

Christos S. Katsanos, Peter W. Grandjean and Robert J. Moffatt

J Appl Physiol 96:181-188, 2004. First published Aug 29, 2003; doi:10.1152/jappphysiol.00243.2003

You might find this additional information useful...

This article cites 41 articles, 23 of which you can access free at:

<http://jap.physiology.org/cgi/content/full/96/1/181#BIBL>

This article has been cited by 2 other HighWire hosted articles:

Acute exercise-induced changes in basal VLDL-triglyceride kinetics leading to hypotriglyceridemia manifest more readily after resistance than endurance exercise

F. Magkos, Y. E. Tsekouras, K. I. Prentzas, K. N. Basioukas, S. G. Matsama, A. E. Yanni, S. A. Kavouras and L. S. Sidossis

J Appl Physiol, October 1, 2008; 105 (4): 1228-1236.

[\[Abstract\]](#) [\[Full Text\]](#) [\[PDF\]](#)

Effect of exercise duration on postprandial hypertriglyceridemia in men with metabolic syndrome

J. Q. Zhang, L. L. Ji, D. L. Fogt and V. S. Fretwell

J Appl Physiol, October 1, 2007; 103 (4): 1339-1345.

[\[Abstract\]](#) [\[Full Text\]](#) [\[PDF\]](#)

Updated information and services including high-resolution figures, can be found at:

<http://jap.physiology.org/cgi/content/full/96/1/181>

Additional material and information about *Journal of Applied Physiology* can be found at:

<http://www.the-aps.org/publications/jappl>

This information is current as of December 1, 2009 .

Effects of low and moderate exercise intensity on postprandial lipemia and postheparin plasma lipoprotein lipase activity in physically active men

Christos S. Katsanos,¹ Peter W. Grandjean,² and Robert J. Moffatt¹

¹Department of Nutrition, Food and Exercise Sciences, Florida State University, Tallahassee, Florida 32304; and

²Department of Health and Human Performance, Auburn University, Auburn, Alabama 36849

Submitted 10 March 2003; accepted in final form 25 August 2003

Katsanos, Christos S., Peter W. Grandjean, and Robert J. Moffatt. Effects of low and moderate exercise intensity on postprandial lipemia and postheparin plasma lipoprotein lipase activity in physically active men. *J Appl Physiol* 96: 181–188, 2004. First published August 29, 2003; 10.1152/jappphysiol.00243.2003.—This study was designed to assess differences in the intensity of exercise to attenuate postprandial lipemia (PPL). Thirteen healthy men (age 23.8 ± 0.9 yr) participated in three random-ordered trials: in low- (25% peak oxygen consumption; Low) and moderate-intensity (65% peak oxygen consumption; Mod) exercise trials, which were completed 1 h before a high-fat meal (1.3 g fat/kg body mass), and a control (Con), fat meal only, trial. Venous blood samples were obtained before the fat meal, and at 2, 4, 6, 8, and 20 h after the fat meal. PPL in the Mod trial (267 ± 50 mg·dl⁻¹·8 h) was lower compared with that in either Con (439 ± 81 mg·dl⁻¹·8 h) or Low (403 ± 91 mg·dl⁻¹·8 h) trials ($P < 0.05$), whereas there was no difference in PPL between Con and Low trials ($P > 0.05$). High-density lipoprotein cholesterol (HDL-C) and HDL subtype 2 cholesterol were not different between or within trials ($P > 0.05$). Postprandial insulinemia was lower in the Mod trial (20.5 ± 5.7 μ IU·ml⁻¹·8 h; $P < 0.05$), but not in the Low trial (31.4 ± 4.7 μ IU·ml⁻¹·8 h), compared with that in the Con trial (34.9 ± 5.0 μ IU·ml⁻¹·8 h). Postheparin lipoprotein lipase activity at 8 h was higher in the Low trial compared with that in either Con or Mod trials, whereas there were no differences between trials at 20 h. These results suggest that, when exercise is performed 1 h before a fat meal, only exercise of moderate but not of low intensity attenuates PPL and that this effect is not associated with changes in postheparin lipoprotein lipase activity.

fat meal; triglycerides; hypertriglyceridemia; insulinemia

CORONARY HEART DISEASE (CHD) remains a leading cause of mortality in Western societies. Although plasma low-density lipoprotein cholesterol has traditionally been linked to an increased risk for CHD (32a), plasma triglycerides (TG) appear to also play a role in the progression of CHD, especially as evidenced during the postprandial period (18, 29). The concept that the process of atherosclerosis is a postprandial phenomenon appeared more than 20 years ago (42). Evidence has been provided that suggests that plasma TG are an important determinant of plasma cholesterol metabolism and that the negative relationship between high-density lipoprotein cholesterol (HDL-C) and CHD is a result of a positive association between CHD and plasma postprandial TG levels (29). Because people spend a major part of their daily life in a postprandial state, use of interventions that could attenuate the magnitude of post-

prandial TG levels would be important in retarding the process of atherosclerosis.

Studies have shown that prolonged aerobic exercise (24, 37–39, 41) decreases the magnitude of postprandial lipemia (PPL) induced by the consumption of a high-fat meal. The energy expended during an exercise session appears to be a major determinant of the decrease in PPL (30). However, factors related to the intensity of the aerobic exercise have not been completely investigated. Exercise of moderate intensity for 90 min, completed some hours before a high-fat meal, reduced PPL to a greater extent compared with exercise of the same duration but of low intensity (37). However, under the same conditions, there was no difference in PPL between exercise sessions of low and moderate intensity when the two exercise sessions were matched for energy expenditure (38). In the latter study (38), the exercise was undertaken several hours before the induction of PPL. However, the timing of exercise relative to the high-fat meal appears to be important in regulating PPL (41), and factors implicated in the attenuation of PPL, such as lipoprotein lipase (LPL) activity (LPLA), appear to be time dependent (20). Also, because the intensity of exercise has an effect on the duration that metabolic changes are sustained, it is possible that, with respect to PPL, the intensity effects of exercise performed several hours before a high-fat meal could be different from those of exercise completed 1 h before the meal.

A single bout of exercise has been shown to affect HDL-C metabolism (14), and it has been suggested that the concentration of HDL-C depends on the metabolism of TG-rich particles (28, 35). Zhang et al. (41) were not able to document any changes in HDL-C during the postprandial lipemic phase when PPL was induced immediately at the end of the exercise, but there was a delayed (~24 h) increase in the HDL subtype 2 cholesterol (HDL₂-C) subfraction.

The mechanisms of the attenuated postprandial plasma TG response after a single bout of exercise are not well understood. It has been shown that exercise attenuates PPL regardless of the relative contributions of fat and carbohydrate to the total energy expended during exercise (25). Furthermore, the attenuated PPL response does not seem to be a result of the energy deficit, per se, induced by the exercise (12). In explaining the attenuated postprandial lipemic response after exercise, the emphasis has been placed on LPLA (16), which is the enzyme responsible for the intravascular hydrolysis of plasma TG. It is known that exercise increases LPLA in skeletal muscle (34), thus providing a means for enhanced removal of plasma TG.

