

The Effect of Physical Activity on Serum Lipids, Lipoprotein, and Apolipoproteins

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Objective: The aim of this study was to measure apolipoprotein-A1 and apolipoprotein-B serum concentrations during a physical activity program. Serum apolipoprotein concentrations may be a more sensitive indicator of coronary heart disease (CHD) risk than total cholesterol, triglycerides, low density lipoproteins and high density lipoproteins. **Design:** Thirty-seven sedentary, healthy adult males were randomly allocated to an exercise group (n=20) who underwent 12 weeks of aerobic physical activity or a sedentary group (n=17) who acted as non-exercising controls. **Results:** The exercise group increased their aerobic capacity (from 33±4 mL•kg⁻¹•min⁻¹ to 40±4 mL•kg⁻¹•min⁻¹) but the sedentary group did not. The percentage of body fat decreased in the exercise group (from 21.8% to 19.5%) but remained unchanged in the sedentary group. Serum cholesterol, lipoprotein and apolipoprotein concentrations did not change but serum triglyceride concentrations were reduced in the exercise group (from 1.8±1.3 mmol•L⁻¹ to 1.2±0.4 mmol•L⁻¹, p<0.05). The Apo-A1:Apo-B ratio increased in the exercise group (from 1.17±0.22 to 1.4±0.27, p<0.05) but not in the sedentary group. **Conclusion:** Apolipoprotein concentrations in sedentary males are no more sensitive than other serum lipid concentrations but are appropriate for monitoring CHD risk-factor change during short-term light exercise interventions.

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INTRODUCTION

Serum lipid concentrations are often used to evaluate the risk of coronary heart disease (CHD). Previous studies have shown that low serum concentrations of high density lipoproteins (HDL) together with high serum concentrations of total cholesterol, triglycerides and lipoproteins are associated with an increased risk of CHD (1). Other findings have suggested that the serum concentration of apolipoproteins may be a more specific and sensitive indicator of CHD risk (2, 3). Apolipoprotein A-1 (Apo-A1), the major protein

component of HDL, is thought to have antiatherogenic potential, whereas apolipoprotein B (Apo-B), the major protein component of low density lipoprotein (LDL), is believed to play an important role in atherogenesis (4, 5). Apo-B concentration also appears to be a good indicator of circulating atherogenic particles and a potentially useful method of evaluating cholesterol transport and independent CHD risk (6). Various studies have indicated that total cholesterol is a less sensitive marker of CHD risk than measured fractions of HDL and LDL; total cholesterol is an even less accurate indicator of CHD risk than

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concentrations of Apo-A1 and Apo-B (6, 7). Ratios between certain circulating lipids are often used to evaluate CHD risk in addition to absolute concentrations. For example, it has been suggested that the lower the Apo-A1:Apo-B ratio the greater the risk of CHD (6). Furthermore, the oxidized phospholipid:Apo-B ratio has been positively correlated with the presence and extent of coronary artery disease on angiographic examination (8).

Previous studies have shown that habitual physical activity helps reduce CHD risk factors, particularly in relation to total cholesterol, triglyceride and lipoprotein concentrations (9, 10). Several studies have investigated the effects of combined exercise and diet on Apo-A1 in the context of weight loss (11, 12) but few have looked at the effects of physical activity alone. Those that have produced inconsistent findings (9, 13) and were limited to Apo-A1 changes among hypercholesterolaemic subjects (14). Epidemiological studies focusing on physical activity and cardiovascular risk have reported an inverse relationship between high levels of physical activity and Apo-B concentrations in young males (15) as well as a positive relationship between physical fitness and HDL:Apo-A1 ratios (16) and favorable levels of both Apo-A1 and Apo-A1:Apo-B ratios in males and females with high levels of physical activity (17). Comparisons between highly active, moderately active, and inactive Japanese males indicated that physical activity may have a greater effect on Apo-A1 than other lipid sub fractions, with Apo-A1 levels increasing in proportion to the amount of physical activity (17).

