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***Research Article***

# SINGLE BOUT MODERATE EXERCISE IS NOT ASSOCIATED WITH ACUTE OR RECOVERY RESPONSE OF CARDIOVASCULAR RISK FACTOR IN ADULT MEN

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## ABSTRACT

It is widely accepted that Obesity is a major health problem and is associated with cardiovascular risk factor and metabolic disorders. This study aimed to determine acute and recovery response of lipid profile markers as cardiovascular risk factors in obese and normal weight men. Subjects was healthy adult obese men (n=15, BMI ≥ 30) and normal weight adult men (n=15, 25 < BMI < 30) matched for age that participated in study by voluntary. Blood samples were collected before and 0, 1 and 24 hours after one bout exercise included 40 min running with moderate intensity of two groups. Data analyzed by (ANOVA) for repeated measures, for continuous variables. Data of independent T test showed that serum triglyceride (TG), total cholesterol (TC) and Low density lipoprotein cholesterol (LDL) was higher in obese group than normal weight subject at baseline, although there was not significant difference in high density lipoprotein cholesterol (HDL) between two groups. No differences were observed in all variables between pre values and acute or recovery response to exercise test in two groups. These data showed that one moderated running test can not affect cardiovascular risk factors immediately and one day's recovery in either obese or normal weight subjects.

***Keywords:*** *Lipid Profile, Body Weight, Acute Exercise*

## INTRODUCTION

The prevalence of overweight and obesity in adults in developed countries is more than 35%. The incidence of obesity is increasing in developing countries as well. Among preventable diseases, death caused by obesity is the second-rate fatal disease after death caused by tobacco. World Health Organization addressed the rapid increase in prevalence of obesity, which was introduced as an epidemic disease. Obesity and the relevant complications are considered as one major global health problem (Diabetes Prevention Program Research Group, 2002).

Effective mechanism in positive energy balance, such as the balance between inflammation and release of several adipocytokine, determine pathophysiology of metabolic abnormalities in the diseases associated with obesity (Alexandraki *et al.*, 2006; Boulet, 2008). Furthermore, impaired secretion and systemic levels of these inflammatory markers are also associated with lipidemic disorders, particularly in obese patients (Fasshauer *et al.*, 2003). Physical activity is introduced as a factor modifying obesity risk factors such as TG, TC, HDL, and LDL. Then, physical activity is addressed as an effective method to improve inflammation. Longitudinal studies have shown that regular exercise has anti-inflammatory effects, which leads to decreased levels of inflammatory markers and improved lipid profile parameters (TG, TC, HDL, LDL) (Moschen *et al.*, 2010). In a recent study, 7-month training exercise increased VO2max and HDL. It was also reported that 7-month training reduced weight, body fat, TNF-a, CRP in obese individuals (Kondo *et al.*, 2006). However, several researchers concluded that exercise is not necessarily associated

with changes in lipid profile markers based on their findings. Furthermore, they reported no changes in these variables in terms of training programs (Durstine *et al*., 2002).

However, despite conflicting findings on response of this inflammatory cytokine to short or long-term training activities, immediate or delayed response of the biochemical variables associated with obesity, such as lipid profile parameters was less assessed. It is not identified whether lipid profile parameters response to one training session is immediate or delayed. There is no consensus in this regard. Hence, the present study not only compared the baseline levels of these variables between obese and normal weight individuals, but also determined the immediate and delayed response of these biochemical variables to an exercise session.

## MATERIALS AND METHODS

**Study population:** Fifteen sedentary, healthy adult (35 – 45 year of old) obese (BMI: 30–36 kg/m2) or normal weight (BMI: 25-30 kg/m2) were recruited through an accessible sampling in present study. All subjects were healthy without history of chronic disease such as arthritis, stroke, diabetes, hypertension, cancer, heart attack, chronic cough, or bronchitis. All participants had not participated in regular exercise/diet programs for the preceding 6 months. Exclusion criteria also included medications or supplementations that alter carbohydrate-fat metabolism and inability to exercise. After introduction and awareness of the subjects of the objectives of the study and once they had completed consent forms, the process of test implementation began.

