HDL intensity and duration of exercise in the present study. The authors

demonstrated that the mechanism of HDL changes following training complex. Enzymes such as lipoprotein lipase (LPL), hepatic TG lipase (HL) and cholesteryl ester transfer protein (CETP) play an important role in HDL concentration. LPL hydrolysis of plasma triglycerides and HDL concentration are the most important factor. The increase in LPL activity after exercise is not responsible for raising HDL. HDL levels immediately after exercise may be associated with decreased CETP activity. CETP molecule is responsible for transporting lipids in HDL and other lipoproteins have been reduced after exercise. Loss of license for which CETP catabolism of HDL (increased half-life) and finally increases HDL levels (36). The findings of the present study showed a significant decrease in blood viscosity increased after 4 weeks of the pyramidal exercises. According to Pearson's test results, this change was associated with decreased plasma levels of LDL cholesterol. The mechanisms involved in the reduction of LDL can be said practice, most of which will affect the lipid profile of higher baseline TG or LDL or low HDL. Participants in this study are relatively high of initial levels of HDL LDL, TG and Tc and why they were in the normal lipid parameters did not show significant changes (36).

CONCLUSION

The results of the present study were observed after 4 weeks of growing pyramid, reduce blood viscosity following plasma LDL cholesterol levels. According to the findings of this study, it can be stated that 4 weeks of growing pyramid can prevent the development of atherosclerotic increased pyramid.

REFERENCES

1. Ogata Y, Taniguchi N. [Vascular function tests and new technology in the early phase of atherosclerosis]. *Rinsho byori The Japanese journal of clinical pathology*. 2012;60(9):887-94. Epub 2012/11/20.

