

The Effects Of Long Term Physical Activity On The Changes In The Rates Of In Apo Proteins A And B In Nonathlete

Karim Salehzadeh¹, Yousef aghdami¹, Morteza Jourkesh²

¹Department of Physical Education, Azerbaijan University of Tarbiat Moallem, Tabriz, Iran; ²Department of Physical Education and Sport Science, Shabestar branch, Islamic Azad University, Shabestar, Iran

Abstract: The Present study aims to evaluate the effects of a one-year-long volleyball practice on the changes in the rates of Apo proteins A and B in the blood serum of non-athlete men. In order to do so, 30 subjects were selected randomly from among non-athlete male students and then were divided into two control and experimental groups. The experimental group on average aged 23 ± 2 . Their average height was 172.2 ± 3 cm and the average weight was 69.6 ± 3.1 kg. On the other hand, the control group aged on average 22 ± 2 and their average height and weight were 170.3 ± 3.8 cm and 69.3 ± 2.7 kg. The experimental group went through a one-year-long volleyball exercise program in which they had to practice for 90 minutes three times in a week. The control group did not have any special practice. The covariance analysis was used to probe the rates of Apo A and B and analyze the data. The rates of Apo proteins, measured before the test in both groups, were taken as the covariate to correct the groups' mean, increase the test's precision and lessen the error risk. The test results revealed that in the experimental group a one-year-long sport exercises has meaningfully changed the level of Apo A in the blood ($P= 0.01$). There was not a significant difference in the rates of Apo A in the posttest measurements in both groups ($P= 0.01$). The amount of Apo B was also meaningfully different in pre and posttest in the experimental groups but the changes in the rates of Apo B in both control and experimental groups did not differ meaningfully. [Karim Salehzadeh, Yousef aghdam, Morteza Jourkesh. **The Effects Of Long Term Physical Activity On The Changes In The Rates Of In Apo Proteins A And B In Nonathlete.** Journal of American Science 2011; 7(6):654-662].(ISSN: 1545-1003). <http://www.americanscience.org>.

Keywords: Long-Term Physical Activity; Apo Protein A; Apo Protein B; Non-Athlete.

1. Introduction

The proteins in the body of lipoproteins are called Apo lipoproteins or Apo proteins (Espinosa-Larranaga, F, et al., 2005). Lipoproteins are composed of so-called lipid proteins which are constructed by free cholesterol, Strifiet cholesterol, triglycerides, phospholipids and quartet lipids (Ernst J. Schaefer, 2002). Although Apo proteins take the minimal amount of 1% of Shilomicrones, they make nearly 60% of the high density HDLs. One or more types of Apo lipoproteins exist in each lipoprotein (L. Holme, et al., 2007). The major Apo protein in the HDL is called A and the major one in the LDL is called B (Martin, R et al., 2002). One of the important prognostic factors in cardiac diseases is the Apo proteins A and B ratio. This ratio must not fall below 0.5 (Mercedes R. 2009). Apo protein A for example acts as the cofactor for the lisitin-cholesterol asile transferaze enzyme (Mestek, M. L. 2009 and Parish S, et al., 2009). It also takes the role of the lipid carrier proteins like Apo protein D in carrying HDL and finally acts as ligands connecting lipoproteins to receiving molecules on the cells in different tissues such as Apo protein B100 and E for LDL receivers and Apo protein A1 for HDL cellular receivers (Durheim, M. T, et al., 2008).

Nowadays cardiovascular diseases are killing many people for they have inactive lifestyles and bad

nutritional habits (Haram, P. et al., 2009). Different studies have revealed that the decrease in Apo protein A and increase in Apo protein B rate is the main cause of cardiovascular diseases (Parish, S et al., 2009). Thus, the rate of Apo protein A is an anti risk factor and its comparison to Apo protein B is a risk factor. This risk is larger in non athletes but about Apo protein B it is the opposite (Kodama, S et al., 2007 and Durheim, M. T et al., 2008). Nicklas, B. et al., (2009); Arthur S. (2009); Amy E. (2009) and other scholars' findings show that Apo protein B in the men and women's blood serum decreased meaningfully after a period of stamina practices.

