Comparison of the effect of different intensity exercise on a bicycle ergometer on postprandial lipidemia in type II diabetic patients

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Original Article

Abstract

BACKGROUND: Postprandial lipid clearance failure and lipoprotein disorders, which are independent risk factors for cardiovascular diseases are well-recognized in type II diabetes. Reduction of fats through exercise has been proved, though the mechanism is not well-defined, and the effects of different intensity exercise on postprandial lipidemia in diabetes type II is unknown. This study aims to find these effects using a cycle ergometer.

METHODS: On three different days, 15 type II diabetics (10 women and 5 men, with a mean age 42.07 ± 6.05 years, weight 94.64 ± 4.37 kg, height 159.78 ± 9.09 cm, and body mass index 29.83 ± 3.93 kg/m^2), consumed a full fat breakfast (750-800 kcal, 85% fat), and 150 min later, blood samples were taken from them to measure their lipid profile. The 1st day was the control day, without any exercises. Seven days later, 90 min after enriched breakfast, they did 30 min of exercise on the cycle ergometer with intensity of 55-70% of maximum heart rate (HRmax), and 14 days later, 90 min after enriched breakfast, they did 30 min of exercise with intensity of 70-85% of HRmax.

RESULTS: According to Friedman non-parametric test, high-density lipoprotein (HDL) cholesterol serum level significantly increased after 30 min of moderate intensity exercise ($P > 0.05$, from 39.4 ± 5.2 to 48.6 ± 9.3), while this increase was insignificant after a higher intensity exercise. Neither intensity levels had any significant effects on triglyceride or on low-density lipoprotein cholesterol.

CONCLUSION: Results showed that moderate intensity exercise was more effective in increasing HDL cholesterol level in type II diabetics.

Keywords: Postprandial Lipidemia, Resistance Exercise, Bicycle Ergometer, Type II Diabetes

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Introduction

Since the middle of the 20th century, the rapid increase in prevalence of diabetes has become a major concern for the World Health Organization,[1,2] and over 246 million current number of diabetes is forecast to increase to 380 million by 2025.[3] Diabetes is a group of metabolic diseases. Over the past three decades, postprandial hyperlipidemia has been the most common diabetes disorder, responsible for pancreatic disorders, insulin resistance, and type II diabetes,[4,5] as well as high incidence of cardiovascular diseases (CVDs).[6] Postprandial lipid increase includes triglyceride (TG) rich lipoproteins, increased very low-density lipoprotein (LDL), and increased less dense, smaller LDL particles in plasma.[6,7] It has been over 30 years since the publication of information identifying relationship between postprandial hyperlipidemia, and CVDs and atherosclerosis.[7,8]

There have been numerous studies on advantages of consistent aerobic exercises on postprandial lipid reduction,[9-16] showing effects remaining 11-18 h after eating fatty food.[16-18] However, no research has been conducted into the effect of different intensity exercise on postprandial lipidemia in diabetes type II patients.[19-23] This study aims to investigate the effect of different intensity of exercises on reducing postprandial lipidemia in patients with diabetes type II.

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Materials and Methods

This applied, cross-sectional, and semi-experimental study was conducted on type II diabetic patients attending Isfahan Cardiovascular Research Center in 2010, with age ranging from 30 to 50 years. After completion of a questionnaire containing such information as age, gender, body mass index, medical history, including kidney, liver, eye, and CVDs; insulin intake, physical activity and diet. Based on blood pressure and electrocardiogram and normal laboratory standards and after completing a consent form, 15 type II diabetic patients (5 males and 10 females) were selected. Those with diabetes complications such as retinopathy, CVDs, skin diseases, joint diseases, and lipid disorders were excluded from the study.

