Journal of Physical Activity and Hormones Vol 1, No. 2, Ser. 2 (June 2017), 039-050

Effect of 8 weeks regular resistance training on attenuation of sdLDL changes after single session of heavy resistance exercise

Hamid Reza Nayeri khoob¹ and Mehrzad Moghadasi^{2*}

Received: 16 April 2017/ Accepted: 10 June 2017

- (1) Department of Exercise physiology, Marvdasht branch, Islamic Azad University, Marvdasht, Iran.
- (2) Department of Exercise physiology, Shiraz branch, Islamic Azad University, Shiraz, Iran
- (*) Associate Professor in Exercise Physiology (PhD) E-mail: mehrzad.moghadasi@gmail.com

Abstract

Introduction: Although heavy exercise can independently increase free radical production that may enhance the susceptibility of LDL to oxidation and create more atherogenic LDL particles such as sdLDL, regular training may attenuate these atherogenic conditions. The aim of present study was to investigate the effect of 8 weeks regular resistance training on attenuation of sdLDL changes after single session of heavy resistance exercise.

Material & Methods: Eleven healthy young men (aged: 26.6 \pm 1.5 years; \pm SD) volunteered to participate in this study. One reparation maximum (1-RM) was measured and the subjects were performed a heavy resistance exercise trial consisted of eight exercises (chest press, triceps extension,

latissimus pull down, shoulder press, arm curls, leg extension, leg curls, and curl-up) of 8 repetitions with 3 sets at 80% of 1RM. Thereafter, the subjects were performed the same 8 stations resistance training in 3 sets with 6-12 maximal repetitions. This training was performed 3 days a week with 65-80% of 1-RM, for 8 weeks. After the 8 weeks intervention, the heavy resistance exercise trial was performed a gain. Blood samples were taken at baseline (1st step), immediately after the first heavy resistance exercise trial (2nd step), 48h after 8 weeks intervention (3rd step) and immediately after the second heavy resistance exercise trial (4th step).

Results: The results showed that sdLDL level was increased after the first heavy resistance exercise trial (P<0.05). After 8 weeks exercise training, sdLDL was decreased compared to 2^{nd} step of blood sampling (P<0.05) and no significant change was observed in sdLDL in this step compare to the baseline. The results indicated that sdLDL level had not significant change after the second heavy resistance exercise trial compare to the 2^{nd} step of blood sampling.

Conclusions: The results suggest regular resistance training with specific intensity and duration utilized in this study, attenuate sdLDL changes after single session of heavy resistance exercise.

Key words: Regular training, sdLDL, Cardiovascular risk factors, Heavy resistance exercise

1. Introduction

Cardiovascular disease (CVD) is a critical, worldwide public health threat (1). Current evidence suggests that in addition to conventional plasma lipoprotein lipid measures, novel measures of plasma lipoprotein subfraction concentration, size, and composition should be considered when evaluating CVD risk, as they may provide a better risk assessment because of their distinct composition and functions (2-4). For example, concentrations of total and small low-density lipoprotein (LDL) particles, large high density lipoprotein (HDL) particles, and large very low density lipoprotein (VLDL) particles better reflect CVD risk than absolute measures of cholesterol concentrations, thus improving the early detection of CVD risk (3-8). In addition, the Adult Treatment Panel (ATP) III report lists small LDL particle concentration as an emerging risk factor and recommends small VLDL particle concentration as a potential target of cholesterol-lowering therapy (7,9-11).

LDL was categorized into subfractions 1 to 7, relative to decreasing size and increasing density. Among them, the small dense subfractions 3 to 7 (sdLDL), not routinely assessed in clinical practice, are presumed to be more atherogenic than larger LDL particles (12,13).

Although it has been reported that acute and high-intensity exercise can increase the susceptibility of LDL to oxidation *in vitro* and create more atherogenic LDL particles such as sdLDL (14), there is a distinct lack of research examining the effect of regular exercise on LDL subfraction. On the other hand, the effect of regular training on acute exercise-induced sdLDL is not clear. Therefore, we evaluated the effect of 8 weeks regular resistance training on attenuation of sdLDL changes after single session of heavy resistance exercise.

