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THE EFFECTS OF THE TYPE OF EXERCISE ON LIPID PROFILES AND ADIPONECTIN LEVEL IN SEDENTARY MEN

UTJECAJ VRSTE AKTIVNOSTI NA LIPIDNI PROFIL I KONCENTRACIJU ADIPONEKTINA U SEDENTARNIH MUŠKARACA

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SUMMARY

The aim of the present study was to compare the effect of the type of exercise on lipid profile and adiponectin level in sedentary young men by 24-hour follow up exercise. Thirty young males with normal body mass index (BMI) were selected randomly and assigned to three groups: aerobic (30 min activity with 60 to 70% maximum oxygen uptake), resistance (3 sets of 10 repetitions at 70% of one maximum repetition), and concurrent (aerobic exercise for 20 min and resistance exercise for 2 sets) exercise. Lipid and adiponectin profiles were measured before and 24-hour after exercise.

Results indicated that the type of exercise had an effect on the lipid response but did not have an effect on the concentration of adiponectin. The concentrations of cholesterol and high density lipoprotein cholesterol (HDL-C) in resistance group reduced significantly 24-hour after exercise in comparison to the basic values (p<0,05). Moreover, the low-density lipoprotein cholesterol (LDL-C) concentration also reduced but it was not significant. The type of exercise had no effect on triglyceride (TG) response. However, when group's data combined, there was a significant reduction in TG concentration. Based on the results, it is concluded that the adiponectin responses to exercise are independent of the type of exercise but resistance exercise has more positive effects on changes in lipid profile than aerobic and concurrent exercises.

Keywords: Adiponectin, lipid profile, sedentary men, type of exercise,

SAŽETAK

Cilj ovog istraživanja bio je usporediti utjecaj vrste vježbanja na lipidni profil i razinu adiponektina u sedentarnih mladića od 24-sata nakon treninga. Trideset mladih muškaraca s normalnim indeksom tjelesne mase (BMI) odabrani su nasumično i razvrstani u tri skupine: aerobnu (30 min aerobnih aktivnosti sa 60 do 70% od maksimalnog primitka kisika), skupinu treninga snage (3 serije po 10 ponavljanja na 70% od 1RM), te mješovitu (aerobna tjelovježba kroz 20 min i trening snage sa 2 serije) Lipidni i adiponektinski profili su mjereni prije i 24 sata nakon vježbanja. Rezultati su pokazali da vrsta vježbe ima utjecaj na lipidni odgovor, ali nema utjecaj na koncentraciju adiponektina. Koncentracije ukupnog kolesterola i HDL-C kolesterola u grupi treninga snage je značajno smanjena 24-sata nakon vježbanja u usporedbi s osnovnim vrijednostima (p<0,05). Osim toga, kolesterol niske gustoće (LDL-C) se također smanjio, ali ne značajno. Vrsta aktivnosti nije imala utjecaja na trigliceride (U skupini mješovitih aktivnosti uočeno je značajno smanjenje koncentracije triglicerida. Na temelju dobivenih rezultata zaključuje se da adiponektinski odgovor ovisi o vrsti vježbe, ali vježbe snage imaju pozitivnije učinke na promjene lipidnog profila od aerobnih vježbi.

Ključne riječi: adiponektin, lipidni profil, sedentarna populacija,vrsta aktivnosti

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INTRODUCTION

World health organization (WHO) has proposed sedentary life style and obesity as two of ten prominent health complications. This lifestyle is a risk factor for cardiovascular diseases which are caused by many factors such as hyperlipemia, hypertension, obesity, fat related hormones, and sedentary life. This multi factorial event has both genetic root and lifestyle basis. Lack of proper physical activity and improper diet are considered as the main reasons of obesity and cardiovascular disease in developed countries (6).