Preliminary tests

Physical examination and measurements of weight, height, body mass index (BMI) were carried out, and under specialist's supervision, treadmill exercise test was conducted according to Bruce protocol and maximum intensity exercise for each patient was determined during the rest after 5-15 min of exercise. Exercise intensity was found by Carnen method as follows:

Heart rate at reserve (HRR) = HR_{max} − heart rate at rest (HR_{rest})

Target heart rate (THR) (intensity %) = HR_{rest} + (intensity %) (HR_{max} – HR_{rest})

Or

THR (intensity %) = HR_{rest} + (intensity %) HRR.

According to exercise test and medical examination, all patients were healthy in cardiovascular terms.

Main tests

Seven days after an exercise test, all patients participated in a 3-day study protocol with 7 days interval. On each day, blood samples were taken 150 min after enriched breakfast. The 1st day was a control day with no exercise. On the other 2 days, 90 min after breakfast, patients exercised on the ergometer bicycle with different intensities for 30 min.

The 1st day

Full fat breakfast included; 150 g bread, 25 g butter, 100 g cream cheese, and 200 ml milk (85% fat), and no exercise. Blood samples were taken 150 min after enriched breakfast.

The 2nd day

Seven days later, 90 min after the same type of enriched breakfast, patients exercised on a cycle ergometer for 30 min with 55-75% maximum heart rate (HRmax) intensity.

The 3rd day

Much the same as the 2nd day, but with 70-85% HRmax intensity.

Exercise sessions and intensities

Each 30 min session involved 5 min of warm-up, 20 min of exercise with specified intensity, and 5 min of cooling down. Caronen method was used according to each patient's intensity result from exercise test, and under specialist's supervision, bicycle ergometer resistance was altered accordingly.

Blood sampling

To determine lipoprotein and lipidemia indices, 10 cc of blood was taken from the antecubital vein of the left arm in seating position, 150 min after enriched breakfast on each day of the test. Blood samples were immediately cooled down to 4° C and centrifuged at 2000 rpm for 15 min, separating its serum. Serum total cholesterol, TG, high-density lipoprotein (HDL) cholesterol, and glucose were measured using Pars Azmoon Company kits, and LDL cholesterol was measured using Randox Company kit.

Data analysis

The SPSS for windows (version 16, SPSS Inc., Chicago, IL, USA) was used for data analysis in two stages; descriptive with mean ± standard deviation and inferential statistics using non-parametric Friedman test. Significance level was set at $P > 0.05$ for all patients.

Results

Participants' characteristics

Fifteen type II diabetic patients (5 men and 10 women) with a mean age of 42.07 ± 6.05 years, BMI of 29.83 ± 3.93 kg/m^2, and with 1.0 ± 0.8 years since their diagnosis with type II diabetes were studied. Exercise test results, biochemical, and clinical details of patients are presented in table 1.

Postprandial lipidemia response after moderate and high intensity physical activity

The mean serum HDL cholesterol increased somewhat after enriched breakfast, and increased significantly after moderate intensity exercise. High intensity exercise increased HDL cholesterol only a little (Table 2). According to the Friedman test, there was a significant difference between the two levels of exercise intensity for serum HDL cholesterol. In fact, moderate intensity exercise increased postprandial HDL more than high intensity exercise (Figure 1). After intake of fat-enriched breakfast, patients' plasma TG concentration increased, but reduced again after exercise (Table 2). Moderate intensity exercise reduced mean TG level more than high intensity (Figure 2). But, the difference between exercise

intensities in reducing TG was insignificant according to Friedman test (P ≥ 0.274) (Table 2).

There was no significant difference in mean postprandial serum LDL level after high and moderate intensity exercise (P < 0.05), though, high intensity exercise more reduced LDL than moderate intensity (Figure 3).

Discussion

In recent years, not only has postprandial hyperlipidemia been taken as the most common sign and risk factor of CVDs and athrosclerosis in type II diabetes, it is also now being taken as the main predictor of CVDs. The relationship between postprandial hyperlipidemia and level of exercise remains unclear,[21-23] in spite of recent studies on the role of exercise in treatment and prevention of postprandial hyperlipidemia.[19-24] Hence, this study attempted to find a suitable exercise intensity level for treatment of this disorder in type II diabetic patients, by comparing two exercise intensity levels.