### *Anthropometric Measurements*

Body weight, height, waist circumference and body fat (%) measurements were obtained by standard methods. Weight and Percentage body fat was measured using body composition monitor (OMRON, Finland). Height of the barefoot subjects was measured to the nearest 0.1 cm. BMI was calculated as weight (kilograms) divided by height squared (square meters). Waist and hip circumferences were measured and a waist-to-hip ratio (WHR) was calculated. Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter.

### *Laboratory Measurements and Intervention*

Exercise test lasted min included running on a flat surface without slope at 65-70 (%) of maximal heart rate. Venous blood samples (5 ml) were collected before, immediately, 1 and 24 hours at end of exercise test. Blood used to determine serum TC, TG, LDL and HDL of two groups. The blood was set into a centrifuge tube and allowed to clot to obtain the serum. Triglyceride, total cholesterol, HDL and LDL-cholesterol was measured directly with enzymatic methods (Randox direct kits) using Kobas Mira auto-analyzer made in Germany.

### *Data Collection*

Statistical analysis was performed with the SPSS software version 15.0. Normality of distribution was assessed by Kolmogorov-Smirnov test. The statistical significance of deference's between the means in the two groups at baseline were evaluated using An Independent sample T-test. Experimental data are presented as means ± SE and were analyzed by two-way analysis of variance with repeated measures over time.

## RESULTS

Table 1 show the descriptive anthropometric and biochemical features of the study groups. At baseline, serum TG, TC and LDL levels were significantly higher in obese subject in comparison to normal weight subjects. But there was no marked difference in serum HDL between obese and none-obese subjects. Acute and recovery response of TC, TG, LDL and LDL to exercise test did not significant change when compared to presets in normal weight (Figure1) and Obese (Figure2) subjects.

**Table 1: Mean and standard deviation of anthropometrical and biochemical features of the study groups**

**Descriptive Statistics of Normal subjects**

| | Mean | Std. Deviation |
|---|---|---|
| Age | 37.93 | 1.280 |
| Height | 171.47 | 2.167 |
| Weight | 67.53 | 1.995 |
| Abdominal 1 | 87.47 | 2.924 |
| Hip | 94.80 | 3.005 |
| WHO | .9237 | .04606 |
| BMI | 22.9660 | .30387 |
| %fat | 21.917 | .9880 |
| Visceral Fat | 7.53 | .990 |
| Total Cholesterol 1 | 170.20 | 25.755 |
| Total Cholesterol 2 | 178.20 | 45.806 |
| Total Cholesterol 3 | 175.53 | 36.492 |
| Total Cholestrol 4 | 165.93 | 28.972 |
| Triglyceride 1 | 132.29 | 63.934 |
| Triglyceride 2 | 145.80 | 66.538 |
| Triglyceride 3 | 141.27 | 70.096 |
| Triglyceride 4 | 127.20 | 66.758 |
| LDL 1 | 88.65 | 25.922 |
| LDL 2 | 99.20 | 13.702 |
| LDL 3 | 97.67 | 12.292 |
| LDL 4 | 90.27 | 13.231 |
| HDL 1 | 48.07 | 4.590 |
| HDL 2 | 49.27 | 4.267 |
| HDL 3 | 49.47 | 4.882 |
| HDL 4 | 47.93 | 2.764 |

