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Full Length Research Paper

Changes in serum lipid profile following moderate exercise

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In this study, 90 middle- aged men were randomly assigned to exercise (E, n= 44) and control groups (C, n= 46) to examine the role of physical exercise on serum lipoproteins. While the exercise group participated in a 2-month training program, the control group maintained their previous exercise habits. Serum triglycerides decreased from 1.54 ± 0.10 to 1.27 ± 0.08 mmol/1 (p<0.001) and high-density lipoprotein cholesterol increased from 1.27 ± 0.04 to 1.41 ± 0.04 mmol/1 (p<0.01) in the exercise group after intervention. As the concentration of apolipoprotein Al stayed constant in both groups, the ratio of HDL cholesterol/apolipoprotein All increased only in the exercise group. While the change in serum triglycerides in the exercise group was not dependent on weight reduction, high-density lipoprotein cholesterol changed based on weight reduction.

Key words: Serum lipoproteins, physical exercise, middle aged men.

INTRODUCTION

A consensus exists that physical inactivity and reduced cardiorespiratory endurance contribute to risk of CHD, coronary hearth disease (Pasternak et al., 1990; Pate et al., 1995). Among the multiple proposed mechanisms for the postulated protective effect of regular physical activity against CHD is a favorable effect on blood lipids, particularly an increase in high- density lipoprotein cholesterol (HDL- C) and a reduction in triglyceride (TG) levels (National Institutes of Health Consensus Development Panel on the Health Implications of Obesity (NIHCDP), 1985; Prong, 1993; USDHHS, 1996). Exercise, primarily aerobic exercise, is a low-cost therapeutic lifestyle change that has been recommended for improving lipid and lipoprotein levels in adults (Rosamond et al., 2008). While previous meta-analytic research has reported significant improvements in lipids and lipoproteins among both men (Kelley and Kelley, 2006) and women (Kelley et al., 2004) as a result of aerobic exercise on lipids and lipoproteins in adults have been underwhelming. For example, previous randomized controlled trials addressing the effects of exercise on lipid and lipoprotein outcomes have reported conflicting findings with regards to HDL-C and TG (Campbell, 1965; Johnson et al., 1983; Boyden et al., 1993; Hersey et al., 1994; Crouse et al., 1997; Prabhakaran et al., 1999; Leon et al., 2001; Tall, 2002; Kodama et al., 2007; Boardley et al., 2007; Kelley and Kelley, 2009). Several of these studies have investigated the effects of combined exercise and diet on lipids and lipoproteins but few have looked at the effects of physical exercise alone (Tall, 2002; Crouse et al., 1997). Given the conflicting findings regarding the effects of exercise on lipids and lipoproteins in adults, in this controlled study, the author aimed to determine the effect of mild-to-moderate physical exercise on HDL and other lipoproteins in middle-aged men.

METHODS

Subjects

The subjects were 100 men aged between 40 to 45 years. All participants were volunteers and had been physically rather inactive during the year preceding the study. Persons who were taking any kind of medications and persons with cardiac or other medical disorders that would contraindicate physical training were excluded. All participants gave informed consent before the study. But ten participants gave up the exercise programme so the total participants were 90.

Design

A questionnaire about physical activity behaviour, smoking and

alcohol consumption level (1-month recall) was administered to the participants in the beginning of the study. The participants came to the laboratory for the baseline determinations (Test 1) of the intervention trial. Blood sample was drawn for serum lipids and lipoproteins in the morning after an overnight fast. A progressive submaximal exercise test was performed in the afternoon on an electrically braked Siemens-Elema bicycle ergometer to familiarize the subjects with the testing procedure and to determine the work intensity for the subsequent tests. The work load was increased stepwise in four consecutive 3 min periods to attain 85% of the age-specific maximal pulse level. Subjective load was evaluated according to Borg (1962). During the test a bipolar CM5 (V5 and manubrium) ECG was continuously monitored and recorded at 3 min intervals. Blood pressure was measured at the end of each 3 min period.

