Physical Exercise Conditioning in the Absence of Weight Loss Reduces Fasting and Postprandial Triglyceride-Rich Lipoprotein Levels

Moshe S. Weintraub, MD, Yacob Rosen, Robert Otto, PhD,
Shlomo Eisenberg, MD, and Jan L. Breslow, MD

The effect of physical exercise conditioning on fasting and postprandial lipoprotein levels was studied in six normolipidemic subjects. The study consisted of two phases: a baseline stabilization phase in which subjects maintained their regular physical activity and an exercise conditioning phase in which subjects had 29 exercise sessions during a 7-week period. Each of these sessions consisted of jogging on a treadmill for 30 minutes. The subjects averaged 15.2 miles/wk. To control for possible confounding factors, such as changes in diet composition and weight loss, we placed the subjects on a metabolic diet and increased their daily caloric intake during the exercise phase. At the end of each phase of the study, a vitamin A–fat loading test was done to specifically label and follow postprandial lipoprotein levels, and a maximum oxygen consumption test was done to evaluate the subjects’ physical fitness. The exercise conditioning phase significantly increased the subjects’ aerobic capacity and postheparin lipoprotein lipase activity, and the phase decreased fasting triglyceride levels. Physical exercise also significantly decreased chylomicon (Sf >1,000) levels by 37%. In summary, this study suggests that physical exercise conditioning reduces fasting and postprandial lipoprotein levels by increasing the catabolism of triglyceride-rich particles. Because these particles may have a role in atherogenesis, this could be a major mechanism by which exercise prevents coronary heart disease.

(79:1007–1014)

Epidemiologic studies suggest that regular physical exercise prevents coronary heart disease.1–4 The exact mechanism is unclear, but physical exercise has had a favorable effect on some of the risk factors associated with coronary heart disease, such as blood pressure, glucose tolerance, and heart rate.5–7 Although lipoprotein levels are major risk factors for the development of coronary heart disease,8–11 the effect of physical exercise on these levels is not clearly established. Some studies have reported favorable effects of exercise on lipoprotein levels,12–17 but others have found no effect.18–23 Many of these studies did not carefully control for confounding variables, such as diet and weight loss. This is the probable explanation for the different results that have been reported. In addition, only a few studies have investigated the effect of physical exercise on postprandial lipoprotein levels. These studies were concerned primarily with the effects of short-term exercise on the turbidity of plasma or triglyceride levels after a fatty meal.24,25 Another study examined the effect of short-term exercise on intravenously administered triglycerides.26 The effects of long-term aerobic conditioning on postprandial lipoprotein levels, while diet and weight loss were carefully controlled, have not been reported.

Most of our lives are spent in the postprandial state during which time the vessel wall is exposed to postprandial lipoproteins. Zilversmit27 has suggested and others have shown in human, animal,28–32 and in vitro studies that these lipoproteins may be
particularly atherogenic. This may be because these particles are metabolized on the endothelial surface of arteries and because their cholesterol becomes incorporated into the vessel wall where it may stimulate lesion formation. Therefore, to understand the relation of physical exercise, lipoprotein levels, and coronary heart disease risk, it is important to measure the effects of exercise on both fasting and postprandial lipoprotein levels.

In this study, we have used the vitamin A–fat loading test, which specifically labels intestinally derived lipoproteins with retinyl palmitate, to measure postprandial lipoprotein levels. Intestinal lipoprotein (chylomicron) metabolism involves a two-step process and includes triglyceride hydrolysis of the newly formed particles by lipoprotein lipase and subsequent uptake of the chylomicron remnants by receptor-mediated recognition of apolipoprotein E on the particle’s surface. As we have shown in our previous studies, the vitamin A–fat loading test, as we have implemented it, can be used to follow both chylomicron and chylomicron remnant metabolism. In addition, the present study design allowed careful control of confounding variables, such as changes in weight and diet composition, so that the effects observed on fasting and postprandial lipoprotein levels were attributable mainly to physical exercise and aerobic conditioning.

