Effect of Kinesiologic Recreation on Plasma Lipoproteins and Apolipoproteins in Fertile Women

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The effect of physical exercise on lipid and apoprotein levels was studied in 31 healthy fertile women (mean age, 39.7 ± 2.3 years) working as civil servants and leading a mostly sedentary way of life (group 1). A control group consisted of 31 age-matched women (mean age, 39.2 ± 2.4 years) with a comparable life-style (group 2). Group 1 performed physical exercise for at least 30 minutes three times per week. They also climbed a 500-m hill at least once per week. The study lasted 6 months, ie, from May to November 1990. Changes in maximum oxygen consumption ($\dot{V}O_2max$), body weight, body mass index (BMI), waist to hip ratio (WHR), and levels of lipids and apolipoproteins (apos) A-I and B were compared between the two groups of subjects. During the May-November period, the control group showed an increase in body weight (*P* < .02), total cholesterol, high-density lipoprotein (HDL) cholesterol, HDL_3, and low-density lipoprotein (LDL) cholesterol (*P* < .01) and a decrease in HDL_2 (*P* < .05). In contrast, group 1 did not show any increase in total cholesterol, and their body weight decreased (*P* < .01). Very–low-density lipoprotein (VLDL) cholesterol and triglyceride levels decreased (*P* < .02), as did LDL cholesterol and HDL_2 levels (*P* < .05), whereas HDL cholesterol and HDL_3 levels increased (*P* < .01). There were no statistically significant changes in WHR and apo A-I level. The findings indicated possible seasonal variations in lipoprotein levels in group 2. In addition, programed kinesiologic recreation appeared to decrease levels of some atherogenic lipoproteins (LDL cholesterol, VLDL cholesterol, total cholesterol, and apo B in fertile women, which is known to prevent the onset of coronary heart disease.

ELEVATED LEVELS of cholesterol, low-density lipoprotein (LDL), and triglycerides and inversely decreased high-density lipoprotein (HDL) levels are risk factors for the development of coronary heart disease.[1,2] Epidemiologic studies have shown that regular physical exercise prevents the development of coronary heart disease,[3] decreases cholesterol, triglyceride, and LDL cholesterol levels, and increases HDL cholesterol levels.[4] Most studies on the effect of physical exercise on plasma lipoproteins have been conducted in males, and to a much lesser extent in females. To the best of our knowledge, the effect of kinesiologic exercise on lipoprotein metabolism in women with a sedentary life-style has hardly been studied at all.

Therefore, we embarked on this study of the effect of a 6-month kinesiologic recreation program on lipoprotein and apolipoprotein metabolism in normal, young, fertile, normolipemic, working women with a sedentary life-style.

SUBJECTS AND METHODS

Subjects

Thirty-one clinically healthy female volunteers aged 35 to 45 years with normal ECG, hepatologic, and renal-function test findings were included in the study (group 1). A control group consisted of 31 age- and job-matched women employed by the same company (group 2). Selection was made by randomization in the registered sample of volunteers engaged in the same type of job and employed by the same firm. Each odd-numbered volunteer was included in the investigation (group 1) and each even-numbered volunteer in the control group (group 2).

All the women reported normal menstrual cycles. None drank alcoholic beverages regularly. Eleven women in group 1 and nine in group 2 smoked up to 20 cigarettes per day. Eighteen women in group 1 and 21 in group 2 drank 1 to 3 cups of coffee per day. None of the women used contraceptives or any other agents that could affect lipoprotein metabolism. All the women were advised not to change their nutritional pattern or, where applicable, reduce smoking or coffee intake during the course of the study. Both group 1 and group 2 subjects worked as civil servants, mostly leading a sedentary way of life. At home, they did their usual housework. All the women were married and had one to three children.

Exercise

The study lasted 6 months, ie, from May to November 1990. Three times per week, group 1 subjects performed a 30- to 60-minute kinesiologic exercise consisting of a 10-minute run, 10 minutes of playing basketball, 10 minutes of lawn exercise, and possibly another 10-minute run. In addition, they climbed a 500-m hill once per week (on Saturdays or Sundays).

Samples

Body weight, body height, and waist and hip circumference were measured, body mass index (BMI) and waist to hip ratio (WHR) calculated, and skinfold thickness caliper-determined. Blood samples were taken in the morning after a 10- to 12-hour fast and collected in EDTA-containing tubes. Plasma was separated by centrifugation, and the samples were stored at −20°C until analysis. Total cholesterol, HDL cholesterol (including HDL_2 and HDL_3 fractions), LDL cholesterol, very–low-density lipoprotein (VLDL) cholesterol, triglyceride, and apolipoprotein (apo) A and B levels were determined.