Table 1. Biochemical and clinical details and exercise test results of type II diabetic patients

Variables	Mean ± SD (n = 15)
Age (year)	42.07 ± 6.05
Height (cm)	159.78 ± 9.09
Weight (kg)	94.64 ± 4.37
BMI (kg/m²)	29.83 ± 3.93
WHR	0.87 ± 0.46
BP (mmHg)	122.60 ± 13.65
BS (mg/dl)	167.06 ± 59.71
TG (mg/dl)	195.93 ± 77.30
Total cholesterol (mg/dl)	184.46 ± 33.32
LDL cholesterol (mg/dl)	113.92 ± 30.21
HDL cholesterol (mg/dl)	39.40 ± 5.27
HbA1c (%)	8.12 ± 1.51

SD: Standard deviation; BMI: Body mass index; WHR: Waist-to-hip circumference ratio; BP: Blood pressure; BS: Blood sugar; TG: Triglyceride; LDL: Low-density lipoprotein; HDL: High-density protein; HbA1c: Hemoglobin A1c

Table 2. Mean and standard deviation of biochemical blood indices before and after breakfast and after 30 min of moderate and high intensity exercise, with Friedman test results, mean scores, and chi-square test and significance level

Variables	Number	Mean ± SD	Mean score	Chi-squared	Significance P > 0.05
Postprandial TG	15	324.53 ± 144.36	2.17		
Postprandial TG and moderate exercise	15	240.93 ± 122.98	1.67	2.586	0.274
Postprandial TG and maximum exercise	15	304.86 ± 141.07	2.17		
Postprandial LDL cholesterol	15	106.26 ± 14.14	2.27		
Postprandial LDL cholesterol and moderate exercise	15	105.46 ± 25.25	1.97	1.932	0.381
Postprandial LDL cholesterol and maximum exercise	15	100.93 ± 18.79	1.77		
Postprandial HDL cholesterol	15	42.06 ± 4.97	1.53		
Postprandial HDL cholesterol and moderate exercise	15	42.06 ± 4.97	1.53	9.404	0.009
Postprandial HDL cholesterol and maximum exercise	15	48.60 ± 9.30	2.60		

SD: Standard deviation; TG: Triglyceride; LDL: Low-density lipoprotein; HDL: High-density lipoprotein

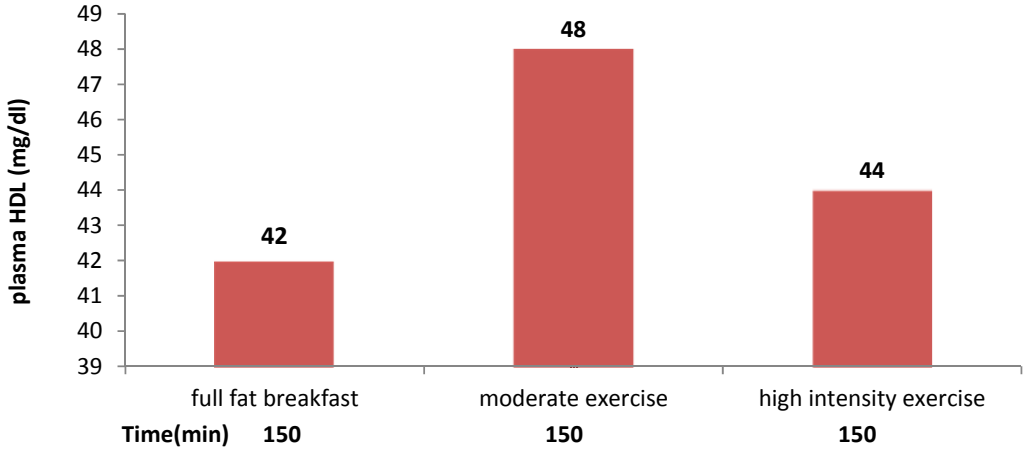

Figure 1. Comparison mean postprandial high-density lipoprotein (HDL) cholesterol concentrations levels, with 30 min of moderate and high intensity exercise

