Changes in Plasma Lipid and Lipoprotein Levels in Men and Women After a Program of Moderate Exercise

KELLY D. BROWNELL, PH.D., PAUL S. BACHORIK, PH.D., AND ROBERT S. AYERLE, M.D.

SUMMARY Levels of high-density lipoprotein (HDL) cholesterol and other lipids and lipoproteins of 24 men and 37 women were measured before and after a 10-week exercise program. The program involved three sessions of aerobic exercise each week, with 15–20 minutes of activity at 70% of maximal heart rate. Men and women had significantly different lipid patterns in response to exercise, despite equivalent increases in maximal oxygen uptake. Men showed a 5.1% increase in HDL cholesterol, a 6% decrease in low-density lipoprotein (LDL) cholesterol, and a 12.4% increase in the HDL/LDL ratio. In contrast, women showed a 1% decrease in HDL cholesterol, a 4.3% decrease in LDL cholesterol, and no significant change in the HDL/LDL ratio. The number of sessions attended correlated positively with HDL/LDL changes in men and correlated negatively with HDL/LDL changes in women. These findings suggest that moderate exercise may have different effects on men and women.

REGULAR PHYSICAL ACTIVITY is associated with decreased incidence of coronary heart disease (CHD), and persons with CHD may benefit from physical training. Exercise may reduce coronary risk by altering plasma lipid and lipoprotein levels. Two of the major lipoproteins that transport cholesterol in plasma are low-density lipoprotein (LDL) cholesterol and high-density lipoprotein (HDL) cholesterol. High levels of LDL cholesterol are related to increased risk of CHD and high levels of HDL cholesterol may protect against CHD. There has been increased interest in HDL cholesterol because its negative relationship with CHD is stronger than the positive relationship between CHD and either total cholesterol or LDL cholesterol.

Highly trained athletes have lower levels of cholesterol and LDL cholesterol, and higher levels of HDL cholesterol than do sedentary persons of the same age and sex; this is true of runners, skiers, and swimmers. Hartung et al. found that marathon runners have higher HDL cholesterol levels than joggers, who in turn have higher levels than sedentary persons. Findings like these underscore the need for prospective studies to determine whether increases in physical activity produce beneficial changes in plasma lipid and lipoprotein levels.

Prospective studies on exercise and lipid changes have yielded conflicting results. Studies of men have shown consistent increases in HDL cholesterol during

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Supported in part by grant HL-22345 from the NHLBI and by funds from the Bell Telephone Company of Pennsylvania. Dr. Brownell is the recipient of a Research Scientist Development Award MH00319 from the National Institute of Mental Health.

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Received February 19, 1981; revision accepted July 1, 1981.

TABLE 1. Changes in Lipid and Lipoprotein Levels and Body Weight for Men (n = 24)

<table>
<thead>
<tr>
<th></th>
<th>Cholesterol (mg/dl)</th>
<th>HDL cholesterol (mg/dl)</th>
<th>LDL cholesterol (mg/dl)</th>
<th>HDL/LDL</th>
<th>Triglycerides (mg/dl)</th>
<th>Body weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean initial values</td>
<td>207.4 ± 8.9</td>
<td>42.2 ± 1.9</td>
<td>132.8 ± 7.9</td>
<td>0.351 ± 0.23</td>
<td>176.4 ± 22.5</td>
<td>85.3 ± 2.3</td>
</tr>
<tr>
<td>Mean change</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>at 10 weeks</td>
<td>-9.9 ± 4.5</td>
<td>1.5 ± 0.9</td>
<td>-8.5 ± 4.3</td>
<td>0.04 ± 0.01</td>
<td>-31.5 ± 12.0</td>
<td>-1.1 ± 0.3</td>
</tr>
<tr>
<td>p</td>
<td>&lt; 0.04</td>
<td>NS</td>
<td>&lt; 0.057</td>
<td>&lt; 0.0015</td>
<td>&lt; 0.02</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Mean percent change</td>
<td>-4.4 ± 2.1</td>
<td>5.12 ± 2.3</td>
<td>-5.97 ± 2.9</td>
<td>12.4 ± 2.9</td>
<td>-9.5 ± 4.8</td>
<td>-1.3 ± 0.4</td>
</tr>
<tr>
<td>at 10 weeks</td>
<td>&lt; 0.04</td>
<td>&lt; 0.04</td>
<td>&lt; 0.05</td>
<td>&lt; 0.0003</td>
<td>&lt; 0.06</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Values are mean ± SEM.
Abbreviations: HDL = high-density lipoprotein; LDL = low-density lipoprotein.

