POSTPRANDIAL HYPERTRIGLYCERIDEMIA (PHTG) has been proposed as a potential risk factor for cardiovascular disease (CVD) (28). Patients with permanent hypertriglyceridemia or PHTG tend to have low values of total high-density lipoprotein (HDL)-cholesterol (Ch), or HDL2-Ch, and subfraction HDL2-Ch (6). Plasma HDL2 concentrations are inversely correlated with the magnitude of postprandial lipemia (19). A single postprandial lipemic event can reduce HDL-Ch by 10% (18).

Evidence suggests that patients with a prolonged PHTG after a fat-meal challenge are more susceptible to CVD (17). The syndrome of PHTG in the presence of low concentration of HDL-Ch and high concentrations of small, dense, low-density lipoprotein (LDL) has been considered as a combined marker for CVD (7). Therefore, prolonged PHTG may be an atherogenic process. It can be postulated that events that can shorten the duration of the PHTG may prevent this atherogenic process.

Investigators have reported that aerobic training tends to promote the rapid removal of triglycerides (TGs) after a fat meal (29). Several studies have demonstrated that acute exercise has a significant effect on attenuating PHTG (1, 8, 23). Tsetsonis and Hardman (26) studied the effect of exercise intensity on PHTG. They found that brisk walking, for 1.5 h at 61% maximal O2 consumption (VO2max), 15 h before a fat meal significantly reduced PHTG, whereas walking at 31% of VO2max lacked the PHTG-lowering effect. Tsetsonis and Hardman (27) also reported that exercise before a fat meal at either low intensity (walking at 32% VO2max) and longer duration (3 h) or moderate intensity (63% VO2max) and shorter duration (1.5 h) significantly attenuated PHTG response. But no exercise-induced changes in HDL2-Ch and HDL2-Ch were found in the two studies.

In two similar studies, Aldred et al. (1) and Hardman and Aldred (8) investigated the effect of a single bout of brisk walking on postprandial lipemia. The two studies had a similar test meal, subject population, and blood sampling time. In the study by Aldred et al. (1), the subjects performed the exercise at 31% VO2max for 2 h at 15 h before the fat meal intake, whereas in the study by Hardman and Aldred (8), the subjects exercised for 1.5 h at 40% VO2max 1.5 h after the fat meal. In comparison, the area under the curve (AUC) score for TGs was higher in the study by Aldred et al. (1) than that in the study by Hardman and Aldred (8) (31 vs. 24% lower in exercise trial than in control trial); this suggests that the premeal exercise may have a better effect on PHTG response. The peak TG concentration response to a fat meal was significantly lower in the premeal-exercise trial than that in the control trial found in the study by Aldred et al. (1). However, this difference did not exist in the postmeal-exercise study by Hardman and Aldred (8). In addition, Aldred et al. reported that HDL-Ch and HDL2-Ch concentrations in the exercise trial were higher (P < 0.05) than those in the control trial during a 3- to 5-h period after a fat-rich meal; however, Hardman and Aldred (8) did not find this difference in their study.

Although the differences in these results may be due to different exercise intensities and durations, it is possible that exercise before or after a fat meal may have a different effect on TG response and HDL metabolism because exercise at different times may elevate lipoprotein lipase (LPL) activity along a different time course. Kiens et al. (12) reported that LPL activity was not elevated immediately after exercise; rather, it increased ~4 h after exercise and approached preexercise concentration at 6 h after exercise. Others have noted that LPL stays elevated for at least 18 h (10) to 24 h (11) during the PHTG may prevent this atherogenic process.
after aerobic exercise. This evidence supports the finding that exercise before ingestion of a fat meal may affect TG response more than exercise after a fat meal. This may be due to the delayed elevation of LPL activity that rises to a high concentration before the postprandial-induced TG peaks and, therefore, dampens PHTG.

Humans spend a great deal of time in the PHTG state. The prolonged PHTG state may contribute to the development of CVD. It is possible that exercising at the right time may elevate LPL activity to a high level before the postprandial-induced TG rises. However, no previous studies have focused on the effect of exercise timing (before or after a fat meal) on PHTG response. Whether an acute exercise bout before or after a fat meal affects HDL metabolism also remains equivocal. Therefore, the purpose of this study was to examine the optimal time to exercise (before or after a fat meal) to magnify the effect of exercise on postprandial TG and HDL responses. It was hypothesized that exercise before ingestion of a fat-rich meal may be more effective in attenuation of PHTG response, and possibly in elevation of HDL\textsubscript{tot-Ch} and HDL\textsubscript{2-Ch} concentrations, than exercise after a fat-rich meal.