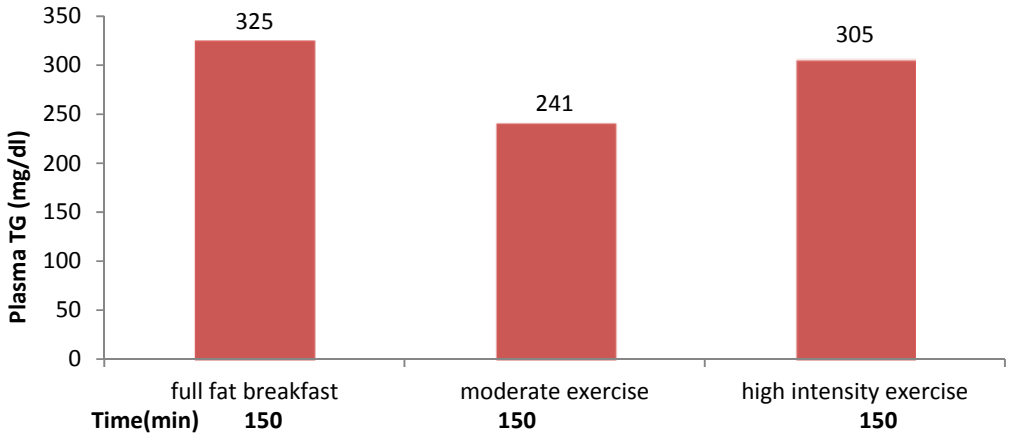

Figure 2. Comparison mean postprandial triglyceride (TG) concentrations levels, with 30 min of moderate and high intensity exercise

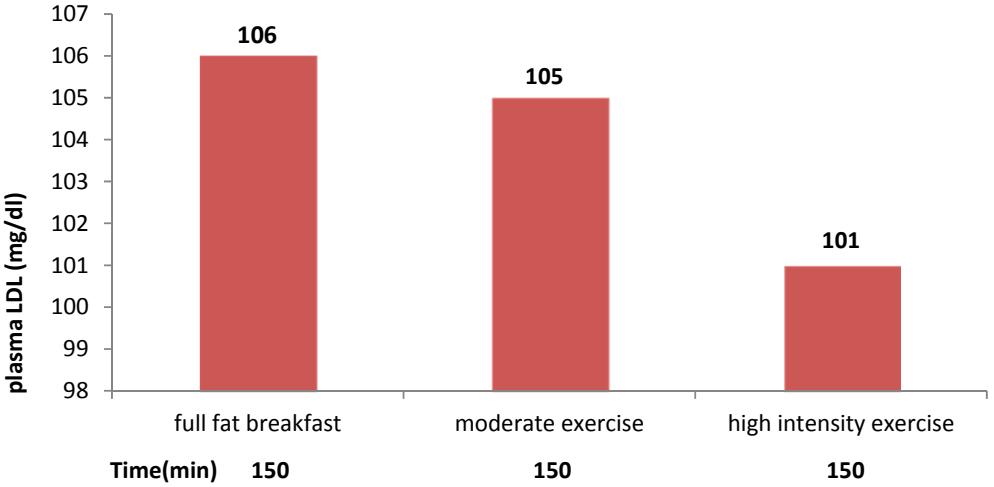

Figure 3. Comparison mean postprandial low-density lipoprotein (LDL) cholesterol concentrations levels, with 30 min of moderate and high intensity exercise