programs of moderate or intensive exercise and have shown either a decrease or no significant change in LDL cholesterol. Even less is known about lipid variations in women. Ballantyne et al. found no change in HDL cholesterol and a decrease in LDL cholesterol in women after moderate exercise. Lewis et al. found no significant change in either measure.

The present study was designed to evaluate lipid and lipoprotein variations in men and women during a program of moderate exercise.

Methods

Subjects

We studied 61 employees of the Bell Telephone Company of Pennsylvania who volunteered for an exercise program. There were 37 women, with a mean age of 35 years (range 20–57 years) and a mean weight of 62.1 kg (range 43–96 kg); the mean percent overweight was 12.2% (range 12% to 69%), calculated for each subject's sex, height and frame size using the Metropolitan Life actuarial tables. There were 24 men, with a mean age of 41.8 years (range 28–60 years), a mean weight of 85.3 kg (range 68–112 kg), and a mean percent overweight of 20% (range 5–47%). To qualify, subjects had to be free of medical conditions that would contraindicate rigorous exercise, not be taking medications that would influence plasma lipid levels or body weight, and be able to score in at least the thirtieth percentile for age and sex on a bicycle ergometer submaximal stress test. None of the women were taking estrogens.

The pretreatment lipid analyses showed that 52 of the 61 subjects were in the normal ranges for cholesterol, triglycerides, HDL cholesterol and LDL cholesterol. Normal was defined as below the ninetieth percentile for age and sex using the data from the Lipid Research Clinics Program Prevalence Study. Eight subjects (five males and three females) had type IV hyperlipoproteinemia (triglycerides above the ninetieth percentile and one man had type II hyperlipoproteinemia (LDL cholesterol above ninetieth percentile). The subjects with elevated lipids were too few in number to allow a separate analysis, so the analyses presented hereafter will include all subjects. For the total sample, the mean initial lipid values were very close to the fiftieth percentiles reported in the LRC study (tables 1 and 2). The ranges of lipids and lipoproteins for men and women respectively were 141–296 and 128–268 mg/dl for cholesterol, 27–61 and 36–93 mg/dl for HDL cholesterol, 61–218 and 63–205 mg/dl for LDL cholesterol, and 69–471 and 23–170 mg/dl for triglycerides.

Exercise Program

All subjects participated in a 10-week exercise program held during the lunch hour at the Philadelphia Central YMCA (one block from the Bell Telephone building). Thirty-minute sessions were held three times weekly for groups of 15–25 subjects. Subjects attended an average of 67% of the scheduled sessions. The groups, led by certified fitness instructors from the YMCA, focused on cardiopulmonary conditioning, flexibility, and muscular strength and endurance, and were conducted according to the YMCA protocol for an aerobic exercise program. The pro-

TABLE 2. Changes in Lipid and Lipoprotein Levels and Body Weight for Women (n = 37)

<table>
<thead>
<tr>
<th></th>
<th>Cholesterol (mg/dl)</th>
<th>HDL cholesterol (mg/dl)</th>
<th>LDL cholesterol (mg/dl)</th>
<th>HDL/LDL</th>
<th>Triglycerides (mg/dl)</th>
<th>Body weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean initial values</td>
<td>186.8 ± 5.9</td>
<td>58.6 ± 2.2</td>
<td>111.8 ± 5.6</td>
<td>0.572 ± 0.37</td>
<td>71.5 ± 4.8</td>
<td>62.1 ± 1.4</td>
</tr>
<tr>
<td>Mean change</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>at 10 weeks</td>
<td>-7.8 ± 3.9</td>
<td>-0.92 ± 1.2</td>
<td>-5.3 ± 3.2</td>
<td>0.43 ± 0.26</td>
<td>3.2 ± 4.7</td>
<td>-0.97 ± 0.27</td>
</tr>
<tr>
<td>p</td>
<td>&lt; 0.05</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>&lt; 0.0009</td>
<td></td>
</tr>
<tr>
<td>Mean percent change</td>
<td>-3.9 ± 2.0</td>
<td>-1.0 ± 2.2</td>
<td>-4.32 ± 2.9</td>
<td>8.19 ± 3.79</td>
<td>14.5 ± 7.4</td>
<td>-1.47 ± 0.4</td>
</tr>
<tr>
<td>at 10 weeks</td>
<td>&lt; 0.06</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>&lt; 0.06</td>
<td>&lt; 0.06</td>
</tr>
</tbody>
</table>