2. Material & Methods

Subjects and inclusion criteria

Eleven healthy young men with a mean (\pm SD) body mass index of 22.5 \pm 3.7 kg/m², volunteered to participate in a 8-week intervention. All the subjects were asked to complete a personal health and medical history questionnaire, which served as a screening tool. The subjects were given both verbal and written instructions outlining the experimental procedure, and written informed consent was obtained. Our participants were not engaged in any systematic exercise programs at least 6 months before the study, none of them had any disease or had been consuming any drugs. The study was approved by the Marvdasht branch, Islamic Azad University Ethics Committee.

Exercise Training 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 (15), as previously described (16). 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 a heavy resistance exercise trial consisted of eight exercises of 8 repetitions with 3 sets at 80% of 1-RM. Thereafter, the subjects were performed the same 8 stations 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. At the end of study, the heavy resistance exercise trial was performed as same as before.

Measurements

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) (17). Body fat percentage was assessed by skinfold thickness protocol. Skinfold thickness was measured sequentially, in chest, abdomen, and thigh by the same investigator using a skinfold caliper (Harpenden, HSK-BI, British Indicators, West Sussex, UK) and a standard technique (17).

Biochemical analyses

Blood samples were taken at baseline (1^{st} step) , immediately after the first heavy resistance exercise trial (2^{nd} step) , 48h after 8 weeks intervention (3^{rd} step) and immediately after the second heavy resistance exercise trial (4^{th} step) . Blood sample was obtained by venipuncture. sdLDL levels were obtain using following formula that previously excogitated by Srisawasdi et al. (2011) (18):

sdLDL (mg/dL) = 0.580 (non-HDL) + 0.407 (dLDL) - 0.719(cLDL) - 12.05

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

The levels of TC, TG, HDL, and dLDL-C 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 (4sulfobutyl)-m-toluidine, disodium salt, and 4-aminoantipyrine generates 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 \pm SD and distributions of all variables were assessed for normality using kolmogorov-smirnov test. 1 × 4 Repeated measures ANOVA was used to evaluate time-course change in variables. Post hoc analyses (Bonferroni) were then performed when warranted. Data were analyzed using SPSS software for windows (version 17, SPSS, Inc., Chicago, IL) and the significance level of this study was set at P< 0.05.

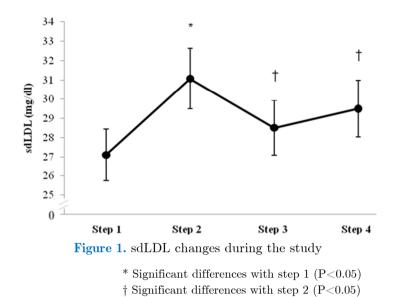
3. Results

Personal characteristics of the subject are presented in the table 1. As shown in the table 1, eleven healthy young men aged 25 to 29 years of old were participated in this study.

Variables	Mean \pm SD
Age (year)	26.6 ± 1.5
Height (cm)	175.6 ± 2.2
Weight (kg)	68.4 ± 9.1
Body mass index (kg/m^2)	22.5 ± 3.7
Body fat (%)	13.1 ± 9.0
WHR	0.89 ± 0.05

Table 1. Personal characteristics of the subject

Changes of sdLDL level during the study were shown in the figure 1. The results indicated that sdLDL level was increased after the first heavy resistance exercise trial (P<0.05). After 8 weeks exercise training, sdLDL was decreased compared to 2^{nd} step of blood sampling (P<0.05) and no significant change was observed in sdLDL in this step compare to the baseline. The results showed that sdLDL level had not significant change after the second heavy resistance exercise trial compare to the baseline and to the 2^{nd} step of blood sampling.



4. Discussion

Even with normal levels of total LDL, cross-sectional studies historically showed a link between sdLDL and CVR (19). This link persisted independently of other lipid parameters (20). Our results indicated hat sdLDL level was increased after the first heavy resistance exercise trial (P<0.05). 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 (21). Yu et al. (1999), however, noted that sdLDL particles decreased significantly by 62% after the triathlon in highly trained athletes. These discrepant results may be attributed to differences in subject populations because our subjects were non-athletes while highly trained athletes were participated in the Yu et al. study.

