

# Cardiorespiratory Fitness and Serum Lipoprotein and Apolipoprotein Profiles in Men and Women

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## Keywords:

*Cardiorespiratory fitness* (心肺持久体力)

*Coronary heart disease (CHD)* (冠状動脈疾患)

*Lipids* (脂質)

*Lipoproteins* (リポプロテイン)

*Apolipoproteins.* (アポリポプロテイン)

## Abstract

*Background:* Previous studies have suggested that vigorous physical activity is associated with a reduced risk of coronary heart disease (CHD). However, the relationship of cardiorespiratory fitness to apolipoproteins has not been well-investigated.

*Objective:* In order to document possible variations of lipoproteins and apolipoproteins in relation to cardiorespiratory fitness, 234 healthy men and 240 women of cardiorespiratory fitness were examined and the serum concentrations of lipoprotein and apolipoprotein A-1, B, and E

investigated.

Methods: Cardiorespiratory fitness using  $\text{VO}_2\text{max}$ , estimated by sub-maximal cycle test, blood pressure, lipid, and lipoprotein profiles, apolipoprotein A-1 (Apo A-1), Apolipoprotein B (Apo B), and other major CHD risk factors were determined. The frequency of weekly leisure activity was reported on in a questionnaire.

Results: Prominent findings for  $\text{VO}_2\text{max}$  were positive associations with HDL-c and Apo A-1 and negative associations with age, triglycerides (TG), Apo-B and Apo-E in both sexes. Also, we found that the activity group had higher levels of  $\text{VO}_2\text{max}$ , serum high density lipoprotein cholesterol (HDL-c) and Apo-A1 to the sedentary group in both genders.

Conclusions: The major finding of this study was that being cardiorespiratory physically fitter improves apolipoprotein profiles, which, in turn, could provide protection against CHD.

## **Introduction**

It is well known that serum lipid and lipoprotein concentrations are determined both genetic and environmental factors, such as diet and physical activity. Elevated serum total cholesterol (TC) level has long been recognized as a risk factor for the development of coronary heart disease (CHD) and atherosclerosis, in general (13). Later studies concerning the distribution of cholesterol between different lipoprotein fractions have shown that, although elevated low density lipoprotein (LDL-c) and triglyceride (TG) were associated with increased risk, high density lipoprotein cholesterol (HDL-c) had a negative correlation with CHD (9,19). HDL-c is supposed to transport cholesterol from peripheral tissues and artery walls to the liver for further catabolism and excretion, thus decreasing cholesterol deposition in the artery walls and helping to prevent the development of coronary sclerosis (25). Recently, a study

indicated that an important part of this relationship resides more specifically in the less dense of the two major HDL subfractions, HDL<sub>2</sub> (14), which contains a relatively high concentration of the important structural protein apolipoprotein A-1 (Apo A-1). An investigator found that Apo A-1 more closely reflects the reduced risk of atherogenesis than any of the HDL-c markers (17). On the other hand, Apo B, the only apoprotein in LDL, is responsible for the specific binding of these particles to the LDL receptor; apolipoprotein E (Apo E) determines serum TC and LDL-c concentrations, and both contribute to CHD risk (15).

Average HDL-c concentrations of endurance athletes are 10–24 mg/dl higher than those of sedentary individuals (12). A positive correlation has been found between exercise conditioning and HDL-c (16). Berg and Keul (2) also showed that HDL-c and Apo A-1 levels correlated primarily with the maximum aerobic capacity in athletes, using stepwise regression analysis. There is little information about the relationship of physical fitness to apolipoprotein levels in the normal population with regular exercise. The mechanism by which a higher level of fitness leads to higher serum concentrations of HDL-c, Apo A-1 and to changes in the levels of other lipoprotein lipids, are not known. For this reason, the data were investigated for relationships which might explain the influence of cardio-respiratory fitness on lipoprotein lipid concentrations, particularly HDL-c, Apo A-1 and Apo E levels.

## **Subjects and Methods**

### Study Subjects

Four hundred and seventy-four healthy Japanese (234 men and 240 women) volunteers participated in our study, aged 25 to 63 years. All of them live in the City of Toyota. They participated mainly in response to

the evaluations of health and fitness. Written informed consent was obtained from the subjects before the examination started. No subject had any illness or abnormality that might have influenced the measurements made in this study.

#### Assessment of Anthropometric Measurements

Subjects were weighed with a digital scale in light clothing without shoes. Body mass index (BMI) was calculated as weight (kg) divided by height squared ( $\text{cm}^2$ ). Skinfold measurements were taken on the right side, using a calipers (made by the National Nutrition Research Institute). Body fat percentage was estimated based on two skinfold (triceps, subscapularis) measurements utilizing a regression equation developed for adults (4,22). Blood pressure was measured from the right upper arm by means of a mercury sphygmomanometer after at least 5 minutes rest, with diastolic pressure recorded at the muffling of sounds; two measurements were made, the lower value being used for analysis, as was the case for resting heart rate (HR) measured at the same time.

