Postprandial lipemia in endurance-trained people during a short interruption to training

ADRIANNE E. HARDMAN, JANET E. M. LAWRENCE, AND SARA L. HERD
Human Muscle Metabolism Research Group, Department of Physical Education, Sports Science and Recreation Management, Loughborough University, Loughborough, Leicestershire LE11 3TU, United Kingdom

Hardman, Adrienne E., Janet E. M. Lawrence, and Sara L. Herd. Postprandial lipemia in endurance-trained people during a short interruption to training. J. Appl. Physiol. 84(6): 1895–1901, 1998.—This study examined changes in postprandial lipemia in endurance-trained people during a short interruption to training. Nine men and one woman (ages 18–55 yr) undertook fat tolerance tests after 15 h, 60 h, and 6.5 days without exercise. The test meal (1.2 g fat, 1.1 g carbohydrate, 66 kJ/kg body mass) was consumed after a 12-h fast. Postprandial lipemia increased rapidly with detraining (area under plasma triacylglycerol vs. time curve: 8.42 ± 1.40, 11.35 ± 1.38, and 11.97 mM × 6 h at 15 h, 60 h and 6.5 days, respectively). In the fasted state, plasma triacylglycerol concentration (0.85 ± 0.15, 1.09 ± 0.12, and 1.10 ± 0.11 mM at 15 h, 60 h and 6.5 days, respectively) and the ratio of total cholesterol to high-density-lipoprotein cholesterol increased with detraining. Values were significantly higher at 60 h and 6.5 days than values at 15 h (P < 0.05) for each of these three variables. The serum insulin response was higher (P < 0.05) at 6.5 days than at 15 h (81.6 ± 11.3, 87.6 ± 11.4, and 94.5 ± 9.4 µIU/ml × 6 h at 15 h, 60 h, and 6.5 days, respectively). Frequent exercise is needed to maintain a low level of postprandial lipemia and insulinemia in trained people.

dietary fat; insulinemia; triacylglycerol; detraining; exercise

A PARTICULAR COMBINATION of characteristics, which has been termed the atherogenic phenotype, has been identified as a strong risk marker for coronary heart disease (2). Its characteristics overlap considerably with those of the metabolic syndrome (also known as the insulin-resistance syndrome) (20, 30) and include hypertriglyceridemia, often relatively mild.

The hypertriglyceridemic state promotes exchange of core lipids; very-low-density lipoproteins (VLDL) exchange their triacylglycerol (TAG) for cholesteryl ester from the cholesterol-rich lipoproteins, which are then lipid depleted through the action of lipases. The end result is a depletion of high-density-lipoprotein (HDL) cholesterol and a preponderance of small, dense low-density lipoproteins (LDL). From the metabolic point of view, postprandial lipemia can be considered as a transient episode of hypertriglyceridemia that occurs several times each day. Recent evidence points to the magnitude of the plasma TAG response to fat intake as a major determinant of LDL heterogeneity measured in the fasted state (17), so it is important to identify the factors that influence this response. Rapid removal of TAG-rich lipoproteins will reduce the severity of postprandial lipemia and, potentially, by inference, its atherogenic sequelae.

Endurance-trained people are reported to exhibit low levels of postprandial lipemia (6, 24), and there is evidence that this may be caused by high rates of TAG removal (5, 6, 11, 29, 32). The rate-limiting step in this process is hydrolysis of lipoprotein TAG by endothelium-bound lipoprotein lipase (LPL). LPL activity may be enhanced in endurance-trained individuals because of their large, well-vascularized skeletal muscle mass. The strong relationship between capillarization and LPL activity measured in the same biopsy samples of muscle (18) provides evidence for this. Although it has been less well explored, the possibility also exists that adipose-tissue LPL activity is enhanced in athletes (27) and/or that muscle blood flow in the basal state (fasting, nonexercised) is higher than in sedentary people (10).

In trained individuals, muscle capillary density remains high for at least 12 wk when training stops (8), and, if muscle capillarization is closely coupled to postprandial lipemia, there should be no change in lipemia during the early weeks of detraining. Two studies (23, 40) provide support for this proposition; they report that the plasma TAG response to a fatty meal in athletes was not changed after 2–3 wk of detraining.

