An Independent, Open Access, Peer Reviewed, Non-Profit Journal
International Journal of Life Sciences
ISSN 2091-0525
website: http://nepjol.info/index.php/IJLS/index
Founded 2007
International Journal of Life Sciences

Research Article

The Effects of a Three-Month College Football Practice on Apo-proteins A and B of Inactive Young Men

Karim Salehzadeh[1]

[1]Assistant Professor, Dept. of Physical Education, Faculty of Educational Sciences, Azarbaijan Shahid Madani University, Tabriz, Iran.

Article Information

Key words:
College football;
Apo protein A;
Apo protein B;
Young men; Inactive

ABSTRACT

This modern age is experiencing increased cardiovascular diseases and higher mortality rates due to inert lifestyles and bad nutrition habits along with stress. Apo lipoproteins A and B are among the most important prognosticating factors of cardiac diseases. Therefore, the present study attempts to study the effects of a three-month college football practice on changes in Apo proteins A and B in inactive young men. Through a public call, 30 subjects were selected randomly from male college students and were divided into two control and experimental groups of 15. The experimental group participated in 3 sessions of exercising per week and the control group had not practice at all. 24 hours before the first and 24 hours after the last exercise session, 10 cc blood samples were taken from both groups after 12 hours of fasting from the left arm vessel. These samples were centrifuged to separate the serum and kept at -70° c to be analyzed at a laboratory. In order to compare Apo proteins A and B of the posttest in both groups, covariance analysis was utilized. Results revealed that physical activities in the form of three-month college football practice change Apo protein A and B levels in blood serum in pre and posttests of experimental group significantly. Yet, the study failed to find significant differences in the level of Apo protein A and B of control and experimental groups.

INTRODUCTION

Lipoproteins are formed by particles called lipids and proteins including free cholesterol, esterified cholesterol, triglyceride and phospholipids along with quadruple lipids (Song et al., 2012). The proteins of lipoprotein structure are called Apo lipoprotein and Apo protein (Niklas et al., 2009). Sixty percent of HDL lipoproteins with high density are generally made up of Apo proteins; however, the percentage falls to modest 1% in chylomicrons (Holme et al., 2007). One or more types of Apo lipoprotein exists in each lipoprotein. Apo lipoprotein B is built in liver and is discharged into plasma as a part of VLDL. It acts in transferring cholesterol to tissues and is a positive risk factor for coronary heart diseases. High density lipoproteins are formed in liver and a high ratio of them consists of Apo lipoprotein A_{-I} and A_{-II} (Ghanbari Niaki, 2013). Apo lipoprotein A_{-I} is the main protein in HDL particles. It is also responsible for transferring the extra cholesterol from tissues and the primary lipoproteins inside intestines. Like HDL, Apo lipoprotein A_{-I} is a negative risk factor for heart attack and coronary diseases (Paoli et al., 2013; Tsekouras et al., 2008). In HDL, the main Apo protein is distinguished with A and in LDL the major Apo protein is shown with B (Segrest et al., 2013). An important prognosticating factor for cardiovascular diseases is the ratio of Apo lipoprotein B to A so that it must be less than 0.5 (Mercedes, 2009). One of the important chemical tasks played by Apo proteins is their role as Enzyme co-factors. For instance, Apo protein A is the enzyme co-factor of lecithin cholesterol acyl transferase (Mestek, 2009; Parish et al., 2009) and plays the role of lipid carrying protein (like Apo protein D as a carrier) HDL. Finally, it plays the role of ligand in linking lipoproteins with receptor molecules on cellule membrane of different tissues like Apo protein B_{100} and E for LDL cell receptors and Apo protein A_1 for HDL cell receptors (Annie et al., 2008; Durheim et al., 2009). This modern age is experiencing increased cardiovascular diseases and higher mortality rates due to inert lifestyles and bad nutrition habits along with stress (Haram et al., 2009; Mestek et al., 2009). Hyperlipidemia is among the