The main finding of this study showed that the serum postprandial HDL cholesterol increased significantly with moderate intensity exercise (Table 2). In contrast, serum HDL did not significantly increase with high intensity exercise (Figure 1). This finding agrees with that of previous studies with regards to the effect of regular aerobic exercise on HDL cholesterol increase.[11] Lipid oxidation[11-27] increased muscular and liver lipoprotein,[12-28] reduced TG concentration,[25,27] increased half-life of HDL cholesterol and its function during regular low-intensity aerobic exercise in patients with type II diabetes[28] are among the main reasons for increased postprandial HDL cholesterol after exercise. Although some studies attributed this increase to TG plasma and lipid metabolic disorders as well as to liver function abnormalities and inadequate and indistinct exercise intensity.[29] Recently, other studies have shown HDL cholesterol can be increased with intensive exercise, believing this to be due to reduction in blood TG resistance, increased insulin sensitivity, and reduced muscle and liver insulin resistance after intensive exercise.[17,30,31] Intensive exercise duration and the effect of consistent exercise and patients' adjustment to it are reasons for differing results of this study.

Another finding was with regards to TG serum level. The difference between exercise intensities in reducing TG was insignificant (Table 2). In fact, moderate intensity exercise reduced mean postprandial TG level in type II diabetic patients more than high intensity exercise. Nonetheless, it was statistically insignificant (Figure 2).

A study in 2006 showed that regular aerobic exercise significantly improved TG concentration due to increased muscle blood circulation and gradual increase in muscle lipid oxidation during exercise.[32] In a similar study, Trenell et al.[17] and Tobin et al.[27] found that a course of regular moderate intensity aerobic exercise can reduce postprandial TG concentration just as well as insulin secretion. This was thought to be due to type II diabetic patients' adjustment to regular aerobic exercise. The effect of adjustment over a few months in previous studies and the small sample size in this study are the reason for the differences.

In other studies,[3,30-34] no significant relationship was found between postprandial serum TG variations and high intensity exercise, which was thought to be due to metabolic differences in type II diabetic patients,[30] increased muscle blood circulation, greater distribution of fatty acids released from adipose tissue onto active muscles during and after strenuous exercise.[32] Other researchers have reported postprandial TG reduction through increased muscular lipoprotein lipase after an intense walking exercise.[3,17,26-34] Again, duration and inadequate number of exercise sessions for effective adjustment caused the differences between the results of this study and those of previous studies.

The last finding of this study showed that one session of high intensity exercise reduced postprandial LDL cholesterol more than one session of moderate intensity exercise. Though it was not statistically significant (Table 2), its importance cannot be undermined (Figure 3). This was also illustrated in previous studies,[3,17,27-35] no significant reduced postprandial LDL cholesterol was found a period of high intensity exercise in type II diabetic patients,[27,30] and emphasized on such reasons as oxidation disorders in liver metabolism, reduced liver LDL receptors, reduced postprandial blood lipid clearance in type II diabetic patients. However, Cohen et al. found a reduction in LDL cholesterol after 14 months of intense physical activity.[34] Also, Harrison et al. showed intensive exercise could reduce LDL cholesterol concentration and level. They considered increased oxidation in liver metabolism and LDL receptors the reasons for this.[30] Therefore, duration and infrequent sessions of high intensity exercise are thought to have made the difference between this and previous studies.

Conclusion

According to the results of this study, moderate intensity aerobic exercise reduces HDL cholesterol level more than high intensity exercise, and postprandial TG serum level is reduced more with regular, consistent, moderate intensity exercise than with high intensity exercise. However, long-term adjustment of the body to regular exercise reduces TG most in type II diabetic patients. Postprandial LDL cholesterol reduction occurred more with high intensity, regular aerobic exercise than with moderate intensity exercise. Generally, postprandial lipid reduction in type II diabetes is best achieved through a combined regular aerobic and consistent exercise.

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Conflict of Interests

Authors have no conflict of interests.