Methods

Subjects

Six healthy normolipidemic adult men were admitted to the General Clinical Research Center of The Rockefeller University in New York. The subjects’ characteristics including age, sex, apolipoprotein E phenotype, height, weight, body mass index, and lipid and lipoprotein levels upon recruitment into the study are listed in Table 1. The subjects had no evidence of cardiac, hepatic, renal, or endocrine diseases and were not receiving medications.

Study Design

The study design is shown in Figure 1. The study included two phases: a baseline stabilization phase and an exercise conditioning phase. In the baseline stabilization phase, subjects were placed on a metabolic diet for 24 days, and their regular physical activity was maintained. On the 15th day, a postheparin lipolytic activity test was done, and on the 20th day, the vitamin A–fat loading test was performed. On the 24th day, to evaluate the subjects’ basal physical fitness level (aerobic capacity), a maximum oxygen consumption test was done. Five blood samples were drawn after a 12-hour fast in the last week of the baseline stabilization phase on days 18, 19, 20, 22, and 23. These were analyzed for lipid, lipoprotein, and apolipoprotein levels. Fasting blood samples were also analyzed for glucose, insulin, and HgbAIC levels. Two-hour postprandial insulin levels were also measured. The average of the five measurements for each variable was used in the data analysis.

Figure 1. A schematic description of study sequences. The study consisted of two phases: baseline stabilization and exercise conditioning. On the baseline and second half of the exercise phase, subjects were placed on a metabolic diet. The daily caloric intake was increased (↑) on the exercise phase to prevent weight loss. On each phase, a postheparin lipolytic activity (PHLA), a vitamin A–fat loading (FTT), and a maximum oxygen consumption test was performed.
In the exercise conditioning phase, subjects were started on an exercise training program for 7 weeks. In the first 25 days, subjects were on ad-lib diets. For the last 24 days, they were again placed on the same metabolic diet as in the baseline stabilization phase, but the daily caloric intake was increased to account for the physical activity so that each subject’s weight would remain stable. At the end of the exercise conditioning phase, the same tests were done as in the baseline stabilization phase (see previous paragraph for exact days on which specific tests were performed). To prevent a possible acute effect of exercise on postprandial lipoprotein levels, the vitamin A–fat loading test was begun 36 hours after the previous exercise session.

Diet Manipulations

The metabolic diet consisted of 42% of calories from fat, 43% from carbohydrate, and 15% from protein. The polyunsaturated to saturated fat (P/S) ratio was 0.08, and the cholesterol content was 200 mg/1,000 calories. The diet consisted of natural foods whose composition had been determined by the USDA and listed in the food tables of Agriculture Handbook 8.42 For variety, a 2-day menu was developed and rotated through the metabolic diet periods. The caloric requirements for each subject for both phases of the study were estimated with the Harris-Benedict equation,43 and adjustments were made, when necessary, to maintain a steady weight during the entire study.

Vitamin A–Fat Loading Test

The vitamin A–fat loading test was performed as recently described.40,41 After an overnight 12-hour fast, subjects were given a fatty meal and 60,000 units aqueous vitamin A/m² body surface. The fatty meal contained 50 g fat/m² body surface consisting of 65% of calories as fat, 20% as carbohydrate, and 15% as protein. The meal contained 600 mg cholesterol/1,000 calories, and the P/S ratio was 0.3. The fatty meal was given as a milkshake, scrambled eggs, bread, and cheese, and it was eaten in 10 minutes. The vitamin A was added to the milkshake. After eating the meal, subjects fasted for 12 hours, but as much drinking water as desired was allowed. To measure levels of retinyl palmitate, blood samples were drawn before the meal, every hour after the meal until 6 hours, and then every 2 hours until 12 hours. The subjects tolerated the meal well, and no one had diarrhea or other symptoms of malabsorption.