#### Assessment of Blood Lipids

Venous blood samples were obtained after a 12-hour overnight fasting, and serum separated according to conventional methods. The serum was recovered after centrifugation at 4 °C, and analyses were completed within 72 hours. TC, TG and HDL-c were analyzed by the enzymatic method. LDL-c was determined according to the Friedewald formula estimated by subtracting HDL-C and one-fifth of the TG value from the TC level (8). Serum Apo A-1, Apo B, and Apo E were determined by single radial immunodiffusion methods, using kits from Daiichi Chemical Company (10).

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### Assessment of Cardiorespiratory Fitness

Cardiorespiratory fitness was measured from a progressive sub-maximal bicycle ergometer exercise test with a pedaling frequency of 60/min. The modified Åstrand and Rhyning protocol of gradually increasing workloads was chosen based upon a previous study (26). The test was terminated when subjects reached 85 per cent of their age-predicted maximal HR. HR was monitored during the test with an ECG using a CM<sub>5</sub> lead, and the last 5 seconds of every minute was recorded on paper. Rate-pressure product (RPP) was determined by multiplying HR and systolic blood pressure. Predicted maximal oxygen uptake ( $\text{VO}_2\text{max}$ ) as a measure of cardiorespiratory fitness was calculated based on HR during the final workload. The test is highly correlated with results of maximal treadmill testing (3). The tests were interrupted prematurely when symptoms such as angina pectoris, dyspnoea, or exhaustion made this necessary. No complications were noted during the exercise tests.

### **Statistical analysis**

Means and standard deviations were calculated for all variables, and the significance of differences tested by the Student *t*-test for unpaired data. The correlations given were computed as linear regression functions. All statistical analyses were performed using commercial software programs.

### **Results**

The mean values and standard deviations of the study population of anthropometrics, predicted  $\text{VO}_2\text{max}$  and serum lipid, lipoproteins and apolipoproteins are presented in Table 1 by each age groups for men and

in Table 2 for women, respectively. Since leisure activity is known to influence  $\text{VO}_2\text{max}$ , the data were also calculated separately for leisure activity and sedentary groups in Table 3 in both genders. It is seen that the leisure activity group in both sexes had significantly higher  $\text{VO}_2\text{max}$ , HDL-c and Apo A-1 values than the sedentary group. Single correlations between the variables are shown in Table 4 for both genders.  $\text{VO}_2\text{max}$  generally decreased with age in both sexes (Fig. 1), as was confirmed in this study, too ( $p < 0.001$ ). Important observations were the significant positive associations between  $\text{VO}_2\text{max}$  with HDL-c and Apo-A1 in both genders, which are shown in more detail in Fig. 2 and 3, respectively. On the other hand,  $\text{VO}_2\text{max}$  showed a significant inverse correlation with TG, Apo B and Apo E in both genders (Table 4). The concentration of HDL-c was significant negatively correlated to TG in both genders.

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**Table 1 Comparison of body mass index, lipid, lipoproteins  
and apolipoproteins in men by age group**

	20-29 n = 18	30-39 n = 72	40-49 n = 78	50-59 n = 54	60-69 n = 12
Age (years)	26.3 ± 1.3	34.9 ± 2.3	43.8 ± 2.2	54.0 ± 2.3	62.5 ± 0.5
Height (cm)	170.6 ± 7.8	172.0 ± 4.7	169.6 ± 3.9	168.4 ± 5.0	168.0 ± 0.2
Weight (kg)	68.9 ± 9.7	65.3 ± 6.2	68.2 ± 3.6	63.3 ± 2.9	62.5 ± 0.5
Body fat (%)	20.3 ± 2.4	18.7 ± 3.4	19.8 ± 2.9	19.3 ± 4.4	18.1 ± 1.6
BMI(kg/m <sup>2</sup> )	23.5 ± 1.4	22.0 ± 1.7	23.6 ± 1.1	22.3 ± 1.1	22.1 ± 0.2
SBP(mmHg)	122.8 ± 11.9	120.4 ± 7.9	119.1 ± 9.9	127.4 ± 8.7	132.5 ± 3.1
DBP(mmHg)	67.5 ± 3.7	74.2 ± 6.6	73.2 ± 8.7	78.7 ± 6.3	78.6 ± 2.3
HR (beats/min)	66.4 ± 6.5	69.2 ± 5.7	70.7 ± 6.3	71.8 ± 5.3	74.0 ± 5.3
RPP (× 10 <sup>3</sup> )	79.7 ± 14.6	83.5 ± 10.6	84.5 ± 12.0	91.5 ± 9.5	98.1 ± 7.9
VO <sub>2max</sub> (ml/kg/min)	48.0 ± 2.4	41.7 ± 3.9	37.9 ± 3.7	37.9 ± 2.1	36.8 ± 1.6
HDL-c (mg/dl)	65.7 ± 9.2	57.3 ± 7.6	52.2 ± 5.9	58.0 ± 10.4	56.0 ± 7.4
LDL-c (mg/dl)	80.2 ± 10.8	101.2 ± 21.0	113.6 ± 13.9	119.8 ± 23.5	101.8 ± 8.8
VLDL-c (mg/dl)	15.9 ± 5.3	18.2 ± 4.0	20.1 ± 5.5	21.7 ± 5.7	17.8 ± 2.9
T-cho (mg/dl)	161.8 ± 15.2	176.7 ± 21.5	186.1 ± 13.3	199.6 ± 19.4	175.6 ± 7.9
TG (mg/dl)	79.6 ± 26.5	91.3 ± 20.1	100.9 ± 27.3	108.7 ± 28.6	89.1 ± 14.2
LDL-c/HDL-c (%)	1.2 ± 0.2	1.8 ± 0.5	2.2 ± 0.4	2.1 ± 0.8	1.8 ± 0.3
T-cho/HDL-c (%)	2.4 ± 0.2	3.1 ± 0.5	3.6 ± 0.5	3.5 ± 1.0	3.1 ± 0.4
Apo A-1 (mg/dl)	155.6 ± 12.6	152.6 ± 23.4	138.2 ± 11.5	154.7 ± 23.9	163.3 ± 5.0
Apo B (mg/dl)	72.0 ± 3.6	79.5 ± 18.2	85.2 ± 10.2	94.5 ± 17.6	81.3 ± 6.7
Apo E (mg/dl)	3.7 ± 0.4	4.5 ± 1.1	5.1 ± 1.3	5.6 ± 1.4	4.9 ± 1.3
Apo A-1/B (%)	2.1 ± 0.2	2.0 ± 0.6	1.6 ± 0.2	1.6 ± 0.4	2.0 ± 0.2