Address for reprint requests and other correspondence: C. S. Katsanos, Metabolism Unit, Shriners Burns Hospital, 815 Market St., Galveston, TX 77550 (E-mail: cskatsan@utmb.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.

LPLA measured in postheparin plasma is elevated after prolonged exercise (9, 17), presumably because of an increase in the muscle isoform of LPL. However, a relationship between an increase in plasma postheparin LPLA and a decrease in PPL after exercise has not been documented. Furthermore, no studies have investigated the effects of the exercise intensity on postprandial plasma postheparin LPLA or hepatic TG lipase activity (HTGLA), with the latter being involved in the metabolism of HDL-C in the liver.

Insulin appears to play a role in the regulation of PPL. Specifically, insulin controls the secretion of very-low-density lipoprotein (VLDL) particles (2, 23), which are an important contributor to the total plasma TG during the postprandial lipemic phase. Also, insulin stimulates adipose tissue LPL during the postprandial period (31), whereas it decreases the activity of muscle LPL (19).

Because exercise of moderate intensity results in greater intramuscular TG utilization than that of low intensity (33), it can be hypothesized that exercise of moderate intensity also results in greater stimulation of muscle LPLA compared with that of low intensity. This would be because LPL liberates chylomicron and VLDL fatty acids that can be used to replenish muscle TG used during exercise. On the other hand, exercise that results in only minimal oxidation of muscle TG, such as that performed at 25% maximal oxygen consumption ($\dot{V}O_{2\max}$) (33), is expected to increase muscle LPLA and attenuate PPL to a lesser degree than exercise performed at moderate intensity. Direct evidence based on published reports (41), as well as research from our laboratory (unpublished observations), suggests that a single bout of exercise performed at moderate intensity immediately before a fat meal attenuates PPL.

The purpose of the present study was to determine whether the attenuated lipemic response to a fat meal ingested 1 h after the end of exercise is affected by the intensity that the exercise is performed. We hypothesized that exercise of moderate intensity attenuates PPL more than exercise of low intensity. Furthermore, a second objective was to determine the effects of exercise intensity on postheparin plasma LPLA, HTGLA, as well as on the metabolism of HDL-C as observed during postprandial lipemic conditions. Insulin response during the postprandial phase was also determined in an effort to investigate physiological mechanisms associated with the PPL response.

METHODS

Subjects. Subjects were recruited through advertisement in the local newspaper and flyers posted in the college community. Thirteen healthy men participated in this study after giving written, informed consent. The study was approved by the Florida State University Human Subjects Committee. Subjects were 23.8 ± 0.9 (mean \pm SE) yr of age, weighed 77.9 ± 3.0 kg, and were 179.1 ± 2.0 cm tall. Peak oxygen consumption ($\dot{V}O_{2\text{peak}}$) was measured at 49.5 ± 2.0 ml \cdot kg $^{-1}\cdot$ min $^{-1}$, and the percent body fat was $11.9 \pm 1.7\%$. All subjects were physically active, were nonsmokers, and were not taking any medication or nutritional supplements known to affect lipid and carbohydrate metabolism. This information was obtained by use of questionnaires that were completed during the subject's first visit to the laboratory. Before participating in the study, subjects were instructed and completed a 3-day (2 weekdays and 1 weekend day) food diary, which was analyzed by use of diet-analysis computer software (Nutritionist Five, First DataBank, San Bruno, CA). One subject was

excluded from the study because of extreme dietary patterns (defined as dietary fat intake either $<15\%$ or $>45\%$). Subject dietary information is presented in Table 1.

Preliminary tests. Subjects took part in two preliminary walking exercise tests performed on a treadmill. First, a 20-min submaximal treadmill test was performed for the purpose of establishing a work-to-oxygen consumption ($\dot{V}O_2$) relationship. Second, a treadmill walking protocol was used to determine each subject's $\dot{V}O_{2\max}$, while the subject walked at a preselected speed. Because in almost all of the subjects an approximation of $\dot{V}O_{2\max}$ was known, a speed was selected with the purpose of reaching exhaustion in 10–12 min (5). During this test, the grade was increased by 2.5% every minute for the first 6 min and by 1.5% thereafter, until exhaustion. Because a plateau for $\dot{V}O_2$ was not documented, the maximal $\dot{V}O_2$ measured was considered as $\dot{V}O_{2\text{peak}}$. The test for $\dot{V}O_{2\text{peak}}$ was considered valid when the following criteria were satisfied: heart rate ± 10 beats/min of $220 - \text{age}$, respiratory exchange ratio value ≥ 1.15 , and voluntary exhaustion (rating of perceived exertion ≥ 19). If the above criteria were not satisfied by the subject, the test was repeated. Throughout the duration of the preliminary tests, expired gases and heart rate were monitored, and average values were recorded in 30-s intervals (True-max 2400 metabolic measurement system, Consentius Technologies, Sandy, UT).

Study trials. All subjects participated in three trials, two of which included exercise whereas the third one served as a control/no exercise trial (Con). During the exercise trials, subjects walked on the treadmill at an intensity corresponding to 25% $\dot{V}O_{2\text{peak}}$ (Low) and 65% $\dot{V}O_{2\text{peak}}$ (Mod). The three trials were performed in a random order, with at least 1 wk separating each trial. The protocol followed during each of the trials is shown in Fig. 1. Subjects reported to the laboratory 5 h before a high-fat meal was consumed (fat meal ingested at ~ 12 noon) and remained for 8 h after that. The time required to expend 1,100 kcal was estimated before each exercise trial and varied according to exercise intensity. This estimation was used to determine when to initiate the exercise session after the baseline blood draw, so that the exercise was completed 1 h before the fat meal ingestion. In the Con trial, subjects rested in the laboratory for 5 h before the fat meal. A light snack (plain bagels, cream cheese, and orange juice) was provided after the baseline blood draw. The amount and the time course of the snack consumption were replicated across the three trials. Besides the test meal and the snack, subjects were allowed to drink only water during the 13-h period spent in the laboratory.

Subjects were asked to abstain from any type of aerobic exercise, weight lifting, and alcohol for 3 days before their participation in this study and throughout each trial. Subjects were requested to record their diet for the day before their first trial and replicate that diet during the day before their subsequent trials. Also, after the 8-h blood sample and until subject's return to the laboratory the next day for the 20-h blood sample, subjects were instructed to record the type and

Table 1. Fasting biochemical variables and dietary information of the subjects

Plasma TG, (mg/dl)	70.5 \pm 4.0
Plasma TC, mg/dl	146.0 \pm 4.9
Plasma HDL-C, mg/dl	51.3 \pm 1.8
Plasma HDL ₃ -C, mg/dl	39.1 \pm 1.1
Plasma HDL ₂ -C, mg/dl	12.3 \pm 1.2
Plasma insulin, μ IU/ml	6.9 \pm 0.6
Blood glucose, mg/dl	85.2 \pm 1.2
Energy intake, kcal/day	2963 \pm 319
Dietary fat total, g/day	109.5 \pm 16.1
Saturated fat, g/day	37.4 \pm 6.0
Cholesterol, mg/day	417.0 \pm 90.9

Values are means \pm SE. TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol. HDL-C₂ and HDL-C₃, HDL subtype 2 and 3 cholesterol.

