

Acute Plasma Glucose and Lipoproteins Responses to a Single Session of Wrestling Techniques-Based Circuit Exercise (WTBCE) in Male Elite Wrestlers

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Abstract

Introduction: It has been shown that regular endurance exercise is a widely recognized modality to raise plasma high-density lipoprotein cholesterol (HDL-C) levels, but the results reported in studies of the effect of supra-maximal /maximal high intensity or power and anaerobic based sports and exercise on lipoprotein are conflicting. **Objectives:** The purposes of the present study were to investigate the effect of a single session of wrestling technique-based circuit exercise (WTBCE) on acute responses of glucose and lipoprotein profiles and the time course change of these metabolites. **Methods:** Twenty young, male elite wrestlers (age $20 \pm .6$ year, height 172.6 ± 2.10 cm, weight 71.05 ± 3.71 kg., and 24.54 ± 0.63 kg/m² in BMI) volunteered to participate in the present study. **Design and Exercise Protocol:** Subjects were asked to complete a single WTBCE (10 techniques or stations separated by 10m, 3 non-stops circuits for 2 sets and one competitive wrestling practice). Blood samples were collected 30min before, immediately after the exercise, and 30min after the exercise. Repeated Measures Analysis of variance was used to analyze the data. **Results:** Plasma glucose and HDL-C were significantly ($p < 0.001, 0.04$) changed. There was no significant change in TG and TC concentrations. **Conclusion:** The present data indicate that a WTBCE was able to generate an acute change in HDL-C and glucose concentration and it can be considered as a stimulus for improvement of plasma HDL-C levels.

Keywords : Plasma glucose, Lipoproteins, HDL-C, Wrestling technique-based circuit exercise.

Introduction

Regular endurance exercise is a widely recognized modality to raise plasma high-density lipoprotein cholesterol (HDL-C) levels [1-3], which is one of the metabolic adaptations contributing to the reduced risk of coronary heart disease (CHD) observed among physically active and fit individuals [4-6]. Although a low plasma HDL-C concentration is often accompanied by an elevated triglyceride (TG) level associated with abdominal obesity and insulin resistance-hyperinsulinemic states [7, 9], some individuals are characterized by low HDL-C levels without hypertriglyceridemia; a condition that has been referred to as isolated hypoalphalipoproteinemia [10, 11]. In addition, a low HDL-C has been reported in cigarette smoking [12], low fat /low cholesterol diet [13,14], self-administrated exogenous testosterone anabolic and androgenic steroids [15, 16] and physical inactivity [17, 18].

There is an agreement about the effects of an aerobic-based sports/endurance exercise on lipid and lipoprotein profiles, particularly on LDL-C, TG, TC, and HDL-C and its subfractions [19, 20, 21, 22, 23]. In contrast to aerobic-based physical activity, the results from the effects of acute exercise (aerobic or anaerobic) and also a power-anaerobic-based exercise/sports on lipid and lipoprotein profile are inconsistent [24, 25, 26, 27, 28, 29]. Additionally, a low level of HDL-C in power athletes, particularly wrestlers has been reported by some investigators [30, 31, 32]. In this regard, Eliakim et al. [30] suggested that in their study only one of the athletes had high levels of HDL-C (75mg/dl) and 24 athletes had mild low HDL-C (35-45mg/dl). They also reported that hypercholesterolemia and low HDL-C were found mainly in power sports (i.e., weight lifting, boxing, wrestling and judo) and anaerobic sports (i.e., tennis, sprints, and jumps, gymnastics, ice skating). According to a comparison by Tsopanakis and et al [26] a lower HDL- concentration has been observed in elites athletes in Olympic sports such as; wrestling, boxing, and sailing (46mg/dl, 41mg/dl,

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and 45mg/dl respectively). Sgouraki et al [31, 32] reported that after the maximal exercise bout [the ergometric test on treadmill ergometer), a preparation phase and then 5% inclination of the treadmill until the end of exercise test (100% VO₂ max, 12-14min the maximum), all groups from basketball, swimming, long distance running and wrestling- the control included showed statistically significant increase in HDL-C levels as compared to rest values (11.4%, 17.1%, 15.0%, 12.7%, and 13.7%). Wallace et al [33] were studied 10 healthy, trained males (25.4 +/- 3.1 yr) before and after 90 min of resistance exercise to determine the acute effects of high volume (HV) and low volume (LV) sessions on alterations in lipid and lipoprotein concentrations as well as the activity of lecithin: cholesterol acyltransferase (LCAT). A significant changes were only found following the HV session. These included increases in HDL-C (11%) and HDL3-C (12%) 24 h post-exercise. Jurimae et al [34] were studied the lipid and lipoprotein responses to a single-circuit weight-training session in 15 untrained male students. In this study subjects performed three circuits using a work-to-rest ratio of 30 s:30s at 70% of one-repetition maximum and the whole program lasted 30 min. The HDL-C increased in 1-h period of recovery compared with the initial level. In the study by Hill et al [35] two intensities of 1RM were employed. To date, in our knowledge, a single wrestling technique-based circuit exercise has not been considered as a stimulus to bring about a change in lipid and lipoprotein metabolism in wrestlers. Thus, the first aim of this study was to see lipid and lipoprotein profiles in elite wrestlers. The second purpose was to investigate the acute responses of plasma glucose and lipoproteins to a single session of WTBCCE program in this subjects.

