J. Phys. Ther. Sci.
26: 1825–1829, 2014

Original Article

Effect of Aerobic Exercise on Risk Factors of Cardiovascular Disease and the Apolipoprotein B / Apolipoprotein A-1 Ratio in Obese Woman

Dae-Young Kim, PhD[1)], Sun-Young Jung, PhD, PT[2)*]

[1)] *Research Institute of Sports Medicine, Department of Protection Science, Kyungwoon University, Republic of Korea*
[2)] *Department of Physiology, College of Medicine, Kyung Hee University: 1 Hoigi-dong, Dongdaemoon-gu, Seoul 130-701, Republic of Korea*

Abstract. [Purpose] The objective of this study was to confirm whether consistent aerobic exercise has an effect on the apolipoprotein B/apolipoprotein A-1 ratio or reduces the risk of cardiovascular disease in obese women. [Subjects and Methods] The participants included 32 obese women between the ages of 40 and 49. Subjects were randomly divided into two groups (n = 16 in each group): the control group and the exercise group. The exercise program in this study corresponded to an intensity of 50 to 60% of the maximum volume of minute oxygen consumption and was performed three times per week over 12 weeks. Physical measurements, measurement of cardiorespiratory fitness and blood pressure, and blood collection were done before and after the 12 weeks of exercise at the same time and under the same conditions. [Results] Based on the results of this study, there were significant interaction effects in both time and group weight, for body mass index, percent body fat, maximum volume of minute oxygen consumption, high-density lipoprotein cholesterol, and the apolipoprotein B/apolipoprotein A-1 ratio. Moreover, waist circumference, total cholesterol, and the atherogenic index decreased significantly after 12 weeks of aerobic exercise. [Conclusion] Regular aerobic exercise effectively improved cardiovascular risk factors and decreased the obesity index in obese women.
Key words: Aerobic exercise, Apolipoprotein, Cardiovascular disease

(This article was submitted Apr. 16, 2014, and was accepted May 21, 2014)

INTRODUCTION

Obesity is increasing rapidly throughout the world and is a threatening public health problem in many countries. The World Health Organization (WHO)[1)] defines obesity as a disease that exerts a negative influence on health and well-being through the accumulation of excess fat. Representative pathologies accompanying obesity include cardiovascular disease (CVD), hypertension, hyperlipidemia, diabetes, stroke, cancer, degenerative arthritis, and lung diseases including sleep apnea. One of the first problems encountered in chronic disease of obese individuals is hyperlipidemia, consisting of elevated blood cholesterol and triglyceride (TG) levels, the most important independent risk factor of atherosclerosis and an important cause of coronary artery disease (CAD)[2)]. Research to date points to high TG, total cholesterol (TC), low-density lipoprotein cholesterol (LDLC), and high-density lipoprotein cholesterol (HDLC) as key risk factors for CAD[3)]. However, after obtaining an enhanced understanding of lipoprotein function and metabolism, rather than measuring traditional risk factors such as TC, TG, HDLC, and LDLC, measuring apolipoprotein, apo- forming blood lipids, and lipoprotein was recently deemed to be more predictive[3)]. Apo is a key protein that forms many types of lipids and acts as a coenzyme, either accelerating or suppressing functions related to transport and re-distribution and metabolism of lipoproteins through the actions of tissue receptor ligands[4)]. Apo A is divided into the A I, AII, and AIV types, which are important components of HDLC formation. Among these, apoA1 has a static correlation with HDLC, and the expression level of apoA1 is determined by the HDLC concentration. Moreover, it eliminates excess cholesterol in the body, converts cholesterol to HDL, and activates lecithin cholesterol acyl transferase (LCAT), which is responsible for reverse cholesterol transport (RCT) to the liver[3)]. It is known that blood HDLC levels are inversely correlated with CVD risk factors. According to related epidemiological studies, for every 1 mg/dL increase in HDLC, the risk for CAD is lowered by 2–3%, and the risk of death is lowered by 6%[5)]. HDLC undergoes a complex biosynthesis and performs many widely known functions within the body, including diverse protective mechanisms against atherosclerosis[6)]. Blood HDLC is composed of diverse types of lipoprotein based on the surface polarity, and apoA1 is known to play

