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The Comparison of Serum Concentrations of LP(a), LDL-c and HDL-c in Speed, Semi-endurance and Endurance Runners

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ABSTRACT

*Increment of lipoprotein(a) [LP(a)] Concentration, as a coronary artery disease risk factor. Probably LP(a) not been affected by physical activity and it is resistant against exercise stimulation. The aim of this cross-sectional study was to determine and compare of LP(a) concentration in selected groups. This analytical study was designed in three groups of subjects (15-25 yrs) who were voluntarily participated and randomly selected from Speed (age: 20.77 ± 3.14, BMI: 19.09 ± 0.97 kg/m²), semi-endurance (age : 19.70±3.42, BMI: 20.25±1.74 kg/m²) and endurance (age: 22.61 ± 2.96, BMI: 19.98 ± 2.32 kg/ m²) runner females (N=3*13). Fasting serum concentration of LP(a) (Speed: 14.00 ± 9.55, semi-endurance: 17.00 ± 10.93 and endurance: 15.30±14.58 mg/dl) analysis with ELISA method. Data compared with Kolmogorov - Smirnov, Levine and ANOVA statistical tests ($p \leq 0.05$).Results demonstrated that differences of LP(a) concentration between groups were not significant ($p = 0.813$, $F_{(2,36)} = 0.208$). Comparison of LP(a) Concentration in speed, semi-endurance and endurance groups, to determine of different exercises training on this risk factor, were not significant. It seems that the findings of previous research and this study, LP(a) not been affected by exercise and it is resistance against training stimulation. Therefore physical activity has not any desirable effect on LP(a) concentration. So, to clarify the possible effect of exercise training on LP(a), more research `must be designed and implemented.*

Key Words: LP(a), LDL_C, HDL_C, Running, Women.

INTRODUCTION

Lipoprotein(a) [LP(a)], very similar to low density lipoprotein cholesterol (LDL-c), is produced in the liver and contain apo(a) and apo(B_{100}). Concentrations of LP(a) in serum is less than 0.05 to 1.90 mmol/liter or even higher. LP(a); Also, unlike other lipids, in addition to participation in the process of atherosclerosis, participate to the atherotrombosis process [1,3,5,9,24]. On the

other hand a reduced serum concentration of high density lipoprotein cholesterol (HDL-c) is risk factor for coronary artery disease [1, 8, and 22]. This phenomenon is caused by a sedentary lifestyle and lack of physical activity and considered as a risk factor for coronary artery disease [5, 6, 14, 16, 18, 19]. Further results showed that physical activity and exercise habits are an important factor in reducing disease severity. Jagroop (2002), Durstine (1996), Buyukazi (2005), Durstine (2001), Lippi G (2006), Thomas (1997), Lee Mora (2007), Heitkamp (2008), Martin (1999), Drowatzky (2001), Montgomery (2002), Lehmann (1997), and Durstine (1996) showed no beneficial effect and relationship between physical activity and exercise levels on lipoprotein (a) levels in men and women with different severity and type of exercise. The results of Mora (2006), the Sai Yong (2008), Ginsburg (1999), Knight (1998), Sattler (2002), Williams (2005), Mercedes (2000) and Hartgens (2004), showed beneficial effect and relationship between physical activity and exercise levels on LP(a) levels in men and women. In summery, Increment of LP(a) and LDL_C and decrement of HDL_C concentrations, are CAD risk factors and Probably LP(a) not been affected by training and it is resistant against exercise stimulation. Therefore what is the Effects of Running Training on Serum Concentrations of LP(a), LDL-C and HDL-C in Female?

MATERIALS AND METHODS

The aim of this cross-sectional study was to determine and compare of LP(a), LDL_C and HDL_C concentrations in selected groups. This analytical study was designed in three groups (N=3*13) of subjects (15-25 yrs) who were voluntarily participated and randomly selected from speed (age: 20.77±3.14, BMI: 19.09±0.97 kg/m²), semi-endurance (age: 19.70±3.42, BMI: 20.25±1.74 kg/m²) and endurance (age: 22.61±2.96, BMI: 19.98±2.32 kg/m²) runner females. Subjects selected from speed (100, 200 and 400 m; n=16), semi-endurance (800 and 1500 m; n=15) and endurance (3000 and 5000 m; n=15) elite runners with at least two years running experience. Fasting blood samples to measure serum concentrations of LP(a) (speed: 14.00±9.55, semi-endurance: 17.00 ± 10.93, endurance: 15.30±14.58 mg/dl), LDL_C (speed: 82.53±17.74, semi-endurance: 77.76±12.57, endurance: 94.38 ±26.00 mg/dl) and HDL_C (speed: 63.92 ± 13.91, semi-endurance: 60.00±17.76, endurance: 63.92±13.37 mg/dl) with ELISA method were done. The normality of distribution and homogeneity of variances determined by Kolmogrov - Smirnov and Levin test, respectively. Mean compared with ANOVA statistical tests ($p \leq 0.05$).

