

Influence of cholesterol status on blood lipid and lipoprotein enzyme responses to aerobic exercise

PETER W. GRANDJEAN,¹ STEPHEN F. CROUSE,² AND J. JAMES ROHACK³

¹Department of Health and Human Performance, Auburn University, Auburn University, Alabama 36849–5323; and ²Applied Exercise Science Laboratory and ³College of Medicine, Texas A&M University, College Station, Texas 77843

Received 24 September 1999; accepted in final form 20 March 2000

Grandjean, Peter W., Stephen F. Crouse, and J. James Rohack. Influence of cholesterol status on blood lipid and lipoprotein enzyme responses to aerobic exercise. *J Appl Physiol* 89: 472–480, 2000.—To compare postexercise changes in plasma lipids and lipoprotein enzymes in 13 hypercholesterolemic (HC) and 12 normocholesterolemic men [total cholesterol (TC) 252 ± 5 vs. 179 ± 5 mg/dl], fasting blood samples were obtained 24 h before, immediately, 24, and 48 h after a single bout of treadmill walking (70% peak O₂ consumption, 500 kcal expenditure). Significant findings ($P < 0.05$ for all) for plasma volume-adjusted lipid and enzyme variables were that TC, low-density-lipoprotein cholesterol, and cholesterol ester transfer protein activity were higher in the HC group but did not influence the lipid responses to exercise. Across groups, TC was transiently reduced immediately after exercise but returned to baseline levels by 24 h postexercise. Decreases in triglyceride and increases in high-density-lipoprotein cholesterol (HDL-C) and HDL₃-C were observed 24 h after exercise and lasted through 48 h. Lipoprotein lipase activity was elevated by 24 h and remained elevated 48 h after exercise. HDL₂-C, cholesterol ester transfer protein activity, hepatic triglyceride lipase, and lecithin: cholesterol acyltransferase activities did not change after exercise. These data indicate that the exercise-induced changes in HDL-C and triglyceride are similar in HC and normocholesterolemic men and may be mediated, at least in part, by an increase in lipoprotein lipase activity.

hypercholesterolemia; lipoproteins; lipoprotein lipase activity; lecithin: cholesterol acyltransferase activity; cholesterol ester transfer protein activity

REGULAR PHYSICAL EXERCISE has been adopted as part of an overall strategy to normalize lipid profiles and reduce coronary heart disease risk in otherwise healthy hypercholesterolemic individuals (18, 44). The basis of such a strategy stems primarily from the exercise training-induced lipid and lipoprotein changes observed in normolipidemic individuals. Indeed, a majority of the investigators who have studied normolipidemic groups have reported reductions in plasma triglyceride (TG) concentrations and increases in high-density-lipoprotein (HDL) cholesterol (HDL-C) after exercise training (17). In addition, when exercise is

accompanied by a loss of body mass or body fat in normolipidemic subjects, total cholesterol (TC) and low-density-lipoprotein cholesterol (LDL-C) concentrations may be reduced (17). The few studies reporting on lipid and lipoprotein responses to exercise training in hypercholesterolemic men indicate that reductions in TC and TG, as well as elevations in HDL-C and HDL₂-C, are possible (44, 45). These results may be somewhat similar to those reported in normolipidemic subjects; however, lipid responses to exercise training in hypercholesterolemic men have been inconsistent, and, at present, there are too few studies from which to draw general conclusions.

Regular aerobic exercise may influence blood lipid profiles by modifying the activities of intravascular enzymes and transfer proteins (6, 17). Elevations in the activities of lipoprotein lipase (LPLa) (31) and lecithin: cholesterol acyltransferase (LCATa) (16) have been shown after exercise training, as have reductions in hepatic TG lipase activity (HTGLa) (40). In addition, a reduction in cholesterol ester (CE) transfer protein (CETP) concentration, which is closely related to CETP activity (CETPa), has been demonstrated after exercise training (42). Greater LPLa or LCATa brought about by exercise training may reduce TG concentrations and facilitate an increase in HDL-C (47). Similarly, an exercise-induced suppression of HTGLa or CETPa may slow the catabolism of HDL particles, thereby enhancing the accumulation of cholesterol in all HDL subfractions (8, 20).

The favorable blood lipid changes that are generally thought to be a result of exercise training may be partially attributed to the short-term lipid modifications that can occur after a single session of aerobic exercise (6, 17). For example, the transient changes observed after one exercise bout have included decreases in TC, LDL-C, and TG and elevations in HDL-C in both the HDL₂ and HDL₃ subfractions (16, 25, 29, 33). As with exercise training, an increase in LPLa (30, 31) and LCATa (16) has been observed after one session of exercise. HTGLa has been reported as decreased (25) or unchanged (30), and, recently, de-

Address for reprint requests and other correspondence: P. W. Grandjean, Dept. of Health and Human Performance, 2050 Beard-Eaves Memorial Coliseum, Auburn Univ., Auburn Univ., AL 36849–5323 (E-mail: grandpw@auburn.edu).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

creases in CETP concentrations have been reported after one bout of prolonged, strenuous exercise (46).

Relatively little attention has been directed toward the lipid and lipoprotein responses to a single session of exercise in hypercholesterolemic men. Crouse and colleagues (12) have reported what may be unique elevations in TC and LDL-C in physically inactive hypercholesterolemic men after one session of stationary cycling. These same researchers demonstrated that exercise training can modify the postexercise changes in TC and LDL-C of hypercholesterolemic men, yet the TC and LDL-C responses to a single bout of exercise in these subjects remained different from that reported for normocholesterolemic individuals (11). It is possible that unique exercise-induced lipid changes observed in hypercholesterolemic men are influenced by distinct lipoprotein enzyme differences from their normocholesterolemic counterparts. For instance, greater CETPa (4, 49), greater HTGLa (37), and lower LPLa (21) in some hypercholesterolemic subjects have been documented. Unfortunately, the lipid and lipoprotein responses to exercise training or after a single bout of exercise cannot be directly compared with those of normolipidemic individuals. First, normolipidemic individuals have not been included in any of the studies that examined either the training-induced changes or the short-term changes in the hypercholesterolemic subjects. Second, no study to date has examined the intravascular lipoprotein enzyme responses to a single session of exercise in hypercholesterolemic individuals. By comparing normo- and hypercholesterolemic groups, unique blood lipid and lipoprotein enzyme responses to exercise can be established, and the role of exercise in the management of some hyperlipidemias may be more clearly defined. Therefore, the purpose of this investigation was to compare blood lipid, lipoprotein lipid, LPLa, HTGLa, LCATa, and CETPa responses to a single session of aerobic exercise between physically inactive normo- and hypercholesterolemic men.