*Corresponding author. Sun-Young Jung (E-mail: strongsun@hanmail.net)

Table 1. Physical characteristics of the participants in this experiment

Variable	Age (years)	Height (cm)	Weight (kg)	BMI (kg/m^2)	Body fat (%)
CON (N=16)	45.8±3.4	156.1±3.2	64.3±3.1	26.3±1.3	34.3±2.7
Exercise (N=16)	47.0±2.9	158.1±4.2	65.1±3.1	26.0±1.2	32.8±3.2

Data are means ± SD.

the most pivotal role in the production and functioning of HDLC[6]. On the other hand, apoB particles constituting LDL, intermediate-density lipoprotein (IDL), and very-low-density lipoprotein (VLDL), combine the LDL particle with the LDL receptor, contributing to the absorption of cellular cholesterol. Apolipoprotein B (apoB) anchors particles responsible for creating atheroma in the arterial walls and pulls in complete lipoproteins, forming foam cells from macrophages and the subendothelium[7]. Therefore, high blood levels of ApoB are known as causal factors that increase the risk of CAD[3]. Such a ratio between apolipoprotein B and apolipoprotein A-1 (apoB/apoA1 ratio) forms a strong correlation with metabolic syndrome and CVD[7, 8]. Recently, blood apoB levels and apoA1 concentrations have been suggested to have a strong impact on traditionally used LDLC and HDLC[9]. Moreover, the apoB/apoA1 ratio has an independent correlation with atherosclerosis, and it has been reported that individuals with high apoB/apoA1 ratios have greater metabolic risk factors[10].

Methods of prevention and treatment of obesity and cardiovascular disease currently include exercise, diet control, and pharmacotherapy. Among these, pharmacotherapy is less effective and has multiple side effects, so it is not widely implemented, whereas diet control is effective for weight loss but lowers the resting metabolic rate, which has the disadvantage of decreasing both body fat and lean body weight.

Especially for women, who have relatively less muscle mass and more body fat, a specific method to improve CVD risk factors and decrease weight is needed. One of the most effective methods is regular exercise, which decreases body fat, lowers blood pressure, improves hyperlipidemia, and lowers HDLC levels. Increases in metabolic equivalents and cardiorespiratory fitness (CRF) decrease metabolic risk factors and effectively prevent CVD[11, 12]. Therefore, this research sought to solidify the exercise-related physiologic evidence for regular aerobic exercise and the subsequent improvement in cardiovascular risk factors in obese women and the apolipoprotein B/A-1ratio in obese women.

SUBJECTS AND METHODS

The study subjects included a total of 32 obese women between the ages of 40 and 49 without medical problems. Subjects were randomly divided into two groups (n = 16 in each group): the control group and the exercise group. They were selected after basic testing, which included collection of information for age, exercise capacity, weight changes over the previous year, smoking status, and currently used medications, and requirements included waist circumference (WC) ≥80 cm and body mass index (BMI) ≥25 kg/m^2 along with exercising less than twice per week. Before testing, the contents and objective of the research were explained, and consent was obtained prior to subject participation. Kyungwoon University approved this study, which complies with the ethical standards of the Declaration of Helsinki. Physical characteristics of the subjects in each group are outlined in Table 1.

