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Comparison the effect of 8 weeks endurance versus resistance exercise on sdLDL concentration in military men

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Abstract

Introduction: Small dense low-density lipoprotein-cholesterol (sdLDL) is an emerging biomarker associated with cardiovascular disease and several comorbidities. The effects of aerobic versus resistance exercise on sdLDL levels are not well known; thus the aim of present study was to investigate effect of 8 weeks aerobic *vs.* resistance exercise on sdLDL concentration in military men.

Material & Methods: Twenty two military men volunteered to participate in this study. The subjects were divided into endurance (n=11) or resistance (n=11) exercise group randomly. The subjects in the endurance exercise group were performed 8 weeks aerobic exercise with 60 to 75% of their hear rate reserve 3 days a week, while the subjects in resistance exercise group were performed resistance training

consisted of eight exercises (chest press, triceps extension, latissimus pull down, shoulder press, arm curls, leg extension, leg curls, and curl-up) of 6-12 maximal repetitions with 3 sets at 65-80% of 1RM for 8 weeks. Blood samples were taken at baseline and 48h after the last session of exercise.

Results: The results showed that sdLDL, TC, TG and LDL decreased and HDL increased only after endurance training. Data also revealed that there were significant differences in changes of sdLDL, TC, LDL and HDL concentration between endurance and resistance exercise.

Conclusions: The results suggest endurance exercise is better than resistance exercise to reduce sdLDL, TC, LDL and increase HDL concentration in the military men.

Keywords: Endurance exercise, Resistance exercise, Cardiovascular disease, sdLDL

1. Introduction

It has been well recognized that the low-density lipoprotein (LDL) is one of the major risk factors of coronary artery disease (1). According to recent guidelines, achieving lower levels of LDL for patients with very high risk has been highly recommended (2,3). However, among patients who reached the appropriate LDL targets, the risk of incident cardiovascular events remains high (4). In fact, LDL particles are heterogeneous in size and density and can be categorized into large, buoyant LDL (lbLDL) and small, dense LDL (sdLDL) (5). A study performed by Hoogeveen et al. (2014) indicated that the sdLDL but not lbLDL was positively associated with the presence of coronary artery disease in Atherosclerosis Risk in Communities (ARIC) study (6).

In hypertriglyceridemic states, excess triglyceride (TG) in LDL is hydrolysised by hepatic lipase and converts to sdLDL (7). Compared with lbLDL, the particles of sdLDL are more atherogenic. The particles of sdLDL have smaller size, higher penetration into the arterial wall, lower binding affinity for the LDL receptor, longer plasma residence time, and increased susceptibility to oxidation (8-11). In some studies on

the relationship between lipid and other coronary artery disease risk markers, data suggested that sdLDL was more significantly associated with inflammatory markers than LDL (12). In the cross-sectional study by Koba and his colleague, sdLDL levels were more powerful than LDL-C levels for the determination of CAD severity (13). This suggests that the atherogenicity of LDL differs among heterogeneous LDL particles and that the association of atherogenic LDL with coronary heart disease is chiefly due to the sd-LDL component (13,14).