Cross-sectional studies of athletes and sedentary controls, as well as data on detraining, are suggestive of a long-lasting effect of endurance training on postprandial lipemia. However, high TAG-removal rates and low levels of postprandial lipemia have typically been observed when athletes were studied 12–24 h after exercise (5, 6, 24, 29, 32). TAG removal has been reported to be increased 18 h (34) and 24 h (1) after a single session of exercise, however, and some of these findings embody the residual effects of the last exercise session. Therefore, the extent to which training stimulates long-term adaptations that lead to chronically low levels of lipemia is unclear.

The time course for the reversal of low levels of lipemia in trained people could be an important consideration in exercise prescription, particularly for individuals who exhibit metabolic risk factors for coronary heart disease. The purpose of the present study was to examine changes in the metabolic capacity for TAG in endurance-trained people on three occasions during a short interruption to training. Partly because of the need to elicit an insulin response, we chose a high-fat mixed meal, rather than an intravenous lipid test, as the challenge to TAG metabolic capacity. Insulin plays an important role in the coordination of postprandial lipid metabolism, and the decline in sensitivity over a
few days without exercise (19) must be a key feature of the detraining process.

**METHODS**

Subjects. Ten normolipidemic endurance-trained volunteers (9 men, 1 woman; ages 18–55 yr) participated in this study, which was approved by the University’s Ethical Advisory Committee. They gave written informed consent after receiving an explanation of the procedures and risks involved. Seven were distance runners, two were triathletes, and one (the woman) was a cyclist. Two competed at the international level, three at the regional level, two at club level, and three were committed recreational runners (two of whom also swam regularly). Subjects had been training for between 4 and 32 yr (median, 7 yr). At the time of the study, they undertook between 4 and 20 sessions each week (median, 6 sessions). The overall training commitment was lowest for one of the recreational runners who ran 20 miles/wk and was greatest for an international triathlete who trained three times on most days (each week running 30 miles, cycling 150 miles, and swimming 10,000 m). Subjects were enrolled only if they met all of the following criteria: nonsmoking, systemic arterial blood pressure <160/90 mmHg, normolipidemic (total cholesterol <6.8 mM, fasting TAG <2.3 mM), no cardiovascular or metabolic disease diagnosed by a physician, and not taking (at the time of the study or during the previous 6 mo) pharmacotherapeutic drugs known to affect lipid or carbohydrate metabolism. Some of the subjects' physical characteristics are shown in Table 1. Five of the subjects (including the woman) exhibited the E3/3 phenotype for apolipoprotein E, two exhibited the E4/3 phenotype, two had the E4/4 phenotype, and one had the E3/2 phenotype.

Study design. Each subject undertook three oral fat tolerance tests over a period of 1 wk. During that time, they refrained from training. Only gentle cycling and walking for personal transportation over short distances were permitted. The fat tolerance tests began 15 h, 60 h, and 6.5 days after each individual’s last training session, which was ≥30 min in duration. Subjects weighed and recorded all food and drink consumed during the 24 h immediately preceding their first test, and they undertook to replicate this intake during the 24 h before each subsequent test. They refrained from alcohol for 24 h before each test.

Fat tolerance tests. Subjects reported to the laboratory at 0800 after a 12-h fast. A cannula was inserted into a forearm or antecubital vein, and the subject rested quietly for 10 min before a baseline blood sample was obtained. The test meal was then consumed within 15 min. This meal comprised whipping cream, fruit, cereal, nuts, and chocolate and was given according to body mass (per kg body mass: 1.2 g fat, 1.1 g carbohydrate, 0.2 g protein, and 66 kJ). Subjects therefore consumed 87 ± 14 (SD) g of fat, 80 ± 13 g of carbohydrate, 14 ± 2 g of protein, and 4.77 ± 0.8 Mj of energy (69% from fat, 27% from carbohydrate). Further blood samples were obtained 0.5 and 1 h after completion of the meal and then hourly for a total of 6 h. The cannula was kept patent by flushing with nonheparinized saline (9 g/l). The first 2 ml of blood withdrawn were always discarded to avoid dilution of the sample. Every subject consumed all of the prescribed meal, and none reported nausea or other gastrointestinal discomfort. Only water was consumed during the 6-h postprandial observation period. Water was available ad libitum during the first trial; the volume ingested was recorded and replicated in subsequent trials. Subjects rested (reading, working quietly, watching television) throughout each observation period and were always seated for at least 10 min before blood samples were taken.