Values are mean ± SEM.
Abbreviations: HDL = high-density lipoprotein; LDL = low-density lipoprotein.
gram became progressively more vigorous, and by the fourth week, the subjects were exercising at approximately 70% of maximal heart rate for 15–20 minutes at each session. The remaining time was used for stretching, warm-up and cool-down activities. We estimate a caloric expenditure of 300–350 kcal for the 30-minute session.

Cardiorespiratory endurance was measured by estimation of maximal oxygen uptake (VO2max) with a constant-torque bicycle ergometer. Subjects were tested at systematically increased work loads to estimate VO2max (l/min). This value was then divided by body weight to yield VO2max expressed in ml/kg/min.

Analytical and Statistical Methods

Blood samples were obtained before and after the 10-week program. Each sample was drawn between 7:00 and 9:00 a.m. after a 12-hour fast and at least 48 hours after the last exercise session. The samples were analyzed by the Lipid Research Clinic of Johns Hopkins University. Total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides were determined using the procedures of the Lipid Research Clinics Program. Blood was collected into evacuated tubes containing solid disodium EDTA (1.5 mg/ml). The plasma was separated at 4°C within 3 hours and shipped unfrozen to the laboratory. Heparin sulfate and MnCl2 were added to plasma to precipitate the apo B–containing lipoproteins. The samples were incubated in an ice bath for 30 minutes and the precipitated lipoproteins were removed by centrifugation at 1500 g for 30 minutes at 4°C. Isopropanol extracts of unfractonated plasma and the HDL-containing heparin MnCl2 supernatant fraction were prepared and treated with a zolite mixture to remove interfering substances. The analyses were performed on the AutoAnalyzer II using a modification of Kessler and Lederer’s method for triglycerides and the Liebermann-Burchard reaction for cholesterol. LDL cholesterol was estimated using the procedure of Friedewald et al. The laboratory procedures were standardized according to the criteria for the Lipid Research Clinics Program.

Overall statistical differences between groups were determined with univariate analyses of variance, and paired t tests were used for pairwise comparisons. Two multivariate techniques were used, each based on a multiple linear regression model. Analyses of covariance were used to adjust the values of the dependent variable to eliminate the effects of confounding variables. The dependent variable was residualized, so the effect of the covariate was subtracted and the significance was calculated on the remaining scores. Second, a multiple correlation coefficient was calculated for each dependent variable using a stepwise procedure to select the variables of most importance. The independent variable that contributes most to the dependent variable is chosen on the first step, and each remaining variable's ability to predict the dependent variable is determined in subsequent steps. Pearson correlation coefficients were used to evaluate the degree of association between two variables.

Dietary, smoking and alcohol consumption patterns were measured before and after the program with self-report questionnaires. Subjects completed a 24-hour dietary recall for the same day of the week before and after the program. These were scored for total calories, cholesterol and saturated fat. The questionnaires also contained items on the number of cigarettes smoked in the past day and the past week, and items on the number of alcoholic drinks for the past week.

Results

The primary focus of this study was on lipid and lipoprotein variations between men and women. Data for men and women were analyzed separately and were then combined for an overall analysis.