Corresponding author Email address: salehzadeh@azaruniv.edu

DOI:dx.doi.org/10.3126/ijls.v10i1.14506

major reasons for people suffering cardiovascular diseases (Song et al., 2012). Several studies have revealed that the main reason for this is the increase in Apo protein B and Apo protein A of blood in these people (Parish et al., 2009; Yourka et al., 2008). Therefore, amount of Apo protein A as an anti-risk factor and the level of Apo protein B as a risk factor in athletes are more significant compared to non-athletes and it is the opposite in case of Apo protein B (Durheim et al., 2008; Kooze Chian et al., 2014). Researchers have reported that high intensity aerobic exercises for 20 minutes with strength training for two periods has a decreasing effect on lipid profiles (Azarbaijani and Abedi, 2012).

Several scholars believe that the level of Apo protein B in blood plasma of men and women decreases significantly after a course of endurance training (Niklas et al., 2009; Arthur, 2009; Amy et al., 2014). In another study on the levels of HDL lipoprotein and Apo proteins A and B in elderly women doing a course of long-term aerobic exercises, it was revealed that blood HDL and Apo protein B decreases significantly, yet; the leve of Apo protein A did not change significantly (Kim and Jung, 2014). Besides, Parish et al., (2009); Ghorbanian et al., (2013) investigated athlete and non-athlete individuals and demonstrated that levels of HDL and Apo protein A in athletes were more than non-athletes. In this regard, Arazi et al., (2012) studied the response of blood factors to a course of resistance exercises with various levels of severity in male students revealed that the amount of HDL and Apo protein A in both athlete groups increased and the level of LDL and Apo protein B in athletes decreased.

Another study on active and inactive students revealed that active students experienced a significant decrease in Apo protein B, LDL, total cholesterol, and serum resting heart beat and BMI of body composition in obese girls after physical activities (Habibzadeh and Rahmani Nia, 2011). However, Amy et al., (2008) revealed that above mentioned variables do not change significantly in men and women after long term aerobic physical activities. As it can be seen, the results are very contradictory. For instance, Haram et al., (2009) reached absolutely different results. Sheikh al-Islami Vatani (2011), in another study, demonstrated that endurance training without losing weight increased HDL by 10 to 20% and Apo protein by 9 to 36%. On the other hand, triglyceride decreased by 7%.

Moreover, Fontana et al., (2007) investigated the effects of aerobic exercises on cardiovascular risk factors and found no significant change in Apo proteins A and B. Accordingly, Segrest et al., (2013) failed to find any significant effect for aerobic and endurance training on Apo proteins A and B. in spite of all these studies and the contradictory results they have obtained and lack of a proper training plan, there is still the need for further studies and numerous ambiguities are yet to be clarified. Apart from their gender, numberless students spend at least four years in universities and decent physical education classes along with proper training programs will guarantee a healthy lifestyle away from cardio-vascular and pulmonary diseases. Thus, the present study aims to find an answer to the question that if a college training program affects the level of Apo proteins A and B.

METHODOLOGY

The present quasi-experimental study was conducted with two control and experimental groups of 15 subjects. The statistical population of the study included all 855 male students aged 23± 2 entering the university in September 2014. The statistical population was called on and 52 students opted to participate in the study. In order to keep the homogeneity 30 students were selected as the statistical sample via physical activity questionnaire and were put into two groups of control and experimental with 15 subjects in each.

Training method

Training program in this study included a three-month college football training that was conducted during the first semester with three sessions of 90 minutes in a week. Each session the severity and the length of the exercise was fixed (Table 1). The sessions were held between 4 and 6 pm in the indoor stadium of Shahid Madani University of Azerbaijan.

In order to find the intensity of running in experimental group, maximum heart rate and heart rate were calculated through Adams formula (2012) to be 190 to 195 beat per minute.