Miller et al. (2006) and Espinosa et al. (2005) studied the amounts of HDL lipoprotein and Apo proteins A and B after long aerobic exercises. They found that in elder women, the HDL and Apo protein B decreased significantly in the subjects' blood however the Apo protein A did not have a big change. Parish et al. (2009), Green J. et al., (2005) and Kodama (2007) investigated athletes and non-athletes and revealed that in spite of other studies' findings, the rate of HDL and Apo protein A in athletes' body was larger than non athletes. Tokmakidis (2003) studied the effect of aerobic and stamina exercises on the rate of Apo protein A and HDL and concluded that their rate increases in both groups (William E. et al., 2003). In

regressions for the experimental group which is $E(y_i) = (B_0 + B_2) + (B_1 + B_3)X_{1i}$ and is $E(y_i) = B_0 + B_1X_{1i}$ for the control group.

Table 1. Volleyball exercises program of a session, three times a week for one year.

Cholesterol, triglyceride, and LDL-HDL-VLDL were measured by an auto analyzer set (Co BAS miRAS) using photometric method. This set was made by Roche in Switzerland. The measurement was done by diagnostic laboratory gates of Pasteur Laboratory. Spectrophotometric method with Randox was used to identify Apo proteins A and B by an auto analyzer set (RA1000) made in England.

exercise	stretches and flexibility	aerobic running	muscles warm-up	skills' practices	game of volleyball	recovery	the whole session
length of session (minutes)	8	12	7	18	40	5	90
severity of exercise (HR max)		70%					

3. Results

The descriptive data for the subjects are presented in table 2. on the other hand other tables show the relationship between Apo proteins A and B in the subjects of both experimental and control groups.

Table 2. least square means of Apo protein A in control and experimental groups accompanied by standard error and P value.

Group	Apo protein A Lsmeans(mg/dl)	Standard Error	P-Value
Control	119.783	1.1185	<0.0001
Experimental	127.951	1.1185	

Table 3. least square means of Apo protein B in control and experimental groups accompanied by standard error and P value.

Group	Apo protein B Lsmeans(mg/dl)	Standard Error	P-Value
Control	108.438	1.0256	0.0006
Experimental	102.362	1.0256	

The difference between two groups in the amount of Apo protein B based on P=0.0006 is meaningful.

Table 4. Means, standard error, t value and P-value of differences of pre and post trial values of Apo proteins in experimental group.

Variable	Mean(mg/dl)	Standard Error	T Value	P-Value
(Apo protein A 1- Apo protein A0)	6.06666	1.9284	3.15	0.0071
(Apo protein B 1- Apo protein B0)	-4.80000	1.1514	-4.17	0.0009

Table 5. Means, standard error, t value and P-value of differences of pre and post trial values of Apo proteins in control group.

Variable	Mean(mg/dl)	Standard Error	T Value	P-Value
(Apo protein A 1- Apo protein A0)	-2.06666	0.98786	-2.09	0.0551
(Apo protein B 1- Apo protein B0)	-2.60000	0.70912	3.67	0.0025

Apo proteins A and B amounts in pretest and posttest of experimental group.

Table 6. comparison of $\frac{B}{A}$ ratio in both control and experimental groups.

Subjects	test phases	Apo B/A ratio	Changes in Apo B/A ratio	meaningful difference
experimental group	pretest	95%		
experimental group	posttest	86%	9.3% decrease	
control group	pretest	79%	3.4% increase	12.7% is not meaningful
control group	posttest	83%		

Apo protein A

The P value for B₁, B₂ and B₃ were meaningful (P<0.05) and the regression slope was not similar in two groups.

