Effect of Moderate Exercise on Serum Lipids in Young Men with Low High Density Lipoprotein Cholesterol

Itamar Raz, Haya Rosenblit, and Jeremy D. Kark

Fifty-five healthy, sedentary, nonsmoking, and nonobese 24- to 26-year-old men who had low plasma concentrations of high density lipoprotein (HDL) cholesterol were selected for a study of the effect of short-term exercise on plasma lipid and lipoprotein concentrations. The participants were randomized into two groups. Of these, 28 were assigned to a 9-week program of submaximal aerobic exercise three times weekly, and 27 were assigned to a nonexercising control group. Changes in physical fitness were assessed by increments in estimated maximal oxygen consumption; this increased by 15% in the exercise group (p<0.001) but remained unchanged in the control group. During the study, body weights and skinfold thicknesses of both groups remained essentially unchanged after 9 weeks. There was no significant difference between the trial groups in total cholesterol, HDL cholesterol, calculated low density lipoprotein cholesterol, or in the HDL2 and HDL3 subfractions. Triglyceride levels were lower by 19 mg/dl in the exercise group as compared to the control group (p<0.05). We conclude that moderate aerobic exercise of 9 weeks duration is an effective and physiologically desirable means of increasing plasma HDL cholesterol concentration, whereas others have not confirmed this finding.

The aim of our study was to evaluate the efficacy of a 9-week program of moderate exercise to raise HDL cholesterol levels in young, nonobese, nonsmoking, sedentary men who initially had low plasma HDL cholesterol concentrations (≤ 40 mg/dl).

Methods

Study Population

Total plasma cholesterol, triglyceride, and HDL cholesterol concentrations were measured in 8624 boys and girls aged 17 to 18 years. This was done in the framework of the Jerusalem Lipid Research Clinic Prevalence Study (LRC).

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Received May 12, 1987; revision accepted December 4, 1987.

during the years 1976 to 1979 at the time of the teenagers' military induction health examination. Approximately 5000 of these participants had a second examination 3 to 4 years later at the end of their military service (Kark JD, unpublished data). Of the initial study population, 864 young people were students at the Hebrew University of Jerusalem during the years 1985 to 1986; 458 were men. The median plasma HDL cholesterol level of 17- to 18-year-old men in the LRC prevalence study was 43 mg/dl; upon discharge from military service, it was 42 mg/dl.

To detect a 4 mg/dl increase in plasma HDL cholesterol, assuming an intraindividual standard deviation of 5 mg/dl, a β level of 0.10, and a one-tailed α level of 0.05, 27 participants were required for each of the two trial groups. All the 329 students in whom the HDL cholesterol concentration was lower than the median in either of the two examinations (168 men were examined only once) were invited for an interview and a repeat HDL cholesterol determination.

Of these, 210 (64%) responded and underwent an initial interview in which 89 were found to be ineligible for the study due to cigarette smoking (n = 37), overweight (n = 19), regular alcohol consumption (n = 8), a health problem (n = 7), regular exercise more than once a week (n = 6), or refusal to participate (n = 12). Plasma lipid determinations were made for the remaining 121 men. At the second stage of selection, 43 students were rejected because their current HDL cholesterol levels exceeded 40 mg/dl, the cut-off point chosen for the study. Another 23 students were rejected from the study due to: reserve military duty planned during the study (n = 6), a body mass index (kg/m²) of more than 25.9 (n = 4), or refusal to be randomly selected to either an exercise or a control group (n = 13). The remaining 55 underwent baseline measurements of plasma lipids, pulse rate, height, weight, skinfold thickness, lung function, and estimated maximal oxygen consumption.
These determinations used enzymatic methods and a bio-
vention group. The blood samples were drawn with mini-
6 days of blood drawing according to the LJpid Research
plasma cholesterol, triglyceride, and HDL cholesterol were
were requested not to change their exercise habits during
self-administered exercise once a week consisting of jog-
ing, bicycle riding, or swimming. A weekly report of com-
pliance was made by each participant and was monitored by
the exercise group supervisor. Men in the control group
were requested not to change their exercise habits during
the trial period and were informed that they would be of-
ered a supervised exercise program at the end of the study.
Both the exercise and control groups were asked not to change the quality of their diet. The control group
was questioned about any unusual exercise or strenuous
work done during the trial period. The duration of the trial
was 9 weeks. No participant in either group was lost to
follow-up. The trial was authorized by the institutional hu-
man subject review committee. Before the beginning of the
study, each participant gave informed consent.

