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The Chronic Effects of Morning Exercise Training on Lipoprotein(a) Levels in Over weight Males

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ABSTRACT

Increment of serum concentration of Lipoprotein(a) [LP(a)] is an independent Coronary Artery Diseases (CAD) risk factor and clinical updates support of LP(a) roles in CAD. We assumed that appropriate and regular physical activity should be reduce the amount of LP(a) and thus reduce the risk of CAD; but research evidences indicated that LP(a) levels not been alter by physical activity and it is resistant to the exercise training stimulations. So, the serum concentration of LP(a) in active and sedentary middle age males is different? What is the effect of physical activity and exercise training on serum concentration of LP(a) in middle age males? The aim of this Cross-sectional study was to determine and compare of LP(a) serum concentration in selected groups of men (40-55 yrs). This descriptive study was designed in three groups of subjects who were voluntarily participated and randomly selected. Fasting serum concentration of LP(a) (Active: 18.06 ± 11.11; Sedentary: 22.06 ± 13.99; CAD: 28.33 ± 7 .20 mg/dl) analysis with Immunoturbidimetric method. LP(a) compared with Kruskal-Wallis Ranks and Mann-Whitney (U) tests (P≤0.05). Results demonstrated that LP(a) differences between Active and CAD groups are significant (p=0.009). Study evidences and results of this study indicated that LP(a) levels not been alter by physical activity and it is resistant to the exercise training stimulations. If LP(a) changes was based on exercise training effects, these differences most are observed between active and sedentary groups, also; but in this study were not seen. Therefore to understanding the effects of exercises training and physical activity on LP(a), more study should be performed.

Key words: LP(a) , Physical Activity , CAD, Exercise.

INTRODUCTION

Increment of serum concentration of Lipoprotein(a) [LP(a)] is an independent Coronary Artery Diseases (CAD) risk factor and clinical updates support of LP(a) roles in CAD [1, 2, 5, 39]. LP(a) is a compound, similar to LDL-c with a concentration of 0.05 to 1.90 mmol/l (20-1500 mg/dl) in normal plasma. It is produced in the liver and includes apo(a) and apo(B_{100}). It is an independent and positive risk factor of coronary artery diseases (CAD) and clinical findings support the role of LP(a) in CAD. LP(a) can participate in atherosclerosis processes, such as LDL-c. LP(a) connects to LDL-c receptor through its apo-B_{100} domain. LP(a), unlike other lipids, not only participates in atherosclerosis, but also plays a role in atherothrombosis processes [3, 4, 18-20]. The hypothesis is that like lipoproteins and other risk factors of cardiovascular diseases, regular exercising and physical activity can decrease LP(a) level and accordingly CAD risk [38]; but, the study shows that probably LP(a) is not affected by exercising and physical activity and is resistant to exercising stimulus. The results of the previous study indicated that there is no significant relationship between physical activity and LP(a) in males and females regardless of duration, severity and type of exercise [3, 6, 7, 8, 10, 13, 14, 17, 20 - 23, 26, 35, 38]. On the other hand, the findings of the other study indicated that there is a significant correlation between exercising in terms of duration, severity and types of exercise with LP(a) concentration in the serum; and physical activity reduces the concentration of the LP(a) and the risk of

cardiovascular diseases [9, 11, 12, 16, 25, 27, 30, 32, 36, 40]. However in the some study LP(a) concentration in serum increased after severe exercise [22, 31]. We assumed that aerobic and continues exercise training and regular physical activity should be reduce the amount of LP(a) and thus reduce the risk of CAD, such as LDL_C; but research evidences indicated that LP(a) levels not been alter by exercise training and it is resistant to the exercise training stimulations. In addition, correlated associations between VO_2max and this risk factor have occasionally, but not consistently, been demonstrated. Therefore, what are the serum concentrations of LP(a) in active, sedentary and with CAD middle aged man? What is the effect of physical activity and exercise training on serum concentration of LP(a) in middle age males?

