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## Relationship Between Physical Conditioning and Plasma High Density Lipoprotein-Cholesterol Concentration

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# THE RELATIONSHIP BETWEEN PHYSICAL CONDITIONING AND PLASMA HIGH DENSITY LIPOPROTEIN-CHOLESTEROL CONCENTRATION

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## ABSTRACT

Five subjects (three females and two males) took part in an exercise regimen in order to determine if aerobic exercise results in an increase in high-density lipoprotein-cholesterol levels (HDL-C) in the plasma.

The exercise regimen consisted of running three miles a day, five days per week for six months. Running speed was at such a pace that the subjects attained a minimum of 60% of their maximal heart rate reserve (MHRR). Before the training program began the following parameters were measured in all of the subjects: height, weight, percent body fat, maximal oxygen consumption ( $V_{O_2}$  max), vital capacity, resting heart rate, resting blood pressure, HDL-C, plasma triglycerides (TG), and plasma cholesterol (TC). These same measurements were retaken every two months and at the conclusion of the study.

The exercise protocol produced significant changes in  $V_{O_2}$  max and resting heart rate. None of the other parameters were significantly changed.

The results of this study have shown that aerobic exercise does not cause significant changes in HDL-C levels.

## INTRODUCTION

HDL-C has been shown to be inversely related to the incidence of cardiovascular disease (Castelli et al., 1977; Gordon et al., 1977; Miller and Miller, 1975; Rhoads et al., 1976). It has also been found that an increase in the HDL-C/TC ratio and a reduction in TG level (which may occur with an increase in HDL-C concentration) may be important indicators of reduced coronary artery disease (Carlson and Ericsson, 1975; Hartung et al., 1981; Huttunen et al., 1979). Physically well-trained individuals have higher levels of HDL-C than sedentary people (Enger et al., 1977; Lehtonen and Viikari, 1978; Lopez-S. et al., 1974; Wood et al., 1976). However, it is not known with certainty that high levels of HDL-C are due to physical activity *per se*. Other parameters, such as genetic factors, may be involved. The effects of aerobic exercise regimens on HDL-C concentrations have shown conflicting results, with some of the studies showing an increase in HDL-C concentrations (Altekruse and Wilmore, 1973; Huttunen et al., 1979) while others have recorded no change (Lipson et al., 1979; Squires et al., 1979; Weltman et al., 1978). Most of the studies completed thus far have been concerned with the effects of rather short term exercise programs on HDL-C concentrations (Enger et al., 1980; Hartung et al., 1981; Lipson et al., 1980).

The primary objective of the present study was to determine the effects of aerobic exercise on plasma lipids and lipoproteins. We were particularly interested in noting the effects of aerobic exercise on plasma HDL-C levels. In addition, we wished to see if an aerobic exercise program resulted in changes in plasma cholesterol and triglyceride levels.

## METHODS AND MATERIALS

Five sedentary volunteers were studied, three females and two males, ages 21-44. None of the subjects smoked and all were of normal body weight according to the Metropolitan Life weight tables. Before the training program began, the following parameters were measured in all of the subjects: height, weight, percent body fat,  $V_{O_2}$  max, vital capacity, resting heart rate, resting blood pressure, plasma HDL-C,

plasma TG, and total plasma TC. The same parameters were also measured at 60, 120 and 180 day intervals during the exercise program. Percent body fat was determined with a skinfold caliper. In the males, skinfold measurements were taken over the chest, abdomen, and thigh while in the female the measurements were recorded over the triceps, thigh, and suprailium (Bau, et al., 1981).

Maximal oxygen consumption was estimated by using the Balke treadmill test (Ellestad, 1980). Our laboratory has no instrumentation for measuring oxygen consumption directly. However, the Balke protocol corrects for this situation since it has determined (by directly measuring oxygen consumption in a large sample population) the oxygen consumption required during each stage of the test. The test was terminated when the heart rate reached 170. Heart rates during the test were recorded with three chest leads hooked to a cardiac preamplifier. The preamplifier in turn was connected to a Physiograph for visual display (Narco Biosystems, Houston, TX).

Vital capacity was determined with a six liter spirometer. The subject first inspired maximally and the amount of gas collected following a maximal exhalation was recorded as the vital capacity. Resting heart rate was measured by direct palpation of the radial artery and resting blood pressure was recorded with a sphygmomanometer.