**Descriptive Statistics of Obese subjects**

| | Mean | Std. Deviation |
|---|---|---|
| Age | 39.67 | 2.289 |
| Height | 173.13 | 4.257 |
| Weight | 93.80 | 6.951 |
| Abdominal 1 | 104.20 | 4.329 |
| Hip | 105.20 | 5.759 |
| WHO | .9915 | .03081 |
| BMI | 31.25 | 1.315 |
| %fat | 32.53 | 1.648 |
| Visceral Fat | 13.40 | 2.324 |
| Total Cholesterol 1 | 214.27 | 24.878 |
| Total Cholesterol 2 | 216.47 | 35.855 |
| Total Cholesterol 3 | 222.73 | 31.001 |
| Total Cholestrol 4 | 210.47 | 27.537 |
| Triglyceride 1 | 195.73 | 48.985 |
| Triglyceride 2 | 204.13 | 51.357 |
| Triglyceride 3 | 193.27 | 49.852 |
| Triglyceride 4 | 188.60 | 49.126 |
| LDL 1 | 129.47 | 16.466 |
| LDL 2 | 133.47 | 16.225 |
| LDL 3 | 132.93 | 15.323 |
| LDL 4 | 130.00 | 15.133 |
| HDL 1 | 47.73 | 2.282 |
| HDL 2 | 47.87 | 9.125 |
| HDL 3 | 48.73 | 3.535 |
| HDL 4 | 47.80 | 2.883 |

