Acute changes in blood lipids and enzymes in postmenopausal women after exercise

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Abstract

Acute changes in blood lipids and enzymes in postmenopausal women after exercise: J Appl Physiol 99: 609–615, 2005. First published March 17, 2005; doi:10.1152/japplphysiol.01354.2004.—The effectiveness of lifestyle intervention strategies to improve blood lipids in women may be dependent on preexisting cholesterol concentrations. We characterized the effects of cholesterol status on blood lipid, lipoprotein lipid, and lipid regulatory enzyme responses to a single session of aerobic exercise in physically active, postmenopausal women. In this study, blood samples were obtained from 12 women with high cholesterol (HC; ≥200 mg/dl) and 13 women with normal cholesterol (NC; <200 mg/dl), 24 h before (Pre), immediately after (IPE), and 24 and 48 h after an exercise session (treadmill walking at 70% peak oxygen consumption, 400 kcal). We found that repeated-measures analysis revealed the following: 1) preexercise cholesterol differences did not influence the lipid or lipoprotein lipid responses to exercise; 2) for both groups, triglyceride was significantly reduced (~8.5%) after exercise; 3) the concentration profile over time for high-density lipoprotein cholesterol was significant for both groups, first falling at IPE then rising back to Pre levels by 24 h after exercise; 4) the lecithin-cholesterol acyltransferase activity (LCATA) exercise response was group dependent, increasing modestly in the NC group at 24 and 48 h, but rising 17% by 48 h in the HC group only and then fell at 24 and 48 h (by 21%) compared with Pre; and 6) cholesterol ester transfer protein activity was unchanged by exercise. From these findings, we conclude that in postmenopausal women, a single session of endurance exercise elicited a short-term, favorable decrease in triglycerides independent of initial blood cholesterol concentrations. However, LCATA and LPLA postexercise changes were influenced by preexercise cholesterol status.

Coronary heart disease (CHD) remains the leading cause of death for women in the United States (50). High blood total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), and low concentrations of high-density lipoprotein cholesterol (HDL-C) are related to risk for CHD development. The National Cholesterol Education Program (NCEP) defines desirable TC and LDL-C as <200 mg/dl and <140 mg/dl, respectively (11). In addition, HDL-C concentrations are often shown to be the strongest predictor of CHD in women; an independent, inverse association of HDL-C concentration and CHD mortality has been demonstrated (10). Moreover, the increased risk of CHD for postmenopausal women has been associated with an overall reduction in HDL-C and an elevation in LDL-C (32).

Endurance exercise may influence blood lipid profiles by altering intravascular enzyme activities. Increased lipoprotein lipase activity (LPLA) and decreased hepatic TG lipase activity (HLA) have been noted after exercise training (17, 24). In addition, increased lecithin-cholesterol acyltransferase (LCAT) activity (LCATA) and reductions in cholesterol ester transfer protein (CETP) concentrations have been reported (15, 42). Elevations in LPLA or LCATA with endurance training may reduce TG and reciprocally increase HDL-C (37). Additionally, reduced HLA or CETP, allowing slowed catabolism of HDL particles with endurance training, may enhance the overall accumulation of cholesterol in HDL subfractions (7). These favorable effects of exercise may contribute to an improvement of the lipid profile.

The majority of exercise and lipid research has been completed on men. Results of exercise training studies in women are inconsistent, with approximately one-half reporting beneficial effects on lipids (8, 16, 28, 30, 33, 34) and with others reporting the opposite or simply no change at all (27, 36, 40). It has been known for some time that blood lipids may respond favorably to a single session of exercise for up to 48 h and that this acute effect of exercise may account for a substantial portion of the benefit often measured in trained persons (17). Compared with studies in men, relatively few studies in women exist on the acute effects of a single session of exercise on lipids, and most include only trained, premenopausal women athletes competing in events of high intensity and long duration. The reported acute changes in lipids and lipoproteins in women include decreases in TC (18, 20, 21, 39, 41), LDL-C (20, 21, 39), and TG (20, 31, 41, 49), with elevations in HDL-C (21, 22, 31, 39, 41, 45) and either HDL-C subtype 3 (HDL3-C) (22, 31, 45) or HDL-C subtype 2 (HDL2-C) (39). Few acute exercise studies have included postmenopausal women, a population that could benefit from a lifestyle therapy to counter the increased lipid-related CHD risk known to accompany menopause.