Recently, it has been indicated that adipose tissue secretes bioactive proteins called adipocytokines which may regulate the metabolism of glucose, lipids and vascular function (10). Adiponectin is an adipocytokine that is exceptionally produced from adipocytes. This protein has antidiabetic, antiatherosclerotic and anti-inflammatory properties (10). Meanwhile, having no similarities with other adipocytokins, the level of serum adiponectin reduces in fat people, diabetics and patients with cardiovascular disease (10). The relation between adiponectin and lipid may also be different based on the size of adipocytes. Two studies reported this relation independent of body mass index or body fat percentage (28,37). Asayama et al. (1) found that the relation between adiponectin and triglycerides is independent of extra weight and body fat percentage, but in fat children and adolescents, it depends on visceral adipose tissue and subcutaneous adipose tissue (1).

Exercise training has a strong effect on preventing and treating the obesity and diabetes (12). It is logical to suppose that such effects may be mediated by regulating the hormones derived from adipose tissue such as adiponectin (16,30,34). The effects of chronic exercise on lipids have been recognized (5,31). However, there are few reports on short-term effects of acute exercise but with contradictory results. Resistance exercises have been proposed as an integral part of physical activity plan for health and preventing skeletal-muscular diseases (23). There is less information about the effect of resistance and combined exercise on lipid profile (24) in comparison with endurance training, It has been reported that resistance exercise will properly reduce LDL-C concentration and increase HDL-C concentration (9,17). There are also contradictory results in this case (21,26). Existing methodological differences, such as exercise methods, test specifications, nutrition control and sampling time after activity may also change the results in exercises (14,38). Therefore, the aim this study was to compare the influence of three types of aerobic, resistance and concurrent exercise on lipid profile and adiponectin of serum in sedentary men with normal body mass index.

SUBJECTS AND METHODS

In a quasi-experimental research model, 30 voluntary men (without any regular exercise history, no body weight changes more than 2kg, no specific disease, and no smoking habit for at least the 6 past months were randomly selected from 100 participants with age range between 18 to 25, and were assigned into three exercise categories: aerobic, resistance, and concurrent. Subjects were informed of the objective, benefits and probable risks of test plan and then completed the agreement form before starting the test. The biochemical and common properties of subjects indicated in table 1.

Test Design

Subjects came to physiological lab, in the morning from 8:00 to 10:00 a.m. in fasting mode to be measured for their body composition and to become familiar with exercise plan and sports equipments. Techniques for proper use of treadmill and weightlifting were taught to the subjects. Then, they began their activity to know and determine a one maximum repeat (1RM) to estimate the maximum oxygen uptake (VO_{2max}) by Bruce maximal exercise testing after 10min special warming up. Three days later determining the one repeat maximum test (1RM) and test for estimating the VO_{2max}, subjects came to the laboratory at 8:00 a.m. in fasting conditions for measuring adiponectin and serum lipids. After sampling their blood, they ate the same breakfast containing 550 kcal and one hour later began their activity. After 24-hour activity, they came to the lab for the second time to be measured for blood factors. The specialist collected their blood samples in the same temporal and spatial conditions (12).

Physiological Evaluations

Body weight was measured using Digital Glass Scale, GES-07, USA with accuracy of ±0.1kg, without shoe and minimum clothing cover. Their height was measured by wall height meter, model 44440, made by Kaveh Co., Iran, ±0.1cm, in standing mode next to the wall without shoes while their shoulders were in normal conditions. Waist size was measured in the thinnest part when subjects were ending their expiration. For measuring the hip size, the most projected part was selected. Waist and hip size were measured by non-tactile tape meter without forcing pressure. Body mass index (BMI) was obtained by dividing the weight (kg) by square height (m^2). For measuring the percentage of fat body, Harpenden's caliper model, (the technique of pinching in three regions, pectoral, abdomen and mid-thigh in the right side of body and it was done fro three times with 20 second intervals between any time for returning back to its initial state and then the average was registered), and Jackson and Pollock's (15) formula and Siri (4) equation were used. For removing the individual errors, all measurements were conducted by the same person.

Maximum muscular power for subjects was determined by maximum weight they could lift for once (12). Estimating the maximum used oxygen (VO_2max) and maximum heart beat for subjects were measured by Bruce Maximal Exercise test 10 min after warming up (12).