The decrease of sdLDL after 8 weeks regular resistance training and no significant change after the second heavy resistance exercise trial compare to the baseline suggested that regular resistance training with specific intensity and duration utilized in this study, attenuate sdLDL changes after single session of heavy resistance exercise. Kraus et al. (2002) that examined the effect of 6 months of exercise training on blood lipids and lipoproteins. Results showed significant changes in LDL

subfractions by reducing the concentration of sdLDL particles and increasing their average size (22). Halle and colleagues (1999) also demonstrated that the men with a VO_{2peak} > 50 ml/kg/min had a 25% lower concentration of sdLDL and apo B compared to the other persons (23). Martin (2008) noted that the trained men had significantly less sdLDL particles and a higher concentration of large LDL subfraction particles despite an equal total LDL particle number. Furthermore, the LDL particles of the trained men had a higher free cholesterol content compared to the LDL of the untrained men (24).

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) (25). 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 (26). 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 (27). 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 (28). 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 (29). Although HTGL may not change with a single exercise session (30), training can result in chronic reduction in HTGL activity (31), which may lead to lower concentrations of sdLDL particles.

5. Conclusion

The results suggest that regular resistance training with specific intensity and duration utilized in this study, attenuate sdLDL changes after single session of heavy resistance exercise.

6. Acknowledgment

The work was supported by grants from the Marvdasht branch, Islamic Azad University. The authors gratefully acknowledge the all subjects whom cooperated in this investigation.

Conflict of interests: No conflict of interests amongst authors.

References

- [1] American Heart Association. Heart disease and stroke statistics 2004 update. Dallas (Tex) 7 American Heart Association; 2003.
- [2] Rifai N, Warnick GR, Dominiczak MH. Handbook of lipoprotein testing. 2nd ed, Washington (DC) 7 AACC Press; 2000.
- [3] Cromwell WC, Otvos JD. Low-density lipoprotein particle number and risk for cardiovascular disease. Curr Atheroscler Rep 2004; 6: 381-387.
- [4] Rosenson RS, Otvos JD, Freedman DS. Relations of lipoprotein subclass levels and low-density lipoprotein size to progression of coronary artery disease in the Pravastatin Limitation of Atherosclerosis in the Coronary Arteries (PLAC-I) trial. Am J Cardiol 2002; 90: 89 - 94.
- [5] Shadid S, LaForge R, Otvos JD, Jensen MD. Treatment of obesity with diet/exercise versus pioglitazone has distinct effects on lipoprotein particle size. Atherosclerosis 2006; 188: 370- 376.
- [6] Cheung MC, Brown BG, Wolf AC, Albers JJ. Altered particle size distribution of apolipoprotein A-I-containing lipoproteins in subjects with coronary artery disease. J Lipid Res 1991; 32: 383- 394.
- [7] Kraus WE, Houmard JA, Duscha BD, Knetzger KJ, Wharton MB, McCartney JS, et al. Effects of the amount and intensity of exercise on plasma lipoproteins. N Engl J Med 2002; 347: 1483 - 1492.
- [8] Zilversmit DB. Atherogenic nature of triglycerides, postprandial lipidemia, and triglyceride-rich remnant lipoproteins. Clin Chem 1995; 41: 153 -158.

- [9] Leon AS and Sanchez OA. Response of blood lipids to exercise training alone or combined with dietary intervention. Med Sci Sports Exerc 2001; 33: S502–S515.
- [10] Leon AS, Sanchez O. Meta-analysis of the effects of aerobic exercise training on blood lipids. Circulation 2001; 104 (Suppl II): II-414– II415.
- [11] Durstine JL, Grandjean PW, Cox CA, Thompson PD. Lipids, lipoproteins, and exercise. J Cardiopulm Rehabil 2002; 22: 385- 398.
- [12] Mattsson N, Magnussen CG, Ronnemaa T, Mallat Z, Benessiano J, Jula A, et al. Metabolic syndrome and carotid intima-media thickness in young adults: roles of apolipoprotein B, apolipoprotein A-I, C-reactive protein, and secretory phospholipase A2: the cardiovascular risk in young Finns study. Arterioscler Thromb Vac Biol 2010, 30: 1861-1866.
- [13] Halle M, Berg A, Garwers U, Baumstark MW, Knisel W, Grathwohl D, et al. Influence of 4 weeks' intervention by exercise and diet on lowdensity lipoprotein subfractions in obese men with type 2 diabetes. Metabolism 1999, 48: 641-644.
- [14] Kaikkonen J, Porkkala-Sarataho E, Tuomainen TP, Salonen JT. Exhaustive exercise increases plasma/serum total oxidation resistance in moderately trained men and women, whereas their VLDL + LDL lipoprotein fraction is more susceptible to oxidation. Scand J Clin Lab Invest 2002; 62: 599-607.
- [15] Kraemer WJ, Häkkinen K, Newton RU, Nindl BC, Volek JS, McCormick M, et al. Effects of heavy-resistance training on hormonal response patterns in younger versus older men. J Appl Physiol 1999; 87: 982-992.
- [16] Ahmadizad S and El-Sayed MS. The effects of graded resistance exercise on platelet aggregation and activation. Med Sci Sports Exerc 2003; 35: 1026-1033.
- [17] American Collage of Sport Medicine. Guidelines for exercise testing and prescription. Philadelphia: Lippincott Williams & Wilkins 2005; 57-90.