It is not clear from the existing literature that women with elevated TC or LDL-C, those who would likely benefit most from beneficial changes in lipids, will respond to exercise interventions. In particular, acute changes in lipids, lipoprotein lipids, and lipid enzymes after a single session of exercise are not well characterized for postmenopausal women with NCEP-defined elevated blood cholesterol. Molecular rationale exists to suggest that exercise may have a different or blunted effect on hypercholesterolemic individuals; those with high-com

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pared with normal cholesterol may differ in lipid-regulating enzyme activities (3, 35, 46) and LDL-C receptor-mediated metabolism (9). Crouse and associates (12, 13) reported reductions in blood TC, LDL-C, and TG accompanied by increases in HDL-C and HDL_3-C concentrations in hypercholesterolemic men for up to 48 h after endurance exercise. In a follow-up study, Grandjean et al. (26) reported similar lipid findings in men coincident with elevated LPLA 24 h after exercise; CETP activity (CETPA), HLA, and LCATA were unchanged. The increases in HDL-C and HDL_2-C or HDL_3-C, along with reductions in TG concentrations, are similar to changes associated with physical activity in normcholesterolemic subjects (5, 17). To our knowledge, comparative studies in postmenopausal women with NCEP-defined normal and elevated cholesterol have not been published. Furthermore, the finding that postmenopausal women who continue to exhibit elevated blood TC and LDL cholesterol, despite regular exercise habits, suggests that they might be unresponsive to exercise therapy. It logically follows that they might also respond differently to acute exercise compared with those with normal cholesterol. Therefore, the purpose of this investigation was to compare and contrast the effects of a single session of exercise on blood lipids, lipoprotein lipids, and lipid enzyme activities between physically active women with high and normal blood cholesterol. We designed this study to test the hypothesis that women with NCEP-defined desirable cholesterol would demonstrate a blunted or no response, whereas those with elevated cholesterol would demonstrate a blunted or no response. The new information we present in this study has clinical relevance for the design of optimal intervention strategies to reduce CHD risk in postmenopausal women.

METHODS

Subjects

Subject recruitment strategies included advertisements placed in newspapers, local network broadcasts, and announcement flyers to departments at Texas A&M University. All methods and procedures were approved by the Texas A&M University Institutional Review Board for Human Subjects in Research. Women volunteers were prospectively recruited for the study if they met the following criteria: 1) were between 40 and 70 yr of age; 2) had either high cholesterol (HC; >200 mg/dl) or normal cholesterol (NC; ≤200 mg/dl) levels; 3) had been postmenopausal for at least 1 yr (natural or surgical); 4) were currently taking a form of hormone replacement therapy; and 5) were performing some type of aerobic exercise at a frequency of at least 2 times per week and duration of at least 20–30 min per session. Volunteers were generally healthy, not taking any medication known to alter lipid metabolism, and reportedly free of diagnosed cardiovascular disease, any contraindication to exercise, or any known metabolic disorder.

Fifty-four volunteers met the requirements and were asked to come to the laboratory for further assessment. The volunteers read and signed the institutionally approved informed consent form; they also were told that laboratory personnel would be blinded to the control group. Women who failed to maintain the prescribed exercise intensity and rate of caloric expenditure were disallowed to continue. A 5-ml blood sample was obtained from each volunteer to verify their cholesterol status. A total of 40 subjects (HC, n = 18; NC, n = 22) were initially selected and fulfilled the requirements for participation in the study. By the conclusion of the study, 15 subjects were eliminated from statistical analysis for the following reasons: 1) hypertriglyceridemia (2 subjects), 2) noncompliance with the dietary and physical activity instructions (4 subjects), 3) problems obtaining usable blood samples (4 subjects), 4) determined not to be on hormone replacement therapy (1 subject); 5) disqualification of hormone replacement therapy during the experimental period (1 subject); and 6) dropped out citing personal difficulty with the exercise protocol (2 subjects) and with the blood draw protocol (1 subject). Therefore, only data from 25 subjects (HC, n = 12; NC, n = 13) who successfully completed the study were statistically analyzed and reported herein. In a general meeting, selected subjects were familiarized with the equipment and the experimental protocol; they were also instructed on the dietary requirements of the study.

