Changes in LPLa and reverse cholesterol transport variables during 24-h postexercise period

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Changes in LPLa and reverse cholesterol transport variables during 24-h postexercise period. Am J Physiol Endocrinol Metab 283: E267–E274, 2002. First published April 2, 2002; 10.1152/ajpendo.00567.2001.—We investigated the time course of exercise-induced lipoprotein lipase activity (LPLa) and reverse cholesterol transport (RCT) during the 24-h postexercise period. Subjects were 10 sedentary normolipidemic males (NTG; fasting triglyceride (TG) = 89.1 ± 8.6 mg/dl) and 6 hyperlipidemic males (HTG; fasting TG = 296.8 ± 64.0 mg/dl). Each subject performed a control trial (no exercise) and 4 exercise trials. In the exercise trials, a subject jogged on a treadmill at 60% of his maximal O2 consumption for 1 h. Pre- and postheparin blood samples were taken before exercise (baseline) and at 4, 8, 12, and 24 h after exercise. There was no group difference in LPLa (P > 0.05) over the time points. When the LPLa data from the two groups were combined, LPLa at 24 h after exercise was higher than baseline or at 4, 8, 12 h after exercise (P < 0.05). Plasma TG and lecithin-cholesterol acyltransferase activity (LCATa) were higher in HTG than in NTG, and the total high-density lipoprotein-cholesterol (HDL2-Chol) lower in HTG than in NTG (P < 0.05). HDL2-Chol, LCATa, and cholesterol ester transfer protein activity did not differ during the 24-h postexercise period (P > 0.05). These results suggest that LPLa is still increasing 24 h after an acute aerobic exercise and that the magnitude of the increase in exercise-induced LPLa in HTG was similar to that in NTG. Furthermore, in the sedentary population with or without HTG, the variables related to RCT do not change during the 24-h period after exercise.

LPL promotes the clearance of TG from the blood by hydrolyzing TG into fatty acids (FAs). These FAs are then taken up by muscle tissues, where they are used as an energy source, or they can be taken up by adipose tissues, reesterified into TG, and stored as lipid droplets (25). This hormone-sensitive enzyme is believed to be stimulated by epinephrine (Epi) and inhibited by insulin (18). Exercise elevates the Epi level and attenuates the insulin level (35).

The ability of an aerobic exercise bout to reduce PPL is related to the length of time between the meal and the exercise session (17, 36). We previously discovered that an aerobic exercise bout 12 h before an HFM was more effective in reducing PPL when compared with bouts immediately (1 h) before or after an HFM (36). This time discrepancy may be because of the delayed elevation of LPLa after the exercise session (30).

LPLa is depressed immediately after exercise (23) and increased at 4 h (24), 18 h (22, 29, 30), 20 h (34), and 24 h (14, 23) postexercise. One limitation of these studies was that LPLa was measured at only one time point. There are several reasons why LPLa is usually determined at only one time point. To measure LPLa in plasma, heparin must be given to the subject. However, heparin is an anticoagulant, and multiple injections of heparin may have detrimental effects on the subject. Also, LPL may not be fully recovered from the previous cleave-off by heparin injection in a short time because LPL is synthesized in muscle and adipose cells and migrates to the lumen of capillary endothelium (25). This may take a certain amount of time. LPLa also can be measured in muscle or adipose tissue samples col-
lected by a needle biopsy. This is an invasive procedure, and multiple sample collections in humans are usually avoided. Because of these reasons, it is difficult to measure the time course of LPLa in humans.

The results of LPLa after a single exercise session from previous studies are difficult to interpret because there were differences in experimental protocols. Some of these previous studies measured LPLa in postheparin plasma, and others measured LPLa in either skeletal or adipose tissue. The source of the sample and the analytical technique used to measure LPLa can result in different values for LPLa. Thus comparisons of LPLa values that are derived from plasma and tissue using different analytical techniques are not valid. The mode, intensity, and duration of the exercise sessions also differed between these previous studies. These differences in exercise sessions also make comparisons among studies difficult. There have been no reports on the time course of postheparin LPLa at specific time points during the 24-h postexercise period. We attempted to collect these data at specific time points by replicating the exercise and postheparin protocol at different time points on different days.

LPLa after an aerobic exercise bout in individuals with HTG has not been investigated. Examination of individual subject data from previous studies completed in this laboratory (34, 36, 37) has shown that the reduction in PPL after an HFM in individuals with HTG may be just as great or greater than in individuals with normal TG (NTG) levels (unpublished results).