Despite a number of scientific evidence supporting the benefits of physical exercises for prevention and management of cardiovascular diseases (15-17), recently Nayeri khoob and Moghadasi (2017) indicated hat sdLDL level was increased after a bout of heavy resistance exercise; however it decreased after 8 weeks regular resistance training (18). Medlow et al. (2016) also reported that an acute bout of moderate intensity exercise can increase sdLDL oxidation potential, independently of age and regardless of a change in selective LDL lipid components in healthy men (19). Intervention studies have also shown the benefits of physical exercise in decreasing sdLDL concentrations (20-22). In a 6-month intervention study, Kotani et al. (2012) studied 30 hyperlipidemic subjects (12 male/18 female subjects, mean age 64 years) and demonstrated a beneficial influence of moderate physical activity on the modulation of LDL particle sizes and oxidative stress status (20). The mean LDL particle size was significantly larger in the post-intervention than in the pre-intervention evaluation (26.9 *vs.* 27.1 mg/dL, $p < 0.01$), suggesting a reduction in sdLDL-C concentrations (20). Dutheil et al. (2014) recruited 100 participants (43 males, 57 females, mean age 59 years, 91.4 kg, BMI 33.4 kg/m^2) with metabolic syndrome from the RESOLVE trial into a one-year study commencing with a three-week residential program combining high exercise volume (15-20 h/week), a restrictive diet (-500 kcal/day) and education; sdLDL-C concentrations markedly decreased after the 3-week residential program (21). Yu et al. (1999), also noted that sdLDL particles decreased significantly by 62% after the triathlon in highly trained athletes (22). The effects of aerobic and resistance exercise on sdLDL levels are not well known; thus the aim of present study was to investigate effect of 8 weeks aerobic *vs.* resistance exercise on sdLDL concentration in military men.

2. Material & Methods

Subjects

Twenty two healthy military men with a mean (±SD) age of 23.7 ± 3.3 years enrolled and volunteered to participate in this study. All the participants were asked to complete a personal health and medical history questionnaire, which served as a screening tool. All the subjects were nonsmokers and free from unstable chronic condition including dementia, retinal hemorrhage, and detachment; and they had no history of myocardial infarction, stroke, cancer, dialysis, restraining orthopedic or neuromuscular diseases. Thereafter, the subjects were randomly assigned to a endurance group (n=11) or resistance group (n=11). The ethic committee of the Islamic Azad University, Marvdasht branch approved the study method and protocol.

Endurance exercise protocol

The 8 weeks endurance exercise training program included 3 training sessions per week. During the 8 weeks intervention, the subjects were trained for 30-45 min per session at a heart rate corresponding to 60-75% individual heart rate reserve (HRR). Each participant was equipped with a heart rate monitor (Polar, FS3c, Finland) to ensure accuracy of the exercise level. Each training session was followed by cool-down.

Resistance exercise protocol

Two familiarization sessions were designed to habituate subjects with the testing procedures and laboratory environment. The main aim of these sessions was to familiarize subjects with different resistance exercises using weight-training machines and also to familiarize them with performing the 1-RM test. Maximal strength was determined using a concentric, 1-RM (23), as previously described (24). The warm-up consisted of running on treadmill for 5 min, two sets of progressive resistance exercises similar to the actual exercises utilized in the main experiment, and 2-3 min of rest accompanied by some light stretching exercises. After the warm-up, subjects performed the 1-RM test, and the heaviest weight that could be lifted once using the correct technique was considered as 1-RM for all the exercises and used to calculate the percentage of resistance. During the familiarization sessions, it was

ensured that all the subjects used the correct techniques for all exercises prior to taking part in the main test sessions. Subjects executed eight resistance exercises selected to stress the major muscle groups in the following order: chest press, triceps extension, latissimus pull down, shoulder press, arm curls, leg extension, leg curls, and curl-up. The subjects were performed resistance training with 65% of 1-RM in 3 sets and 12 maximal repetitions during the first 4 weeks of intervention. 1-RM test was performed again and at the 4 second weeks of intervention, resistance training was performed with 80% of new 1-RM in 3 sets and 6 maximal repetitions. Each training session was followed by cool-down.

Anthropometric and body composition measurements

Height and body mass were measured, and body mass index (BMI) was calculated by dividing body mass (kg) by height (m^2). Waist circumference was determined by obtaining the minimum circumference (narrowest part of the torso, above the umbilicus) and the maximum hip circumference while standing with their heels together. The waist to hip ratio (WHR) was calculated by dividing waist by hip circumference (cm). Body fat percentage was assessed by skinfold thickness protocol. Skinfold thickness was measured sequentially, in triceps, abdominal, and suprailiac by the same investigator using a skinfold caliper (Harpenden, HSK-BI, British Indicators, West Sussex, UK) and a standard technique.