METHODS AND PROCEDURES

Subjects

Adult male volunteers from Brazos County, Texas, were recruited by posted flyers, advertisements in local newspapers, presentations to civic organizations, and appearances on local television talk shows. All volunteers were initially screened by phone or personal interviews and considered for the study if they were between 35 and 55 yr of age, apparently healthy, and physically inactive. Volunteers were considered healthy if they were not presently taking any medication known to affect lipid metabolism and reported being free of metabolic disorders, diagnosed cardiovascular disease, and any contraindication to exercise. Volunteers were characterized as physically inactive if they participated in less than two exercise sessions per week and each exercise session lasted <20 min.

Seventy-five volunteers who met the above requirements visited our laboratory, read and signed an institutionally approved informed consent, and completed a health history questionnaire. On this same visit, height and mass were

measured, body composition was estimated via seven-site skinfold (2), and a 5-ml blood sample was obtained from each volunteer to determine TC and TG status.

Twenty-eight subjects with TG concentrations <200 mg/dl and either hypercholesterolemia (HC; TC >240 mg/dl, $n = 13$) or normocholesterolemia (NC; TC <200 mg/dl, $n = 15$) fulfilled the requirements for entry into the study. All subjects were selected for similarities in their age, body mass, and relative body fat, because these factors are known to influence plasma lipid and lipoprotein concentrations (6). Two NC subjects were subsequently excluded from this study after it was determined that they reported for the experimental blood sampling in a postprandial state. In addition, all plasma samples for one NC subject were found to be unreliable for the measurement of several variables of interest due to difficulty in blood sampling. Therefore, 25 subjects (HC: $n = 13$, NC: $n = 12$) were included in the final data analysis.

Procedures

General protocol. All subjects underwent a physical examination, conducted by a cardiologist, and completed preliminary physiological testing. Within 2 wk of physiological testing, subjects reported to the laboratory on 4 consecutive days for experimental blood sampling in association with a single bout of exercise. Self-reported dietary records were used to assess the daily variation in each subject's eating habits over the experimental blood-sampling period.

Preliminary physiological testing. Peak oxygen consumption ($\dot{V}O_{2\text{ peak}}$) and work rate were determined from a standardized graded exercise test performed on a motor-driven treadmill (model Q-65, Quinton Instrument, Seattle, WA) (9). Throughout the test, heart rate and rhythm were monitored continuously by using a 12-lead electrocardiogram (model Q-4100, Quinton Instrument), blood pressure was determined manually, and ratings of perceived exertion were obtained during the last 30 s of every stage and at maximal exercise. Respiratory gas exchange [O_2 consumption ($\dot{V}O_2$) and CO_2 production] was measured on a breath-by-breath basis and averaged over 30-s intervals by using an automated gas analysis system (CPX/D Exercise Stress Testing System, Medical Graphics, Minneapolis, MN). The test was considered a maximal effort if two of the following criteria were met: the achieved maximum heart rate was within 10 beats/min of the individual's age-predicted maximum, the respiratory exchange ratio (RER) was >1.1, the rating of perceived exertion was ≥ 18 , or a plateau for $\dot{V}O_2$ was achieved despite a further increase in workload (2).

Hydrostatic weighing was used to estimate body density and percent fat (26). Waist and hip girth measurements were obtained, and a waist-to-hip ratio was calculated to estimate regional body fat distribution (2).

Experimental exercise session. The $\dot{V}O_2$ (l/min) and RER at 70% of $\dot{V}O_{2\text{ peak}}$ were obtained from the graded exercise test data and used to estimate the exercise duration needed to elicit an energy expenditure of 500 kcal. The kilocalorie equivalent was determined from the RER, and the rate of energy expenditure was calculated by multiplying the kilocalorie equivalent by the corresponding $\dot{V}O_2$ (l/min). Exercise duration was estimated by dividing 500 kcal by the calculated rate of energy expenditure.

After a brief warm-up period, the treadmill speed and grade were increased to meet the predicted intensity of 70% of $\dot{V}O_{2\text{ peak}}$ for each subject. Respiratory gas-exchange data and exercise heart rates were obtained initially and at 15-min intervals throughout the exercise session to verify the subject's exercise intensity and rate of energy expenditure.

The total energy expenditure and the remaining exercise time were calculated from the respiratory gas exchange data at each interval. Adjustments were made in either speed or grade to maintain the prescribed exercise intensity (2). The average exercise intensity for the HC and NC groups was 67.6 ± 0.9 and $71.2 \pm 1.6\%$, respectively. The energy expenditure for the HC group was 504 ± 2 kcal, whereas the NC group spent 503 ± 5 kcal of energy. No differences were determined between groups for either exercise intensity or energy expenditure.

Blood sampling. Blood samples were obtained 24 h before (baseline), immediately after, 24 h after, and 48 h after the experimental exercise session. Each subject reported to the laboratory at approximately the same time each morning after a 12-h fast in which water was allowed ad libitum. All blood samples were obtained from an antecubital vein by using a Teflon catheter (no. 3828781 Angiocath, Becton Dickinson, Franklin Lakes, NJ) and multiple-sample vacutainer system (no. 7210, 22 gauge \times 1 in., Becton Dickinson). First, blood was drawn into two chilled 10-ml vacutainer tubes containing 10.5 mg Na-EDTA (no. 310547, Sherwood Medical, St. Louis, MO) and immediately placed on ice. Next, 75 IU/kg heparin (1,000 IU/ml, Elkins-Sinn, St. Louis, MO) were administered and allowed to circulate for 10 min to release endothelial-bound lipoprotein lipase (51). A "postheparin" blood sample was drawn into a chilled vacutainer containing 143 USP sodium-heparin (no. 320751, Sherwood Medical). Plasma from pre- and postheparin blood was isolated by centrifugation at 1,500 g for 20 min at 4°C, and 0.01% NaN₃ solution was introduced into all plasma samples (4 μ l/ml plasma) for preservation. Aliquots of pre- and postheparin plasma were sealed separately in 2-ml cryovial tubes and stored at -70°C for later analysis.

Biochemical analysis. Before freezing, HDL and HDL₃ subfractions were separated from fresh aliquots of plasma as described by Warnick and Albers (52) and Gidez et al. (24), respectively. Plasma samples were analyzed enzymatically for TC, free cholesterol (FC), HDL-C, HDL₃-C, and TG concentrations (1, 10). HDL₂-C was calculated as the difference between HDL-C and HDL₃-C. LDL-C was calculated from TC, TG, and the HDL-C (23). Total plasma lipase activity and HTGLa were assayed according to Krauss et al. (32) and Belfrage and Vaughn (5) with modifications described by Thompson et al. (51). The rate of cholesterol esterification, an estimate of LCATa, was assayed as described by Stokke and Norum (43). CETPa was determined according to the methods described by Tato et al. (49). Measures of hematocrit and hemoglobin were determined from fresh blood samples and used to estimate changes in plasma volume relative to the baseline blood sample (14). All plasma lipid concentrations and enzyme activities were then adjusted for the estimated plasma volume changes that occurred over the blood sampling period.