Dietary and Physical Activity Records

Self-reported dietary records were kept to assess the daily variation in each individual’s eating. All subjects were instructed in the use of the American Dietetic Association food exchanges (2) to standardize dietary documentation; instructions and advice were given by a trained dietician during a 3-day practice and the experimental session. For the investigation, subjects were asked to maintain dietary guidelines a total of 7 days, beginning 4 days before the baseline blood sample, and then until completion. All daily food records were analyzed for caloric consumption and nutrient proportion using the Minnesota Nutrition Data Systems (NDS, version 2.9, Food Data Base 10A, Nutrition Data Base 25, Minneapolis, MN) software. Physical activity, using a 7-day physical activity record (6), and dietary records were kept during the same duration of the experimental time line.

Demographics and Physiological Testing

Each subject underwent a physical examination by a cardiologist before testing. Physiological testing was completed at least 1 wk before the beginning of the experimental blood sampling and exercise session. Measurements included: 1) height and body weight, 2) percent body fat (29), and 3) waist and hip girth (1). Peak oxygen consumption (VO_2_peak) was assessed with an automated metabolic gas-analysis system (CPX/D Exercise Stress Testing System, Medical Graphics, Minneapolis, MN) during a maximal effort graded exercise test (GXT) on a motor-driven treadmill according to the Balke and Ware protocol (4). Heart rate and rhythm were monitored continuously from a 12-lead electrocardiogram. Ratings of perceived exertion and manual blood pressures were obtained during the last 30 s of every stage and at maximal exercise. The test was considered a maximal effort if at least two parameters for test termination criteria were met, according to published American College of Sports Medicine guidelines (1). All physiological measurements were consistently performed by the same skilled laboratory personnel.

Experimental Exercise Sessions

Subjects were asked not to engage in any planned exercise other than that prescribed as a part of this study at least 72 h before the experimental exercise session and until all blood sampling had been completed. The respiratory exchange ratio, treadmill work rate, heart rate, and peak oxygen consumption (l/min) at 70% of VO_2_peak were determined from the GXT data for each individual. From these data, the exercise duration required to expend 400 kcal of energy at 70% VO_2_peak was calculated for each subject. From these data, the exercise duration required to expend 400 kcal of energy at 70% VO_2_peak was calculated for each subject. From the experimental exercise session, each subject reported to the laboratory in the morning after a 12-h fast restricted to water only. After the subject performed a 3-min warm-up on the treadmill, a stopwatch was started, and the speed and grade were adjusted to the prescribed work rate calculated to elicit 70% of VO_2_peak. The prescribed rate of energy expenditure was confirmed by automated metabolic gas analysis, both at the initiation of exercise and at 15-min intervals throughout the exercise session. As necessary, adjustments were made in treadmill speed or grade to maintain the prescribed exercise intensity and rate of caloric expenditure.

Blood Sampling and Biochemical Analyses

Blood samples were obtained on 4 consecutive days for the measurement of lipid and lipoprotein lipid concentrations, and for lipid
enzyme activity levels. Detailed descriptions of our procedures and quality control for blood sampling, for lipid and lipoprotein lipid assays, and for LCATA and CETPA and HLA have been previously published (26). Briefly, blood was drawn for analysis 1 day before exercise (Pre) and then immediately (IPE), 24 h (24 HR), and 48 h (48 HR) after the 400-kcal experimental exercise session. As in previous studies, plasma lipid concentrations and lipid enzyme activities were adjusted for plasma volume shifts from the Pre measurement time point (13, 14, 26). Estradiol concentrations were determined in duplicate by 125I radioimmunoassay using a commercially available kit (Coat-A-Count Estradiol-6, Diagnostic Products, Los Angeles, CA). The repeated use of a trilevel control system (CON65 Multivalent Control Module, Diagnostic Products, Los Angeles, CA) during estradiol assay runs was used for quality control. Our intra-assay coefficient of variation for the estradiol assay was 1.82%.