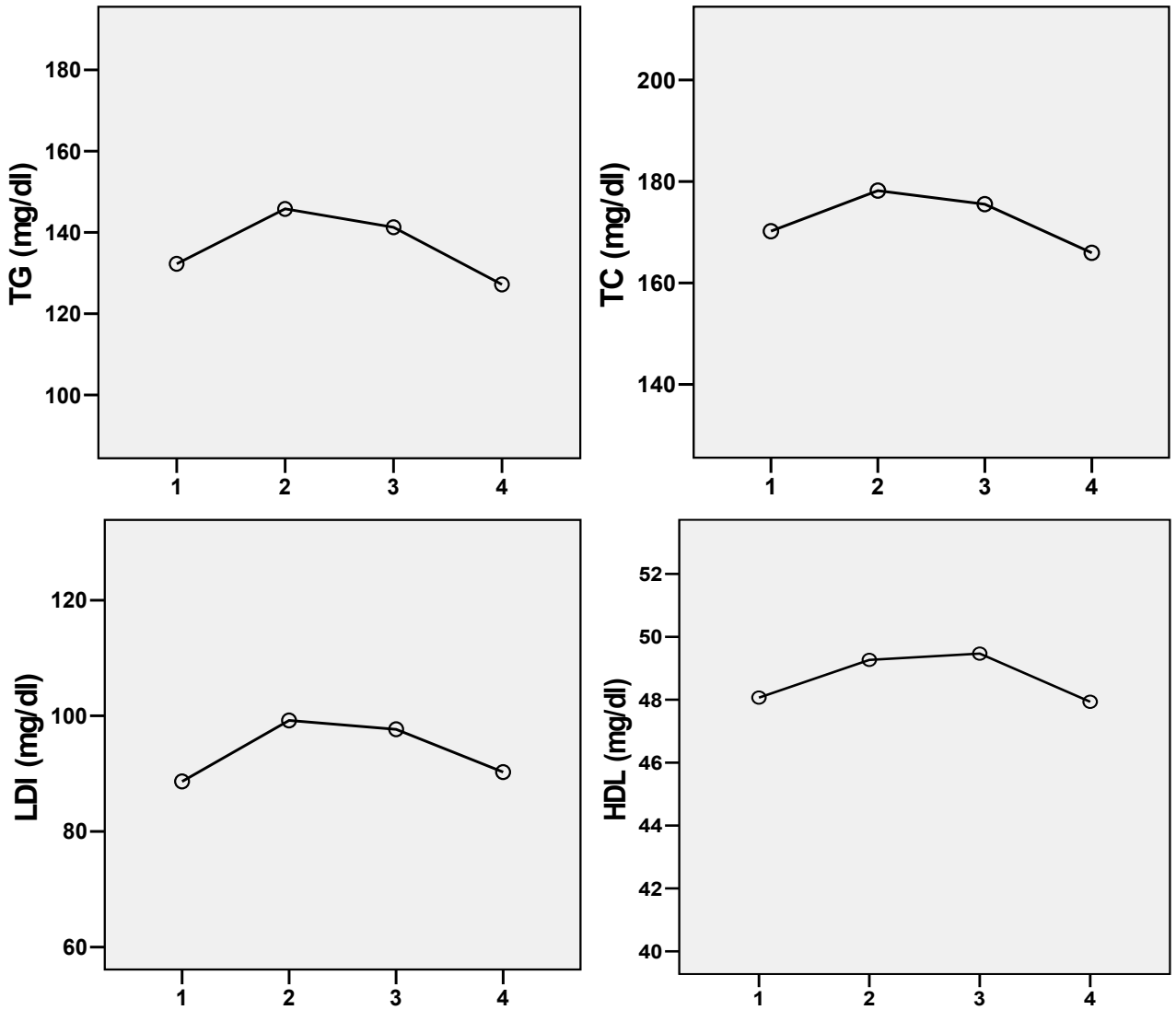


**Figure 1: The change pattern of acute and recovery response of Lipid profile markers to exercise test in normal-weight subjects. No significant difference in all measurement compared to baseline. Numbers on the horizontal axis (1: blood samples before exercise, 2: immediately after exercise, 3: I hours recovery, 4: 24 hours recovery)**

