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Effect of a strenuous aerobic exercise on sdLDL concentration in healthy men

Nooshin Khajeian*

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(*) MS in Exercise Physiology, Department of Exercise physiology, Marvdasht branch, Islamic Azad University, Marvdasht, Iran.
E-mail: Nooshinkh2020@gmil.com

Abstract

Introduction: Clinical studies indicated that small dense LDL (sdLDL) levels are more powerful than LDL levels for the determination of severe stable coronary heart disease (CHD). The effects of intensive aerobic exercise on sdLDL levels are not well known; thus the aim of present study was to investigate effect of a strenuous aerobic exercise on sdLDL concentration in healthy men.

Material & Methods: Eleven healthy young men (aged: 20.8 ± 1.8 years; ± SD) volunteered to participate in this study. All the subjects were performed Repeated High-Intensity Endurance Test (RHIET) as a strenuous aerobic exercise. Blood samples were taken at baseline and immediately after the RHIET. Wilcoxon and paired-sample t-test was used to analyze the data.

Results: The results showed that sdLDL (38.8±11.3 mg/dl *vs.* 39.9±11.3 mg/dl), TC (188.6±36.2 mg/dl *vs.* 194.1±42.2 mg/dl), TG (139.6 mg/dl ± 55.0 *vs.* 157.7 ± 79.7 mg/dl),

LDL (109.1 ± 33.4 mg/dl *vs.* 121.5 ± 53.0 mg/dl) and HDL (44.0 ± 13.6 mg/dl *vs.* 44.6 ± 14.0 mg/dl) remained unchanged in response to strenuous aerobic exercise. Significant correlation was observed between changes of sdLDL with TC (r = 0.74, P = 0.008), TG (r = 0.65, P = 0.02) and LDL (r = 0.64, P = 0.03) levels.

Conclusions: The results suggest strenuous aerobic exercise had not significant effect on blood lipids and lipoprotein subfractions.

Keywords: Coronary heart disease, Intensive exercise, Lipoprotein subfractions, sdLDL

1. Introduction

Reducing the concentration of low-density lipoprotein cholesterol (LDL) in serum is the primary target for the prevention of coronary heart disease (CHD) under national and international guidelines (1-3). Among patients with established CHD, the guidelines recommend a target LDL level of <100 mg/dL (1-3). Lipid-lowering agents such as 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (statins) reduce CHD events, but the level of LDL on statin treatment is not always predictive of outcome (4). Small dense LDL (sd-LDL) particles have been suggested to be highly atherogenic due to their greater ability to penetrate the arterial wall, their lower affinity for the LDL receptor, their prolonged half-life in plasma, and their lower resistance to oxidative stress compared to large-LDL (5,6). The Québec cardiovascular study has confirmed that a greater proportion of sd-LDL at baseline is a strong and independent predictor of CHD in the first 7 years of follow-up (7). In contrast, an elevated concentration of large-LDL is a poor predictor of CHD and seems to be associated with a low CHD risk (7). This suggests that the atherogenicity of LDL differs among heterogeneous LDL particles and that the association of atherogenic LDL with CHD is chiefly due to the sd-LDL component (8).

Regular exercise can reduce blood pressure, improve insulin sensitivity, and increase HDL and apolipoprotein B (9-11), thus help reduce the

prevalence of CHD. Although it has been reported that regular aerobic exercise can reduce LDL (9-11), there is a distinct lack of research examining the effect of intensive aerobic exercise on sdLDL. 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 (12). 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 (13). Yu et al. (1999), however, noted that sdLDL particles decreased significantly by 62% after the triathlon in highly trained athletes (14). The effects of intensive aerobic exercise on sdLDL levels are not well known; thus the aim of present study was to investigate effect of a strenuous aerobic exercise on sdLDL concentration in healthy men.

2. Material & Methods

Subjects

Following ethical approval from Marvdasht branch, Islamic Azad University Ethics Committee, eleven apparently healthy young men (aged: 20.8 ± 1.8 years; ± SD) participants were recruited for the study (Anthropometric and body composition data are provided in Table 1). A screening questionnaire obtaining medical and exercise history was completed by each participant. Participants were excluded if they used multi-vitamins or antioxidants in the past two months, were using lipid lowering/modifying medication, had a diagnosis of heart disease, dyslipidaemia, type 1 or type II diabetes mellitus, orthopaedic limitations or had any other health problem that may interfere with exercise. All participants were non-smokers and they were not engaged in any systematic exercise programs at least 6 months before the study as assessed by a physical activity questionnaire.

