

## Effect of Various Intensities of Circuit Resistance Training on Plasma Levels of High-Density Lipoprotein-Associated Apolipoprotein O, Total Cholesterol and Triglyceride in Untrained Men

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### ABSTRACT

**Background and Objectives:** Apolipoprotein O (apoO) is a 198 amino acids protein that exists predominantly in high-density lipoprotein (HDL). It may exert cardioprotective effects via decreasing fat accumulation and increasing removal of cholesterol from macrophages. Although the health benefits of exercise are well documented, no study has yet investigated the effects of various types of training, including resistance training on apoO level. Therefore, we aimed to determine effects of five weeks of circuit resistance training with different intensities on plasma levels of apoO, HDL, total cholesterol (TC) and triglyceride (TG) in young untrained men.

**Methods:** Forty-five age- and weight-matched healthy untrained men were randomly assigned to a control group (n=10) and four training groups: training at 20% intensity (n=9), training at 40% intensity (n=8), training at 60% intensity (n=7) and training at 80% intensity (n=8). The subjects performed circuit resistance training at 10 stations (30 seconds at each station) with three repetitions, without rest between stations and with 3-minute active rest between the repetitions. The training was carried out for 45 minutes per session, three sessions a week, for five weeks. Venous blood samples were taken 48 hours before the first exercise session and 48 hours after the last training session. Plasma levels of apoO, HDL, TC and TG were measured using commercial kits. Data were analyzed using repeated measures ANOVA and Tukey's post hoc test at significance level of 0.05.

**Results:** After the training intervention, mean plasma level of TC and TG did not differ significantly between the study groups ( $P>0.05$ ). Training at 80% of one-repetition maximum (1RM) caused a slight decrease in the apoO concentrations. Moreover, apoO concentration was significantly higher in the 20% 1RM training group compared to other study groups ( $F=11.599$ ,  $P<0.002$ ).

**Conclusion:** Our results indicate that circuit resistance training at 80% of 1RM can decrease HDL-associated apoO level but does not significantly alter other parameters.

**KEYWORDS:** Circuit resistance training, HDL-O, TC, TG, Young men.

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## INTRODUCTION

Coronary artery disease is one of the leading causes of death in both developed and developing countries. Increased levels of total cholesterol (TC), low-density lipoprotein (LDL) and triglyceride (TG) as well as lowered amount of high-density lipoprotein-cholesterol (HDL-C) are strong independent markers for coronary artery disease.

Studies have shown that resistance training can change protein components of lipoproteins, such as apolipoprotein O (apoO). ApoO is mainly found in HDL but can be also present in LDL and VLDL in low amounts. This apolipoprotein can exert cardioprotective effects by decreasing fat accumulation and increasing the removal of cholesterol from macrophage cells (1).

Alteration of lipids and apoO levels are considered risk factors of cardiovascular disease. Minor structural or functional alteration of apoO can cause major problems in metabolism of lipids (2).

Regular physical activity is one of the best ways to improve lifestyle and reduce risk of cardiovascular disease (3,4). The effects of physical activity on lipid and lipoprotein levels are well-established (5). Resistance training increases muscle strength, muscle mass, free fatty acids metabolism and energy expenditure. Therefore, it can be effective in improving the metabolic risk factors associated with cardiovascular disease (6). Previous studies indicated that resistance training could lower hepatic and circulating

lipid levels (7). In ovariectomized rats, 12 weeks of resistance training decreased liver and muscle lipid levels and improved blood lipid profiles (8). In addition, eight weeks of resistance training improved lipid profiles in rats with a normal or high-fat diet (9). Despite these findings, there is little known about the effects of resistance training on apoO and HDL levels. Therefore, we aimed to investigate effects of five weeks of resistance training on levels of apoO, HDL-C, TC and TG in untrained young men.