Dietary instructions that recommended reduced usage of saturated fats and simple carbohydrates and avoidance of excessive alcohol consumption were given all subjects. Weight loss was not encouraged. These recommendations were made to make the diet of the test persons as uniform as possible and to avoid nonspecific diet-induced changes later during the tests. After these determinations 100 participants (but ten of them gave up the programme later) selected for the intervention trial were randomly assigned to two groups: Exercise (group E) and control (group C). The biochemical studies and the exercise testing were repeated on all subjects 2 months after the beginning of the intervention (Test 2). The subjects were advised to avoid physical exercise on the day before the tests. Body weight was recorded during all visits.

Intervention (training) program

The subjects assigned to the exercise group were given an individualized training program that consisted of walking, jogging, swimming, or cycling. During 8 weeks the program included three weekly training sessions at an intensity that was adjusted to get the previously nonactive participants accustomed to physical exercise. The subjects were instructed to determine their training heart rates from 10 s pulse counts estimated several times during the exercise. The prescribed intensity during the training period was calculated from the modified Balke's formula (Balke, 1974): resting heart rate + 0.40 X (maximal heart rate - resting heart rate). The participants were advised to have a warm-up period of 15 min before and a 10 min slow-down period after the 30 min exercise period to avoid the risks of too-vigorous changes of physical activity.

Maximal oxygen uptake

Maximal oxygen uptake (VO_2) (1/min) was calculated using the indirect method. Work load/heart rate pairs recorded during the graded exercise were extrapolated to the predicted age-specific maximal heart rate and the corresponding work load and oxygen uptake were estimated according to Lange-Andersen et al. (1971).

Cholesterol and triglyceride concentration

Cholesterol and triglyceride concentration in serum and in various lipoprotein fractions was measured by an Autoanalyzer II apparatus (Technicon Instruments, Tarrytown, New York) using enzymatic assay (Boehringer Mannheim GmBH, Germany) . The Autoanalyzer results were calculated by taking into account the carryover and baseline corrections in all determinations. Serum lipoprotein fractionation was carried out using the procedure recommended in LRCMLO (1974). Heparin-manganese was replaced with dextran-sulphate-magnesium chloride (1.0 ml serum + 50 µl of 2% dextran-sulphate (m.w. 500,000) + 50 µl of 2.0 mol/1 $MgCl_2$) in the precipitation of very low density lipoproteins (VLDL) and low-density lipoproteins (LDL), as this method gave more reproducible results combined with the enzymatic cholesterol determination (Kostner,1976).

The concentration of apolipoproteins Al and All

The concentration of apolipoproteins Al and All was measured with a radial immunodiffusion procedure similar to the method described by Cheung and Albers (1977). Fifty microliters of plasma were mixed with an equal volume of tetramethylurea. After adding 650 µl of a solution containing 0.01 M Tris chloride, pH 8.0, and 6 M urea, the mixture was incubated overnight at room temperature. Fourmicroliter samples were pipetted in duplicate on agarose plates containing antiserum (4% anti-Al and 8% anti-All) and 0.02 M Tris chloride, pH 8.0, 0.15 M NaCl, 1 mM EDTA and 1% bovine serum albumin. After 48 h diffusion the immunoprecipitates were measured with a calibrating viewer. Standard preparations of Al and All were included in all plates.

Statistical methods

The results were mean ± SEM. Differences in the mean values were tested with the t test and a paired t test and moreover, the correlations between parameters were analyzed.

RESULTS

There were significant correlations between maximal oxygen uptake calculated per kg body weight (ml/kg * min) and total serum triglyceride ($r = -0.28$, $n = 90$) and VLDL cholesterol ($r = -0.26$, $n = 100$) concentrations in the baseline studies before the beginning of the intervention. Serum triglyceride levels were significantly lower in nonsmokers than smokers (1.30 ± 0.06 vs 1.89 ± 0.21 mmol/l, $p < 0.05$), while the levels of the other serum lipids were not related to the smoking status ($p>0.05$). A weak positive correlation ($r = 0.26$, $n = 100$) was present between HDL cholesterol level and alcohol consumption measured by 1-month recall technique ($p<0.05$). There were no significant correlations between alcohol consumption and total serum cholesterol, triglyceride or LDL cholesterol concentrations in the all participants before intervention ($p>0.05$) (Table 1).