Although the Apo-A1:Apo-B ratio is a potentially effective tool to monitor changes in risk among CHD patients undertaking prescribed physical activity, what has not been established is whether apolipoprotein concentrations can be used to evaluate changing risk among otherwise healthy sedentary people undertaking mild physical activity. The aim of this study was to measure the extent to which apolipoprotein concentrations are sensitive to mild increases in physical activity. It was hypothesized that mild aerobic exercise among previously sedentary males would provoke an increase in serum Apo-A1 concentration and an increased Apo-A1:Apo-B ratio.

MATERIAL AND METHODS

Design and Participants

A between-subjects experimental design was used in which thirty-seven sedentary, healthy male participants were randomly assigned to either an exercise condition ($n = 20$, age 42.4 ± 1.4 years, height 177.7 ± 1.2 cm and body mass 80.6 ± 1.9 kg) or a no

exercise control condition ($n = 17$, age 44.5 ± 1.5 years, height 175.7 ± 1.7 cm and body mass 85.7 ± 3.3 kg). A power analysis for an ANOVA design with two independent groups and three measurement trials was performed for sample size estimation using the SAS macro program FPOWER.SAS. The outcome indicated that 18 participants per condition (30 total) would be needed to achieve a statistical power of >0.8 assuming a moderate effect size of 0.6.

Participants had no recorded history of medical problems and had not participated in sports or exercise during the six months preceding the study. Only aerobically unfit participants were included in this study. Volunteers with a peak oxygen uptake of greater than $40 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ were also excluded. Participants provided their written informed consent to participate in the study. All of the procedures used were approved by the University of Wollongong ethics committee and conducted in accordance with the Declaration of Helsinki.

Protocol

Participant measurements were taken prior to the intervention (week 0), halfway through the intervention (week 6) and at the end of the intervention (week 12). On each occasion participants' anthropometric profiles were measured, resting heart rate and blood pressure measurements were taken, a blood sample was taken for lipid analysis and an ECG stress test incorporating a test was conducted. A 24-hour diet recall was completed by each participant for the period immediately prior to each testing session to establish any differences in participants' diets. Participants were asked to refrain from smoking for at least one hour prior to each exercise test and to fast for 12-16 hours in preparation for the serum lipid analyses. Originally, twenty participants were recruited for each group, but three subjects from the sedentary group were subsequently excluded from the study after they reported that they had taken up physical activity or had markedly modified their diets as indicated by the 24-hour dietary recalls.

Body Composition and Anthropometric Testing

Participants' height was measured using a stadiometer (Somatometre 211daPD, USA) and body mass was measured using calibrated portable scales (Scout 2, Ohaus, USA). Four site skinfold measurements (triceps, subscapular, suprailiac, and medial calf) were taken by a qualified anthropometrist using Harpenden skinfold calipers (Harpenden, West Sussex, UK). All skinfold measurements were taken to the nearest

0.1mm and an average of three readings for each site was used. Skinfold measurements were used to estimate the percentage of body fat using previously established conversion equations (18).

Lipid Assessment

In each subject a fasting venous blood sample was taken from the antecubital vein immediately prior to the stress test. This blood sample was analyzed for total serum cholesterol (CHOL), triglyceride (TRIG), HDL, LDL, Apo-A1 and Apo-B. With the exception of Apo-A1 and Apo-B, the serum concentration of each component was determined using an Ektachem DT60 analyzer (Kodak, USA). Apo-A1 and Apo-B concentrations were measured using a Beckman Array Protein System (Beckman, USA).

Stress Testing and Cardiovascular Analysis

Each participant performed a stress test during which aerobic capacity was measured using an integrated Quinton cardiac stress testing system (Q-5000 EKG/Stress Test System, USA). A modified Bruce treadmill protocol with a constant treadmill speed (5.5 km• h⁻¹) and increases in treadmill gradient (1% per minute) was used during the stress test. On-line gas analysis was completed during stress testing. Inspired air volume was measured by a calibrated turbine Ventilation Monitor (Morgan Mk 2, USA). Participants' expired air was collected in 3L mixing chambers via a Hans Rudolph low resistance valve. Expired air samples were drawn from a mixing chamber and dried. Each minute sample was analysed for O₂ and CO₂ using O₂ (Morgan Mk 701) and CO₂ (Morgan) gas analysers calibrated against two alpha standard gases of known compositions. The stress test was terminated when the participant reached his peak aerobic capacity as indicated by a heart rate plateau at high work intensities. As described in the study design, only participants with a peak aerobic capacity () of 40 ml• kg⁻¹• min⁻¹ or less were included in this study.

