Hyperlipemic Response of Young Trained and Untrained Men after a High Fat Meal

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To test the hypothesis that endurance training is associated with a decreased lipemia after a high fat meal, 16 young men [22 to 34 years old, nine of whom were trained (T) and seven of whom were untrained (UT)] were recruited. T ran >30 or biked >100 miles a week, while UT had been sedentary for at least the preceding 3 months. Daily caloric intake and daily caloric expenditure during exercise were 35% and 704% greater, respectively, in T than in UT. VO2max was 31% greater, while percent body fat was 36% lower in T than in UT. Dietary composition and body height and weight were similar. After a fasting blood sample was taken, the men ate a high fat meal (approximately 56% of total calories as fat in 1100 kcal adjusted to body weight) and additional blood samples were taken hourly for 8 hours. Fasting lipids were similar. Postprandial peak triglyceride (TGmax), percent TG increase (%TG), and total lipemic response (TLR, the area under the lipemia curve in excess of fasting TG) were 42%, 54%, and 75% greater, respectively, in UT vs. T. Stepwise regression analysis showed that the same three-variable model (training status, fasting TG, and VO2max) described the variation in TGmax (R²=0.97), %TG (R²=0.75), and TLR (R²=0.92). Furthermore, this same analysis showed that after adjustment for fasting TG and VO2max, the UT group had a significantly greater postprandial lipemia whether expressed as TGmax (p<0.0001), %TG (p=0.0002), or TLR (p=0.0002). Thus, endurance training appears to be associated with a diminished lipemia after a high fat meal in young adult men. (Arteriosclerosis 9:217–223, March/April 1989)

Atherosclerosis is the major underlying cause of death and disability in Western society. Most research dealing with the etiology of atherosclerotic vascular disease has linked elevated postabsorptive blood cholesterol and low density lipoproteins with an increased risk of developing the disease. In contrast, little attention has been paid to fat tolerance, which may be defined as the plasma triglyceride (TG) response to a fatty meal. However, the magnitude of the triglyceremic response to a standard fatty meal (postprandial lipemia) differs substantially among apparently healthy individuals who are considered normolipidemic on the basis of fasting blood lipid values. Even among apparently normolipidemic individuals, the variability in postprandial lipemia may be pathophysiologic, since a significant lipemia may persist throughout most of the day in normal adults consuming fatty foods over three meals. In fact, recent work by Ziewersm suggests that postprandial metabolism of TG-rich lipoproteins may constitute an atherogenic process in individuals who chronically eat a diet rich in fat and cholesterol. Furthermore, Engelberg has recently reviewed some older literature, which suggests that tissue hypoxia can also result from this postprandial lipemia and that this hypoxia may also be atherogenic. Thus, if the rate of atherogenesis is related to either the peak values or the duration of lipemia after a high fat meal in a total dose or dose-time relation, then improving an individual's fat tolerance (i.e., decreasing the magnitude of postprandial lipemia) could suggest a mechanism for protecting against atherosclerotic plaque formation.

Two previous studies by Attekruse and Wilmore and Patsch et al. have suggested that chronic endurance exercise may improve fat tolerance. While these two studies have shown promising results, a critical analysis of their experimental designs reveals that a number of variables known to affect lipoprotein metabolism were not fully addressed (e.g., alcohol intake, medications, smoking, gender, caloric intake, adiposity). Furthermore, older individuals over larger age ranges were considered in both studies. Since atherogenesis is a process beginning in childhood, it is of interest to study fat intolerance in younger individuals if exercise is to be considered as an effective tool in the primary prevention of atherosclerosis.

Thus, one purpose of this study was to test the hypothesis that endurance exercise training is associated with a decreased lipemia after the ingestion of a high fat meal in young adult males. The study design attempts to control for factors known to affect lipoprotein metabolism. A second purpose was to develop a regression model to describe postprandial lipemia with simultaneous consideration of training status and other physiological parameters that may affect lipoprotein metabolism. Portions of these data have been presented previously as a brief technical note.
Methods

Subjects

Sixteen [nine trained (T) and seven untrained (UT)] nonsmoking, nonobese, healthy asymptomatic men, 22 to 34 years old, served as subjects for this investigation. No subject consumed more than two cups of caffeinated or two glasses of alcoholic beverages per day; none took prescription or nonprescription medications. In addition, the T subjects had to be currently either running a minimum of 30 miles per week, cycling at least 100 miles per week, or performing an equivalent combination of running and cycling. Conversely, the UT subjects currently had to be taking no regular vigorous aerobic exercise. All subjects must have maintained their exercise patterns for at least 3 months before starting the investigation. Subjects were matched as closely as possible for age, height, and weight.