The order in which subject plasma samples were analyzed was randomly selected before each biochemical analysis. Interassay variation was avoided by analyzing all samples from each subject within a single assay run. Average absorbances in all spectrophotometric analyses were calculated from replicate measurements within an absorbance range of 0.01 nm. When differences between replicate absorbances were >0.01 nm, all four samples for the subject were reanalyzed. Absorbances for normal and abnormal control plasma were determined at regular intervals throughout each assay run. Replicate measurements of radioactivity within a range of 10% were accepted for enzyme assays. When differences between replicate counts per minute were $>10\%$, all samples for the subject were reanalyzed. Control samples were included at

regular intervals throughout each assay run. Coefficients of variation (CVs) for spectrophotometric and radiolabeled assays were calculated from the control absorbances and counts per minute, respectively, and used as indicators of inter- and intra-assay reliability.

The interassay CVs determined from control serum measured with each assay run were as follows: TC, 2.42%; HDL-C, 2.40%; HDL₃-C, 1.08%; TG, 3.75%; total plasma lipase activity, 4.53%; HTGLa, 11.74%; LCATa, 5.47%; and CETPa, 3.30%. There was no interassay CV for FC, as all samples were analyzed in a single assay run. Intra-assay CVs, determined from control serum measured multiple times within each assay run were as follows: TC, 1.08%; FC, 1.38%; HDL-C, 1.53%; HDL₃-C, 1.88%; TG, 1.35%; total plasma lipase activity, 4.39%; HTGLa, 4.16%; LCATa, 5.37%; and CETPa, 2.97%.

Diet records. Subjects recorded their diet over a 3-day period (2 weekdays and 1 weekend day) before data collection. The food records were analyzed for caloric intake and nutrient composition, and subjects were provided with an individualized diet plan based on their 3-day dietary report. Subjects were asked to adhere to their individualized diet plan and record their dietary intake starting 4 days before the baseline blood sample and continuing until they completed the experiment. All daily food records were analyzed for caloric intake and nutrient composition by using the Minnesota Nutrition Data Systems software (version 2.8, Food Database 10A, Nutrition Database 25, Minneapolis, MN).

Statistical analysis. Baseline differences between HC and NC groups for the physiological, lipid, and enzyme variables were determined by using one-way ANOVA. The exercise data were analyzed by using a 2×4 (group \times time) ANOVA with repeated measures on the second factor. The lipid-dependent variables of interest were plasma volume-adjusted concentrations of TC, FC, TG, LDL-C, HDL-C, HDL₂-C, and HDL₃-C. LPLa, HTGLa, LCATa, and CETPa were also adjusted for shifts in plasma volume and analyzed in the same manner. Dietary data were analyzed for daily caloric intake and composition of fats, carbohydrates, protein, and cholesterol by using a 2×7 (group \times time) ANOVA with repeated measures on the second factor. This investigation was exploratory in nature; therefore, the comparison-wise error rate was set at $P < 0.05$. Each test for significance was conducted independently; therefore, it is recognized that the experimentwise error rate may be somewhat higher than $P < 0.05$. Duncan's new multiple range test was used to determine significant time effects where appropriate.

Relationships among the physiological characteristics, baseline plasma lipid and enzyme values, and changes in the dependent variables with exercise were determined by using Pearson product-moment correlation coefficients. Change scores for the dependent variables in the exercise data set were calculated as 24 h postexercise minus baseline and 48 h postexercise minus baseline. All data were analyzed by using the Statistical Analysis System (SAS for Windows, version 6.11, SAS Institute, Cary, NC).

RESULTS

Our purpose was to examine the influence of plasma cholesterol status on the lipid and lipoprotein enzyme responses to a single bout of exercise in physically inactive men. Every effort was made to recruit subjects who were of similar age, weight, and percent body fat to reduce the impact of these factors on the potentially different group lipid responses to exercise. As pre-

Table 1. HC and NC group data at baseline

	HC Group	NC Group
Age, yr	46 ± 2	43 ± 1
Height, cm	174.8 ± 1.67	178.9 ± 2.32
Weight, kg	89.1 ± 5.1	82.7 ± 4.3
Body fat, %	26 ± 2	22 ± 2
W/H	0.94 ± 0.01	0.91 ± 0.02
$\dot{V}O_{2max}$, l/min	2.8 ± 0.1	2.9 ± 0.2
$\dot{V}O_{2max}$, ml · kg ⁻¹ · min ⁻¹	31.3 ± 1.0	35.4 ± 1.6*
TC, mg/dl	252 ± 5*	179 ± 5
CE, mg/dl	66 ± 1*	50 ± 2
FC, mg/dl	186 ± 5*	129 ± 4
LDL-C, mg/dl	179 ± 5*	109 ± 5
TG, mg/dl	155 ± 13	132 ± 17
HDL-C, mg/dl	41 ± 2	43 ± 3
HDL ₂ -C, mg/dl	8 ± 1	9 ± 2
HDL ₃ -C, mg/dl	33 ± 2	34 ± 2
LPLa, μmol FFA · ml ⁻¹ · h ⁻¹	14.2 ± 1.3	11.9 ± 1.2
HTGLa, μmol FFA · ml ⁻¹ · h ⁻¹	21.3 ± 2.0	21.0 ± 3.2
LCATa, μmol chol est · l ⁻¹ · h ⁻¹	76.8 ± 8.2	60.6 ± 7.1
CETPa, %CE transferred/4 h	34.4 ± 1.7*	28.2 ± 2

Values are means ± SE. HC, hypercholesterolemic group, n = 13; NC, normocholesterolemic group, n = 12; W/H, waist circumference-to-hip circumference ratio; $\dot{V}O_{2max}$, maximal oxygen uptake; TC, total plasma cholesterol; FC, free cholesterol; CE, cholesterol ester; LDL-C, low-density-lipoprotein cholesterol; TG, triglyceride; HDL-C, high-density-lipoprotein cholesterol; LPLa, lipoprotein lipase activity in μmol free fatty acid (FFA) formed per ml per h; HTGLa, hepatic triglyceride lipase activity; LCATa, lecithin: cholesterol acyltransferase activity in μmol cholesterol esterified (chol est) per liter per h; CETPa, cholesterol ester transfer protein activity expressed as a percentage of CE transferred from a common donor [³H-HDL₃-CE (75 mg/dl)] to a common acceptor [combined very-low and low-density lipoprotein fractions (187 mg/dl)] in a 20:1 acceptor-to-donor ratio. *Significant difference between HC and NC group, P < 0.05 for all.

sented in Table 1, no differences were found between groups for any of the physiological variables except relative $\dot{V}O_{2peak}$. As expected, the HC group exhibited greater concentrations of TC, LDL-C, FC, and CE than did their NC counterparts. CETPa was also higher in the HC group (see Table 1). Significant relationships between selected physiological variables and baseline lipid and enzyme activities are shown in Table 2.

No group × time interactions were observed for any of the plasma lipid or enzyme variables, indicating that cholesterol status did not influence the magnitude or direction of lipid and enzyme responses to exercise (Table 3). A significant time effect was observed for the estimated shifts in plasma volume, TC, TG, HDL-C, HDL₃-C, and LPLa. The exercise-induced changes in

plasma volume and the plasma lipid and enzyme variables are illustrated as combined group means in Figs. 1-5.