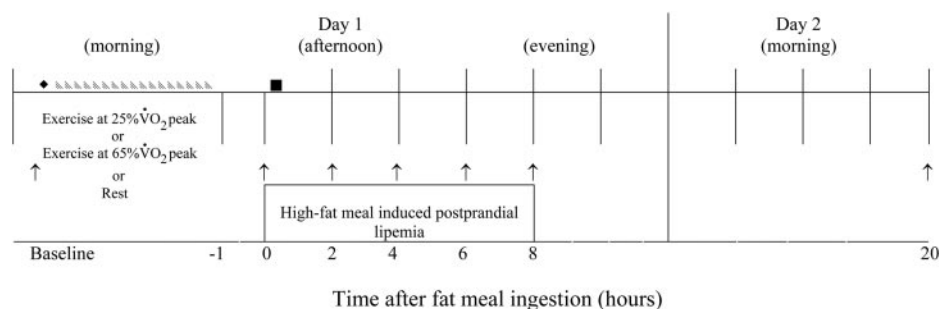


Fig. 1. Study protocol. Baseline, ~5 h before the fat meal ingestion at 0 h. $\dot{V}O_{2\text{peak}}$, peak oxygen consumption; \uparrow , venous blood draws; \blacklozenge , standardized snack; \blacksquare , test meal.

amount of any food or drink consumed, as well as the time it was consumed, with the purpose of replicating the recorded diet during the following trials. Compliance with the instructions was verbally verified each time the subject arrived to the laboratory.

The exercise sessions were performed in the morning of *day 1* (Fig. 1). Fine adjustments on the treadmill grade were made during the first 20 min of the exercise sessions, and when necessary thereafter, so that the $\dot{V}O_2$ remained always close to the desired one ($\pm 5\%$). During each exercise session heart rate, expired gases, and the calculated energy expenditure were continuously monitored for the first 20 min of exercise by using the metabolic measurement system indicated earlier. After that, physiological exercise responses were monitored for 5 min, in 15-min intervals for the first 1.5 h and in 30-min intervals thereafter, until the end of exercise. During exercise subjects were allowed to consume water ad libitum.

Blood samples were obtained by venous puncture at baseline, immediately before the test meal ingestion (0 h), and at 2, 4, 6, 8, and 20 h thereafter (Fig. 1). Blood samples were collected into vacutainer tubes after the subject remained seated for 15 min. The baseline blood sample was drawn after a 12-h overnight fast and the 20-h blood sample after an 8-h overnight fast. The 8-h and 20-h blood samples were collected by use of an infusion set, which allowed for the infusion of heparin. Specifically, the following procedure was employed for the 8-h and 20-h blood samples: after initial blood samples were obtained, 75 IU of sodium heparin (1,000 USP IU/ml, Elkins-Sinn, Cherry Hill, NJ) per kilogram of body weight were infused over a 1-min period. Ten minutes after the intravenous heparin infusion (36), blood was drawn into a 10-ml sodium heparin tube for the determination of plasma LPLA and HTGLA. Blood samples for the determination of baseline LPLA and HTGLA were taken on a separate day from the three trials and under the same controlled conditions of the baseline blood sample during the trials. This approach deemed it necessary to avoid any increase in the rate of intravascular hydrolysis of TG during the PPL period because of the heparin infusion for the collection of baseline blood samples in each trial. This single sample was used as a baseline for LPLA and HTGLA for all three trials.

Test meal. The test meal consisted of whipping cream and ice cream and was given in a milkshake form. The amount of ice cream and whipping cream were weighted so that the amount of fat provided by the meal was according to the subject's body weight [1.3 g of fat per kg body mass (24, 38)]. The test meal remained the same for the subject's three trials. Fat (mainly long chain fatty acids), carbohydrate, and protein contributed 84% (saturated fat 53%), 15%, and 4% of the total calories, respectively. Subjects were asked to consume the test meal within 15 min. Subjects were encouraged throughout their presence in the laboratory to report any symptoms that might relate to the test meal ingestion, such as diarrhea or gastrointestinal discomfort. With the exception of two subjects who reported some gastrointestinal discomfort, the test meal was tolerated well by the subjects.

Blood chemistry analyses. Blood collected in a 3-ml sodium heparin tube was analyzed immediately for hemoglobin, hematocrit, and blood glucose. Hemoglobin was determined in duplicate (OMNI 3

modular analyzer, AVL Scientific, Roswell, GA) and hematocrit in triplicate (by use of the microcapillary method), and they were both used for estimation of plasma volume changes (7). Blood glucose concentration was determined in duplicate by using a blood glucose analyzer (model 2300 STAT, Yellow Springs Instruments, Yellow Springs, OH).

The remaining blood samples were immediately centrifuged (2,100 g) for 15 min at 4°C by use of a refrigerated centrifuge (Sorvall RT7, DuPont Sorvall Products, Newtown, CT). After centrifugation, plasma samples for the determination of TG, insulin, HDL-C, HDL subtype 3 cholesterol (HDL₃-C), LPLA, and HTGLA were placed in labeled microcentrifuge tubes and stored at -70°C for later analysis. Determination of true TG was based on an enzymatic procedure using a commercially available kit (procedure no. 337, Sigma Diagnostics, St. Louis, MO). The determinations of HDL-C and HDL₃-C were performed in 1.0 ml of plasma according to methods by Warnick and Albers (40) and Gidez et al. (10), and only at baseline and 0, 4, 8, and 20 h. Plasma HDL₂-C was calculated as the difference between HDL-C and HDL₃-C. Plasma insulin was assayed in 25 μ l of plasma by use of a commercially available ELISA kit (cat no. 008-10-1113-01, ALPCO Diagnostics, Windham, NH). Total plasma lipase activity and HTGLA were determined in the postheparin plasma on the basis of methods by Krauss et al. (21) and Belfrage and Vaughan (1), with modifications reported by Thompson et al. (36). Plasma LPLA was determined as the difference between total plasma lipase activity and HTGLA. Samples from the same subject were assayed in one run. Intra-assay coefficients of variation were 2.7% for TG, 1.8% for HDL-C, 2.1% for HDL₃-C, 5.8% for insulin, 5.4% for total plasma lipase activity, and 6.6% for HTGLA.

Calculations and statistical analyses. PPL was quantified by calculating the area under the plasma TG curve (TGAUC) over the 8-h postprandial period adjusted to the baseline TG value, according to the formula (27)

$$\text{PPL (mg}\cdot\text{dl}^{-1}\cdot\text{8 h)} = 2[n_2 + n_4 + n_6] + n_8 - 7n_B$$

In the formula, n_B represents the plasma TG value at baseline, and n_2 to n_8 represent plasma TG values from 2 to 8 h after the meal ingestion. The same procedure was also used to quantify postprandial insulinemia ($\mu\text{IU}\cdot\text{ml}^{-1}\cdot\text{8 h}$).