## MATERIALS AND METHODS

In this quasi-experimental study, 45 non-active male medical students (average age:  $19.52 \pm 0.96$  years, average weight:  $16.40 \pm 78.52$  Kg) from the Golestan University of Medical Sciences (Gorgan, Iran) were enrolled. Prior to participation, all procedures and research objectives were explained to the subjects and written consent was obtained. Inclusion criteria included lack of regular exercise for at least six months and no history of chronic diseases (liver, diabetes, kidney and cardiovascular disease), no history of drug/alcohol addiction and lack of physical disability. After homogenization based on individual characteristics, especially total strength, the subjects were randomly assigned to five groups of control (n=10), training at 20% intensity (n=9), training at 40% intensity (n=8), training at 60% intensity (n=7) and training at 80% intensity (n=8) (Table 1).

Table 1- Characteristics of the subjects in each study group

Group	Control (n=10)	Training at 20% intensity (n=9)	Training at 40% intensity (n=8)	Training at 60% intensity (n=7)	Training at 80% intensity (n=8)
	Mean $\pm$ standard error	Mean $\pm$ standard error	Mean $\pm$ standard error	Mean $\pm$ standard error	Mean $\pm$ standard error
<b>Variable</b>					
Age (years)	20 $\pm$ 1	19 $\pm$ 1	19 $\pm$ 1	19 $\pm$ 1	18 $\pm$ 1
Height (Cm)	177 $\pm$ 8	178 $\pm$ 3	176 $\pm$ 6	183 $\pm$ 7	181 $\pm$ 4
Weight (Kg)	76 $\pm$ 17	77 $\pm$ 20	76 $\pm$ 16	81 $\pm$ 13	81 $\pm$ 15
BMI (Kg/m <sup>2</sup> )	24 $\pm$ 4	24 $\pm$ 6	24 $\pm$ 3	24 $\pm$ 2	24 $\pm$ 5
Back extensions (Kg)	24 $\pm$ 5	21 $\pm$ 5	18 $\pm$ 6	18 $\pm$ 10	18 $\pm$ 8
Abdomen (Kg)	28 $\pm$ 6	22 $\pm$ 8	25 $\pm$ 6	24 $\pm$ 7	21 $\pm$ 10
Back arm (Kg)	42 $\pm$ 7	31 $\pm$ 5	37 $\pm$ 7	44 $\pm$ 15	35 $\pm$ 6
Barbell Bench Press (Kg)	30 $\pm$ 6	26 $\pm$ 11	28 $\pm$ 10	33 $\pm$ 11	24 $\pm$ 10
leg Squat (Kg)	57 $\pm$ 11	54 $\pm$ 15	53 $\pm$ 12	64 $\pm$ 14	54 $\pm$ 11
Leg (Kg)	95 $\pm$ 35	145 $\pm$ 209	61 $\pm$ 14	63 $\pm$ 12	61 $\pm$ 11
Front arm (Kg)	16 $\pm$ 3	13 $\pm$ 5	11 $\pm$ 5	14 $\pm$ 6	11 $\pm$ 6
Back foot (Kg)	21 $\pm$ 2	23 $\pm$ 9	16 $\pm$ 5	19 $\pm$ 2	18 $\pm$ 5
Front leg (Kg)	34 $\pm$ 7	36 $\pm$ 12	31 $\pm$ 12	38 $\pm$ 4	35 $\pm$ 10
Arm pit (Kg)	49 $\pm$ 6	47 $\pm$ 8	44 $\pm$ 5	48 $\pm$ 6	44 $\pm$ 6

Our training protocol was based on a protocol designed by Abbas Ghanbari and Abolfazl Aghababaiyan (10). The subjects first became familiar with the training environment and equipment. Value of one-repetition maximum (1RM) of the intended movements (bench press, seated cable row, arm cable curl, triceps cable curl, lying leg curl, barbell squat, lumbar extension, abdominal, decline sit-up, quadriceps) was calculated using the following formula and through trial and error:

$$1rm = \frac{\text{Displaced weight}}{\frac{95}{0} - (\text{repid2}) \times 25\%}$$

