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Lipid profile and chronic exercise training in Cigarette smoke

Mirakhori Fatemeh, Mirakhori Zahra and Zand Alireza
Department of Physical Education and Sport Science,
Shahr-e-Qods Branch, Islamic Azad University, Tehran, IRAN

(Corresponding author: Mirakhori Fatemeh)
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ABSTRACT: Cigarette smoke (CS) is a main risk factor in cardiovascular diseases, metabolic syndrome and other chronic diseases. The main purpose of present study was to assess the effect of long term exercise training on lipid profile markers in males with CS. Fating blood samples was collected and serum separated in twenty two adult males with CS that divided into exercise (3 months aerobic training) or control subjects. The serum was used for triglyceride (TG), total cholesterol (TC), high-density lipoprotein (HDL-cholesterol) and low-density lipoprotein (LDL-cholesterol) as lipid profile markers. Pre and post training data were compared with Paired sample T test. All statistical tests were performed and considered significant at a $P \leq 0.05$. At baseline there were no differences in the age, body weight, and other anthropometrical or lipid profile indexes between the two groups. We observed a significant decrease for HDL-cholesterol ($p = 0.001$) and anthropometrical markers by exercise program in exercise group. All other parameters remained unchanged in exercise group. All variables remained unchanged in the control group. It seems that exercise training can be improve these metabolic markers only when smoking is stopped. Further, long-term, well-conducted studies are still required to elucidate the significance of exercise training on hormonal or metabolic mediators in presence to smoking.

Keywords: Cigarette smoke, Lipid profile, Exercise

INTRODUCTION

Tobacco use is one of the most common habits on the rise that has affected people's life style more than 4 centuries [1]. Currently smoking is increasing at a dramatic rate in the world and is known as a risk factor in the prevalence of chronic diseases such as atherosclerosis, diabetes and respiratory diseases such as chronic obstructive pulmonary disease (COPD) [1, 2, 3]. In addition to the impact of smoking on mood and lifestyle, a relationship between smoking and systemic inflammation and cardiovascular diseases has been repeatedly reported [4]. Furthermore, some previous studies suggest a direct relationship between smoking and total cholesterol (TC), low density lipoprotein (LDL) and cholesterol to Hidensity lipoprotein (TC/HDL), and a negative relation with HDL [5].

However, other studies reported increased number of white blood cells (WBC) and leukocytes in response to smoking [6, 7]. Some studies also pointed to the direct and indirect association between smoking and increased lipid levels. These findings suggest that smoking is related to increased abdominal fat, increased waist to hip ratio [8. 9], increased free fatty acid and glycerol stimulation, the dyslipidemia phenomenon, in other words, increased LDL and decreased HDL, endothelial dysfunction and increased blood viscosity [10, 11].

Some recent studies showed that 8 week of reduced smoking is associated with dramatic changes in the blood levels of LDL, HDL and TC/HDL [12]. However, other studies also pointed out that although some changes caused by smoking are reversible after quitting smoking, the levels of C - reactive protein (CRP), as one of cardiovascular risk factors, are still kept at high levels even after 10 to 20 years of quitting smoking [1]. Among external interventions, the role of short-term training programs on the levels of inflammatory mediators and cardiovascular risk factors in patient populations or smokers is of considerable importance. In this context, significant improvements in HDL and other cardiovascular risk factors in response to long-term training programs in the overweight populations or obesity-related diseases have already been reported by some studies, so that these studies pointed to significantly increased HDL following training programs [13, 14, 15]. Some others reported no change in these variables because of physical training [16]. Despite these contradictory findings on the response of cardiovascular risk factors to physical training in other populations, studies in this field are also limited in smokers. This study aimed to investigate the effects of three months of aerobic exercise on the levels of cardiovascular risk factors in male smokers.

MATERIAL AND METHODS

Study subjects: Twenty two adult men with cigarette smoke participated in the study by accessible sampling and selected for exercise (age, 40 ± 6.6 year; height, 176 ± 2.6 cm, n=11) or control (age, 40 ± 1.9 year; height, 175 ± 1.4 cm, n=11) groups.

Anthropometry and study inclusion: All subjects were otherwise in good health were taking no medications. Participants were included if they had not been involved in regular physical activity in the previous 6 months. Patients with known history of respiratory infections, neuromuscular disease, cardiopulmonary disease and type II diabetes or other chronic diseases were excluded.