Blood for lipid determination was drawn from the antecubital vein after a 12-14 h fast. Samples, drawn on two days (within a week of each other), were averaged to provide baseline values. High density lipoproteins were determined by using a heparin-manganese precipitation procedure (Warnick and Alberts, 1978). Total cholesterol concentrations were measured by using the orrho-phthaldehyde technique (Rudel and Morris, 1973). Serum triglycerides were analyzed by the method described by Sardesai and Manning (1968).

The training protocol consisted of having the subjects run for three miles per day, five times per week for six months. Since the subjects had previously led a sedentary life style it took them one to two months to build their stamina to the point that they could complete their daily exercise regimen without stopping. The subjects were encouraged to run at a speed that would exert a minimum of 60% of their MHRR. This was calculated in the following manner:  $60\% \text{ MHRR} = \text{MHR} - \text{RHR} \times 0.6 + \text{RHR}$  where MHRR equals maximal heart rate

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reserve, MHR equals maximum heart rate and RHR equals resting heart rate (Karvonen, et al., 1957). Maximal heart rate was calculated by subtracting the subject's age from 220.

Statistical significance ( $P < 0.05$ ) between means was determined with a t-test. Results are expressed as mean  $\pm$  standard deviation.

RESULTS

Physical characteristics for the subjects are shown in Table 1. The exercise program did not result in a significant change in weight, percent body fat, vital capacity or resting blood pressure in any of the subjects. A significant change in resting heart rate however, was recorded.

Table 2 shows the changes that occurred in the  $V_{O_2}$  max during the study. The post-training  $V_{O_2}$  max of 56.8 ml/kg/min was significantly greater than the pre-training mean of 50.0. One subject (#5) had to discontinue running after four months because of a cold weather induced asthmatic condition. Therefore a four month  $V_{O_2}$  max figure was used as the final reading in this case.

The changes that occurred in TC, HDL-C, and TG during the study are reflected in Table 3. TC levels fell from  $175.6 \pm 9.2$  to  $167.8 \pm 23.8$  mg/dl. HDL-C levels declined from  $56.8 \pm 8.9$  to  $51.6 \pm 9.8$  mg/dl. TG decreased from  $50.6 \pm 30.9$  to  $42.6 \pm 9.8$  mg/dl. However, none of these changes were significant. One subject (#3) had started on a protein sparing diet a short time before the conclusion of the project. The diet probably caused the massive changes seen in his blood chemistry profile.

Table 1. Physical characteristics for individual subjects at the beginning (B) and end (E) of the experiment.

Subject	1		2		3		4		5		x SD	t
	B	E	B	E	B	E	B	E	B	E		
Sex	Female	Female	Female	Male	Female	Male	Female	Male	Female	Male		
Age (yrs)	21	21	29	29	34	34	30	37	43	44	32.8 $\pm$ 7.3	23.0 $\pm$ 7.7
Height (cm)	163	163	170	170	183	183	173	173	188	188		
Weight (kg)	54.6	52.7	55.8	54.1	103.3	105.1	68.1	67.3	90.0	91.8	74.8 $\pm$ 13.4	74.2 $\pm$ 20.9
Percent body fat	21.7	21.8	23.2	23.0	25.7	26.0	23.8	25.8	18.2	17.8	22.4 $\pm$ 2.0	22.8 $\pm$ 3.0
$V_{O_2}$ max (ml/kg/min)	46.5	58.0	50.8	58.0	52.5	58.1	52.8	51.1	47.8	55.5	50.0 $\pm$ 12.5	56.8 $\pm$ 11.0
Vital capacity (liters)	2.4	2.8	3.3	3.4	4.3	4.4	3.7	3.7	4.7	4.6	3.9 $\pm$ 0.5	3.8 $\pm$ 0.4
Resting heart rate (beats/min)	70	64	75	67	64	66	72	68	68	60	69 $\pm$ 4.4	59 $\pm$ 2.8
Blood pressure-resting (mmHg)												
a) systolic	120	100	114	110	110	112	120	118	119	120	113.6 $\pm$ 9.4	112.0 $\pm$ 9.0
b) diastolic	82	60	68	65	78	70	80	80	72	82	71.8 $\pm$ 9.9	71.8 $\pm$ 9.7

\* Mean  $\pm$  standard deviation

Table 2. Changes in  $V_{O_2}$  max (ml/kg/min) during the study.