Note. Results are means ± SD.

Abbreviation: BMI; body mass index.

HR; heart rate.

SBP; systolic blood pressure.

DBP; diastolic blood pressure.

RPP; rate pressure product (heart × systolic BP).

VO<sub>2max</sub>; maximal oxygen uptake

Table 2 Comparison of body mass index, lipid, lipoproteins and apolipoproteins in women by age group

	20-29 n = 24	30-39 n = 180	40-49 n = 36
Age (years)	28.2 ± 0.4	32.4 ± 2.4	44.5 ± 2.6
Height (cm)	159.2 ± 8.5	164.0 ± 5.2	159.6 ± 3.4
Weight (kg)	52.7 ± 3.4	54.3 ± 6.4	53.3 ± 4.3
Body fat (%)	19.4 ± 3.7	18.5 ± 2.8	19.0 ± 3.3
BMI(kg/m <sup>2</sup> )	21.0 ± 2.9	20.1 ± 1.9	21.0 ± 2.6
SBP(mmHg)	106.5 ± 3.8	109.6 ± 6.8	113.5 ± 5.2
DBP(mmHg)	65.0 ± 3.9	65.7 ± 6.3	71.1 ± 4.8
HR (beats/min)	68.0 ± 5.2	68.0 ± 5.1	66.4 ± 3.4
RPP ( × 10 <sup>3</sup> )	72.5 ± 6.5	74.6 ± 7.7	75.5 ± 6.1
VO <sub>2</sub> max (ml/kg/min)	37.1 ± 2.5	36.5 ± 2.2	32.6 ± 5.7
HDL-c (mg/dl)	70.4 ± 5.6	70.1 ± 8.6	65.5 ± 7.7
LDL-c (mg/dl)	90.5 ± 12.3	92.5 ± 14.0	107.9 ± 20.3
VLDL-c (mg/dl)	13.5 ± 4.7	13.2 ± 5.3	19.9 ± 4.8
T-cho (mg/dl)	174.6 ± 9.4	175.9 ± 14.0	193.4 ± 19.3
TG (mg/dl)	67.7 ± 23.3	64.4 ± 21.6	99.5 ± 23.8
LDL-c/HDL-c (%)	1.3 ± 0.3	1.3 ± 0.3	1.6 ± 0.5
T-cho/HDL-c (%)	2.5 ± 0.3	2.5 ± 0.4	3.0 ± 0.5
Apo A-1 (mg/dl)	161.7 ± 18.1	155.4 ± 16.2	154.4 ± 13.0
Apo B (mg/dl)	70.2 ± 12.2	68.0 ± 11.0	93.0 ± 25.3
Apo E (mg/dl)	4.0 ± 0.6	4.3 ± 0.9	5.2 ± 1.3
Apo A-1/B (%)	2.3 ± 0.4	2.3 ± 0.4	1.8 ± 0.9

Note. Results are means ± SD.

Abbreviation: BMI; body mass index.

HR; heart rate.

SBP; systolic blood pressure.

DBP; diastolic blood pressure.

RPP; rate pressure product (heart × systolic BP).