No group × time interactions or group effects were found for any of the dietary variables. A significant time effect was observed for daily caloric intake, grams of fat, protein, and cholesterol. However, no significant differences in the daily nutrient composition of the diet were observed when nutrients were expressed as a percentage of the daily caloric intake. The average daily caloric intake and the nutrient composition of the diet are presented in Table 4.

DISCUSSION

To our knowledge, this study is the first to compare the lipid and lipoprotein enzyme changes that occur in normo- and hypercholesterolemic men after a single session of exercise. Our laboratory has previously shown, as have others, that one bout of exercise can favorably modify blood lipid concentrations (12, 17). However, the primary and unique finding of this investigation is that preexisting cholesterol status does not influence acute blood lipid and lipoprotein enzyme responses to exercise in sedentary men.

The hypercholesterolemic men had baseline TC and LDL-C values in the 85th percentile for their age (38). Conversely, the average TC and LDL-C concentrations in the normocholesterolemic men placed them in the 15th percentile (38). The remaining components of the lipid profile were similar between groups, with the average HDL-C at approximately the 50th percentile for men of comparable age (38). The greater baseline CETPa in the hypercholesterolemic group was directly related to higher TC and LDL-C concentrations. Elevated CETPa in the hypercholesterolemic men and the inverse relationship between CETPa and HDL-C and HDL₂-C concentrations in the present cohort concur with what has been previously reported (49).

Some of the metabolic conditions that underlie HC (e.g., liver function, apolipoprotein B receptor deficiency or dysfunction, enzyme synthesis or secretion, etc.) may not be influenced by a single session of exercise. In contrast, the elevated TC, LDL-C, and CETPa observed in hypercholesterolemic men may allow for greater lipid responses to exercise than those with normal lipid profiles. Indeed, the earlier findings of Crouse and co-workers (12) presented evidence that

Table 2. Correlations for selected physiological, lipid, and lipoprotein enzyme data at baseline

	TC	FC	LDL-C	HDL-C	HDL ₂ -C	HDL ₃ -C	TG	LPLa	HTGLa
Weight, kg				-0.432			0.479	0.425	
W/H		0.437			-0.325				0.602
Body fat, %							0.398	0.609	
TG				-0.761	-0.523	-0.607			0.461
HTGLa				-0.474	-0.534				
LCATa		0.428							
CETPa	0.447	0.401	0.449	-0.422	-0.435				

Pearson product-moment correlations were determined with the combined data from HC and NC groups. All correlations shown are significant, P < 0.05 for all.

Table 3. Changes in blood lipid and lipoprotein enzyme variables with exercise

	Baseline	IPE	+24 h	+48 h
TC, mg/dl				
HC	252 ± 5	247 ± 5	248 ± 7	251 ± 7
NC	179 ± 4	171 ± 4	182 ± 4	180 ± 5
LDL-C, mg/dl				
HC	179 ± 5	175 ± 5	176 ± 6	180 ± 7
NC	109 ± 5	100 ± 4	109 ± 5	107 ± 5
TG, mg/dl				
HC	155 ± 13	148 ± 8	140 ± 12	136 ± 11
NC	132 ± 17	131 ± 18	115 ± 14	117 ± 13
HDL-C, mg/dl				
HC	41 ± 2	41 ± 2	43 ± 2	46 ± 2
NC	43 ± 3	45 ± 3	49 ± 3	50 ± 4
HDL ₂ -C, mg/dl				
HC	8 ± 1	8 ± 1	8 ± 2	9 ± 1
NC	9 ± 2	8 ± 1	11 ± 2	11 ± 2
HDL ₃ -C, mg/dl				
HC	33 ± 2	34 ± 1	35 ± 2	36 ± 2
NC	34 ± 2	37 ± 3	38 ± 2	39 ± 2
LPLa, $\mu\text{mol FFA} \cdot \text{ml}^{-1} \cdot \text{h}^{-1}$				
HC	14.2 ± 1.3	16.0 ± 1.4	17.1 ± 1.6	14.9 ± 1.2
NC	11.9 ± 1.2	12.1 ± 1.3	14.6 ± 1.7	14.5 ± 1.4
HTGLa, $\mu\text{mol FFA} \cdot \text{ml}^{-1} \cdot \text{h}^{-1}$				
HC	21.3 ± 2.0	20.5 ± 2.0	20.6 ± 2.2	20.5 ± 2.0
NC	21.0 ± 3.2	18.8 ± 3.0	21.4 ± 3.3	20.0 ± 2.9
LCATa, $\mu\text{mol chol est} \cdot \text{l}^{-1} \cdot \text{h}^{-1}$				
HC	76.8 ± 8.2	77.5 ± 8.0	79.2 ± 10.8	84.7 ± 8.2
NC	60.6 ± 7.1	56.1 ± 5.9	55.7 ± 6.0	60.2 ± 7.7
CETPa, %CE transferred/4 h				
HC	34.4 ± 1.7	34.5 ± 1.5	34.8 ± 1.9	35.3 ± 2.1
NC	28.2 ± 1.9	28.4 ± 1.9	29.6 ± 2.4	30.4 ± 2.7

Values are group means at each time point ± SE. HC, $n = 13$; NC, $n = 12$; Baseline, 24 h before experimental exercise session; IPE, immediately postexercise; +24 h and +48 h, 24 and 48 h after exercise, respectively.

hypercholesterolemic men may respond uniquely to a single session of aerobic exercise.

Our TC results are in agreement with those reported previously by Crouse et al. (12), in that the decrease in TC was of similar magnitude (3 vs. 5%) and occurred immediately after the exercise session. Yet the findings reported by Crouse et al. differed from the present study in that TC and LDL-C values continued to increase after exercise and were significantly greater

than baseline levels 48 h after the exercise session. Our results indicate that TC and LDL-C concentrations can be reduced immediately after exercise, return to pre-exercise levels within hours after the exercise session, and remain stable thereafter. Furthermore, the present findings provide evidence that the TC and LDL-C responses after exercise are the same in sedentary men, regardless of their initial cholesterol status.

The magnitude and direction of the TG response in the present study were similar to the 10% decrease

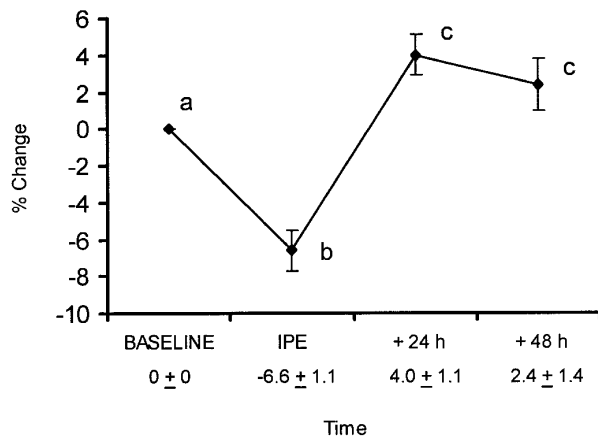


Fig. 1. Percent change in plasma volume with exercise. Baseline, 24 h before exercise; IPE, immediately postexercise; +24 h and +48 h, 24 and 48 h after exercise, respectively. Data are combined group means ± SE. ^{a,b,c}Significant difference between times ($P < 0.05$).