Maximum Oxygen Consumption Test

Maximum oxygen consumption tests were done in the Human Performance Laboratory of the Adelphi University in Long Island with a treadmill. During the test an electrocardiogram recording was used to derive the heart rate, and open circuit spirometry was used to measure oxygen and carbon dioxide concentrations with Beckman OM11 and LB2 gas analyzers (Fullerton, California), respectively. The speed of the treadmill was increased until the subject’s heart rate was 60–70% of the predicted maximum. This rate was achieved in 4–5 minutes with the treadmill at a speed of 5–6 mph. When a steady state was achieved, the work load was increased by elevating the treadmill by 2° every 2 minutes until the subject was too exhausted to continue.

Exercise Program

The exercise sessions were strictly supervised. The main goal of the program was to improve aerobic capacity. Exercise was performed on a Trotter 440 motorized treadmill (Hopedale, Massachusetts). Each session consisted of a 5-minute warm-up period, 20 minutes of intense exercise at 80% of predicted maximum heart rate, and a 5-minute, slow running, cool-down period. Heart rate was monitored during the whole session by a pulsimeter (CIC) belt (Model Exersentry III; Respironics, Monroeville, Pennsylvania). Each of the subjects had 29 exercise sessions during the 7 weeks of the exercise program. The average mileage per week was 15.2.

Analysis of Samples

Venous blood was drawn from the forearm and transferred to a tube containing sodium ethylenediaminetetraacetic acid (EDTA). Samples were immediately centrifuged at 1,500g for 15 minutes, and 0.5 ml plasma was stored wrapped in foil at −20° C for retinyl ester assay. Another 0.5 ml was stored at 4° C for triglyceride determinations. An aliquot of 2.5 ml plasma was transferred into a 0.5×2-in. cellulose nitrate tube and layered with 2.5 ml NaCl solution (d=1.006 g/ml). Tubes were subjected to preparative ultracentrifugation for 1.6×10⁶g/min in a Beckman SW-55 rotor to float chylomicron particles of Sf>1,000.44,45 The chylomicron-containing supernatant was removed and brought to a total volume of 2 ml with saline. The infranatant was brought to a volume of 5 ml with saline. Aliquots (0.5 ml) of supernatant and infranatant were wrapped in foil and assayed for retinyl ester. Additional aliquots were assayed for triglyceride concentration. As discussed elsewhere, the procedure appears to separate a predominantly chylomicron population from a predominantly remnant population.40,41 Retinyl esters were quantified as previously described with retinyl acetate as an internal standard.40,41

Lipid, Lipoprotein, and Apolipoprotein Determinations

Cholesterol and triglycerides were measured enzymatically with Boehringer Mannheim reagents (Cholesterol 236691, Triglyceride 126012) (Indianapolis, Indiana). Total and high-density lipoprotein (HDL) cholesterol measurements were standardized by the Lipid Standardization Program of the Center for Disease Control, Atlanta, Georgia. HDL was determined after precipitation of whole plasma with
Table 2. Effects of Physical Exercise Conditioning on Fasting Plasma Lipids and Lipoproteins