Mean values for metabolic variables of interest were compared by using either 3×7 (TG, insulin, glucose), 3×5 (HDL-C, HDL₂-C, HDL₃-C), or 3×3 (LPLA, HTGLA) (trial \times time) ANOVA with repeated measures. Statistically significant interactions were followed by Tukey's honestly significant difference post hoc procedure. One-way ANOVA with repeated measures was used to compare mean areas under the curve between the three trials and analyze differences in means for physiological variables between the two exercise sessions. Correlation analyses were performed by using the Pearson's product-moment correlation coefficient. Statistical significance was set at $P < 0.05$, and data are reported as means \pm SE.

RESULTS

Exercise sessions. Subjects exercised for a mean duration of 237.5 ± 9.0 min during the Low trial and 90.8 ± 3.7 min during the Mod trial. Mean $\dot{V}O_2$ during the exercise sessions for the Low and Mod trials corresponded to 25.1 ± 0.3 and $65.0 \pm 0.5\%$ $\dot{V}O_{2\text{ peak}}$, respectively. Respiratory exchange ratio values were 0.88 ± 0.01 and 0.94 ± 0.01 for the Low and Mod trials, respectively. Data comparing mean metabolic responses between the two exercise sessions are presented in Table 2.

Blood chemistry. ANOVA results revealed a statistically significant trial \times time interaction ($P < 0.05$) for plasma volume changes, and, therefore, all plasma/blood chemistry values reported in this investigation have been adjusted to reflect these changes.

TG was the variable of main interest in this investigation. Figure 2A shows the TG response over the course of the three trials. Mean TG value reached a peak at 4 h after the high-fat meal in all three trials (Con = 151.8 ± 18.5 mg/dl; Low = 140.8 ± 21.7 mg/dl; Mod = 120.6 ± 11.84 mg/dl). Statistical analysis indicated a significant trial \times time interaction for the TG response. Follow-up post hoc procedure indicated that at 4 h the mean TG concentration for the Mod trial, but not that of the Low trial, was lower compared with the Con trial ($P < 0.05$). Information regarding the TGAUC for the three trials is presented in Fig. 2B. In the Mod trial, TGAUC was significantly lower by 39% compared with the Con trial ($P < 0.01$) and by 34% compared with the Low trial ($P < 0.05$). Compared with the control trial the TGAUC for the Low trial was lower by only 8% ($P > 0.05$). Mean plasma values for HDL-C, HDL₃-C, and HDL₂-C are shown in Table 3. There was a statistically significant trial \times time interaction only for the HDL₃-C subfraction ($P < 0.05$).

Plasma insulin response during the course of the three trials is shown in Fig. 3A. Postprandial insulinemia was quantified the same way as PPL, by using the area under the plasma insulin curve over the 8-h postprandial period adjusted to the baseline value (INAUC). Compared with the Con trial, only the mean INAUC for the Mod trial (moderate exercise intensity) was significantly lower (by 41%; $P < 0.05$). Mean INAUC for the Mod trial was also 35% lower compared with that of the Low trial, but this difference was not statistically significant (Fig. 3B). Mean glucose data collected during the trials are shown in Fig. 4. Statistical analysis showed a significant trial \times time interaction. Post hoc procedure indicated that the glucose concentration at 2 h was higher in the Low trial compared with that in the Con trial ($P < 0.05$).

Table 2. Metabolic responses estimated by indirect calorimetry during the exercise sessions

	Low-Intensity Exercise	Moderate-Intensity Exercise
CHO _{ox} , % cal	60.6 ± 3.1	79.5 ± 2.4*
CHO _{ox} , cal·kg ⁻¹ ·min ⁻¹	37.2 ± 2.6	127.7 ± 6.7*
FAT _{ox} , % cal	39.4 ± 3.1	20.5 ± 2.4*
FAT _{ox} , cal·kg ⁻¹ ·min ⁻¹	23.9 ± 1.8	32.5 ± 3.7†

Values are means \pm SE; CHO_{ox}, carbohydrate oxidation; FAT_{ox}, fat oxidation; Estimation of the contribution of carbohydrate and fat oxidation to the total energy expenditure was done according to the methods of Lusk (23a), with the assumption of no protein oxidation. Significant differences between the low- and moderate-intensity exercise sessions. * $P < 0.01$; † $P < 0.05$.

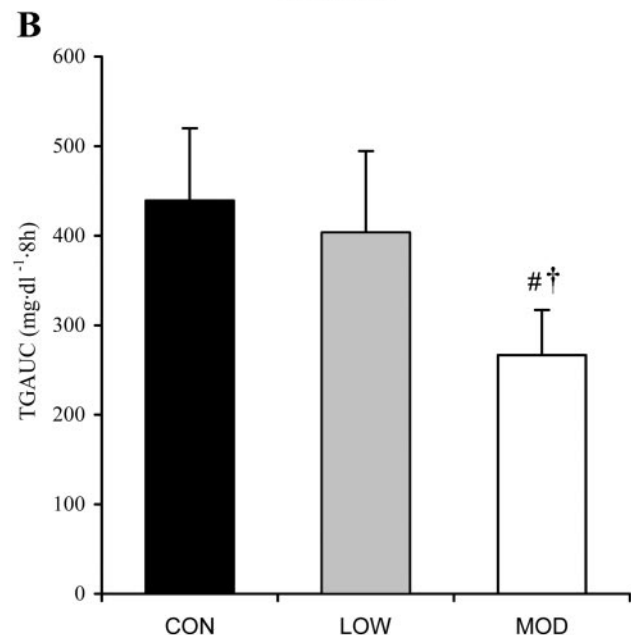
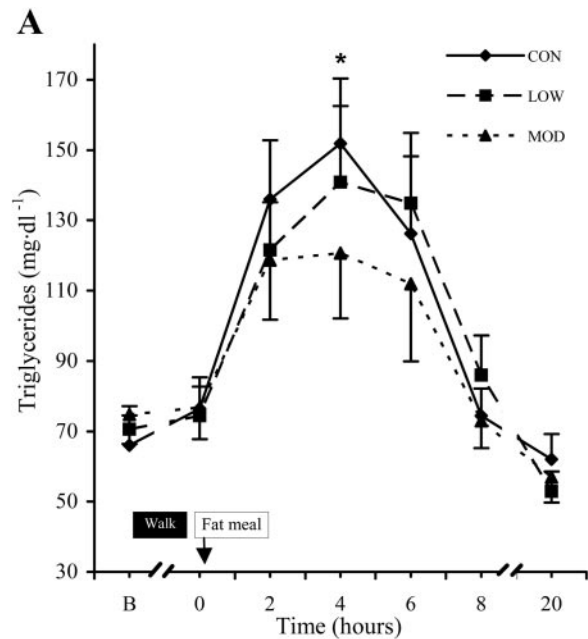


Fig. 2. Mean plasma triglyceride response after the high-fat meal ingestion (A) and triglyceride area under the curve (TGAUC; B) for the low (Low)- and moderate-intensity (Mod) walking exercise and control (Con) trials. B, baseline blood sample. *Significant difference between Con and Mod trials at this time point ($P < 0.05$); Mod trial significantly different from Con (# $P < 0.01$) and Low († $P < 0.05$) trials.