Lipoprotein Determinations

Total cholesterol, HDL cholesterol, VLDL cholesterol, and triglyceride levels were determined using commercial kits according to the manufacturer's methods (Abbott Laboratories, Wiesbaden-Delkenhaim, Germany). LDL cholesterol was determined as follows: LDL cholesterol = total cholesterol − (HDL cholesterol + VLDL cholesterol).[5,6] HDL_3 was determined using a commercial kit (Immuno, Vienna, Austria), and HDL_2 was calculated as HDL_2 = HDL cholesterol − HDL_3 cholesterol.[7] Apolipo-

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proteins were determined with radioimmunologic kits[8] (Pharmacia Diagnostics, Uppsala, Sweden).

Maximum oxygen consumption ($\dot{V}O_2max$) was determined by a progressive test on a motor-driven treadmill, using the method reported by Saltin and Astrand.[9] The proportion of fatty tissue was determined by caliper measurement of skinfold thickness. WHR was also determined for all subjects.

Statistical Analysis

The variables studied were analyzed using the paired *t* test for time-related within-group changes and independent *t* test for between-group comparison. Linear regression correlation was determined among all the variables to evaluate interrelationships.

RESULTS

Subjects' characteristics are listed in Table 1. Initially, there were no statistically significant differences between group 1 and group 2 according to age, body height, body weight, BMI, WHR, and $\dot{V}O_2max$. No statistically significant differences were found in plasma cholesterol, HDL cholesterol, HDL cholesterol subfraction (HDL_2 and HDL_3), LDL cholesterol, VLDL cholesterol, triglyceride, and apo A-I and B levels between groups 1 and 2 (Table 2).

However, at the end of the study (Table 1) body weight showed a statistically significant decrease and increase in groups 1 and 2, respectively. A statistically significant increase in $\dot{V}O_2max$ was recorded in group 1, whereas no change was observed in group 2. Lipoprotein concentrations also changed at the end of the study (Table 2). In group 1, a statistically significant decrease was recorded for LDL cholesterol, VLDL cholesterol, and triglyceride levels, whereas HDL cholesterol and HDL_3 cholesterol levels showed a statistically significant increase. Total cholesterol and apo A-I levels remained unchanged, whereas apo B levels showed only a slight decrease. HDL_2 levels showed a statistically significant decrease.

However, in group 2, total cholesterol, LDL cholesterol, HDL cholesterol, and HDL_3 cholesterol levels showed a statistically significant increase. In group 2, and likewise in group 1, HDL_2 concentrations also showed a statistically significant decrease. No statistically significant changes were observed in VLDL cholesterol, triglyceride, and apo A-I and apo B concentrations.

Statistically significant differences in certain parameters observed in group 2 resulted in statistically significant differences in total cholesterol, LDL cholesterol, VLDL cholesterol, triglyceride, and apo B levels between groups 1 and 2.

No correlation was found between body weight, BMI, and WHR and the levels of HDL and HDL_2 and HDL_3 subfractions in either group.

DISCUSSION

When comparing lipoprotein concentrations determined in different seasons, possible seasonal variations should be taken into account. Controversial findings have been reported in the literature. Most investigators report higher winter concentrations of cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides,[10-13] whereas others have failed to observe such variations.[14,15] Recent studies have revealed that even daily and monthly oscillations of lipids and apolipoproteins occur,[16-18] but this observation has not been confirmed by all who study the issue.[19] In our control group (group 2), concentrations of total cholesterol, LDL cholesterol, HDL cholesterol, and HDL_3 were observed to increase in a statistically significant manner, whereas the level of HDL_2 decreased.

For the time being, the etiology and mechanism of these seasonal variations remain unknown. We can only speculate about the possible external causes (eg, length of daylight and temperature)[20] and internal causes (eg, ethnic differences, hormone state, and activities of certain enzymes).[21]

Data on the effect of physical exercise on lipoprotein levels in normal subjects are controversial. Whereas some investigators report positive effects,[22,23] others failed to observe them.[24,25] However, this controversy may be due to differences in the intensity of exercise, duration of performance, and characteristics of subjects. Thus, attempts were made to determine the lowest exercise intensity at which positive effects on lipoprotein levels may be expected.[26] In our group 1, the level of total cholesterol did not change, whereas in group 2, a statistically significant increase was recorded. However, a statistically significant decrease was recorded in LDL cholesterol levels in group 1, in contrast to group 2.