After warm up, the subjects performed the movements in 10 stations at different intensities (20, 40, 60 and 80%) of 1RM for 30 seconds without rest between the stations. The trainings were performed at medium speed and finished with cool down. The exercises were performed in three sets with 3-minute active rest between each set. The heart rate of the subjects was assessed within 6 seconds immediately after completing each set. Fasting blood samples (10 cc) were taken 48 hours before the first training session and 48 hours after the last training session in sitting position. The samples were collected in EDTA-coated tubes. After centrifugation at 1500 g for 15 minutes, supernatant was transferred to a separate tube and kept at -70

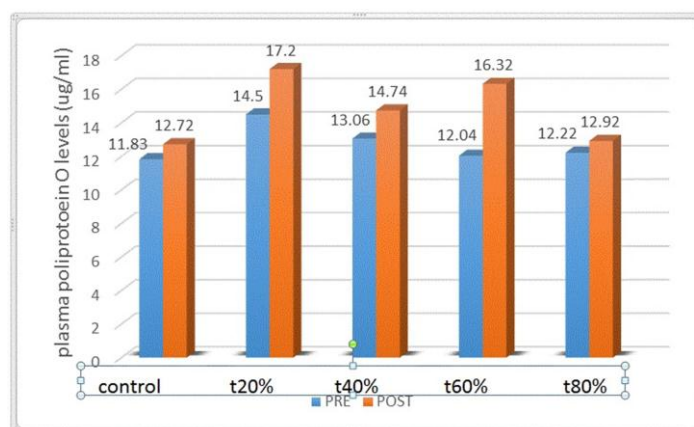
°C. ApoO level was measured using commercial ELISA kits. HDL-C was measured by photometric method (Pars Test, Tehran, Iran) with coefficient of variation and sensitivity of 0.81% and 1 mg/ml. Level of TC and TG was determined by enzymatic (CHOD-PAP) calorimetric method (Pars Test Tehran, Iran), respectively. Coefficient of variation and sensitivity of the method was 61% and 5 mg/ml for TC and 1.04% and 5 mg/ml for TG, respectively. All mentioned factors were measured using the Prestige 24i chemistry analyzer (Japan).

Normality of data was assessed using the Kolmogorov-Smirnov test. Data analysis was performed with SPSS 16 using one-way ANOVA, two-way repeated measures ANOVA Tukey's test at significance of 0.05.

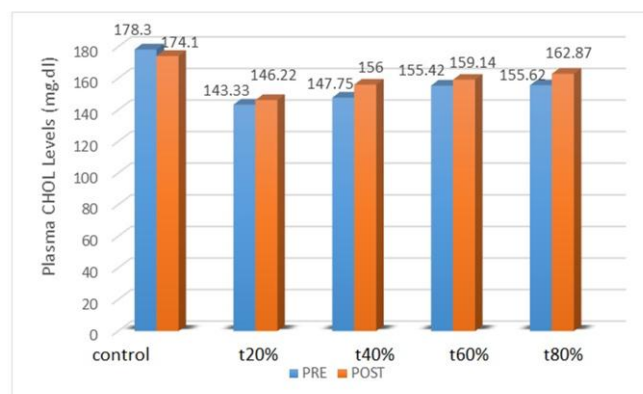
## RESULTS

The five-week training program at all intensities caused a significant increase in HDL-associated apoO level. Although plasma apoO increased in all training groups, this increase was statistically significant only in the 20% 1RM group ( $F=11.599$ ,  $P<0.002$ , Figure.1). The training at various intensities caused no significant change in the plasma concentrations of TC and TG (Figures 2 and 3).

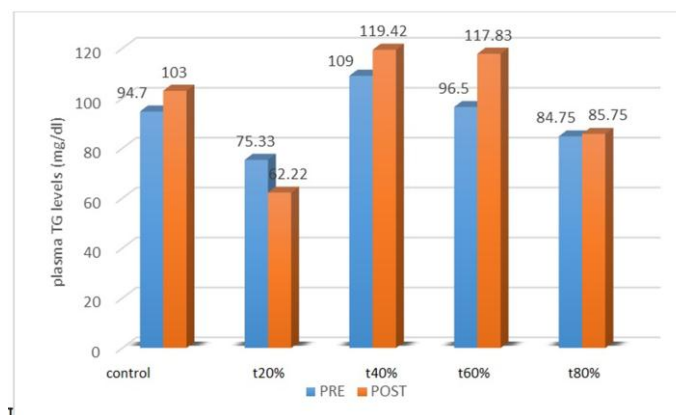
Figure 1- Mean plasma concentration of apoO in different groups after five weeks of circuit resistance training