2. Willerson JT, Ridker PM. Inflammation as a cardiovascular risk factor. *Circulation*. 2004;109(21 Suppl 1):II2-10. Epub 2004/06/03.
3. Guo Y, Lip GY, Apostolakis S. Inflammation in atrial fibrillation. *Journal of the American College of Cardiology*. 2012;60(22):2263-70. Epub 2012/12/01.
4. Barbeau P, Litaker MS, Woods KF, Lemmon CR, Humphries MC, Owens S, et al. Hemostatic and inflammatory markers in obese youths: effects of exercise and adiposity. *The Journal of pediatrics*. 2002;141(3):415-20. Epub 2002/09/10.
5. Stuijver DJ, van Zaane B, Romualdi E, Brandjes DP, Gerdes VE, Squizzato A. The effect of hyperthyroidism on procoagulant, anticoagulant and fibrinolytic factors: a systematic review and meta-analysis. *Thrombosis and haemostasis*. 2012;108(6):1077-88. Epub 2012/09/28.
6. Mather KJ, Steinberg HO, Baron AD. Weight Loss and Endothelial Function in Obesity. *Diabetes Care*. 2003;26(6):1927-8.
7. Wolberg AS. Determinants of fibrin formation, structure, and function. *Current opinion in hematology*. 2012;19(5):349-56. Epub 2012/07/05.
8. Abramson JL, Vaccarino V. Relationship between physical activity and inflammation among apparently healthy middle-aged and older US adults. *Archives of internal medicine*. 2002;162(11):1286-92. Epub 2002/06/01.
9. Hovland A, Lappégard KT, Mollnes TE. LDL apheresis and inflammation--implications for atherosclerosis. *Scandinavian journal of immunology*. 2012;76(3):229-36. Epub 2012/06/08.
10. Lassila R. Role and management of coagulation disorders in peripheral arterial disease. *Scandinavian journal of surgery : SJS : official organ for the Finnish Surgical Society and the Scandinavian Surgical Society*. 2012;101(2):94-9. Epub 2012/05/25.
11. Bastard JP, Jardel C, Bruckert E, Blondy P, Capeau J, Laville M, et al. Elevated levels of interleukin 6 are reduced in serum and subcutaneous adipose tissue of obese women after weight loss. *The Journal of clinical endocrinology and metabolism*. 2000;85(9):3338-42. Epub 2000/09/22.
12. Bastard JP, Jardel C, Bruckert E, Vidal H, Hainque B. Variations in plasma soluble tumour necrosis factor receptors after diet-induced weight loss in obesity. *Diabetes, obesity & metabolism*. 2000;2(5):323-5. Epub 2001/02/28.
13. Ghanbari-Niaki A, Tayebi SM. Effects of a Light Circuit Resistance Exercise Session on Some Hematological Parameters of Male Collage Students. *Annals of Applied Sport Science*. 2013;1(1):6-11.
14. Ghanbari AR, Tayebi SM, Delrouz H. The Effect of A Single Session Eccentric Resistance Exercise On Some Blood Coagulation Factors of Inactive Male Students. *Sci J Blood Transfus Organ*. 2011;8(3):195-206 [Article in Farsi].
15. Tayebi SM, Agha-Alinejad H, Kiadaliri K, Ghorbanalizadeh F, Ghaziani. Assessment of CBC in physical activity and sport: a brief review. *Sci J Blood Transfus Organ*. 2011;7(4):249-65 [Article in Farsi].
16. Beavers KM, Nicklas BJ. Effects of lifestyle interventions on inflammatory markers in the metabolic syndrome. *Frontiers in bioscience (Scholar edition)*. 2011;3:168-77. Epub 2011/01/05.
17. Imhof A, Koenig W. Exercise and thrombosis. *Cardiology clinics*. 2001;19(3):389-400. Epub 2001/09/26.
18. Koenig W, Ernst E. Exercise and thrombosis. *Coronary artery disease*. 2000;11(2):123-7. Epub 2000/04/12.
19. Borer KT, Huang J, Sanford T, Fay W. Increased Plasma Fibrinogen and Decreased Plasmonogen Activator Inhibitor-1 (Pai-1) After 15 Weeks of Training in Postmenopausal Women. *Medicine & Science in Sports & Exercise*. 2001;33(5):S51.
20. Furukawa F, Kazuma K, Kojima M, Kusakawa R. Effects of an off-Site walking program on fibrinogen and exercise energy expenditure in women. *Asian Nursing Research*. 2008;2(1):35-45.
21. Ribeiro J, Almeida-Dias A, Ascensao A, Magalhaes J, Oliveira AR, Carlson J, et al. Hemostatic response to acute physical exercise in healthy adolescents. *Journal of science and medicine in sport / Sports Medicine Australia*. 2007;10(3):164-9. Epub 2006/07/18.
22. Lippi G, Maffulli N. Biological influence of physical exercise on hemostasis. *Seminars in thrombosis and hemostasis*. 2009;35(3):269-76. Epub 2009/05/20.
23. Ghanbari Niaki A. Effect of an Interval Progressive (Pyramidal) Aerobic Training On Lipid and Lipoprotein Profiles in San Shou Athletes. *International Journal of Humanities*. 2004;12(1):97-110.
24. Eterović D, Pintarić I, Tocilj J, Reiner Z. Determinants of plasma viscosity in primary hyperlipoproteinemias . *Clinical hemorheology and microcirculation*. 1995;15(6):841-50.
25. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the Concentration of Low-Density Lipoprotein Cholesterol in Plasma, Without Use of the Preparative Ultracentrifuge. *Clinical Chemistry*. 1972;18(6):499-502.

26. Hilberg T, Glaser D, Reckhart C, Prasa D, Sturzebecher J, Gabriel HH. Blood coagulation and fibrinolysis after long-duration treadmill exercise controlled by individual anaerobic threshold. *European journal of applied physiology*. 2003;87(1-2):100-103. Epub 2003/07/29.
27. El-Sayed MS, Lin X, Rattu AJ. Blood coagulation and fibrinolysis at rest and in response to maximal exercise before and after a physical conditioning programme. *Blood coagulation & fibrinolysis : an international journal in haemostasis and thrombosis*. 1995;6(8):747-52. Epub 1995/12/01.
28. El-Sayed MS, Sale C, Jones PG, Chester M. Blood hemostasis in exercise and training. *Medicine and science in sports and exercise*. 2000;32(5):918-25. Epub 2000/05/05.
29. Smith JE. Effects of strenuous exercise on haemostasis. *Br J Sports Med*. 2003;37(5):433-5. Epub 2003/09/30.
30. Smith JE, Garbutt G, Lopes P, Pedoe DT. Effects of prolonged strenuous exercise (marathon running) on biochemical and haematological markers used in the investigation of patients in the emergency department. *Br J Sports Med*. 2004;38(3):292-4. Epub 2004/05/25.
31. Prerost MR, Feldman BF, Herbert WG. Homocysteine, fibrinogen and physical activity in human males with coronary artery disease. *Comp Haematol Int*. 1999;9(1):25-30.
32. Balagopal P, George D, Sweeten S, Mann KJ, Yarandi H, Mauras N, et al. Response of fractional synthesis rate (FSR) of fibrinogen, concentration of D-dimer and fibrinolytic balance to physical activity-based intervention in obese children. *Journal of thrombosis and haemostasis : JTH*. 2008;6(8):1296-303. Epub 2008/05/30.
33. Boutcher SH, Meyer BJ, Craig GA, Astheimer L. Plasma lipid and fibrinogen levels in aerobically trained and untrained postmenopausal women. *The Journal of sports medicine and physical fitness*. 2003;43(2):231-5. Epub 2003/07/11.
34. Adamopoulos S, Parissis J, Kroupis C, Georgiadis M, Karatzas D, Karavolias G, et al. Physical training reduces peripheral markers of inflammation in patients with chronic heart failure. *European heart journal*. 2001;22(9):791-7.
35. Ye Y, Hou XH, Pan XP, Lu JX, Jia WP. Serum vaspin level in relation to postprandial plasma glucose concentration in subjects with diabetes. *Chinese medical journal*. 2009;122(21):2530-3. Epub 2009/12/03.
36. Kraus WE, Houmard JA, Duscha BD, Knetzger KJ, Wharton MB, McCartney JS, et al. Effects of the amount and intensity of exercise on plasma lipoproteins. *The New England journal of medicine*. 2002;347(19):1483-92. Epub 2002/11/08.