Table 1. Participant characteristics of the subjects

Variables	mean	SD
Body weight (kg)	69.4	13.0
Body mass index (kg/m^{2})	22.2	3.2

Body fat (%)	8.2	2.7
WHR	0.83	0.1

Strenuous aerobic exercise

All the subjects were performed Repeated High-Intensity Endurance Test (RHIET) as a strenuous aerobic exercise. Each subject was allowed 10 minutes to complete his own specific warm-up. Four beacons were placed 5 meters apart in a straight line to cover a total distance of 15 meters. Subjects were instructed to avoid pacing and perform with a maximal effort throughout the whole test. Each subject started the test in line with the first beacon, and upon an auditory signal sprinted 5 m to a second beacon, touched the ground adjacent to the beacon with their hand and returned back to the first beacon, touching down on the ground adjacent to the beacon with the hand again. The subject then sprinted 10 m to the third beacon, and back to the first beacon etc. until an exercise period of 30 seconds had elapsed. No instruction was given as to which hand should touch during each turn. The subjects performed 43 repeat bouts of this protocol with a 30 second rest between bouts.

Biochemical analyses

Blood samples were taken at baseline and immediately after the RHIET. Blood sample was obtained by venipuncture. sdLDL levels were obtain using following formula that previously excogitated by Srisawasdi et al. (2011) (15):

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

dLDL: Direct low-density lipoprotein-cholesterol

cLDL: Calculated low-density lipoprotein-cholesterol

The levels of TC, TG, HDL, and dLDL-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 (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:

$$\text{cLDL} = \text{TC} - \text{HDL} - (\text{TG}/5).$$

Statistical Analysis

Results were expressed as the mean ± SD and distributions of all variables were assessed for normality. Paired t-test and Wilcoxon test were used to compute mean (± SD) changes in the variables before and after the intervention. Pearson and spearman correlations and general linear regression analysis were performed to calculate a correlation. 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 17, SPSS, Inc., Chicago, IL).

3. Results

The results showed that sdLDL (38.8 ± 11.3 *vs.* 39.9 ± 11.3) remained unchanged in response to strenuous aerobic exercise (Figure 1). Significant correlation was observed between changes of sdLDL levels with TC ($r = 0.74$, $P = 0.008$), TG ($r = 0.65$, $P = 0.02$) and LDL levels ($r = 0.64$, $P = 0.03$). General linear regression indicates that change of TG is an independent predictor for changes of sdLDL levels ($t = 2.8$, $P = 0.02$).

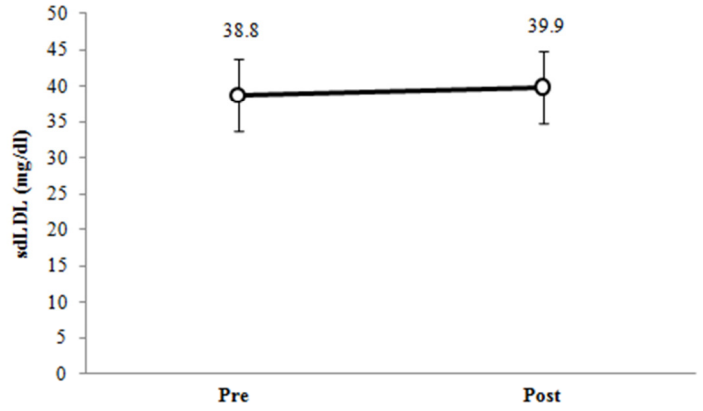

Figure 1. Changes of sdLDL in response to intensive aerobic exercise

The changes of TC concentration in response to intensive aerobic exercise are presented in the Figure 2. The results indicated that there were no significant differences in TC concentration after the intervention compare to the baseline levels (188.6 ± 36.2 *vs.* 194.1 ± 42.2).