The exercise program in this study consisted of walking at 50–60% of the maximum volume of minute oxygen consumption (VO_{2max}) three times per week for 12 weeks. After a VO_{2max} treadmill test, oxygen consumption and heart beats per minute were calculated, as were individual pulse rates/minute corresponding to 50–60% of the VO_{2max} and individual logistic regression analysis were performed. To confirm the target calorie expenditure obtained with a Polar Heart Monitor (Polar S610i, Polar Electro, Kempele, Finland), the energy expenditure was calculated using the reached targeted pulse designated during the walking exercise program. To standardize the expended energy per session as 400 kcal and 1,200 kcal/week (3 times per week), the amount of time spent performing exercise was adjusted to between 60 and 120 minutes depending on the individual's ability. Physical measurements taken included height (cm), weight (kg), and the calculated body mass index (BMI), which was calculated as BMI = weight (kg)/height (m^2). Percent body fat was assessed using the InBody 720 bioelectrical body composition analyzer (Biospace, Seoul, Republic of Korea) following the procedures recommended by the American College of Sports Medicine (ACSM)[11]. Waist circumference was measured with a tape, while blood pressure was measured with an automatic machine (Tensoval Comfort, Paul Hartmann AG, Heidenheim, Germany) and included systolic (SBP) and diastolic (DBP) blood pressure measurements. Mean arterial pressure (MAP) was calculated by using the following equation: MAP = DBP+((SBP−DBP)/3). The VO_{2max} as an index of cardiorespiratory fitness (CRF), was measured on a motor driven treadmill using the Bruce protocol, as described in the ACSM guidelines (2010)[11]. Oxygen consumption (VO_2) was measured using standard breath-by-breath techniques of pulmonary gas exchange variables (Metabolic Gas Analyzer System; Quark b^2, Cosmed, Italy), and exercise heart rate was measured using 12-lead electrocardiography (Q-4500, Quinton Cardiology Systems, Bothell, WA, USA). VO_{2max} was considered the highest oxygen consumption attained at the exhaustion moment.

Blood sample analyses were performed simultaneously under the same conditions before and after the exercise program. The obtained blood samples were separated by centrifugation at 4 °C, and TG (triglyceride; Vitros TRIG DTD, Johnson & Johnson, NY, USA), and TC (total cholesterol; Vitros CHOL DTD, Johnson & Johnson, NY, USA) were analyzed on an slide using a Vitros DT60II chemistry

Table 2. Changes in obesity indices and blood pressure with 12 weeks aerobic exercise

Measured variable	Group	Pre	Post	Δscore
Weight (kg)	CON	64.3±3.1	63.5±3.5	−0.82±1.93
	Exercise	65.1±3.1	62.0±3.0†††	−3.11±1.24***
BMI (kg/m^2)	CON	26.3±1.3	26.0±1.5	−0.33±0.80
	Exercise	26.0±1.2	24.8±1.3†††	−1.24±0.47***
WC (cm)	CON	89.5±5.2	89.2±4.9	−0.36±0.83
	Exercise	88.3±4.6	86.7±4.5	−1.58±2.47*
Body fat (%)	CON	34.3±2.7	33.4±3.1	−0.87±1.54
	Exercise	32.8±3.2	30.6±3.5†	−2.18±1.96***
SBP (mmHg)	CON	124.5±12.3	123.9±8.6	−0.56±7.99
	Exercise	121.9±13.6	117.0±9.7	−4.84±10.23
DBP (mmHg)	CON	80.8±10.3	79.2±10.7	−1.56±9.42
	Exercise	77.2±9.7	80.4±7.8	3.18±6.45
MAP (mmHg)	CON	95.3±10.3	94.1±9.1	−1.22±7.11
	Exercise	92.1±10.5	92.6±7.8	0.51±7.37

Data are means. Δscore = change in the score from before the exercise program to 12 weeks * p value of paired t-test (*p<0.05; **p<0.01; ***p<0.001). † p value of two-way ANOVA test (group×time; †p<0.05; ††p<0.01; †††p<0.001). BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure

system (Johnson & Johnson, NY, USA). With HDLC using magnesium chloride and dextran sulfate, the cholesterol-containing apoB was precipitated, and using the HDLC (HDL cholesterol; Vitros HDLC DTD, Johnson & Johnson, NY, USA) slide, analysis was performed with a Vitros DT60II chemistry system (Johnson & Johnson, NY, USA). The atherogenic index was calculated by using the following equation: AI = (TC − HDLC) / HDLC[13]. ApoB and ApoA1 were analyzed by using immunoturbidimetry (Hitachi Auto-Analyzer, Model 705, Hitachi, Tokyo, Japan).