**METHODS**

**Subjects**

Twenty-one recreationally trained male subjects, ages 21–39 yr, participated in this study (Table 1). A physical activity questionnaire was used to screen each subject to determine his eligibility for participation in the study. Recreationally trained subjects were defined as having regular, noncompetitive type of aerobic exercise for 3–6 times/wk for at least 2 yr before participation. Subjects who were not qualified as recreationally trained or who had any major CVD disease risk factors and symptoms were eliminated from the study. Body composition was assessed by using skinfold measurements for all individuals in an attempt to avoid subjects with extreme body fat (acceptable range: between 9 and 27%). Three skinfold sites (abdominal, chest/pectoral, and thigh) were used to estimate percentage of body fat. The percent body fat was calculated by using the equation described by Jackson and Pollock (9).

Before participation in the study, subjects were instructed to record 3 days of their diet (2 weekdays and 1 weekend) to determine the composition of their habitual diet. The dietary record was analyzed by using the Food Processor IV computer program (version 7.0). To avoid the potentially confounding effect of diet on the lipid profile, individuals with an extreme diet style were not allowed to participate in this study. The criteria for the dietary nutrients were: 1) average fat in the diet within the range of 20–40% and 2) saturated, monounsaturated, and polyunsaturated fat intake each between 7 and 14% of total fat calories. The fasting TG concentrations of all the subjects were within the normal range (51–135 mg/dl) except for one subject who had a fasting TG level of 238 mg/dl. Each subject signed an informed consent form as approved by the Institutional Review Board of the University of Missouri-Columbia.

**Vo\textsubscript{o2max} Test**

Subjects were habituated to the use of the treadmill and performed an exhaustive treadmill test to determine the exercise intensity for the subsequent submaximal exercise in the exercise trials. After a 5-min warm-up at a self-elected pace at level treadmill grade, the initial speed was set at 4 miles/h (mph) and held constant for the first 2 min. Then the speed was increased by 0.5 mph in 1-min increments up to 6.5 mph. Thereafter, the speed remained constant, and the treadmill grade was raised by 2% every 1 min until exhaustion. The highest O\textsubscript{2} consumption value obtained during the test was considered as the Vo\textsubscript{o2max}.

**Preparatory Dietary Control**

Subjects were instructed to maintain their usual diet for 1 day before each trial. To ensure that subjects had the same kind of food, quantity, and drinks at the same time for each trial, they were required to record their diet 1 day before the first trial, and then they duplicated the diet 1 day before each of the rest of the trials. Subjects were fasted 12 h before each trial. No exercise or alcohol was allowed 3 days before the experiment. Caffeine intake was not allowed 24 h before the experiment.

**Test Meal**

To induce PHTG in each testing trial, the standard fat-rich meal was given in a milkshake form and consumed within 10 min. The milkshake consisted of a combination of 270 ml of whipping cream and 65 g of specialty ice cream with walnuts (980 kcal, 100 g fat, 17 g carbohydrate, and 3 g protein).

**Experimental Trials**

Each subject performed 1) control trial (Control; fat meal only), 2) exercise trial at 1 h after a fat meal (Post), 3) exercise trial at 1 h before a fat meal (1 h-Pre), and 4) exercise trial at 12 h before a fat meal (12 h-Pre). The order of the trials was randomized. Each subject was given 1–2 wk for recovery after each trial. Subjects reported to the laboratory after 12 h of fasting. A baseline blood sample (0 h) was taken immediately before the standard fat-rich meal intake. Additional blood samples were taken at 2, 4, 6, 8, and 24 h after the meal. In the exercise trials, subjects performed a single bout of exercise at 60% of their Vo\textsubscript{o2max} for 1 h on a treadmill at the designated time in each trial (Fig. 1). A standard normal meal (75% carbohydrate, 16% protein, and 9% fat), consisting of a Subway sandwich (595 kcal, 76 g carbohydrate, 24 g protein,