Electrocardiograms (ECGs) were recorded at rest and during exercise during each of the three testing sessions and were conducted and interpreted by a cardiologist. A 12-lead ECG was taken immediately prior to commencing the exercise test with the participant remaining stationary and standing on the treadmill. The exercising electrocardiograms were recorded during the treadmill exercise tests. Interpretation of both the resting and exercising electrocardiographs involved identifying persistent ST segment depression (at least 1mm that is horizontal or down-sloping at 0.08 seconds after the J point) because, according to the American College of Sports Medicine (19), an ST segment depression that occurs

early and remains throughout testing is more likely to be due to coronary heart disease than an ST depression that occurs only during high intensity exercise. Heart rates were determined directly from the ECG trace. Resting systolic blood pressure (SBP) and resting diastolic blood pressure (DBP) recordings were determined indirectly, with a sphygmomanometer cuff placed on the left arm using the Quinton 5000 Exercise Test Monitor. All cardiovascular variables were monitored for 30 minutes after completion of the exercise test.

Physical Activity Programme

Participants in the experimental group completed a 12-week aerobic physical activity program that was designed in accordance with American College of Sports Medicine guidelines (19). Each week comprised a combination of supervised and unsupervised walking or jogging that gradually increased during the first eight weeks from 10 minutes performed 3 days per week to 45 minutes performed 5 days per week. Target heart rate reserve values of 50-60% were calculated for each participant based upon resting and maximum values recorded during their stress tests. Participants monitored their own heart rates throughout each exercise session and regulated their efforts accordingly. Individual relative exercise intensity was more difficult to control during the less frequent supervised group-based training sessions, with the result that some participants occasionally exceeded the intended 50-60% heart rate reserve.

Statistical analysis

Between-group comparisons (exercise vs. sedentary) were made using between-subjects one-way ANOVAs with an alpha level of 0.05 to indicate significance. Comparisons between the trials (0, 6, and 12 weeks) were made using repeated-measures ANOVAs and post-hoc paired samples t-tests with a Bonferonni corrected alpha level of 0.0167 to indicate significance. All values are presented as the mean ± one standard deviation and effect sizes for significant outcomes were calculated using Hedge's adjusted g.

RESULTS

Body Composition

At week 0 (pre-trial) there were no differences between the exercise and sedentary groups for age (42.4±1.4 years vs. 44.5±1.5 years, p>0.05), height (177.7±1.2 cm vs. 175.7±1.7 cm, p>0.05), body mass (80.6±1.9 kg vs. 85.7±3.3 kg, p>0.05) or percentage body fat (21.5±0.7% vs. 21.8±0.5%, p>0.05). There

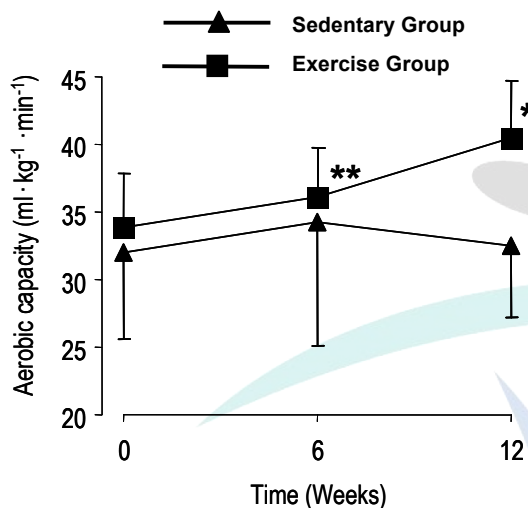


Figure 1. Change in aerobic capacity between exercise and sedentary groups. ** $p < 0.01$ (compared with week 0)

was no significant reduction in body mass between week 0 and week 12 among either the sedentary group (86.7±3.3 kg vs. 85.8±3.4 kg, $p > 0.0167$) or the exercise group (80.6±1.9 kg vs. 78.7±1.7 kg, $p > 0.0167$). However, a reduction in the percentage of body fat between week 0 and week 12 was evident among the exercise group (from 21.8±0.5% to 19.5±0.5%, $p < 0.0167$, $g = -4.5$) but not among the sedentary group (21.5±0.7% vs. 22.4±0.7%, $p > 0.05$).