In addition, PPL often is associated with a low level of HDL-Chol and a high level of low-density lipoprotein-Chol (13). HDL-Chol is responsible for transporting cholesterol ester to the liver for excretion, a process known as reverse cholesterol transport (RCT). Sustained PPL may impair RCT, resulting in atherosclerosis. Lecithin-cholesterol acyltransferase (LCAT), an enzyme synthesized in the liver, is responsible for cholesterol ester synthesis. The elevation of LCAT activity (LCATa) may result in the elevation of HDL-Chol (25). However, whether exercise training or a single session of exercise causes changes in LCATa still remains equivocal (14, 16). Therefore, the purpose of this study was to examine the time course of LPLa and RCT-related variable changes after an acute bout of aerobic exercise in sedentary HTG and NTG individuals. We hypothesized that exercise may induce a significant elevation in plasma heparin-releasable LPLa and a possible improvement in RCT at 12 and 24 h postexercise. The LPLa in patients with HTG might be less sensitive to acute exercise stimulation than normolipemic individuals.

METHODS

Subjects. Sixteen sedentary male subjects were recruited for this study (Table 1). Sedentary was defined as participating in no more than one routine exercise session per week over the last 2 yr. Subjects were informed of the risks associated with the study and were required to complete an informed-consent form approved by the University of Missouri Health Sciences Institutional Review Board. Each subject completed a medical history questionnaire, a diet questionnaire, a physical activity questionnaire, and a diet habit survey. Subjects were disqualified if they were taking any form of TG-lowering medication or displaying more than one CVD risk factor as defined by the American College of Sports Medicine’s Guidelines for Exercise Testing and Prescription (1).

Preliminary testing. Subjects were divided into two different groups based on their fasting TG screen. Subjects with a fasting TG level between 50 and 180 mg/dl were placed in the NTG group and those with fasting TG levels between 75 and 150 mg/dl were placed in the HTG group (n = 10). Subjects with fasting TG levels between 200 and 400 mg/dl were placed in the HTG group (n = 6). Percent body fat was estimated from the measurement of three skinfold sites (abdomen, chest, and thigh; see Ref. 21).

Each subject performed a maximal oxygen consumption (Vo2max) test (36) to determine the appropriate exercise intensity to be used in the subsequent exercise sessions. Briefly, subjects warmed up for 5 min at a self-selected pace at a level treadmill grade. The initial speed of the treadmill was 4 mph for the first 2 min of the test. After the initial 2 min, the treadmill speed was increased by 0.5 mph/min until the treadmill speed was up to 6.5 mph. Thereafter, the speed remained constant, and the treadmill grade was raised by 2% every minute until exhaustion. The highest Vo2 obtained was considered the subject’s Vo2max.

Subject preparation. To reduce intrasubject variability, each subject completed a 24-h dietary record during the day immediately before the first trial. A copy of this diet was given to each subject before each subsequent trial. Each subject was required to repeat the same diet during the 24-h period before each additional trial. Telephone calls were made to each subject 2 days before each trial to remind them to follow the same 24-h diet that they recorded before their first trial and to refrain from alcohol and caffeine use for the next 24 h.

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Age, yr</th>
<th>Weight, kg</th>
<th>Body fat, %</th>
<th>V02max, ml·kg⁻¹·min⁻¹</th>
<th>Total calories used in exercise, kcal/h</th>
<th>Body mass index, kg/m²</th>
<th>Waist-to-hip ratio</th>
<th>RER</th>
<th>Fat calories used in exercise</th>
<th>Cholesterol calories used in exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>19 ± 2</td>
<td>14.3 ± 4.4</td>
<td>22.0 ± 1.6</td>
<td>36.5 ± 1.5</td>
<td>507 ± 20</td>
<td>25.9 ± 1.1</td>
<td>0.87 ± 0.01</td>
<td>0.90 ± 0.01</td>
<td>171 ± 19</td>
</tr>
<tr>
<td>n = 10</td>
<td></td>
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<tr>
<td>Hypertriglyceridemic</td>
<td>84 ± 2</td>
<td>92 ± 5</td>
<td>23.0 ± 1.9</td>
<td>29.4 ± 1.8</td>
<td>479 ± 34</td>
<td>28.2 ± 1.2</td>
<td>0.91 ± 0.02</td>
<td>0.92 ± 0.01</td>
<td>130 ± 35</td>
</tr>
<tr>
<td>n = 0</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>88 ± 2</td>
<td>90 ± 4.5</td>
<td>23.0 ± 1.3</td>
<td>30.8 ± 1.4</td>
<td>497 ± 18</td>
<td>26.8 ± 0.9</td>
<td>0.89 ± 0.01</td>
<td>0.91 ± 0.01</td>
<td>155 ± 15</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. V02max, maximal O2 consumption; Cho, carbohydrate; TG, triglyceride; TC, total cholesterol; SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid; RER, respiratory exchange ratio.