The triglyceride level was significantly lower in group E than in group C at the end of the training (1.27 ± 0.08 mmol/l vs 1.58 ± 0.13 mmol/l, $p < 0.05$), whereas HDL cholesterol level in group E (1.41 ± 0.04 mmol/l) was significantly higher than group C (1.26 ± 0.03 mmol/l) (Table 2). As no significant change occurred in the concentration of apolipoprotein Al in either group, the HDL-C/apolipoprotein Al ratio increased in the exercise group but not in the control group. On the other hand the level of apolipoprotein All decreased in both groups (Table 2). The correlations between the changes in serum triglycerides, HDL cholesterol, body weight and VO2 (ml/kg - min) in group E after the training period are shown in Table 3. A significant negative correlation was observed between the changes in HDL cholesterol and

Table 1. Baseline (Test 1) results of groups E and C.

	Group E	Group C
Body weight (kg)	78.4 ± 1.5	79.9 ± 1.3
V02 (ml/kg * min)	43.0 ± 1.2	43.9 ± 0.9
Cholesterol (mmol/1)	6.8± 0.2	6.8± 0.2
Triglycerides (mmol/1)	1.54± 0.10	1.43± 0.14
VLDL cholesterol (mmol/1)	0.61± 0.06	0.54± 0.07
LDL cholesterol (mmol/1)	4.8± 0.2	4.8± 0.1
HDL cholesterol (mmol/1)	1.27± 0.04	1.24± 0.04
HDL cholesterol/apolipoprotein Al (mmol/mg X 104)	7.9± 1.0	8.1± 1.1
Smoking status	13/44	13/46
Alcohol consumption (ml/month)*	303 ± 45	343 ± 63

Values are mean ± SEM; *Alcohol consumption was estimated with 1-month recall technique and is expressed as ml of absolute alcohol used per month.

Table 2. Group comparisons of test 1 and test 2 (n=90).

Groups	Tests	HDL cholesterol (mmol/l)	Trygliceride (mmol/l)	Apolipoprotein Al (mg/dl)	Apolipoprotein AII (mg/dl)	HDL cholesterol/apolipoprotein Al (mmol/mg X 104)
E	I	1.27 ± 0.04	1.54±0.10	159±3	40.0 ± 1.0	7.9 ± 1.0
	II	1.41 ± 0.04	1.27± 0.08	161±2	35.8 ± -0.81	8.4 ± 0.4
C	I	1.24 ± 0.04	1.43±0.14	155±3	37.8 ± 1.0	8.1 ± 1.1
	II	1.26 ± 0.03	1.58± 0.13	158±4	35.9 ± 1.0	8.0 ± 1.0

Values are mean ± SEM; There were significant differences in HDL cholesterol and trygliceride levels between groups E and C ($p<0.05$).

Table 3. Correlations between the changes in serum triglycerides, HDL cholesterol, body weight and $V0_2$ in the exercise group (n = 44).

Parameters	Correlation coefficient (r)
HDL cholesterol-body weight	+0.33*
HDL cholesterol-V02	-037*
HDL cholesterol-triglycerides	-0.37*
Triglycerides-V02	+0.23
Triglycerides-body weight	-0.08

*p <0.05.

fasting serum triglycerides (r = - 0.37). A negative correlation (r = -0.37) was present between the HDL cholesterol and VO_2. Furthermore, a positive correlation (r = 0.33) was present between the changes in HDL cholesterol and body weight. On the other hand there were not correlations between triglycerides-V02 (r = +0.23) and triglycerides-body weight (r = -0.08).

DISCUSSION

A decrease in serum triglycerides after exercise has been described in the study of Larson-Meyer et al. (2008). In another one, Slentz et al. (2007) did a research covered sedentary, overweight subjects (n = 240) were randomized to 6-mo control or one of three exercise groups: 1) high-amount/vigorous-intensity exercise; 2) low-amount/vigorous-intensity exercise; or 3) low-amount/moderate-intensity exercise. Moderate-intensity but not vigorous-intensity exercise resulted in a sustained reduction in triglyceride over 15 days of detraining. Yet, exercise-induced reduction in serum triglyceride concentration is not clear. The decrease cannot be attributed solely to weight reduction. It was earlier shown that serum triglyceride concentration decreases during an exercise program, even though the participants increase

their caloric intake to compensate for additional caloric expenditure (Lapman et al., 1977; Gyntelberg et al., 1977).