Sex Differences

The mean and percent changes among the 24 men for cholesterol, HDL cholesterol, LDL cholesterol, the HDL/LDL ratio, triglycerides and body weight are presented in table 1. The men showed decreases of 4.4% in cholesterol (p < 0.04), 5.97% in LDL cholesterol (p < 0.05), 9.5% in triglycerides (p < 0.06) and 1.3% in body weight (p < 0.001). Combined with an increase of 5.1% in HDL cholesterol (p < 0.04), the significant reduction in LDL cholesterol resulted in a 12.4% increase in the HDL/LDL ratio (p < 0.0003).

The lipid and lipoprotein changes among the women were different from those among the men (table 2). The 37 women showed significant decreases of 3.9% in cholesterol (p < 0.06) and 1.5% in body weight (p < 0.06), and nonsignificant decreases of 1.0% in HDL cholesterol and 4.3% in LDL cholesterol. Consequently, there was a nonsignificant increase of 8.2% in the HDL/LDL ratio. There was a surprising 14.5% increase in triglycerides (p < 0.06).

Because there was great variability in lipid and lipoprotein responses for both men and women, analyses of variance were used to compare the differences shown in tables 1 and 2. Men and women differed in their initial values for each lipid measure and for body weight. Initial levels were higher in men than in women for cholesterol (p < 0.05), and LDL cholesterol (p < 0.03), triglycerides (p < 0.0001), and body weight (p < 0.02), but were lower for men in HDL cholesterol (p < 0.03) and the HDL/LDL ratio (p < 0.0001). Men showed significantly greater percent changes in HDL cholesterol (p < 0.08) and triglycerides (p < 0.01), and significantly greater changes in the HDL/LDL ratio (p < 0.02) and triglycerides (p < 0.01). To account for initial differences, analyses of covariance using initial values as the covariate were used to compare men and women. After adjusting for initial values, the only significant difference was a greater increase in the HDL/LDL ratio for men (p < 0.02).

A stepwise multiple regression was used to separate the effects of five factors on changes in lipids; the fac-
tors were initial values, initial body weight, weight changes, the number of sessions attended and age. In women, none of these factors contributed significantly to changes or percent changes in cholesterol or LDL cholesterol. High initial values were associated with the greatest reductions in triglycerides (p < 0.0002). Age made an independent contribution to triglyceride changes (p < 0.03; older persons had the greatest reduction). The number of sessions attended was associated with changes in HDL cholesterol (p < 0.04) and the HDL/LDL ratio (p < 0.04), and with percent changes in HDL cholesterol (p < 0.07). Women who attended the most sessions tended to have the largest decreases in HDL cholesterol (r = −0.34, p < 0.04) and the HDL/LDL ratio (r = −0.35, p < 0.04), and the largest percent decreases in HDL cholesterol (r = −0.41, p < 0.01).

In men, none of the factors contributed to changes in cholesterol. Low initial values were associated with the greatest increases in HDL cholesterol (p < 0.0008); the high initial values were associated with the greatest reductions in triglycerides (p < 0.0001). Higher ages were associated with the greatest reductions in LDL cholesterol (p < 0.03) and the greatest increases in HDL/LDL (p < 0.04). The number of sessions attended was related to several lipid measures. Men who attended the most sessions tended to show the greatest reductions in LDL cholesterol (r = −0.38, p < 0.065), the greatest increases in the HDL/LDL ratio (r = 0.52, p < 0.01) and the greatest percent increases in the HDL/LDL ratio (r = 0.51, p < 0.01).

Men and women showed significant increases in VO₂max during the program (p < 0.05 in both cases). The increases for men (3.2 ml/kg min) did not differ significantly from the increases for women (2.7 ml/kg min). The initial values for VO₂max for men (31.3 ml/kg min) and women (30.5 ml/kg min) did not differ. Men and women did not differ in the number of sessions attended (21.8 vs. 21.2). These data indicate that men and women performed the same amount of exercise and showed equivalent increases in aerobic capacity. Surprisingly, the number of sessions attended was not correlated significantly with changes in VO₂max. Furthermore, the number of sessions attended correlated with several measures of lipid changes, but changes in VO₂max did not correlate with any measure of lipid changes.

There were no significant changes in men or women in their reported dietary patterns. Total calories were higher in men than in women both before and after the program, but the sexes did not differ in the percentage of total calories contributed by cholesterol or saturated fat. Men and women also did not differ in their number of cigarettes or alcoholic drinks either before or after the program. Neither sex showed significant changes in smoking or drinking during the program.