Statistical Analysis

All initial measures of lipid and physiological variables were tested for normality before hypothesis testing. Log transformations were necessary to normalize the HDL-C and HDL2-C concentration distributions. The results of the subsequent statistical analyses were not altered after normalizing the distributions of these variables; thus results from the raw data are presented. Significance of baseline differences between the HC and NC groups for each of the physiological and Pre lipid variables were determined using ANOVA. Simple linear regression was utilized to determine whether baseline TC would predict changes in variables from baseline to 24 h after exercise. Data for daily caloric and nutrient intake, as well as for daily physical activity, were analyzed using a 2 (group) × 7 (day) repeated-measures analysis of variance. Plasma TC, free cholesterol, TG, LDL-C, HDL-C, HDL2-C, HDL3-C concentrations, as well as LPLA, HLA, LCATA, and CETPA data, were analyzed with group (HC and NC) by time (Pre, IPE, 24 HR, 48 HR) two-factor factorial ANOVA performed using the Huynh-Feldt correction factor when the sphericity assumption held; and the multivariate test statistic was performed utilizing Wilks’ lambda.

Dietary and Physical Activity

Dietary records were analyzed for 1) total daily caloric intake (in kcal); 2) nutrient composition as a percentage of the total daily caloric intake (%carbohydrate, %fat, and %protein); and 3) total daily grams of carbohydrate, fat, and protein. No significant group differences or changes as a function of the day measured were found for any of the dietary variables. Thus the dietary intakes of the women were stable throughout the experimental period, which verifies their compliance with our dietary prescription. Analysis of the physical activity records showed significantly greater energy expenditure for all women on the sixth recording day, the day of the experimental exercise session, a finding consistent with the design of the study.

Exercise and Plasma-Derived Variables

Experimental exercise session and changes in plasma volume. As designed, the average exercise energy expenditure for the HC and NC groups was 400 ± 0.43 and 400 ± 0.09 kcal, respectively; the combined range was 399–404 kcal. The average exercise duration to complete this volume of exercise was 85.1 ± 6.9 min for the HC group and was 81.1 ± 3.8 min for the NC group. The range was 59–147 min for combined groups. Our statistical analysis of these data showed that exercise energy expenditure and exercise duration were the same for women in the NC and HC groups. Changes in plasma volume were significant (P < 0.01) between IPE and both 24 HR and 48 HR time points in both groups of women. Therefore, lipid, lipoprotein lipid, and lipid enzyme activities were corrected for changes in plasma volume across time, and all results are reported as plasma volume-corrected values.

Lipids, lipoprotein lipids, enzymes, and CETP. Blood lipid and lipoprotein lipid concentrations at the IPE, 24 HR, and 48 HR time points did not depend on preexercise cholesterol status of the women. Our results show that the blood lipid and lipoprotein-lipid responses to exercise were the same for women in the HC and NC groups. Regardless of group membership, the primary lipid change noted in the women was a postexercise reduction (−8.5%) in TG concentration 24 h after exercise. Although still below Pre values at 48 HR, the reduc-
HDL2-C, or HDL3-C (Table 3). The postexercise LCATA and values were noted for blood concentrations of TC, FC, LDL-C, (Fig. 2). No significant changes over time from preexercise respective patterns of the NC women (Figs. 3 and 4). Exercise LPLA response patterns in the HC women differed from the not significantly different (P0.05). Missing data due to technical difficulties and the statistical procedure for handling missing values (SPSS, version 11.5) resulted in smaller subject numbers in each group for the enzyme variables.

Values are means ± SE; n, no. of subjects. TC, total cholesterol; FC, free cholesterol; TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; HDL2-C, HDL-C subtype 2; HDL3-C, HDL-C subtype 3; LPLA, lipoprotein lipase activity; HLA, hepatic lipase activity; LCATA, lecithin-cholesterol acyltransferase activity; CETPA, cholesterol ester transfer protein activity. *Significant differences between HC and NC groups, P < 0.05. Values are means ± SE. Pairwise comparisons approached significance between IPE and 24 HR (P = 0.083).