Cardiovascular Measurements

At week 0 there were no differences between the groups in resting heart rate ($p > 0.05$), SBP ($p > 0.05$) or DBP ($p > 0.05$). Between week 0 and week 12 there was a reduction in resting heart rate among the exercise group (from 73±2 beats·min⁻¹ to 68±2 beats·min⁻¹, $p < 0.0167$, $g = -2.5$) but not among the sedentary group (72±3 beats·min⁻¹ vs. 73±3 beats·min⁻¹, $p > 0.0167$).

Cardiovascular Measurements

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(from 85 mm±2 Hg to 79±2 mm Hg, $p < 0.01$, $g = -2.9$) was observed among the sedentary group.

At week 0 there was no difference between the groups in ($p > 0.05$). Between week 0 and week 12 there was a significant increase in among the exercise group (from 33±4 mL·kg⁻¹·min⁻¹ to 40±4 mL·kg⁻¹·min⁻¹, $p < 0.01$, $g = 5$) but not among the sedentary group (32±5 mL·kg⁻¹·min⁻¹ vs. 32±5 mL·kg⁻¹·min⁻¹, $p > 0.0167$). Changes in are presented in Figure 1.

Serum Lipid Concentrations

At week 0 there were no differences between the groups for CHOL ($p > 0.05$) or TRIG ($p > 0.05$). Between week 0 and week 12 there was a decrease in CHOL among both the sedentary group (from 6.1±0.3 mmol·L⁻¹ to 5.3±0.2 mmol·L⁻¹, $p < 0.01$, $g = -3.1$) and the exercise group (from 6.1±0.3 mmol·L⁻¹ to 5.0±0.2 mmol·L⁻¹, $p < 0.01$, $g = -4.2$). Between week 0 and week 12 there was a reduction in TRIG among the exercise group (from 1.8±0.3 mmol·L⁻¹ to 1.2±0.2 mmol·L⁻¹, $p < 0.01$, $g = -2.3$) but not among the sedentary group (1.7±0.1 mmol·L⁻¹ vs. 1.7±0.1 mmol·L⁻¹, $p > 0.0167$).

At week 0 there were no differences between the groups for Apo-A1 ($p > 0.05$) or Apo-B ($p > 0.05$). Between week 0 and week 12 there were no changes in Apo-A1 among the exercise group (1.15±0.06 g·L⁻¹ vs. 1.10±0.06 g·L⁻¹, $p > 0.0167$) or the sedentary group (1.06±0.03 g·L⁻¹ vs. 1.06±0.03 g·L⁻¹, $p > 0.0167$). Compared with week 0, Apo-B levels among the exercise group had decreased by week 6 (from 0.94±0.18 g·L⁻¹ to 0.81±0.18 g·L⁻¹, $p < 0.0167$, $g = -0.7$) and were still lower at week 12 (from 0.94±0.18 g·L⁻¹ to 0.78±0.18 g·L⁻¹, $p < 0.0167$, $g = -0.9$). There were no changes in Apo-B levels among the sedentary group between week 0 and week 6 (0.95±0.27 g·L⁻¹ vs. 0.89±0.22 g·L⁻¹, $p > 0.0167$, $g = -0.2$) or between week 0 and week 12 (0.95±0.27 g·L⁻¹ vs. 0.88±0.36 g·L⁻¹, $p > 0.0167$). All serum lipid and apolipoprotein concentration values are presented in Table 1.

Between week 0 and week 12 there was an increase in the Apo-A1:Apo-B ratio among the exercise group (from 1.17±0.22 to 1.4±0.27, $p < 0.0167$, $g = 0.9$) but not among the sedentary group (1.28±0.49 vs. 1.41±0.49, $p > 0.0167$). There were no trial or group differences in the HDL:CHOL ratio ($p > 0.05$), the LDL:CHOL ratio ($p > 0.05$) or the LDL:HDL ratio ($p > 0.05$). All serum lipid and apolipoprotein ratio values are presented in Table 2.