**Laboratory and Measurement Procedures**

Twelve-hour fasting blood samples were drawn at the
start, in the middle, and at the end of the study. The blood samples drawn at the middle and at the end of the study were
taken 48 hours after the last exercise sessions of the inter-
vention group. The blood samples were drawn with mini-
al veinostasis into vacutainer tubes containing 1.5 mg/ml
disodium EDTA and into empty tubes while the subject
remained in a sitting position. Plasma and serum were
prepared from blood within 2 hours of blood drawing. Plas-
ma lipids were measured at baseline, at the midpoint, and
at the end of the study on refrigerated aliquots within 6
days of blood drawing according to the Lipid Research
Clinic Protocol. Plasma cholesterol and triglyceride were
determined by the Technicon (USA) autoanalyzer II (AA-
II), which uses the Liebermann-Burchard method for cho-
esterol and a fluorometric procedure for triglyceride. HDL
cholesterol was measured directly from the plasma after precipita-
tion of the apo B-containing lipoproteins with heparin and manganese chloride. Determinations of plasma cholesterol, triglyceride, and HDL cholesterol were
repeated in a single assay on serum samples stored at
−30°C for 16 months after the termination of the study.
These determinations used enzymatic methods and a bio-
trol kit (Paris, France).

In 18 participants randomly from each trial group, HDL2 and HDL3 were also measured before and at the end of the study. The number of participants was re-
stricted due to limited laboratory facilities. Plasma HDL2
(d = 1.07 to 1.125 g/ml) and HDL3 (d = 1.125 to 1.21 g/ml)
were separated by preparative and zonal ultracentrifuga-
tion as described by Patsch et al.

The estimated maximal oxygen consumption was meas-
ured by using the bicycle ergometer test described by As-
strand and Astrand and Kaare. Briefly, pulse rate and
blood pressure were measured at rest. Each participant
was exposed to an initial load of 1 watt per kilogram of body
weight, until the pulse reached a steady rate. Every 3
minutes, the work load was increased by 25 watts, while the
rate of pedaling was kept constant at 50 rpm. The target
pulse (maximal pulse) for each subject was calculated as
200 minus the age of the subject (about 175 beats/min). All
the participants reached the target pulse. The maximal
oxygen consumption was estimated by using the partici-
rant’s weight, maximal work load, and pulse according to
the Astrand and Kaare nomogram. Anthropometric mea-
surements were made on men wearing shorts but without
shoes. The body mass index was calculated as weight
divided by the square of height (kg/m2). The skinfold thick-
ness was measured to the nearest millimeter with a Har-
pend pal (Holtain Ltd.) at the biceps, triceps, sub-
scapular, and supra-iliac sites on the right side of the body
as the subject stood in a relaxed position. Physical mea-
surements and determination of oxygen consumption
were repeated at the end of the study.

A brief, 26-item dietary questionnaire that was intended to
assess the change in intake during the trial was applied
at the midpoint and at the end of the trial. Participants were
asked if they had altered their intake of food items chosen
to cover the major sources of dietary fat, protein, and car-
bohydrates in our population. This included three items for
alcohol intake. Five summary questions were asked about
fat, sugar, starch, and alcohol. A simple three-point scale of measurement (increased, no change, reduced) was
used.

Differences between the baseline and the 9-week val-
ues within each trial group were assessed by the paired
r test. The mean differences between trial groups were
assessed for statistical significance by t tests for the dif-
ference between the paired differences in each trial

**Results**

The average age (± standard deviation) of the study participants was 24.8 ± 0.8 years (range 24 to 26 years). The average height was 1.76 ± 0.06 meters (range 1.64 to
1.93) and the weight was 70.8 ± 7.8 kg (range 54 to 87 kg).
The average body mass index was 22.8 ± 2.2 kg/m2
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The average estimated oxygen consumption was 38.3 ± 7.5 ml/kg/min (range 25 to 63 ml/kg/min).