All data are expressed as averages and standard deviations, and to observe the transitional changes in the groups before and after exercise, two-way repeated measures ANOVA was conducted. For comparison of changes in the average values of measured variables before and after the 12 weeks of exercise training, the paired samples t-test was used. Moreover, the change in values between before and after 12 weeks of exercise was extracted (Δscore = changed score between before and after 12 weeks of exercise). All statistical analyses were performed by using SPSS for Window (version 18.0), and values of p=0.05 were considered statistically significant.

RESULTS

A comparison of the changes in blood pressure and obesity index after 12 weeks of aerobic exercise is shown in Table 2. Weight (p<0.001), BMI (p<0.001), and percent body fat (p<0.05) demonstrated statistically significant interactions (p<0.05). WC decreased statistically significantly after 12 weeks of aerobic exercise (p<0.05). However, blood pressure (SBP, DBP, MAP) did not demonstrate significant effects.

The cardiovascular fitness and cardiovascular risk factor comparison results are shown in Table 3. A marker of cardiovascular fitness, VO_{2max} (p<0.05), demonstrated statistically significant interactions. HDLC (p<0.05), apoB (p<0.001), and the apoB/apoA1 ratio (p<0.05) demonstrated statistically significant interactions. TC (p<0.05) and AI (p<0.05) demonstrated statistically significant decreases after 12 weeks of exercise. However, TG and apoA1 did not demonstrate statistically significant differences.

DISCUSSION

The ACSM[11] recommends 45 to 60 minutes of exercise daily, which consumes 300 to 400 kcal, to improve obesity and reduce weight. Fogelholm[14] also reported that to effectively decrease weight, a minimum of 35 to 45 minutes of exercise per day for a total of 250 to 300 minutes per week are required. In this study's results, 12 weeks of aerobic exercise in obese women significantly decreased obesity indices such as weight, BMI, and percent body fat. It is thought that it was appropriate to set the exercise volume at a level that does not cause fatigue in the weight control program for obese women. However, there were no changes in blood pressure (SBP, DBP, MAP). This is thought to be due to the fact that the subjects had normal blood pressures.

Generally speaking, indices evaluating cardiorespiratory fitness (CRF) include VO_{2max}, which represents the oxygen-carrying capacity of the active muscles during exercise. VO_{2max} decreases by 1% per year (setting 25 years as the standard), and the decrease in VO_{2max} displays an intimate relationship with chronic diseases and CVD[15]. Regular aerobic exercise is a good method for enhancing VO_{2max}, enhancing cardiovascular function, and effectively preventing and delaying CVD[11]. This study's results show a significant increase in VO_{2max} after 12 weeks of aerobic exercise. In reality, Katzmarzyk et al.[16] reported that aerobic exercise resulted in an increase in CRF in 19,173 obese subjects. Moreover, LaMonte et al.[17] reported that VO_{2max} is an independent and strong predictor of CVD and a meta-

Table 3. Changes in the cardiorespiratory fitness and apoB/apoA1 ratio with 12 weeks aerobic exercise