DISCUSSION

Compared with sedentary controls, participants who exercised showed an increase in peak aerobic capacity and Apo A-1:Apo B ratios as well as lower levels of

Table 1. Exercise and sedentary group serum lipid concentration values.

	Week 0		Week 6		Week 12	
	Sedentary	Exercise	Sedentary	Exercise	Sedentary	Exercise
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
<i>Cholesterol (mmol·L⁻¹)</i>	6.1 (1.3)	6.1 (1.3)	5.9 (1.3)	5.4 (1.3)*	5.3 (0.9)**	5.0 (0.9)**
<i>Triglyceride (mmol·L⁻¹)</i>	1.7 (0.4)	1.8 (1.3)	1.7 (0.9)	1.5 (0.9)	1.7 (0.4)	1.2 (0.4)**
<i>H.D.L (mmol·L⁻¹)</i>	1.34 (0.4)	1.28 (0.27)	1.17 (0.4)*	1.08 (0.18)*	1.15 (0.53)	1.16 (0.22)
<i>L.D.L (mmol·L⁻¹)</i>	4.01 (1.3)	3.97 (1.12)	3.80 (1.34)	3.43 (0.98)*	3.34 (1.34)*	3.29 (0.94)*
<i>Apo A-1 (mmol·L⁻¹)</i>	1.15 (0.27)	1.06 (0.13)	1.14 (0.22)	1.04 (0.13)	1.10 (0.27)	1.06 (0.13)
<i>Apo B (mmol·L⁻¹)</i>	0.95 (0.27)	0.94 (0.18)	0.89 (0.22)	0.81 (0.18)*	0.88 (0.36)	0.78 (0.18)*

* $p < 0.0167$ ** $p < 0.01$ (from baseline at Week 0)

adiposity, serum triglyceride, and Apo-B, which is consistent with our hypothesis. However, both sedentary and exercise groups showed reductions in total serum cholesterol and LDL. These results suggest that sedentary people who regularly participate in light aerobic exercise for even a short period can reduce their CHD risk, as indicated by changes in the Apo A-1:Apo B ratio, an accepted measurement of CHD susceptibility (5-11). Contrary to other studies (25, 27), our findings add that light exercise, typically prescribed to people with CHD risk factors, can have beneficial effects within a relatively short period.

The observed changes in body composition in both the sedentary and the exercise groups were as expected and correspond with similar alterations in weight and percentage of body fat that have been observed in adult males during 20- and 24-week aerobic exercise programmes (13, 20, 21). Only the exercise group showed a reduction in adiposity during the current study, suggesting that even light regular exercise can cause beneficial physiological changes among sedentary people in a relatively short period.

Peak aerobic capacity also responded as expected in both the sedentary and the exercise groups. When the changes observed in the exercising participants are compared with the sedentary controls, it appears likely that the aerobic exercise regime followed during this study contributed to the change. Many studies have shown that maximal aerobic capacity is positively correlated with levels of activity in an individual (13, 20-23). However, studies also show that there is no direct association between plasma lipoprotein concentrations and cardiorespiratory fitness, particularly in 'low exercise' study groups (24-26). Heart rate and blood pressure responses were as expected and are consistent with other well-documented studies (13, 27, 28).

Serum triglyceride levels fell only among the exercising participants but, unexpectedly, serum

cholesterol levels fell among both groups. Our findings also suggest that changes in LDL concentration are more sensitive to low intensity aerobic physical activity than changes in HDL since both the sedentary and the exercise groups showed no change in HDL. Previous studies have suggested that lipoprotein measurements can be influenced by sampling time and method, suppressed baseline lipoprotein levels and factors such as body weight, age, diet, smoking and intensity of exercise (9, 24, 29, 30). Since all of these factors were carefully controlled in our study, we conclude that light exercise has selected short-term benefits, with serum triglyceride responding the most. In fact, it is apparent from other studies that traditional lipoprotein measures do not change consistently in response to various exercise programs. While one study found that LDL fell among 30-45 year old males after a one-year exercise program (31), other studies observed reductions in CHOL, LDL and non-HDL only after a 24-week high intensity exercise program (20) or among men aged over 50 years (29). We conclude from our results, along with the inconsistent outcomes of other studies, that the use of traditional lipoprotein levels to evaluate CHD risk factors may be an unreliable predictor, especially when low intensity short-duration exercise interventions are used.