Biochemical analyses

Blood samples were taken at baseline and 48h after the last session of intervention. Blood sample was obtained by venipuncture. sdLDL levels were obtain using following formula that previously excogitated by Srisawasdi et al. (2011) (25):

$$\text{sdLDL (mg/dL)} = 0.580(\text{non-HDL}) + 0.407\ (\text{dLDL}) - 0.719(\text{cLDL}) - 12.05$$

dLDL: Direct low-density lipoprotein-cholesterol
cLDL: Calculated low-density lipoprotein-cholesterol

The levels of TC, TG, HDL, and dLDL were measured on the Siemens Dimension RxL Max by using the Siemens enzymatic methods (Siemens Medical Solution Diagnostics, Tarrytown, NY). For the dLDL-C assay (Siemens Medical Solution Diagnostics), the method uses a reagent 1 containing a detergent that solubilizes only non-LDL particles. The

cholesterol released is consumed by cholesterol esterase and cholesterol oxidase in a non-color forming reaction. The second detergent contained in reagent 2 solubilizes the remaining LDL particles. The soluble LDL is then oxidized by the action of cholesterol esterase and cholesterol oxidase forming cholestenone and hydrogen peroxide. The enzymatic action of peroxidase on hydrogen peroxide in the presence of N, N-bis (4-sulfobutyl)-m-toluidine, disodium salt, and 4-aminoantipyrine generate a colored product. We calculated the cLDL (in mg/dL) by using the Friedewald formula:

$$cLDL = TC - HDL - (TG/5).$$

Statistical Analysis

Results were expressed as the mean ± SD and distributions of all variables were assessed for normality. Paired t-test and independent sample t-test were used to compute mean (± SD) changes in the variables before and after the intervention. The level of significance in all statistical analyses was set at $P \leq 0.05$. Data analyses were performed using SPSS software for windows (version 22, SPSS, Inc., Chicago, IL).

3. Results

Changes in anthropometric variables

Anthropometric and body composition characteristics of the subjects at baseline and after the intervention are presented in Table 1. Before the intervention, there were no significant differences in any of variables among the two groups. Body fat percent ($t = -2.1$, $P = 0.04$) and WHR ($t = -2.7$, $P = 0.01$) decreased after 8 weeks endurance training compared to the resistance training group.

Changes in biochemical variables

Changes in TG, TC, LDL, sdLDL and HDL of the participants are presented in the Table 2. Data revealed that TC ($t = -4.8$, $P = 0.001$), LDL ($t = -3.8$, $P = 0.001$) and sdLDL ($t = -2.1$, $P = 0.04$) decreased and HDL ($t = -2.3$, $P = 0.02$) increased after endurance training compared to the resistance training group. Although TG concentration decreased only after endurance training ($t = -2.8$, $P = 0.16$), but no

significant differences were observed between two types of training (t = 0.9, P = 0.3).

Table 1. Anthropometric and body composition characteristics (mean ± SD) of the subjects

	Endurance training (mean±SD)		Resistance training (mean±SD)	
	Pretraining	Posttraining	Pretraining	Posttraining
Body weight (Kg)	69.4 ± 13.0	68.6 ± 12.2	68.4 ± 9.1	68.5 ± 8.9
BMI (Kg/m^2)	22.2 ± 3.2	22.0 ± 3.0	22.5 ± 3.7	22.5 ± 3.6
Body fat (%)	8.2 ± 2.7	7.9 ± 2.6 [ab]	13.1 ± 9.0	13.0 ± 8.9
WHR	0.83 ± 0.1	0.82 ± 0.1 [ab]	0.89 ± 0.05	0.89 ± 0.05

[a] P<0.01 for between-group differences.

[b] P<0.01, pretraining vs. posttraining values.