**DISCUSSION**

The primary purpose of this investigation was to compare and contrast the effects of a single session of exercise on blood lipids, lipoprotein lipids, and lipid enzyme activities between physically active women with NCEP panel III-defined high and normal blood cholesterol. To address this purpose, we recruited two groups of women, one with high and another with normal blood cholesterol, but who were otherwise similar in demographic and physiological characteristics known to influence blood lipoproteins (44, 47) (Tables 1 and 2).

**Cholesterol Status and Lipid and Lipoprotein Lipid Exercise Response**

The women in our study with NCEP-defined elevated blood TC and LDL-C concentrations before exercise exhibited lipid and lipoprotein lipid response patterns after exercise that were not different from those in women with normal blood cholesterol. Regression analysis provided further evidence that pre-exercise TC concentrations did not influence the postexercise lipid profiles in the women in our study. We were not able to predict from preexercise TC values any 24-h postexercise changes in lipid and lipoprotein lipid concentrations. Thus the preexercise cholesterol status of the women in our study did not affect their postexercise lipid and lipoprotein profiles, a finding consistent with reports for men with similar lipid profiles (26).

With respect to specific exercise effects, there were no statistically significant changes in TC or LDL-C concentrations after the 400-kcal endurance exercise session in either group of women in our study. These findings were consistent with the results of several published studies (23, 31, 39, 43, 49). Others have shown that TC and LDL-C concentrations remained stable in untrained postmenopausal (25) and in untrained mildly obese women (31) up to 48 h after an acute exercise session at an intensity of 60–70% of maximal oxygen uptake. Furthermore, exercise intensity appears not to influence the postexercise lipid response (39, 49). In contrast, significant decreases in TC and LDL-C concentrations have been noted in trained women after a prolonged, exhaustive exercise session, such as running a marathon (18, 20, 21, 41). Additionally, TC and LDL-C concentrations were reduced immediately after exercise in hypercholesterolemic men (12, 13, 26). These data taken with ours suggest that gender and volume of exercise may influence the acute lipid response to exercise.

The most noteworthy finding related to lipids in our study was the beneficial 8.5% reduction in TG concentrations in both HC and NC women 24 h after exercise. In prior studies, significant reductions in blood TG concentrations after exercise...
have been reported in men and in women with some degree of regularity (17, 19, 20, 31, 41, 49). After high-volume, ultraendurance exercise, TG concentrations were reduced up to 70% (20, 41). Furthermore, after treadmill walking at an intensity ranging from 30 to 60% of maximal oxygen consumption, premenopausal women exhibited a decrease in TG concentrations ranging from 15 to 26% (19, 49). In contrast, some have failed to find significant reductions in blood TG concentrations in pre- and postmenopausal women after exercise (22, 39, 45). Increases in blood TG concentrations after exhaustive, high volume exercise have also been reported in premenopausal women (21, 18, 39). Reasons for these divergent findings are not readily apparent, but they may be related to interstudy subject differences in training status, menopausal and hormonal status, diets, and volume of exercise performed.

To the best of our knowledge, we are the first to report a blood TG reduction after a single session of endurance exercise in postmenopausal women with NCEP panel III-defined elevated cholesterol. Comparable TG reductions have been reported in hypercholesterolemic sedentary and trained men 24–48 h after a single session of endurance exercise (12, 13, 26). Our present results confirm postexercise reductions in TG concentrations, even in women with elevated blood cholesterol.

On the basis of previous research, we expected either no change or an increase in HDL-C concentration after exercise (12, 13, 26, 39). Although the repeated-measures ANOVA for differences in the HDL-C profile after exercise for the women in our study was statistically significant (P < 0.04), post hoc pairwise comparisons were not (P > 0.05). Thus it is with caution that we interpret our HDL-C postexercise data. As shown in Fig. 2, HDL-C concentration first fell nearly 5% (~3 mg/dl) IPE, and then it rose to return to preexercise values by 24 and 48 h after exercise (P = 0.083). The IPE-to-24 HR increase in HDL-C concentration was primarily due to a nearly 20% increase (~2.3 mg/dl) in the HDL2-C subfraction, whereas HDL3-C concentrations rose only 1% (~0.8 mg/dl) during this same 24-h period (Table 3). Although not statistically significant, the average HDL2-C concentration was still elevated 16% over baseline at the 24-h measurement period, whereas HDL3-C was ~2% lower. Our results support those of Pronk et al. (39), who reported increased HDL2-C concentra-