The estimated regression for Apo protein A in control group was:

$$\text{Apo A} = 16.153 + 0.850413(\text{Apo A}_0)$$

The estimated regression for the experimental group is as follows:

$$\text{Apo A} = 52.7027 + 0.61753(\text{Apo A}_0)$$

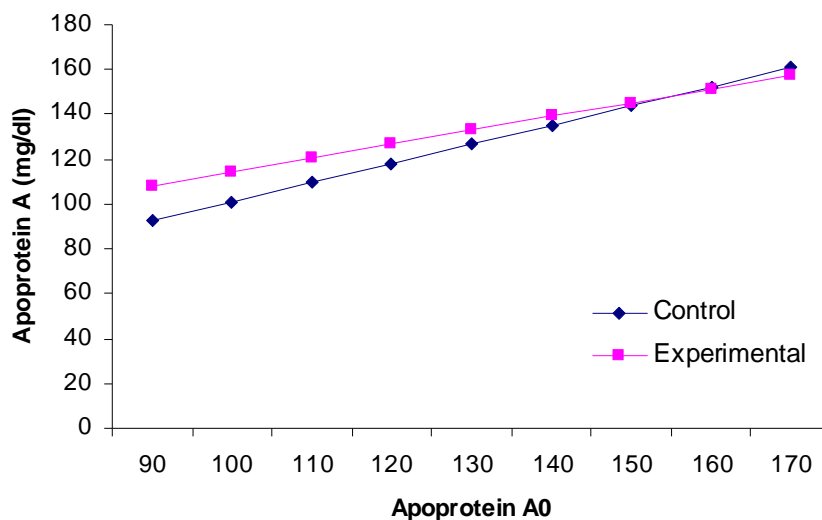


Figure 1. The changes in Apo protein A in both experimental groups dependent on initial Apo protein measurements in two pretests.

Apo protein B

The P value for B₁ and B₂ were meaningful in P<0.05, but for the B₃ that was not meaningful. The regression slope for different groups were different and the estimated regression on Apo protein B in the control group is as follows:

$$\text{Apo B} = 11.20778 + 0.9112599(\text{Apo B}_0)$$

The estimated regression in the experimental group is as follows:

$$\text{Apo B} = 2.9028 + 0.983645 (\text{Apo B}_0)$$

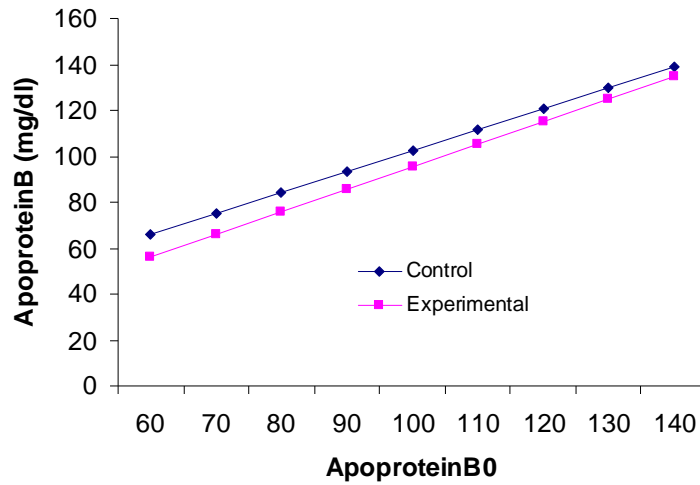


Figure 2. Apo protein B changes in both control and experimental groups dependent on initial Apo protein measurements in the pretest

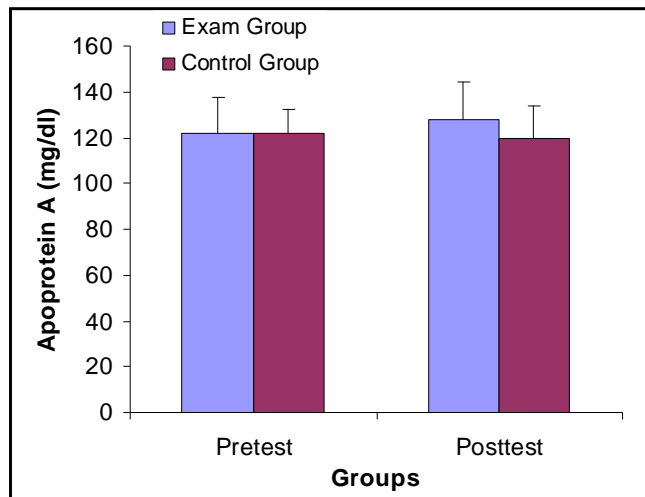


Figure 3. Comparison of Apo protein A changes in both control and experimental groups