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**Table 1. Physical characteristics and fasting lipemic status of subjects**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value ± SE</th>
</tr>
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<tbody>
<tr>
<td>Age, yr</td>
<td>27 ± 1.7</td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>82 ± 1.6</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>16 ± 1.1</td>
</tr>
<tr>
<td>Fasting TG, mg/dl</td>
<td>86 ± 9.1</td>
</tr>
<tr>
<td>Fasting HDL\textsubscript{tot-Ch}, mg/dl</td>
<td>44 ± 1.5</td>
</tr>
<tr>
<td>Fasting HDL\textsubscript{2-Ch}, mg/dl</td>
<td>39 ± 1.1</td>
</tr>
<tr>
<td>Fasting HDL\textsubscript{3-Ch}, mg/dl</td>
<td>56 ± 0.7</td>
</tr>
<tr>
<td>Vo\textsubscript{o2max} ml·kg\textsuperscript{-1}·min\textsuperscript{-1}</td>
<td>48 ± 1.0</td>
</tr>
<tr>
<td>Exercise energy expenditure, kcal/day</td>
<td>2,860 ± 374</td>
</tr>
<tr>
<td>Dietary fat, %</td>
<td>29 ± 1.3</td>
</tr>
<tr>
<td>SFA, %</td>
<td>9.3 ± 0.7</td>
</tr>
<tr>
<td>MUFA, %</td>
<td>8.5 ± 0.7</td>
</tr>
<tr>
<td>PUFA, %</td>
<td>4.8 ± 0.4</td>
</tr>
<tr>
<td>Carbohydrate, %</td>
<td>54 ± 1.3</td>
</tr>
<tr>
<td>Protein, %</td>
<td>17 ± 3.2</td>
</tr>
</tbody>
</table>

Values are means ± SE. TG, triglycerides; HDL, high-density lipoprotein; HDL\textsubscript{tot-Ch}, total HDL-cholesterol; HDL\textsubscript{1}, HDL\textsubscript{2}, subtypes of HDL; Vo\textsubscript{o2max}, maximal O\textsubscript{2} consumption; SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid.
Precipitation of lipoprotein containing apolipoprotein B. Lipoprotein containing apolipoprotein B was precipitated with heparin-MnCl$_2$ (12.57 mg/ml, 7.92 g/ml) reagent. Fifty microliters of heparin-MnCl$_2$ reagent were mixed with 0.5 ml plasma. The mixed sample was incubated at 4°C for 20 min and centrifuged at 1,500 g for 20 min at 0°C. An aliquot of the supernatant was used for analysis of HDL$_{3}$-Ch. Another aliquot of the supernatant was used for precipitation of HDL$_2$. HDL$_2$-Ch precipitation. The heparin-MnCl$_2$ supernatant (200 µl from the previous step) was mixed with 20 µl of dextran sulfate solution (14.3 mg/ml) to precipitate HDL$_2$-Ch. After standing at room temperature for 20 min, the sample was centrifuged at 1,500 g at 20°C for 20 min. An aliquot of the supernatant was used for HDL$_3$-Ch analysis.

Ch analysis. The Ch of HDL$_{34}$ and HDL$_3$ was measured enzymatically by using the Ch reagent (procedure no. 353, Sigma Diagnostics). The measured value for HDL$_{34}$-Ch was multiplied by 1.1, and that for HDL$_3$-Ch was multiplied by 1.21 to correct for dilution by the reagents.

HDL$_2$-Ch calculation. HDL$_2$-Ch was calculated as the difference between HDL$_{34}$-Ch and HDL$_3$-Ch. The sum of the samples from each subject were assayed in one run. The intra-assay coefficients of variation for HDL$_{34}$-Ch, HDL$_2$-Ch, and HDL$_3$-Ch analysis were 0.3, 1.0, and 3.2%, respectively.

Data Analysis

The concentrations of plasma TG, HDL$_{34}$-Ch, HDL$_3$-Ch, and HDL$_2$-Ch were analyzed by using a two-way (times $\times$ trials) ANOVA with repeated measures on two factors. Significant F ratios (P < 0.05) were followed by one-way ANOVA and Tukey post hoc tests to identify the differences among the times and/or trials.