*Significant difference between groups (P < 0.05).

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Experimental design. Each subject completed five different trials. One trial was used to determine the baseline (preexercise level) of LPLa, hepatic lipase activity (HLa), TG, HDL-Chol subfractions, LCATa, cholesteryl ester transfer protein activity (CETPa), insulin, and Epi. The purpose of the other four trials was to determine changes in these variables at 4, 8, 12, and 24 h after an aerobic exercise session. The trial flow chart is shown in Fig. 1. The order of these five trials was randomized. Each trial was separated by at least 1 wk. Subjects reported to the laboratory after a 12-h fast. An initial blood sample was taken immediately before heparin injection for the analysis of TG, HDL-Chol subfractions, LCATa, CETPa, insulin, and Epi. The heparinized blood sample was collected 15 min after the injection of sodium heparin [1,000 U/ml (Elkins-Sinn, Cherry Hill, NJ); 100 IU/kg body wt] in each trial at the designated time to determine LPLa and HLa (Fig. 1). To assess the postexercise Epi level, a preheparin sample was also collected immediately after one of the exercise trials. The exercise session consisted of a 60-min treadmill jog at 60% of the subject’s $V_{\text{O2 max}}$. Metabolic and heart rate data were collected during the first exercise session to determine the treadmill speed to maintain the subject at 60% of their $V_{\text{O2 max}}$. This speed was used for all subsequent exercise trials.

Standard meal. A standardized meal was given to the subjects every 4 h during each exercise trial period (Fig. 1). The meal consisted of 4 oz (dry weight) of Hodgson Mill Whole Wheat Pasta (Hodgson Mill, Gainesville, MO), 0.5 cup of Enrico’s Fat Free Pasta Sauce (Venture Paking, Syracuse, NY), four Fat Free Natural Ry Krisp Crackers (Bremner, Princeton, KY), and 1.0 oz of Kraft Natural Reduced Fat Mild Cheddar Cheese (Kraft Foods, Glenview, IL). Each meal contained 650 kcal, of which 62% was carbohydrate, 23% was protein, and 15% was fat. Saturated FAs accounted for 40% of the fat calories, and unsaturated FAs accounted for 30%. The meals were designed to have a low glycemic index to minimize the insulin response (10).

Blood collection. All blood samples were collected via a butterfly needle inserted in an antecubital vein. All nonheparinized blood samples were collected in 10-ml tubes containing EDTA. Postheparin blood was collected in 10-ml tubes containing heparin. All samples were separated by centrifugation at 4°C for 15 min at 2,000 g in a Marathon 22100R centrifuge (Fisher Scientific, Pittsburgh, PA). The plasma was stored at −70°C until analyzed.

TG, insulin, Epi, CETPa, and HDL-Chol subfraction analysis. Plasma TG concentrations were measured enzymatically using diagnostic kits (Infinity TG Reagent; Sigma, St. Louis, MO). Plasma insulin levels were measured using an Insulin$^{125}$ RIA kit (ICN Pharmaceuticals, Costa Mesa, CA). Plasma Epi levels were analyzed using an enzyme-linked immunosorbent assay kit supplied by KMI Diagnostics (Minneapolis, MN). CETPa was determined by a CETPa kit with spectrofluorometric quantitation (Roar Biomedical, New York, NY). HDLtot-Chol and HDLsub-Chol were measured by using the modified heparin-MnCl$_2$-dextran sulfate method, as described elsewhere (36).

LPLa and HLa analysis. LPLa and HLa were measured as previously described (20). Heparinized plasma (30 μl) was incubated with a radioactive TG ([14C]triolein) emulsion at 37°C for 1 h. Total lipase activity was measured by the liberation of labeled (14C) free fatty acids (FFAs) from the radioactive emulsion. HLa was measured by inhibiting LPLa with a 1.0 M NaCl solution. The difference between the total activity and the HLa was equal to the LPLa. Activity levels were measured via a scintillation counter (Beckman LS5800) and reported as micromoles FFA per milliliter of plasma per hour.