Apparatus and Procedures

All experimental procedures were conducted in the Human Performance Laboratory at the University of California at Davis with the exception of the blood lipid, lipoprotein, and apoliipoprotein analyses, which were performed in the Clinical Chemistry Laboratory at the Medical College of Virginia Hospitals. All subjects gave written informed consent.

Before performing any other tests, all subjects were screened for resting electrocardiogram abnormalities and syncope during phlebotomy. Two subjects were excluded for syncope while another was excluded for high grade ectopy. These three subjects were untrained. All others then performed a graded maximal exercise test to determine their maximal oxygen uptake (VO2max). Body composition analysis was performed on each subject to determine percent body fat. This consisted of the hydrostatic weighing technique as described by Willmore and Behnke, along with residual lung volume determination via the oxygen dilution technique. Height and weight were also measured. Finally, to determine the composition of their normal diets and to quantify the amounts of aerobic exercise normally taken, subjects recorded all food and beverages consumed, as well as all the exercise they performed for 7 days. These records were checked after 3 days for consistency and accuracy.

After analysis of their 7-day records, subjects were asked not to deviate from their normal dietary and exercise patterns for 3 days before their fat tolerance test, except as noted below. In an attempt to prevent subjects from markedly deviating from their normal diet during the control period, they were given a list of the foods and beverages they had consumed while keeping their food records and asked to choose their meals from this list during the 3-day period. As before, subjects were instructed to record all food and beverages consumed, as well as all exercise taken during these 3 days. These records were later analyzed to determine how closely subjects adhered to their normal diet. Subjects were also asked to consume little or no alcohol on the third and second days before their fat tolerance test, and then to avoid alcohol completely on the day before the test. In addition, subjects were told to avoid vigorous exercise the day before the test, to be well rested, and to take as little exercise as possible while getting to the lab. Thus, all subjects went through a similar 3-day control period, with each subject serving as his own control.

Subjects reported to the laboratory after a 12-hour fast to begin their fat tolerance test. After resting quietly for 10 minutes, a venous blood sample was obtained from an antecubital vein. Immediately after phlebotomy, subjects consumed a high fat breakfast purchased from McDonald's restaurant consisting of approximately two sausage McMuffins with eggs, one order of hash brown potatoes, and a glass of ice water. The meal, standardized to a 70 kg individual, contained approximately 70 grams of fat, 560 mg of cholesterol, and 1100 calories, with 56% of the calories coming from fat. Subjects finished consuming their meals within 10 to 15 minutes after receiving their food.

The quantity of food (and thus the quantity of fat) given to each subject differed depending on the subject's body weight. This was done in an attempt to control for the differences in the rates of digestion and metabolism that could occur between subjects due to differences in body size alone. Therefore, principles taken from dimensional analysis theory were utilized to determine how much fat each individual should consume. Based on the literature, 70 grams was chosen as the amount of fat that a 70 kg man should consume for a physiological fat tolerance test. Furthermore, this was the amount of fat contained in this McDonald's breakfast, which seems to be a representative breakfast for a large number of Americans. The following proportion was used to calculate the amount of fat consumed by each subject:

\[
\text{grams of fat} = \frac{70 \text{ grams of fat}}{(70 \text{ kg subject})^{0.75}} \times \left(\frac{\text{weight of subject}^{0.75}}{\text{weight of subject}^{0.75}}\right)
\]

Each subject received one sausage McMuffin with egg and one order of hash brown potatoes (a total of 39.9 grams of fat), with the remaining amount of fat coming from a calculated amount of an additional sausage McMuffin with egg.