Diet Records

Subjects were prescreened prior to entry into the exercise study to ensure compliance with the typical American Heart Dietary intake recommendations (i.e., 50–60% carbohydrate, <30% fat, 10–15% protein) (7). Three-day (two weekday and one weekend day) dietary recalls were used for this analysis. Subjects were also given standard dietary instructions for nutrient intake during the 3 days prior to the exercise trial. Intake instructions were based on American Heart Association Guidelines. Total megajoule (MJ) (total kilocalories/238.95) intake range recommendations were based on age, gender and body weight and from estimated resting metabolic rate Harris and Benedict (13). Information from physical activity questionnaires (high, low, moderate activity) was also used to aid in the calculation of total MJ intake to ensure that subjects were in energy balance prior to the exercise trial (7). Food exchange lists with serving sizes were used for nutrient recommendations (Health Management Resources, Boston, Mass., USA). Subjects were asked to complete dietary records for all 3 days prior to the exercise trial. Nutrient intake and distribution (total MJ intake, % fat, % carbohydrate, and % fat) was completed using Software Dorosty Food Processor (NШ, FP2).

One session aerobic, resistance and concurrent exercise

One session of aerobic, resistance and concurrent exercise included general warming up (10min), special warming up (3 to 5min), related exercise and strain exercise and cooling down (5min). Aerobic exercise included running on treadmill for 30 minutes in 60-70% VO_{2max}, resistance exercise with intensity of 70% of 1RM with 10 repeats per each movement for 3 sets with 30-second resting time between stations and 2 minutes between each round of exercise, and concurrent exercise including aerobic exercise, running on treadmill for 20minutes in 60- 70% of VO_{2max} and resistance exercise with intensity of 70% of 1RM with 10 repeats per each movement for 3 sets with 30-seconds resting time between stations and 2minutes between each round. Resistance exercises include 10 station movements in circle form. Stations included: (1) leg flexion, (2) leg extension, (3) leg press, (4) squat, (5) lat pull down, (6) bench press, (7) cross movement by dumbbell, (8) biceps curl, (9) triceps push-down, (10) sit-up, respectively

Collecting and analyzing the blood

After 8 to 10 hours of fasting in two stages, i.e. before beginning the activity (pre-exercise) and 24 h post-exercise, 5ml of venous blood was collected from each subject in sitting and rest mode and immediately centrifuged in 3000rpm and stored in -70°C. For blood sampling, subjects were asked not to have any activities two days before the test.

The level of serum adiponectin was measured by ELISA from sandwich competitive method type (adipogen kit, made in Korea, sensitivity 1ng/ml, and intra and inter assay changes 3.99 and 2.89, respectively).

Total serum cholesterol level was measured by enzymatic colorimetric method, and triglyceride level was measured by enzymatic colorimetric method in the presence of glycerol phosphate oxidase and HDL-C level was measured by enzymatic method and after precipitating remaining lipoproteins containing Apo-B by phosphotangestic acid solution and magnesium chloride. In all cases, the kit derived from Pars Azemon Tehran Co., Iran was used. Serum level of LDL-C was measured by using Friedewald formula (8) for samples with their triglyceride level less than 400mg/ml. subjects with triglycerides higher than 400mg/ml were omitted from the study.

Statistical Analysis

Initially, all data were tested to determine their normal distribution by using Shapiro- Wilk method. For determining the basic differences and effects of type of exercise on blood factors, one-way analysis of variance (ANOVA) was used and for determining the difference between groups Bonferroni post hoc test was used. Paired t-test was also used for determining the difference before and after exercise in the groups. In all cases, a p-value less than 0.05 were considered for indicating the statistical significance. All data were analyzed by using SPSS12 software.

Table 1- Descriptive characteristics of participants at baseline.
Tablica 1. Deskriptivni pokazatelji ispitanika prije aktvnosti