Data for postheparin LPLA and HTGLA are presented in Table 4. With respect to the LPLA, statistical analysis indicated a significant interaction between the trial and time factors. Post hoc procedure indicated that at 8 h mean LPLA in the Low trial was higher than that in the Con trial as well as that in the Mod trial (for both $P < 0.05$). Within trials, there were no significant changes for LPLA over time in the Con trial. Within the Low trial, LPLA at 8 h was higher than that at baseline ($P < 0.05$), but it was not different from baseline at 20 h, whereas within the Mod trial LPLA at 20 h was higher

Table 3. Plasma HDL-C and its subfractions during the trials

	B	0 h	4 h	6 h	20 h
HDL-C					
Con	50.5±3.3	50.8±3.4	49.7±3.2	51.7±3.4	52.6±3.3
Low	52.5±3.2	54.7±3.1	53.4±3.1	53.7±3.3	53.7±3.2
Mod	50.9±3.4	51.6±3.3	50.9±3.6	52.2±3.5	53.0±3.4
HDL₃-C					
Con	38.4±1.9	37.9±1.7	37.5±1.7	39.4±1.9	39.5±1.7
Low	40.0±1.9*	41.8±1.8*†	40.5±1.8*†	40.7±1.9	40.5±1.8
Mod	38.8±1.9	39.5±1.8*	38.6±2.0	40.3±1.9	40.1±1.8
HDL₂-C					
Con	12.1±2.1	12.9±2.3	12.2±2.3	12.3±2.7	13.1±2.3
Low	12.5±2.3	12.9±2.1	12.9±2.3	13.0±2.4	13.2±2.3
Mod	12.2±1.9	12.1±2.1	12.3±2.2	11.8±2.4	12.9±2.2

Values are means ± SE; all values are in mg/dl and have been adjusted for plasma volume changes. Low, low-intensity exercise trial; Mod, moderate-intensity exercise trial; Con, control trial; B, baseline blood sample (~5 h before the fat meal ingestion); 0 h, time of the fat meal ingestion (1 h after the end of exercise for the exercise trials). Significant time effect for HDL-C when means were collapsed across trials; *significantly different from Con trial at this time point ($P < 0.05$); †significantly different from Mod trial at this time point ($P < 0.05$).

than that at baseline ($P < 0.05$), but it was not different from baseline at 8 h. Regarding the HTGLA response, no statistically significant differences were observed.

Correlational analyses. $\dot{V}O_{2\text{ peak}}$ correlated with HDL₂-C ($r = 0.70$; $P < 0.01$) but not with HDL-C ($r = 0.50$; $P > 0.05$). It was found that the change in HDL-C (20-h value – baseline value) was positively correlated with the corresponding changes in both HDL₂-C and HDL₃-C in the two exercise trials (in the Low trial, with HDL₂-C, $r = 0.61$, $P < 0.05$; with HDL₃-C, $r = 0.60$, $P < 0.05$; and in the Mod trial, with HDL₂-C, $r = 0.81$, $P < 0.001$; with HDL₃-C, $r = 0.74$, $P < 0.01$). However, in the Con trial the change in HDL-C was correlated only with the change in HDL₃-C ($r = 0.57$; $P < 0.05$) and not with the change in HDL₂-C ($r = 0.43$; $P > 0.05$).

Correlations of postheparin LPLA and HTGLA with several variables of interest were examined. LPLA at 8 h was positively correlated with the change in INAUC (Con value – exercise trial value) only in the moderate exercise trial ($r = 0.69$; $P < 0.05$). LPLA correlated significantly with HDL-C, but not with HDL₂-C. Specifically, there was a positive correlation between LPLA and HDL-C at the 8-h time point for the Low trial ($r = 0.66$; $P < 0.05$) and at the 20-h time point for the Mod trial ($r = 0.60$; $P < 0.05$). On the other hand, HTGLA correlated significantly with HDL₂-C but not with HDL-C. Specifically, at baseline, individuals with higher HTGLA had lower HDL₂-C ($r = -0.80$; $P < 0.01$). Also, in the exercise trials and at 8 h there was an inverse relationship between HTGLA and HDL₂-C (Low trial, $r = -0.82$; $P < 0.001$; and Mod trial, $r = -0.73$; $P < 0.01$).

DISCUSSION

The main finding of this investigation is that the intensity of an exercise session (completed 1 h before the consumption of a high-fat meal) plays a role in attenuating the magnitude of postprandial TG response. Low-intensity exercise did not affect the magnitude of PPL. On the other hand, moderate-intensity exercise attenuated the postprandial lipemic response. With respect to the latter, the findings of the present study are

in agreement with those that have previously been reported by Zhang et al. (41), when subjects completed 1 h of exercise at 60% of $\dot{V}O_{2\text{ max}}$ immediately before the consumption of a fat-rich meal. Specifically, in the present study there was a 39% decrease in the magnitude of PPL compared with the control trial, which is comparable to the 38% decrease reported in the study by Zhang et al.

Because it is known that the initial TG concentration is a determining factor of the magnitude of PPL (4), and because exercise has previously been shown to affect plasma TG under conditions similar to those of this study (41), the fasting TG