<table>
<thead>
<tr>
<th>Subject</th>
<th>Weight (kg)</th>
<th>$V_O^{max}$ (ml/kg · min)</th>
<th>TC (mg/dl)</th>
<th>TG (mg/dl)</th>
<th>VLDL (mg/dl)</th>
<th>LDL (mg/dl)</th>
<th>HDL (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Base</td>
<td>Exer</td>
<td>Base</td>
<td>Exer</td>
<td>Base</td>
<td>Exer</td>
<td>Base</td>
</tr>
<tr>
<td>1</td>
<td>76.2</td>
<td>76.5</td>
<td>42.8</td>
<td>54.2</td>
<td>166</td>
<td>150</td>
<td>60</td>
</tr>
<tr>
<td>2</td>
<td>61.8</td>
<td>62.5</td>
<td>42.8</td>
<td>55.6</td>
<td>164</td>
<td>172</td>
<td>60</td>
</tr>
<tr>
<td>3</td>
<td>81.3</td>
<td>82.6</td>
<td>32.6</td>
<td>42.6</td>
<td>160</td>
<td>166</td>
<td>107</td>
</tr>
<tr>
<td>4</td>
<td>77.3</td>
<td>77.7</td>
<td>35.0</td>
<td>57.0</td>
<td>187</td>
<td>170</td>
<td>57</td>
</tr>
<tr>
<td>5</td>
<td>64.9</td>
<td>65.6</td>
<td>15.0</td>
<td>35.0</td>
<td>194</td>
<td>184</td>
<td>115</td>
</tr>
<tr>
<td>6</td>
<td>85.2</td>
<td>85.2</td>
<td>41.3</td>
<td>55.9</td>
<td>177</td>
<td>170</td>
<td>66</td>
</tr>
<tr>
<td>Mean</td>
<td>74.4</td>
<td>75.0</td>
<td>34.9</td>
<td>50.0*</td>
<td>174</td>
<td>168</td>
<td>81</td>
</tr>
<tr>
<td>SD</td>
<td>9.2</td>
<td>9.1</td>
<td>10.6</td>
<td>9.1</td>
<td>13</td>
<td>11</td>
<td>14</td>
</tr>
</tbody>
</table>

These fasting lipids and lipoprotein concentrations are the average of five measurements for each subject in the last week of both the baseline and exercise conditioning phases of the study.

TC, total cholesterol; TG, triglyceride; VLDL, very low-density lipoprotein; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

*Significantly different from the baseline stabilization phase by two-tailed simple sign test, $p<0.05$.

dextran sulfate-magnesium. HDL plus low-density lipoprotein (LDL) cholesterol was measured on the infranatant after a 2-hour spin in a Beckman air furnace to float the very low-density lipoprotein (VLDL). Appropriate corrections were made for dilution factors, and VLDL, LDL, and HDL levels were calculated. Three frozen aliquots of plasma obtained during the last week of each study phase were assayed for apoprotein A-I and B levels by sandwich enzyme-linked immunosorbent assay as previously described.

Postheparin Plasma Lipolytic Activities

Lipolytic activities were determined 1 week before the vitamin A–fat loading test. Lipoprotein lipase and hepatic triglyceride lipase were released into the circulation by intravenous heparin injection at a dose of 60 units/kg body wt. After 15 minutes, blood was drawn into tubes containing 4 mM EDTA. The plasma was immediately separated at 4°C by centrifugation at 2,700 rpm for 12 minutes and promptly frozen at −70°C. The assay itself is a modification of the method of Krauss et al.

Statistical Analysis

The differences between the two study phases were analyzed for significance with a two-tailed simple sign test with an $\alpha$ criterion of 0.05. Correlations between all measured variables and retinyl palmitate responses were calculated by linear regression analysis with the least-squares methods. Calculations were performed on the Rockefeller University Hospital CLINFO system.

Results

The effects of the exercise program on weight, maximum oxygen consumption, and fasting lipid and lipoprotein levels is shown in Table 2. Subjects' weights were stable during the entire study with no difference observed between the baseline stabilization and exercise conditioning phases. Exercise significantly increased maximum oxygen consumption from 34.9±5 to 50.0±9 mg/kg/min and significantly decreased fasting triglyceride levels from 81±14 to 70±16 mg/dl. Although exercise tended to lower the levels of total cholesterol, VLDL, and LDL and to raise the level of HDL, these effects were not significant.