Anthropometry. Height and weight were determined by using standard methods. Skinfold thicknesses were measured at four sites, i.e., biceps, triceps, subscapular, and suprailliac.

Analytical methods. At each sampling point, blood samples were collected into precooled 9-ml potassium-EDTA Monovettes (Sarstedt, Leicester, UK) and were kept on ice before centrifugation. A separate sample was dispensed into a plain tube for preparation of serum. Plasma was separated and stored at −20°C until analyzed for glucose, TAG, cholesterol, HDL cholesterol (Boehringer Mannheim, Lewes, UK) and nonesterified fatty acids (NEFA; Wako, Neuss, Germany) by enzymatic, colorimetric methods with the use of a centrifugal analyzer (Cobas-Bio; Roche, Basel, Switzerland). Serum was separated and stored at −20°C for determination of serum insulin concentration by using a solid-phase [125I] radiomunnoassay available in a commercial kit (COAT-A-COUNT Insulin; Diagnostic Products, Los Angeles, CA). Samples from all three trials for each subject were always analyzed in the same batch. Hemoglobin concentration and hematocrit were determined in baseline and 6-h samples so that changes in plasma volume could be estimated (9). Phenotypes of apolipoprotein E were determined in a human genetics laboratory by isoelectric focusing with the use of Western blot techniques.

Accuracy and precision were maintained by using quality-control sera (Boehringer Mannheim and Roche). Within-batch coefficients of variation were 1.9% for total cholesterol, 1.2% for TAG, 1.2% for glucose, 1.3% for NEFAs, 4.2% for insulin, and 3.1% for HDL cholesterol.

Analysis of weighed food inventories. Weighed food inventories were analyzed by using a computerized version (COMPEAT, Nutrition Systems, London) of food-composition tables (15).

Calculations and statistics. In plasma samples obtained in the fasted state only, VLDL-cholesterol concentration was calculated from the plasma TAG concentration by using the formula of Friedwald et al. (13), taking into account the fact that plasma TAG was expressed in Système International units. LDL-cholesterol concentration was then calculated by subtraction of VLDL- and HDL-cholesterol from total cholesterol.

The total lipemic and insulimemic responses were determined as the areas under curves for the plasma/serum concentration vs. time by using the trapezoidal rule. The incremental responses were determined by the same method, but they were normalized to the 0-h level.

Comparisons between trials were made by using analysis of variance, followed by post hoc tests to assess the significance of differences between pairs of means. Relationships were evaluated by using Pearson’s product-moment correlation coefficient. A 5% level of significance was adopted throughout, and results are expressed as means ± SE unless otherwise stated.

**Table 1. Some physical characteristics of the subjects**

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th></th>
<th>Woman</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>39.8 (20.9–55.4)</td>
<td></td>
<td>18.3</td>
<td></td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>70.0 (66.0–95.1)</td>
<td></td>
<td>57.5</td>
<td></td>
</tr>
<tr>
<td>Height, m</td>
<td>1.77 (1.60–1.81)</td>
<td></td>
<td>1.57</td>
<td></td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>22.4 (20.4–29.0)</td>
<td></td>
<td>23.3</td>
<td></td>
</tr>
<tr>
<td>Σ 4 skinfolds, mm</td>
<td>35.7 (20.4–83.4)</td>
<td></td>
<td>49.0</td>
<td></td>
</tr>
</tbody>
</table>

Values are medians with range in parentheses; n = 9 men and 1 woman.
RESULTS

The time taken for the subjects to consume the meal was similar in all trials, i.e., 9.5 ± 0.6, 9.7 ± 0.6, and 9.0 ± 0.3 min at 15 h, 60 h, and 6.5 days, respectively (P > 0.05).