Some researchers consider that an exercise-induced im-

Table 1. Effect of Exercise on Body Weight, Waist and Hip Circumferences, WHR, BMI, and $\dot{V}O_2max$

	May		November	
	Group 1	Group 2	Group 1	Group 2
Age (yr)	39.7 ± 2.3	39.2 ± 2.4	—	—
Height (cm)	164.29 ± 6.68	161.74 ± 4.99	164.29 ± 6.68	161.74 ± 4.99
Weight (kg)	70.38 ± 13.34	71.66 ± 11.13	67.79 ± 12.44†§	72.75 ± 11.23*
BMI (kg/m²)	26.02 ± 6.13	27.10 ± 6.19	25.28 ± 6.01	27.86 ± 6.18
Waist circumference (cm)	81.23 ± 11.00	82.52 ± 8.87	79.90 ± 10.86	82.68 ± 8.75
Hip circumference (cm)	105.35 ± 10.00	107.00 ± 8.75	104.06 ± 9.81	107.23 ± 8.05
WHR	0.77 ± 0.05	0.77 ± 0.05	0.77 ± 0.05	0.77 ± 0.05
$\dot{V}O_2max$ (L/min)	2.29 ± 0.53	2.45 ± 0.49	2.53 ± 0.43†‡	2.42 ± 0.49

NOTE. Mean ± SD.

*$P < .02$, †$P < .01$: within-group comparisons.

‡$P < .05$, §$P < .01$: between-group comparisons.

Table 2. Effect of Exercise on Lipoprotein and Apolipoprotein Levels (mean ± SD)

	May		November	
	Group 1	Group 2	Group 1	Group 2
Total cholesterol	5.11 ± 0.88	5.72 ± 0.76	5.14 ± 0.69	6.59 ± 0.73‡¶
HDL cholesterol	1.40 ± 0.27	1.41 ± 0.31	1.67 ± 0.36‡	1.63 ± 0.38‡
HDL_2	0.43 ± 0.16	0.42 ± 0.18	0.35 ± 0.16*	0.34 ± 0.16*
HDL_3	0.98 ± 0.20	0.99 ± 0.19	1.34 ± 0.29‡	1.30 ± 0.29‡
LDL cholesterol	3.41 ± 0.74	3.68 ± 0.62	3.05 ± 0.64*	4.34 ± 0.71‡¶
VLDL cholesterol	0.50 ± 0.20	0.64 ± 0.29	0.42 ± 0.12†	0.62 ± 0.29‖
Triglycerides	1.30 ± 0.43	1.42 ± 0.63	0.93 ± 0.28†	1.45 ± 0.73¶
Apo A-I	1.43 ± 0.27	1.45 ± 0.25	1.47 ± 0.29	1.43 ± 0.24
Apo B	0.82 ± 0.21	0.85 ± 0.23	0.75 ± 0.20	0.86 ± 0.23§

*$P < .05$, †$P < .02$, ‡$P < .01$: within-group comparisons.
§$P < .05$, ‖$P < .02$, ¶$P < .01$: between-group comparisons.

provement in lipid levels might be due to a reduction in body weight or changes in the composition of body structures.[27] Different effects of exercise- or diet-induced body weight reduction on lipid levels have been reported.[28] In our study, a statistically significant body weight reduction was observed in group 1, in contrast to group 2, in whom body weight increased. The effect of physical exercise of decreasing the cardiovascular disease incidence is considered to reflect the effect of exercise on the level of HDL cholesterol. Physical exercise is known to increase the concentration of HDL cholesterol,[29,30] although opposite findings have also been reported.[31] Some studies have suggested that the effect of physical exercise on HDL concentration does not induce body weight changes,[27] but instead a reduction in abdominal fatty tissue.[32] Measurement of the proportion of abdominal fat and the composition of body structures provides a better correlation for HDL cholesterol level than obesity itself.[33] In our study, neither group 1 nor group 2 subjects were obese. No correlation was found between HDL level and BMI or WHR. Most researchers who investigated the effect of exercise on HDL levels in females failed to observe any statistically significant changes,[34-36] although opposite reports are also found in the literature.[37]

In this study, no changes were observed in group 1 as compared with group 2. The difference in the lipoprotein response to exercise between males and females might be due to the fact that women leading a sedentary way of life have a better lipoprotein profile than men, which might be consequential to the level of endogenous sex steroids. In group 1, no significant changes were observed in the level of apo A-I, whereas the level of apo A-II was not determined. Several studies have demonstrated that the level of apo A-II is increased by physical exercise.[30,38]

Some investigators observed a decrease in apo A-I in women who exercised,[38] but others failed to confirm it.[37,39] Our findings are consistent with studies that report a decrease in LDL cholesterol and apo B.[40,41]

Accordingly, further investigations of the effect of exercise, especially programed kinesiologic recreation, on levels of HDL cholesterol, HDL_2 and HDL_3 subfractions, and apoproteins in fertile women might prove interesting and intriguing.

In conclusion, possible seasonal variations in lipoprotein levels were observed in fertile women. Programed kinesiologic recreation performed over a prolonged period also appeared to decrease levels of atherogenic lipoprotein fractions in fertile women, thus preventing the development of coronary heart disease.

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