Age – 220= HR_{Max}
% HRR = HR_{Max} - HR
% THR= HR+ 0.70 (HR_{Max} - HR)

Table 1. One session of the three month college football training with three session a week during one semester

Type	Stretching	Aerobic	Warming up joints	skills	Game of football	recovery	Whole session
Length	10	15	10	10	40	5	90
Intensity		0.70					

Taking blood samples from subjects

48 hours before blood sampling, necessary advice was given to the subjects including fasting, not taking any medicine and not doing any physical activity before sampling. 10 cc of blood sample was taken from subjects 24 hours before the first training session and 24 hours after the final session from left arm vessel of all subjects in both groups using sterilized tubes containing anti-clotting EDTA chemical. The subjects were fast for 17 hours. The temperature in blood sampling site was 23-25° c. and after sampling the samples were centrifuged by experts and taken to Pasteur Laboratory in a subzero temperature.

Gauging device

Gauging cholesterol, triglyceride, and HDL, LDL and VLDL variables was conducted using CoBAS miRAS Auto analyzer with photometry method made by Rosche Company in Switzerland and via pathology kits of Pasteur Laboratory. Randox kit with spectrophotometer method used by RA 1000 auto analyzer made in England to measure Apo A1 and Apo B.

Statistical method

Descriptive statistics were used to analyze data for mean and standard deviation. Due to the difference in Apo proteins of the subjects in their pre and posttests along with comparing Apo proteins A and B in the post test and Apo proteins A and B in both control and experimental groups, covariance analysis was used. Apo protein levels of the pretest for each individual were taken as covariate to correct the mean of the groups and increase its precision through decrease error probability.

The statistical model used is as follows:

$Y_{ij}=B_l+B_iX_{ij}+T_i+E_{ij}$ I= l, ..., a J= l, ..., n Y_{ij} observed= j in group i X_{ij} = Apo protein variable in pretest μ_x (covariate with variable)

E_{ij} = random error T_i = fixed effect of group I (post test) (pretest) B_0= intercept

B_l= regression coefficient and total mean equals: $\mu = B_0 + B_{l\mu x}$ and the mean of the group is $\mu = B_0 + B_1\mu_x + T_i$ where μx is the mean of x covariate.

In order to test the difference in Apo protein in pretest and post test pendent samples statistical method was applied for two samples before and after the experiment were dependent to each other i.e. subjects gauged in pretest were the same people gauged in post test. The difference in Apo protein changes in control and experimental groups were tested through a multiple regression model where the grouped variable was taken as a binominal variable with 0 and 1 assigned for control and experimental groups respectively. The multiple regression model used was as follows:

$$Y_i = B_0 + B_1X_{1i} + B_2X_{2i} + B_3X_{1i}X_{2i} + E_i$$

In this regard, Apo proteins A and B are given to B_3, B_2, B_1 and B_0 mg/dl regression parameters and Apo proteins in pretest X_{ii}. The number defined for groups was X_{2i}, for control group it was 1 and for the experimental group it was 0. The mutual effect of Apo proteins on group pretest was X_{2i} and X_{li} and for the random error, it was E_i. Calculated regression for experimental group was as follows:

$$E(y_i) = (B_0 + B_2) + (B_1 + B_3)X_{1i}$$

And for the control group, it was:

$$E(y_i) = B_0 + B_1X_{1i}$$

RESULTS

Descriptive data from tests are presented in table 2. Yet, results found in other tables are demonstrating Apo protein A and B of the subjects in control and experimental groups.

Table 2. Mean of minimum squares of Apo protein a in control and experimental groups with standard error and level of probability

Groups	Sd	Apo protein A Lsmeans (mg/dl)	sig
Control	1.1185	119.783	0.0001
Experimental	1.1185	127.951	

Table 3. Mean of minimum squares of Apo protein B in control and experimental groups with standard error and level of probability

Groups	Sd	Apo protein B Lsmeans (mg/dl)	sig
Control	1.0256	108.438	0.0006
Experimental	1.0256	102.362	

The difference between control and experimental groups based on P= 0.0006 in the level of Apo protein B is significant.