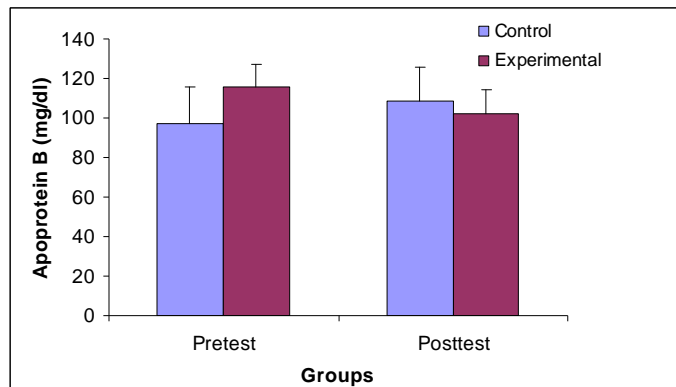


Figure 4. Comparison of Apoprotein B changes in both control and experimental groups

4. Discussion

Different studies in modern countries have revealed that Apoproteins A and B ratio is an important factor in estimating the risk of cardiovascular diseases (Miller G. et al., 2006; Konstantinos et al., 2009). Several methods have been used to change this ratio and the most important one is physical activities. However different studies have reached different conclusions. Each of these studies have focused on a specific sport. Green et al., (2005) studied 24 weeks of cycling with 50 and 80 percent of Vo_{2max} on the ratio of

Apoproteins $\frac{B}{A}$ and reached similar results (Green J. et al., 2005).

Despite the results of the above-mentioned study and other similar ones demonstrate that regular physical activities increase the HDL and decrease LDL and VLDL. the increase in HDL hinders cholesterol sedimentation in blood vessels (O'Donovan, G and et al., 2005; Jacobs et al., 2006; Durham et al., 2008; Haram et al., 2009; Jenkins et al., 2009; Konstantinos et al. 2009; Michael L et al., 2009). As the HDL raises, Apoprotein A being a major part of it raises as well (Press et al., 2003; Sharma, A et al., 2003; O'Donovan, G et al., 2005; Michael L. Mestek., 2009; Parish S et al., 2009) LDL triglycerides catabolize as the lipoprotein lipase enzymes begin activity through Apoprotein A on the blood vessels. Accordingly, as the Apoproteins increase by physical activities, catabolism of LDL and VLDL triglycerides grows (Tall, A, 2002; Sharma, A et al., 2003; Von Stengel, Simon 2004; Stefan Branth et al., 2006; Parish S et al., 2009). Most of the studies conducted approve the positive role of physical activities and show that they affect the increase in Apoprotein A and decrease in Apoprotein B meaningfully. In spite of these findings, some other studies have rejected them (Green J. et al. 2005; Espinosa-Larranaga F. et al. 2005; Miller G. et al. 2006; Fontana L. et al. 2007; Kodama S. and et al. 2007; Parish S. et al. 2009). In order to clarify a bit more on the topic and considering the fact that many people have turned from individual sports to ball games to block the risk of cardiovascular diseases, the present study have studied the effects of a long-term volleyball exercise on the changes in the Apoprotein A and B rates in non-athlete men. the results reveal that these activities do not change the Apoprotein A's amount in non-athlete subjects ($P=0.01$). The difference in the control and experimental groups' Apoprotein A is not meaningful ($P=0.01$). on the other hand, comparison of Apoprotein B in both control and experimental group in pretest and posttest shows a meaningful difference ($P=0.01$). This meaningful difference in the pretest and posttest of control group have been caused by the one-year-long volleyball exercises in which many factors