References

1. Regensteiner JG, Reusch JE, Stewart KJ, Veves A. Diabetes and Exercise. Berlin, Germany: Springer; 2009. p. 317.
2. Burns N, Finucane FM, Hatunic M, Gilman M, Murphy M, Gasparro D, et al. Early-onset type 2 diabetes in obese white subjects is characterised by a marked defect in beta cell insulin secretion, severe insulin resistance and a lack of response to aerobic exercise training. Diabetologia 2007; 50(7): 1500-8.
3. Zanuso S, Jimenez A, Pugliese G, Corigliano G, Balducci S. Exercise for the management of type 2 diabetes: a review of the evidence. Acta Diabetol 2010; 47(1): 15-22.
4. Kommoju UJ, Reddy BM. Genetic etiology of type 2 diabetes mellitus: a review. International Journal of Diabetes in Developing Countries 2011; 31(2): 51-64.
5. Skoczynska A, Kreczynska B, Poreba R. Postprandial lipemia in diabetic men during hypolipemic therapy. Pol Arch Med Wewn 2009; 119(7-8): 461-8.
6. Enkhmaa B, Ozturk Z, Anuurad E, Berglund L. Postprandial lipoproteins and cardiovascular disease risk in diabetes mellitus. Curr Diab Rep 2010; 10(1): 61-9.
7. Carstensen M, Thomsen C, Gotzsche O, Holst JJ, Schrezenmeir J, Hermansen K. Differential postprandial lipoprotein responses in type 2 diabetic men with and without clinical evidence of a former myocardial infarction. Rev Diabet Stud 2004; 1(4): 175-84.
8. Lievre MM, Moulin P, Thivolet C, Rodier M, Rigalleau V, Penfornis A, et al. Detection of silent myocardial ischemia in asymptomatic patients with