Table 4. Mean of minimum squares of Apo proteins A and B in pretest of control and experimental group with standard error and level of probability

Variable	Mean (mg/dl)	Sd	t	sig
Apo $_{A1}$- Apo $_{A0}$	6.0666	1.9284	3.15	0.0071
Apo $_{B1}$- Apo $_{B0}$	-4.8000	1.1514	-4.17	0.0009

Table 5. Mean, errors, T levels and level of probability of Apo proteins A and B difference in pretest and posttests of experimental group

Variable	Mean (mg/dl)	Sd	t	sig
Apo $_{A1}$- Apo $_{A0}$	-2.0666	0.9878	-2.09	0.0551
Apo $_{B1}$- Apo $_{B0}$	-2.6000	0.7091	3.67	0.0025

Table 6. Comparing the ratio of Apo proteins B/A in control and experimental groups

Subjects	Test step	B/A	B/A	Sig/d
Control	Pretest	95 %		
Control	Posttest	86 %	9.3% decrease	
Experi	Pretest	79 %		
Experi	Posttest	83 %	3.4 % increase	12.7 %

Results related to Apo protein A:
Level of probability for B_1, B_2 and B_3 was significant (P<0.05) the regression slope for both groups is not similar. The estimated regression for Apo A in control group is:

Apo $_A$= 16.153+ 0.850413 (Apo$_{A0}$)
For experimental group it is:
Apo $_A$= 52.7027+ 0.61753 (Apo$_{A0}$)

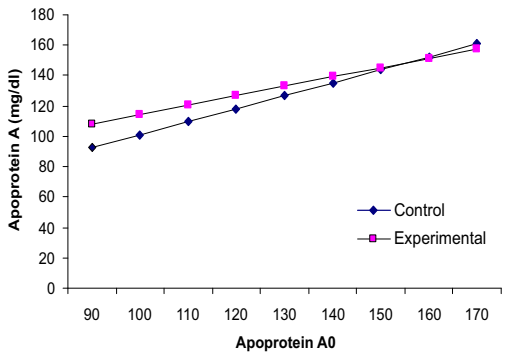

Figure 1. Apo protein A changes in both experimental groups depending on the level of Apo protein in pretests

Results for the Apo protein B:
Level of probability for B_1, B_2 is significant when P<005; yet in case of B_3 it is not significant. Regression slope for groups is different and the regression estimated FOR Apo protein B in control group is:

Apo $_B$= 11.20778+ 0.9112599 (Apo $_{B0}$)
For experimental group it is:
Apo $_B$= 2.9028+ 983645 (Apo $_{B0}$)

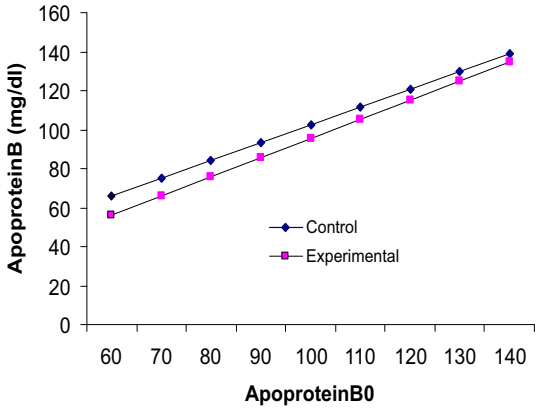

Figure 2. Apoprotein B changes in both experimental groups depending on the level of Apo protein in pretests