Between-trial differences in plasma volume were estimated by using average values for hemoglobin concentration and hematocrit measured before and after the fat tolerance test. Relative to the first trial, plasma volume was increased by 0.2 ± 1.1% at 60 h and by 0.6 ± 2.5% at 6.5 days. Plasma concentrations of TAG were adjusted for these differences on an individual basis, and the corrected values are reported.

Plasma and serum concentrations in the fasted state. Concentrations of TAG, NEFA, insulin, glucose, cholesterol, and HDL cholesterol measured in the fasted state are shown in Table 2. TAG concentrations increased by an average of 47% during 6.5 days without exercise, and most of this increase occurred between the trials at 15 h and at 60 h. VLDL cholesterol increased with detraining, as did the ratio of total cholesterol-to-HDL cholesterol (Table 2). Concentrations of NEFAs were markedly lower at 60 h than at 15 h, with little change thereafter.

Table 2. Concentrations of plasma triacylglycerol; non-esterified fatty acids; total cholesterol; HDL, LDL, and VLDL cholesterol; glucose; and serum insulin in the fasted state measured 15 h, 60 h, and 6.5 days after last exercise session

<table>
<thead>
<tr>
<th>Time After Last Exercise</th>
<th>15 h</th>
<th>60 h</th>
<th>6.5 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triacylglycerol, mM</td>
<td>0.85 ± 0.15</td>
<td>1.09 ± 0.12</td>
<td>1.10 ± 0.11</td>
</tr>
<tr>
<td>Nonesterified fatty acids, mM</td>
<td>0.62 ± 0.05</td>
<td>0.39 ± 0.04</td>
<td>0.35 ± 0.06</td>
</tr>
<tr>
<td>Total cholesterol, mM</td>
<td>4.56 ± 0.24</td>
<td>4.71 ± 0.21</td>
<td>4.75 ± 0.26</td>
</tr>
<tr>
<td>HDL cholesterol, mM</td>
<td>1.17 ± 0.06</td>
<td>1.14 ± 0.08</td>
<td>1.13 ± 0.09</td>
</tr>
<tr>
<td>Ratio of total to HDL cholesterol</td>
<td>3.99 ± 0.28</td>
<td>4.27 ± 0.30</td>
<td>4.29 ± 0.25</td>
</tr>
<tr>
<td>LDL cholesterol, mM†</td>
<td>3.01 ± 0.20</td>
<td>3.07 ± 0.17</td>
<td>3.12 ± 0.19</td>
</tr>
<tr>
<td>VLDL cholesterol, mM†</td>
<td>0.39 ± 0.07</td>
<td>0.50 ± 0.06</td>
<td>0.50 ± 0.05</td>
</tr>
<tr>
<td>Insulin, µIU/ml</td>
<td>6.6 ± 0.7</td>
<td>6.5 ± 0.6</td>
<td>7.1 ± 1.1</td>
</tr>
<tr>
<td>Glucose, mM</td>
<td>4.57 ± 0.11</td>
<td>4.62 ± 0.12</td>
<td>4.61 ± 0.11</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 10 subjects. HDL, high-density lipoprotein; LDL, low-density lipoprotein; VLDL, very-low-density lipoprotein. *Significantly different from 15 h, P < 0.05. †Estimated by using Friedewald formula (13).

Postprandial concentrations of NEFAs, insulin, and glucose are shown in Fig. 3. Suppression of NEFAs (difference between concentration at baseline and at the lowest point of the postprandial response) was significantly reduced at 60 h and at 6.5 days after exercise compared with 15 h (0.34 ± 0.04, 0.15 ± 0.02, and 0.10 ± 0.03 mM at 15 h, 60 h, and 6.5 days, respectively). Both insulinemic responses were higher (P < 0.05) after 6.5 days without exercise than at 15 h postexercise. The total response was 81.6 ± 11.3, 87.6 ± 11.4, and 94.5 ± 9.4 µIU/ml × 6 h at 15 h, 60 h, and 6.5 days, respectively. The incremental response was 41.8 ± 8.2, 48.5 ± 9.9, and 52.8 ± 7.5 µIU/ml × 6 h. There were no significant changes in plasma glucose concentrations.