The magnitude of TG response was also quantified as the TG AUC score as defined by two lines: one connecting the individual TG values and one originating at the baseline sample concentration parallel to the abscissa. This area was calculated by the trapezoidal rule, using the formula

$$\text{TG AUC score} = 2 \times (L_2 + L_4 + L_6) + L_0 - 7L_0,$$

where $L_0$ is the length representing the TG concentration at $n$ h (7a).

The TG AUC score is a conventional index to indicate the plasma TG response to a fat-meal intake. In 1 h-Pre, the TG concentration measured immediately before the exercise ($-1$ h) was used as baseline for calculation of TG AUC score because the exercise caused significant increase in TG at 0 h. The TG AUC scores were analyzed by using a one-way ANOVA with repeated measures followed by Tukey's post hoc tests. Relationships between variables were evaluated by using Pearson's product-moment correlation coefficient. All data are reported as means ± SE.

RESULTS

The physical characteristics and the fasting lipemic status of subjects are shown in Table 1. Figure 2A shows the TG concentrations over time among the four trials. At 0 h (before the fat-meal intake), TG concentrations were not different among the four trials. The plasma TG concentration curves in Control and 1 h-Pre have similar patterns in which TGs peak at 4 h. The TG curve in Post shows two peaks: one at 2 h and one at 6 h. The TG curve in 12 h-Pre is the lowest in all four trials.

Compared with the control trial, the TG AUC score in 1 h-Pre was 38% lower, 12 h-Pre was 51% lower,
whereas Post was only 5% lower (P > 0.05; Fig. 2B).

There was no significant difference in TG AUC scores between 1 h-Pre and 12 h-Pre trials. The TG AUC scores in both 1 h-Pre and 12 h-Pre trials were significantly higher than that in Post, which did not differ from Control (Fig. 2B).

Figure 3 depicts the changes of HDL$_{\text{tot}}$-Ch and HDL$_2$-Ch in the four trials. There were no significant differences in HDL$_{\text{tot}}$-Ch at 0, 4, and 24 h among the four trials. At 8 h, HDL$_{\text{tot}}$-Ch in 12 h-Pre was higher than that in both Control and Post but was not different from that in 1 h-Pre. Control, Post, and 1 h-Pre were not different from each other. In Control, the only difference in HDL$_{\text{tot}}$-Ch was between 4 h and 24 h. In Post, there were no significant differences in HDL$_{\text{tot}}$-Ch at the four sample times. In both 1 h-Pre and 12 h-Pre, HDL$_{\text{tot}}$-Ch at 8 and 24 h was significantly higher than at 0 and 4 h (Fig. 3A).

HDL$_2$-Ch concentrations in the four trials did not differ from each other at 0, 4, and 8 h. At 24 h, HDL$_2$-Ch concentrations in both the 1 h-Pre and 12 h-Pre trials were significantly higher than in Control. In Control, HDL$_2$-Ch concentrations at 8 and 24 h were significantly lower than at 0 h. In Post, there were no differences in HDL$_2$-Ch concentrations among all the time points. Compared with their respective baseline concentrations, HDL$_2$-Ch in both 1 h-Pre and 12 h-Pre tended to increase at 24 h but the levels did not reach significance (Fig. 3B).
The fasting TG plasma concentration was significantly \((r = 0.41)\) correlated with the TG AUC score in Control (Table 2). Fasting HDL\(_{\text{tot}}\)-Ch and HDL\(_{2}\)-Ch were not correlated with the TG AUC score in Control. The TG AUC scores in all the three exercise trials were significantly correlated with that in Control (Table 2).

### DISCUSSION

Exercising before the fat meal had a significant effect on postprandial TG response. This finding was consistent with that reported by Aldred et al. (1), who found that exercising 15 h before a fat-rich meal significantly reduced PHTG. However, the magnitude of TG reduction (compared with the control trial), as indicated by the TG AUC score, was much larger in 12 h-Pre trial of the present study than that in the Aldred et al. study (51 vs. 31\%, respectively). This difference may be caused by the discrepancy in exercise intensities and modes as well as by the exercise times. The exercise performed in the study by Aldred et al. was brisk walking at 31\% of \(V\dot{O}_{2}\text{max}\) for 2 h, whereas the subjects in our study jogged at 60\% of \(V\dot{O}_{2}\text{max}\) for 1 h. Indeed, a recent study (27) reported that, acute, moderate-intensity aerobic exercise at 61\% \(V\dot{O}_{2}\text{max}\) significantly lowered PHTG response, whereas the exercise at 32\% \(V\dot{O}_{2}\text{max}\) did not lower PHTG. Thus, exercise intensity may be a factor in the attenuation of the PHTG response.