VO<sub>2</sub>max; maximal oxygen uptake



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Table 3 Comparison of body mass index, lipids, lipoproteins and apolipoproteins in samples of active and inactive groups by gender

	Men			Women		
	Leisure activity ( $\geq 1$ times/wk) n = 120	Sedentary n = 114	between- group p value	Leisure activity ( $\geq 1$ times/wk) n = 53	Sedentary n = 187	between- group p value
Age (years)	39.6 $\pm$ 9.3	46.6 $\pm$ 8.8		33.3 $\pm$ 5.9	33.9 $\pm$ 5	
Height (cm)	170.7 $\pm$ 5.3	169.4 $\pm$ 4.3		162.2 $\pm$ 3.5	163 $\pm$ 6.2	
Weight (kg)	65.2 $\pm$ 5.4	66.7 $\pm$ 5.3		52.3 $\pm$ 5.3	54.5 $\pm$ 6	
Body fat (%)	18.4 $\pm$ 3.1	20.2 $\pm$ 3.5		18.1 $\pm$ 2.7	18.8 $\pm$ 3	
BMI(kg/m <sup>2</sup> )	22.3 $\pm$ 1.2	23.2 $\pm$ 1.6		19.9 $\pm$ 2.1	20.5 $\pm$ 2.2	
SBP(mmHg)	121 $\pm$ 10.2	123.9 $\pm$ 9.1		110.1 $\pm$ 6.8	109.8 $\pm$ 6.5	
DBP(mmHg)	73.4 $\pm$ 7.4	75.9 $\pm$ 7.7		66.1 $\pm$ 6.6	66.5 $\pm$ 6	
HR (beats/min)	68.4 $\pm$ 5.9	72 $\pm$ 5.9		68.3 $\pm$ 4.1	67.6 $\pm$ 5.1	
RPP ( $\times 10^3$ )	83 $\pm$ 11.7	89.5 $\pm$ 11		75.2 $\pm$ 6.5	74.3 $\pm$ 7.6	
VO <sub>2</sub> max (ml/kg/min)	42.9 $\pm$ 3.8	36.5 $\pm$ 2.1	***	39.5 $\pm$ 1.8	34.9 $\pm$ 3	***
HDL-c (mg/dl)	60 $\pm$ 7.8	52.5 $\pm$ 8	***	76.5 $\pm$ 5.4	67.5 $\pm$ 8	***
LDL-c (mg/dl)	104.7 $\pm$ 23.5	111.5 $\pm$ 18.3		86.2 $\pm$ 12.5	97 $\pm$ 16	
VLDL-c (mg/dl)	18 $\pm$ 5.2	21 $\pm$ 4.8		13.3 $\pm$ 5.7	14.5 $\pm$ 5.3	
T-cho (mg/dl)	182.8 $\pm$ 22.3	185 $\pm$ 18.4		176 $\pm$ 12.3	179.1 $\pm$ 16.7	
TG (mg/dl)	90.2 $\pm$ 26.2	105.1 $\pm$ 24.1		60.6 $\pm$ 16.5	72.7 $\pm$ 26.7	
LDL-c/HDL-c (%)	1.7 $\pm$ 0.5	2.1 $\pm$ 0.6		1.1 $\pm$ 0.2	1.4 $\pm$ 0.4	
T-cho/HDL-c (%)	3.1 $\pm$ 0.6	3.6 $\pm$ 0.7		2.3 $\pm$ 0.2	2.6 $\pm$ 0.4	
Apo A-1 (mg/dl)	156 $\pm$ 18.6	141.6 $\pm$ 19.9	***	166.6 $\pm$ 15.2	152.8 $\pm$ 14.9	***
Apo B (mg/dl)	81.7 $\pm$ 15.6	87.1 $\pm$ 15.8		66.5 $\pm$ 9.4	73.5 $\pm$ 17.9	
Apo E (mg/dl)	4.3 $\pm$ 1	5.6 $\pm$ 1.3		3.7 $\pm$ 0.5	4.6 $\pm$ 1	
Apo A-1/B (%)	1.9 $\pm$ 0.5	1.6 $\pm$ 0.4		2.5 $\pm$ 0.5	2.1 $\pm$ 0.5	

Note. Results are means  $\pm$  SD. \*\*\* p < 0.001 between-group, leisure activity vs. sedentary.

Abbreviation: BMI; body mass index.

HR; heart rate.

SBP; systolic blood pressure.

DBP; diastolic blood pressure.

RPP; rate pressure product (heart  $\times$  systolic BP).