including nutrition, physical activities and heredity are also important. As it can be seen, this exercise may change the Apoprotein rate slightly in the subjects in experimental group in pretest and posttest. Although this slight increase in the amounts measured in the pretest and posttest is equal (+6.06666), in the control group this rate shows a little fall (+2.60000). This difference in the mean of the subjects in both control and experimental groups have made the $T=3.15$ and $P=0.0071$ so it can be inferred that the change in the Apoprotein A rate has been sufficient in the experimental group and it can be defined and thus it is meaningful. the findings of the present study are in complete concordance with the findings of Green (2005); Haram et al. (2009); Jacobs et al. (2006); O'donovan et al. (2005); Parish et al. (2009); Arthur S. Leon (2009); Micheal et al. (2009); Von Stengel (2004); Slentz et al. (2007); Nicholas et al. (2009); Metsios et al. (2008) and disagrees with findings of Espinosa et al. (2005); Fahlman (2002); Haram et al. (2009); Kodama (2007); Fontana et al. (2007); Amy E. et al. (2008) and William et al. (2003). The present findings show that although the rate of Apoprotein has changed in the experimental group significantly, analyzing the results on Apoprotein B present no meaningful differences in both control and experimental groups in the pretest and posttest ($P=0.01$). In this group Apoprotein B has decreased significantly (-4.8000) and this decrease may have huge effects on the cardiovascular diseases' risk factors and reduce them (Fahlman, 2002; William, E et al., 2003; Von Stengel, Simon., 2004; O'Donovan, G. et al., 2005; Stefan Branth et al., 2006; Miller, G. et al., 2006; Fontana, L et al., 2007; L.Holme, A., 2007; Yourka D Tchoukalova et al., 2008). Thus it can be said that these activities had a meaningful effect on the Apoprotein B rate in the subjects under study. Overall it is proved that the Apoprotein B to A ratio is a prognostic factor in cardiac muscles' defects (Green j et al., 2005; Miller. G et al., 2006; Parish S et al., 2009; Jenkins et al 2009). In the subjects of the control group

this ratio $\left\{ \frac{\text{pretest}}{\text{posttest}} = \frac{0/79}{0/83} \right\}$ had a 0.43 raise and in

the subjects of the experimental group $\left\{ \frac{\text{pretest}}{\text{posttest}} = \frac{95}{86} \right\}$ had a 9.3% fall. The difference in the B/A ratio was 12.7%, which was a huge difference, compared to the standard difference, and may be the cause of a big risk. It can be concluded that activities like the ones tested here cannot be supposed as the hindering factors against cardiovascular diseases risk factors (Miller. G et al., 2006; Kodama, S et al., 2007; Amy E. Griel et al., 2008; Parish S et al., 2009). However, a huge fall can be seen for Apo protein B (Table 6), and findings of this research are in concordance with Michael et al.,