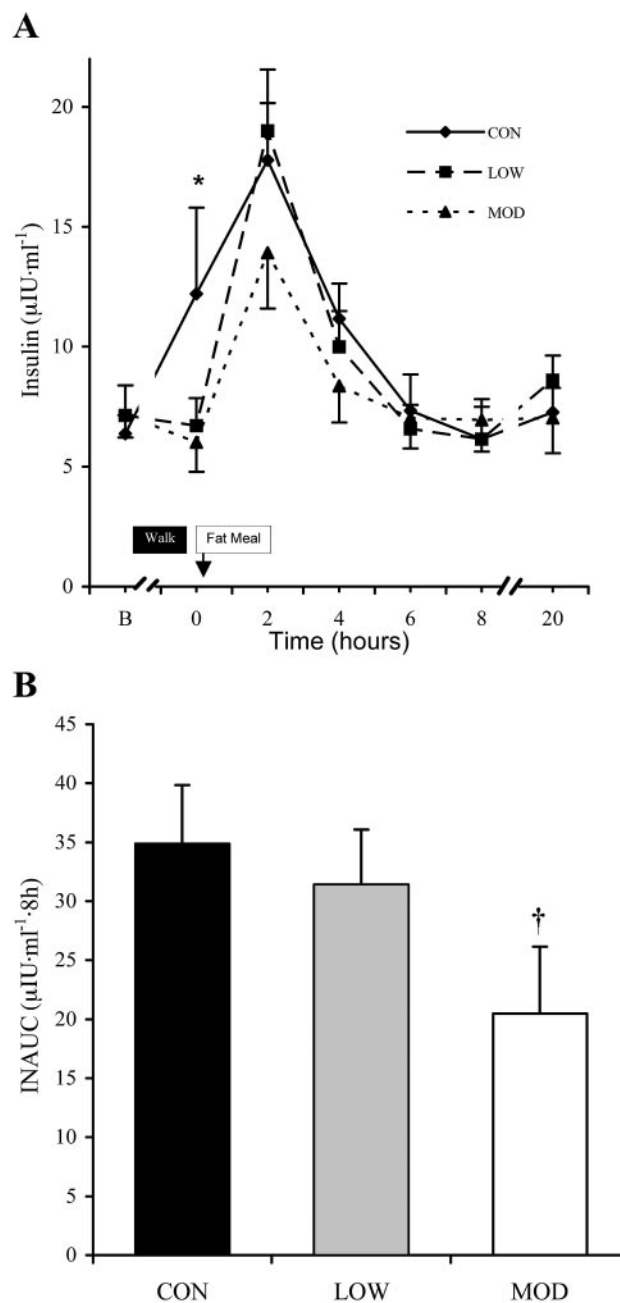


Fig. 3. Mean plasma insulin response after the high-fat meal ingestion (A) and insulin area under the curve (INAUC; B) for the Low, Mod, and Con trials. *Significantly different from Low and Mod at this time point ($P < 0.05$); †Mod trial significantly different from Con trial ($P < 0.05$).

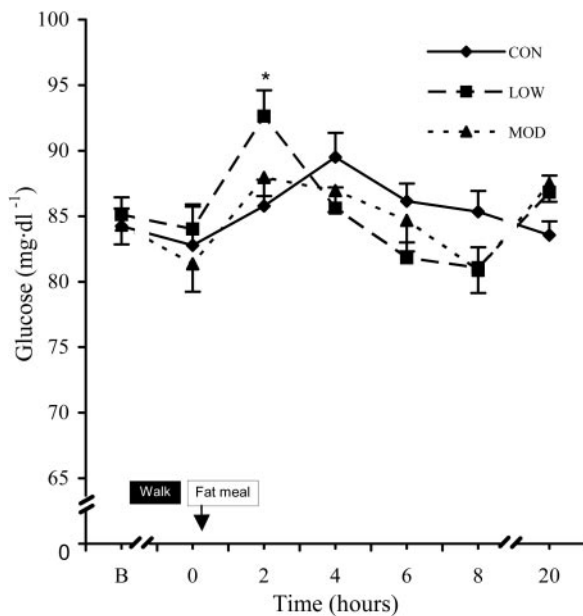


Fig. 4. Mean blood glucose response after the high-fat meal ingestion for the Low, Mod, and Con trials. *Significant difference between Low and Con at this time point ($P < 0.05$).

concentration is more appropriate to be used as baseline for normalizing the TGAUC. In the present investigation, mean postprandial lipemic response was the highest during the Con trial, whereas it was the lowest during the Mod trial. In the Mod trial, PPL was lower compared not only to the Con trial (39% lower) but also to the Low trial (34% lower). Previous research has concluded that the decrease in PPL is independent of exercise intensity when the total energy expended remains constant (38). Although there is an apparent discrepancy with respect to the low-intensity exercise between the findings of the present investigation and those reported by Tsetsonis and Hardman (38), in the latter study PPL was induced the day after the exercise whereas in the present study it was induced immediately after exercise. The different findings are likely a result of differences in the timing of the meal relative to the exercise, because Zhang et al. (41) have showed that the timing of exercise relative to a high-fat meal is a determining factor in the magnitude of PPL. However, other factors such as gender differences in subject populations cannot be excluded, because we studied only men whereas Tsetsonis and Hardman (38) studied a group of both men and women.

Energy deficit induced by exercise has previously been shown to significantly reduce PPL, whereas energy deficit of the same magnitude induced by caloric restriction does not (12). This suggests that the exercise-induced energy deficit is important in regulating PPL. This may emphasize the important role of the local contractile activity in regulating muscle LPL (15), which, in turn, has been suggested to have central role in the regulation of PPL (16). If the energy expended during exercise was the most important factor in regulating PPL, one would expect similar effects from exercise sessions of different intensities but matched for energy expenditure. However, this was not the case in the present study. When exercise was undertaken before the high-fat meal, the exercise intensity-determined rate of energy expenditure was also im-

portant in regulating PPL. Therefore, under these conditions, there should be other physiological and metabolic factors affected by the higher exercise intensity that influence PPL. Factors such as the TG entry into the circulation and their removal at the muscle level are discussed later in the paragraphs that follow.

As expected, the relative contribution of fat to the total energy expended during exercise was higher in the Low trial compared with the Mod trial (Table 2). However, despite the greater total fat oxidation during exercise in the Low trial, PPL was lower in the Mod and not in the Low trial, indicating that the total amount of fat oxidized is not an important contributor to the lower lipemic response. Malkova et al. (25) have provided direct evidence that the attenuated lipemic response of prior exercise is, indeed, independent of the relative contributions of fat and carbohydrate to the energy requirements of exercise.

Although the contribution of fat oxidation to the total exercise energy expenditure was higher during the low-intensity exercise, the rate of fat oxidation was higher during the moderate-intensity exercise. Because our data are limited to rates of total body oxidation, it is not possible to differentiate with respect to rates of fat oxidation between tissues, such as adipose, plasma, or muscle. Romijn et al. (33), however, who used exercise intensities the same as in this investigation, provide evidence that during exercise of moderate intensity the oxidation rate of fat from intramuscular sources is higher compared with that during exercise of low intensity. Although not during, but rather 6 h after the end of exhaustive exercise, Kiens and Richter (20) have reported a decrease in intramuscular TG coinciding with an increase in muscle LPLA. It is possible that, in the present study, an intramuscular TG depletion after moderate-intensity exercise, compared with that of low-intensity exercise, could have caused an increase in the rate of chylomicron- and VLDL-originating free fatty acid (FFA) uptake by the muscle to restore muscle TG, through an increase in muscle LPLA. Because we did not determine muscle LPLA, but rather measured LPLA in postheparin plasma, which includes LPLA associated with adipose tissue, this effect of moderate-intensity exercise on muscle LPLA remains a possibility.