Most likely, the attenuated TG response is due to the exercise-induced increase in the activity of LPL, which is located at the endothelial surface of capillaries in both muscle and adipose tissues. Exercise enhances LPL activity in both muscle and adipose tissues by means of epinephrine (Epi) (15) and glucagon (4), which activate adenylate cyclase, resulting in an increase in cAMP. A cAMP-independent protein kinase, in turn, phosphorylates and thereby activates hormone-sensitive LPL (13). The increased LPL activity in muscle may play the most important role in the attenuated TG response (14). Others have shown that exercise induces human LPL gene expression predominantly in skeletal muscles, and more vigorous skeletal muscle activity may result in greater increase in LPL production (24). Because jogging involves more muscle activity than walking does, jogging may induce greater LPL activity than walking does. This also may explain why the 12 h-Pre trial in the present study had a better PHTG-lowering effect than the study by Tsetsonis and Hardman (27), which used walking (51 \% vs. 26\% reduction in PHTG, respectively). Furthermore, exercise at a higher intensity not only causes more vigorous muscle activity but also induces greater Epi and glucagon release, which may stimulate LPL activity through the cAMP cascade for lipolysis (15).

Plasma TG not only can be hydrolyzed by the LPL in muscles but also can be broken down by LPL in the bloodstream. Oscai et al. (16) observed that exercise had a heparin-like effect in releasing LPL from endothelial capillaries of the active muscle tissue to the blood circulation. With abundant LPL, the blood circulation could become an active site for TG hydrolysis (21). Therefore, the apparent decrease in TG response in the postmeal-exercise trials may be, in part, caused by the exercise-induced release of LPL from skeletal muscle into blood circulation.

In our study, subjects in Control had the highest TG AUC score among the four trials; this indicates slower removal of plasma TG than in the exercise trials. Compared with Control, the TG AUC score in 1 h-Pre was 38\% lower, 12 h-Pre was 51\% lower, whereas Post was only 5\% lower \((P > 0.05)\). The significant difference in TG AUC scores between post- and premeal-exercise trials may be because of the delayed increase of LPL activity by the action of exercise. Investigators have observed that LPL activity is not elevated immediately after exercise; rather, it increases \(4 \text{ h after exercise} (12)\) and stays elevated up to 18 h (10) to 24 h (11) after exercise. Our data showed that plasma TG peaked \(4 \text{ h after the fat meal (Control). On the basis of this evidence, it is possible that in Post the delayed increase of LPL activity may miss the rising of PHTG because TG peaked at \(4 \text{ h after the fat meal, whereas the LPL activity might be elevated to a high level at later time.}\)

During exercise, the hormone-sensitive LPL hydrolyzes intracellular TGs in skeletal muscle and myocardium, as well as adipose tissue, to provide free fatty acids (FFAs) as an energy source (20). After exercise, the expended endogenous TGs need to be replenished from exogenous TGs (2). This may cause rapid uptake of FFAs from the blood circulation. Therefore, exercise before a fat meal may favor the biochemical reaction of TG hydrolysis by LPL due to 1) the rapid removal of the reaction product (FFAs) and 2) the abundant substrates (TGs) provided by the fat meal (mass action effect). On the other hand, the postmeal exercise may impair the hydrolysis of circulating TG from the fat meal due to the elevated intracellular TG hydrolysis during exercise (15). This may explain the high response to dietary TG and slow removal of plasma TGs in Post. However, our data from Post did not support the observation by Hardman and Aldred (8), who reported that exercise (6 men and 6 women walking at 40\% \(V\dot{O}_{2}\text{max}\), 1.5 h after a fat meal reduced PHTG by 24\%. This discrepancy may be caused by the differences in the fat loading as well as in the exercise mode and duration. The fat meal used in the study by Hardman and Aldred was 84 g of fat, whereas we used 100 g of fat. The exercise used in the study of Hardman and Aldred