Measured variable	Group	Pre	Post	Δscore
VO_{2max} (mL/kg/min)	CON	28.7±2.8	29.9±3.8	1.20±2.48
	Exercise	30.9±3.4	34.3±3.2†	3.32±2.72***
TG (mg/dL)	CON	116.0±49.6	121.5±60.3	5.50±52.19
	Exercise	120.4±94.1	93.0±41.8	−27.37±71.53
TC (mg/dL)	CON	210.0±38.2	202.9±35.0	−7.12±26.30
	Exercise	201.6±44.1	185.1±41.4	−16.43±23.46*
HDLC (mg/dL)	CON	46.6±15.4	44.3±10.2	−2.31±11.09
	Exercise	45.0±13.1	51.1±15.0†	6.15±8.53*
Atherogenic index	CON	4.01±2.13	3.80±1.41	−0.20±1.54
	Exercise	3.87±1.94	2.98±1.58	−0.89±1.17*
Apolipoprotein B	CON	79.2±17.8	81.1±18.9	1.86±10.59
	Exercise	84.5±17.1	73.2±15.9†††	−11.35±8.44***
Apolipoprotein A-1	CON	152.3±26.5	153.3±28.8	0.96±23.73
	Exercise	151.0±28.9	156.2±32.6	5.18±14.60
ApoB/ApoA-1 ratio	CON	0.53±0.16	0.54±0.16	0.01±0.12
	Exercise	0.58±0.17	0.49±0.19†	−0.08±0.09**

Data are means. Δscore = change in the score from before the exercise program to 12 weeks, * p value of paired t-test (*p<0.05; **p<0.01; ***p<0.001). † p value of two-way ANOVA test (group×time; †p<0.05; ††p<0.01; †††p<0.001). TG, triglycerides; TC, total cholesterol; HDLC, high density lipoprotein cholesterol

bolic risk factor in both women and men.

HDLC plays a critical role in maintaining homeostasis of the TC quantity. This role of HDLC is due to RCT, through which HDLC may block the formation of atherosclerosis[18]. RCT eliminates surplus cholesterol residing in the macrophages of the arterial walls, transports it to the liver, and exports it in vitro, basically eliminating the production of atherosclerotic plaque[19]. RCT is the only mechanism that can eliminate surplus cholesterol within the body, and is therefore a critical factor in maintaining appropriate concentrations and functions of HDLC in prevention of CVD. Specifically, low HDLC concentrations are known to independently raise the frequency of CVD along with other risk factors[20]. Based on these results, HDLC demonstrated significant interactions. Jenkins et al.[21] reported that aerobic exercise effectively increased HDLC concentrations, while Katzmarzyk et al.[16] reported that HDLC concentrations increased in obese patients with metabolic syndrome, which was in line with our research. TC and AI decreased significantly after exercise training in the present study. Because the equation AI = (TC−HDLC/HDLC) was used, increases in HDLC due to exercise and decreases in TC yielded a decrease in AI. Normally, an AI value < 3 is noted as normal[13]; after exercise in the present study, the values normalized, which indicates that the exercise training was effective.

ApoA1 is one of the most basic units in the formation of HDLC and is a critical component of HDLC. ApoA1 is synthesized in the liver and gastrointestinal tract and secreted into the plasma, and through the action of the cell membrane protein of the peripheral tissues, known as ATP-binding cassette transporter A1 (ABCA1), cholesterol within cells is transported to apoA1 and forms HDLC[22]. It is known that as the synthesis of apoA1 is increased by long-term exercise, catabolism decreases[23]. However, in this research, there were no significant increases in apoA1. Such results are thought to be due to the exercise volume or length not being sufficient to change the blood concentration of apoA1. Additionally, those kinetics of apoA1 may be attributable to the fact that the subjects were obese. In obese individuals, as compared with normal individuals, hypercatabolism occurs relative to apoA1 synthesis speed[24]. In fact, the changes in apoA1 in this study are thought to be related to the apoA1 synthesis speed. However, it is difficult to arrive at conclusions based on insufficient data.

ApoB is a critical protein component of LDLC that increases the absorption of cellular cholesterol by combining LDL particles with the LDL receptor. Such apoB is intimately associated with blood concentrations of LDLC and CVD, and apoB plays an important role in improving blood lipid concentrations[9]. ApoB and the apoB/apoA1 ratio are clinical indices for metabolic risk factors, specifically predicting CVD risk and lowering cholesterol, which are useful markers for treatment progress. Recently, regular exercise has been shown to decrease the apoB level and apoB/apoA1 ratio, directly influencing CVD prevention[25]. Based on this study's results, apoB and the apoB/apoA1 ratio demonstrated significant interactions. Holme et al.[25] reported that in a study with overweight males, regular aerobic exercise decreased apoB levels and the apoB/apoA1 ratio, and Ben Ounis et al.[26] reported the same findings. Also, Crouse et al.[27], in a study involving adults with hypercholesterolemia, reported that aerobic exercise decreased apoB, and Janssen et al.[28] showed that aerobic exercise and diet control in obese women decreased the apoB/apoA1 ratio. However, Henderson et al.[29] reported that solitary exercise did