Variables	Aerobic group (n=10)	Resistance group (n=10)	Concurrent group (n=10)
Age (years)	22,3±1,3	23,1±1,4	22,9±1.7
Weight (kg)	68,4±3,6	67,1±5,6	70,7±8,6
Height (m)	168,6±4,2	167,8±4,5	173,8±10,3
BMI ($kg.m^{-2}$)	24,03±0,86	23,83±1,15	23,3±1
Fat body%	18,9±1,2	18,7±1,6	18,1±1,3
VO_{2max} ($ml.kg^{-1}.min^{-1}$)	37,3±2,8	39,9±3,3	37,06±4,6
Waist to Hip (W/H) (cm)	0,89±0,009	0,88±0,02	0,88±0,02
HDL-C ($mg.dl^{-1}$)	47,1±1,4	48,7±2,05	48,8±3
LDL-C ($mg.dl^{-1}$)	97,8±4,9	94,4±4,5	95,3±5,9
TC ($mg.dl^{-1}$)	165,7±4,9	170,5±10,1	164,2±6,4
TG ($mg.dl^{-1}$)	94,9±5,7	93,5±9,1	88,5±11,3
Adiponectin ($\mu g.ml^{-1}$)	7,3±1,4	7,08±1,8	7,44±1,04

RESULTS

Table 1 shows the results of ANOVA test for all variables (pre-exercise). The results indicated that there is no significant difference between three groups, aerobic, resistance and concurrent ($p<0.05$). These results also indicated that three groups were very homogenous. The rates of received calorie in three groups were also compared using repeated measures ANOVA test and it was revealed that there is no significant difference in the rate of calorie absorption among three groups (data were not indicated).

Table 1 shows common properties of subjects. Data analysis indicated that the type of exercise influenced the short-term response of lipids' profile, but it is ineffective for serum adiponectin ($p \leqslant 0.05$) (figure 1). The concentrations of HDL-C (48.7±2.05 before vs. 47.3±1.6 after) and TC (170.5±10.08 before vs. 166.1±7.65 after) were significantly reduced 24 hours after exercise in resistance group (figure 2). The concentration of triglyceride (TG) regardless of the type of exercise (92.3±9.14 before vs. 90.43±8.9 after) considerably reduced 24 hours after exercise. The concentration of LDL-C reduced in resistance group but not significantly (figure 3).

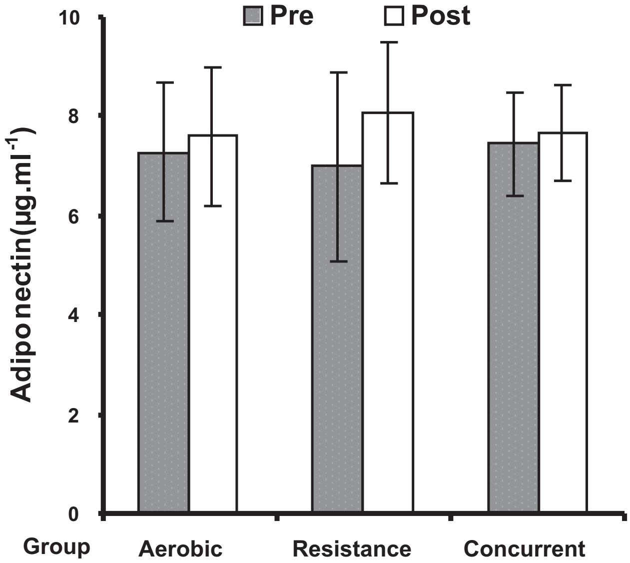
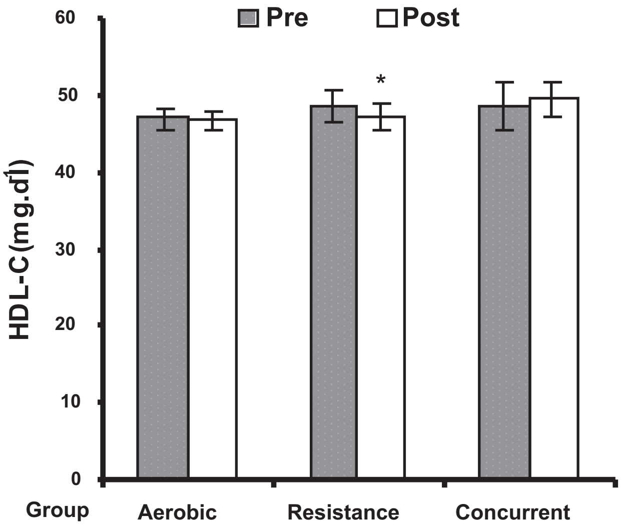


Figure 1. Changes in serum adiponectin concentration before and after exercise. Information has been reported based on mean and standard deviation.
Slika 1. Promjene u koncentraciji serumskog adiponektina prije i poslije aktivnosti.