MATERIALS AND METHODS

The aim of this Cross-sectional study was to determine and compare of serum concentration of LP(a) in selected groups of men (40-55 yrs). Invitation letters including objectives and methodology of research, letters of satisfaction and voluntary participation, Health and Disease Risk Questionnaire, National Health Interview Survey (NHIS) and Physical Activity Rating (PA-R) were distributed among the participants. In this descriptive and analytical study, subjects (N = 3*15) were voluntarily participated, was based on NHIS and PA-R questionnaire [19, 28]; one as active [age: 47.86 ± 5.33 (yrs), BMI: 27.96 ± 2.26 (kg/m^2)] and the second as sedentary [age: 43.53 ± 4.34 (yrs), BMI: 26.26 ± 2.96 (kg/m^2)] and the other as men with CAD [age: 48.13 ± 5.85 (yrs), BMI: 26.44 ± 2.34 (kg/m^2)]. This subjects randomly selected out of 19 men who were participated in morning training, 18 employees and 17 men of CAD outpatients, respectively. The activity level was based on American College of Sports Medicine (ACSM) standard in NHIS and Physical Activity Rating in PA-R questionnaire [19, 28]. Elderly men (active and sedentary) lacked any external and clinical symptom of cardiovascular diseases, diabetes, hypertension, etc. and had not taken any special medication and supplement and didn't have any special diet. Estimated VO_2max (Active: 39.04 ± 2.56, Sedentary: 32.64 ± 3.05 and CAD: 30.37 ± 4.27 $mL.Kg^{-1}.min^{-1}$) as a physical activity rating index (independent variable) estimated was based on non exercise prediction equations for VO_2max, was developed by researchers at the university of Houston [19, 28]; using age, physical activity status and body mass index. Blood sampling was done to measure fasting serum concentration of Lp(a) after 9 to 12 hours of fasting, 7-8 am, from left Antecubital vein at medical diagnosis laboratory. Dependent variable included fasting serum concentration of LP(a) (Active: 18.06 ± 11.11 and Sedentary: 22.06 ± 13.99 and CAD: 28.33 ± 7.20 mg/dl) analysis with Immunoturbidimetric method, using Biomerio full automated immunoanalyser. The normality of the distribution and homogeneity of variances tested with Kolmogorov–Smirnov and Levene's tests respectively. Pearson correlation equations measured between estimated VO_2max and Lp(a) in groups. Pearson correlation coefficient significance was determined using Fisher's T test. Correlation coefficients were compared using Fisher's Z test. Estimated VO_2max and LP(a) compared with ANOVA and LSD tests and with Kruskal-Wallis Ranks and Mann-Whitney (U) tests, respectively ($P \leq 0.05$).

RESULTS AND DISCUSSION

Table 1 show the descriptive characteristic of the groups and variables. Age was significantly different between groups ($p = 0.034$, $F(2, 42) = 3.06$*). Body mass index (BMI) was not significantly different between groups ($p = 0.144$, $F(2, 42) = 2.029$). Differences of estimated VO_2max between the groups were significant ($p \leq 0.001$, $F(42, 2) = 26.54$*). Results of LSD test showed that the VO_2max differences between active and sedentary groups ($p \leq 0.001$) and between active and CAD groups ($p \leq 0.001$) were significant. LP(a) differences between groups were significant ($p = 0.03$, $\chi^2(15, 2) = 7.047$*). The results of Mann-Whitney U test showed that the LP(a) differences between active and CAD groups were significant ($p = 0.009$). Table 2, show the correlation coefficients of LP(a) with estimated VO_2 max in active ($r = 0.122$, $p = 0.665$), sedentary ($r = 0.027$, $p = 0.924$) and CAD groups ($r = 0.014$, $p = 0.962$). None of the correlation coefficients were significant. Comparison of correlation coefficients of estimated VO_2max and LP(a) showed that the differences of correlation coefficients between groups were not significant.

Table 1. ANOVA test for equality of means (M ± SD)

Groups	n	Age (yrs)	BMI (kg/m²)	VO_2max (ml.kg^{-1}.min^{-1})	LP(a) (mg/dl)
Active	15	47.86 ± 5.33*	27.96 ± 2.26	39.04 ± 2.56**	18.06 ± 11.11 ***
Sedentary	15	43.53 ± 4.34*	26.26 ± 2.96	32.64 ± 3.05**	22.06 ± 13.99
CAD	15	48.13 ± 5.85*	26.44 ± 2.34	30.37 ± 4.27**	28.33 ± 7.20 ***

**Active and Sedentary groups (p = 0.028), Sedentary and CAD groups (p = 0.020)*

***Active and Sedentary groups (p = 0.001), Active and CAD groups (p = 0.001)*

**** Active and CAD groups (p = 0.009)*

Table 2. Pearson correlation of estimated VO_2max with LP(a).