VO<sub>2</sub>max; maximal oxygen uptake

**Table 4 Correlation matrix of the variables investigated in both sexes**

♂ ♀	Age	Fat %	BMI	SBP	DBP	HR	RPP	VO2 max	HDL .c	LDL .c	Total .c	TG	Apo A-1	Apo B	Apo E
Age		0.14 *	0.07	0.23 ***	0.29 ***	0.27 ***	0.31 ***	-0.50 ***	-0.11	0.39 ***	0.41 ***	0.23 ***	0.05	0.29 ***	0.31 ***
Fat %			0.51 ***	0.04	0.05	0.02	0.04	-0.18 **	-0.18 **	-0.10	-0.16 *	0.07	-0.32 ***	-0.07	0.14 *
BMI				-0.04	-0.05	0.04	0.01	-0.21 **	-0.26 ***	0.08	0.02	0.21 **	-0.40 ***	0.07	0.34 ***
SBP					0.54 ***	0.36 ***	0.81 ***	-0.12	0.10	-0.04	0.06	0.22 **	0.04	0.15 *	0.17 *
DBP						0.47 ***	0.61 ***	-0.21 **	0.04	0.20	0.28 ***	0.23 ***	-0.06	0.25 ***	0.17 *
HR							0.84 ***	-0.38 ***	-0.19 **	0.22 **	0.25 ***	0.43 ***	-0.15 *	0.20 **	0.35 ***
RPP								-0.34 ***	-0.07	0.12	0.19 **	0.40 ***	-0.08	0.22 **	0.32 ***
VO2 max									0.60 ***	-0.27 ***	-0.14 *	-0.44 ***	0.37 ***	-0.31 ***	-0.58 ***
HDL .c										-0.38 ***	-0.06	-0.35 ***	0.63 ***	-0.21 **	-0.33 ***
LDL .c											0.92 ***	0.17 *	-0.32 ***	0.26 **	0.13 *
Total .c												0.28 ***	-0.12	0.67 ***	0.13 *
TG													0.22 **	0.27 ***	0.51 ***
Apo A-1														-0.19 **	-0.16 *
Apo B															0.26 **
Apo E															

\* p < 0.05. \*\* p < 0.01. \*\*\* p < 0.001

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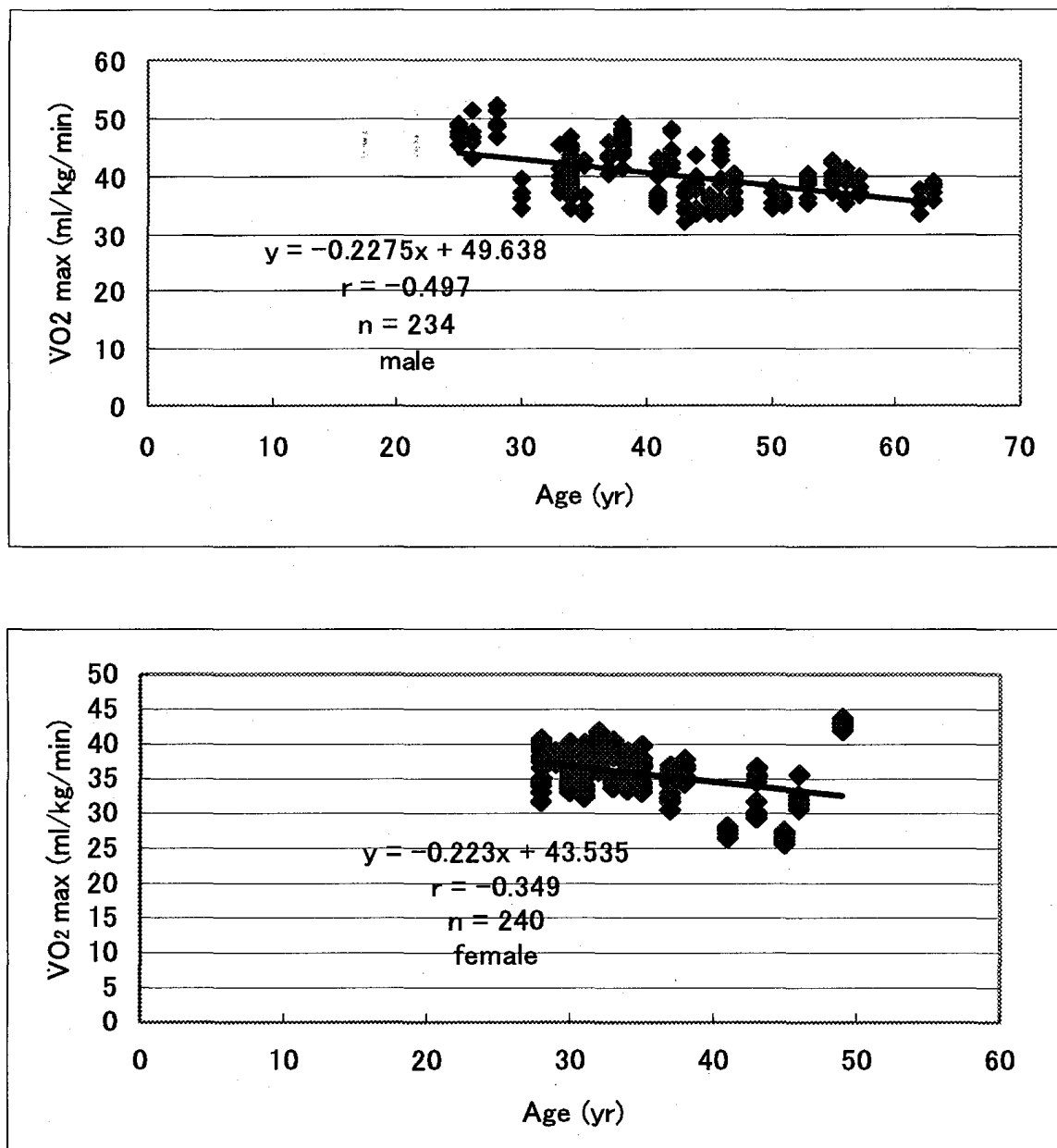


Fig. 1. Relation between cardiorespiratory fitness (VO<sub>2</sub>max) and age in men (top) and women (bottom).