DISCUSSION

Several studies have used the ratio of Apoprotein B to A as an important cardiovascular risk factor (Konstantinos et al., 2009; Parish *et al.*, 2009). Researchers use different methods to change this ratio the most important of which is physical activities. However, findings on this issue are contradictory in many cases for they study different sports. the present study showed that a three-month general physical education two in college affects the level of Apo A of blood serum significantly (Table 2). The finding concords with findings of Greene *et al.*, (2009) on the effects of a 24-week pedaling on Ergometer with 50 and 80% intensities of Vo_{2max} on the changes in ratio of Apoproteins B/A. Several other studies finding similar results show that regular physical activities increase levels of HDL and decrease VLDL and LDL levels. This increase in HDL prevents cholesterol depositing in blood vessels (Segrest, 2013; Haram *et al.*, 2009; Jenkins et al., 2010 and Michelle, 2009). Increasing HDL increases the level of Apo A as its major component (Ghorbanian et al., 2013; Song et al., 2012). Moreover, studies approve of the positive effects of physical activities and believe that physical activities significantly increase Apoprotein A and decrease Apoprotein B levels (Arazi, 2012). Furthermore, circular endurance and resistance exercises may have desirable effects on lipid profile, hematology and resting heart rate and offer a proper non-medicinal solution for deterring cardiovascular diseases and disorders induced by obesity in adolescent obese boys.

Several other studies have disproved of this idea (Ghanbari Niaki, 2014 and Poorveghar, 2013). They demonstrated that participating in activities with low intensities and short lengths will not increase Apoprotein A in non-athletes significantly (P=0.01). moreover, the difference in the levels of Apoprotein A in control and experimental groups was not significant (P=0.01), furthermore, some studies have revealed that intense and short activities in young inactive men does not affect weight and body composition and decrease VLDL density,

fasting triglycerides in plasma through preventing VLDL secretion in the liver and triglyceride for 48 hours (Terrados et al., 2010). In this regard, it seems LDL triglycerides are catabolized through various processes after Lipase Lipoproteins are activated by Apo protein A. therefore, increase in Apo proteins after physical activities expand catabolism of LDLD and VLDL triglycerides (Azarbaijani & Abedi, 2012 and Habib Zadeh & Rahmani Nia, 2011).

The present study, on the other hand, found a significant difference in the level of Apoproteins B of control and experimental groups in their pre and posttests (P=0.01). This difference in pre and posttests of the control group may be induced by the 3-month length of the training course (table 3). Various factors may affect the result in this length of time including other physical activities, nutrition and some hereditary factors. As it can be seen, trainings increased Apoprotein A slightly in the experimental group subjects in their posttest compared to the pretest (table 4). Despite the fact that increase in pre and posttests is +6.06666, control group experienced a +2.60000 decrease and this difference in the means of subjects in control and experimental groups have caused T= 3.15 and P= 0.0071. This may show that the training have affected the increase in Apoprotein A in the experimental group sufficiently and significantly (Table 4). This findings concord with findings of Mogharnasi et al., (2014); Poorveghar et al., (2014); Sheikh al-Islami et al., (2011); Haram et al., (2009); Parish et al., (2009); Arthur S Leon (2009); Michelle et al., (2009) and Nikolas et al., (2009). They concluded that there is a significant difference between means of serum density of Apo lipoproteins A and B in pretest and after aerobic activities. At the same time, a significant difference was observed between mean density of serum triglyceride of the subjects and mean densities of high density cholesterol (HDL-C) and low density cholesterol (LDL-C) in pre and posttests. Accordingly, findings from this study show that in spite of observing increase in the level of Apoprotein A in experimental group, in case of Apoprotein B, like most of the studies mentioned above, no significant difference is seen in the level of Apoprotein B of the control and experimental groups in pre and posttests (P=0.01). Yet, the level of Apoproteins B in this group have decreased by -4.8000 (table 4). This difference in the level of Apoprotein B may have great effects on heart risks and decrease the probable dangers significantly (Mogharnasi et al., 2014; Sheikh al-Islami, 2011). Data show that these activities significantly affect Apo protein B levels of the subjects under this study (table 4). However, this finding, contradicts with findings of Haram et al., (2009); Fontana et al., (2007) and Amy E et al., (2008). They demonstrated that in spite of the significant difference in the amount of Lipoprotein HDL and Apo protein B of the subjects after long-term training, levels of Apoprotein a does not change significantly. Nonetheless, the ratio of Apo protein B to A is a precise prognosticating factor of damages to heart muscle (Tsekouras et al., 2008; Ghanbari Niaki, 2013). This ratio has increased in subjects of control group by 83%/79%= 0.43 and decreased by 86%/95%= 9.3% in subjects of the experimental group. This ratio was 12.7% in case of A/B ratio of control and experimental groups (table 6). This difference is much higher than its standard level and may pose extreme dangers (Mercedes, 2009 and Ghanbari Niaki, 2013). This shows that despite expectations, these activities cannot be decent activities for decreasing cardiovascular risks (Terrados et al., 2010 and Poorveghar, 2013). Moreover, a significant decrease is observed in the level of Apo protein B (table 6). This finding concords with findings of Ghorbanian et al., (2013); Kim and Jung (2013); Arthur S. Leon (2009) and Matsios et al., (2008). They believe that in order to decrease the level of Apo protein B after long-term physical activities, other factors like, nutrition, number and length of sessions in a week, caloric expenditure, smoking and drugs (which were not under control in the present study) must be considered for caloric expenditure, body fat percentage, heredity and type of training are among the most important factors affecting the level of Apo protein A and B in pre and posttests of studies (Habib Zadeh & Rahmani Nia, 2011 and Song et al, 2012). This finding does not concord with findings from studies by Poorveghar et al., (2014); Azarbaijani & Abedi (2012); Arazi et al., (2012) and Parish et al., (2009). They demonstrated that the difference in mean densities of Apo lipoproteins A and B of serum after intense aerobic exercises. Besides, there is a significant difference in mean density of serum triglyceride of the subjects and mean densities of HDL-C and LDL-C before and after training (P=0.0001). It seems as if these trainings increase secretion of Catecholamine and sympathetic nerves' activity and decrease insulin secretion resulting in increase in growth hormones, cortisol, epinephrine and glucagon. Finally, this provides the ground for freeing fatty acids from fatty tissues (Shahsavar et al., 2011 and Poorveghar, 2014).