Table 2. Biochemical variables (mean ± SD) of the subjects

	Endurance training (mean±SD)		Resistance training (mean±SD)	
	Pretraining	Posttraining	Pretraining	Posttraining
TG (mg/dl)	208.2 ± 97.4	139.6 ± 55.0 [a]	166.8 ± 91.2	132.5 ± 41.0
TC (mg/dl)	188.6 ± 36.2	161.3 ± 24.7 [ab]	158.9 ± 31.3	157.2 ± 27.1
LDL (mg/dl)	109.8 ± 33.4	83.7 ± 29.0 [ab]	95.2 ± 20.7	93.8 ± 26.2
sdLDL (mg/dl)	38.8 ± 11.3	32.5 ± 8.6 [ab]	0.89 ± 0.05	0.89 ± 0.05
HDL (mg/dl)	39.1 ± 11.1	44.0 ± 13.6 [ab]	35.9 ± 4.3	36.1 ± 3.8

[a] P<0.01 for between-group differences.

[b] P<0.01, pretraining vs. posttraining values.

4. Discussion

Our results show positive effects of aerobic endurance exercise on changes in lipid profile, and this is generally consistent with previous reports where authors examined longer training programs. For example, a 6-month study of Dunn et al. (2009) who examined a mixed sample of healthy, sedentary men and women, showed promising results, with significantly positive changes in TC, LDL, and the TC/HDL ratio (26). One of the rare studies exclusively examining females evaluated the effects of 16 weeks of endurance exercise and a significant increase in HDL and decrease in the concentration of triglycerides were reported

(27). In another study with healthy young females, Kyrolainen et al. (2018) evidenced changes in lipid panel indicators as a result of 9-week endurance training (28). More specifically, TG changes were not significant; TC was significantly reduced, while HDL levels significantly increased (28). Amouzad Mahdirejei et al. (2015) studied the effect of endurance and resistance exercise training on PON1 and lipid profile levels in obese men. The authors reported a significant decrease in LDL levels (9.89 %) in response to aerobic training, while cholesterol, triglyceride and HDL levels did not significantly change. As well cholesterol, triglyceride, HDL and LDL levels did not significantly change in resistance training and control groups (29). Although we expected that endurance training would have better effects on lipid profile variables than resistance-training, reports which highlight significant improvements in cardiovascular risk factors as a result of resistance-based physical exercise are not rare. For example, Prabhakaran et al. (1999) studied premenopausal women and reported significant decreases in total cholesterol and LDL cholesterol as a result of 14-week resistance training (30). Similarly, in a study with healthy males, high-resistance training and moderate-intensity resistance physical exercise performed over 6 weeks were equally effective with regard to reductions of LDL, total cholesterol, and the TC/HDL ratio (31). These discrepancies probably relate to differences in duration of intervention studies; sample size or inherent characteristics of the populations. According to involved mechanisms which decrease LDL measure we can say that exercise training performances increase the activity of lipoprotein lipase enzyme and decrease the catabolism of rich-triglyceride lipoprotein. Fat tissues possess numerous capillaries and autonomic nervous system (ANS), so all their metabolic functions are controlled by thyroid, sexual, neural hormones, and we can not account just one reason for increase or decrease of one variant (32). Stimulating beta adrenergic receptor is one of the important reasons that increase lipolise which decreases in aerobic training. Finally, it increases lipolise which leads to an increase in Cholesterol LDL particles and a decrease in the protein level of these particles (33). As a result, LDL particles increases and their density will decrease. Therefore, decrease in LDL particles by exercise training may demonstrate the positive effect of exercise on

cardiovascular system (34). In this research, exercise intensity and time duration may not significantly change HDL in obese people (35). The researchers showed that HDL changes mechanism following exercise is complicated (35). Enzymes such as lipoprotein lipase and cholesterylester transfer protein play a key role to change HDL concentration. Lipoprotein lipase through hydrolysis triglyceride plasma is the most factors to change HDL concentration (36). A slight increase in HDL concentration after exercise period may relate to changes of activity density of cholesterylester transfer protein (CETP). CETP is responsible for transporting fat in HDL molecule and other lipoproteins. A change in CETP is permission for change in HDL catabolism procedure, and finally changes HDL concentration (37). On the other hand, participants' HDL primary level was relatively high and cholesterol and triglyceride levels were normal in this study; may be that is why lipid parameters did not represent different changes (36,37).