Fig. 1. Plasma triacylglycerol response during fat tolerance tests conducted 15 h, 60 h, and 6.5 days after last exercise session. Values are means ± SE for 10 subjects.

Table 2. Concentrations of plasma triacylglycerol; non-esterified fatty acids; total cholesterol; HDL, LDL, and VLDL cholesterol; glucose; and serum insulin in the fasted state measured 15 h, 60 h, and 6.5 days after last exercise session

<table>
<thead>
<tr>
<th>Time After Last Exercise</th>
<th>15 h</th>
<th>60 h</th>
<th>6.5 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triacylglycerol, mM</td>
<td>0.85 ± 0.15</td>
<td>1.09 ± 0.12</td>
<td>1.10 ± 0.11</td>
</tr>
<tr>
<td>Nonesterified fatty acids, mM</td>
<td>0.62 ± 0.05</td>
<td>0.39 ± 0.04</td>
<td>0.35 ± 0.06</td>
</tr>
<tr>
<td>Total cholesterol, mM</td>
<td>4.56 ± 0.24</td>
<td>4.71 ± 0.21</td>
<td>4.75 ± 0.26</td>
</tr>
<tr>
<td>HDL cholesterol, mM</td>
<td>1.17 ± 0.06</td>
<td>1.14 ± 0.08</td>
<td>1.13 ± 0.09</td>
</tr>
<tr>
<td>Ratio of total to HDL cholesterol</td>
<td>3.99 ± 0.28</td>
<td>4.27 ± 0.30</td>
<td>4.29 ± 0.25</td>
</tr>
<tr>
<td>LDL cholesterol, mM†</td>
<td>3.01 ± 0.20</td>
<td>3.07 ± 0.17</td>
<td>3.12 ± 0.19</td>
</tr>
<tr>
<td>VLDL cholesterol, mM†</td>
<td>0.39 ± 0.07</td>
<td>0.50 ± 0.06</td>
<td>0.50 ± 0.05</td>
</tr>
<tr>
<td>Insulin, µIU/ml</td>
<td>6.6 ± 0.7</td>
<td>6.5 ± 0.6</td>
<td>7.1 ± 1.1</td>
</tr>
<tr>
<td>Glucose, mM</td>
<td>4.57 ± 0.11</td>
<td>4.62 ± 0.12</td>
<td>4.61 ± 0.11</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 10 subjects. HDL, high-density lipoprotein; LDL, low-density lipoprotein; VLDL, very-low-density lipoprotein. *Significantly different from 15 h, P < 0.05. †Estimated by using Friedewald formula (13).
Dietary analysis. There were no significant differences in the intake of energy or macronutrients during the day before each oral fat tolerance test (Table 3), although there was a small decrease (4%) in energy intake.

**DISCUSSION**

Exercise-trained people have high TAG clearance rates and a low plasma TAG response to a dietary fat challenge, but little is known about the length of time these characteristics persist if training is interrupted. We sought to examine the changes in postprandial lipemia on three occasions during the early phase of detraining. Our findings demonstrate the important contribution of a recent session of exercise to the low levels of lipemia that are characteristic of trained people. In comparison with values measured 15 h after the last training session, lipemia was 45% greater after 60 h without exercise, with little further increase after nearly 1 wk without exercise. These increases in lipemia are, by an order of magnitude, greater than differences seen on a test-retest basis (mean difference between two trials for eight subjects: 1.9%, 95% confidence interval, −20 to +16%; J. M. R. Gill and A. E. Hardman, unpublished data).