### Table 2. Correlation between control TG AUC score and other relevant variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Correlation with Control TG AUC Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting TG</td>
<td>0.41*</td>
</tr>
<tr>
<td>Fasting HDL(_{\text{tot}})-Ch</td>
<td>0.13</td>
</tr>
<tr>
<td>Fasting HDL(_2)-Ch</td>
<td>0.06</td>
</tr>
<tr>
<td>TG AUC score</td>
<td>0.74†</td>
</tr>
<tr>
<td>Post</td>
<td>0.74†</td>
</tr>
<tr>
<td>1 h-Pre</td>
<td>0.62*</td>
</tr>
<tr>
<td>12 h-Pre</td>
<td>0.74†</td>
</tr>
</tbody>
</table>

AUC, area under the curve. *Significant at \(P < 0.01\); †significant at \(P < 0.001\).
was brisk walking at 40% of $V_{O_2max}$ for 1.5 h. In our study, the subjects jogged at 60% of $V_{O_2max}$ for 1 h.

Our data showed that TG concentrations in 12 h-Pre were the lowest at the 8-h period among the four trials after the fat meal. The TG AUC score in 12 h-Pre was 21% lower than that in 1 h-Pre and 49% lower than that in Post. The TG peak in the 12 h-Pre trial was also the lowest among trials. This phenomenon of low response to a fat meal in 12 h-Pre may be caused by the following factors. First, in 12 h-Pre, LPL activity may be preactivated by the exercise bout 12 h before the fat-meal intake and stay elevated during the trial. During the moderate-intensity exercise in the present study, the intracellular TG may be the primary energy source for the working muscles. Second, after a prolonged exercise bout, the metabolism of the body may remain elevated for ~12 h and may also consume intracellular TGs of muscular and adipose tissue (25). Therefore, the working muscle and adipose tissues in 12 h-Pre may deplete more intracellular TG by the prestimulated LPL activity and the elevated postexercise metabolism than is depleted in 1 h-Pre. This may subsequently cause the muscle and adipose tissues "hunger" for exogenous TG to replenish their diminished storage, which may induce rapid removal of the circulating TG and result in an attenuation of the PHTG response. In contrast, in the 1 h-Pre trial, subjects had the fat meal only 1 h after the exercise. Consequently, the removal rate of the plasma TG in 1 h-Pre may not be as effective as in 12 h-Pre. This may explain why the 12 h-Pre trial tended to have a lower response to a fat meal than did the 1 h-Pre trial.

Tsetsonis and Hardman (26) observed that exercise 15 h before a fat meal, at low intensity (32% $V_{O_2max}$) and for a longer time (3 h), significantly attenuated PHTG, whereas exercise at the same intensity but for a shorter period (1.5 h) did not affect PHTG (27). This evidence may verify that exercise for a longer period at lower intensity causes more intracellular TG depletion and subsequently induces rapid uptake of plasma TGs, resulting in an attenuation of PHTG. These findings suggest that exercise duration also may be a factor in attenuation of PHTG response.

Our data did not reveal a relationship between Control TG AUC score and fasting HDL$_{tot}$-Ch or HDL$_2$-Ch. This finding is in agreement with the studies by Aldred et al. (1) and by Hardman and Aldred (8) but did not support the study by Patsch et al. (19), who found that the fasting HDL$_2$-Ch was inversely correlated ($r = -0.63$) with the magnitude of TG AUC score. This discrepancy may be due to different amounts of fat loading. The amount of fat loading in the present study and in the two confirming studies (1, 8) was 100, 82, and 84 g, respectively. In contrast, Patsch et al. (19) used 130 g of fat in their study. The lower fat loading in the present study and the two supporting studies (1, 8) may not be high enough to reveal the lipid-carrying capability of HDL$_2$-Ch. Whether HDL$_2$-Ch has a negative correlation with postprandial lipemia still remains equivocal.

The significant difference in HDL$_{tot}$-Ch among trials was found only at 8 h after the fat meal, at which time the HDL$_{tot}$-Ch level in 12 h-Pre was significantly higher than that in both Control and Post but not different from that in 1 h-Pre. Aldred et al. (1) reported that HDL$_{tot}$-Ch in the 15-h premeal-exercise trial was significantly ($P < 0.05$) higher than that in the nonexercise trial at 3, 4, and 5 h after ingestion of the fat meal. Although there is a time discrepancy, there is agreement on the increase in HDL$_{tot}$-Ch induced by exercise in the study by Aldred et al. (1) and in the present study. The discrepancy in the time of HDL$_{tot}$-Ch increase may be caused by the different exercise time. In the study of Aldred et al., the subjects exercised 3 h earlier relative to the meal than did subjects in the present study. In two similar studies by Tsetsonis and Hardman (26, 27), no changes in HDL$_{tot}$-Ch were observed in a 6-h period after the fat meal. This observation is compatible with our findings.