**Analyses for the Total Sample**

The mean and percent changes in lipids and lipoproteins for all subjects are shown in table 3. These data are presented to allow comparisons with previous studies and to show that combining data for men and women may mask important differences. There were significant decreases of 4.1% in cholesterol (p < 0.006) and 1.4% in body weight (p < 0.0001), and a significant increase of 9.8% in HDL/LDL (p < 0.0004). The 1.6% increase in HDL cholesterol and the 5.1% decrease in triglycerides did not reach significance.

**Discussion**

The inverse relationship between regular physical activity and the prevalence of coronary heart disease has been well established. Paffenbarger et al. suggest that this association cannot be explained by inherent physical attributes and that the exercise itself is responsible for the reduced risk. Regular exercise may influence coronary risk by altering plasma lipids. Plasma lipid levels are associated with risk for coronary heart disease, and highly trained athletes tend to have more favorable lipid patterns than do sedentary persons. Prospective studies on sedentary persons who increase their physical activity are necessary to establish the effects of exercise on lipids. Several studies of this nature have been done; some report increases in lipids, others report decreases, and others report no changes. The few studies of women suggest that they may differ from men in lipid changes.

Studies on lipid changes in response to exercise are summarized in table 4. Because the focus of our study was on HDL cholesterol, only studies reporting this measure were included in the table. Among men, physical training has been found to produce either a

**Table 3. Changes in Lipid and Lipoprotein Levels and Body Weight for Men and Women Combined (n = 61)**

<table>
<thead>
<tr>
<th></th>
<th>Cholesterol (mg/dl)</th>
<th>HDL cholesterol (mg/dl)</th>
<th>LDL cholesterol (mg/dl)</th>
<th>HDL/LDL (mg/dl)</th>
<th>Triglycerides (mg/dl)</th>
<th>Body weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean initial values</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mean change at 10 weeks</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-8.6 ± 2.9</td>
<td>0.02 ± 0.83</td>
<td>-6.6 ± 2.6</td>
<td>0.42 ± 0.17</td>
<td>-10.4 ± 5.9</td>
<td>-1.04 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>&lt; 0.005</td>
<td>NS</td>
<td>&lt; 0.01</td>
<td>&lt; 0.01</td>
<td>&lt; 0.08</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td><strong>Mean percent change at 10 weeks</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-4.1 ± 1.5</td>
<td>1.57 ± 1.6</td>
<td>-4.97 ± 2.13</td>
<td>9.77 ± 2.59</td>
<td>-5.1 ± 5.1</td>
<td>-1.4 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>&lt; 0.006</td>
<td>NS</td>
<td>&lt; 0.02</td>
<td>&lt; 0.0004</td>
<td>NS</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

Values are mean ± SEM.

Abbreviations: HDL = high-density lipoprotein; LDL = low-density lipoprotein.
TABLE 4. Changes in Plasma Lipid and Lipoprotein Levels (mg/dl) in Studies Reporting Values for Cholesterol, High-density Lipoprotein Cholesterol, Low-density Lipoprotein Cholesterol and Triglycerides