diabetes: results of a randomized trial and meta-analysis assessing the effectiveness of systematic screening. Trials 2011; 12: 23.
9. Codario RA. Type 2 Diabetes, Pre-Diabetes, and the Metabolic Syndrome. Berlin, Germany: Springer; 2010. p. 367.
10. Ferreira AP, Ferreira CB, Souza VC, Cordova CO, Silva GC, Nobrega OT, et al. The influence of intense intermittent versus moderate continuous exercise on postprandial lipemia. Clinics (Sao Paulo) 2011; 66(4): 535-41.
11. Colberg SR. Physical activity: the forgotten tool for type 2 diabetes management. Front Endocrinol (Lausanne) 2012; 3: 70.
12. Hurren NM, Balanos GM, Blannin AK. Is the beneficial effect of prior exercise on postprandial lipaemia partly due to redistribution of blood flow? Clin Sci (Lond) 2011; 120(12): 537-48.
13. Praet SF, van Loon LJ. Exercise therapy in type 2 diabetes. Acta Diabetol 2009; 46(4): 263-78.
14. Colberg SR, Sigal RJ, Fernhall B, Regensteiner JG, Blissmer BJ, Rubin RR, et al. Exercise and type 2 diabetes: the American College of Sports Medicine and the American Diabetes Association: joint position statement. Diabetes Care 2010; 33(12): e147-e167.
15. Morrato EH, Hill JO, Wyatt HR, Ghushchyan V, Sullivan PW. Physical activity in U.S. adults with diabetes and at risk for developing diabetes, 2003. Diabetes Care 2007; 30(2): 203-9.
16. Wagner H, Degerblad M, Thorell A, Nygren J, Stahle A, Kuhl J, et al. Combined treatment with exercise training and acarbose improves metabolic control and cardiovascular risk factor profile in subjects with mild type 2 diabetes. Diabetes Care 2006; 29(7): 1471-7.
17. Trenell MI, Hollingsworth KG, Lim EL, Taylor R. Increased daily walking improves lipid oxidation without changes in mitochondrial function in type 2 diabetes. Diabetes Care 2008; 31(8): 1644-9.
18. Rimbert V, Boirie Y, Bedu M, Hocquette JF, Ritz P, Morio B. Muscle fat oxidative capacity is not impaired by age but by physical inactivity: association with insulin sensitivity. FASEB J 2004; 18(6): 737-9.
19. MacEneaney OJ, Harrison M, O'Gorman DJ, Pankratieva EV, O'Connor PL, Moyna NM. Effect of prior exercise on postprandial lipemia and markers of inflammation and endothelial activation in normal weight and overweight adolescent boys. Eur J Appl Physiol 2009; 106(5): 721-9.
20. Kadoglou NP, Iliadis F, Angelopoulou N, Perrea D, Ampatzidis G, Liapis CD, et al. The anti-inflammatory effects of exercise training in patients with type 2 diabetes mellitus. Eur J Cardiovasc Prev Rehabil 2007; 14(6): 837-43.
21. Okada S, Hiuge A, Makino H, Nagumo A, Takaki H, Konishi H, et al. Effect of exercise intervention on endothelial function and incidence of cardiovascular disease in patients with type 2 diabetes. J Atheroscler Thromb 2010; 17(8): 828-33.
22. Conn VS, Hafdahl AR, Moore SM, Nielsen PJ, Brown LM. Meta-analysis of interventions to increase physical activity among cardiac subjects. Int J Cardiol 2009; 133(3): 307-20.
23. Holt RG, Cockram C, Flyvbjerg A, Goldstein BJ. Textbook of Diabetes. New Jersey, NJ: Wiley; 2010. p. 1119.
24. Singhal A, Trilk JL, Jenkins NT, Bigelman KA, Cureton KJ. Effect of intensity of resistance exercise on postprandial lipemia. J Appl Physiol (1985) 2009; 106(3): 823-9.
25. Nicklas B. Endurance Exercise and Adipose Tissue. London, UK: Taylor & Francis; 2001. p. 179.
26. Castaneda C, Layne JE, Munoz-Orians L, Gordon PL, Walsmith J, Foldvari M, et al. A randomized controlled trial of resistance exercise training to improve glycemic control in older adults with type 2 diabetes. Diabetes Care 2002; 25(12): 2335-41.
27. Tobin LW, Kiens B, Galbo H. The effect of exercise on postprandial lipidemia in type 2 diabetic patients. Eur J Appl Physiol 2008; 102(3): 361-70.
28. Zois C, Tokmakidis SP, Volaklis KA, Kotsa K, Touvra AM, Douda E, et al. Lipoprotein profile, glycemic control and physical fitness after strength and aerobic training in post-menopausal women with type 2 diabetes. Eur J Appl Physiol 2009; 106(6): 901-7.
29. Harrison M, O'Gorman DJ, McCaffrey N, Hamilton MT, Zderic TW, Carson BP, et al. Influence of acute exercise with and without carbohydrate replacement on postprandial lipid metabolism. J Appl Physiol (1985) 2009; 106(3): 943-9.
30. Harrison M, Murphy RP, O'Connor PL, O'Gorman DJ, McCaffrey N, Cummins PM, et al. The endothelial microparticle response to a high fat meal is not attenuated by prior exercise. Eur J Appl Physiol 2009; 106(4): 555-62.
31. Miyashita M, Burns SF, Stensel DJ. Accumulating short bouts of brisk walking reduces postprandial plasma triacylglycerol concentrations and resting blood pressure in healthy young men. Am J Clin Nutr 2008; 88(5): 1225-31.
32. Middlebrooke AR, Elston LM, Macleod KM, Mawson DM, Ball CI, Shore AC, et al. Six months of aerobic exercise does not improve microvascular function in type 2 diabetes mellitus. Diabetologia 2006; 49(10): 2263-71.
33. Mensink M, Blaak EE, van Baak MA, Wagenmakers AJ, Saris WH. Plasma free Fatty Acid uptake and oxidation are already diminished in subjects at high risk for developing type 2 diabetes. Diabetes 2001; 50(11): 2548-54.

34. Cohen ND, Dunstan DW, Robinson C, Vulikh E, Zimmet PZ, Shaw JE. Improved endothelial function following a 14-month resistance exercise training program in adults with type 2 diabetes. Diabetes Res Clin Pract 2008; 79(3): 405-11.
35. Galbo H, Tobin L, van Loon LJ. Responses to acute exercise in type 2 diabetes, with an emphasis on metabolism and interaction with oral hypoglycemic agents and food intake. Appl Physiol Nutr Metab 2007; 32(3): 567-75.

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