We have no evidence concerning whether the increased lipemia was caused by faster appearance rates of TAG-rich lipoproteins or to reduced disappearance rates (or both), but marked increases in appearance rates are unlikely. Substrate delivery to the liver is the major determinant of VLDL secretion (37), and concentrations of NEFAs were lower (in the early postprandial phase) or unchanged (later postprandial phase) when exercise was withdrawn, with little variation in plasma glucose concentrations. Effects of greater anxiety during the first trial could conceivably alter gastrointestinal handling of dietary fat, relative to subsequent trials, and, hence, chylomicron appearance rates. All subjects approached the first trial calmly, however, and there does not appear to be a systematic effect on postprandial lipemia with repeated testing (16). Therefore, the most likely explanation for our findings is a decrease in rates of catabolism of TAG-rich lipoproteins. Chylomicrons and VLDL are cleared by a common saturable pathway (4). The fasting pool size increased with detraining, so the increase in lipemia probably reflects increased competition by VLDL for LPL and so reduced overall TAG-removal rate.

Decreased catabolism could not be linked with major structural changes in either adipose tissue or skeletal muscle over only 1 wk (8). One possible explanation relates to changes in the activity of LPL as a conse-

**Table 3.** Amounts of energy, fat, carbohydrate, and protein ingested during day before fat tolerance tests conducted 15 h, 60 h, and 6.5 days after last exercise session

<table>
<thead>
<tr>
<th>Time After Exercise</th>
<th>Energy, MJ</th>
<th>Fat, g</th>
<th>Carbohydrate, g</th>
<th>Protein, g</th>
</tr>
</thead>
<tbody>
<tr>
<td>15 h</td>
<td>11.5 ± 1.0</td>
<td>97 ± 10</td>
<td>382 ± 36</td>
<td>105 ± 12</td>
</tr>
<tr>
<td>60 h</td>
<td>11.4 ± 1.1</td>
<td>93 ± 12</td>
<td>389 ± 43</td>
<td>102 ± 10</td>
</tr>
<tr>
<td>6.5 days</td>
<td>11.0 ± 1.1</td>
<td>89 ± 11</td>
<td>363 ± 40</td>
<td>110 ± 15</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 10 subjects.
sequence of removal of some aspect(s) of the exercise response, perhaps a mechanism for replenishing muscle TAG stores reduced by exercise (1). The runners’ last training session would have stimulated a delayed but transient increase in muscle LPL activity, although there is uncertainty about the time course of this increase. Our data are consistent with reports of increases in muscle LPL activity evident at >8 h but <20 h after an exercise session (35) and at 14–18 h after exercise (34). The small difference in lipemia between 60 h and 6.5 days is in keeping with earlier studies that showed that the effects of exercise on plasma LPL activity and TAG removal cannot be detected 2 days after the exercise (1).

There are few reports of the effect of an interruption to endurance training on postprandial lipemia. Patsch et al. (28) present data for one well-trained man who was forced to cease training for surgery; 4 wk later, postprandial lipemia was markedly increased. In contrast, two other studies report no change with 2–3 wk of detraining in groups of 8 and 11 runners (23, 40). One reason for these apparently conflicting findings may be the timing of measurements in relation to the last exercise session and so to changes in LPL activity. Verstraete et al. (40) do not give any information on this. In the study by Mankowitz and colleagues (23), measurements in the trained state were made >20 h after exercise. Therefore they might not reflect the effects of acute exercise as strongly as measurements in the present study. Differences from the untrained state (2–3 wk later) would have been correspondingly less. Furthermore, Mankowitz and colleagues found an increase of 40% in the chylomicron-retinyl ester response to the test meal after detraining; this finding is difficult to reconcile with an unchanged plasma TAG response if >80% of the rise in the plasma TAG concentration after a fatty meal is caused by increases in the chylomicron fraction (7).

The metabolic capacity for TAG is a strong determinant of HDL-cholesterol concentrations, because the residence time of TAG-rich lipoproteins in the circulation dictates the opportunity for exchange of their lipid with cholesterol-rich lipoproteins. Although we found no significant changes in HDL cholesterol on detraining (Table 2), there were indications of altered cholesterol metabolism. Both the concentration of VLDL cholesterol and the ratio of total cholesterol to HDL cholesterol increased with detraining; the pattern of change was similar to that for lipemia. One reason that the decrease in HDL cholesterol was not more marked may be that it takes >1 wk for changes in HDL cholesterol to be detected. Decreases have been reported with detraining if the period without exercise has been longer, i.e., 2–3 wk (23) and 1 mo (25).