(2009); Parish et al., (2009); Arthur S. Leon, (2009); Metsios et al., (2008); Jacobs et al., (2006); Haram P. et al., (2009); and do not concord with the findings of Von, (2004); Green (2005); Parish S. et al., (2009); Miller et al., (2006). It should be noted that beside long-term physical activities, other factors like nutrition, sessions' number in a week, length and severity of exercises, caloric cost, smoking and medicines which were not controlled are of high importance in decreasing the amount of Apo protein B (Lee, I-M et al., 2003; [Eisenmann JC.](#), 2004; Fontana, L et al., 2007; Annie Motard and et al., 2008; Mercedes R. Carnethon, 2009). It is highly important for the caloric cost of individuals, amount of fat in the body, heredity, personal exercising methods are among the factors affecting amount of Apo proteins A, and B in both pretest and posttest levels and influence the research results (Sharma A. et al. 2003; [Tokmakidis SP](#), [Volaklis KA](#), 2003; Metsios, G.et al.,2008; [Parish S](#) et al.,2009; Mestek, M.L. 2009; Michael L. Mestek,2009). Overall conclusion can be drawn from this study that even if a one-year-long physical activity in the form of a three-session volleyball exercise increases Apo protein A rate meaningfully, higher increase may have a huge role in safety from cardiovascular diseases. Besides, these exercises may decrease the amount of Apo protein B meaningfully and bring the Apo protein B to Apo protein A ratio to the standard one. Accordingly, in both cases it can be said that biochemical, physiological, and physical shape is in a way that can be effective on cardiovascular condition and reduce the risk of diseases (Lee, I-M et al., 2003; William, E et al., 2003; Miller G et al., 2006; Slentz, C et al., 2007; Yourka D Tchoukalova et al., 2008; Parish S et al ., 2009; Michael L. Mestek, 2009). Changing lifestyles from an idle and static one to a more dynamic and active one may increase the energy usage (Martin R. et al., 2002; Jenkins et al. 2009; Parish S. et al., 2009; Mercedes R. Carnethon, 2009) and help have a healthier heart. Finally, it should be noted that there are many factors affecting the amount of Apo protein A and B as cardiovascular risk factors, and more studies are needed to clarify more on these factors and identify mechanisms of changes in Apo proteins A and B.

Corresponding Author:

Morteza JOURKESH

Department of Physical Education and Sport Science,
Shabestar branch, Islamic Azad University, Shabestar,
Iran

Telephone 00989143124670

mjourkesh@iaushab.ac.irE-mail

Jourkesh_2006@yahoo.com

References:

1. Amy, E., Griel, Yumei Cao., Deborah D., Bagshaw, Amy M., Cifelli, Bruce Holub and Penny M. Kris-Etherton. (2008). A Macadamia Nut-Rich Diet Reduces Total and LDL-Cholesterol in Mildly Hyper cholesterolemic Men and Women. *J. Nutr* 138, 761-767.
2. Annie Motard-Belanger., Amelie Charest., Genevieve Grenier., Paul Paquin., Yvan Chouinard., Simone Lemieux., Patrick Couture and Benoit Lamarche.(2008) Study of the effect of trans fatty acids from ruminants on blood lipids and other risk factors for cardiovascular disease.Patrick Couture, and Benoit Lamarche. *Am J Clin Nutr* 87, 593-9.
3. Arthur,S. Leon.(2009) Biological Mechanisms for the Cardioprotective Effects of Aerobic Exercise. *A J OF LMED* 3(1), 32-34.
4. Durham, Michael T; Cris, A Slentz; Lori, A Bateman; Stephanie, K Mabe; William, E Kraus.(2008) Relationships between exercise-induced reductions in thigh intermuscular adipose tissue, changes in lipoprotein particle size, and visceral adiposity. *Am. J. Physiol. Endocrinol. Metab* 295, 407- 412.
5. [Eisenmann JC.](#) (2004) Physical activity and cardiovascular disease risk factors in children and adolescents: an overview. *Can J Cardiol* 1;20(3) , 295-301.
6. Ernst J ,Schaefer. (2002) Lipoproteins, nutrition, and heart disease. *Am J Clin Nutr* 75, 191-212.
7. Fahlman Boardly,j Gerontol A boil. (2002) Effects of endurance training and resistance training on plasma lipoprotein profile in elderly women. *Sci med .Sci. Eeb* 57 (2), 85.
8. Francisco Espinosa-Larrañaga. (2005) The importance of low serum levels of high-density lipoprotein cholesterol (HDL-C) as a cardiovascular risk factor. *j Diabetes and Vascular Disease Research* 2, 1-8.
9. Fontana, Luigi, Dennis T, Villareal., Edward P, Weiss., Susan B, Racette., Karen Steger-May., Samuel Klein., John O, Holloszy (2007) Calorie restriction or exercise: effects on coronary heart disease risk factors. A randomized, controlled trial. *Am. J. Physiol. Endocrinol. Metab* 293, 197-202.
10. Green, John S. FACSM., Lowe, Robert C., Pronk, Nico FACSM., Jacobsen, Dennis FACSM., Rohack, James J., Crouse, Stephen F, FACSM. (2005) Low and High intensity Endurance exercise training does not significantly alter the apolipoprotein-B / apolipoprotein-A1 ratio in hyper cholesterolemic men. *Med sci sport Exer* 37(5) , 470.