Table 1 presents the baseline data and demonstrates that the characteristics of the trial groups were generally similar. It should be noted that, despite the similarity in body mass index in both groups, there was a significant difference in body weight and height notwithstanding the random allocation. The exercise group weighed less, and was shorter than, the control group. However, there was no appreciable or statistically significant difference in skinfold thickness between the groups, suggesting equal adiposity. The mean HDL cholesterol concentration of 33 to 34 mg/dl and the mean proportion of HDL cholesterol to total cholesterol (22% to 23%) reflect the selection criteria for HDL cholesterol of less than or equal to 40 mg/dl.

Although the distribution of HDL cholesterol was truncated by design, the well-described inverse association with plasma triglyceride persisted at baseline (r = -0.31, p = 0.01).

The intervention group participated in an average of 79% of the instructed exercise sessions and reported self-administered training in 65% of the requested sessions. In the control group, three students reported that they exercised on an average once every 2 weeks notwithstanding study instructions.

From Table 2 it can be seen that in both groups there was a very slight, but equal, increase in body mass index by the end of the study. There was no significant change in the measures of skinfold thickness either within or between the study groups. The estimated maximal oxygen consumption in the exercise group increased by 15% (p<0.001) but did not change at all in the control group. In the exercise group, the resting pulse rate decreased by 5 beats per minute (p = 0.004). No change in resting pulse rate was evident in the nonexercising group. However, the difference between the pulse rate differences was not statistically significant (p = 0.08). At the end of the study, the work load in the exercise group increased by 28 watts, while in the control group it decreased by 7 watts (p<0.001).

Changes in reported dietary intake were very small. The differences between the trial groups were not substantial and were not statistically significant (data not shown). There was no evidence for differential changes in intake of fats and carbohydrates.

The increase in estimated maximal oxygen consumption in the exercise group (compared with the controls) demonstrates that the regular moderate exercise done by this group resulted in a modest, but significant, improvement in their fitness.

Figure 1 and Table 3 describe the changes in plasma lipid and lipoprotein concentrations. At the end of the study, HDL cholesterol increased equally in both groups (4 mg/dl, p = 0.88 for the difference between the differences). In the exercise group, a small decrease in total

Table 1. Baseline Values for Exercise and Control Groups

<table>
<thead>
<tr>
<th></th>
<th>Exercise (n = 28)</th>
<th>Control (n = 27)</th>
<th>Significance (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>24.7 ± 0.8</td>
<td>25.0 ± 0.8</td>
<td>0.09</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.6 ± 2.3</td>
<td>23.1 ± 2.0</td>
<td>0.46</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>68.6 ± 7.4</td>
<td>73.1 ± 7.7</td>
<td>0.03</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.74 ± 0.06</td>
<td>1.78 ± 0.06</td>
<td>0.02</td>
</tr>
<tr>
<td>Biceps (mm)</td>
<td>5.1 ± 2.1</td>
<td>5.4 ± 2.4</td>
<td>0.71</td>
</tr>
<tr>
<td>Triceps (mm)</td>
<td>9.4 ± 3.5</td>
<td>10.1 ± 3.3</td>
<td>0.44</td>
</tr>
<tr>
<td>Subscapular (mm)</td>
<td>14.2 ± 4.7</td>
<td>13.5 ± 4.2</td>
<td>0.58</td>
</tr>
<tr>
<td>Supra-iliac (mm)</td>
<td>13.1 ± 6.1</td>
<td>12.1 ± 4.3</td>
<td>0.39</td>
</tr>
<tr>
<td>Skinfold (mm)</td>
<td>41.8 ± 14.2</td>
<td>41.2 ± 12.1</td>
<td>0.71</td>
</tr>
<tr>
<td>Total (mm)</td>
<td>38.6 ± 7.9</td>
<td>37.6 ± 6.9</td>
<td>0.76</td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td>34.4 ± 4.5</td>
<td>33.1 ± 6.2</td>
<td>0.37</td>
</tr>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>147.6 ± 25.8</td>
<td>149.4 ± 18.7</td>
<td>0.85</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>104.3 ± 41.2</td>
<td>98.3 ± 36.3</td>
<td>0.32</td>
</tr>
<tr>
<td>HDL/cholesterol</td>
<td>0.23 ± 0.05</td>
<td>0.22 ± 0.04</td>
<td>0.40</td>
</tr>
<tr>
<td>LDL-C (mg/dl)</td>
<td>93.4 ± 19.6</td>
<td>96.5 ± 17.6</td>
<td>0.64</td>
</tr>
</tbody>
</table>

Values are means ± SD. BMI = body mass index; EVo^max = estimated maximal oxygen consumption; HDL = high density lipoprotein; C = cholesterol; LDL = low density lipoprotein.