* Indication of significance level, $p< 0.05$



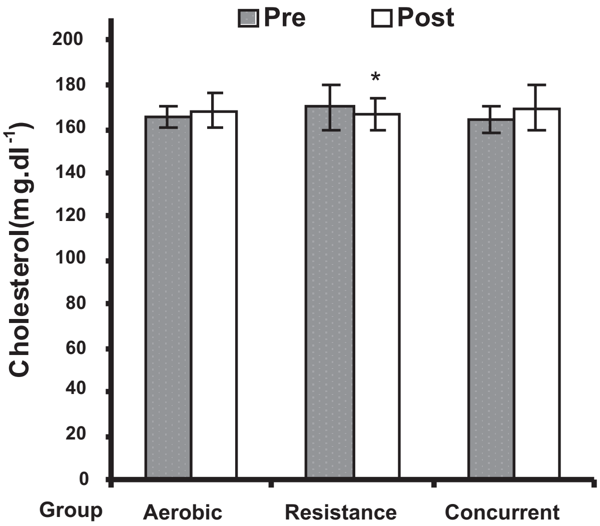


Figure 2 Changes of HDL-C, and total cholesterol concentration before and after exercise. Information has been reported based on mean and standard deviation.
Slika 2. Promjene u koncentraciji HDL-C prije i poslije aktivnosti.

* Indication of significance level, $p< 0.05$

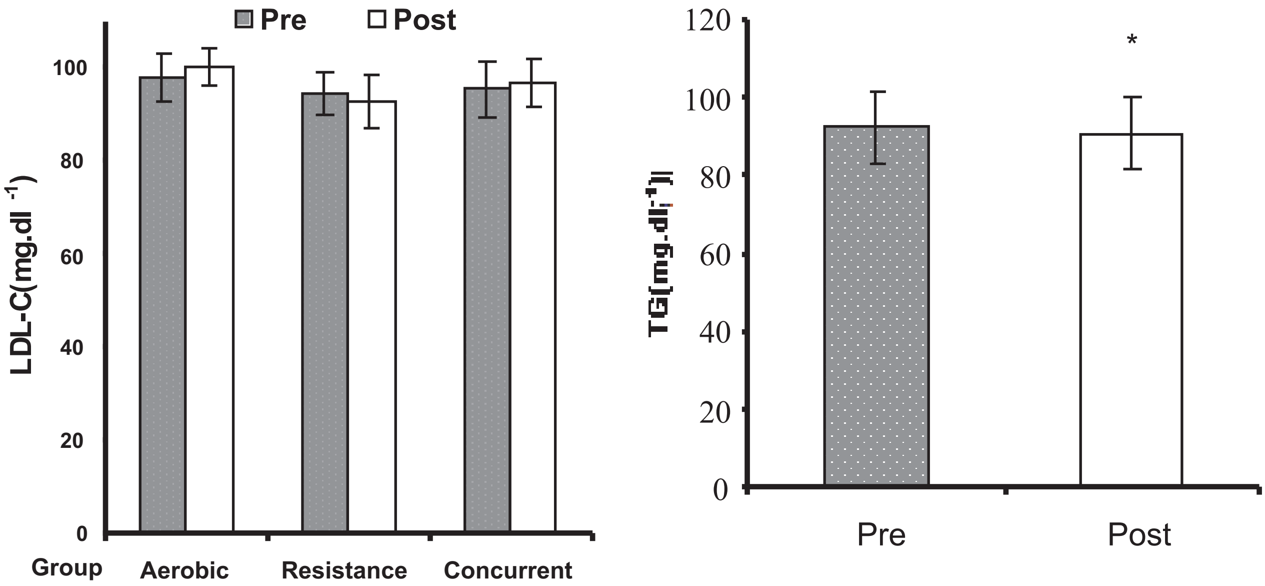


Figure 3. Changes of LDL-C, and triglyceride concentration before and after exercise. Information has been reported based on mean and standard deviation.
Slika 3. Promjene u koncentraciji LDL-C prije i poslije aktivnosti.
* Indication of significance level, $p< 0.05$