LCATa analysis. LCATa was determined as described by Gillett and Owen (12). Briefly [7(n)-3H]cholesterol (Amercham) was dried and then mixed with human albumin (Sigma). Blood plasma was incubated with albumin-cholesterol emulsion and Ellmann reagent (Sigma). Next, mercaptoethanol was added for enzyme reactivation. The reaction was stopped by adding chloroform-methanol. Lipid precipitates were separated by thin-layer chromatography (TLC). The fraction on TLC containing free and esterified cholesterol was determined by liquid scintillation. The results were reported as the percentage of nonesterified cholesterol esterified per 4 h.

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**Fig. 1.** Trial flow chart. EX, exercise; Arrow, blood draw. * Standard meal ingestion.
To eliminate interassay variability, all samples from a single subject were analyzed together in each assay.

Statistical analysis. ANOVA with repeated measures was used for data analysis. SAS software (version 8.02; SAS Institute, Cary, NC) was used to perform the analysis. Significant F ratios were followed with post hoc contrast comparisons with specifically designated error terms. P < 0.05 was considered statistically significant. Relationships between variables were evaluated by using Pearson's product-moment correlation coefficient. All data are presented as means ± SE.

RESULTS

Table 1 depicts the subject characteristics. The VO_2_MAX was significantly higher in the NTG group than in the HTG group, whereas the fasting TG levels in the HTG group were significantly higher than their counterparts (89.1 ± 8.6 vs. 296.8 ± 64.0 mg/dl). The 0-h (baseline) values of LPL_a were not significantly different between the two groups (NTG, 3.82 ± 0.62 vs. HTG, 4.25 ± 0.83 μmol FFA·ml plasma⁻¹·h⁻¹). There were no significant (P > 0.05) differences in either LPL_a or HLa between groups over any time period. When the two groups were combined, LPL_a was significantly elevated at 24 h compared with 0, 4, 8, and 12 h (Fig. 2), and HLa was higher at 8, 12, and 24 h compared with 4 h (Fig. 3). TG levels in the two groups did not differ across the time points (P > 0.05). When trials were combined, TG in the NTG group was higher than the HTG group (P < 0.001). Combined group TG did not differ across the time points (P = 0.086; Fig. 4). The postexercise Epi levels were higher (P < 0.05) than preexercise levels in both groups, although the result did not reveal group differences in Epi (Fig. 5).

![Figure 2](image1.png)

**Fig. 2.** Effect of exercise timing on lipoprotein lipase activity (LPL_a). There was no difference in LPL_a between normal triglyceride (NTG) and high plasma triglyceride (HTG) groups (P > 0.05). FFA, free fatty acid. *Combined LPL_a at 24 h was significantly higher than at 0, 4, 8, and 12 h (P < 0.05). #Combined LPL_a at 4 h was significantly lower than at 0 h (P < 0.05).

![Figure 4](image2.png)

**Fig. 4.** Effect of exercise timing on triglyceride (TG). TG levels within either group did not differ across the time points (P > 0.05). *When trials were combined, TG in the HTG group was higher than in the NTG group (P < 0.001). Combined group TG did not differ across the time points (P = 0.086).
at each time point were combined. There were no significant differences in HDL$_2$-Chol between groups and across the time period (Fig. 8). The LCAT$_a$ was not affected by exercise (Fig. 9). Nonetheless, the LCAT$_a$ was significantly higher ($P < 0.05$) in the HTG group than the NTG group at all time points ($P < 0.05$), but there were no differences for either group across the time points. The CETPa data did not show any significant differences between groups and across the time points (Fig. 10). The baseline insulin level was correlated with baseline LPL$_a$ ($r = 0.56$, $P < 0.05$) and postexercise LPL$_a$ ($r = 0.35$, $P < 0.01$). The postexercise insulin level also was correlated with baseline LPL$_a$ ($r = 0.68$, $P < 0.01$) and postexercise LPL$_a$ ($r = 0.62$, $P < 0.01$; Table 2).