Table 2. Values in Exercise and Control Groups at Baseline and at Trial Completion

<table>
<thead>
<tr>
<th></th>
<th>Exercise group (n = 28)</th>
<th>Control group (n = 27)</th>
<th>p*</th>
<th>Net diff†</th>
<th>p diff‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>Mean SD</td>
<td>Mean SD</td>
<td>Mean SD</td>
<td>Diff SD</td>
<td></td>
</tr>
<tr>
<td>EVo^max (ml/kg/min)</td>
<td>36.8 7.9</td>
<td>44.1 7.6</td>
<td>5.5 5.7</td>
<td>0.001</td>
<td>37.6 6.9</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.6 2.3</td>
<td>22.8 2.4</td>
<td>0.2 0.43</td>
<td>0.05</td>
<td>23.1 2.0</td>
</tr>
<tr>
<td>Pulse (rate/min)</td>
<td>70.3 8.4</td>
<td>65.1 7.8</td>
<td>-5.2 8.8</td>
<td>0.01</td>
<td>69.5 12.2</td>
</tr>
<tr>
<td>Maximal load (watt)</td>
<td>159 33.5</td>
<td>187 35.0</td>
<td>25 35.1</td>
<td>&lt;0.001</td>
<td>188 30.5</td>
</tr>
<tr>
<td>Biceps (mm)</td>
<td>5.1 2.1</td>
<td>5.0 1.9</td>
<td>-0.1 1.4</td>
<td>0.70</td>
<td>5.4 2.4</td>
</tr>
<tr>
<td>Triceps (mm)</td>
<td>9.4 3.5</td>
<td>9.0 3.4</td>
<td>-0.4 2.0</td>
<td>0.42</td>
<td>10.1 3.3</td>
</tr>
<tr>
<td>Subscapular (mm)</td>
<td>14.2 4.7</td>
<td>13.9 4.1</td>
<td>-0.3 1.7</td>
<td>0.52</td>
<td>13.5 4.2</td>
</tr>
<tr>
<td>Supra-iliac (mm)</td>
<td>13.1 6.1</td>
<td>12.3 5.1</td>
<td>-0.8 3.1</td>
<td>0.14</td>
<td>12.1 4.3</td>
</tr>
<tr>
<td>Skinfold (mm) (total)</td>
<td>41.8 14.2</td>
<td>40.3 12.4</td>
<td>-1.5 5.0</td>
<td>0.12</td>
<td>41.2 12.1</td>
</tr>
</tbody>
</table>

The trial lasted 9 weeks.

*Paired t test for difference between 9 weeks and baseline.
†Difference between the differences (Exercise 9 weeks - Exercise baseline) - (control 9 weeks - control baseline).
‡t test for the difference between the differences.
There was no significant difference between the increment to total cholesterol increased similarly in both groups. The ratio of HDL cholesterol to total cholesterol increased similarly in both groups. There was no significant difference between the increment in the groups (p = 0.48).

The triglyceride concentration in the exercise group decreased by 4%, while at the same time it increased in the control group by 13% (p = 0.023). An inverse correlation between the change in estimated maximal oxygen consumption and triglyceride was noted in the exercise group (r = -0.43, p = 0.01) but not in the control group.

The similar pattern of increase in HDL cholesterol in both groups was evident as soon as 4 weeks into the trial (Figure 1). In the exercise group at the study midpoint, there was a tendency toward reduced triglyceride levels; in the control group, there was marked elevation. The difference in total cholesterol was more marked at midpoint than at the end of the study, but it was not statistically significant.