Table 3. Nonsignificant variable results across time

<table>
<thead>
<tr>
<th>Lipids</th>
<th>Pre (n = 25)</th>
<th>IPE (n = 25)</th>
<th>24 HR (n = 25)</th>
<th>48 HR (n = 25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC, mg/dl</td>
<td>198.8±6.7</td>
<td>193.6±8.5</td>
<td>194.4±7.0</td>
<td>193.8±7.6</td>
</tr>
<tr>
<td>FC, mg/dl</td>
<td>61.2±1.8</td>
<td>58.2±2.2</td>
<td>60.2±2.0</td>
<td>60.1±2.0</td>
</tr>
<tr>
<td>LDL-C, mg/dl</td>
<td>108.5±6.6</td>
<td>106.0±7.4</td>
<td>106.1±6.4</td>
<td>105.2±6.8</td>
</tr>
<tr>
<td>HDL2-C, mg/dl</td>
<td>13.7±1.9</td>
<td>11.3±1.2</td>
<td>13.9±1.8</td>
<td>13.1±1.7</td>
</tr>
<tr>
<td>HDL3-C, mg/dl</td>
<td>51.1±1.6</td>
<td>50.7±1.8</td>
<td>51.1±1.3</td>
<td>50.7±1.5</td>
</tr>
<tr>
<td>Lipid enzymes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HLA, μmol free fatty acid·ml⁻¹·h⁻¹</td>
<td>13.7±1.96</td>
<td>12.62±1.74</td>
<td>13.02±1.82</td>
<td>13.81±2.10</td>
</tr>
<tr>
<td>CETPA, %cholesterol ester transferred/4 h</td>
<td>19.40±1.65</td>
<td>21.56±1.66</td>
<td>19.46±1.50</td>
<td>19.69±1.81</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. Pre, 24 h before an exercise session; IPE, immediately after an exercise session; 24 HR, 24 h after an exercise session; 48 HR, 48 h after an exercise session. Missing data due to technical difficulties and the statistical procedure for handling missing values (SPSS, version 11.5) resulted in smaller subject numbers in each group for HLA and CETPA.

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Fig. 3. Group membership influenced lecithin-cholesterol acyltransferase activity (LCATA) after exercise (P = 0.032). LCATA values at each of the time points were as follows: 1) high cholesterol (HC): Pre = 3.99 ± 0.46 μmol cholesterol est (cholesterol est)·ml⁻¹·h⁻¹, IPE = 4.05 ± 0.48 μmol cholesteryl est·ml⁻¹·h⁻¹, 24 HR = 4.27 ± 0.54 μmol cholesteryl est·ml⁻¹·h⁻¹; 2) normal cholesterol (NC): Pre = 4.78 ± 0.46 μmol cholesteryl est·ml⁻¹·h⁻¹, IPE = 4.35 ± 0.48 μmol cholesteryl est·ml⁻¹·h⁻¹, 24 HR = 5.12 ± 0.54 μmol cholesteryl est·ml⁻¹·h⁻¹. Values are means ± SE.

Fig. 4. Lipoprotein lipase activity (LPLA) response to exercise was dependent on group membership (P = 0.008). LPLA values at each of the time points were as follows: 1) HC: Pre = 16.27 ± 2.41 μmol free fatty acids (FFA)·ml⁻¹·h⁻¹, IPE = 19.06 ± 2.16 μmol FFA·ml⁻¹·h⁻¹, 24 HR = 13.19 ± 2.04 μmol FFA·ml⁻¹·h⁻¹, 48 HR = 12.80 ± 2.68 μmol FFA·ml⁻¹·h⁻¹; 2) NC: Pre = 13.39 ± 2.25 μmol FFA·ml⁻¹·h⁻¹, IPE = 14.72 ± 2.02 μmol FFA·ml⁻¹·h⁻¹, 24 HR = 13.48 ± 2.63 μmol FFA·ml⁻¹·h⁻¹, 48 HR = 14.55 ± 2.50 μmol FFA·ml⁻¹·h⁻¹. Values are means ± SE.
tions after exercise in postmenopausal women. In contrast, others have found HDL3-C concentrations to be elevated after exercise in premenopausal women (22, 31, 45). These results taken together suggest that menopausal status may influence the postexercise cholesterol distribution among the HDL subfractions. More research is needed to confirm this hypothesis.