The effect of the exercise program on postheparin lipolytic activity, fasting apolipoprotein B, apolipoprotein A-I, glucose, HgbAIC, insulin, and postprandial insulin levels are shown in Table 3. Exercise significantly increased lipoprotein lipase activity and glucose levels. There were trends for exercise to decrease hepatic triglyceride lipase activity and apolipoprotein B levels, but these were not significant. There were no consistent effects of exercise on the levels of apolipoprotein A-I, HgbAIC, and fasting and postprandial insulin.

The effects of the exercise program on postprandial lipoprotein levels are shown in Table 4 and Figure 2. Postprandial lipoprotein levels, as measured by the area below the retinyl palmitate curve or retinyl palmitate peak levels, were lower after exercise for both the chylomicron fraction (37% reduction) and nonchylomicron fraction (28% reduction). The former was statistically significant, whereas the latter barely missed significance.

This study also provides information about the relation of aerobic capacity and indexes of lipoprotein metabolism. This information was obtained by seeking correlations between the maximum oxygen consumption measurements and the corresponding levels of fasting and postprandial lipoproteins, postheparin lipolytic activities, fasting glucose levels, and fasting and postprandial insulin levels. As shown in Figure 3, significant correlations of maximum oxygen consumption after exercise were only found with lipoprotein lipase activity, chylomicron retinyl palmitate area, and fasting triglyceride levels. The correlation with lipoprotein lipase activity was positive, whereas the correlations with the latter two were negative. In the baseline stabilization phase,
TABLE 3. Effects of Physical Exercise Condition on Plasma Lipolytic Activity, Apolipoprotein A-I and B Levels, and Glucose and Insulin Levels

<table>
<thead>
<tr>
<th>Subject</th>
<th>Lipolytic activity (μmole FFA/ml/hr)</th>
<th>Apolipoprotein B mg/dl</th>
<th>Apolipoprotein A-I mg/dl</th>
<th>Glucose mg/dl</th>
<th>Fasting insulin μIU/ml</th>
<th>ΔInsulin μIU/ml</th>
<th>HgbA1c (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LPL Exer</td>
<td>HTGL Exer</td>
<td>Base Exer</td>
<td>Base Exer</td>
<td>Base Exer</td>
<td>Base Exer</td>
<td>Base Exer</td>
</tr>
<tr>
<td>1</td>
<td>10.2</td>
<td>11.7</td>
<td>13.8</td>
<td>15.3</td>
<td>82</td>
<td>62</td>
<td>96</td>
</tr>
<tr>
<td>2</td>
<td>10.0</td>
<td>11.3</td>
<td>18.5</td>
<td>10.8</td>
<td>71</td>
<td>69</td>
<td>136</td>
</tr>
<tr>
<td>3</td>
<td>4.9</td>
<td>6.2</td>
<td>5.3</td>
<td>5.0</td>
<td>69</td>
<td>71</td>
<td>121</td>
</tr>
<tr>
<td>4</td>
<td>10.2</td>
<td>10.6</td>
<td>6.1</td>
<td>5.5</td>
<td>98</td>
<td>74</td>
<td>102</td>
</tr>
<tr>
<td>5</td>
<td>4.1</td>
<td>5.7</td>
<td>25.0</td>
<td>26.1</td>
<td>103</td>
<td>93</td>
<td>137</td>
</tr>
<tr>
<td>6</td>
<td>8.3</td>
<td>9.6</td>
<td>16.6</td>
<td>8.7</td>
<td>73</td>
<td>67</td>
<td>120</td>
</tr>
<tr>
<td>Mean</td>
<td>7.9</td>
<td>9.2*</td>
<td>14.2</td>
<td>11.9</td>
<td>83</td>
<td>73</td>
<td>119</td>
</tr>
<tr>
<td>SD</td>
<td>2.7</td>
<td>2.6</td>
<td>7.5</td>
<td>7.9</td>
<td>14</td>
<td>10</td>
<td>17</td>
</tr>
</tbody>
</table>

FFA, free fatty acid; LPL, lipoprotein lipase; HTGL, hepatic triglyceride lipase; ΔInsulin, the difference between postprandial insulin taken 2 hours after the fatty meal and the fasting level; Base, baseline; Exer, exercise.