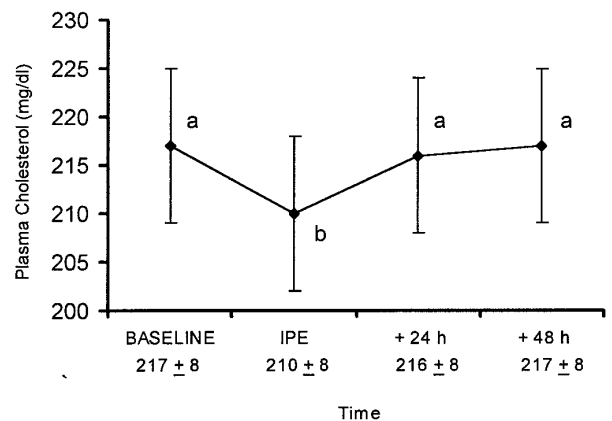


Fig. 2. Average change in plasma volume-adjusted total cholesterol concentrations. Data are combined group means ± SE. ^{a,b}Significant difference between times ($P < 0.05$).

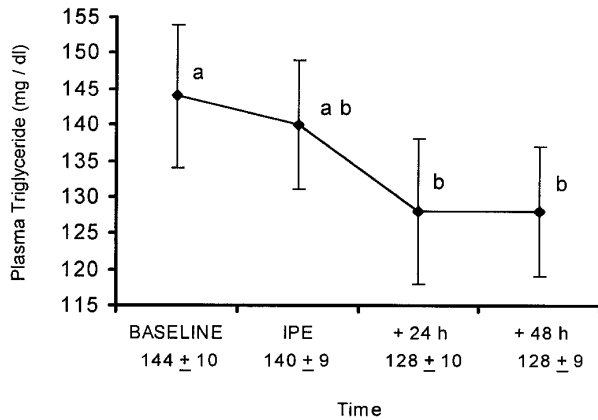


Fig. 3. Average change in plasma triglyceride concentrations. Data are plasma volume-adjusted means ± SE. ^{a,b}Significant difference between times (*P* < 0.05).

reported in inactive subjects (27) and somewhat below the 18% decrease reported previously for sedentary hypercholesterolemic men (12). In contrast, others have reported that TG concentrations did not change in sedentary subjects after a single exercise session (3, 13, 30). Reasons for the discrepancies are unclear. In trained individuals, reductions in TG seem to occur most frequently when the energy requirement of the exercise bout is large (17). However, the TG response in sedentary individuals has occurred when the energy expenditure was either low (27) or moderate, as reported in the present study and elsewhere (12). Moreover, investigators have failed to observe TG responses in sedentary individuals using a variety of exercise interventions (3, 13, 30). Therefore, discrepancies in the TG response for sedentary subjects may not be solely a function of the exercise stimulus. It may be that the baseline TG concentrations influence the TG response to aerobic exercise in untrained individuals. For example, when inactive subjects had low initial TG concentrations (range 89–123 mg/dl), the TG values did not change after exercise (13, 30); however, exceptions do exist (27). Our findings and those reported by

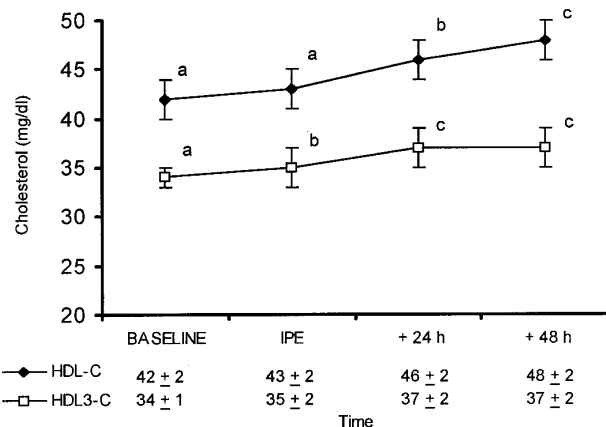


Fig. 4. Average change in plasma high-density-lipoprotein cholesterol (HDL-C) and HDL₃-C concentrations. Data are plasma volume-adjusted means ± SE. ^{a,b,c}Significant difference between times (*P* < 0.05).

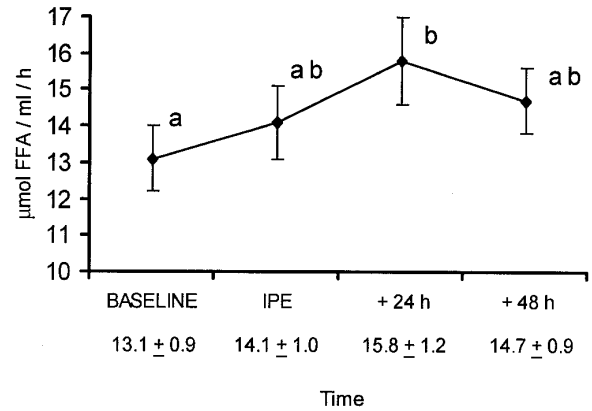


Fig. 5. Average change in postheparin plasma lipase activity. FFA, free fatty acid. Data are plasma volume-adjusted means ± SE. ^{a,b}Significant difference between times (*P* < 0.05).

Crouse et al. (12) demonstrate that, when initial TG values are moderately elevated (~170–177 mg/dl) in sedentary individuals, a delayed reduction in TG concentrations can persist for up to 48 h after a single bout of exercise.

Baseline TG concentrations in the present study were inversely related to HDL-C, HDL₂-C, and HDL₃-C. These relationships have been described previously and are well established (20). In addition, the decrease in TG concentrations observed in the present study was accompanied by a concomitant 14% elevation in HDL-C that lasted 48 h into recovery. The elevation in HDL-C was primarily due to an 11% increase in HDL₃-C. In trained individuals, the decreases in TG concentrations are rarely accompanied by an increase in HDL-C after an exercise bout (16, 29). In many cases, decreases in TG occurred without changes in HDL-C (15, 28, 34, 50), or HDL-C increased without significant modifications in TG concentrations (25, 33). Similarly, aerobic exercise has been shown to increase HDL-C in sedentary subjects without changing TG concentrations (30). However, increased HDL-C and reduced TG concentrations after a single exercise bout in sedentary hypercholesterolemic individuals have been reported (12). The changes in TG and HDL-C concentrations observed in the present study are in agreement with the previous findings in hypercholesterolemic men. Our results illustrate, however, that these changes are not necessarily unique to hypercholesterolemic individuals, because TG and HDL-C were altered in a similar manner in both normo- and hypercholesterolemic men. It is also noteworthy that the elevations in HDL-C, with or without TG modifications, have largely been attributed to greater HDL₃-C concentrations in sedentary subjects (12, 30). Our HDL₃-C results are in agreement with these findings as well.