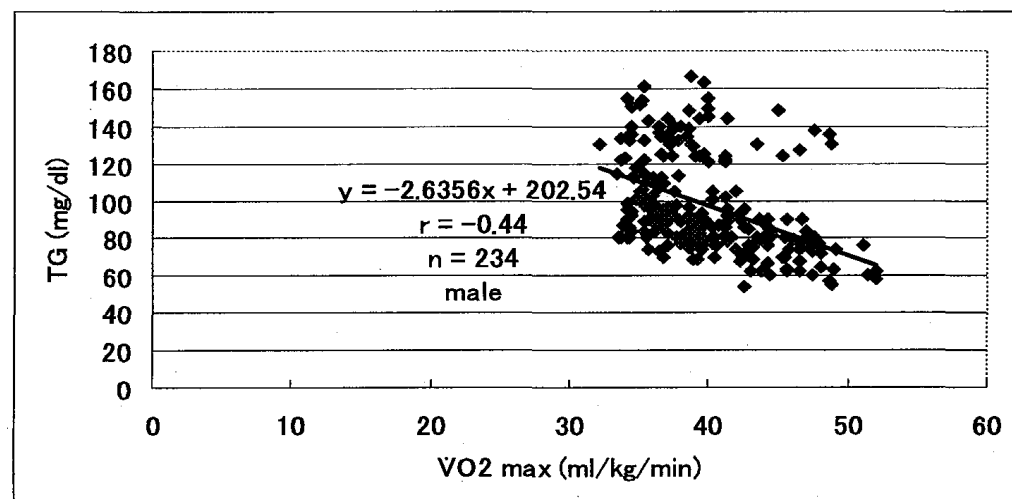
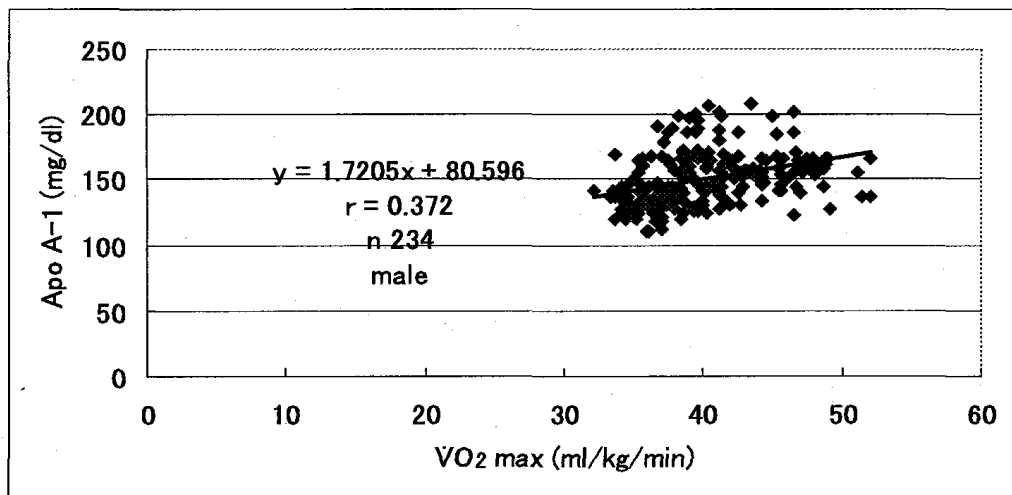
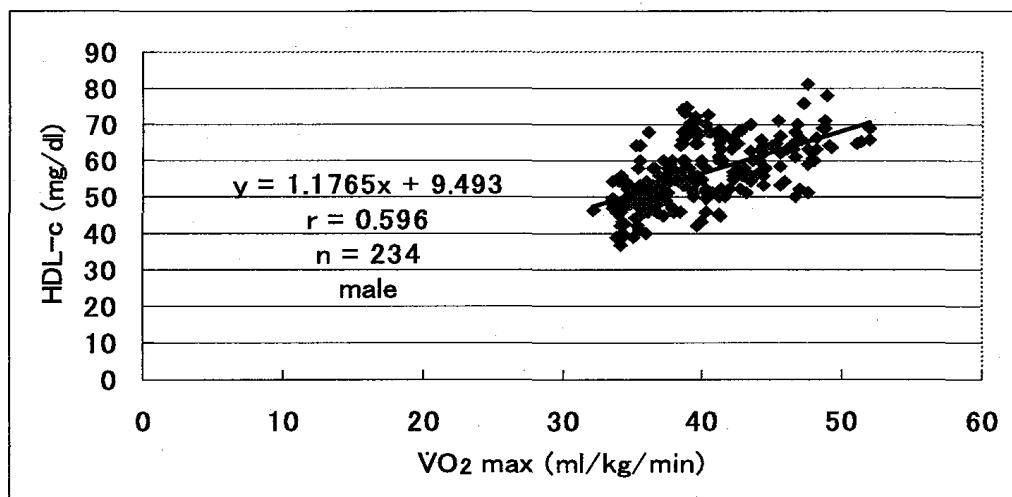