Throughout the study, Apo-A1 concentrations remained constant among the exercise and the sedentary groups. Apo-B concentrations were lower in the exercise group but remained unchanged in the sedentary group. These results differ from previous apolipoprotein studies. For instance, in one such study, a group of active males showed significantly higher Apo-A1 levels than matched controls when subjects had been exercising for a minimum of two years (17). This result was accompanied by a significant reduction in HDL, but no change in LDL. Similar data was obtained in a study into the effect of inactivity on

Table 2. Exercise and sedentary group serum lipid ratio values.

	Week 0		Week 6		Week 12	
	Sedentary	Exercise	Sedentary	Exercise	Sedentary	Exercise
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
HDL:CHOL	0.23 (0.09)	0.22 (0.09)	0.21 (0.09)	0.21 (0.04)	0.23 (0.13)	0.24 (0.04)
LDL:CHOL	0.64 (0.09)	0.64 (0.09)	0.65 (0.09)	0.64 (0.09)	0.62 (0.13)	0.64 (0.09)
LDL:HDL	3.26 (1.36)	3.64 (1.07)	3.54 (1.79)	3.75 (1.21)	3.51 (2.28)	3.19 (1.03)
Apo-A1:Apo-B	1.28 (0.49)	1.17 (0.22)	1.36 (0.49)	1.33 (0.31)*	1.41 (0.49)	1.40 (0.27)*

* p<0.0167 (from baseline at Week 0)

serum lipid concentrations (32). Patients who were confined to hospital beds showed highly significant reductions in both Apo-A1 and HDL after 3-6 weeks. Remarkably, serum cholesterol concentrations actually decreased significantly in comparison with those of active controls. It was concluded that daily activity was accompanied by an increase in both HDL and Apo-A1, but may not be accompanied by a decrease in serum cholesterol (32). However, these studies involved either long-term exercise or extremely sedentary behavior, which makes direct comparisons with our own findings difficult. What our findings add is that in the short term light physical activity has a beneficial effect on Apo-B but not on Apo-A1 concentrations.

More recent studies report more varied and inconsistent findings. For example, Fung et al. (33) studied a large group of healthy men in relation to the impact that physical activity level (using average metabolic equivalent-hours per week) had upon lipid profiles. They found that Apo-A1 level variation was not related to average physical activity level, despite a positive association between level of physical activity and HDL (33). Fung et al. did, however, identify an inverse relationship between sedentary activity (hours spent watching television) and Apo-A1 levels in the same group of individuals. Couillard et al. (30) discovered that favorable effects on lipid profiles following a 20-week exercise programme (decreases in CHOL, LDL, TRIG and Apo-B levels, and an increase in Apo-A1 and HDL) were restricted to men with high baseline levels of triglyceride and LDL. As a result of this study, Couillard et al. proposed that discrepancies in HDL and Apo-A1 in response to exercise are independent of one another and that there is a significant metabolic influence on cholesterol response (30).

CONCLUSION

An increase in the Apo-A1:Apo-B ratio was evident only among the exercising group, suggesting that this measure is sensitive to light exercise and responds in a relatively short period. The increase in the ratio

appears to be due mainly to a greater reduction of Apo-B concentration among the exercising group than has been previously discussed. As other studies have suggested, the Apo-A1:Apo-B ratio appears to be an effective way of evaluating CHD risk (6, 34). Our study adds that this method is appropriate for monitoring CHD risk factor change during short-term light-exercise interventions.

It is a strength of the present study that the treatment conditions involved mild aerobic physical activity to reflect the intensity of exercise that is considered to be safe and which is typically prescribed to improve the prognosis of patients with CVD risk factors. The main limitation of our study is the relatively short data collection period. What is needed in the future is a more detailed examination of how light exercise affects apolipoprotein responses over a longer period.