Material and Methods

Subjects and Research Design

The study was approved by the ethic committee of the School of Medical Sciences of Tarbiat Modares University and conducted in accordance with the policy statement of the Declaration of the Iranian Ministry of health. Written consent was obtained from the twenty male young elite wrestlers (age 20 ± .6 year, height 172.6 ± 2.10 cm, weight 71.05 ± 3.71 kg, and 24.54 ± 0.63 kg/m² in BMI, 5 ± 1.5 years history in wrestling) who volunteer to participate in the present study. The wrestlers had a high experiences in the national and international wrestling competitions. All subjects were asked to complete a medical examination and a medical questionnaire to ensure that they were not taking

any medication, were free of cardiac, respiratory, renal, and metabolic diseases, and were not using steroids. Also, all the subjects were completely familiarized with all of the experimental procedures.

Exercise Testing Procedures

Before the main trial, participants were taken to the wrestling club three times. The first and second visits of all the participants performed a practice a wrestling-technique-based circuit exercise (WTBCCE) (8 techniques/or stations) for familiarization. On the third visit, the subjects completed a practice session to insure that each participant was able to complete the entire exercise session and also to confirm that the program was producing fatigue at the end of the session. This was confirmed by visual and verbal feedback from the participants. The subjects were allowed to take as long time as they felt necessary to recover from each attempt. The experiment protocol was started at 08.30 AM and finished at 11.30 AM to avoid the effects of circadian rhythm. Subjects were asked to perform 2sets of 3 non-stop circuits exercise (8 techniques/stations with one- repetition for each exercise at their maximum speed) with a 4 minute rest between sets which followed by one competitive wrestling (2×3min with 30s rest between)(Figure.1). In meantime, the distance between each station was 10 meters and whole WTBCCE exercise session lasting for 25 min the maximum (Fig.1). All the exercises were conducted after an overnight fast state. The subjects were instructed to follow a normal lifestyle maintaining daily habit-s, to avoid any medications, and to refrain from exercise 3 days before the experimental day.

Biochemical analyses: Blood samples were obtained from antecubital vein 30 minutes before exercise, immediately after the exercise and 30min following the exercise. Plasma was separated by centrifugation within 15 minutes of collection and divided into three aliquots. The aliquots were frozen and stored at -20°C and -80°C for subsequent analyses (within 3-4 weeks). The samples were analyzed for glucose, TG, TC, HDL-C and LDL-C and VLDL-C. High density lipoproteins (HDL-C) by precipitation method with MgCl₂-Na Phosphotungstate (Men Com Cat No 100, Tehran Iran) , Serum glucose(glucose oxidase, Men Com Cat No 428), TG (lipoprotein lipase and glycerolkinase, Men Com Cat No 337) and TC (Cholesterol esterase, Men Com Cat No 258). LDL-C was also calculated by using two equations as previously described (36, 37). [(TC-[VLDLC + HDLC]=LDLC or (LDLC= TC-(TG/5

+ HDLC)]. The TC/HDLC and LDL/HDLC ratios were adopted according to the atherogenic and CAD (CHD) risk factor indexes were calculated.

Statistics: the data were analyzed using SPSS package (version 10.1) by personal computer. The obtained data for plasma HDL-C glucose, GTG, TC, LDL-C, and VLDL-C were analyzed using analysis variance (ANOVA) with repeated measure variables. Statistical significance was accepted at $p < 0.05$. Significant effects were followed by appropriate planned comparison.

Results

Table 1 shows the mean values (\pm SE) of age, height, body weight (BW), body mass index and other physiological, physical performance, and related parameters. Plasma glucose concentrations significantly changed ($P < 0.001$) from 91 ± 1.2 mg/dl to 153.68 ± 7.5 mg /dl immediately after exercise and still higher and significant ($P < 0.023$) when compared with pre-exercise value (Fig.1). A significant changes were observed in HDL-C levels ($F = 5.018$, $P < 0.025$). HDL-C increased from 43.5 ± 2 mg/dl to 48.2 ± 1.5 mg/dl immediately after exercise. However, an insignificant ($P < 0.4$) reduction was observed in HDL-C levels following 30 min of the exercise recovery period (Fig.1). As presented in Table.2, plasma TG, TC, LDL-C, VLDL-C levels showed an insignificant increase after a single session of WT BCE. An insignificant reduction in LDL/HDL (-0.681 or 20.4%) and in TC/HDL (-0.12) ratios were also observed.