<table>
<thead>
<tr>
<th>Study</th>
<th>Description</th>
<th>Cholesterol</th>
<th>HDL cholesterol</th>
<th>LDL cholesterol</th>
<th>Triglycerides</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leon et al.25</td>
<td>6 obese young men; 5 days/week for 16 weeks; treadmill at up to 3.2 mph</td>
<td>-6.0</td>
<td>-5.0</td>
<td>-8.0</td>
<td>-21.0</td>
</tr>
<tr>
<td></td>
<td>at 10% grade</td>
<td>NS</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Gyntelberg et al.31</td>
<td>5 subjects with type IV hyperlipoproteinemia; 4 sessions with 30 minutes</td>
<td>-12.0</td>
<td>6.0</td>
<td>0</td>
<td>-129.0</td>
</tr>
<tr>
<td></td>
<td>at 70% max. O2 over 6 days; treadmill</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>Huttunen et al.29†</td>
<td>50 physically inactive asymptomatic men; walking, jogging, cycling,</td>
<td>-25.9</td>
<td>5.4</td>
<td>-22.6</td>
<td>-24.3</td>
</tr>
<tr>
<td></td>
<td>swimming or skiing 3-4 days/week for 16 weeks approx. 70% max. heart rate</td>
<td>&lt;0.05</td>
<td>&lt;0.01</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td></td>
<td>by week 8</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ballantyne et al.36*</td>
<td>20 middle-aged, nonobese, nonsmoking, asymptomatic men; unsupervised</td>
<td>10.1</td>
<td>6.96</td>
<td>3.1</td>
<td>-27.4</td>
</tr>
<tr>
<td></td>
<td>aerobic activity 3 days/week for 6 months</td>
<td>NS</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>&lt;0.002</td>
</tr>
<tr>
<td>Streja and Mymin20</td>
<td>32 men with coronary artery disease; walking/jogging at 70-85% max.</td>
<td>11.0</td>
<td>13.5</td>
<td>10.0</td>
<td>-17.0</td>
</tr>
<tr>
<td></td>
<td>heart rate for 3 days/week for 13 weeks</td>
<td>&lt;0.05</td>
<td>&lt;0.01</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Altekruse and Wilmore26</td>
<td>39 asymptomatic, sedentary men; walking/jogging 3 days/week for 10 weeks</td>
<td>-23.7</td>
<td>18.6§</td>
<td>-13.0§</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>NS</td>
</tr>
<tr>
<td>Lopez et al.27</td>
<td>13 asymptomatic male medical students; 4 days/week for 7 weeks of jogging</td>
<td>-7.0</td>
<td>16.0§</td>
<td>-12.7§</td>
<td>-27.0</td>
</tr>
<tr>
<td></td>
<td>and calisthenics at 70% max. heart rate (7 mets)</td>
<td>&lt;0.03</td>
<td>&lt;0.01</td>
<td>&lt;0.02</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Brownell et al.</td>
<td>24 asymptomatic men; 3 days/week of aerobic activities for 10 weeks; 70%</td>
<td>-9.9</td>
<td>1.5</td>
<td>-8.5</td>
<td>-31.5</td>
</tr>
<tr>
<td>(present study)</td>
<td>max. heart rate for 15-20 min. by week 4</td>
<td>&lt;0.04</td>
<td>NS</td>
<td>&lt;0.005</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ballantyne et al.36*</td>
<td>16 middle-aged, nonobese, nonsmoking, asymptomatic women; unsupervised</td>
<td>-6.19</td>
<td>1.9</td>
<td>16.6</td>
<td>-23.9</td>
</tr>
<tr>
<td></td>
<td>aerobic activities 3 days/week for 6 months</td>
<td>NS</td>
<td>&lt;0.04</td>
<td>&lt;0.01</td>
<td>NS</td>
</tr>
<tr>
<td>Lewis et al.32</td>
<td>22 obese women; 17-week program; 2 days/week of walking/jogging at 80%</td>
<td>-2.3</td>
<td>4.7</td>
<td>-4.9</td>
<td>7.4</td>
</tr>
<tr>
<td></td>
<td>max. heart rate plus 2 days/week of calisthenics</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Brownell et al.</td>
<td>37 asymptomatic women; 3 days/week of aerobic activities for 10 weeks; 70%</td>
<td>-7.8</td>
<td>-0.92</td>
<td>-5.3</td>
<td>3.2</td>
</tr>
<tr>
<td>(present study)</td>
<td>max. heart rate for 15-20 min. by week 4</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Values listed for lipid and lipoprotein changes are converted from mmol/l to mg/dl using 38.7 mg/dl = 1 mmol/l for cholesterol, HDL cholesterol, and LDL cholesterol, and 88.5 mg/dl = 1 mmol/l for triglycerides.
†Values taken from graphs provided in original paper.
§Indirect measurement of total lipoprotein mass. HDL cholesterol calculated from total cholesterol minus cholesterol in beta and prebeta lipoprotein. LDL cholesterol calculated as 46.9% of beta lipoprotein. 
Abbreviations: HDL = high-density lipoprotein; LDL = low-density lipoprotein; max. = maximal.