HDL$_{tot}$-Ch concentrations in both Control and Post at 8 and 24 h were different from the baseline concentrations (0 h). In comparison, HDL$_{tot}$-Ch concentrations at 8 and 24 h in both premeal-exercise trials were higher than their respective baseline concentrations. These observations support the previous findings by Kantor et al. (10, 11), who found HDL-Ch was increased at 24 and 42 h after an exercise bout.

HDL$_2$-Ch concentrations at 8 and 24 h in the Control trial were significantly lower than baseline values, whereas HDL$_2$-Ch values in Post remained unchanged. This result somewhat supported the findings reported by Aldred et al. (1) and Tsetsonis and Hardman (27). They found that HDL$_2$-Ch remained unchanged in a 6-h postprandial period in the exercise trials performed either 1.5 h after a fat meal (1) or 15 h before a fat meal (27). However, in neither study was HDL$_2$-Ch measured at 24 h after the meal. In contrast, in the present study, HDL$_2$-Ch in both the premeal-exercise trials tended to increase at 24 h after the meal. The 12 h-Pre trial had the highest HDL$_2$-Ch concentration at 24 h. These observations were compatible with the findings reported by Kantor et al. (10), who showed that the concentration of HDL$_{tot}$-Ch and HDL$_2$-Ch increased 18 h after a marathon. Kantor et al. also reported that HDL$_{tot}$-Ch and HDL$_2$-Ch increased even 48 and 72 h after the exercise. Because the formation of HDL$_2$-Ch increases the Ch-carrying capacity (18), the higher level of HDL$_2$-Ch induced in the premeal-exercise trials may be beneficial in the prevention of atherosclerosis.

Acute exercise may stimulate the activity of lecithin-cholesterol acyl transferase (LCAT) (5), which converts HDL$_2$-Ch to HDL$_3$-Ch. The increased HDL$_{tot}$-Ch and HDL$_2$-Ch observed in the premeal-exercise trials in the present study may be due to abundant substrates from TG hydrolysis by LPL and enhanced LCAT activity. Also, this may explain why the HDL$_2$-Ch concentrations in the premeal-exercise trials were higher than those in Control. The pattern of HDL$_2$-Ch concentrations in the four trials at 24 h (Fig. 3B) is similar to TG concentrations at 8 h (Fig. 2A) and the TG AUC score (Fig. 2B) in a reversed fashion, indicating that lower
TG response corresponds to higher HDL_{2-Ch} concentration or vice versa. This phenomena may be due to the increased components from TG breakdown and elevated LCAT activity through exercise for HDL_{2-Ch} synthesis (5). Because elevated PHTG concentrations are associated with increased LDL concentrations and reduced HDL-Ch concentrations, hypertriglyceridemic patients are often at increased risk for development of CVD (17, 18). The prolonged exposure of arterial wall to TG-rich lipoprotein remnants may enhance the atherogenic process (22). The premeal-exercise intervention, especially exercise 12 h before a fat meal, may prevent this detrimental effect.

In conclusion, the results from the present study indicate that exercising before a fat-rich meal can significantly attenuate the PHTG response, whereas exercising after the fat meal does not have an effect on PHTG. Exercising 12 h before the fat meal may have a better overall TG-lowering effect than exercising 1 h before the fat-meal intake. The TG-lowering effect in the premeal-exercise trials is accompanied by elevation of HDL_{2-Ch} and HDL_{3-Ch}. These findings suggest that the detrimental effects of eating fat during a meal can be decreased by exercise before the meal. Furthermore, exercising 12 h before a fat-rich meal intake may magnify this beneficial effect on the TG response and HDL metabolism, which may more efficiently blunt the postprandial-induced atherosclerotic process.

We thank Dr. Richard H. Cox for advice on the statistical procedures.

This study was supported by a grant from American College of Sports Medicine.

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Received 3 March 1998; accepted in final form 1 June 1998.

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