Discussion

The purpose of the present study was to investigate acute plasma glucose and lipoprotein responses to a single session of WT BCE program. The main findings of this study were a significant increased plasma glucose and an elevation in HDL-C level ($+ 4.2$ mg/dl) immediately after exercise. An insignificant changes in plasma TC and TG, and LDL-C concentrations in the present study are in disagreement with Sgouraki et al [32] who observed a significant increase in TG, TC, and LDL-C concentrations after an acute treadmill ergometer test. Hughes et al [38] found no significant difference in TC, TG, and LDL-C measures after 15, 30, and 45 min of an acute exercise at VO_{2max} 20% blow VT. Davis et al [39] did not observed any significant changes in blood lipid variables and LDL-C after an acute exercise at 50% (lasting 90min) and 75% VO_{2max} (lasting 60min). As reported by Imamura et al [40] the concentrations of serum TC, TG, and LDL-C

showed no significant changes after an acute moderate exercise intensity ($60\% VO_{2max}$) for 30 or 60min in duration in sedentary young women. Our results also are in agreement with Jürimäe et al [34] and Wallace et al [33]. In the present study a significant increase in plasma glucose was observed immediately after the exercise program and still significantly higher during 30min of recovery period when compared to before exercise value. Increased in blood glucose are consisted with some of the previous studies using different resistance exercise protocols [34, 43, 44, 45] and it also is disagreement with other reported results [46, 47]. Robergs et al. [43] reported glucose increases due to plasma volume shifts. In regards to HDL-C, post-exercise increased in HDL-C have previously been reported following prolonged endurance events lasting ≥ 2 hr [41, 42]. Sgouraki et al [31] reported that after the maximal exercise bout of the ergometric test on treadmill for 12-14min the maximum, wrestling and control groups showed statistically significant increase in HDL-C levels as compared to rest values (12.7%, and 13.7%). In other study by Sgouraki et al [32] reported that after maximal effort wrestling and control groups showed significant increase in HDL-C levels compared to rest values (13% and 14.4% respectively). Gordon et al [42] reported no significant change sin HDL-C levels immediately and 1h after a single exercise on tread mill at 60 and 75% VO_2 max. the same results also were reported by Gordon et al (1996)[48]. Angelopoluos et al [49] evaluated the effect of single bout and repeated bouts (30min) of treadmill exercise on HDL-c and its subfractions. They pointed out that total HDL-C remained higher significantly than the pre-exercise values 5 min after the exercise. In the study by Hughes et al [38] who used different exercise duration (15, 30, 40min) at certain intensity (VO_{2max} 20% below ventilatory threshold) to investigate any changes in serum lipoprotein metabolism. They were found no significant changes in HDL in all exercise durations. Davis et al [39] studied the effects of acute exercise intensity on plasma lipid s in well trained runner in a high-intensity (at 75% VO_{2max} and for 60min) and low intensity session (at 50% VO_{2max} and for 90min). They did not observed any significant changes in HDL-C and HDL-C2 levels after exercise. Ferguson et al [51] who reported that HDL-C concentration was significantly elevated immediately after and 48h after exercise in the 1500kcal session. An increase in HDL-C levels (2 ± 4 mg/dl) after a bicycle ergometer exercise at 80% of HR max in trained men was reported by Kantor et al [52]. Wallace et

Set	Rounds	Stations	Total performed Techniques
1	3	8	24
3min rest (semi-active recovery)			
2	3	8	24
3min rest (semi-active recovery)			
3	2x3min (30s rest between) Competitive wrestling		

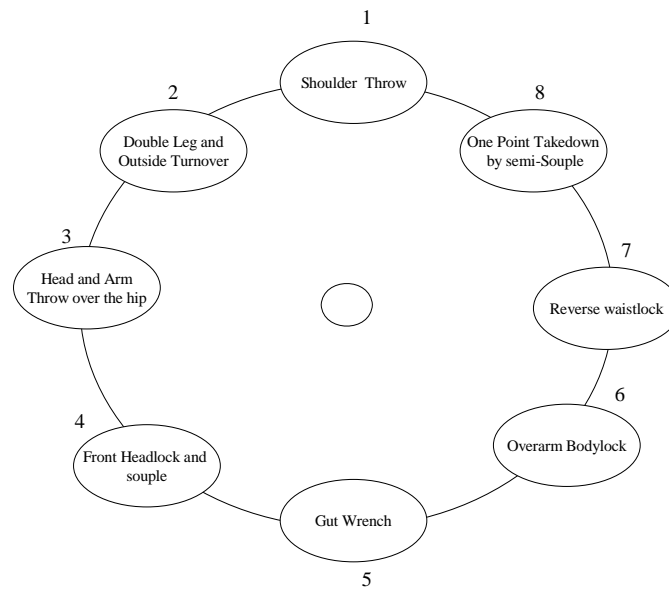


Figure 1: An experimental working plan

Table1: Physical characteristics of the participants (mean \pm standard error, n =20)

Variables	Values
Age (year)	20 \pm 0.69
Weight (kg)	74 \pm 3.71
Height (cm)	172.5 \pm 2.10
BMI (Kg/m ²)	24.54 \pm 0.63
HR _{Rest} (beat/min ⁻¹)	69 \pm 2
CHR _{max1} (beat/min ⁻¹)	173 \pm 3
CHR _{max2} (beat/min ⁻¹)	177.0 \pm 2
Wrestling HR _{Rest}	126.71 \pm 4.35
Wrestling HR _{max}	179.23 \pm 3.87
Avrage Time1 (min)	1.40 \pm 0.029
Avrage Time2 (min)	1.42 \pm 0.042
Practice sessions/week	3-4
Wrestling Experiences (y)	7.62 \pm 1.23

Table2: Biochemical variables: plasma TC, TG, LDL-C, VLDL-C concentrations and TC/HDL and LDL/HDL-C ratios. Values are mean \pm SE. * $p < 0.05$, ** $p < 0.01$ compared with pre-exercise. + $p < 0.05$, 30-Postexercise compared with pre-exercise value.