Results from this study demonstrate that men and women differ markedly in their lipid and lipoprotein responses to an exercise program, and that combining data for men and women can obscure these important differences (table 1-3). Men showed significant reductions of 4.4% in cholesterol, 6% in LDL cholesterol, and 9.5% in triglycerides, and a 5.1% increase in HDL cholesterol. Women showed a 4.1% decrease in cholesterol, no significant changes in HDL cholesterol or...
LDL cholesterol, and a 14.5% increase in triglycerides. The differences between men and women were statistically significant for each of the measures except LDL cholesterol, but the differences were no longer significant after adjusting for initial lipid values with an analysis of covariance. However, the HDL/LDL ratio, an important predictor of coronary heart disease, increased substantially in men (p < 0.0003) but did not change significantly in women. The difference in the ratio between men and women remained significant even after adjusting for initial values.

The differential changes in lipids for men and women could not be attributed to differences in amount of exercise or level of conditioning. Men and women showed equivalent increases in \( \text{VO}_{2} \max \) and did not differ in the number of sessions attended during the program. Interestingly, some lipid and lipoprotein changes occurred in proportion to the number of sessions attended, but not in proportion to increases in maximal aerobic capacity. In men, the number of sessions attended correlated negatively with changes in LDL cholesterol and positively with changes in the HDL/LDL ratio. In women, the number of sessions attended correlated negatively with changes in HDL cholesterol and the HDL/LDL ratio. Changes in \( \text{VO}_{2} \max \) did not correlate significantly with changes in any lipid or lipoprotein measure. It is surprising and puzzling that lipid changes show a stronger relationship to the number of sessions attended than to increases in maximal aerobic capacity. This highlights the importance of discovering the mechanisms by which exercise influences conditioning and lipid changes.

Several factors other than exercise may have influenced the results of this study. Plasma lipid levels can vary with diet, smoking, and alcohol intake, and it is possible that participants in exercise programs will make collateral changes in these areas. We collected questionnaire data on these three factors. The patients reported no significant changes in diet, smoking, or alcohol intake. Furthermore, men and women did not differ in their reports of changes. We recognize, however, the weakness of self-report data, so we look to other studies in which prospective and retrospective measurements have been made of these factors. Streja and Mymin found no changes in caloric intake or in the constitution of the diet in sedentary men during a 13-week program of moderate exercise. Gyntelberg et al. found that diet did not contribute to lipid changes during exercise in five hyperlipidemic men, and two retrospective studies showed that diet did not account for any of the differences in lipids between active and sedentary persons. Smoking did not influence lipids in one prospective test of an exercise program, and a study on weight reduction showed that smoking and alcohol did not contribute to lipid changes. These studies suggest that exercise per se influences lipids, but careful measurement of other health habits is still necessary.

Recent research suggests that certain subclasses of HDL cholesterol, namely HDL\(_2\) and HDL\(_4\), may be important considerations in predicting risk for coronary heart disease and relating specific health practices (e.g., exercise) to changes in lipoproteins. We did not analyze our samples for HDL\(_2\) and HDL\(_4\), and it is possible that changes in HDL cholesterol masked important effects of physical activity on the two subclasses. There is some evidence that men and women differ in the subclasses, particularly in HDL\(_4\), so further research is needed to specify the precise effect of physical activity on lipoproteins in men and women.

This study suggests that a short-term exercise program in a work setting can improve plasma lipid and lipoprotein patterns more in men than in women, although the effect of exercise on the HDL subclasses is not known. During our 10-week exercise program, subjects took part in three lunch-hour sessions each week at a YMCA near their place of employment. The subjects were primarily sedentary office workers who averaged 16% above ideal body weight. The program produced small but significant weight changes in men and women, and a highly significant increase in the HDL/LDL ratio in men. Interestingly, in another study, weight reduction produced more positive lipid and lipoprotein changes in men than women. The long-term maintenance of lipid and lipoprotein changes is important to measure. The degree to which subjects continue to exercise after such a program is unknown, as is the metabolic adaptation to prolonged exercise. Thompson et al., for example, found that lipid changes during weight reduction had disappeared 8 months after the program, even though weight loss remained constant. Because exercise is one of the few hygienic measures available to the individual for the control of coronary risk, further research on the relationship between exercise and lipids is important.