**DISCUSSION**

Our LPL$_a$ data support the previous findings that LPL$_a$ was elevated after a single exercise bout (14, 22–24, 29, 30, 34) and remained elevated for up to 24 h after exercise (14, 23). LPL$_a$ in the NTG group tended to increase gradually over the postexercise period, whereas the increase in LPL$_a$ in the HTG group was only observed at 24 h after exercise (Fig. 2). The less sensitive response of LPL$_a$ to an acute exercise bout observed in the HTG group may explain their high fasting plasma TG levels. When data from the NTG and HTG groups were combined, LPL$_a$ increased 26% at 24 h postexercise. Likewise, previous studies reported an 11% (23) and 21% (14) increase in LPL$_a$ at 24 h postexercise in untrained men.

However, studies using biopsy techniques revealed different results in exercise-induced LPL$_a$ changes (24, 30). Seip et al. (30) reported that exercise significantly increased heparin-nonreleasable skeletal muscle LPL$_a$.
but did not increase heparin-releasable mLPLa at 14–18 h postexercise. Kiens et al. (24) observed that mLPLa increased at 4 h postexercise and tended to decrease at 8 h. Nonetheless, our data showed a tendency to increase in plasma postheparin LPLa at 24 h postexercise (Fig. 2). These discrepancies are likely the result of differences in the experimental techniques. In the study by Seip et al. (30), the tissues from biopsy were incubated in heparin solution in vitro to release LPL, whereas we injected heparin in vivo. In vivo blood circulation may release LPL differently from in vitro incubation. Thus the direct comparison of heparin-releasable LPLa from biopsy and plasma may be invalid.

By examining the compiled data from muscle biopsy studies (30a), it is reasonable to speculate that skeletal mLPL mRNA peaks at ~4 h postexercise (31), and mLPL protein mass peaks between 8 and 16 h postexercise (at moderate intensity for 60–90 min; see Refs. 30, 31, 32a). Data from previous studies (14, 22, 34) and the current study also suggest that plasma LPLa may peak between 18 and 24 h postexercise. The generation of the capillary surface-bound LPL is a multiple-step process, beginning from gene expression to LPL translocation (4), which may explain the delayed increase in postheparin LPLa after an acute exercise session.

Studies of exercise-induced changes in HLa revealed conflicting results. Some studies reported decreases in HLa 24 h (12a, 34a) after exercise. Others, however, found no changes in HLa at 24 h postexercise compared with the baseline levels (8b, 14, 23), which is in agreement with our current data. Nonetheless, our data showed that HLa was significantly elevated at 8, 12, and 24 h compared with 4 h after exercise (Fig. 3). Kantor et al. (23) observed an increase in HLa in trained individuals at 72 h postexercise. Comparisons of the results are difficult because of differences in subject population and experimental conditions. For instance, some studies used trained individuals (12a, 34a) and others used untrained individuals (14); and the caloric expenditures used in the exercise session varied from ~500 (14) to 800 (8b, 12a, 34a) kcal.

Whether HLa is proatherogenic or antiatherogenic still remains undetermined (8a). The decrease in HLa may favor the conversion of HDL3-Chol to HDL2-Chol (6), which has been considered as antiatherogenic. However, a recent study (8a) reported that patients with CVD had lower HLa than their healthy counterparts and suggested that low HLa was a novel risk factor for CVD. Exercise training resulted in a 46% increase in HLa in patients with type 2 diabetes, which was accompanied by an increase in HDLtot-Chol (24a).

It has been proposed that exercise-induced LPLa may be regulated by Epi (24), which results in a cascade of events leading to the elevation of LPLa (25). In support of this classic notion, Pedersen et al. (27a) reported that infusion of Epi induced elevation in mLPLa in humans. However, this concept has been challenged by Greiwe et al. (14a), who demonstrated that mLPL protein mass was markedly increased at 22 h postexercise regardless of α- and β-adrenergic

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**Table 2. Correlation between LPLa and other relevant variables**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Correlation with Baseline LPLa</th>
<th>Correlation with Postexercise LPLa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline insulin</td>
<td>0.56*</td>
<td>0.35†</td>
</tr>
<tr>
<td>Postexercise insulin</td>
<td>0.68†</td>
<td>0.62†</td>
</tr>
<tr>
<td>Baseline Epi</td>
<td>0.15</td>
<td>0.11</td>
</tr>
<tr>
<td>Postexercise Epi</td>
<td>−0.04</td>
<td>−0.18</td>
</tr>
<tr>
<td>Baseline TG</td>
<td>−0.01</td>
<td>−0.05</td>
</tr>
<tr>
<td>Postexercise TG</td>
<td>−0.02</td>
<td>−0.16</td>
</tr>
</tbody>
</table>