Variables	Pre-Exe	Post-Exe	30 min Post-Exe
TC (mg/dl)	163 \pm 5.6	175 \pm 6.3	165 \pm 5.6
TG (mg/dl)	108 \pm 11	121 \pm 11	115 \pm 12
LDL-C (mg/dl)	99 \pm 6.4	102 \pm 7.4	97 \pm 6
VLDL-C(mg/dl)	21.7 \pm 2	24.3 \pm 2	22.5 \pm 2.4
TC/HDL-C Ratio	3.9 \pm 0.3	3.8 \pm 0.2	4.1 \pm 0.2
LDL/HDL-C Ratio	2.4 \pm 0.22	3 \pm 0.8	2.5 \pm 0.2

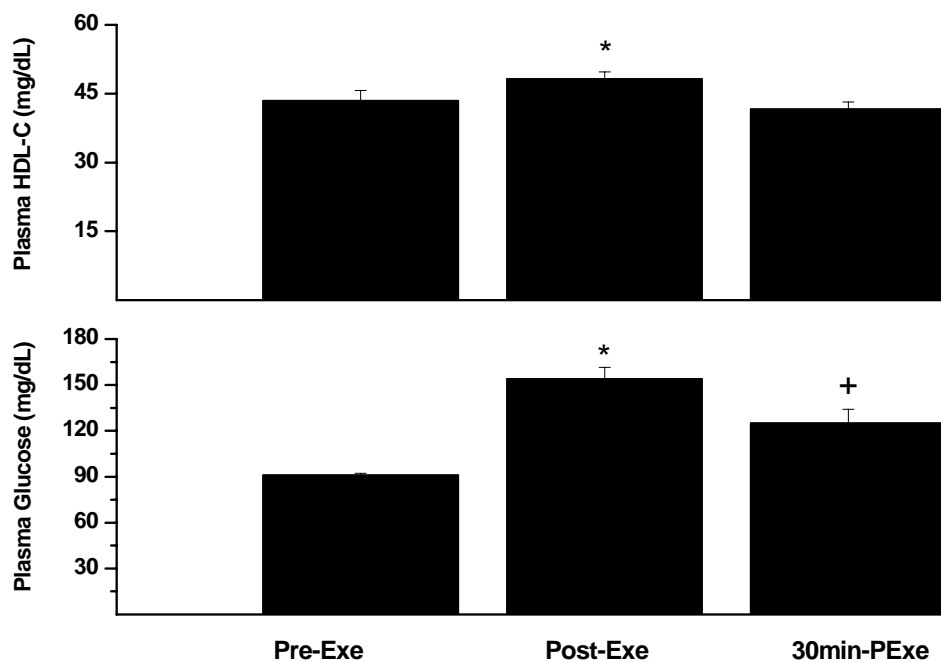


Figure 2: Plasma glucose and HDL-C concentrations to a single wrestling technique-based circuit exercise (WTBCE) plus a competitive wrestling practice before, immediately after exercise, and after 30min of recovery. Values are mean \pm SE. * $p < 0.05$, ** $p < 0.01$ compared with pre-exercise. + $p < 0.05$, 30min Post-exercise compared with pre-exercise value.

al [33] were studied the effects of 90 min of resistance exercise with different volumes (high volume and low volume) on alterations in lipid and lipoprotein concentrations as well as the activity of lecithin: cholesterol acyltransferase (LCAT). A significant changes were only found following the high volume session. These included increases in HDL-C (11%) and HDL3-C (12%) 24 h post-exercise. Jürimäe et al [34] who reported that the plasma HDL-C levels were insignificantly increased during a circuit resistance exercise (ten exercises, three circuits using a work-to-rest ratio of 30 s:30s at 70% of one-repetition maximum and for 30min), but after a 1-h period of recovery the concentration was significantly higher than before exercise. As reported by Burns et al [53] plasma HDL-C concentration were significantly lower after a single resistance exercise trail than in the

control trail. Wooten et al [54] did not find a significant change in HDL-C after an acute circuit resistance exercise and training. Our result is also in agreement with by Hill et al [35] who pointed out that the only significant effect of exercise in their experimental condition was to acutely increase in HDL-C in the immediate post exercise sample compared with the control. A discrepancies between our results with other previous reported findings, first of all could be explained by the general factors such as duration, intensity or energy expenditure per session [39, 55], interest resting period, mode of exercise [56], and diet [57]. The initial levels of plasma HDL-C is also can be considered as a determining factor. As reported by Eliakim et al [30] how suggested that in their study the hypercholesterolemia and low HDL-C were found mainly in power sports (i.e.,