The second aim of this study was to investigate the effect of endurance versus resistance exercise on sdLDL concentration in military men. The results of present study indicated that sdLDL decreased only in response to endurance exercise. Nayeri khoob and Moghadasi (2017) indicated hat sdLDL level was increased after a bout of heavy resistance exercise; however it decreased after 8 weeks regular resistance training (18). Medlow et al. (2016) also reported that an acute bout of moderate intensity exercise can increase sdLDL oxidation potential, independently of age and regardless of a change in selective LDL lipid components in healthy men (19). In a 6-month intervention study, Kotani et al. (2012) studied 30 hyperlipidemic subjects and demonstrated a beneficial influence of moderate physical activity on the modulation of LDL particle sizes and oxidative stress status (20). The mean LDL particle size was significantly larger in the post-intervention than in the pre-intervention evaluation, suggesting a reduction in sdLDL-C concentrations (20). Dutheil et al. (2014) recruited 100 participants with metabolic syndrome from the RESOLVE trial into a one-year study commencing with a three-week residential program combining high exercise volume (15-20 h/week), a restrictive diet (-500 kcal/day) and education; sdLDL-C concentrations markedly decreased after the 3-week residential program (21). Yu et al. (1999), also noted that sdLDL

particles decreased significantly by 62% after the triathlon in highly trained athletes (22). These discrepant results may be attributed to differences in study populations and type of the intervention.

Basically, the formation of sdLDL particles may arise through the exchange of cholesterol esters for TG, between LDL and these large VLDL. This action is mediated by CETP, which ultimately produces TG-rich LDL particles, which are then lipolyzed by hepatic triglyceride lipase (HTGL) (38). SdLDL particles may also be generated when excess TG on VLDL are exchanged for cholesterol esters on LDL by CETP, producing TG-rich LDL, which then undergoes lipolysis by HTGL to produce smaller and denser LDL particles (39). In a cross-sectional investigation Zambon et al. (1993) reported that high HTGLa is associated with an increase in sdLDL particles and a decrease in HDL2-C (40). In the Familial Atherosclerosis Treatment Study, treatment with colestipol / lovastatin and colestipol / niacin significantly decreased HTGLa with a concomitant conversion of sdLDL to buoyant LDL, which was the strongest predictor of angiographic regression (41). Alterations in LDL composition associated with training may be mediated by changes in HTGL activity. High HTGL activity has been correlated with increased sdLDL and phenotype B in patients with CHD (42). Although HTGL may not change with a single exercise session (43), training can result in chronic reduction in HTGL activity (44), which may lead to lower concentrations of sdLDL particles.

This study has several limitations that must be mentioned. First, we compared two types of exercise (endurance *vs.* resistance) on sdLDL and the other blood lipid in military men at the beginning and at the end of the study, but did not continuously monitor diets, and this investigation did not include strict control of the participants' diet. Next, we did not observe daily physical activity of military men involved in the investigation, which may also contribute to changes in studied variables. Moreover, the mechanism of effect of exercise on improving lipid profile is related to the enzymatic processes involved in lipid metabolism However, one limitation of the present study was the lack of measurement of these enzymes.

5. Conclusion

The results of this research indicate that endurance training can be used as an effective factor to reduce the risks of cardiovascular disease in military men.

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Conflict of interest: The authors declare that they have no conflict of interest relating to the publication of this manuscript.

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