Anthropometric measurements of height, weight, percent body fat, and circumference measurements were taken study. Weight was measured to the nearest 100 g using digital scales. Standing height was measured to the nearest 0.1 cm with the use of a wall-mounted stadiometer. The BMI was calculated as the weight in kilograms divided by the square of the height in meters. Abdominal and hip circumference were determined in a standing position at the end of normal expiration

Blood Collection: Blood samples were collected, via the cannulated antecubital vein, between 8:00–9:00 a.m. after an overnight fasting for all groups before and after (48 hour) the exercise program. The serum was used for triglyceride, total cholesterol, HDL-cholesterol and LDL-cholesterol as lipid profile markers by enzymatic methods (Randox direct kits) using Kobas Mira auto-analyzer made in Germany.

Training protocol: Subjects of exercise group achieved a 12-week individualized aerobic training program consisting of three sessions of walking, running and/or cycling, without any dietary intervention. In each session, after a warm-up (5-10 min), subjects trained for approximately 35-45 min 60-80% of maximal heart rate then cool up for 5 min. For all subjects, the first training session was performed under professional physiologist supervision. Control subjects were instructed to maintain their habitual activities. The intensity of the activity of any person was controlled using the Polar heart rate tester (made in the US).The participants of two groups were asked to remain their diet during the study.

Statistical analysis: Data were analyzed by computer using the Statistical Package for Social Sciences (SPSS) for Windows, version 11.5. We verified normal distribution of variables with a Kolmogorov–Smirnov test, and the parametric variables with skewed distribution were expressed as mean ± SD. Differences between groups were calculated using the independent samples t-test. Student's paired 't' test was applied to compare the pre and post training values. The differences between the groups were considered to be significant at a p-value of ≤ 0.05.

RESULTS

As previous mentioned, in present study, lipid profile markers were compared between before and after three aerobic training in smokers. Body weight and other anthropometrical parameters during experimental protocol are shown in Table 1. No significant difference was observer in anthropometrical markers between two groups at baseline.

Aerobic exercise training results in significant decrease in body weight ($p = 0.000$), BMI ($p = 0.000$), body fat % ($p = 0.006$) and other anthropometrical markers in exercise group, but not in control subjects (Table 1).

Table 2 contains the changes in lipid profiles markers by aerobic training program in exercise group as well as control subjects. All lipid profile markers were also same between two groups at baseline.

Exercise training led to a significant increase in serum HDL-cholesterol when compared with baseline ($p = 0.001$, Fig 1). No significant differences were found in TG ($p= 0.181$), TC ($p = 0.690$) and LDL-cholesterol ($p = 0.234$) by exercise program with compared to baseline in exercise group. All anthropometrical and clinical parameters remained unchanged in control group.

Table 1: Mean and SD of anthropometrical parameters of two groups at pre and post training.

group	**Weight** (kg)	**AC** (cm)	**HC** (cm)	**Systolic BP** (mmHg)	**Diastolic BP** (mmHg)	**BMI** (kg/m2)	**BF** (%)
Exercise (*pre-training)*	***92.4*** *(5.44)*	***103*** *(6)*	***103*** *(5.3)*	***127*** *(9)*	***87*** *(6)*	***29.9*** *(1.97)*	***28.8*** *(1.96)*
Exercise *(Post-training)*	***88.9*** *(5)*	***98.6*** *(5.4)*	***98*** *(4.1)*	***125*** *(7)*	***84*** *(5)*	***28.8*** *(1.88)*	***27.8*** *(1.94)*
Control *(pre-training)*	***90*** *(5.69)*	***100*** *(3.1)*	***99.6*** *(2.7)*	***132*** *(10)*	***89*** *(7)*	***29.5*** *(1.9)*	***28.6*** *(1.7)*
Control *(pos-training)*	***90.3*** *(5.73)*	***99.9*** *(2.7)*	***99.5*** *(2.8)*	***130*** *(10)*	***90*** *(6)*	***29.6*** *(1.9)*	***28.6*** *(1.8)*

AC*, Abdominal circumference;* ***HC****, Hip circumference deviation;* ***BMI****, body mass index;* ***BF,*** *Body fat percentage;*

Table 2: Mean and SD of lipid profile markers of two groups at pre and post training.

group	Total cholesterol (mg/dl)	Triglyceride (mg/dl)	LDL cholesterol (mg/dl)	HDL cholesterol (mg/dl)
Exercise (*pre-training)*	***166*** *(32)*	***165*** *(58)*	***108*** *(26)*	***43*** *(3.7)*
Exercise *(Post-training)*	***167*** *(31)*	***157*** *(46)*	***101*** *(14)*	***45.6*** *(3.6)*
Control *(pre-training)*	***160*** *(27)*	***164*** *(42)*	***113*** *(16)*	***41*** *(2.6)*
Control *(pos-training)*	***163*** *(20)*	***159*** *(40)*	***116*** *(21)*	***41*** *(2.4)*