DISCUSSION

Results indicated that acute exercise could change the lipid profile response. Concentrations of both TC and LDL-C reduced 24 hours after resistance exercise while there was a slight increase in lipids for two other groups of aerobic and concurrent exercise. LDL-C reduction following the resistance exercise was not significant. In most studies, significant reduction has been observed in TC and LDL-C following acute exercise in trained subjects who performed endurance exercise of long-duration that required a large expenditure of calories (6,24). Reduction in TC and LDL-C in untrained subjects with normal level (9) and hypercholesteremia (2) following low size and short-term exercise has also been indicated. Crouse et al. (2) observed initial reduction in TC and LDL-C in untrained men with hypercholesteremia immediately after acute exercise (450 kcal). The concentration of these lipids frequently increased to such extent that LDL-C concentration was significantly increased 24 hours after and TC 48 hour after short-term exercise in comparison basic values (2). In addition, Kantor et al. (19) reported slight increase in TC and LDL-C concentration, although it was not significant after 24 hours after training for one houre of cycle ergometer exercise in untrained men (9). Therefore, in this study, due to TC and LDL-C reduction by resistance exercise, it is recommended that probably these subjects used more energy or their exercise size was higher than previous studies.

In this study, significant reduction in HDL-C concentration in acute resistance exercise was observed while such a reduction was not observed in aerobic and concurrent exercises. Jurimae et al. (18) reported that HDL-C concentration in untrained subjects did not significantly change after 5min of acute exercise 30min of resistance circle exercise (18). Wallace et al. (36) reported no significant change in HDL-C concentration immediately after one acute resistance exercise with low and high volume in trained men (7). While an increase in the concentration of HDL-C after 24 hours was indicated in acute resistance exercise (800 kcal) with high volume (32). Generally, it was determined that specific volume of exercise is needed to increase the concentration of HDL-C (4). Reports in untrained men with high and normal cholesterol indicate that one session of aerobic exercise consuming 350 to 500kcal energy results in a significant increase in the concentration of HDL-C 24 hours after exercise bout (6). Data of this study propose that regardless of the type of exercise, a significant increase in HDL-C depends on the rate of energy consumption and activity in healthy people.

In this study, the mode of exercise had no effect on the short-term response of TG in healthy and sedentary men. When three exercise groups were combined, a significant reduction in TG 24 hours after exercise was indicated. The same result was observed for the untrained people with hypercholesteremia and normal cholesterol (2,4,11,18,23,36). In comparison with other studies, a reduction in TG concentration following acute endurance exercise in sedentary subjects was observed (9). It has been proposed that people with maximum initial TG tend to show more reduction after exercise (2,9,18). While this study, like some others (2) that used sedentary subjects, indicated that natural concentration of initial TG did not make considerable reduction following acute exercise.

Another study indicated significant difference in the rate of serum adiponectin before and 24 hours after three exercises (aerobic, resistance and concurrent). Previous studies that tested the effect of acute exercise on adiponectin response reported that trained rowers experienced significant reduction in the adiponectin levels immediately after an acute exercise as well as significant increase 30 min after exercise. Kriketos et al. (23) reported a significant increase in the adiponectin levels in sedentary and fat men after a short term exercise program (nearly one week) (23). On the contrary, Ferguson et al. (7) reported the inefficiency of a session of submaximal aerobic training on the concentration of adiponectin in healthy men with normal body weight (7). Similarly, Kraemer et al. (22) proved that there is no significant increase in the adiponectin levels in healthy young men after continuous and heavy running (22). Punyadeera et al. (29) also reported no significant change in the adiponectin levels after the exercise with returning to the initial state in healthy men with normal body weight (29). Exercise studies indicated that exercise might not be the main reason of reducing the adiponectin. It seems that reduction in body weight or body composition may be a changing factor in adiponectin levels. Some reports indicated that following the improvement in the body composition of young fat men, their adiponectin level increased and this is more important than any conducted exercise programs. However, the mechanism of regulation of plasma adiponectin by body weight changes has not yet been indicated. This study investigated the acute exercise, where there was no evidence for body weight changes, and this can be considered as an explanation for the lack of changes on adiponectin levels.