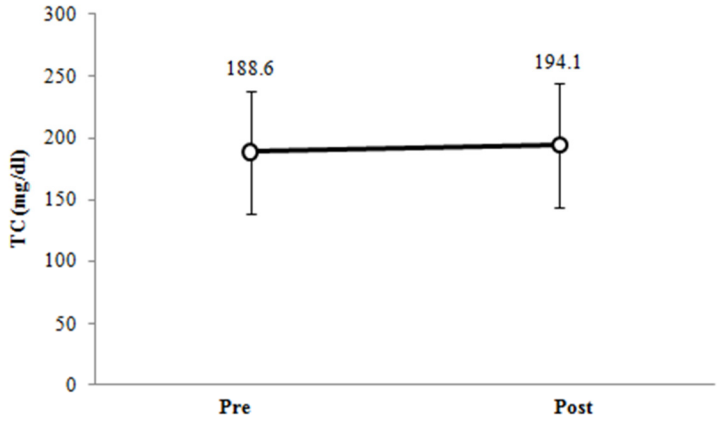

Figure 2. Changes of TC in response to intensive aerobic exercise

As shown in the Figure 3, no significant differences were observed in TG concentration after the intensive aerobic exercise compare to the baseline levels (139.6 ± 55.0 *vs.* 157.7 ± 79.7).

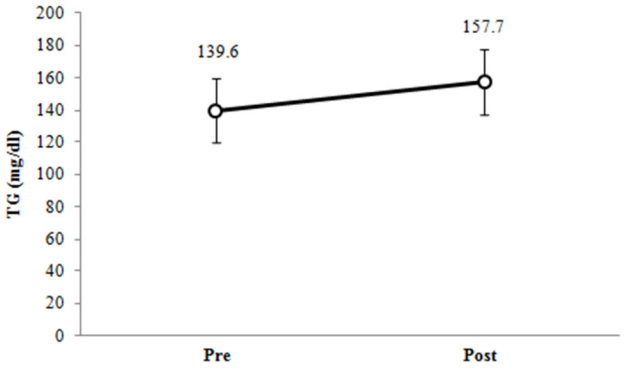

Figure 3. Changes of TG in response to intensive aerobic exercise

The results showed that LDL (109.1 ± 33.4 *vs.* 121.5 ± 53.0) sdLDL remained unchanged in response to strenuous aerobic exercise (Figure 4).

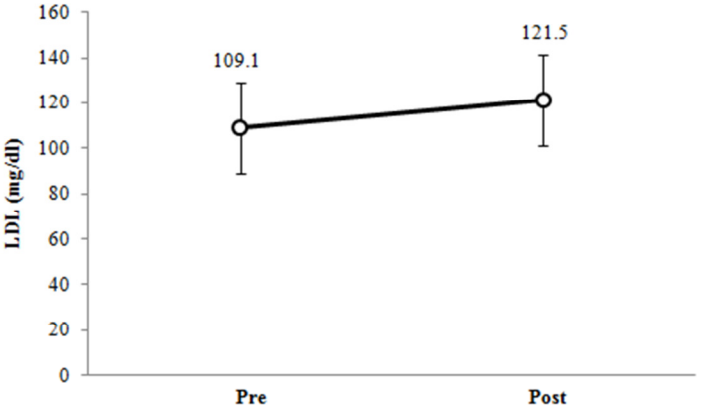

Figure 4. Changes of LDL in response to intensive aerobic exercise

The changes of HDL concentration in response to intensive aerobic exercise are presented in the Figure 5. The results indicated that there were no significant differences in HDL concentration after the intervention compare to the baseline levels (44.0 ± 13.6 *vs.* 44.6 ± 14.0).

