Postprandial lipemia: effects of exercise and restriction of energy intake compared\textsuperscript{1–3}

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ABSTRACT

Background: The mitigating effect of exercise on postprandial lipemia may be attributable to the energy deficit incurred.

Objective: We aimed to compare the effects of prior exercise and an equivalent energy intake deficit on postprandial lipemia.

Design: Eleven postmenopausal women participated in 3 oral-fat-tolerance tests after undergoing different treatments on the preceding day: control (subjects refrained from exercise and consumed a prescribed diet), exercise (subjects consumed the same diet but walked briskly for 90 min), and intake restriction (subjects' food intake was restricted to induce the same energy deficit, relative to control, as brought about by the 90-min walk). Venous blood samples were obtained after subjects fasted overnight, 30 min after they ate a mixed, high-fat meal (1.70 g fat, 1.65 g carbohydrate, and 99 kJ/kg fat-free body mass), and hourly for the next 6 h.

Results: In the exercise trial, the mean fasting triacylglycerol concentration was 19% and 17% lower than the control and intake restriction values, respectively \((P < 0.05\) for both). Compared with the control trial, exercise reduced postprandial lipemia by a mean of 20% \((P < 0.05)\), whereas intake restriction reduced it by 7\% (NS). In the exercise trial, fasting and postprandial fatty acid concentrations were higher than control values \((P < 0.05)\). Exercise, but not intake restriction, reduced postprandial insulin concentrations.

Conclusions: The results suggest that the effect of exercise on postprandial lipid metabolism was greater than and different from that attributable to the energy deficit incurred. Am J Clin Nutr 2000;71:465–71.

KEY WORDS Exercise, triacylglycerol, women, energy deficit, lipemia, fat ingestion, lipid metabolism, insulin, fatty acids

INTRODUCTION

In the 20 y since Zilversmit (1) proposed that atherogenesis was a postprandial phenomenon, there has been increasing interest in postprandial lipid metabolism. Epidemiologic evidence (2), case-control clinical studies (3–5), and a growing body of scientific research suggest that exaggerated postprandial lipemia is linked to the progression of atherosclerosis, either directly via the deposition of postprandial lipoprotein remnants onto artery surfaces (6) or indirectly by contributing to a predominance of small, dense LDL particles and HDL\(_3\) cholesterol (7). Thus, interventions that can moderate the postprandial lipid response may be beneficial.

Several studies have shown that in both males and females of different ages, a single session of exercise before the ingestion of a fat-rich meal attenuates postprandial lipemia (8–13), and the magnitude of this reduction appears to be linked to the energy expended during the exercise session (9–11). However, in all these investigations, food intake was standardized during the day before the oral-fat-tolerance test so that exercise brought about a short-term energy deficit relative to the control trials.

It is possible that in response to an energy deficit, the body may clear triacylglycerol more rapidly from the circulation to replace energy stores. Increased activity of lipoprotein lipase (LPL), a key enzyme in the hydrolysis of triacylglycerol, may play a role in this process. In humans it has been shown that after restriction of energy intake, the postprandial rise in adipose tissue LPL activity was increased (14). Moreover, in the postprandial state, adipose tissue LPL activity was higher in previously energy-restricted rats than in control rats ad libitum (15, 16).

In addition, loss of body mass, a consequence of prolonged energy deficit, has been shown to increase adipose tissue LPL activity (17). Consequently, it is possible that the attenuation of lipemia by exercise is attributable to the associated energy deficit rather than to the exercise per se. Therefore, the purpose of this study was to compare the effects on postprandial lipemia of prior exercise and an equivalent energy deficit induced by restriction of food intake.

Postmenopausal women were studied because, despite the popular perception that coronary heart disease is a male disease, heart disease is responsible for 23\% of female mortality and is the leading cause of death in women <60 y of age in the United Kingdom (18). With the onset of menopause, women lose much of the cardioprotective effect of endogenous estradiol and their coronary heart disease risk approaches that of

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men (19); therefore any intervention that reduces coronary heart disease risk is particularly relevant for this group. In addition, the possible effects of cyclic hormone concentrations on lipid and carbohydrate metabolism may confound the study of premenopausal women.

SUBJECTS AND METHODS

Subjects

The subjects were 12 moderately active but untrained postmenopausal (with amenorrhea for > 2 y) volunteers aged 60.2 ± 3.8 y. For the subject group, the following mean (±SD) values were obtained: body mass index (in kg/m²), 24.8 ± 1.3; sum of 4 skinfold thicknesses, 60.9 ± 11.5 mm; waist circumference, 78.3 ± 6.4 cm; and estimated maximal oxygen uptake (VO₂max), 30.7 ± 3.2 mL·kg⁻¹·min⁻¹. The study was conducted with the approval of the Loughborough University Ethical Advisory Committee. All subjects were fully informed of the procedures and risks involved and gave their written consent to participate. One subject did not complete the study, so data are presented for 11 women. All the women were apparently healthy nonsmokers and none was receiving hormone replacement therapy or any drugs thought to affect lipid or carbohydrate metabolism.