CONCLUSION

Overall results reveal that although three months of college football training with three sessions in a week is a regular and classic training program with somewhat low intensity, it increased the level of Apo protein A significantly and this could have positive effects on cardiovascular health. The effect of these trainings on the level of Apo protein B was decreasing and significant. The ratio of Apo B/Apo A gets closer to standard levels after its decrease. In conclusion, both results demonstrate that beneficial changes in biochemical, physiologic and body composition affect cardiovascular condition and decrease the risk of diseases. Change in lifestyle and adding activity to the inactive life could increase calorie expenditure and cardiovascular health condition would improve. In any case, variety of factors affecting Apo proteins A and B and levels of the indices of heart risks,

more precise studies with higher controls over these factors could give better insights to human health.

ACKNOWLEDGMENTS

Hereby the researcher would like to express his gratitude for the collaboration of all the participants in this study.

REFERENCES

Adams Gene M (2012). A Guide to the Laboratory of Sports Physiology. First Ed. Translated by: Farhad Rahmani Nia, Hamid Rajabi, Abbasali Gayini and Hossein Mojtehedi. Asre Entezar Publication, Tehran. 207-218.

Amy E G, Yumei C, Deborah D B, Amy M C, Bruce H, and Penny M, Kris-Etherton (2008). A Macadamia Nut-Rich Diet Reduces Total and LDL-Cholesterol in Mildly Hypercholesterolemic Men and Women. J Nutr 138: 761-767.

Annie M-B, Amélie Ch, Geneviève G, Paul P, Yvan Ch, Simone L, Patrick Ce, and Benoît L (2008). Study of the effect of Trans fatty acids from ruminants on blood lipids and other risk factors for cardiovascular disease. Am J Clin Nutr 87: 593–9.

Arazi H, Salehi A, Hosseini Y, Jahanmahin M (2012). The response of hematological factors to a circuit resistance training program with various intensities in athlete male students. Sci J Iran Blood Trans Org 9(1): 54-62.