REFERENCES

1. Pekkanen J, Linn S, Heiss G et al. Ten year mortality from cardiovascular disease in relation to cholesterol level among men with and without pre-existing cardiovascular disease. *N Eng J Med* 1990; 322: 1700-1707.
2. Rifkind BM. High-density lipoprotein cholesterol and coronary artery disease: survey of the evidence. *Am J Cardiol* 1990; 66: 3A-6A.
3. Bairaktari ET, Seferiadis IK, & Elisaf MS. Evaluation of methods for the measurement of low-density lipoprotein cholesterol. *J Cardiovasc Pharmacol Ther* 2005; 10: 45-54.
4. Insull W, Marais AD, Aronson R, & Manfreda S. Efficacy and safety of fluvastatin ER 80 mg compared with fluvastatin IR 40 mg in the treatment of primary hypercholesterolaemia. *Br J Cardiol* 2004; 11: 148-155.
5. O'Connell BJ & Genest J. High-density lipoproteins and endothelial function. *Circulation* 2001; 104: 1978-1983.
6. Sniderman AD, Furberg CD, Keech A, Roeters van Lenep JE et al. Apolipoproteins versus lipids as indices of coronary risk and as targets for statin treatment. *Lancet* 2003; 361:777-780.
7. Wald NJ, Law M, Watt HC, Wu T et al. Apolipoproteins and ischaemic heart disease: Implications for screening. *Lancet* 1994; 343: 75-79.
8. Tsimikas S, Brilakis ES, Miller ER, McConnell JP, Lennon RJ, Kornman KS, Witztum JL, Berger PB. Oxidized phospholipids, Lp(a) lipoprotein, and coronary artery disease. *N Eng J Med* 2005; 353: 46-57.
9. Tall A. Exercise to reduce cardiovascular risk: how much is enough? *N Eng J Med* 2002; 347: 1522-1525.
10. Dowling EA. How exercise affects lipid profiles in women: what to recommend for patients. *Phys Sports Med* 2001; 29: 45-52.