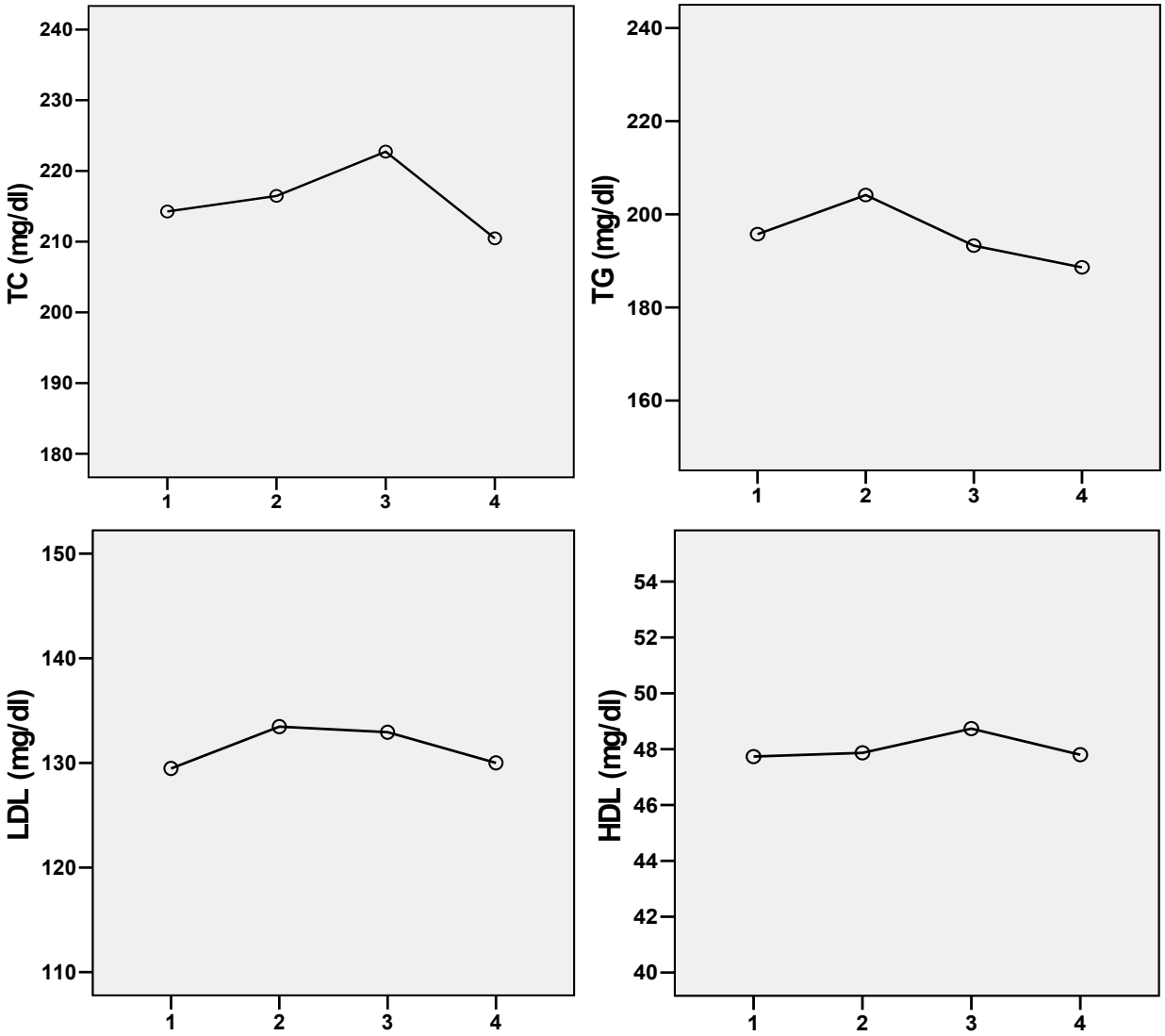


**Figure 2: The change pattern of acute and recovery response of Lipid profile markers to exercise test in obese subjects. No significant difference in all measurement compared to baseline. Numbers on the horizontal axis (1: blood samples before exercise, 2: immediately after exercise, 3: I hours recovery, 4: 24 hours recovery)**

## DISCUSSION

Although the immediate or delayed response of these variables to an exercise session was less studied, a training test did not significantly change the levels of lipid profile parameters in obese and normal weight men in the present study. However, these findings showed that normal weight men had lower levels of triglycerides, total cholesterol and low-density lipoprotein compared to obese men. Although high-density lipoprotein levels were similar in both groups, the two groups had significantly different weight and fat percentage. This issue is relatively controversial.

Consistent with these results, two exercise tests (one moderate intensity strength exercise and one submaximal aerobic exercise) did not change the levels of lipid profile parameters in students in another study (Sevgi *et al.,* 2009). However, contrary to these findings, other findings showed that 30 minutes exercise in the form of cycling increased HDL-C by 10% and 24% respectively at 10 and 24 hours after the exercise (Kumar *et al.,* 2003). In another study, Berg *et al* showed a slight increase in HDL levels immediately after 30 minutes desert running (Berg *et al.,* 1983). Pi *et al* also concluded that long-term walking is associated with immediate changes in lipoprotein levels in both healthy adult and young men (Pay, 1991). The exact mechanisms linking exercise to increased HDL are not known yet. Exercise increases energy costs and promotes those catabolic processes, which change phosphorylation state of the regulatory enzymes involved in both synthesis and degradation of cholesterol or lipoprotein. It should be noted that HDL levels increase by exercise or even an exercise session (Kumar *et al.,* 2003). In another study, no immediate change in HDL immediately after marathon running was reported in women. However, increased delay in HDL levels at 24 h after exercise was observed in the former study (Goodyear, 1990). Overall, increased energy consumption due to exercise changes fat and carbohydrate storage or transmission. Exercise can change enzymatic activity of lipoprotein lipase, hepatic lipase or lecithin cholesterol acetyltransferase, which are involved in metabolism of plasma triglyceride (Kumar *et al.,* 2003).

Although these findings confirmed improved lipid profile parameters in response to one training session, the findings obtained from this study showed that there are no significant changes in both immediate and delayed levels of lipid profile parameters after 40 minutes moderate-intensity running in both obese and normal-weight men. In this regard, clinical studies have shown that exercise sometimes slightly changes or do not change blood HDL levels (Stensvold *et al.,* 2010; Sillanpää *et al.,* 2009; ). No change in HDL and other lipid profile parameters in response to exercise were also reported in several recent studies (Romero *et al.,* 2013). On the other hand, a significant increase in HDL levels in response to one exercise session may be attributed to a decrease in plasma volume because several researchers showed that one exercise session is associated with decreased plasma volume. This supports increased HDL due to reduced plasma volume observed in several studies (Van *et al.,* 1972). Although most believe that exercise activities are associated with reduced blood cholesterol, this hypothesis is not thoroughly confirmed. Exercise activities do not necessarily decrease cholesterol. Most studies reported that blood cholesterol levels are not reduced through exercise (Durstine *et al.,* 2002). However, exercise along with dietary modification lower blood cholesterol.

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