CONCLUSION

Results of this study indicated that response of lipid profile to a session of exercise is a function of type of exercise while type of exercise has no influence on the response of serum adiponectin. According to the results of this study, it is recommended that sedentary young men can enjoy temporary improvement in their lipid profile in addition to attaining the positive muscular-skeleton effects even by one session of resistance exercise. Moreover, if their activity is repeated, it is likely to institutionalize such responses to make them become more stable. In addition, this may reduce the possibility of being affected by chronic diseases.

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References

1. Asayama K, Hayashibe, H, Dobashi K, et al. Decrease in serum adiponectin level due to obesity and visceral fat accumulation in children. Obesity Research, 2003;11(9):1072-9.
2. Crouse SF, O'Brien BC, Rohack, JJ et al. Changes in serum lipids and apolipoproteins after exercise in men with high cholesterol: influence of intensity. J Appl Physiol, 1995;79 (1):279- 86.
3. Dridi S, Taouis M. Adiponectin and energy homeostasis: consensus and controversy. J Nutrit Biochem,2009; 20:831–9.
4. Durstine JL, Haskell WL. Effects of training on plasma lipids and lipoproteins. Exerc Sport Sci Rev,1994;22:477- 521.
5. Elmahgoub SM, Lambers S, Stegen S et al. 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, 2009,168:1327–33.
6. Ferguson MA, Alderson NL, Trost SG et al. Effects of four different single exercise sessions on lipids, lipoproteins, and lipoprotein lipase. J ApplPhysiol,1998; 85 (3):1169-1174.
7. Ferguson MA, White LJ, McCoy S et al. Plasma adiponectin response to acute exercise in healthy subjects. Eur J ApplPhysiol,2004;91(2–3):24–9.
8. Friedewald, WT, Levy RI, Fredrickson S. Estimation of the concentration of low- density lipoprotein cholesterol in plasma without use of preparative ultracentrifuge. Clin Chem,1979;18: 499– 502.
9. Goldberg L, Elliot DL, Schutz RW et al. Changes in lipid and lipoprotein levels after weight training, JAMA, 1984;252(4):504-10 .
10. Goropashnaya AV, Herron J, Sexton, M.et al. Relationships between plasma adiponectin and body fat distribution, insulin sensitivity, and plasma lipoproteins in Alaskan Yup'ik Eskimos: the Center for Alaska Native Health Research study. Metabol Clinic Experim, 2009; 58: 28–9.
11. Grandjean PW, Crouse SF, Rohack JJ. Influence of cholesterol status on blood lipid and lipoprotein enzyme responses to aerobic exercise. J Appl Physiol, 2000;89(2):472- 80.
12. Gregory BD, Shala ED, Walter RT. (2008). ACSM's Health-Related Physical Fitness Assessment Manual. (2 rd edition). Philadelphia, Baltimor: Lippincott Williams & Wilkins.
13. Harris J, Benedict FA. (1919). Biometric study of basal metabolism in man. Washington, DC: Carnegie Institution of Washington.
14. Hernandez-Torres RP, Ramos-Jim´enez, A, Torres-Duran, PV et al. .Effects of single sessions of low-intensity continuous and moderate-intensity intermittent exercise on blood lipids in the same endurance runners. J Sci Med Sport, 2009;12: 23- 31.
15. Jackson AS, Pollock MI. . Generalized equations for predicting body density of men. Brit J Nutrit 1978;40:497-504.