During the 8 hours after consumption of the standard meal, subjects rested quietly in the laboratory and consumed no food or beverage except water. One venous blood sample was obtained hourly.

All blood samples were collected in 10 ml MONOJECT tubes (Sherwood Medical Industries, St Louis, MO) containing EDTA anticoagulant. Samples were centrifuged for 15 minutes to separate the plasma, which was then transferred to and stored in 1 ml plastic plasma vials with screw caps. Plasma samples were stored at −20°C until the analyses were performed, except during one 24-hour period when they were shipped in dry ice (−78.5°C) from California to Virginia. All samples were analyzed within 4 months. Stability of apoliipoprotein (apo) for 2 years and lipoprotein concentrations of cholesterol and TG for 6 months at −20°C have been previously documented.

Blood Analyses

All plasma samples from the fat tolerance test were enzymatically analyzed for TG with correction for endogenous glycerol. The fasting samples were also analyzed for total cholesterol, high density lipoprotein (HDL) cho-
lesterol, and apo A-I and apo B, as previously described. These data (see Table 3) are reproduced in part from a prior manuscript for the convenience of the readers and to enter the stepwise regression analysis that relates various fasting plasma constituents to measures of the postprandial lipemia—an original usage of these data.

**Nutritional Analysis**

Both the 7- and 3-day food records were analyzed to determine total daily caloric intake and the percent of total calories from fat, protein, carbohydrate, and alcohol. This was accomplished by using a nutrition analysis computer program based on the United States Department of Agriculture (USDA) Handbook No. 8, Table 1.17 USDA Handbook No. 45618 was used to convert household units to gram weights, and additional references19,20 were used for food items not found in USDA Handbook No. 8.

**Caloric Expenditure Estimates during Exercise**

Based on the 7-day exercise records, the amount of vigorous or prolonged aerobic exercise performed by subjects on a daily basis was determined. Data obtained from exercise records were used to determine speed of walking, hiking, cycling, and swimming. With these values, the type of exercise and each subject's body weight and daily caloric expenditure during exercise were determined from tables presented by Williams,21 Katch and McArdle,22 Adams,23 Pugh,24 and the American College of Sports Medicine.25

**Statistical Analyses**

To facilitate comparisons of the lipemic response to the high fat meal between groups, the magnitude of lipemia was quantified in the following three ways: 1) total lipemic response (TLR), which was equal to the area bounded by the line connecting the nine TG values during the fat tolerance test in excess of the 0-hour value (TG0); 2) maximum TG (TGmax) concentration attained during the fat tolerance test; and 3) percentage TG increase (%TGI), which was calculated according to the formula: %TGI = \frac{TG_{max} - TG_0}{TG_0} \times 100\%  

A stepwise regression analysis was performed for each of these lipemic response measures after first including a training status indicator (TSI), which indicated whether a subject was in the T or UT group. The TSI was included first because it is a designed factor that we intended to evaluate a priori. The stepwise procedure then selected other covariates related to lipemic response. The full list of regressors applied to the procedure was derived from the variables presented in Tables 1, 2, and 3 and included those previously reported to affect lipoprotein metabolism: daily exercise caloric expenditure,26 daily caloric intake,27 age,29 percent of total calories from fat,29 percent of total calories from carbohydrate,50 percent of total calories from alcohol,31 percent body fat,27 VO2max or aerobic fitness,7,32 and fasting concentrations of TG, cholesterol, apo B, apo A-I, and HDL cholesterol.33 Order of entrance into the regression model was determined by maximum R2 improvement.32 For each lipemic response measure, a “best” model was chosen based on consideration of the R2 increment at each step, Mallows' Cp statistic, and the number of variables entered.

**Table 1. Subject Characteristics**

| Group       | Age (yrs) | Height (cm) | Weight (kg) | Body fat (%) | VO2max (ml/kg/min) |
|-------------|-----------|-------------|-------------|--------------|--------------------|
| Trained     | 30.2±2.9  | 181.4±4.8   | 71.2±7.5    | 13±4         | 67.4±8.8           |
| Untrained   | 25.9±4.8  | 176.3±12    | 73.3±16.2   | 20±7         | 51.6±6.2           |

Values are means±1 SD. There were 9 trained and 7 untrained men.