11. Haram, P.M. Al Share., Bendheim ., Britton., Gilligan., Kemi ., Koch., Lee., Najjar., Waldum., Wisloff. (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.
12. Jacobs Kevin, A., Ronald M, Krauss., Jill A, Fattor., Michael A, Horning., Anne L, Friedlander., Timothy A, Bauer., Todd A, Hagobian., Eugene E, Wolfel and George A, Brooks. (2006) Endurance training has little effect on active muscle free fatty acid, lipoprotein cholesterol, or triglyceride net balances. *Am. J. Physiol. Endocrinol. Metab* 291, 656-665.
13. Jenkins, Nathan T., Jennifer A, McKenzie., Coleen M, Damcott., Sarah, Witkowski., and James M, Hagberg .(2009) Endurance exercise training effects on body fatness, VO₂max, HDL-C subfractions, and glucose tolerance are influenced by a PLIN haplotype in older Caucasians. *J Appl Physiol* doi:10.1152.
14. Konstantinos, Dimitrios Tambalis, Demosthenes B, Panagiotakos ., Stavros A, Kavouras ., and Lambros S, Sidossis. (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.
15. Kodama, Satoru., Shiro, Tanaka., Kazumi, Saito., Miao, Shu., Yasuko, Sone., Fumiko, Onitake., Emiko, Suzuki., Hitoshi, Shimano., Shigeru, Yamamoto., Kazuo, Kondo., Yasuo, Ohashi., Nobuhiro, Yamada., Hirohito, Sone.(2007) Effect of Aerobic Exercise Training on Serum Levels of High-Density Lipoprotein Cholesterol: A Meta-analysis. *Arch Internal Med* 167, 999-1008.
16. L.Holme, A. T., Hostmark and Anderssen, S. A. (2007) ApoB but not LDL-cholesterol is reduced by exercise training in overweight healthy men. Results from the 1-year randomized Oslo Diet and Exercise Study. *Journal of Internal Med* 262(2), 235 – 243.
17. Lee Min, Howard D. Sesso, Yuko Oguma, Ralph S., Paffenbarger, Jr. (2003) Relative Intensity of Physical Activity and Risk of Coronary Heart Disease. *Circulation* 107, 1110-1116.
18. Martin,R.P., Haskell,W.L and Wood,PD.(2002) Blood chemistry and lipid profiles of elite distance runners. *Annals of New York Academy of science* 301, 346-360.
19. Mercedes R, Carnethon. (2009) Physical Activity and Cardiovascular Disease: How Much Is Enough? *A J OF L Med* 3(1), 44-49.
20. Mestek, M. L. (2009) Physical Activity, Blood Lipids, and Lipoproteins. *AM JO OF LIF MED* 3, 279-283.
21. Michael L, Mestek.(2009) Physical Activity, Blood Lipids, and Lipo proteins .*American Journal of Lifestyle Medicine* 3(4), 279-283.
22. Miller, G. S., Martin, S. E., Glowacki, S., Womack, J. W., Green, J. S. FACSM and Crouse, S. F. FACSM. (2006) The Effect of Training Modality on Apo-A1, Apo-B, and the Apo-B/Apo-A1 Ratio in Men. *Medicine & Science in Sports & Exercise* 38 (5), 484.
23. Metsios, G. S. A., Stavropoulos-Kalinoglou, J. J. C. S., Veldhuijzen van Zanten, G. J., Treharne, V. F., Panoulas, K. M. J., Douglas, Y. Koutedakis and G. D. Kitas.(2008) Rheumatoid arthritis, cardiovascular disease and physical exercise: a systematic review. *Rheumatology (Oxford)* 47, 239-248.
24. [Nicklas, BJ.](#), [Wang , X.](#), [You, T.](#), [Lyles, MF.](#), [Demons, J.](#), [Easter, L.](#), [Berry, MJ.](#), [Lenchik, L.](#), [Carr, JJ.](#) (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.
25. O'Donovan., Gary., Andrew, Owen., Steve, R. Bird., Edward, M. Kearney., Alan, M. Nevill., David, W. Jones and Kate Woolf-May. (2005) 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* 98, 1619-1625.
26. [Parish Sarah.](#), Richard, Peto., Alison, Palmer., Robert, Clarke., Sarah, Lewington., Alison, Offer., Gary, Whitlock., Sarah, Clark., Linda, Youngman., Peter, Sleight.(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.
27. Press, V. Freestone and C.F. George. (2003) Physical activity the evidence of benefit in the prevention of coronary heart disease. *jour of Med* 4, 245-251.
28. Pynn, Marianne., Katrin, Schäfer., Stavros, Konstantinides and Martin Halle.(2004) Exercise Training Reduces Neointimal Growth and Stabilizes Vascular Lesions Developing After Injury in Apolipoprotein E-Deficient Mice. *Circulation* 109, 386-392.
29. Savvas, P. Konstantinos A.(2003) Training and Detraining Effects of a Combined-strength and Aerobic Exercise Program on Blood Lipids in Patients With Coronary Artery Disease. *Journal*