To establish whether the statistically significant increase in plasma HDL cholesterol seen in each of the two study groups at 9 weeks might have been caused by laboratory drift, measurements of total cholesterol, HDL cholesterol, and triglyceride were made again on frozen serum specimens stored at -30°C for 16 to 18 months (Table 4). The samples were paired (baseline and 9 weeks), and the order was randomized. All laboratory determinations were performed on a single day. The mean values of total cholesterol and triglyceride at baseline (on the frozen serum specimens) were close to those determined on the fresh plasma specimens during the trial. (The expectation that serum cholesterol values would exceed plasma values by about 3% was not realized.) Serum HDL cholesterol concentrations were lower by about 2 mg/dl than on fresh

Figure 1. Total cholesterol (TC), triglyceride (TG), and HDL-cholesterol (HDL-C) levels at baseline and after 4 and 9 weeks of the study in the exercise and the control group. Values are given as means ± SEM.

Table 3. Plasma Lipids and Lipoproteins in Exercise and Control Groups at Baseline and at Trial Completion

<table>
<thead>
<tr>
<th>Exercise group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline</strong></td>
<td><strong>Completion</strong></td>
</tr>
<tr>
<td>HDL-C</td>
<td>34.4</td>
</tr>
<tr>
<td>Mean</td>
<td>4.5</td>
</tr>
<tr>
<td>SD</td>
<td>4.2</td>
</tr>
<tr>
<td>Diff</td>
<td>p*</td>
</tr>
<tr>
<td>TG</td>
<td>104.3</td>
</tr>
<tr>
<td>Mean</td>
<td>41.2</td>
</tr>
<tr>
<td>SD</td>
<td>100.4</td>
</tr>
<tr>
<td>Diff</td>
<td>-3.9</td>
</tr>
<tr>
<td>p*</td>
<td>0.27</td>
</tr>
<tr>
<td>CHOL</td>
<td>87.9</td>
</tr>
<tr>
<td>Mean</td>
<td>19.6</td>
</tr>
<tr>
<td>SD</td>
<td>87.1</td>
</tr>
<tr>
<td>Diff</td>
<td>-7.2</td>
</tr>
<tr>
<td>p*</td>
<td>0.001</td>
</tr>
<tr>
<td>HDL-C/CHOL</td>
<td>0.23</td>
</tr>
<tr>
<td>Mean</td>
<td>0.05</td>
</tr>
<tr>
<td>SD</td>
<td>0.04</td>
</tr>
<tr>
<td>Diff</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

The trial lasted 9 weeks. The measurements were made on fresh samples.

HDL-C = high density lipoprotein cholesterol; TG = triglycerides; CHOL = cholesterol; LDL = low density lipoprotein.

*Paired t test for difference between 9 weeks and baseline.

†Difference between the differences (Exercise 9 weeks - Exercise baseline) - (control 9 weeks - control baseline).

‡‡ net difference (p = 0.93).

Table 4. Serum Lipids and Lipoproteins in Exercise and Control Groups at Baseline and at Trial Completion

<table>
<thead>
<tr>
<th>Exercise group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline</strong></td>
<td><strong>Completion</strong></td>
</tr>
<tr>
<td>HDL-C</td>
<td>32.5</td>
</tr>
<tr>
<td>Mean</td>
<td>5.5</td>
</tr>
<tr>
<td>SD</td>
<td>1.3</td>
</tr>
<tr>
<td>Diff</td>
<td>5.4</td>
</tr>
<tr>
<td>TG</td>
<td>102.2</td>
</tr>
<tr>
<td>Mean</td>
<td>48.3</td>
</tr>
<tr>
<td>SD</td>
<td>87.8</td>
</tr>
<tr>
<td>Diff</td>
<td>45.8</td>
</tr>
<tr>
<td>p*</td>
<td>-14.4</td>
</tr>
<tr>
<td>CHOL</td>
<td>92.6</td>
</tr>
<tr>
<td>Mean</td>
<td>25.8</td>
</tr>
<tr>
<td>SD</td>
<td>86.9</td>
</tr>
<tr>
<td>Diff</td>
<td>25.2</td>
</tr>
<tr>
<td>p*</td>
<td>15.3</td>
</tr>
<tr>
<td>HDL-C/CHOL</td>
<td>0.24</td>
</tr>
<tr>
<td>Mean</td>
<td>0.1</td>
</tr>
<tr>
<td>SD</td>
<td>0.25</td>
</tr>
<tr>
<td>Diff</td>
<td>0.04</td>
</tr>
</tbody>
</table>

The trial lasted 9 weeks. The measurements were made on frozen samples stored 16 to 18 months.