We cannot compare levels of postprandial lipemia in trained people with those in the sedentary population because there is no standard fat tolerance test, and normative data are not available from the literature. However, the postprandial TAG response at 6.5 days in our trained men (12.5 ± 1.6 mM × 6 h for n = 9; omitting the female subject) is certainly not low in relation to data from our laboratory for 22 healthy, normolipidemic men of average or below-average physical activity level, i.e., 10.1 ± 0.8 mM × 6 h. These untrained men were 32.2 ± 2.2 yr of age, with body mass index values of 24.3 kg/m² and fasting TAG concentrations of 0.99 ± 0.08 mM (Refs. 15 and 23, and unpublished data). None of them exercised during the 2 days before a fat tolerance test.

Apolipoprotein E phenotype influences postprandial lipoprotein metabolism (39). Both the ε2 allele (38) and the ε4 allele (3) have been reported to be associated with perturbations of the lipolytic process, compared with the ε3 allele. In the present study, the individual who did not show an increase in lipemia when training ceased (15 vs. 60 h) possessed the E4/E4 phenotype. However, this may not have been linked to this characteristic, because the other individual with this phenotype showed a 50% increase in lipemia over the same period. Moreover, the two subjects who were heterozygous for the ε4 allele also showed a clear increase (27 and 103%) in lipemia with detraining.

In this study, we used a meal containing fat with carbohydrate, because it maintained the interactions of changes in insulin sensitivity with changes in lipid metabolism. Postprandial insulin responses did not differ between 15 and 60 h but had increased (by 16%, P < 0.05) after 6.5 days without training, in line with recent evidence in trained middle-aged people that the improved insulin action after exercise persists for 3 days but not for 5 days (19). However, the time course of the changes in insulinemia differed from that in lipemia (Figs. 1 and 3), and the correlations between these changes were weak (15 vs. 60 h, r = 0.26; 15 h vs. 6.5 days, r = 0.03), so these two were probably unrelated. Decrements in insulin action could have contributed to the weaker suppression of NEFA postprandially over the detraining period; but, again, the time course does not fit well.

Insulin influences LPL, stimulating its activity in adipose tissue but with the opposite effect in skeletal muscle (12). The interactions with training are complex, however, and improved insulin sensitivity has been reported to be associated with a reduction in adipose tissue LPL activity (21). Detraining appears to upregulate LPL in adipose tissue, with reciprocal decreases in muscle LPL. In athletes, the ratio of (heparin-releasable) LPL activity in these tissues increased fourfold after 2 wk without exercise (36), favoring storage in adipose tissue. In the present study, the relative importance of adipose tissue and muscle as sites for TAG uptake may therefore have changed during the days without training. Our data show only the net effect of changes in all tissues on the plasma TAG response.

The importance of skeletal muscle as a site of TAG removal is a matter of debate. This tissue has been reported to take up 50% of an intravenous fat load, compared with 13% in adipose tissue (31), but other studies that used arteriovenous differences to determine responses to normal meals have found muscle, presumably nonexercised muscle, to be much less impor-
Postprandial Lipemia: Interruption to Training

The parallel between rapid exercise-related changes in gene expression for LPL and for proteins critical for the transport and metabolism of glucose, i.e., GLUT-4 and hexokinase, has already been drawn (35). All these changes influence substrate acquisition by skeletal muscle and the transport and metabolism of glucose, i.e., GLUT-4 transport and metabolism in skeletal muscle. The consequences for glucose tolerance and insulin sensitivity, particularly in insulin-resistant individuals, are well documented.


REFERENCES

The authors thank the subjects for their willingness to interrupt their training. Supported by the British Heart Foundation. Address for reprint requests: A. E. Hardman, Reader in Human Exercise Metabolism, Dep. of Physical Education, Sports Science and Recreation Management, Loughborough Univ., Loughborough, Leicestershire, LE11 3TU, UK (E-mail: a.e.hardman@lboro.ac.uk). Received 25 September 1997; accepted in final form 29 January 1998.

Downloaded from http://ajpphysiology.org/ by 10.220.32.246 on April 29, 2017.