**Table 2. Diet Composition and Caloric Expenditure during Exercise**

| Group       | Food/exercise record | Exercise-caloric expenditure (kcal/day) | Caloric intake (kcal/day) | % Calories as protein | % Calories as carbohydrate | % Calories as fat | % Calories as alcohol |
|-------------|----------------------|----------------------------------------|---------------------------|-----------------------|---------------------------|------------------|----------------------|
| Trained     | 7-day                | 868±587                                | 3368±817                 | 14±4                  | 49±10                     | 32±7             | 5±6                  |
| Untrained   | 7-day                | 108±131                                | 2507±573                 | 15±4                  | 47±11                     | 32±9             | 7±8                  |
| Trained     | 3-day                | —                                      | 3116±93                 | 17±7                  | 51±9                      | 32±7             | 1±3                  |
| Untrained   | 3-day                | —                                      | 2485±616                | 15±4                  | 47±10                     | 33±9             | 5±6                  |

Values are means±1 SD. There were 9 trained and 7 untrained men.

**Table 3. Postabsorptive Lipid, Lipoprotein, and Apo-lipoprotein Levels**

| Variable     | Trained     | Untrained  |
|--------------|-------------|------------|
| Cholesterol  | 156±26      | 158±35     |
| Triglyceride | 64±15       | 69±35      |
| HDL-C        | 48±9        | 47±11      |
| Apo A-I      | 135±15      | 126±18     |
| Apo B        | 93±14       | 103±22     |

Values are means±1 SD (in mg/dl). There were 9 trained and 7 untrained men.

HDL-C = high density lipoprotein cholesterol, apo = apolipoprotein.
The three lipemic response models were tested for overall significance using the $F$ statistic. The TSI, which assesses our hypothesis that endurance training is associated with a decreased postprandial lipemia, was examined by testing the null hypothesis that the parameter (i.e., coefficient) associated with TSI is equal to zero. For this component and the other components of each model, a two-tailed $t$ test was used to test the null hypothesis. Significance was accepted when $p<0.05$. Due to the large number of variables examined, no other tests of statistical significance were performed. Other variables were examined using descriptive statistics only.

**Results**

Age, height, weight, percent body fat, and VO$_{2\text{max}}$ for the T and UT groups are shown in Table 1. An attempt was made to match our T and UT subjects as closely as possible for age, height, and weight. Although there were slight differences in these three variables between groups, they appear to be biologically insignificant in regards to this study. On the other hand, percent body fat was 36% lower, while VO$_{2\text{max}}$ was 31% higher in the T group compared to the UT group.

Data obtained from the 7-day and the 3-day control period food records, as well as data obtained from the 7-day exercise records, are presented in Table 2. Daily caloric intake and exercise energy expenditures were 35% and 704% greater, respectively, in the T group relative to the UT group. Percent of total calories from protein, carbohydrate, fat, and alcohol were similar between groups. Data appeared to be well-controlled in the 3 days preceding the fat tolerance test relative to the 7-day diet histories, except that calories from alcohol had decreased as requested.

The concentrations of cholesterol, TG, HDL cholesterol, apo A-I, and apo B in the postabsorptive plasma are summarized in Table 3. These data have been previously reported. Therefore, there were essentially no differences in any of these measures between the T and UT groups.

TG values for each of the nine plasma samples collected over the 8-hour fat tolerance test are presented graphically in Figure 1. In general, the curve for the UT group shows an exaggerated lipemic response (i.e., a poorer fat tolerance) with more variability relative to the lipemic response of the T group. TG peaked at approximately 3 hours in both groups (3.1±1.2 hours in T vs. 3.7±1.0 hours in UT). By 8 hours after the high fat meal, TG concentration had decreased to within 7% of the fasting value in the T group but was still elevated 20% in the UT group.