*Significantly different from the baseline stabilization phase by two-tailed simple sign test, p=0.032.

The same trends occurred, but these were either not significant or less significant (Figure 3).

The mechanisms controlling postprandial lipoprotein levels were explored by correlating the chylomicron retinyl palmitate area with the corresponding levels of lipoprotein lipase activity, HDL, and fasting triglycerides. As shown in Figure 4, after exercise, chylomicron retinyl palmitate area significantly correlated inversely with lipoprotein lipase activity and directly with fasting triglyceride levels. There were no correlations with HDL levels after exercise, but a trend occurred before exercise for an inverse correlation of chylomicron retinyl palmitate area and HDL levels.

Discussion

This study explores the relation between aerobic physical fitness and fasting and postprandial lipoprotein levels under conditions controlled for diet composition and weight change, which are two factors that have confounded most previous studies of this subject. During the baseline stabilization and exercise conditioning phases, subjects were on a metabolic diet with exactly the same composition. The only difference was that during the exercise conditioning phase the caloric intake was increased by 300–500 calories/day to prevent weight loss. In addition, acute effects of exercise on postprandial lipoprotein levels would be diminished by performing the vitamin A-fat loading 36 hours after the last exercise session. In view of these precautions, exercise conditioning was probably the main factor causing the changes in lipid metabolism in this study.

This study showed that a 43% increase in maximum oxygen consumption, achieved by regular aerobic physical exercise during a 7-week period by running an average of 15.2 miles/wk, significantly reduced total postprandial lipoprotein levels by 32%. The exercise program also significantly decreased fasting triglyceride levels and increased postheparin lipoprotein lipase activity, both by 16%. Because exogenous and endogenous triglyceride-rich lipoproteins are catabolized by lipoprotein lipase, this supports the hypothesis that the reduction in fasting triglyceride and postprandial lipoprotein levels was due to increased clearance. The correlations of maximum oxygen consumption positively with lipoprotein lipase activity and negatively with chylomicron retinyl palmitate area and fasting triglyceride levels (Figure 3) suggest that the change in aerobic physical fitness was the

TABLE 4. Effects of Physical Exercise Condition on Postprandial Lipoprotein Levels

<table>
<thead>
<tr>
<th>Subject</th>
<th>Area below retinyl palmitate curves μg/l/hr</th>
<th>Retinyl palmitate peak levels (μg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Chylomicron fraction</td>
<td>Nonchylomicron fraction</td>
</tr>
<tr>
<td></td>
<td>Base Exer</td>
<td>Base Exer</td>
</tr>
<tr>
<td>1</td>
<td>13,307</td>
<td>5,033</td>
</tr>
<tr>
<td>2</td>
<td>4,788</td>
<td>3,970</td>
</tr>
<tr>
<td>3</td>
<td>9,557</td>
<td>7,714</td>
</tr>
<tr>
<td>4</td>
<td>6,156</td>
<td>4,989</td>
</tr>
<tr>
<td>5</td>
<td>15,328</td>
<td>8,934</td>
</tr>
<tr>
<td>6</td>
<td>9,654</td>
<td>6,665</td>
</tr>
<tr>
<td>Mean</td>
<td>9,798</td>
<td>6,217*</td>
</tr>
<tr>
<td>SD</td>
<td>4,033</td>
<td>1,885</td>
</tr>
</tbody>
</table>

*Significantly different from the baseline stabilization phase by two-tailed simple sign test, p=0.032.
direct cause of the changes observed in the fasting and postprandial lipoprotein levels.