Subject	initial	2 months	4 months	6 months	percent increase
1	46.5	45.0	53.5	58.0	25.0
2	50.5	60.8	60.9	58.0	14.9
3	52.5	56.0	54.0	59.5	13.2
4	52.8	60.0	60.0	61.3	16.1
5	47.8	56.0	55.0	47.0	16.0
x $\pm$ SD	50.0 $\pm$ 2.8	55.6 $\pm$ 5.9	56.8 $\pm$ 4.8	56.8 $\pm$ 12.1	17.1 $\pm$ 4.0

\* took the four months reading to calculate % increase

\*\* Mean  $\pm$  standard deviation

DISCUSSION

A negative correlation between coronary artery disease (CAD) and plasma HDL-C levels has been well documented (Castelli et al., 1977; Gofman et al., 1966; Gordon et al., 1977). In addition, Rhoads et al. (1976) demonstrated that the negative correlation between CAD and HDL-C was independent of other chemical or risk factors. In the 1950's and 1960's several investigators also discovered a negative correlation between high levels of physical activity and CAD (Breslow and Buell, 1960; McDonough et al., 1965; Morris et al., 1953; Morris and Crawford, 1958; Taylor et al., 1962).

Recently several studies have focused on the possible relationship between exercise and HDL-C levels. Cross sectional studies, in which different populations are compared, have revealed that middle-aged runners and cross country skiers have significantly higher levels of HDL-C than do their more sedentary counterparts (Enger et al., 1977; Hartung et al., 1978; Wood et al., 1976). Cross sectional studies, however, do not reveal whether the high levels of HDL-C observed in physically active people are the result of exercise or some other mediator, perhaps genetic in nature (Hartung et al., 1980). Several studies have indicated that increased levels of physical activity cause increases in HDL-C levels (Altekruse and Wilmore, 1973; Enger et al., 1980; Lopez-S. et al., 1974). In contrast to these findings, the present study revealed no correlation between HDL-C levels and physical activity. Even after an increase in aerobic fitness was clearly established, there was no corresponding increase in HDL-C levels in any of our five subjects.

There are two possible explanations for the discrepancies between the present study and earlier studies in regard to the correlation between physical activity and HDL-C levels. First, the earlier studies were not conducted for as long a period of time as our study. It has been shown that increases in  $V_{O_2}$  max do not start to appear until after 15 to 20 weeks of exercise (Pollock et al., 1969; and Pollock et al., 1969). None of the earlier studies exceeded three months in duration while our study was conducted over a six month time period. Secondly, in the earlier studies there was always a weight loss associated with the increased physical activity. Many investigations have shown that HDL-C is inversely related to body weight and this may explain the increased HDL-C levels recorded in the earlier studies (Avogaro et al., 1978; Carlson and Ericsson, 1975; Gordon et al., 1977; Gulley et al., 1979; Rhoads et al., 1976). There was no significant weight loss in our study or in a study conducted by Lipson et al. (1980) in which the diet was rigidly controlled. Finally, Lipson et al. (1980) also did not find an increase in HDL-C levels as a result of exercise.

There were no significant changes in TC or TG levels in this study. Other investigations have shown that along with an increase in HDL-C concentrations, there is also an increase in the HDL-C/TC ratio and a decrease in the TG levels (Carlson and Ericsson, 1975; Hartung et al., 1981; Huttunen et al., 1979). However, those types of findings could not be confirmed in our study since there were no significant changes in HDL-C, TC, or TG levels.

In summary, the exercise regimen utilized in the present study resulted in an increased level of aerobic fitness after 180 days as revealed by the significantly increased  $V_{O_2}$  max in all five subjects. The TG and TC

Table 3. Changes in TC, HDL-C and TG that occurred during the experiment. B designates beginning and E designates the end of the study.

SUBJECT	1		2		3		4		5		x SD	t
	B	E	B	E	B	E	B	E	B	E		
SEX	FEMALE	FEMALE	FEMALE	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE	MALE		
TC (mg/dl)	177	158	168	157	193	181	171	195	169	196	171.9 $\pm$ 19.2	167.8 $\pm$ 23.8
HDL-C (mg/dl)	66	60	52	48	42	54	65	58	38	60	56.8 $\pm$ 8.9	51.6 $\pm$ 9.8
TG (mg/dl)	35	38	23	33	111	77	52	54	48	40	50.6 $\pm$ 30.8	42.6 $\pm$ 9.8

\* Mean  $\pm$  standard deviation

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levels of the plasma, however, remained unchanged throughout the study. Likewise, we could not establish a positive relationship between aerobic fitness and HDL-C levels.

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