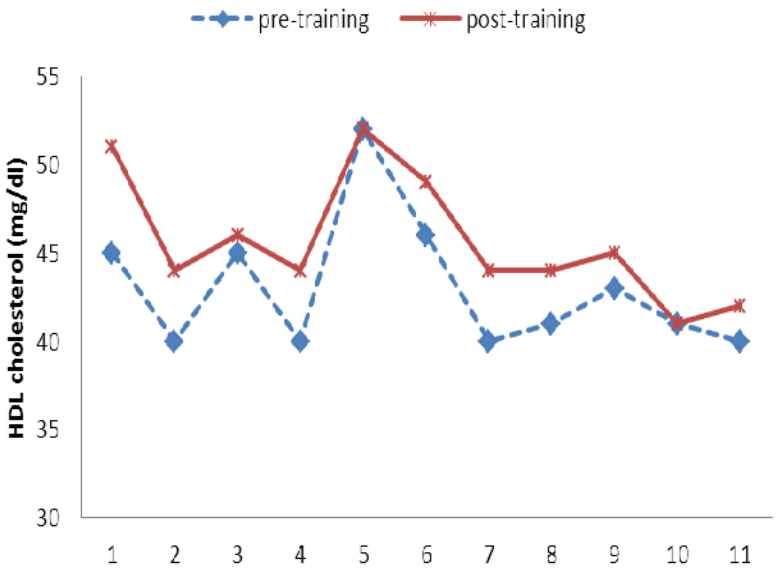

Fig 1: Pre and post training HDL-cholesterol of subjects in exercise group.

DISCUSSION

The non-change of TG, TC, and LDL is among the findings of this study. In other words, a three-month aerobic training did not result in changes in cardiovascular risk factors in male smokers. Continuous use of tobacco is the second leading cause of death worldwide [1]. If smoking continues in the same way, it is expected that in 2030, it directly victimizes an annual average of 9 million people [17]. Tobacco use leads to higher triglycerides and lower serum HDL [12]. Most previous studies pointed that smoking is associated with certain metabolic disorders such as dyslipidemia [18, 19]. According to some studies, smokers are exposed to metabolic disorders characterized by insulin resistance syndrome which is associated with increased postprandial triglyceride followed by increased small dense HDL and small dense LDL particles [20]. The researchers emphasized that increased postprandial triglyceride levels and vLDL in smokers is much higher than non-smokers.

Some scientific sources claimed that after 8 weeks of reduced smoking, significant changes occur in the levels of LDL and the HDL/LDL ratio [21]. However, some studies pointed that although some changes caused by smoking are reversible after quitting, the CRP inflammatory mediator are maintained at high levels even after 10 to 20 years after quitting [1].

In the present study, non-change of cardiovascular risk factors in the studied smokers was observed while some other studies reported a significant reduction of these variables in other populations, such as diabetics and obese people. In a recent study, 3 and 6 months of aerobic and resistance exercise led to a significant decrease in TG, TC, TC/HDL along with the increase of HDL in patients with cardiovascular diseases [15]. In another study, 24 weeks of physical exercise led to an increase in HDL and decrease in TG, TC in patients with metabolic syndrome [14]. However, in line with the present study, there are some studies that reported the non-change of lipid profile parameters in response to long-term training programs such as 12 weeks of aerobic exercise [16]. The lack of significant change in these variables may be attributed to the low sample size. However, despite the non-change of TG, TC and LDL as cardiovascular risk factors in this study, HDL levels increased significantly in response to the training program.

In other words, a three-month aerobic training led to a significant increase in HDL as good cholesterol in the studied male smokers. The non-change of TG, TC and LDL levels despite a significant improvement in HDL in response to a long-term training program was not only observed in the present study but also other studies on other nonsmokers patients reported findings consistent with this study. For example, in a recent study, 12 weeks of exercise training as brisk walking by men with metabolic syndrome did not change the levels of TG, TC and LDL while HDL levels increased significantly and the CRP levels as another cardiovascular risk factor tended to reduce [22]. In another study, 6 months of exercise combined with diet control did not affect LDL levels in men with metabolic syndrome [23]. No change in cardiovascular risk factors following long-term training programs was reported by some other studies [16]. Despite the said contradictory findings, the non-change of cardiovascular risk factors in response to the training program by male smokers in this study may be attributed to their smoking during the training course. In other words, it seems that smoking during exercise training neutralizes the positive effects of exercise program.

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