- of *Cardiopulmonary Rehabilitation & Prevention* 23 (3) 193 – 200.
30. Sharma, Arya M., Arno, Schmidt., Luca, Mascitelli., Francesca, Pezzetta., Cris A. Slentz., William, E. Kraus., Alan, R. Tall. (2003) Effects of Exercise on Plasma Lipoproteins *NEJM* 348, 1494-1496.
 31. Slentz., Cris A. Slentz., Joseph A. Houmard., Johanna L. Johnson., Lori A. Bateman., Charles J. Tanner., Jennifer S. McCartney., Brian D. Duscha and William E. Kraus. (2007) Inactivity, exercise training and detraining, and plasma lipoproteins. STRRIDE: a randomized, controlled study of exercise intensity and amount *J. Appl. Physiol* 103, 432-442.
 32. Susanne, Dimitriou., von duvillard, Serge P., Paul, weber., Bernhard, Stadlmann., Monika, Emura., Linda M., Pek Kayla and Mueller, Erich. (2007) Nine months aerobic fitness induced changes on blood lipids and lipoproteins in untrained subjects versus controls. *European journal of applied physiology* 99(3),291-299.
 33. Stefan, Branth., Sjodin, Anders., Foslund, Anders., Hambraeus, Leif , Holmback 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.
 34. Tall, A. R. (2002) Exercise to Reduce Cardiovascular Risk . How Much Is Enough? *NEJM* 347, 1522-1524.
 35. Tokmakidis, SP., Volaklis KA. (2003) Training and detraining effects of a combined-strength and aerobic exercise program on blood lipids in patients with coronary artery disease. *J Cardiopulm Rehabil* 23(3) 193-200.
 36. William, E.Kraus., Joseph, A. Houmard., Brian, D. Duscha Kenneth J. Knetzger., Michelle B. Wharton., Jennifer S. McCartney., Connie W. Bales., Sarah Henes., Gregory P. Samsa., James D. Otvos., Krishnaji R. Kulkarni and Cris A. Slentz. (2003) Effects of the Amount and Intensity of Exercise on Plasma Lipoproteins. *N Engl J Med* 348,1494-1496.
 37. Von Stengel, simon. (2004) Exercise effects on CHD-Risk-Factors in Early postmenopausal women with increased cholesterol levels- preliminary 4-year-results. *Med Sci.Sport,Exer* 36(5) , 212.
 38. Yourka, D Tchoukalova., Christina, Koutsari., Maksym, V Karpyak., Susanne, B Votruba., Eliana, Wendland and Michael, D Jensen.(2008) Subcutaneous adipocyte size and body fat Distribution. *Am J Clin Nutr* 87, 56-63.