weight lifting, boxing, wrestling and judo) and anaerobic sports (i.e., tennis, sprints, and jumps, gymnastics, ice skating). The same result was reported by Tsopanakis et al [25]. The underlying biochemical mechanism responsible for acute increase in HDL-C after a single WT BCE plus wrestling practice session is complex. Enzymes such as lipoprotein lipase (LPL), hepatic triglyceride lipase (HL), and lecithin: cholesterol acyltransferase (LCAT) and cholesterol ester transport protein (CETP) play a role in mediating HDL-C concentration change. Acute increase in HDL-C probably related to catabolism of triglyceride rich lipoproteins via lipoprotein lipase [58, 52, 59, 51]. It has been suggested that the increases reported for HDL-C after endurance exercise, may be partly due to reduce recycling and catabolism velocities of lipoprotein in athletes compared to controls, than to lipoprotein increases *per se* (60). In addition to lipoprotein lipase (LPL), lecithin: cholesterol acyltransferase (LCAT) is known as a plasma factor in HDL-C remodeling which esterifies cholesterol in HDL particles, permitting its transports in HDL core and increase in cholesterol per HDL particles. An increased LCAT activity was reported by several investigators [61, 62, 63]. A post-exercise higher level of HDL-C has been attributed to a significant changes in HDL-C subpopulations, such as HDL2-C and HDL3-C [4, 58, 49, 42, 48, 27, 55, 51, 31, 32, 64, 65, 66]. The HDL-C increased HDL-C concentration immediately after exercise may have been related to decrease CETP activity or concentration. CETP is responsible for the shuttling of lipid between HDL and other lipoproteins, and has been shown to decrease after exercise [67, 68]. The reverse cholesterol transport (RCT) has been reported after physical exercise in untrained subjects and athletes [69, 70, 71, 72, 73]. In this regard, Campaigne et al [74] reported that HDL-C was significantly increased after exercise for 30min on a cycle ergometer at 60% VO_{2max} . They also pointed out that cholesterol efflux was higher to HDL-C obtained from sedentary group compared with runner group before exercise. They conclude that acute exercise increased HDL's ability to act as an acceptor of cellular cholesterol in runner, whereas it decreased in sedentary group. In addition to above factors, the effects of exercise on apolipoproteins, particularly Apo-AI and AII [26, 75, 76] and pre beta-HDL has been reported by recent studies [77, 78]. This is the first report demonstrating that wrestling technique -based circuit exercise plus a competitive wrestling practice resulted in an increased HDL-C and glucose

concentrations.

Our results suggested that as single wrestling technique-based circuit exercise plus a competitive wrestling was able to bring a change in plasma glucose, lipid and lipoprotein profiles. Although we found an insignificant increase in TC, TG, and LDL-C, but the exercise program was more effective on the plasma HDL-C concentration. The HDL-C concentration between subjects was varied from 29-64 mg/dl with averages 41-47mg/dl. Thus, our data partially initially low HDL-C in wrestlers. The present results also suggest that this exercise program can be considered as a stimulus for lipid and lipoprotein changes. It has pointed out that we did not measure HDL-C changes during the exercise program and other plasma factors which are involve in HDL-C remodeling. Further work should investigate the possible role of pre-beta HDL and ATP-binding cassette transporter (ABC) family, particularly A-I (ABCA1) in cholesterol efflux and RCT processes during and after the WT BCE program.

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References

- 1-Durstine JL, Grandjean P, Davis PG, et al (2001). Blood lipid and lipoprotein adaptation to exercise: A quantitative analysis. *Sports Med* 31(15):1033-1062.
- 2-Taylor PA, Ward A (1993). Women, high density lipoprotein cholesterol, and exercise. *Arch Intern Med* 153: 1178-1184.
- 3-Schwartz RS, Cain KC, Shuman WP, et al (1992). Effect on intensive endurance training on lipoprotein in young and older men. *Metabolism* 41(6): 649-654.
- 4-Dufaux B, Assmann G, Hollmann W (1982). Plasma lipoproteins and physical activity [review]. *Int J sports Med* 3:123-126.
- 5-Suzuki I, Yamada H, Sugiura T, et al (1998). cardiovascular fitness, physical activity and selected coronary heart disease risk factor in adults. *J Sports Med Phys Fitness* 38(2):149-157.
- 6-Williams PT (1996). High-density lipoprotein cholesterol and other risk factors for coronary heart disease in female runner. *N Engl J Med* 334:1298-1303.
- 7-Rashid S, Watanabe T, Sakaue T, et al (2003). Mechanism of HDL lowering in insulin resistant, hypertriglyceridemic states: the combined effect of HDL triglyceride enrichment and elevated hepatic