not impact lipoproteins such as apoB and apoA1, so long-term aerobic exercise is thought to be relatively effective. Based on this study's results, regular aerobic exercise significantly improved the obesity index, CRF, blood HDLC, and apoB/apoA1 ratio in obese women. Such results suggest that regular exercise can effectively improve the obesity index along with cardiovascular risk factors. However, TG and apoA1 levels did not change significantly, so exercise intensity, frequency, and duration may need to be considered and increased.

REFERENCES

1) World Health Organization: Obesity and overweight. Global strategy on diet, physical activity and health, 2011.
2) Wooten JS, Biggerstaff KD, Anderson C: Response of lipid, lipoprotein-cholesterol, and electrophoretic characteristics of lipoproteins following a single bout of aerobic exercise in women. Eur J Appl Physiol, 2008, 104: 19–27. [Medline] [CrossRef]
3) Walldius G, Jungner I: Apolipoprotein B and apolipoprotein A-I: risk indicators of coronary heart disease and targets for lipid-modifying therapy. J Intern Med, 2004, 255: 188–205. [Medline] [CrossRef]
4) Durstine JL, Grandjean PW, Cox CA, et al.: Lipids, lipoproteins, and exercise. J Cardiopulm Rehabil, 2002, 22: 385–398. [Medline] [CrossRef]
5) Chapman MJ, Assmann G, Fruchart JC, et al. European Consensus Panel on HDL-C: raising high-density lipoprotein cholesterol with reduction of cardiovascular risk: the role of nicotinic acid—a position paper developed by the European Consensus Panel on HDL-C. Curr Med Res Opin, 2004, 20: 1253–1268. [Medline] [CrossRef]
6) Tsompanidi EM, Brinkmeier MS, Fotiadou EH, et al.: HDL biogenesis and functions: role of HDL quality and quantity in atherosclerosis. Atherosclerosis, 2010, 208: 3–9. [Medline] [CrossRef]
7) Sniderman AD, Faraj M: Apolipoprotein B, apolipoprotein A-I, insulin resistance and the metabolic syndrome. Curr Opin Lipidol, 2007, 18: 633–637. [Medline] [CrossRef]
8) Wallenfeldt K, Bokemark L, Wikstrand J, et al.: Apolipoprotein B/apolipoprotein A-I in relation to the metabolic syndrome and change in carotid artery intima-media thickness during 3 years in middle-aged men. Stroke, 2004, 35: 2248–2252. [Medline] [CrossRef]
9) Vaverkova H, Karasek D, Novotny D, et al.: Apolipoprotein B versus LDL-cholesterol: association with other risk factors for atherosclerosis. Clin Biochem, 2009, 42: 1246–1251. [Medline] [CrossRef]
10) Sierra-Johnson J, Somers VK, Kuniyoshi FH, et al.: Comparison of apolipoprotein-B/apolipoprotein-AI in subjects with versus without the metabolic syndrome. Am J Cardiol, 2006, 98: 1369–1373. [Medline] [CrossRef]
11) American College of Sports Medicine: ACSM's Guidelines for exercise testing and prescription, 8th ed. Philadelpia: Lippincott Williams and Wilkins, 2010.
12) Kim DY, Jung SY, Seo BD: Effect of exercise intervention on changes in free Fatty Acid levels and metabolic risk factors in stroke patients. J Phys Ther Sci, 2014, 26: 275–279. [Medline] [CrossRef]
13) Haglund O, Luostarinen R, Wallin R, et al.: The effects of fish oil on triglycerides, cholesterol, fibrinogen and malondialdehyde in humans supplemented with vitamin E. J Nutr, 1991, 121: 165–169. [Medline]