The lipemic responses observed in the present study might have been mediated in part by insulin. It is known that,

Table 4. Postheparin plasma lipoprotein lipase and hepatic triglyceride lipase activities during the trials

	B		8 h	20 h
LPLA	22.7 ± 3.9	Con	24.2 ± 4.2	26.5 ± 4.4
		Low	37.9 ± 3.9*†	30.8 ± 4.0
		Mod	30.2 ± 3.9	32.0 ± 2.9†
HTGLA	85.3 ± 9.4	Con	84.0 ± 9.8	81.6 ± 10.1
		Low	83.4 ± 9.4	82.9 ± 8.9
		Mod	83.9 ± 8.9	81.5 ± 9.9

Values are means ± SE; all values are in $\mu\text{mol free fatty acids}\cdot\text{ml}^{-1}\cdot\text{h}^{-1}$ and have been adjusted for plasma volume changes. LPLA, postheparin plasma lipoprotein lipase activity; HTGLA, postheparin hepatic triglyceride lipase activity; the 8-h and 20-h postheparin blood samples were collected 8 and 20 h, respectively, after the fat meal ingestion; B, baseline postheparin blood sample collected on a separate day than the 3 trials. *Significantly different from Con and Mod trials at this time point ($P < 0.05$); †significantly different from baseline ($P < 0.05$).

although exercise is a positive stimulus for muscle LPLA (34), insulin decreases muscle LPLA (19). On the basis of this evidence, it is reasonable to hypothesize that in the Mod trial the effects of exercise on muscle substrate utilization, which were discussed in the previous paragraph, together with the attenuated postprandial insulinemic response, might have mediated an increase in the muscle LPLA. The postheparin plasma LPLA at 8 h, however, was elevated in the Low but not in the Mod trial, and this was observed despite high postprandial insulin in the Low trial. Because LPLA was measured in postheparin plasma, we cannot determine the relative contribution of muscle LPLA to the measured postheparin LPLA. It is possible that the higher LPLA measured at 8 h in the postheparin plasma in the Low trial may reflect mainly an increase in adipose tissue LPL, because it is known that insulin stimulates adipose tissue LPL during the postprandial period (31). Malkova et al. (24) showed that the attenuation in PPL several hours after exercise could not be explained by the leg uptake of plasma TG. Because muscle LPLA appears to be higher several hours compared with immediately after exercise (20), the findings by Malkova et al. (24) raise the possibility that in the present study an increased uptake of plasma TG by the muscles, because of increased muscle LPLA, might not have been the major factor determining the decrease in PPL in the Mod trial.

A possible determining factor of the postprandial lipemic response is the rate at which either chylomicrons or VLDL enter the circulation. Decreased rate of TG entry in the circulation will result in diminished lipemia. Although it is possible that the rate of chylomicron-TG entry into the circulation might have been affected by the moderate-intensity exercise to a greater extent than the low-intensity exercise, there is no evidence to suggest any effects of exercise intensity on the production, secretion, or entrance of the chylomicrons into the systemic circulation. On the other hand, hepatic VLDL-TG secretion may be affected by a single bout of exercise, but conclusive evidence for that, and under conditions similar to those of the present study, is lacking. In the postabsorptive state, total VLDL concentration has been found to be falling during the 4.5-h period that followed moderate-intensity exercise (3). Also, Gill et al. (11) have provided indirect evidence to indicate that the decrease in postprandial plasma TG with prior exercise is, at least in part, a result of a decrease in VLDL-TG.

The site (muscle vs. adipose tissue) where plasma TG hydrolysis takes place plays an important role in the uptake of the liberated FFA by the tissues. Evans et al. (8) showed that, after a fat load, there was an increased "escape" in the circulation of LPL-liberated FFA in the adipose tissue whereas such an escape of the liberated FFA was not observed in the muscle. It is possible that an increase in plasma FFA after the fat load, because of FFA release into the circulation after TG hydrolysis in the adipose tissue, likely increased the rate of VLDL-TG secretion by the liver (22) and could have contributed to the higher lipemic response observed in the Low trial compared with the Mod trial.

Postprandial insulinemia was affected by the exercise intensity, and it was lower only in the Mod trial compared with the Con trial. When comparing the results by Tsetsonis and Hardman (38), in which postprandial insulinemia did not decrease during low-intensity exercise whereas PPL did, with the results

of the present investigation, it is evident that the timing of exercise relative to the fat meal affects changes in postprandial insulinemia and PPL differently. It is possible that in the present investigation insulin might have played a role in regulating PPL, because both postprandial insulinemia and PPL decreased in the Mod trial. However, we were not able to document any statistically significant correlation in the changes between insulin and PPL as a result of exercise. In accordance with that, Gill et al. (13) have provided data to indicate that the exercise induced changes in the postprandial TG response are not associated with those in the postprandial insulin response.

The mechanisms associated with the lower insulin response after the moderate-intensity exercise can only be speculated. On the basis of available evidence, muscle glycogen contributes substantially to the total energy production during exercise at 65% $\dot{V}O_{2\max}$, whereas its contribution during exercise at 25% $\dot{V}O_{2\max}$ is minimal (33). Decreased muscle glycogen content is a stimulus for an increase in GLUT-4 glucose transporters in the muscle cell surface (6), which increases glucose transport to the cell and, therefore, decreases the requirements for insulin-mediated glucose disposal to the muscle. However, alternative mechanisms for the insulin response associated with the three trials cannot be excluded. It is very likely, for example, that the decrease in fat accumulation in the blood with moderate-intensity exercise contributed to an improved insulin sensitivity (32) and, therefore, decreased need for insulin secretion compared with the low-intensity exercise.

In conclusion, our results indicate that when the exercise is completed 1 h before ingestion of a high-fat meal, exercise of moderate intensity, but not an equal energy expenditure exercise of low intensity, attenuates the postprandial lipemia in physically active men. The observed responses in postprandial lipemia after the exercise are not associated with the changes in lipoprotein lipase activity measured in postheparin plasma.

GRANTS

This project was supported in part by research grants from the Life Fitness Academy and the Gatorade Sports Science Institute.

REFERENCES

1. **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.
2. **Bianda T, Zapf J, Froesch ER, and Schmid C.** Insulin-like growth factor-I lowers fasting and postprandial triglyceride levels without affecting chylomicron clearance in healthy men. *J Intern Med* 245: 455–462, 1999.
3. **Borsheim E, Knardahl S, and Hostmark AT.** Short-term effects of exercise on plasma very low density lipoproteins (VLDL) and fatty acids. *Med Sci Sports Exerc* 31: 522–530, 1999.
4. **Brunzell JD, Hazzard WR, Porte D Jr, and Bierman EL.** Evidence for a common, saturable, triglyceride removal mechanism for chylomicrons and very low density lipoproteins in man. *J Clin Invest* 52: 1578–1585, 1973.
5. **Buchfuhrer MJ, Hansen JE, Robinson TE, Sue DY, Wasserman K, and Whipp BJ.** Optimizing the exercise protocol for cardiopulmonary assessment. *J Appl Physiol* 55: 1558–1564, 1983.
6. **Derave W, Lund S, Holman GD, Wojtaszewski J, Pedersen O, and Richter EA.** Contraction-stimulated muscle glucose transport and GLUT-4 surface content are dependent on glycogen content. *Am J Physiol Endocrinol Metab* 277: E1103–E1110, 1999.
7. **Dill DB and Costill DL.** Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol* 37: 247–248, 1974.