See legend of Table 3 for abbreviations.

*Paired t test for difference between 9 weeks and baseline.

†Difference between the differences (Exercise 9 weeks - Exercise baseline) - (control 9 weeks - control baseline).

‡‡ test for the difference between the differences.
plasma in both groups, probably reflecting the effects of frozen storage. The baseline difference between the groups for HDL cholesterol measured on fresh plasma (exercise group higher by 1.3 mg/dl) was almost identical to that of frozen stored serum (1.4 mg/dl). By 9 weeks, HDL cholesterol had increased by 1.3 mg/dl in the exercise group (not significant) compared with an increase of 3.2 mg/dl \((p<0.01)\) in the control group. The difference in increment, 1.9 mg/dl higher in the control group, was not statistically significant \((p = 0.15)\). The absence of a statistically significant exercise effect on HDL cholesterol and on total cholesterol (as determined by the net difference between the exercise and control groups) and the presence of such an association for triglycerides is consistent with the findings for the fresh plasma specimens.

Changes in HDL2 cholesterol and HDL3 cholesterol concentrations, measured in 18 participants in each trial group on fresh plasma, did not differ significantly between the groups at the end of the study (Table 5).

Upon termination of the study, the exercising students were allowed continued free access to the university sports facilities and were encouraged to engage in regular aerobic exercise. Only 40% of the trial group continued to exercise at a similar frequency for 2 months, and by 6 months only 15% continued to exercise regularly.

**Discussion**

The present study demonstrates that moderate exercise over a short term (9 weeks) in young, healthy, nonobese men with relatively low HDL cholesterol levels improved their fitness but did not significantly affect their plasma HDL cholesterol, estimated LDL cholesterol, or total cholesterol concentrations, as evidenced by the absence of a differential change in these measures between groups. We infer from the similar results in both trial groups that exercise was not the reason for the plasma HDL cholesterol elevation seen in each study group. There was no differential change in body weight or skinfold thickness measurements between the two groups; weight change in each group was minimal. Although the trial groups differed significantly in weight and height on intake into the study, there was little difference in measures of adiposity at baseline, and no differential change in adiposity by the end of the study.

The equal increase in HDL cholesterol measured in fresh plasma in both trial groups is perplexing, although our main analysis related not to changes within each group but to differences in change between the groups. These differences were, as noted, trivial. The equal reduction in LDL cholesterol in the study groups is almost certainly a complementary reflection of the increase in HDL cholesterol, because the estimation of LDL cholesterol is not independent of HDL cholesterol measurement. We postulate three possible explanations for the equal increments in plasma HDL cholesterol: 1) regression to the mean, 2) seasonal effects, and 3) laboratory drift. The selection process of the sample, in which a central criterion was HDL cholesterol concentrations at or below the population median on one or two examinations and below 41 mg/dl at the medical examination, would not exclude regression to the mean as an explanation for the increment. The presence of a randomized, concurrent control group, however, prevents regression to the mean from biasing the central comparison of interest. Seasonal effects may have played a partial role. Among men examined in the Jerusalem Lipid Research Clinic Study,27 plasma HDL cholesterol increased by 1 to 2 mg/dl over the months parallel to the trial (without taking the effects of chance into account). We were able to examine directly the issue of laboratory drift by using a very precise enzymatic method of determination on frozen stored serum specimens in a single assay procedure on one day. The results suggest that laboratory drift explained between 30% to 70% of the increment seen in HDL cholesterol measured in fresh plasma. We consider it possible that the statistically significant increase of 3.2 mg/dl still apparent in serum HDL cholesterol measured in the frozen stored specimens in the control group relates to a slightly stronger effect of regression to the mean in this group, which at baseline had an HDL cholesterol mean value lower by 1.4 mg/dl than the exercise group.