At present, it is unclear why there are varying HDL-C and TG responses reported after an exercise bout. It is possible that subject characteristics, such as body composition, regional body fat distribution, cardiovascular fitness, state of training, preexisting lipid concentrations, and a variety of genetic influences on

Table 4. Daily caloric intake and nutrient composition data

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
kcal	2,298 ± 151*	2,045 ± 143	2,126 ± 139	1,901 ± 103	2,079 ± 125	2,006 ± 129	1,930 ± 119
Carbohydrate, g/day	260 ± 19	242 ± 19	264 ± 20	238 ± 16	240 ± 15	259 ± 21	236 ± 22
Fat, g/day	94 ± 9*	83 ± 7	81 ± 7	70 ± 6	86 ± 8	72 ± 6	74 ± 6
Protein, g/day	107 ± 8†	87 ± 5	91 ± 5	87 ± 5	92 ± 6	86 ± 6	86 ± 5
Carbohydrate, %	46 ± 2	47 ± 2	49 ± 2	50 ± 2	47 ± 2	52 ± 2	48 ± 3
Fat, %	36 ± 2	36 ± 2	34 ± 2	33 ± 2	36 ± 2	33 ± 2	35 ± 2
Protein, %	18 ± 1	18 ± 1	18 ± 1	19 ± 1	18 ± 1	17 ± 1	19 ± 1
P/S	0.63 ± 0.06	0.57 ± 0.05	0.71 ± 0.09	0.76 ± 0.06	0.69 ± 0.07	0.72 ± 0.08	0.61 ± 0.06

Values are combined group means ± SE. Nutrients expressed as percentages are percentages of the daily caloric intake (kcal). P/S, polyunsaturated-to-saturated fat ratio. Blood samples were collected on days 5–7. *Day 1 > days 4, 6, and 7 ($P < 0.05$); †day 1 > all other days ($P < 0.05$).

lipid metabolism and transport, play important roles. It is likely that experimental procedures, such as the exercise stimulus (intensity, duration, and mode), the timing of the blood collection, analytic techniques, and controls; subject sample size; and the extent to which dietary habits, nutrient intake, and physical activity levels are taken into account, will have substantial influences on the HDL-C and TG changes reported after an exercise session. Given the available information, it is not possible to quantify from the existing literature the independent or interactive effect that these variables have on HDL-C and TG changes. However, as mentioned previously, a common postulation is that a threshold for caloric expenditure, rather than a specific exercise intensity or duration, may be critical for inducing changes in HDL-C (17). The caloric threshold seems to vary directly with the functional capacity of the subjects (12, 19) and is thought to alter lipid concentrations primarily by inducing postexercise increases in LPLa (39).

A delayed increase in plasma LPLa was observed in the present cohort at 24 and 48 h after the exercise session. Others have shown increases in plasma (25, 29, 30), muscle (36, 48), and adipose tissue LPLa (41, 48) after aerobic exercise. In some studies, the LPLa response was delayed (25, 29, 30) but not always (36, 41, 48). The augmented LPLa accelerates the hydrolysis of TG-rich lipoproteins and is associated with decreases in TG and elevations in HDL-C concentrations (25, 29, 30). The results of the present study support these findings, as greater LPLa was observed in concordance with the lower TG and elevated HDL-C concentrations.

Acute reductions in HTGLa may occur after prolonged exercise (25, 42), but more frequently this enzyme activity is unaltered (29, 30). A decrease in HTGLa may result in higher HDL₂-C concentrations or a greater HDL₂-to-HDL₃-C ratio by diminishing the conversion of HDL₂ to HDL₃. The inverse correlation we observed between baseline measures of HTGLa and HDL-C and HDL₂-C in this study would corroborate this notion. In addition, the lack of change in HTGLa after the exercise session may partially explain the stable HDL₂-C levels observed in this study. Based on our findings, we suggest that the LPLa and HTGLa responses to aerobic exercise are not necessarily differ-

ent in normo- and hypercholesterolemic men. LPLa is augmented after a single bout of prolonged exercise in sedentary men; however, HTGLa in these subjects does not seem to change after a single exercise session.

This study is the first to show that one bout of aerobic exercise does not alter CETPa in sedentary men. The unaltered CETPa may also partially explain the lack of change in the postexercise HDL₂-C concentrations, because the measured CETP-mediated transfer of CE from this fraction was not abated after the exercise session. In contrast, Takanami et al. (46) reported lower CETP concentrations and elevated HDL₂-C concentrations in athletes immediately and 1 day after a triathlon competition. The differences in subject characteristics, the exercise stimulus, and the measures of CETPa vs. concentrations prohibit direct comparisons from being made between these studies. However, given that CETP concentrations are related to CETPa (49), our results appear to contradict the findings of Takanami et al. (46). Factors other than the CETP concentration can influence CETPa. For example, Bagdade et al. (4) have demonstrated that the TG composition of very-low-density-lipoprotein is a major influence on the rate of CETP-mediated lipid transfer among the lipoprotein fractions. Thus a single session of exercise may alter CETPa by changing the lipid composition of the various lipoprotein fractions. CETPa in the present investigation was assayed in the presence of a common donor (³H-HDL₃-CE) and a common acceptor (combined very-low- and low-density lipoprotein fractions). As such, the influence of the lipoprotein lipid composition on CETPa is minimized. When CETPa was measured by similar methodology, it was shown to be closely related to CETP concentrations (49). Therefore, it may be indicated from these results that CETPa, as influenced by CETP protein concentration, is not altered in sedentary subjects after prolonged exercise. However, this theory cannot be fully substantiated because we did not measure CETP concentrations. Whether prolonged exercise can alter CETPa by changing the lipid composition of the various lipoprotein fractions has yet to be determined.

LCATa was not modified by exercise in the present investigation. These results are in agreement with the findings of Berger and Griffiths (7), who reported that LCATa did not change in recreationally active subjects

after a moderate exercise session. Conversely, Frey et al. (22) observed increased LCATa in trained and untrained subjects immediately after prolonged exercise. Similarly, Dufaux et al. (16) reported that LCATa increased acutely after an endurance run yet decreased below baseline values 2 days later. Reasons for the disparate results are not altogether clear. One possibility may be that the changes observed by Dufaux's group were accompanied by elevations in FC and the FC-to-CE ratio. Berger and Griffith (7) reported that the FC-to-CE ratio was not modified with exercise in their study. Therefore, it may be that an increase in available substrate precedes any modification in LCATa. This theory is consistent with the fact that LCATa can be greatly influenced by the substrates necessary for this reaction. For instance, the availability of FC in the HDL fraction can have a measured effect on LCATa (20). The findings of Frey and co-workers (22) may further support this contention, because they reported that baseline LCATa was related to FC and CE concentrations. Baseline LCATa was related to FC concentrations in the present study; however, changes in FC and CE concentrations were not observed after the exercise session.