11. Williams PT, Krauss RM, Vranizan KM, & Wood PD. Changes in lipoprotein subfractions during diet-induced and exercise-induced weight loss in moderately overweight men. *Circulation* 1990; 81: 1293-304.
12. Williams PT, Krauss RM, Vranizan KM, Albers JJ, & Wood PD. Effects of weight loss by exercise and by diet on apolipoproteins A-I and A-II and the particle-size distribution of high-density lipoproteins in men. *Metabolism* 1992; 41: 441-449.
13. Crouse SF, O'Brien BC, Grandjean PW, Lowe RC, Rohack JJ, & Green JS. Effects of training and a single session of exercise on lipids and apolipoproteins in hypercholesterolaemic men. *J App Physiol* 1997; 83: 2019-2028.
14. Crouse SF, O'Brien BC, Grandjean PW, Lowe RC, Rohack JJ, Green JS, & Tolson H. Training intensity, blood lipids, and apolipoproteins in men with high cholesterol. *J App Physiol* 1997; 82: 270-277.
15. Ratakari OT, Taimela S, Porkka KVK, Telama R, Valimaki I, Akerblom HK, & Viikari JSA. Associations between physical activity and risk factors for coronary heart disease: The Cardiovascular Risk in Young Finns Study. *Med Sci Sports Exerc* 1997; 29: 1055-61.
16. MacAuley D, McCrum EE, Stott G, Evans AE, Duly E, Trinick TR, Sweeney K, & Boreham CAG. Physical fitness, lipids, apolipoproteins in the Northern Ireland Health and Activity Survey. *Med Sci Sports Exerc* 1997; 29: 1187-91.
17. Nagao N, Imai Y, Arie J, Sawada Y, & Karatsu K. Comparison of serum apolipoproteins and lipoproteins in active and inactive males. *J Sports Med Phys Fitness* 1988; 28: 67-73.
18. Durmin JGVA & Rahaman MM. The assessment of the amount of fat in the human body from measurements of skin fold thickness. *Br J Nutr* 1967; 21: 681-689.
19. American College of Sports Medicine. *Guidelines for Exercise Testing and Exercise Prescription (7th Edition)*. Philadelphia: Lippincott Williams & Wilkins, 2005.
20. O'Donovan G, Owen A, Bird SR, Kearney EM, Nevill AM, Jones DW, & Woolf-May K. Changes in cardiorespiratory fitness and coronary heart disease risk factors following 24 wks of moderate- or high-intensity exercise of equal energy cost. *J App Physiol* 2005; 98: 1619-1625.
21. Skinner JS, Wilmore KM, Krasnoff JB, Jasolski A, Jasolska A, Gagnon J, Province MA, Leon AS, Rao DC, Wilmore JK, & Bouchard C. Adaptation to a standardized training program and changes in fitness in a large, heterogeneous population: The HERITAGE Family Study. *Med Sci Sports Exerc* 2000; 32: 157-161.
22. Jae SY, Fernhall B, Heffernan KS, Jeong M, Chun EM, Sung J, Lee SH, Lim YJ, & Park WH. Effects of lifestyle modifications on C-reactive protein: contribution of weight loss and improved aerobic capacity. *Metabolism: Clin Exper*. 2006; 55: 825-31.
23. Berthouze SE, Minaire PM, Castells J et al. Relationship between mean habitual daily energy expenditure and maximal oxygen uptake. *Med Sci Sports Exerc* 1995; 27: 1170-1179.
24. Kraus WE, Houmard JA, Duscha BD et al. Effects of the amount and intensity of exercise on plasma lipoproteins. *N Eng J Med* 2002; 247: 1483-1492.
25. Leon AS, Rice T, Mandel S, Després J-P, Bergeron J, Gagnon J, Rao DC, Skinner JS, Wilmore JH, & Bouchard C. Blood lipid response to 20 weeks of supervised exercise in a large biracial population: The HERITAGE Family Study. *Metabolism* 2000; 49: 513-520.
26. Israel RG, Sullivan MJ, Marks RHL, Cayton RS, & Cheiner TC. Relationship between cardiorespiratory fitness and lipoprotein(a) in men and women. *Med Sci Sports Exerc* 1994; 26: 425-431.
27. Wilmore JH, Stanforth PR, Gagnon J, Rice T, Mandel S, Leon AS, Rao DC, Skinner JS, & Bouchard C. Heart rate and blood pressure changes with endurance training: The HERITAGE Family Study. *Med Sci Sports Exerc* 2001; 33: 107-116.
28. Dickhuth H, Horstmann T, Staiger J, Reindell H, & Keul J. The long term involution of physiological cardiomegaly and cardiac hypertrophy. *Med Sci Sports Exerc* 1989; 21: 244-149.
29. Ainslie PN, Reilly T, Maclaren DPM, & Campbell IT. Changes in plasma lipids and lipoproteins following 10 days of prolonged walking: influence of age and relationship to physical activity level. *Ergonomics* 2005; 48:1352-1364.
30. Couillard C, Després JP, Lamarche B et al. Effects of endurance exercise training on plasma HDL cholesterol levels depends on levels of triglycerides: evidence from men of the Health, Risk Factors, Exercise Training and Genetics (HERITAGE) family study. *Arterioscler Thromb Vasc Biol* 2001; 21: 1226-1232.
31. Nye ER, Robertson MC, & Sutherland WH. Clinical features of ischaemic heart disease correlated with high density lipoprotein cholesterol levels in men with hyperbetalipoproteinaemia. *NZ Med J* 1984; 97: 437-442.
32. Nikkila EA, Kuusi T, & Myllynen P. High density lipoprotein and apolipoprotein A-I during physical inactivity. *Atherosclerosis* 1988; 37: 457-462.
33. Fung TT, Hu FB, Yu J, Chu NF, Spiegelman D, Tofler GH, Willett WC, & Rimm EB. Leisure-time physical activity, television watching, and plasma biomarkers of obesity and cardiovascular disease risk. *Am J Epidemiol* 2000; 152: 1171-1178.
34. Talmud PJ, Hawe E, Miller GJ, & Humphries SE. Nonfasting apolipoprotein B and triglyceride levels as a useful predictor of coronary heart disease risk in middle-aged UK men. *Arterioscler Thromb Vasc Biol* 2002; 22: 1918-1923.