Values are means ± SE and are computed from both groups. LPLa, lipoprotein lipase activity; Epi, epinephrine. *P < 0.05 and †P < 0.01.
receptor blockage. Likewise, our data did not reveal any correlations between Epi and postexercise LPLα despite the increase in Epi immediately after exercise. The exercise-induced elevation of LPLα might be the result of muscle contraction itself rather than Epi stimulation (14a). However, the signaling pathway of muscular contraction-induced elevation of mLPL remains unknown.

Our data revealed strong correlations between postexercise LPLα and both preexercise (r = 0.35, P < 0.01) and postexercise (r = 0.62, P < 0.01) insulin levels (Table 2). This finding may suggest that the elevated LPLα may be mainly from adipose tissues, because insulin downregulates mLPLα and upregulates adipose LPLα (24). The increased adipose LPLα may help replenish the energy consumed during exercise (27). Additionally, the subjects used in the present study were physically inactive, and their mLPLα may be lower than adipose tissue LPLα. However, because adipose and mLPLα cannot be differentiated in heparinized plasma samples, this is only a speculation. In support of this speculation, Herd et al. (18) reported that an acute exercise bout attenuated a fat meal-induced increase in plasma TG without elevating mLPLα. Nonetheless, Kiens et al. (24) reported that infusion of insulin significantly attenuated mLPLα.

The TG level of the HTG group was 14% lower at 24 h compared with the baseline level (P = 0.06). These results were compatible with the results for LPLα, which were of the highest at 24 h postexercise. Herd et al. (18) reported a 17% decrease in TG at 16 h after 90 min of cycling at 60% VO2 max. However, we did not observe TG reduction in the NTG group after exercise, which agrees with the findings by Angelopoulos and Robertson (3), Cullinane et al. (7), and Kantor et al. (23), who also observed no changes in TG levels in sedentary subjects after a single session of exercise. The TG reduction observed in the HTG group may suggest that sedentary individuals with HTG may benefit more from a session of exercise than their NTG counterparts.

The exercise session used in the current study did not boost HDLtot-Chol or HDL2-Chol levels during the 24-h postexercise period. These observations support the study by Kantor et al. (23), which reported that an acute exercise session did not result in changes in either HDLtot-Chol or in HDL2-Chol in the untrained subjects. Nevertheless, the increase in HDLtot-Chol levels was observed in the trained subjects 18 h (29) and 48 h (23) postexercise. The exercise-induced changes in HDLtot-Chol may be related to the physical fitness levels of subjects.

LCATα has been considered as an important factor in the RCT system (19). Our data showed that the HTG group had significantly higher levels of LCATα compared with the NTG group. Similarly, Tato et al. (33) also observed higher LCATα in HTG subjects than in normal subjects. The high LCATα level may be caused by high plasma TG (2, 28). However, whether acute exercise induces an increase in LCATα still remains equivocal. Some researchers reported that LCATα increased immediately after a maximal exercise (11) and 3 h after a prolonged exercise bout (8). Others observed no changes in LCATα after exercise (5, 15). The acute session of exercise in our study did not yield an increase in LCATα for either group (Fig. 9).

CETP has been proposed to reduce the cholesterol-carrying capacity of HDL and thus is often considered atherogenic (32). However, the effect of exercise on CETPα remains inconclusive. Gupta et al. (16) reported that athletes had higher levels of CETPα compared with sedentary controls, whereas Seip et al. (32) observed a decrease in CETPα after exercise training. After a bicycle marathon, both CETP mass and activity were notably reduced at 24 and 48 h (9). In contrast, the present study did not reveal exercise-induced changes in CETPα (Fig. 10). These incompatible data may be because of different subject populations (trained vs. sedentary) and/or a different volume of exercise activities (bicycle marathon vs. 60 min jogging).

In conclusion, this study demonstrated that LPLα was significantly elevated 24 h after a 1-h exercise session at a moderate intensity in the sedentary population. The magnitude of the exercise-induced increase in LPLα in patients with HTG was similar to that of their NTG counterparts at 24 h postexercise. These results suggest that patients with HTG may have an exercise benefit in LPLα elevation similar to that of NTG individuals at 24 h postexercise.

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