**Figure 2- Mean plasma concentrations of TC in different groups after five weeks of circuit resistance training**



**Figure 3- Mean plasma concentration of TG in different groups after five weeks of circuit resistance training**



## DISCUSSION

The results of this study showed that performing five weeks of circuit resistance training at different intensities can affect some of the cardiovascular risk factors (11). Although much research has been done on the impact of endurance and strength training on lipoprotein and lipid levels (12-14), there is little known about the effects of circuit resistance training on apoO and plasma lipid profile. In this study, the five-week resistance training protocol caused a significant increase in apoO level in all training groups but did not significantly alter other tested parameters. There was no significant difference in plasma TC levels between the study groups. This finding is in line with findings of Turkman et al. (15) and Yektayar et al. (16).

Plasma TG levels did not differ significantly between the study groups, which were consistent with some studies (17, 18) but contrary to the results of some other studies (19,20). Exercise increases lipoprotein lipase (LPL) and decreases hepatic lipase (HL)

activity. Considering that the increased activity of LPL increases catabolism of TG-rich lipoproteins, it is expected to observe TG rise after physical activity (21). In addition, regular exercise can inhibit HL activity (22), which could decrease the TG content of VLDL and LDL.

Enzymes such as LPL, hepatic TG lipase and cholesteryl ester transfer protein play an important role in alteration of HDL concentrations. Plasma LPL changes lipoprotein concentration through hydrolysis of TG. However, increased lipoprotein levels immediately after exercise is not due to LPL activity but possibly related to a decrease in the concentration of cholesteryl ester transfer proteins, which are responsible for carrying fats in HDL molecules and other lipoproteins. This in turn slows down lipoprotein catabolism (half-life) and ultimately increases lipoprotein concentrations (23). Given that adipose tissues are supplied with capillaries and innervated by sympathetic nerve fibers, which can regulate

lipid metabolism, one cannot consider a single mechanism responsible for alteration of lipid parameters. Another important cause of lipolysis is stimulation of beta-adrenergic receptors in adipose tissue. Increased sympathetic nervous system activity and immediate release of both epinephrine and norepinephrine after exercise lead to lipolysis (24). In a previous study (unpublished), we investigated effects of four weeks of circuit resistance training and supplementation with milk, whey cheese and protein whey on plasma HDL-apoO levels in high school students. The results showed that apoO level decreased significantly at the end of the study in all groups except for the milk supplementation group. However, apoO level did not differ significantly between the study groups (25).

Similar to apolipoprotein B, apoO secretion has a microsomal triglyceride transfer protein (MTP)-dependent mechanism. In fact, apoO is secreted in VLDL and then transferred to HDL. Moreover, apoO expression is significantly associated with MTP expression (1). It has been reported that patients with acute coronary syndrome has twice as high apoO level than healthy individuals (26). The decrease in HDL-apoO level could be attributed to a possible down-regulation effect

on MTP expression in the liver since a study has shown that resistance training (treadmill running) decreases MTP mRNA levels in ovariectomized or non-ovariectomized rats treated with estradiol (27, 28). The decrease in HDL-apoO level observed in our subjects might be due to hepatic TG-rich VLDL secretion. Given that administration of sex hormones can affect apoO level (25) and the increase in apoO level was relatively higher in the training groups than in the control group, it can be inferred that training-induced changes in the sex hormones may have contributed to the apoO alteration. Considering the lack of enough studies in this regard, it is not yet possible to propose an exact mechanism for the decreased HDL-apoO level following resistance training. Nevertheless, the effects observed in the training groups could also reflect the relative cardioprotective effects of the training protocol used in the present study.

## CONCLUSION

Our results indicate that resistance training may reduce risk of developing cardiovascular disease.

## CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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