The three measures calculated to quantify the magnitude of the postprandial lipemia are presented in Table 4. The TLR, TG$_{\text{max}}$, and %TG were 75%, 42%, and 54%, respectively, greater in UT than in T. Table 5 presents the stepwise regression analysis of the variables that accurately describe the variation in TLR ($R^2=0.92$), TG$_{\text{max}}$ ($R^2=0.97$), and %TG ($R^2=0.75$) in our subjects when variables were combined and entered in the following order:

$$\text{TLR} = -984.1 + 344.7 \text{ TSI} + 10.00 \text{ TG}_0 + 9.85 \text{ VO}_{2\text{max}}$$

$$\text{TG}_{\text{max}} = -231.7 + 76.6 \text{ TSI} + 3.58 \text{ TG}_0 + 2.18 \text{ VO}_{2\text{max}}$$

$$\%\text{TG} = -131.6 + 107.3 \text{ TSI} + 0.89 \text{ TG}_0 + 2.93 \text{ VO}_{2\text{max}}$$

where TSI is the training status indicator, which takes on the value 0 for the T group and 1 for the UT group. TG$_0$ is the fasting TG concentration in mg/dl, and VO$_{2\text{max}}$ is expressed in ml/kg/min. The stepwise regression analysis selected the same three-variable model for TLR, TG$_{\text{max}}$, and %TG. In each case, fasting TG and VO$_{2\text{max}}$ were selected in addition to the TSI, which was forced into the model as previously discussed. Note that the UT group indicator (which identifies the UT vs. T group) is significant in all models: TLR ($p=0.0002$), TG$_{\text{max}}$ ($p<0.0001$), and %TG ($p=0.0002$). That is, after adjustment for fasting TG and VO$_{2\text{max}}$ differences, UT subjects had a significantly higher postprandial lipemia whether measured as TLR, TG$_{\text{max}}$, or %TG.

**Discussion**

One purpose of this study was to determine whether endurance training is associated with a decreased lipemia after a high fat meal in young adult men. A second purpose was to develop a model to describe this postprandial response. In comparing the T and UT groups, we attempted to control for variables known to affect lipoprotein metabolism.

Our T and UT subjects were of similar height and weight (Table 1) and consumed a similar diet (Table 2). However, the T subjects were older and had a lower percent body fat and higher VO$_{2\text{max}}$ than the UT subjects had (Table 1). While the difference in age in subjects this young has little effect on fasting lipid levels, the overall influence of age on the postprandial lipemia would be to cause an exaggerated response in the older T group relative to the younger UT group. This is the opposite of what was seen. Although height and weight were similar,
percent body fat was 36% less in the T than in the UT group, indicating the hazard of using height and weight relationships or weight alone to estimate adiposity. While adiposity might be thought to influence the postprandial lipemia, it did not significantly improve $R^2$ in any of the regression models. As expected, $V_{O_{2\text{max}}}$ was greater (by 31%) in the T than in the UT group. However, the $V_{O_{2\text{max}}}$ of the UT group was surprisingly high (51.6 ml/kg/min) for such a relatively sedentary population (daily exercise energy expenditure of 108 kcal) (Table 2). We are unable to explain the high $V_{O_{2\text{max}}}$ of our UT group except to note that our subjects were selected from an extremely fitness-conscious community and even our "sedentary" group had a mean weekly exercise energy expenditure equivalent to jogging 7 miles. While dietary composition was similar between groups, daily caloric intake was 35% and energy expenditure during exercise was 70% greater, respectively, in the T versus UT group (Table 2). The excess daily caloric expenditure during exercise of the T group compared to the UT group (760 kcal/day) was closely matched by an excess caloric intake (881 kcal/day) in the T group, at least to the level of accuracy of these caloric estimates (Table 2). Therefore, the diets of these two groups appear to be matched as closely as possible given the excess exercise energy expenditure of the T group.

Fasting levels of total cholesterol, TG, HDL cholesterol, apo A-I, and apo B in both groups are presented in Table 3. Somewhat surprisingly, there appeared to be no difference of biologic significance between the groups in these variables, although as we have previously reported, the ratio of apo A-I to apo B is greater in the T than in the UT group, indicating perhaps a lower overall coronary artery disease (CAD) risk in the T group.