Differences in daily dietary intake can influence the effect of a single exercise session on plasma lipids and enzyme activity. For example, increased carbohydrate ingestion can reduce LPLa and HDL-C and increase TG concentrations (35). Conversely, increased cholesterol or fat intake can elevate cholesterol concentrations in all the lipoprotein fractions (38). Lithell et al. (35) demonstrated that dietary changes could influence plasma lipid concentrations in as little as 3 days. Thus the purpose of having subjects record their diet on a daily basis was to ensure stable caloric and dietary habits and nutrient intake during the period in which blood samples were obtained. The daily dietary records indicated that the normo- and hypercholesterolemic groups reported similar dietary nutrient composition and caloric intake over the blood sampling period. In the combined group, however, differences in daily caloric intake and the total grams of fat, protein, and cholesterol were observed during the experimental protocol. Further analyses indicated that the dietary variables were generally greater on the first day of record keeping but were stable for 3 days before the first blood draw and throughout the remainder of the blood sampling period. Thus it is unlikely that the different nutrient intake on the first day of dietary record keeping had a bearing on the lipid response to exercise. It can be argued that the differences found on the first day merely reflect a period of adjustment to a stable dietary regimen and that this practice prevented a greater daily variation than what might have occurred without this protocol.

Based on the results of this study, we can conclude that a single bout of aerobic exercise can modify lipid concentrations and the lipoprotein enzyme activities similarly in sedentary normo- and hypercholesterolemic men. A single exercise session can reduce TC and LDL-C, but these changes are transient. More

persistent changes included a reduction in TG and elevations in HDL-C, which can last for up to 48 h after exercise. The greater HDL-C concentrations are primarily due to increases in HDL₃-C. Furthermore, the lipid changes seem to be the result of an increase in LPLa, as HTGLa, LCATa, and CETPa remained unaltered after exercise. These changes are consistent with the notion that exercise can favorably alter the lipid markers of cardiovascular disease risk by augmenting the reverse cholesterol transport pathway. However, the optimal exercise training strategy for ameliorating atherogenic lipid profiles in otherwise healthy hypercholesterolemic men remains to be determined.

We thank Joann Kutchna, Nikki Jackson, and Joan Bush for help in data collection.

This research was supported by Texas A&M Interdisciplinary Research Initiatives Grant 96-57.

REFERENCES

1. **Allain C, Poon L, Chan C, Richmond W, and Fu P.** Enzymatic determination of total cholesterol. *Clin Chem* 20: 470-474, 1974.
2. **American College of Sports Medicine.** Clinical exercise testing. In: *ACSM's Guidelines for Exercise Testing and Prescription*, edited by Kenney WL. Philadelphia, PA: Williams & Wilkins, 1996.
3. **Angelopoulos T and Robertson R.** Effect of a single exercise bout on serum triglycerides in untrained men. *J Sports Med Phys Fitness* 33: 264-267, 1993.
4. **Bagdade J, Ritter M, and Subbiah P.** Accelerated cholesterol transfer in plasma of patients with hypercholesterolemia. *J Clin Invest* 87: 1259-1265, 1991.
5. **Belfrage P and Vaughan M.** Simple liquid-liquid partition system for isolation of labeled oleic acid from mixtures with glycerides. *J Lipid Res* 10: 341-344, 1969.
6. **Berg A, Frey I, Baumstark M, Halle M, and Keul J.** Physical activity and lipoprotein disorders. *Sports Med* 17: 6-21, 1994.
7. **Berger G and Griffiths M.** Acute effects of moderate exercise on plasma lipoprotein parameters. *Int J Sports Med* 8: 336-341, 1987.
8. **Bleicher J and Lacko A.** Physiologic role and clinical significance of reverse cholesterol transport. *J Am Osteopath Assoc* 92: 625-632, 1992.
9. **Bruce R, Kusumi F, and Hosmer D.** Maximal oxygen intake and nomographic assessment of functional aerobic impairment in cardiovascular disease. *Am Heart J* 85: 546-562, 1973.
10. **Bucolo G and Davis H.** Quantitative determination of serum triglycerides by the use of enzymes. *Clin Chem* 19: 476-482, 1973.
11. **Crouse S, O'Brien B, Grandjean P, Lowe R, Rohack J, and Green J.** Effects of exercise training and a single session of exercise on lipids and apolipoproteins in hypercholesterolemic men. *J Appl Physiol* 83: 2019-2028, 1997.
12. **Crouse S, O'Brien B, Rohack J, Lowe R, Green J, Tolson H, and Reed J.** Changes in serum lipids and apolipoproteins after exercise in men with high cholesterol: influence of intensity. *J Appl Physiol* 79: 279-286, 1995.
13. **Cullinane E, Lazarus B, Thompson P, and Saritelli A.** Acute effects of a single exercise session on serum lipids in untrained men. *Clin Chim Acta* 109: 341-344, 1981.
14. **Dill D and Costill D.** Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol* 37: 247-248, 1974.
15. **Dufaux B, Assmann G, Order U, Hoederath A, and Hollmann W.** Plasma lipoproteins, hormones, and energy substrates during the first days after prolonged exercise. *Int J Sports Med* 2: 256-260, 1981.
16. **Dufaux B, Order U, Muller R, and Hollmann W.** Delayed effects of prolonged exercise on serum lipoproteins. *Metabolism* 35: 105-109, 1986.