- lipase activity. *Clin Biochem* 36:421-429.
- 8-Taskinen M-R. Diabetic dyslipidemia (2002). *Atherosclerosis Suppl* 3:47-51.
- 9-Pascot A, Lemieux I, Prud'homme D, et al (2001). Reduced HDL particle size as an additional feature of the atherogenic dyslipidemia of abdominal obesity. *J Lipid Res* 42:2007-2014.
- 10-Hersberger M and Eckardstien (2003). Low high density lipoprotein cholesterol. *Drug* 63(18):1907-1945.
- 11-Goldbourt U, Yaari S, Medaile JH (1997). Isolated low HDL cholesterol as a risk factors for coronary disease mortality: a 21-year follow-up of 8000 men 17:107-113.
- 12-Imamura H, Teshima K, Miyamoto N, Shirota T (2002). Cigarette smoking, high density lipoprotein cholesterol subfractions, and lecithin:cholesterol acyltransferase in young women. *Metabolism* 51(10):1313-1316.
- 13-Brinton EA, Eisenberg S, Breslow JL (1990). A low fat diet decrease high density lipoprotein (HDL) cholesterol levels by decreasing HDL apoprotein transport rates. *J Clin Invest* 85:144-151.
- 14-Gorge W (1996). High-fat, high cholesterol diet raises plasma HDL cholesterol: studies on mechanism of this effect. *Nurt Rev* 54(1):34-37.
- 15-Allen M, et al (1984). Reduced high density lipoprotein cholesterol in power athletes use male sex hormone derivatives an atherogenic factor. *Int J Sports Med* 5:341-342.
- 16-Allen M, et al (1985). Serum lipid in power athletes self-administration reduce high density lipoprotein cholesterol in power athletes use of male sex hormones derivatives an atherogenic factor. *Int J Sports Med* 6:139-144.
- 17-Tikkanen HO, Hämäläinen E and Härkönen (1999). Significant of skeletal muscle properties on fitness, long-term physical training and serum lipids. *Atherosclerosis* 142: 367-378.
- 18-Wood PD, Haskell WL, Blair SN, et al (1983). Increased exercise levels and plasma lipoprotein concentration: a 1 years, randomized controlled study in sedentary, middle-aged men. *Metabolism* 32:31.
- 19-Williams PT, Krauss RM, Wood PD, Lindagren FT, Giotas C and Vraizan KM (1986). Lipoprotein subfractions of runners and sedentary men. *Metabolism* 35(1): 45-52.
- 20-Thompson PD, Cullinane EM, Sady SP, et al (1991). HDL metabolism in endurance athletes and sedentary men. *Circulation* 84: 140-152.
- 21-Williams PT, Stefanick ML, Vranizan KM, et al (1994). The effects of weight loss by exercise or dieting on plasma high density lipoprotein (HDL) levels in men with low, intermediate and normal to high at baseline. *Metabolism* 43:917-924.
- 22-Hartung GH (1995). Physical activity and high density lipoprotein cholesterol. *J Sports Med Phys Fitness* 35(1):1-5.
- 23-Couillard C, Despres J-P, Lamarche B, et al (2001). Effects of endurance exercise training on plasma cholesterol levels depend on levels of triglycerides. *Arterioscler Thromb Vas Biol* 21: 1226-1232.
- 24-Aellen R, Hollmann W, Boutellier U (1993). Effects of aerobic and anaerobic training on plasma lipoproteins. *Int J Sports Med*. Oct;14(7):396-400.
- 25-Tsopanakis C, Kotsarellis D, Tsopanakis A (1986). Lipoprotein and lipid profiles of elite athletes in Olympic sports. *Int J Sports Med* 7(6):316-321.
- 26-Jauhiainen M, Laitinen M, Pettula I, et al (1985). lipids and apolipoproteins A-I, B, and C-II and different rapid weight loss programs (weight lifters, wrestlers, boxers and judoka). *Int j Biochem* 17(2): 167-174.
- 27-Pronk NP, Crouse SF, O'Brien BC, Rohack JJ (1995). Acute effects of walking on serum lipids and lipoproteins in women. *J Sports Med Phys Fitness* 35:50-58.
- 28-Hurley BF, Seals DR, Hagberg JM, Goldberg AC, et al (1984). High-density-lipoprotein cholesterol in bodybuilders v powerlifters. Negative effects of androgen use. *JAMA*. 252(4):507-13.
- 29-Giada G, Zuliani G, Baldo-Enzi G, et al (1996). Lipoprotein profiles, diet and body composition in athletes practicing mixed an anaerobic activities. *J Sports Med Phys Fitness* 36:211-6.
- 30-Eliakim A, et al (2002). Screening blood test in member national Olympic team. *J Sports Med Phys fitness* 42: 250-255.
- 31-Sgouraki A, Tsopanakis A, Tsopanakis C (2001). Acute exercise: response of HDL-C subfractions levels in selected sport disciplines. *J Sports Med Phys Fitness* 41:386-389.
- 32-Sgouraki A, Tsopanakis A, Kioussis A, Tsopanakis C (2004). Acute effects of short duration maximal endurance exercise on lipid, phospholipid and lipoprotein levels. *J Sports Med Phys Fitness* 44:444-450.
- 33-Wallace MB, Moffatt RJ, Haymes EM, Green NR (1991). Acute effects of resistance exercise on parameters of lipoprotein metabolism. *Med Sci Sports Exerc* 23 (2):199-204.
- 34-Jürimäe T, Karelson K, Smirnova T, Viru A (1990). The effect of a single-circuit weight-training session on the blood biochemistry of untrained university students. *Eur J Appl Physiol Occup Physiol*. 61(5-6):344-8.
- 35-Hill S, Birmingham MA, Knight PK (2005). Lipid metabolism in young men after acute resistance exercise at two different intensities. *J Sci Med Sports* 8(4):441-445.
- 36-Friedewald Wt, Levy RI, Fredrickson DS (1972). Estimation of the concentration of low intwnsity lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. *Clin Chem* 18:499-504.
- 37-Bachorick PS (2001). Method for determining LDL cholesterol In *Clinical diagnosis and management by Laboratory Methods*. Edited by Henry JB 12th edition W.B. Saunders Company. USA, chapter,12, pp234-238.
- 38-Hughes RA, Thorland WG, Eyford T, Hood T (1990). The acute of exercise on serum lipoprotein metabolism. *J Sports Med Phys Fitness* 30(1): 37-44.
- 39-Davis PG, Bartoli WP, Durstine JL (1992) Effects of acute exercise intensity on plasma lipids and