14) Fogelholm M: Walking for the management of obesity. Dis Manag Health Outcomes, 2005, 13: 9–18. [CrossRef]
15) Buskirk ER, Hodgson JL: Age and aerobic power: the rate of change in men and women. Fed Proc, 1987, 46: 1824–1829. [Medline]
16) Katzmarzyk PT, Church TS, Janssen I, et al.: Metabolic syndrome, obesity, and mortality: impact of cardiorespiratory fitness. Diabetes Care, 2005, 28: 391–397. [Medline] [CrossRef]
17) LaMonte MJ, Barlow CE, Jurca R, et al.: Cardiorespiratory fitness is inversely associated with the incidence of metabolic syndrome: a prospective study of men and women. Circulation, 2005, 112: 505–512. [Medline] [CrossRef]
18) Shah PK, Kaul S, Nilsson J, et al.: Exploiting the vascular protective effects of high-density lipoprotein and its apolipoproteins: an idea whose time for testing is coming, part I. Circulation, 2001, 104: 2376–2383. [Medline] [CrossRef]
19) Fielding CJ, Fielding PE: Molecular physiology of reverse cholesterol transport. J Lipid Res, 1995, 36: 211–228. [Medline]
20) Singh IM, Shishehbor MH, Ansell BJ: High-density lipoprotein as a therapeutic target: a systematic review. JAMA, 2007, 298: 786–798. [Medline] [CrossRef]
21) Jenkins NT, McKenzie JA, Damcott CM, et al.: Endurance exercise training effects on body fatness, VO_{2max}, HDL-C subfractions, and glucose tolerance are influenced by a PLIN haplotype in older Caucasians. J Appl Physiol 1985, 2010, 108: 498–506. [Medline] [CrossRef]
22) Attie AD, Kastelein JP, Hayden MR: Pivotal role of ABCA1 in reverse cholesterol transport influencing HDL levels and susceptibility to atherosclerosis. J Lipid Res, 2001, 42: 1717–1726. [Medline]
23) Olchawa B, Kingwell BA, Hoang A, et al.: Physical fitness and reverse cholesterol transport. Arterioscler Thromb Vasc Biol, 2004, 24: 1087–1091. [Medline] [CrossRef]
24) Ooi EM, Watts GF, Farvid MS, et al.: High-density lipoprotein apolipoprotein A-I kinetics in obesity. Obes Res, 2005, 13: 1008–1016. [Medline] [CrossRef]
25) Holme I, Høstmark AT, Anderssen SA: ApoB but not LDL-cholesterol is reduced by exercise training in overweight healthy men. Results from the 1-year randomized Oslo Diet and Exercise Study. J Intern Med, 2007, 262: 235–243. [Medline] [CrossRef]
26) Ben Ounis O, Elloumi M, Makni E, et al.: Exercise improves the ApoB/ApoA-I ratio, a marker of the metabolic syndrome in obese children. Acta Paediatr, 2010, 99: 1679–1685. [Medline] [CrossRef]
27) Crouse SF, O'Brien BC, Grandjean PW, et al.: Effects of training and a single session of exercise on lipids and apolipoproteins in hypercholesterolemic men. J Appl Physiol 1985, 1997, 83: 2019–2028. [Medline]
28) Janssen I, Fortier A, Hudson R, et al.: Effects of an energy-restrictive diet with or without exercise on abdominal fat, intermuscular fat, and metabolic risk factors in obese women. Diabetes Care, 2002, 25: 431–438. [Medline] [CrossRef]
29) Henderson GC, Krauss RM, Fattor JA, et al.: Plasma triglyceride concentrations are rapidly reduced following individual bouts of endurance exercise in women. Eur J Appl Physiol, 2010, 109: 721–730. [Medline] [CrossRef]