17. **Durstine J and Haskell W.** Effects of exercise training on plasma lipids and lipoproteins. In: *Exercise and Sports Science Reviews*, edited by Holloszy J. Philadelphia, PA: Williams & Wilkins, 1994, p. 477–521.
18. **Durstine J and Moore G.** Hyperlipidemia. In: *Exercise Management for Persons with Chronic Diseases and Disabilities/American College of Sports Medicine*, edited by Durstine J. Champaign, IL: Human Kinetics, 1997, p. 101–105.
19. **Ferguson M, Alderson N, Trost S, Essig D, Burke J, and Durstine J.** Effects of four different single exercise sessions on lipids, lipoproteins, and lipoprotein lipase. *J Appl Physiol* 85: 1169–1174, 1998.
20. **Fielding P and Fielding C.** Dynamics of lipoprotein transport in the circulatory system. In: *Biochemistry of Lipids, Lipoproteins and Membranes*, edited by Vance D and Vance J. Edmonton, Alberta, Canada: Elsevier, 1991, p. 427–459.
21. **Frederickson D and Lees R.** A system for phenotyping hyperlipoproteinemia. *Circulation* 31: 321–326, 1965.
22. **Frey I, Baumstark M, Berg A, and Keul J.** Influence of acute maximal exercise on lecithin: cholesterol acyltransferase activity in healthy adults of differing aerobic performance. *Eur J Appl Physiol* 62: 31–35, 1991.
23. **Friedewald W, Levy R, and Frederickson D.** Estimation of the concentration of low-density-lipoprotein cholesterol in plasma without the use of preparative centrifugation. *Clin Chem* 18: 499–502, 1972.
24. **Gidez L, Miller G, Burstein M, Sagle M, and Eder H.** Separation and quantitation of subclasses of human plasma high density lipoproteins by a simple precipitation procedure. *J Lipid Res* 23: 1206–1223, 1982.
25. **Gordon P, Goss F, Visich P, Warty V, Denys B, Metz K, and Robertson R.** The acute effects of exercise intensity on HDL-C metabolism. *Med Sci Sports Exerc* 26: 671–677, 1994.
26. **Heyward V.** Assessing body composition and anthropometric components of fitness. In: *Advanced Fitness Assessment and Exercise Prescription*, edited by Frey R, Roy M, Anderson J, and Levy D. Champaign, IL: Human Kinetics, 1991, p. 141–183.
27. **Hughes R, Thorland W, Housh T, and Johnson G.** The effect of exercise intensity on serum lipoprotein responses. *J Sports Med Phys Fitness* 30: 254–260, 1990.
28. **Kaminsky L, Kanter M, Nequin N, Lesmes G, and Laham-Saeger J.** Lipids and lipoproteins after an ultramarathon road race. *Ann Sports Med* 4: 41–44, 1988.
29. **Kantor M, Cullinane E, Herbert P, and Thompson P.** Acute increase in lipoprotein lipase following prolonged exercise. *Metabolism* 33: 454–457, 1984.
30. **Kantor M, Cullinane E, Sady S, Herbert P, and Thompson P.** Exercise acutely increases high density lipoprotein-cholesterol and lipoprotein lipase activity in trained and untrained men. *Metabolism* 36: 188–192, 1987.
31. **Kiens B and Lithell H.** Lipoprotein metabolism influenced by training-induced changes in human skeletal muscle. *J Clin Invest* 83: 558–564, 1989.
32. **Krauss R, Levy R, and Fredrickson S.** Selective measurement of two lipase activities in postheparin plasma from normal subjects and patients with hyperlipoproteinemia. *J Clin Invest* 54: 1107–1124, 1974.
33. **Kuusi T, Kostiaainen E, Vartiainen E, Pitkanen L, Ehnholm C, Korhonen H, Nissinen A, and Puska P.** Acute effects of marathon running on levels of serum lipoproteins and androgenic hormones in healthy males. *Metabolism* 33: 527–531, 1984.
34. **Lamon-Fava S, McNamara J, Farber H, Hill N, and Schaefer E.** Acute changes in lipid, lipoprotein, apolipoprotein, and low-density-lipoprotein particle size after an endurance triathlon. *Metabolism* 38: 921–925, 1989.
35. **Lithell H, Jacobs I, Vessby B, Hellsing K, and Karlsson J.** Decreases of lipoprotein lipase activity in skeletal muscle in man during a short-term carbohydrate-rich dietary regime. With special reference to HDL-cholesterol, apolipoprotein and insulin concentrations. *Metabolism* 31: 994–998, 1982.
36. **Lithell H, Orlander J, Schele R, Sjodin B, and Karlsson J.** Changes in lipoprotein lipase activity and lipid stores in human skeletal muscle with prolonged heavy exercise. *Acta Physiol Scand* 107: 257–261, 1979.
37. **Moriguchi E, Tamachi H, and Goto Y.** Hepatic lipase activity and high density lipoproteins in familial hypercholesterolemia: adaptational mechanisms for LDL-receptor deficient state. *Toikai J Exp Clin Med* 15: 401–406, 1990.
38. **National Cholesterol Education Program.** Second report of the expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel II). *Circulation* 89: 1331–1445, 1994.
39. **Oscail L, Tsika R, and Essig D.** Exercise has a heparin-like effect on lipoprotein lipase activity in muscle. *Can J Physiol Pharmacol* 70: 905–909, 1992.
40. **Peltonen P, Marniemi J, Hietanen E, Vuori I, and Ehnholm C.** Changes in serum lipids, lipoproteins, and heparin releasable lipolytic enzymes during moderate physical training in man: a longitudinal study. *Metabolism* 30: 518–526, 1981.
41. **Savard R, Despres J-P, Marcotte M, Theriault G, Tremblay A, and Bouchard C.** Acute effects of endurance exercise on human adipose tissue metabolism. *Metabolism* 36: 480–485, 1987.
42. **Seip RL, Moulin P, Cocke T, Tall A, Kohrt WM, Mankowitz K, Semenkovich CF, Ostlund R, and Schonfeld G.** Exercise training decreases plasma cholesteryl ester transfer protein. *Arterioscler Thromb* 13: 1359–1367, 1993.
43. **Stokke K and Norum K.** Determination of lecithin: cholesterol acyltransferase in human plasma. *Scan J Clin Lab Invest* 27: 21–27, 1971.
44. **Superko H.** The most common cause of coronary heart disease can be successfully treated by the least expensive therapy—exercise. *ACSM Certified News* 8: 1–5, 1998.
45. **Superko H and Haskell W.** The role of exercise training in the therapy of hyperlipoproteinemia. *Cardiol Clin* 5: 285–310, 1987.
46. **Takanami Y, Iwane H, Kawai Y, Katsumura T, and Shimomitsu T.** Influence of strenuous endurance exercise on cholesteryl transfer protein and HDL metabolism in serum (Abstract). *Med Sci Sports Exerc* 28: S29, 1996.
47. **Taskinen M-R and Nikkila E.** High density lipoprotein subfractions in relation to lipoprotein lipase activity of tissues in man—evidence for reciprocal regulation of HDL₂ and HDL₃ levels by lipoprotein lipase. *Clin Chim Acta* 112: 325–332, 1981.
48. **Taskinen M-R, Nikkila E, Rehunen S, and Gordin A.** Effect of acute vigorous exercise on lipoprotein lipase activity of adipose tissue and skeletal muscle in physically active men. *Artery* 6: 471–483, 1980.
49. **Tato F, Vega G, Tall A, and Grundy S.** Relation between cholesterol ester transfer protein activities and lipoprotein cholesterol in patients with hypercholesterolemia and combined hyperlipidemia. *Arterioscler Thromb Vasc Biol* 15: 112–120, 1995.
50. **Thompson P, Cullinane E, Henderson O, and Herbert P.** Acute effects of prolonged exercise on serum lipids. *Metabolism* 29: 662–665, 1980.
51. **Thompson P, Kantor M, Cullinane E, Sady S, Saritelli A, and Herbert P.** Postheparin plasma lipolytic activities in physically active and sedentary men after varying and repeated doses of intravenous heparin. *Metabolism* 35: 999–1004, 1986.
52. **Warnick G and Albers J.** A comprehensive evaluation of the heparin-manganese precipitation procedure for estimating high density lipoprotein cholesterol. *J Lipid Res* 19: 65–76, 1978.