مقاله اصیل

تاریخ دریافت: ۱۳۹۲/۰۵/۱۰

تاریخ پذیرش: ۱۳۹۲/۰۸/۱۵

تازه‌های علوم کاربردی ورزش

دوره اول، شماره سوم

صص ۴۷-۵۶، پاییز ۱۳۹۲

تأثیر تمرین هرمی فزآینده بر سطوح نیمرخ لیپیدی و فیبرینوژن پلاسما، و ویسکوزیته خون در مردان جوان تمرین نکرده

عباس قنبری نیای،^۱ مصطفی بهزاد خامسلو^{*}،^۳ اسید مرتضی طیبی

۱. استاد گروه فیزیولوژی ورزشی، دانشکده تربیت‌بدنی و علوم ورزشی، دانشگاه مازندران، بابلسر، ایران.
۲. کارشناس ارشد فیزیولوژی ورزشی، آموزشکده فنی حرفه‌ای سماء، دانشگاه آزاد اسلامی، واحد ساری، ساری، ایران.
۳. استادیار گروه فیزیولوژی ورزشی، دانشکده تربیت‌بدنی و علوم ورزشی، دانشگاه علامه طباطبائی، تهران، ایران.

چکیده

هدف پژوهش حاضر بررسی اثر تمرین فزآینده (هرمی) کوتاه مدت بر سطوح نیمرخ لیپیدی و فیبرینوژن پلاسما، و ویسکوزیته خون بود. تعداد ۱۹ مرد جوان غیرورزشکار سالم به طور تصادفی به دو گروه تجربی (۱۰ نفر) و کنترل (۹ نفر) تقسیم شدند. گروه تمرینی به مدت ۴ هفته به یک برنامه تمرینی فزآینده (هرمی) با شدت‌های ۱۰۰٪- ۲۵٪ حداکثر ضربان قلب خودشان، ۴۲ دقیقه در هر جلسه، ۳ بار در هفته پرداختند. تفاوت معناداری در پارامترهای ترکیب‌بدنی بین گروه تمرینی و کنترل مشاهده نشد. سطوح کلسترل کل (TC) و LDL-C و ویسکوزیته خون به طور معناداری در گروه تمرینی در مقایسه با گروه کنترل کاهش یافت. تغییرات غلظت‌های HDL-C، تری‌گلیسیرید (TG) و فیبرینوژن دو گروه معنادار نبود. نتایج نشان می‌دهد که برنامه تمرین فزآینده (هرمی) کوتاه مدت می‌تواند برخی از ریسک فاکتورهای قلبی- عروقی را احتمالاً از طریق کاهش در ویسکوزیته خون تغییر دهد.

واژگان کلیدی: تمرین فزآینده هرمی، فیبرینوژن، ویسکوزیته خون، نیمرخ لیپیدی.

* - نویسنده مسئول:

مصطفی بهزاد خامسلو

پست الکترونیک: mostafabehzadkh@yahoo.com