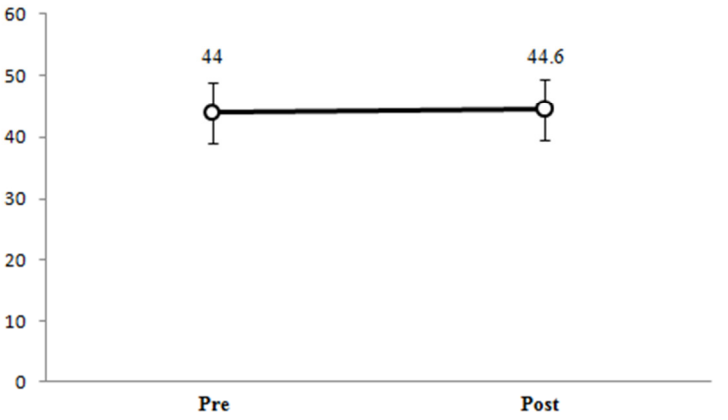

Figure 5. Changes of HDL in response to intensive aerobic exercise

4. Discussion

The primary aim of this study was to investigate the effect of a strenuous aerobic exercise on sdLDL concentration in healthy men. The results of present study indicated that sdLDL remained unchanged in response to strenuous aerobic exercise. Nayeri khoob and Moghadasi (2017) however indicated hat sdLDL level was increased after a bout of heavy resistance exercise and it decreased after 8 weeks regular resistance training (12). 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 (13). Yu et al. (1999), however, noted that sdLDL particles decreased significantly by 62% after the triathlon in highly trained athletes (14). 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) (16). 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 (17). 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 (18). 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 (19). 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 (20). Although HTGL may not change with a single exercise session (21), training can result in chronic reduction in HTGL activity (22), which may lead to lower concentrations of sdLDL particles. The result indicated that there is a significant correlation between changes of sdLDL concentration with TC, TG and LDL levels. General linear regression indicates that change of TG is an independent predictor for changes of sdLDL levels. The results showed that TC, TG, LDL and HDL remained unchanged in response to strenuous aerobic exercise, thus it seems that the lack of effect of strenuous aerobic exercise on sdLDL in the present study might be due to the absence of reductions in blood lipids especially TG levels.

On the other hand, there were virtually no changes in TC, TG, LDL and HDL levels in response to a strenuous aerobic exercise. Long-term effects of continuous exercise have been known on lipid profile (23), but the short-term effects of exercise in this regard are too little and contradictory. Previously Greene et al. (2012) in line with the results of present study indicated that lipid profile (TC, TG, LDL, LDL_3, LDL_4, vLDL, HDL, HDL_{2a} and HDL_{2b}) had no significant changes after a session of aerobic exercise on a treadmill with 70% VO_{2max} in obese and overweight adults (24). Hojjati and Shahsavari (2015) however indicated that serum TG and TC increased immediately after a single bout of aerobic exercise in type II diabetic females, however for LDL and HDL no significant changes were observed (25). Although muscle needs less fat compare to carbohydrates during exercise but oxidation of free fatty acids is very important for

performing endurance activities (26,27). Catecholamine, growth hormone, and cortisol rise in the bloodstream, but insulin level reduces. The consequences of these occurrences are increased glycogenolisis and glycolysis in muscle and liver, lipolysis in adipose tissue and muscles, and gluconeogenesis in liver and also increased protein breakdown in muscle and liver (25). The pure effect of this process is that the amount of glucose in the blood remains relatively constant (at least for 60-90 min), fatty acids, glycerol, ketones and also the amino acids increase in the blood during endurance exercise. These are energy sources that are used by the muscle (28). One reason for increased HDL is increasing the activity of the lipoprotein lipase enzyme and decreasing the activity of hepatic lipase enzyme. HDL level was rose by lipoprotein lipase activity, that is a key enzyme for converting vLDL to HDL. The storage and synthesis of hepatic triglycerides and LDL is going to decrease by hepatic lipase activity reduction. In this context, it is likely that other mechanisms, such as decreased insulin sensitivity and lipoprotein changes in lipid levels can be mentioned (29). It should be considered that continuous activity can increase the blood volume and an exercise session can decrease it. Therefore, in the primary steps of the exercise, by increasing the blood pressure a certain amount of plasma enter into the interstitial water and blood concentration has increased up to 20% in some capillaries in the first 10 min of the exercise. It is possible that no changes in TG, TC, LDL and HDL even with reduction plasma volume lead to some changes in concentration of serum lipids (25).

5. Conclusion

The results suggest strenuous aerobic exercise had not significant effect on blood lipids and lipoprotein subfractions in healthy young men.

6. Acknowledgment

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References

1. Grundy SM, Cleeman JI, Merz CN, Brewer HB Jr, Clark LT, Hunninghake DB, et al. National Heart, Lung, and Blood Institute; American College of Cardiology Foundation; American Heart Association: Implications of recent clinical trials for the National Cholesterol Education Program Adult Treat ment Panel III guidelines. Circulation 2004; 110: 227-239.