- apolipoproteins in trained runners. *J Appl Physiol*; 72(3): 914-919.
- 40-Imamura H, Katagiri S, Uchid K, et al (2000). Acute effects of moderate exercise on serum lipids, lipoproteins and apolipoproteins in sedentary young women. *Clin Exp Pharmacol Physiol* 27(12): 975-979.
- 41-Lennon DL, Stratman FW, Shrago E, et al (1983). Total cholesterol and HDL-C changes during acute moderate intensity exercise men and women. *Metabolism. Clin Exper* 32 (30):244-249.
- 42-Gordon PM, Goss FL, Visich PS, et al (1994). The acute effects of exercise intensity on HDL-C metabolism. *Med Sci Sports Exerc* (6):671-7.
- 43-Robergs RA, Pearson DR, Costill DL, et al (1991). Muscle glycogenolysis during differing intensities of weight-resistance exercise. *J Appl Physiol* 70(4):1700-1706.
- 44-Kraemer RR, Durand RJ, Hollander DB, et al (2004). Ghrelin and other glucoregulatory hormone responses to eccentric and concentric muscle contractions. *Endocrine* 24(1): 93-98.
- 45-Kraemer WJ, Volek JS, Bush JA, et al (1998). Hormonal responses to consecutive days of heavy-resistance exercise with or without nutritional supplementation. *J Appl Physiol* 85(4).
- 46-Haff GG, Koch AJ, Potteiger JA, et al (2000). Carbohydrate supplementation attenuates muscle glycogen loss during acute bouts of resistance exercise. *Int J Sports Nutr Exerc Metab* 10:326-339.
- 47-Haff GG, Schroder CA, Koch AJ, et al (2001). The effects of supplemental carbohydrate ingestion on intermittent isokinetic leg exercise. *J Sports Med Phys Fitness* 41:216-222.
- 48-Gordon PM, Visich PS, Goss FL, et al (1996). Comparison of exercise and normal variability on HDL cholesterol concentrations and lipolytic activity. *Int J Sports Med* 17(5): 332-337.
- 49-Angelopoulos TJ, Robertson RJ, Goss FL, et al (1993). Effect of repeated exercise bouts on high density lipoprotein cholesterol and its subfractions HDL-2C, and HDL-3C. *Int J Sports Med* 14(4): 196-201.
- 50-Hughes RA, Thorland WG, Eyford T, Hood T (1990). The acute effects of exercise duration on serum lipoproteins metabolism. *J Sports Med Phys Fitness* 30(1): 37-44.
- 51-Ferguson MA, Alderson NL, Trost SG, et al (1998). Effects of four different single exercise on lipids, lipoproteins, and lipoprotein lipase. *J Appl Physiol* 85(3): 1169-117.
- 52-Kantor MA, Cullinane EM, Sady SP, et al (1987). Exercise acutely increase high density lipoprotein-cholesterol and lipoprotein lipase activity in trained and untrained men. *Metabolism* 36(2); 188-192.
- 53-Burns SF, Corrie H, Holder E, et al (2005). A single session of resistance exercise does not reduce postprandial lipaemia. *J Sports Sci* 23(3): 251-260.
- 54-Wooten JS, Hook CP, Henderson CL, et al (2003). Acute response of lipid and lipoprotein concentrations following treadmill and circuit resistance exercise. *Med Sci Sports Exerc* 35(5): S86.
- 55-Carous SF, O'Brien BC, Grandjean PW, et al (1997). Effect of training and a single session of exercise on lipids and apolipoproteins in hypercholesterolemic men. *J Appl Physiol* 83(6): 2019-2028.
- 56-Altana TS, Michaelson JL, Ball SD, Thomas TR (2004). Single sessions of intermittent and continuous exercise and postprandial lipemia. *Med Sci Sports Exerc* 36(8):1364-1371.
- 57-Boss MC, Davis SC, Puhl SM, et al (2004). Effects of zone diet macronutrient proportions on blood lipid, blood glucose, body composition, and treadmill exercise performance. *Nutr Res* 24:521-530.
- 58-Kantor M, Cullinane EM, Herbert PN, Thompson (1984) Acute increase in lipoprotein lipase following prolonged exercise. *Metabolism* 33:454-457.
- 59-Visich PS, Goss L, Gordon PM, et al (1996) Effects of exercise with varying energy expenditure on high density lipoprotein cholesterol. *Eur J Appl Physiol* 72: 242-248.
- 60-Herbert PN, Bernier DN, Cullinane EM, et al (1984). High density lipoprotein metabolism in runner and sedentary men. *JAMA* 252(8): 1034-1037.
- 61-Thomas TR, Adeniran SB, Iltis PW, Aquiar CA, Albers JJ (1985). Effects of interval and continuous running on HDL-cholesterol, apoproteins A-1 and B, and LCAT. *Can J Appl Sport Sci* 10(1):52-59.
- 62-Grandjean PW, Crouse SF, O'Brien BC, Rohack JJ, Brown JA (1998). The effects of menopausal status and exercise training on serum lipids and the activities of intravascular enzymes related to lipid transport. *Metabolism* 47(4):377-83
- 63-Weise SD, Grandjean PW, Rohack JJ, Womack JW, Crouse SF (2005). Acute changes in blood lipids and enzymes in postmenopausal women after exercise. *J Appl Physiol* 99(2):609-615.
- 64-Ferguson MA, Alderson NL, Trost SG, et al (2003). Plasma lipid response during exercise. *Scand J Clin Lab Invest* 63:73-80.
- 65-Park DH and Ransone JW (2003). Effect of submaximal exercise on high density lipoprotein cholesterol subfractions. *Int J Sports Med* 24: 245-251.
- 66-Kelley GA, Kelley KS (2005). Aerobic exercise and HDL₂-C: A meta-analysis of randomized controlled trials. *Atherosclerosis xxx:xxx-xxx*.
- 67-Seip RL, Moulin P, Cocke T, Tall A, Kohrt WM, Mankowitz K, Semenkovich CF, Ostlund R, Schonfeld G (1993). Exercise training decreases plasma cholesteryl ester transfer protein. *Arterioscler Thromb Sep* 13(9):1359-67.
- 68-Wilund KR, Ferrell RE, Phares DA, Goldberg AP, Hagberg JM (2002). Changes in high-density lipoprotein-cholesterol subfractions with exercise training may be dependent on cholesteryl ester transfer protein (CETP) genotype. *Metabolism. Jun* 51(6):774-8
- 69-Gupta AK, Ross EA, Myers JN, et al (1993). Increased reverse cholesterol transport in athletes. *Metabolism* 42:684-690.
- 70-Leaf DA (2003). The effect of physical exercise on reverse cholesterol transport. *Metabolism* 52(8): 950-957.
- 71-Brites F, Verona J, De Geitere C, Fruchart JC, Castro