RESULTS AND DISCUSSION

Age differences between groups were not significant (p=0.074, F (2, 36) =2.809). Body mass index differences between groups were not significant (p=0.226, F (2, 36) = 1.549). Lipoprotein(a) differences between groups were not significant (p=0.813, F (2,36)=0.208). HDL-c differences between groups were not significant (p=0.749, F (2, 36) =0.291). LDL-c difference between groups were not significant (p=0.098, F (2, 36) =2.485). Results demonstrated that differences of LP(a), LDL_C and HDL_C concentrations between groups were not significant. Comparison of serum concentrations of LP(a), LDL_C and HDL_C in speed, semi-endurance and endurance with control groups to determine the training effects on these risk factors were not significant. Cardoso (1996) stated that the concentration of LP(a) in endurance runners was higher than strength training and control groups [4]. Mackinnon (1999) stated that the concentration of LP(a) in endurance runners was higher than other athletes [17]. Durstine (2001) stated that concentrations of LP(a) at different levels of fitness and physical activity are not different [7]. Mora (2006) showed weak association between physical activity and concentrations of LP (a) in Women [19]. So, it seems that the effect of physical activity and exercise on the LP(a), are scattered and inconsistent. The result, according to the nature of this type of training

and energy systems involved in implementing such training is not unexpected. Gruden (1996) stated that excessive speed and exercises that stimulate the adrenergic system may cause acute changes on LP(a). Hubinger (1996) stated that the 12 weeks endurance training with moderate intensity in endurance man runners alter the levels of LP(a) [13]. Lee Mora (2007) also stated that the concentration of LP (a) levels after 9 months of endurance training did not differ [28]. While the results of Ginsburg (1999) showed that the concentrations of LP(a) increased immediately after intense endurance race [30]. Parano (1998) also stated that after the implementation of a course of cardiac rehabilitation programs, reduction of LP(a) levels were observed [20]. Lippi G (2006) stated that in endurance athletes compared with non-athlete control group, serum LP(a) has not difference [16]. So, it seems that the results of Hubinger (1996) Thomas, Ziogas and Harris (1997), Lee Mora (2007) and Heitkamp (2008) have a good correlation. While the results of Parano (1998), Ginsburg (1998), Lippi G (2006) have no agreement. It seems that the effect of endurance exercise training on long-term changes of LP(a) are scattered.

Different factors interact with physical activity and exercise on concentrations of lipoprotein (a) levels. Mackinnon and Hubinger and McCarthy (1996) stated that low-fat diet recommended for weight loss may cause slight changes in serum LP(a) [13]. Martin (1999) stated that a family history of coronary artery disease and congenital and hereditary hyperlipoproteinemia determined levels of LP(a) and energy consumption in physical activity and not effect on LP(a) [18]. Mercedes (2000) stated that LP(a) responses to exercise is important [21]. Williams (2005) stated that activity level and diet changes have similar effects on LP(a) [29]. Fallah Mohammadi (2006) stated that the combined strength and endurance training reduces LP(a) in men [8]. The elite runners were not related to any underlying disease. They have not a history of taking any medication, supplements, sports nutrition and on diet. Family history and genetic in subjects were also negative. Some cross-sectional studies have consistently shown a weak correlation between levels of LP(a) and regular moderate physical activity. Other research indicated that effects and associated high levels of cardiopulmonary fitness and decreasing LP(a) levels. Some intervention studies have reported a slight increase to moderate levels of LP(a), from 9 to 12 months of intense exercise training .

CONCLUSION

The role of lipoprotein(a) and details of its metabolism process well not clear; therefore, we can not comment about how the process of metabolism during exercise and the possible effects of exercise and physical activity on this risk factors. The optimal level of intensity, duration and type of exercise training is very important unanswered question. It seems that the findings of previous and this study, LP(a) not been affected by exercise and it is resistance against training stimulation. Therefore physical activity has not any desirable effect on LP(a) concentration. So, to clarify the possible effect of exercise on LP(a), more study must be designed and implemented.

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