2. Ballantyne C, Arroll B, Shepherd J. Lipids and CVD management: towards a global consensus. Eur Heart J 2005; 26: 2224-2231.

3. Saito Y. The Japan Atherosclerosis Society Guidelines for Diagnosis and Treatment of Atherosclerotic Cardiovascular Disease 2002 (Editorial). J Atheroscler Thromb 2004; 11: 101-103.

4. Sniderman AD. How, when, and why to use apolipoprotein B in clinical practice. Am J Cardiol 2002; 90: 48i-54i.

5. Griffin BA. Lipoprotein atherogenicity: an overview of current mechanisms. Proc Nutr Soc 1999; 58: 163-169.

6. Berneis KK, Krauss RM. Metabolic origins and clinical significance of LDL heterogeneity. J Lipid Res 2002; 43: 1363-1379.

7. St-Pierre AC, Cantin B, Dagenais GR, Mauriège P, Bernard PM, Després JP, et al. Low-density lipoprotein subfractions and the long-term risk of ischemic heart disease in men, 13-year follow-up data from the Québec Cardiovascular Study. Arterioscler Thromb Vasc Biol 2005; 25: 553-559.

8. Koba S1, Yokota Y, Hirano T, Ito Y, Ban Y, Tsunoda F, et al. Small LDL-cholesterol is superior to LDL-cholesterol for determining severe coronary atherosclerosis. J Atheroscler Thromb 2008; 15: 250-260.

9. Myers J. Cardiology patient pages. Exercise and cardiovascular health. Circulation 2003; 107: e2-e5.

10. Ghasemianpoor J, Nematollahzadeh Mahani M. Effects of short term lifestyle activity modification on lipid profiles in obese and overweight middle aged men with type 2 diabetes. J Physic Act Horm 2017; 1: 1-10.

11. Eskandary S, Rahimi E. Effects of eight weeks aerobic training, resistance training and concurrent training on the metabolic syndrome and HbA1c in men with type 2 diabetes. J Physic Act Horm 2017; 1: 51-64.

12. Nayeri khoob HR, Moghadasi M. Effect of 8 weeks regular resistance training on attenuation of sdLDL changes after single session of heavy resistance exercise. J Physic Act Horm 2017; 1: 39-50.

13. 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.

14. Yu HH, Ginsburg GS, O'Toole ML, Otvos JD, Douglas PS, Rifai N. Acute changes in serum lipids and lipoprotein subclasses in triathletes as assessed by proton nuclear magnetic resonance spectroscopy. Arterioscler Thromb Vasc Biol 1999; 19: 1945-1949.

15. 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.

16. 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.

17. 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.

18. 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.

19. 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.

20. 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.

21. 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.

22. 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.

23. Elmahgoub SM, Lambers S, Stegen S, Van Laethem C, Cambier D, Calders P. The influence of combined exercise training on indices of obesity, physical fitness and lipid profile in overweight and obese adolescents with mental retardation. Eur J Pediatr 2009; 168: 1327-1333.

24. Greene NP, Martin SE, Crouse SF. Acute exercise and training alter blood lipid and lipoprotein profiles differently in overweight and obese men and women. Obesity (Silver Spring) 2012; 20: 1618-1627.

25. Hojjati Z, Shahsavari S. Acute effects of aerobic and combined exercise on serum lipid profile in type II diabetic females. Iran J Health Sci 2015; 3: 31-37.

26. Chapman MJ, Ginsberg HN, Amarenco P, Andreotti F, Boren J, Catapano AL, et al. Triglyceride-rich lipoproteins and high density lipoprotein cholesterol in patients at high risk of cardiovascular disease: evidence and guidance for management. Eur Heart J 2011; 32: 1345-1361.

27. Daly RM, Miller EG, Dunstan DW, Kerr DA, Solah V, Menzies D, et al. The effects of progressive resistance training combined with a whey-protein drink and vitamin D supplementation on glycaemic control, body composition and cardiometabolic risk factors in older adults with type 2 diabetes: study protocol for a randomized controlled trial. Trials 2014; 15: 431-443.

28. MacLaren D, Morton J. Biochemistry for sport and exercise metabolism. 1st ed. London, UK: Wiley, 2011; 185-187.

29. Jensen TE, Richter EA. Regulation of glucose and glycogen metabolism during and after exercise. J Physiol 2012; 590: 1069-1076.