- [18] Srisawasdi P, Chaloeysup S, Teerajetgul Y, Pocathikorn A, Sukasem C, Vanavanan S, et al. Estimation of plasma small dense LDL cholesterol from classic lipid measures. Am J Clin Pathol 2011; 136: 20-29.
- [19] Coresh J, Kwiterovich PO Jr, Smith HH, Bachorik PS: Association of plasma triglyceride concentration and LDL particle diameter, density, and chemical composition with premature coronary artery disease in men and women. J Lipid Res 1993; 34: 1687–1697.
- [20] Lamarche B, Tchernof A, Moorjani S, Cantin B, Dagenais GR, Lupien PJ, Despres JP: Small, dense low-density lipoprotein particles as a predictor of the risk of ischemic heart disease in men. Prospective results from the Quebec Cardiovascular Study. Circulation 1997; 95: 69–75.
- [21] Medlow P, McEneny J, Murphy MH, Trinick T, Duly E, Davison GW. Lipoprotein subfraction oxidation in acute exercise and ageing. Free Radical Res 2016; 50: 345-353.
- [22] Kraus W, Houmard J, Duscha B, Knetzger K, Wharton M, McCarthy J, et al. Effects of the amount and intensity of exercise on plasma lipoproteins. N Engl J Med 2002; 347: 1483-1492.
- [23] Halle M, Berg A, Garwers U, Baumstark MW, Knisel W, Grathwohl D, Konig D, Keul J. Influence of 4 weeks' intervention by exercise and diet on low-density lipoprotein subfractions in obese men with type 2 diabetes. Metabolism 1999; 48: 641-644.
- [24] Martin SE. The effect of resistance, endurance, and combination exercise on lipid metabolism and non-traditional cardiovascular disease risk markers in previously untrained men. Doctoral thesis, 2008.
- [25] Skoglund-Andersson C, Tang R, Bond MG, de Faire U, Hamsten A, Karpe F. LDL particle size distribution is associated with carotid intima-media thickness in healthy 50-year-old men. Arterioscleros Thrombos Vas Biol 1999; 19: 2422-2430.

- [26] Tsimikas S, Willerson JT, Ridker PM. C-reactive protein and other emerging blood biomarkers to optimize risk stratification of vulnerable patients. J Am Coll Cardiol 2006; 47: C19-C31
- [27] Zambon A, Hokanson JE, Brown BG, Brunzell JD. Evidence for a new pathophysiological mechanism for coronary artery disease regression: hepatic lipase-mediated changes in LDL density. Circulation 1999; 99: 1959-1964.
- [28] Zambon A, Austin MA, Brown BG, Hokanson JE, Brunzell JD. Effect of hepatic lipase on LDL in normal men and those with coronary artery disease. Arterioscler Thromb 1993; 13: 147-153.
- [29] Zambon A, Austin MA, Brown BG, Hokanson JE, Brunzell JD. Effects of hepatic lipase on LDL in normal men and those with coronary artery disease. Arterioscler Thromb 1993; 13: 147–153.
- [30] Johansson J, Carlson LA, Landou C, Hamsten A. High-density lipoproteins and coronary atherosclerosis: a strong inverse relation with the largest particles is confined to normo-triglyceridemic patients. Arterioscler Thromb 1991; 11: 174-182.
- [31] Herbert PN, Bernier DN, Cullinane EM, Edelstein L, Kantor MA, Thompson PD. High-density lipoprotein metabolism in runners and sedentary men. JAMA 1984; 252: 1034 –1037.