Fig. 2. Relation between cardiorespiratory fitness (VO<sub>2</sub> max) and Apo A-1 (top), HDL-c (middle) and TG (bottom) in men.

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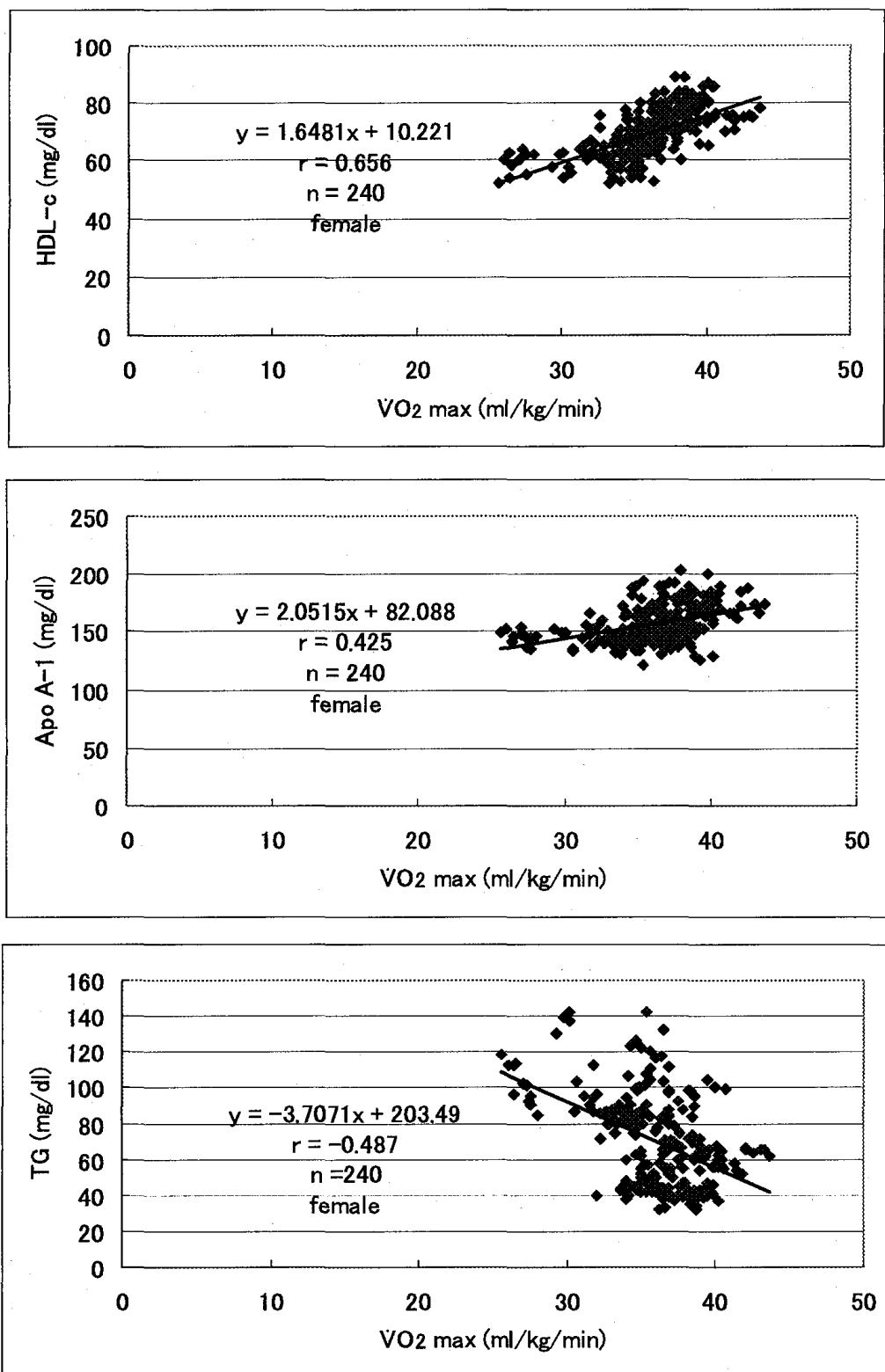


Fig. 3. Relation between cardiorespiratory fitness (VO<sub>2</sub> max) and Apo A-1 (top), HDL-c (middle) and TG (bottom) in women.