At first glance, these findings appear to differ from those of other studies carried out in sedentary, healthy, middle-aged men. In a number of well-controlled studies, body weight reduction in the exercise group seems to play a major role in the response of HDL cholesterol levels to exercise. A meta-analysis of 95 studies18 carried out before 1985 showed that HDL cholesterol increase in the exercise groups was dependent on weight reduction. There are, however, contradictory data28 that suggest that aerobic exercise causes an increase in HDL cholesterol and apo A-I proportionally. Our findings from a randomized controlled clinical trial in a population that had low HDL cholesterol concentrations, no weight loss during the trial, and no change in skinfold thickness are consistent with the meta-analysis conclusion.

In designing and carrying out our study, we made an attempt to investigate the relation between a single, modifi-
able factor (exercise) and HDL cholesterol levels. The effect of other factors that may influence HDL cholesterol concentrations including smoking, change in body weight, change in skinfold thickness, alcohol consumption, and major changes in diet (as assessed by a questionnaire) were excluded as likely explanations.

Our study differed from all prior investigations in our choice of the study population. Our central question of interest was whether moderate exercise can influence HDL cholesterol concentrations in young men whose only obvious risk factor for coronary heart disease was low HDL cholesterol levels. If low HDL cholesterol concentrations can be modified by moderate exercise, this could encourage participation in regular exercise as a valuable preventive measure for such individuals.

Our study demonstrated that 9 weeks of moderate exercise in young men with relatively low HDL cholesterol concentrations had no effect on total cholesterol, HDL cholesterol, or LDL cholesterol concentration, in spite of the fact that it improved their fitness. Change in oxygen consumption in the exercise group was weakly correlated with change in HDL cholesterol ($r = 0.16, p = 0.21$). Similar correlations ($r = 0.24$) in a short-term exercise study have been described by others. The fact that exercise induced a modest decrease in triglyceride levels, but did not at the same time affect HDL cholesterol levels, suggests the possibility that: 1) exercise did not decrease triglyceride levels by a mechanism of exchange between cholesterol and triglyceride in HDL and 2) the change in triglyceride level was independent of HDL cholesterol levels.

In assessing the lack of HDL cholesterol response in our study, several issues deserve consideration: 1) The exercise period of 9 weeks was relatively short and, on the average, the subjects attended only 14 supervised sessions but two unsupervised sessions. It is possible that a longer period of activity would eventually have raised the HDL cholesterol level as has been shown in previous studies. However, in most studies that showed an elevation of HDL cholesterol in exercise groups, the elevation was evident after only a short period of exercise (4 to 8 weeks). Furthermore, in some other studies, the elevated HDL cholesterol level tended to vanish during a longer period of follow-up. 2) More vigorous exercise may be needed in a better response, as has been seen in other studies.

In our study, the decrease in heart rate and the moderate increase in estimated maximal oxygen consumption in the exercise group were taken as evidence that the exercise program was effective in achieving improved fitness. Even if it had been possible to raise HDL cholesterol levels in our study group by very vigorous exercise, this would be of little public health importance in our population, because it is unlikely that habits could be changed so drastically. This is supported by the fact that only 40% of the exercise group continued to participate in moderate exercise at the end of the study, and only 15% continued to exercise after 6 months, although the potential benefit of such exercise had been emphasized and continued supervision had been offered.

One may speculate that men with consistently low HDL cholesterol levels may respond less to exercise than those with higher concentrations. This possibility of heterogeneity in response to exercise would be a worthwhile avenue to pursue.

Acknowledgments

The authors thank Hillel Ruskin who made available the University sports facilities for this trial, Yekiel Friedlander who assisted with the randomization, and Mario Baras who helped in data processing. We thank the reviewers for their helpful and constructive comments.

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Index Terms: HDL • cholesterol • exercise
Effect of moderate exercise on serum lipids in young men with low high density lipoprotein cholesterol.
I Raz, H Rosenblit and J D Kark

Arterioscler Thromb Vasc Biol. 1988;8:245-251
doi: 10.1161/01.ATV.8.3.245

Arteriosclerosis, Thrombosis, and Vascular Biology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1079-5642. Online ISSN: 1524-4636

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