8. **Evans K, Clark ML, and Frayn KN.** Effects of an oral and intravenous fat load on adipose tissue and forearm lipid metabolism. *Am J Physiol Endocrinol Metab* 276: E241–E248, 1999.
9. **Ferguson MA, Alderson NL, Trost SG, Essig DA, Burke JR, and Durstine JL.** Effects of four different single exercise sessions on lipids, lipoproteins, and lipoprotein lipase. *J Appl Physiol* 85: 1169–1174, 1998.
10. **Gidez LI, Miller GJ, Burstein M, Slagle S, and Eder HA.** Separation and quantitation of subclasses of human plasma high density lipoproteins by a simple precipitation procedure. *J Lipid Res* 23: 1206–1223, 1982.
11. **Gill JM, Frayn KN, Wootton SA, Miller GJ, and Hardman AE.** Effects of prior moderate exercise on exogenous and endogenous lipid metabolism and plasma factor VII activity. *Clin Sci (Lond)* 100: 517–527, 2001.
12. **Gill JM and Hardman AE.** Postprandial lipemia: effects of exercise and restriction of energy intake compared. *Am J Clin Nutr* 71: 465–471, 2000.
13. **Gill JM, Herd SL, Tsetsonis NV, and Hardman AE.** Are the reductions in triacylglycerol and insulin levels after exercise related? *Clin Sci (Lond)* 102: 223–231, 2002.
14. **Grandjean PW, Crouse SF, and Rohack JJ.** Influence of cholesterol status on blood lipid and lipoprotein enzyme responses to aerobic exercise. *J Appl Physiol* 89: 472–480, 2000.
15. **Hamilton MT, Etienne J, McClure WC, Pavey BS, and Holloway AK.** Role of local contractile activity and muscle fiber type on LPL regulation during exercise. *Am J Physiol Endocrinol Metab* 275: E1016–E1022, 1998.
16. **Hardman AE.** The influence of exercise on postprandial triacylglycerol metabolism. *Atherosclerosis* 141, Suppl 1: S93–S100, 1998.
17. **Kantor MA, Cullinane EM, Sady SP, Herbert PN, and Thompson PD.** Exercise acutely increases high density lipoprotein-cholesterol and lipoprotein lipase activity in trained and untrained men. *Metabolism* 36: 188–192, 1987.
18. **Karpe F, Hellenius ML, and Hamsten A.** Differences in postprandial concentrations of very-low-density lipoprotein and chylomicron remnants between normotriglyceridemic and hypertriglyceridemic men with and without coronary heart disease. *Metabolism* 48: 301–307, 1999.
19. **Kiens B, Lithell H, Mikines KJ, and Richter EA.** Effects of insulin and exercise on muscle lipoprotein lipase activity in man and its relation to insulin action. *J Clin Invest* 84: 1124–1129, 1989.
20. **Kiens B and Richter EA.** Utilization of skeletal muscle triacylglycerol during postexercise recovery in humans. *Am J Physiol Endocrinol Metab* 275: E332–E337, 1998.
21. **Krauss RM, Levy RI, and Fredrickson DS.** Selective measurement of two lipase activities in postheparin plasma from normal subjects and patients with hyperlipoproteinemia. *J Clin Invest* 54: 1107–1124, 1974.
22. **Lewis GF.** Fatty acid regulation of very low density lipoprotein production. *Curr Opin Lipidol* 8: 146–153, 1997.
23. **Lewis GF and Steiner G.** Acute effects of insulin in the control of VLDL production in humans. Implications for the insulin-resistant state. *Diabetes Care* 19: 390–393, 1996.
- 23a. **Lusk G.** *The Elements of the Science of Nutrition*. Philadelphia, PA: W. B. Saunders, 1928.
24. **Malkova D, Evans RD, Frayn KN, Humphreys SM, Jones PR, and Hardman AE.** Prior exercise and postprandial substrate extraction across the human leg. *Am J Physiol Endocrinol Metab* 279: E1020–E1028, 2000.
25. **Malkova D, Hardman AE, Bowness RJ, and Macdonald IA.** The reduction in postprandial lipemia after exercise is independent of the relative contributions of fat and carbohydrate to energy metabolism during exercise. *Metabolism* 48: 245–251, 1999.
27. **Patsch JR.** Postprandial lipaemia. *Baillieres Clin Endocrinol Metab* 1: 551–580, 1987.
28. **Patsch JR, Gotto AM Jr, Olivercrona T, and Eisenberg S.** Formation of high density lipoprotein2-like particles during lipolysis of very low density lipoproteins in vitro. *Proc Natl Acad Sci USA* 75: 4519–4523, 1978.
29. **Patsch JR, Miesenbock G, Hopferwieser T, Muhlberger V, Knapp E, Dunn JK, Gotto AM Jr, and Patsch W.** Relation of triglyceride metabolism and coronary artery disease. Studies in the postprandial state. *Arterioscler Thromb* 12: 1336–1345, 1992.
30. **Pattit JS and Cureton KJ.** Effects of prior exercise on postprandial lipemia: a quantitative review. *Metabolism* 52: 418–424, 2003.
31. **Picard F, Naimi N, Richard D, and Deshaies Y.** Response of adipose tissue lipoprotein lipase to the cephalic phase of insulin secretion. *Diabetes* 48: 452–459, 1999.
32. **Randle PJ, Garland PB, Hales CN, and Newsholme EA.** The glucose-fatty acid cycle: its role in insulin sensitivity and the metabolic disturbances of diabetes mellitus. *Lancet* 1: 785–789, 1963.
- 32a. **Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. The Expert Panel.** *Arch Intern Med* 148: 36–69, 1988.
33. **Romijn JA, Coyle EF, Sidossis LS, Gastaldelli A, Horowitz JF, Enderit E, and Wolfe RR.** Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration. *Am J Physiol Endocrinol Metab* 265: E380–E391, 1993.
34. **Seip RL, Mair K, Cole TG, and Semenkovich CF.** Induction of human skeletal muscle lipoprotein lipase gene expression by short-term exercise is transient. *Am J Physiol Endocrinol Metab* 272: E255–E261, 1997.
35. **Tall AR.** Plasma high density lipoproteins. Metabolism and relationship to atherogenesis. *J Clin Invest* 86: 379–384, 1990.
36. **Thompson PD, Kantor MA, Cullinane EM, Sady SP, Saritelli A, and Herbert PN.** Postheparin plasma lipolytic activities in physically active and sedentary men after varying and repeated doses of intravenous heparin. *Metabolism* 35: 999–1004, 1986.
37. **Tsetsonis NV and Hardman AE.** Effects of low and moderate intensity treadmill walking on postprandial lipaemia in healthy young adults. *Eur J Appl Physiol* 73: 419–426, 1996.
38. **Tsetsonis NV and Hardman AE.** Reduction in postprandial lipemia after walking: influence of exercise intensity. *Med Sci Sports Exerc* 28: 1235–1242, 1996.
39. **Tsetsonis NV, Hardman AE, and Mastana SS.** Acute effects of exercise on postprandial lipemia: a comparative study in trained and untrained middle-aged women. *Am J Clin Nutr* 65: 525–533, 1997.
40. **Warnick GR and Albers JJ.** A comprehensive evaluation of the heparin-manganese precipitation procedure for estimating high density lipoprotein cholesterol. *J Lipid Res* 19: 65–76, 1978.
41. **Zhang JQ, Thomas TR, and Ball SD.** Effect of exercise timing on postprandial lipemia and HDL cholesterol subfractions. *J Appl Physiol* 85: 1516–1522, 1998.
42. **Zilvermit DB.** Atherogenesis: a postprandial phenomenon. *Circulation* 60: 473–485, 1979.