The postexercise HDL-C and HDL-subfraction profiles we report here for postmenopausal women differ from those in men, where HDL-C and the HDL3-C subfraction concentrations were increased 24 and 48 h after a similar session of endurance exercise (12, 13, 26). We have no direct comparative data from men in our present study to explain this apparent gender difference. However, it is noteworthy that the preexercise average HDL-C concentration in the women in our study (~65 mg/dl) was 44–55% higher than the baseline averages of men in similarly designed studies [45 mg/dl in Crouse et al. (13) and 42 mg/dl in Grandjean et al. (26)]. Hence, a further increase after exercise in women with such relatively high HDL-C concentrations would be unlikely.

**Cholesterol Status, Exercise, Enzymes, and CETP**

The response profiles for LCATA and LPLA after exercise for these two lipid regulatory enzymes were significantly different in the HC compared with the NC women (Figs. 3 and 4). The primary difference in LCATA profiles between the two groups was a 9% fall from the preexercise value at the IPE measurement period in the NC subjects compared with a respective 1.5% increase in the HC subjects (Fig. 3). With regard to LPLA, the HC group exhibited a 17% increase at IPE, followed by a 19% reduction by 24 HR compared with the Pre value, and a further reduction to 21% below Pre by 48 HR. In the NC group, the LPLA increased only 10% IPE, and remained slightly elevated through the 48-h period (~9%). We are aware of no other published data for women showing this differential LPLA effect related to preexercise cholesterol status. Published research generally shows that LCATA is relatively resistant to acute changes with exercise unless a relatively large volume of exercise is completed (24). We are currently unable to explain the 9% drop in LCATA immediately after exercise in the NC women in our study. However, the increase in HDL3-C coupled with LCATA rise at 24 h in both HC and NC women is consistent with the hypothesized role of LCAT in reverse cholesterol transport (24).

Comparative studies in men have shown that, regardless of preexercise cholesterol status, LPLA increases after endurance exercise and remains elevated for up to 48 h; this increase is accompanied by a decrease in circulating TG (26). This raises the issue of different enzyme responses to exercise in men compared with women. In this regard, Perreault et al. (38) reported that the acute LPLA response after exercise is gender specific, increasing in men but not in premenopausal women. Our results additionally show that LPLA may actually decrease after exercise in postmenopausal women who have elevated cholesterol. The decrease in LPLA along with a decrease in blood TG noted in our study is not consistent with the hypothesized role of LPLA in the regulation of circulating lipids (37, 48). This suggests that TG concentrations may be modulated by factors other than LPL in postmenopausal women.

The role of CETP in intravascular remodeling of circulating very low-density lipoproteins, LDL, and HDL is well established. To our knowledge, we are the first to show that CETP is not changed after exercise in postmenopausal women, regardless of their preexisting cholesterol status. Furthermore, postheparin HLA was not altered with exercise in the women in our study. Both of these findings are consistent with the majority of published research showing no change after exercise in either of these lipid regulatory proteins (24, 26).

In summary, the primary finding in our study in physically active, postmenopausal women with NCEP panel III-defined high and normal blood cholesterol was that TG concentrations were reduced 24 h after performing 400 kcal of treadmill exercise, regardless of the women’s preexercise cholesterol status. Other lipid risk markers were essentially unchanged by exercise, but cholesterol status did influence the manner in which LPLA and LCATA responded to the exercise stimulus. These enzyme findings are novel and require additional research to verify. Thus practitioners recommending exercise for coronary artery disease risk reduction in postmenopausal women, even for women with elevated cholesterol, can be confident that a single session of endurance exercise will not worsen the lipid profile and would likely have a favorable influence on circulating lipid risk markers.

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