16. Jamurtas AZ, Theocharis V, Koukoulis G. et al. The effects of acute exercise on serum adiponectin and resistin levels and their relation to insulin sensitivity in overweight males. Eur J ApplPhysiol, 2006;97:122–6.
17. Joseph LJO, Davey SL, Evans WJ et al. Differential effect of resistance training on the body composition and lipoprotein-lipid profile in older men and women. Metabolism,1999;48(11):1474-80.
18. Jurimae T, Karelson K, Smirnova T et al. The effect of a single-circuit weight training session on the blood biochemistry of untrained university students. Eur J Appl Physiol, 1990;61:344-8.
19. Kantor MA, Cullinane EM, Sady SP et al. Exercise acutely increases high- density lipoprotein-cholesterol and lipoprotein lipase activity in trained and untrained men. Metabolism, 1987;36 (2):188-92.
20. Kim C, Jina P, Jongsuk, P et al. Comparison of body fat composition and serum adiponectin levels in diabetic obesity and non-diabetic obesity. Obesity,2006;14:1164 – 71.
21. Kokkinos PF, Hurley BF, Smutok MA et al. Strength training does not improve lipoprotein-lipid profiles in men at risk for CHD. Med Sci Sports Exerc, 1991;23(10):1134- 9.
22. Kraemer, RR, Aboudehen KS, Carruth AK, et al. Adiponectin responses to continuous and progressively intense intermittent exercise. Med Sci Sports Exerc,2003;35(8):1320–5.
23. Kriketos AD, Gan SK, Poynten AM et al. Exercise increases adiponectin levels and insulin sensitivity in humans. Diabetes Care,2004;27(2):629–30.
24. Lamon-Fava S, McNamara JR, Farbe. HW et al. Acute changes in lipid, lipoprotein, apolipoprotein, and low-density lipoprotein particle size after an endurance triathlon. Metabolism,1999;38(9):921-25.
25. Lauber RP, Sheard NF. The American Heart Association Dietary Guidelines for 2000: a summary report. Nutr Sci,2001; 59:298–306
26. LeMura LM, Von Duvillard SP, Andreacci,J et al. Lipid and lipoprotein profiles, cardiovascular fitness, body composition, and dieting during and after resistance, aerobic and combination training in young women. Eur J Appl Physiol,2000; 82:451- 58.
27. Marques E, Carvalho J, Soares JMC et al. Effects of resistance and multi component exercise on lipid profiles of older women. Maturitas,2009;63:84–8.
28. Pilz S, Horejsi R, Molle, R et al. Early atherosclerosis in obese juveniles is associated with low serum levels of adiponectin. J Clin Endoc Metab,2005;90(8):4792-6.
29. Punyadeera C, Zorenc AH, Koopman R et al. The effects of exercise and adipose tissue lipolysis onplasma adiponectin concentration and adiponectin receptor expression in human skeletal muscle. Eur J Endocrinol,2005;152(3):427–36.
30. Rokling-Andersen MH, Reseland JE, Veiered MB et al. Effects of long-term exercise and diet intervention on plasma adipokine concentrations. Am J ClinNutr, 2007;86:1293- 301.
31. Sabbaghian Rad L, Gholami M . Impact of exercise training and/or diet on the lipoprotein–lipid profiles in young overweight women. Br J Sports Med, 2010;44:i20 doi:10.1136/bjsm.2010.078972.60.
32. Sallis JF, Haskell WL, Wood PD, Fortmann SP, Rogers T, Blair SN, Paffenbarger RS J.r. . Physical activity assessment methodology in the Five-City Project. Am J Epidemiol,1985;121:91– 106.
33. Siri WE. (1956). Body composition from fluid spaces and density: analysis of methods. University of California Radiation Laboratory Report UCRL. no. 3349.
34. Tongjian Y, Barbara JN. Effects of Exercise on Adipokines and the Metabolic Syndrome.Current Diabetes Reports,2008;8:7- 11.
35. US Department of Health and Human Services. (1996). Physical activity and health: a report of the surgeon general. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention. National Center for Chronic Disease Prevention and Health Promotion.
36. Wallace MB, Moffatt RJ, Haymes EM. et al. . Acute effects of resistance exercise on parameters of lipoprotein metabolism. Med Sci Sports and Exerc,1991;23(2):199-204.
37. Weiss R, Taksali SE, Dufour S. et al. The "obese insulin-sensitive" adolescent: Importance of adiponectin and lipid partitioning. J Clinic Endoc Metab,2005;90(6):3731- 7.
38. Wooten JS, Kyle D, Biggerstaff KD, et al. Response of lipid, lipoprotein-cholesterol, and electrophoretic characteristics of lipoproteins following a single bout of aerobic exercise in women. Eur J ApplPhysiol,2008;104:19–27.