The lipemia after a high fat meal in T and UT subjects is illustrated in Figure 1. The three measures calculated to quantify the magnitude of the postprandial lipemia, TLR, TG, and %TGI, were greater in the UT than in the T group (Table 4). These results are consistent with the classic investigation of Altekruse and Wilmore,7 which suggested that a 10-week physical conditioning program resulted in a more rapid clearing of serum TG after the ingestion of a high fat meal in a group of previously sedentary men. Specifically in that study,7 TG peaked at lower values and returned to fasting levels more rapidly at the end of the conditioning program compared to pre-conditioning values, findings which parallel those of our T and UT groups.

The pioneering work of Altekruse and Wilmore7 and Patsch et al.8 have stimulated us to look more critically at the relationship between exercise and postprandial lipemia. We attempted to do this as follows: 1) matching subjects as closely as possible for age, height, and weight, and 2) by entering variables known to influence lipoprotein metabolism into a stepwise regression analysis, since it is unlikely that one single variable can adequately characterize a complex metabolic process like postprandial lipemia. The results of this stepwise regression modeling (Table 5) show that 92%, 96%, and 75% of the variation in TLR, TGI, and %TGI, respectively, can be accounted for by the same three variables: training status, fasting TG, and $V_{O_{2\text{max}}}$. It is important to note the following: 1) Although these equations account for a large amount of the variation in lipemic response, the small sample size and cross-sectional design preclude generalization and cause-effect attribution. 2) While training status, fasting TG, and $V_{O_{2\text{max}}}$ might all be thought to vary together,29 collinearity diagnostics showed there was little dependency among these independent variables. Thus, they all appeared to identify qualitatively distinct factors affecting the lipemic response. 3) The TSI was included as the first step in the regression models, since we intended to evaluate this factor a priori. Thus, the inferences concerning training status are more powerful since they are made after adjustment for the significant covariates. 4) In these models, higher $V_{O_{2\text{max}}}$ values are associated with a greater lipemic response. We feel this is an artifact of the rela-
tively small differences between the $V_{o_{\text{max}}}$ values of our T and UT subjects. Furthermore, the majority of the variation in lipemic response is accounted for by training status and fasting TG, while $V_{o_{\text{max}}}$ acts to "fine-tune" the regression model. 5) Patsch et al. showed significant bivariate relationships between TLR and HDL cholesterol, HDL2, apo A-I, and apo B, while none of these variables entered our multivariate model, although we did not measure HDL2. However, they did find that weekly running mileage and fasting TG correlated significantly with TLR, and both training status and fasting TG were significant predictors in all three of our lipemic response models. While HDL2 described 74% of the variation in TLR in the model of Patsch et al., they described here accounts for 92% of the variation in TLR. It is also worth noting that the variables in this model (TSI, fasting TG, and $V_{o_{\text{max}}}$) are also much more easily assessed than HDL2. This analysis illustrates the importance of considering the combined effects of variables and accomplishes the purpose of developing improved models for describing the lipemic response. 6) Finally, the original purpose of determining whether the trained condition is associated with a decreased lipemic response in young men also appears to be accomplished in that the trained condition was significantly associated with a lesser postprandial lipemia.

Although the exact mechanism is unknown, it appears that exercise, specifically habitual endurance-type exercise, may attenuate the postprandial lipemia that follows a high fat meal. These exercise-induced adaptations appear to be mediated by changes in enzymes involved in cholesterol and TG synthesis, transport, and catabolism. In fact, the activity of lipoprotein lipase (LPL), the enzyme that catabolizes TG-rich chylomicrons and very low density lipoproteins, has been shown to be higher in adipose tissue and skeletal muscle of endurance athletes compared to sedentary controls. Furthermore, several studies have demonstrated that endurance exercise training can significantly increase adipose tissue and postheparin LPL activity in previously sedentary men. Thus, this increased LPL activity may speed the clearance of TG-rich particles from the blood after the ingestion of fatty foods and, thereby, both decrease the extent of postprandial lipemia and possibly protect against atherogenesis. Finally, since we are particularly interested in exercise as a tool in this primary prevention of CAD, we would like to emphasize that if an elevated postprandial lipemia places an individual at increased risk of CAD, even in individuals traditionally considered at low CAD risk but who chronically eat a high fat diet, regular endurance exercise may decrease this risk.

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