Arthur S L (2009). Biological Mechanisms for the Cardio protective Effects of Aerobic Exercise. A J OF LMED 3: 32S. DOI:10.1177.

Azarbayjani MA, Abedi B (2012). Comparison of Aerobic, Resistance and Concurrent Exercise on Lipid Profiles and Adiponectin in Sedentary Men. Knowle & Heal 7(1): 32-8.

Durheim MT, Cris AS, Lori AB, Stephanie KM and William E K (2008). Relationships between exercise-induced reductions in thigh intramuscular adipose tissue, changes in lipoprotein particle size, and visceral adiposity. Am J Physio Endocr Metab 295: E407-E412.

Espinosa-Larranaga F, Margarita V-J, Roberto M-S (2005). The importance of low serum levels of high-density lipoprotein cholesterol (HDL-C) as a cardiovascular risk factor. j Diabe and Vasc Dis Res 2: S1-S8.

Fontana L, Villareal DT, Weiss EP, Racette SB, Steger-May K, Klein S, Holloszy JO (2007). Calorie restriction or exercise: effects on coronary heart disease risk factors. A randomized, controlled trial. Am J Physiol Endoc Metab 293: E197-E202.

Ghanbari-Niaki A (2013). Plasma Apo-M Response to a Circuit Resistance Training Program. An of App Sport Sci 1(4): 1-4.

Ghorbanian B, Ravassi AA, Kordi M R, Hedayati M (2013). The Effects of Rope Training on Lymphocyte ABCA1 Expression, Plasma Apo_{A1} and HDL_c in Boy Adolescents. Int J Endocr Metab 11(2): 76-81.

Greene NP, Lambert BS, Greene ES, Carbuhn AF, Green JS & Crouse SF (2009). Comparative efficacy of water and land treadmill training for overweight or obese adults. Med & Scie in Sports & Exer 41(9): 1808-1815 (PMID:19657288)

Haram PM, Ole JK, Sang JL, Marianne OB, Qusay Y Al-Sh, Helge LW, et al (2009). Aerobic interval training vs. continuous moderate exercise in the metabolic syndrome of rats artificially selected for low aerobic capacity. Cardiovasc Res 81: 723-732.

Habibzadeh N, Rahmaninia F (2011). A Study of Two Months Walking Exercise on Serum Lipoprotein and Body Mass Index in Obese Girls. Medic J of Mashhad U of Medic Sci 54(2): 100-104.

Jenkins NT, Mc Kenzie JA, Damcott CM, Witkowski S, Hag berg JM (2010). Endurance exercise training effects on body fatness, VO_2max, HDL-C subfractions, and glucose tolerance are influenced by a PLIN haplotype in older Caucasians. J Appl Physiol 1: 0:01018.doi:10.1152.

Kim DY and Jung SY (2014). Effect of Aerobic Exercise on Risk Factors of Cardiovascular Disease and the Apo lipoprotein B/Apo lipoprotein A-1 Ratio in Obese Woman. J Phys Ther Sci 26(11): doi:10.1589/jpts.26.18251825–1829.

Konstantinos Tambalis DB, Panagiotakos DB, Kavouras SA, Sidossis LS (2009). Responses of Blood Lipids to Aerobic, Resistance, and Combined Aerobic With Resistance Exercise Training: A Systematic Review of Current Evidence. Angiology 60(5): 614-632.

Koozehchian Majid S, Nazem F, Kreider RB, Roberts WJ, Best Thomas M, Yi R and et al (2014). The role of exercise training on lipoprotein profiles in adolescent males. Lip Heal Dis 13: 95. doi:10.1186/1476-511X-13-95

Holme L, Hostmark A. T & Anderssen S. A (2007). Apo_B but not LDL-cholesterol is reduced by exercise training in overweight healthy men. Results from the 1-year randomized Oslo Diet and Exercise Study. J of Int Med 262(2): 235–243.

Mercedes RC (2009). Physical Activity and Cardiovascular Disease: How Much Is Enough? A J OF L Med. 3(1): 44S-49S.