## Discussion

The decline in cardiorespiratory fitness, indexed as  $\text{VO}_2\text{max}$ , generally seen with aging (5,7), was observed in this population in both genders. The results of the present study also agreed with those previously reported, that HDL-c and  $\text{VO}_2\text{max}$  were statistically significantly higher in the leisure activity group than in the sedentary group (12). The association among low resting heart rate, low RPP, low LDL-c and low incidence of CHD is often ascribed to  $\text{VO}_2\text{max}$ , since exercise training decreases LDL-c, heart rate, and systolic blood pressure at rest. In this study, we observed a weakly negative, but, significant correlation between  $\text{VO}_2\text{max}$  and resting heart rate, body fat percentage and LDL-c in both genders. It is well known that cholesterol is principally carried by LDL-c in the blood and has been positively associated with development of premature CHD. Thus, it is believed that leisure activity is one of the important factors that can improve cardiorespiratory fitness and reduce CHD risk.

Generally, regular participation in physical activity is associated with lower plasma TG concentrations. The present study found that the leisure activity group tended to have lower TG levels in both gender than did the sedentary group. This is consistent with earlier work by Martin, et al. (18) who reported relatively constant mean plasma TG concentrations in runners, as compared with an inactive group.

The primary function of HDL-c has become clearer in recent years. There are several reports indicating that exercise conditioning is one of the factors that increase HDL-c. It serves as the cholesterol acceptor in the reverse transport and excretion of cholesterol. The result is a constant flux in HDL composition and a transport mechanism for net movement of cholesterol from the peripheral vascular compartment and tissues to the

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liver for excretion as bile. Patients who have cardiovascular disorders have been found to have reduced HDL-c and Apo A-1, as compared with normal, healthy individuals (1,24). The consistency of these results is noteworthy, and it implies that HDL-c and Apo A-1 levels may be useful predictors of heart disease potential in apparently healthy individuals. Although little information exists about the exercise-induced mechanism of increased  $\text{VO}_2\text{max}$  on lipoprotein and apolipoprotein metabolism, reports on the antiatherogenic effect of endurance training have been previously published (2,16). In most case control studies where endurance athletes are compared with less active controls, there have been differences in lipids and lipoproteins associated with differences in  $\text{VO}_2\text{max}$  (6,21). The present study found that the leisure activity group had significantly higher serum HDL-c, Apo-A1 and  $\text{VO}_2\text{max}$  values in both genders than did the sedentary group. Moreover, Miller et al. (20) mentioned that there was a strong positive correlation between aerobic capacity and HDL-c ( $r = 0.81$ ) in healthy males of varying degrees of habitual physical activity. Schwarts, et al., (27) who reported that baseline  $\text{VO}_2\text{max}$  was related to HDL-c level, also found that the relationship between HDL-c and Apo-A1 had a very good correlation ( $r = 0.82$ ) before exercise training in all 26 subjects, after exercise training, this relationship was almost identical and most subjects seem to move up the line representing the baseline relationship ( $r = 0.84$ ). Our study showed that the level of  $\text{VO}_2\text{max}$  is positively correlated with HDL-c, and also positively correlated with Apo-A1 concentrations in both genders. On the other hand, our study showed the level of  $\text{VO}_2\text{max}$  was significantly negatively correlated to TG and body fat percentage in both genders. Consequently, these results may support the hypothesis that a higher level of  $\text{VO}_2\text{max}$  influences lipoprotein metabolism by modifying body

fatness (28). Also, the present study strongly suggested that being physically fitter may contribute toward lessening the risk of cardiovascular disorders.

A cross-sectional data analysis indicates much higher serum Apo A-1 levels in endurance trained athletes than in age-matched controls (28). One study showed that dynamic exercise programs elevate HDL-c and reduce LDL-c and VLDL-c levels in adults, and that there are significant positive correlations between Apo A-1 as well as HDL-c and individual maximum aerobic capacity in well-trained male athletes (11). The present study found higher Apo A-1 levels in the leisure activity group compared with the sedentary group, and also found good relationships among Apo A-1 concentration, HDL-c and  $\text{VO}_2\text{max}$  in both genders. On the other hand, Apo B may play an important role in the etiology of atherosclerosis (23). Yet, research on the effect of exercise training on Apo B is sparse. In the Stanford study, Wood et al. (29) observed no change in Apo B after one year of training, however, its change was negatively correlated with distance running ( $r = -0.29$ ,  $p < 0.05$ ). The findings of the present study support those previously observed that serum Apo B levels were slightly lower in the leisure activity group in both genders, and found a similar negatively significant relationship between  $\text{VO}_2\text{max}$  and Apo B in both sexes. Although the mechanism by which physical activity may alter apolipoprotein profiles is unclear, it is postulated that one way may be through increased activation of lipoprotein lipase activity, thereby causing catabolism of triglycerides.

It can be concluded that regular leisure activity can benefit serum apolipoprotein profiles, as well as lipid and lipoproteins profiles, and also contribute toward lessening the risk of cardiovascular disorders.

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