- G, Wikinski R (2004). Enhanced cholesterol efflux promotion in well-trained soccer players. *Metabolism*. 53(10):1262-7.
- 72-Olchawa B, Kingwell BA, Hoang A, Schneider L, Miyazaki O, Nestel P, Sviridov D (2004). Physical fitness and reverse cholesterol transport. *Arterioscler Thromb Vasc Biol* 24(6):1087-91.
- 73-Wei C, Penumetcha M, Santanam N, Liu YG, Garelnabi M, Parthasarathy S (2005). Exercise might favor reverse cholesterol transport and lipoprotein clearance: potential mechanism for its anti-atherosclerotic effects. *Biochim Biophys Acta*. 1723(1-3):124-7
- 74-Campaigne BN, Fontaine RN, Park MS, Rymaszewski ZJ (1993). Reverse cholesterol transport with acute exercise. *Med Sci Sports Exerc* 25(12):1346-51.
- 75-Lamon-Fava S, Fisher EC, Nelson ME, Evans WJ, Millar JS, Ordovas JM, Schaefer EJ (1989) Effect of exercise and menstrual cycle status on plasma lipids, low density lipoprotein particle size, and apolipoproteins. *J Clin Endocrinol Metab* Jan;68(1):17-21.
- 76-Wilund KR, Colvin PL, Phares D, Goldberg AP, Hagberg JM (2002). The effect of endurance exercise training on plasma lipoprotein AI and lipoprotein AI:AII concentrations in sedentary adults. *Metabolism* 51(8):1053-60.
- 77-Jafari M, Leaf DA, Macrae H, Kasem J, O'conner P, Pullinger C, Malloy M, Kane JP (2003). The effects of physical exercise on plasma prebeta-1 high-density lipoprotein. *Metabolism* 52(4):437-42.
- 78-Sviridov D, Kingwell B, Hoang A, Dart A, Nestel P (2000). Single session exercise stimulates formation of pre beta 1-HDL in leg muscle. *J Lipid Res* 44(3):522-6.