Mestek ML (2009). Physical Activity, Blood Lipids, and Lipoproteins. AM JO OF LIF MED 3: 279-283.

Michael L (2009). Mestek Physical Activity, Blood Lipids, and Lipoproteins .American Journal of Lifestyle Medicine 3(4): 279-283.

Metsios GS, Metsios GS, Stavropoulos-Kalinoglou A, Veldhuijzen van Zanten JJ.CS, Treharne GJ, Panoulas VF and et al (2008). Rheumatoid arthritis, cardiovascular disease and physical exercise: a systematic review. Rheumatology (Oxford) 47: 239-248.

Mogharnasi M, Eslami R, Behnam B (2014). Effects of Endurance and Circuit Resistance Trainings on Lipid Profile, Heart Rate, and Hematological Parameters in Obese Male Students. An of AP Sport Sci 2(4): 11-22

Nicklas BJ, Wang X, You T, Lyles MF, Demons J, Easter L and et al (2009). Effect of exercise intensity on abdominal fat loss during calorie restriction in overweight and obese postmenopausal women: a randomized, controlled trial. Am J Clin Nutr 89: 1043-1052.

Paoli A, Quirico FP, Tatiana M, Giuseppe M, Marco N, Giuseppe Band et al (2013). Effects of high-intensity circuit training, low-intensity circuit training and endurance training on blood pressure and lipoproteins in middle-aged overweight men. Lipi Heal Dis 12: 131. doi:10.1186/1476-511X-12-131

Parish S, Peto R, Palmer A, Clarke R, Lewington S, Offer Aand et al (2009). The joint effects of apolipoprotein B, apolipoprotein A1, LDL cholesterol, and HDL cholesterol on risk: 3510 cases of acute myocardial infarction and 9805 controls. Eur Heart J 30(17): 2137-46.

Pourvaghar MJ, Shahsavar AR, Bahram ME (2015). The effect of a single bout of severe aerobic exercise on Apo lipoproteins A, B and some serum lipid profiles. Feyz, J of Kashan U of Medl Sci 18(6): 585-591.

Segrest JP, Cheung MC, Jones MK (2013). Volumetric determination of Apo lipoprotein stoichiometry of circulating HDL subspecies. J of lipid resea. 54(10): 2733-44.

Shahsavar AR. Pourvaghar MJ (2011). Follow-up Response of Epinephrine and nor Epinephrine to an Exhausting Physical Activity. Int Confe on Sport Med and Sport Scie June, Paris, France.

Sheikh ol-eslami Vatani D, Ahmadi S, Mojtahedi H, Marandi M, Ahmadi Deharshid K, Faraji H, et al (2011). Effect of moderate and high intensity resistant exercises on cardiovascular risk factors in none athlete university students. Kowsar Med J 16(2): 115-21.

Song F, Poljak A, Crawford J, Kochan NA, Wen W, Cameron B, et al (2012). Plasma Apo lipoprotein levels are associated with cognitive status and decline in a community cohort of older individuals. Plo S one 7(6): e34078.

Stefan B, Sjödin A, Forslund A, Hambraeus L, Holmbäck Ulf (2006). Minor changes in blood lipids after 6 weeks of high-volume low- intensity physical activity with strict energy balance control. Eur J Appl Physiol 96: 315–321.

Terrados N, Valcárcel G, Venta R (2010). New Cardiovascular Risk Factors and Physical Activity. Apunts Med E Sport 45(167): 201-8.

Tsekouras YE, Magkos F, Kellas Y, Basioukas KN, Kavouras SA, Sidossis LS (2008). High-intensity interval aerobic training reduces hepatic very low-density lipoprotein-triglyceride secretion rate in men. Am J Physiol Endocr Metab 295(4): E851-8.

Yourka DT, Christina K, Maksym VK, Susanne BV, Eliana W and Michael DJ (2008).

Subcutaneous adipocyte size and body fat Distribution. Am J Clin Nutr 87: 56-63.