Acknowledgment

The authors thank Elizabeth Venditti, Janet Albaum, and Lois Boyer for the testing. Robert Fiske, Ken Zarsky, and George Burger of the Philadelphia YMCA for coordinating the exercise program, Drs. Peter O. Ketterovich and Peter Wood for comments on the manuscript, Marion Weston for the data analysis, and Robert Walker, Sharon Ensley, and David Widman of the Division of Lipid Research, Johns Hopkins University, for analyzing the blood samples.

References


54. Levy RI, Rifkind BM: The structure, function and metabolism of lipoproteins.
Left Ventricular Function in Trained and Untrained Healthy Subjects

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SUMMARY Left ventricular function was compared in 18 normal sedentary controls (mean age 28 years, range 22–34 years) and nine endurance-trained athletes (mean age 19 years, range 15–25 years) at rest and during supine bicycle exercise. Gated radionuclide angiograms were performed at rest and at each level of graded maximal supine bicycle exercise. Heart rate, blood pressure, left ventricular ejection fraction and the relative changes in left ventricular end-diastolic and end-systolic volumes were assessed. Athletes attained a much greater workload than controls (mean 22.1 kpm/kg body weight vs 13 kpm/kg body weight). Both groups achieved similar increases in heart rate, blood pressure and ejection fractions. In the controls, the mean end-diastolic volume increased to 124% of that at rest (p < 0.02) during exercise and the mean end-systolic volume decreased to 81% of the rest level (p < 0.02). In contrast, the mean end-diastolic volume did not significantly change during exercise in the athletes, and the mean end-systolic volume decreased to 64% of rest (p < 0.05). Thus, although trained and untrained healthy subjects had similar increases in the left ventricular ejection fraction during exercise, different mechanisms were used to achieve these increases. Untrained subjects increased end-diastolic volumes, whereas trained subjects decreased the end-systolic volumes. The ability of athletes to exercise without increasing preload may be an effect of training and might have important implications in reducing myocardial oxygen demand during exercise.

ALTHOUGH the human left ventricular response to exercise has been the focus of many studies in the last several decades, the data on some aspects of this subject conflict. Until computer-assisted gated radionuclide cardiac angiography became available, accurate serial assessment of left ventricular volume at rest and during exercise was not technically possible.

To address the question of normal left ventricular volume and ejection fraction response to exercise, we studied 18 healthy untrained subjects at rest and during supine bicycle exercise. We also studied nine endurance-trained athletes to determine whether exercise training alters the left ventricular response to exercise.

Materials and Methods

Subjects

The study population consisted of 18 normal sedentary control subjects and nine endurance-trained athletes. The control group was made up of 14 males, mean age of 28 years (range 22–34 years). The athletes, seven males and two females, were scullers who had represented Canada in various international competitions and were involved in vigorous daily training. The daily training schedule included at least 4 hours of rowing, cycling and jogging, and each athlete had been in training for at least 3 years before the study. The mean age of the athletes was 19 years (range 15–25 years). No subject had a history of medical problems, and all subjects were normal by physical examination and standard resting ECG.

Exercise Protocol

After patients were placed supine on the exercise table, with feet positioned on the ergometer pedals, the baseline measurements of heart rate, blood pressure and multiple-lead ECG were taken. A gated blood pool radionuclide angiogram was then done as described below. The subjects performed graded supine bicycle exercise to the point of exhaustion, using a Quinton 845 ergometer, with increments of 300 kpm every 3 minutes. Blood pressure, heart rate, multilead ECG and radionuclide angiograms were repeated serially during each level of exercise.

Imaging Technique

Radionuclide angiography was done with a Ohio Nuclear Sigma Series 420 scintillation camera, using
Changes in plasma lipid and lipoprotein levels in men and women after a program of moderate exercise.
K D Brownell, P S Bachorik and R S Ayerle

Circulation. 1982;65:477-484
doi: 10.1161/01.CIR.65.3.477
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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