LP(a) / VO_2max		n	r	sig	r^2	Z_{obs} / Z_r			
Active	A	15	0.122	0.665	0.015	0.123	A		
Sedentary	S	15	0.027	0.924	0.001	0.027	0.235	S	
CAD	C	15	0.014	0.962	0.001	0.014	0.267	0.032	C

Active, sedentary and CAD groups were defined and compared on the basis of the differences in physical activity and estimated VO_2max as independent variable. Thus, estimated VO_2max values are naturally different between active, sedentary and CAD groups. VO_2max calculated according to non exercise estimated VO_2max formula of the University of Houston in terms of age, physical activity, body mass index, sex and a constant coefficient. In this study, sex coefficient was fixed; since all the participants were male. According to table 1, the three groups were not significantly different in terms of body mass index. The participants age were between 40-55, and the age differences of active and sedentary groups (p = 0.028) and active and CAD groups (p = 0.02) were significant. With regard to age coefficient in the formula (-0.381), maximum difference of estimated VO_2max due to age difference (4.33 years) in two groups of active and sedentary is 1.65 ml.kg^{-1}.min^{-1} ; which is ignorable. Mean difference of physical activity rating in the three groups were significant, (PA-R mean of active, sedentary and CAD groups were 5.73, 0.80 and 1.00 respectively). Despite the age mean difference between groups, the significant differences between active and sedentary groups and between active and CAD groups in terms of estimated VO_2max was due to the significant differences of physical activity rating in groups.

Not only clinical findings support the role of LP(a) in CAD [1, 3, 5, 39]; but also the results of some previous study indicated that the effect of high concentration of LP(a) on CAD [29, 33, 34, 36]. The results of the some previous study indicated that the concentration of LP(a) in active ischemic patients, was insignificantly lower than inactive patients [23, 24]. In this study, difference of LP(a) concentration in active group (18.06) and CAD group (28.33) was significant (p = 0.009). The concentration of LP(a) in active group was less than sedentary and CAD groups. But, the findings of the previous study and this study indicated that LP(a) is not under the effect of physical activity and it is resistant to exercise training. If LP(a) changes were affected by physical activity and exercise training, the difference should be observed in active and sedentary groups. Yet, the present study didn't show any difference between groups. Also, no significant correlation was observed between VO_2max and LP(a) concentration in the groups. Comparison of correlation coefficients didn't show any significant difference between groups. The outcomes can be attributed to other factors increasing LP(a) concentration in the CAD group; and physical activity and exercise training has not effect to decreasing LP(a) in active group. LP(a) levels in the serum which is mainly determined genetically approximately remains fixed in human being. It doesn't seem that identified factors that affect lipoproteins, such as medication, decreasing the amount of fat and changing body mass and exercise; can affect LP(a) [19, 20, 28, 37].

CONCLUSION

Some cross-sectional and population studies indicate a weak relationship between LP(a) concentration and regular physical activity. Some other studies support the decreasing effects of exercise training and high levels of cardio respiratory fitness on serum LP(a). Some other cross-sectional and interventional study administered in 12 weeks to 4 years have shown that serum LP(a) does not change despite increasing in VO_2max and decreasing other serum lipoproteins. Also, some other cross-sectional study indicated that serum LP(a) probably increases in sever physical activity like distance running or weight lifting for more than several months to several years. Some interventional studies have shown average decrease of LP(a) (10-15%) after 9 to 12 months of severe exercising. Recent study evidences and results of this study indicated that probably LP(a) levels not been alter by aerobic and continues exercise training and regular physical activity and it is resistant to the exercise stimulations. If LP(a) changes was based on exercise training effects, this differences most be observed between active and sedentary groups, also; but this differences in this study was not seen. In addition, any reasonable correlation association between VO_2max and LP(a) in groups was not seen. The comparison of correlation equation between groups by use Fisher Z test indicated

that were not differences. Recent studies have demonstrated the same results. LP(a) level is resistant to exercise stimulation and will not affect by regular physical activity. Since this difference was observed between active and CAD groups, it can be concluded that other factors such as genetics or related disease are probably responsible for LP(a) increment in CAD group and probably LP(a) decrement is not as a result of exercise training in active groups. Yet the quality of LP(a) metabolism during exercise training in not clear. More studies need to be done to clarify the optimum levels of intensity, duration and type of exercise and also to determine the probable effects of exercises training on LP(a). Therefore; to understand the associations and probably effects of exercises training and physical activity on LP(a), more study should be performed.

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