The exercise group had an increase in HDL cholesterol after training programme. Several previous investigations have reported high serum HDL cholesterol levels in subjects who practice strenuous physical exercise (Lehtonen and Viikari, 1978; Bunout et al., 2001; Castaneda et al, 2002; Durak et al., 1990; Elliott et al., 2002; Fahlman et al., 2002; Fenkci et al., 2006). In Slentz et al's study (2007), continued inactivity resulted in significant increases in low-density lipoprotein (LDL) particle number, small dense LDL, and LDL-cholesterol. A modest amount of exercise training prevented this deterioration. The high-amount group had significant improvements in high-density lipoprotein (HDL)-cholesterol, HDL particle size, and large HDL levels that were sustained for 15 days after exercise stopped. They concluded that physical inactivity has profound negative effects on lipoprotein metabolism. Modest exercise prevented this. Thirty minutes per day of vigorous exercise, like jogging, has sustained beneficial effects on HDL metabolism. Kodama et al (2007), did a meta-analitical study searching Electronic database of MEDLINE (1966-2005) for randomized controlled trials that examined the effect of exercise training on HDL-C level. Twenty-five articles were included. Mean net change in HDL-C level was statistically significant. Minimal weekly exercise volume for increasing HDL-C level was estimated to be 120 min. Univariate regression analysis indicated that every 10-minute prolongation of exercise per session was associated with an approximately 1.4-mg/dL (0.036-mmol/L) increase in HDL-C level. In contrast, there was no significant association between exercise frequency or intensity. The general conclusion was that regular aerobic exercise increases HDL-C level (Hagerman et al., 2000; Hong, 2004; Katznelson et al., 2006; LeMura et al., 2000; Maesta et al., 2007).

Although HDL cholesterol increased in the exercise group, no change occurred in the serum concentration of apolipoprotein Al, the major protein component of HDL. The concentration of apolipoprotein All, another peptide of HDL, decreased slightly in both groups. The cause of the divergent changes in the various components of HDL is unknown. It has earlier been reported that the ratio of HDL cholesterol/ apolipoprotein Al varies in different disease states (Rapoport, 1978) and in fact, there is some evidence that the level of HDL cholesterol is a better predictor of coronary heart disease than the concentration of apolipoprotein Al (Ishikawa, 1978). The results of this study clearly demonstrate that weight reduction is the cause of the increase in HDL cholesterol induced by physical activity. Thus, a positive correlation was observed between the changes in the body weight and HDL cholesterol concentration. The variable most strongly associated with the increase in HDL cholesterol in the exercise group was the decrease in serum triglycerides. A correlation has earlier been demonstrated in crosssectional studies between serum triglyceride and HDL cholesterol levels (Castelli et al., 1977; Olson et al., 2006; Sallinen et al., 2007; Sigal et al., 2007; Thomas et al., 2005; Vincent et al., 2003; Wosornu et al., 1996).

Contrary to these results Williams et al (1983) reported that among runners, one- year changes in plasma HDL-C concentrations correlated strongly with their body weight changes ($r = -0.53$, $P < 0.001$). Curve-fitting procedures and regression analysis suggested that processes associated with weight change produce much of the plasma HDL-C changes induced by moderate exercise and that changes in HDL-C concentration predominantly reflect changes in the reputedly anti-atherogenic HDL2 sub-component. Further, the interaction between weight change and plasma HDL-C concentration was significantly different ($p<0.001$) in exercisers and controls suggesting that the metabolic consequences of exercise-induced weight change are different from the consequences of weight change in the sedentary state. As a conclusion, there are some uncertainity about the correlations between the parameters of this study. However, based on the results of this study, one can claim that physical exercise has effects on serum lipoproteins and middle aged people could practice regular moderate (aerobic) exercise. Further studies comprising different methodology are needed to clarify this issue.

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