In our study, postexercise chylomicron retinyl palmitate area significantly and positively correlated with fasting triglyceride levels, and it inversely correlated with lipoprotein lipase activity (Figure 4). This is in agreement with current concepts that chylomicrons and endogenous triglycerides compete for clearance by a common saturable pathway regulated by lipoprotein lipase.\textsuperscript{49–51} Before exercise, the chylomicron retinyl palmitate area correlated inversely with HDL levels. This is expected because HDL levels are dependent on the rate of transfer to HDL of surface remnants from chylomicrons and VLDL formed during lipolysis.\textsuperscript{52,53} The lack of correlation between postexercise chylomicron retinyl palmitate area and HDL levels may be because the change in triglyceride-rich lipoprotein metabolism occurs before a change in HDL levels. In fact, the increase in physical fitness in our study was not associated with a significant increase in HDL or apolipoprotein A-I levels. We speculate that the initial effect of exercise is to increase lipolysis and lower fasting and postprandial triglyceride levels and that the increase in HDL concentration is a later event that may reflect changes in body composition. In support of this hypothesis, Patsch et al.\textsuperscript{52} in a longitudinal study of vigorous exercise in two subjects, found that HDL levels increased only after 6 months of exercising. In addition, Wood et al.\textsuperscript{54} recently reported that weight loss, achieved either through dieting or exercise, was responsible for changes in HDL levels. These studies are consistent with our findings. Alternatively, the lack of an observed effect of exercise conditioning on HDL or apolipoprotein A-I levels may reflect the limited statistical power to detect small differences in our study design.

Population-based studies have shown that physically active people have higher HDL levels than sedentary individuals and that the level of physical exercise correlates with HDL levels.\textsuperscript{55–58} Other studies have also shown correlations between physical activity and postheparin lipoprotein lipase activity,\textsuperscript{53,59,60} and inverse correlations between HDL and postprandial lipoprotein levels.\textsuperscript{51,52} The present metabolic study suggests that the initial events after beginning exercise conditioning include improved aerobic capacity, increased lipoprotein lipase activity, and increased catabolism of endogenous and exogenous triglyceride-rich lipoproteins.

The inverse association of coronary heart disease incidence and HDL levels in many populations has been used as evidence that HDL “protects” against atherosclerosis. The proposed mechanism is through a reverse cholesterol transport pathway mediated.
by HDL. However, the current metabolic study shows that the **earliest** effect of physical exercise is to decrease the levels of fasting and postprandial lipoproteins and that this most probably precedes an effect on HDL levels. Thus, the protective effect of physical exercise, and other interventions that affect HDL levels, on coronary heart disease incidence may be through a primary effect on triglyceride-rich lipoproteins. This is plausible because VLDL, intermediate-density lipoproteins, and postprandial lipoproteins have atherogenic properties, and decreasing vessel wall exposure to them may be beneficial.

In this study, we isolated the effect of physical fitness on fasting and postprandial lipoprotein levels by controlling diet and preventing weight loss. However, it is well known that body mass is a principal determinant of fasting and postprandial lipoprotein levels, and from a clinical view, the greatest benefit would be to combine exercise and weight loss.

**Acknowledgments**

We thank the Nursing Service of the Clinical Research Center of Rockefeller University for their help in performing the vitamin A–fat loading tests, especially Carmen Schmidt, RN; and Lori Cersosimo and Lorraine Duda for their expertise in preparing the manuscript.

**References**


60. Sady SP, Cullinane EM, Her fret PN: Training, diet and physical characteristics of distance runners with low or high concentrations of high density lipoprotein cholesterol. *Atherosclerosis* 1984;53:273–281


**KEY WORDS**: postprandial lipoproteins; physical exercise conditioning; chylomicrons; chylomicron remnants
Physical exercise conditioning in the absence of weight loss reduces fasting and postprandial triglyceride-rich lipoprotein levels.
M S Weintraub, Y Rosen, R Otto, S Eisenberg and J L Breslow

Circulation. 1989;79:1007-1014
doi: 10.1161/01.CIR.79.5.1007

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/79/5/1007