Preliminary testing

Subjects were familiarized with walking on the motorized treadmill in the laboratory and then, on a separate occasion, performed a 4-stage, 20-min submaximal incremental treadmill test. Expired air samples were collected by using standard Douglas bag techniques and heart rates were monitored with electrocardiography (modified lead I). The relation of oxygen uptake (VO₂) to heart rate was extrapolated up to the age-predicted maximum heart rate [mean of (220 – age) beats/min and (210 – 0.5 × age) beats/min] and VO₂max was estimated.

During a separate visit, subjects performed a test designed to estimate the net energy expenditure resulting from a 90-min walk at 60% VO₂max. When the subjects arrived at the laboratory, they sat quietly for 30 min to ensure that they were in a rested state. A 6-min expired air sample was then obtained so that their resting metabolic rates could be estimated, via indirect calorimetry. Subjects then walked on the treadmill for 90 min at a speed and intensity calculated to elicit 60% of estimated VO₂max. Two-minute expired air samples were obtained starting at 13, 28, 58, and 88 min into the walk. Rates of perceived exertion (21) and heart rates were also recorded at these time points. On completion of the walk, subjects were seated and expired air samples were collected continuously for 15 min to estimate the excess postexercise VO₂ (a pilot study had indicated that VO₂ returned to baseline values within this time period). The net energy expenditure of exercise (ie, the energy expenditure over and above the resting metabolic rate) was calculated on an individual basis by using indirect calorimetry.

Subjects weighed and recorded their food intake for 3 d (2 weekdays and 1 weekend day) before the study so that their normal dietary intake could be assessed. Food diaries were analyzed with a computerized version of food composition tables (COMP-EAT 5; Nutrition Systems, London; 22).

Main trials: study protocol

Subjects participated in 3 oral-fat-tolerance tests with different preceding interventions (control, exercise, and intake restriction) in a balanced design.

Control trial

On the day before the oral-fat-tolerance test, subjects refrained from exercise and carried out only the activities of daily living. They were asked to sit quietly for 90 min during the afternoon, a period equivalent to the time they would spend walking in the exercise trial, because the additional energy expenditure of the walk was calculated relative to sitting. They consumed only food provided by the experimenters; the energy value of the food corresponded to their usual energy intake. For all subjects, 50% of energy was from carbohydrate, 36% was from fat, and 14% was from protein.

Exercise trial

On the day before the oral-fat-tolerance test, subjects attended the laboratory during the afternoon (midway between lunch and dinner) and walked on the treadmill for 90 min at the same speed and gradient as in the earlier walk to estimate energy expenditure. Subjects consumed the same diet as in the control trial; the foods were provided by the experimenters.

Intake restriction trial

On the day before the oral-fat-tolerance test, subjects performed only the activities of daily living and sat quietly for 90 min during the afternoon, as in the control trial. However, they consumed a diet (provided by the experimenters) with the same macronutrient composition as the diets consumed in the other 2 trials but reduced in energy by the net energy expenditure of exercise (ie, energy expended during exercise and the postexercise period over and above resting energy expenditure). Thus, the energy deficit (relative to the control trial) elicited in this trial was similar to that elicited in the exercise trial. The energy intakes at lunch and dinner were each reduced by half the total net expenditure of exercise, so that the deficit occurred at similar times of day in the exercise and intake restriction trials.

On the day before the intervention in the first trial, subjects weighed and recorded their dietary intakes. This diet was replicated before subsequent trials. In addition, for the 2 d before the intervention in each trial, subjects refrained from exercise.

Oral-fat-tolerance tests

Subjects reported to the laboratory after an overnight fast of ≥12 h, having traveled by car to ensure that they were in a rested state. A cannula was introduced into an antecubital or forearm vein under local anesthesia and, after a 10-min interval, a blood sample was obtained [9 mL into a precooled EDTA-coated Monovette tube (Sarstedt, Leicester, United Kingdom), 4.5 mL into a non-heparin-containing serum Monovette tube (Sarstedt), and 5 mL into a precooled EDTA-coated Monovette tube (Sarstedt) containing 250 μL Trasylol (Bayer Plc, Newbury, United Kingdom)]. A cereal-based, high-fat test meal composed of whipping cream, oats, nuts, coconut, chocolate, and fruit (1.70 g fat, 1.65 g carbohydrate, 0.25 g protein, and 99 kJ/kg fat-free body mass) was then consumed. Additional blood samples [9 mL into a precooled EDTA-coated Monovette tube (Sarstedt) and 4.5 mL into a non-heparin-containing serum Monovette tube (Sarstedt) and 4.5 mL into a non-heparin-containing serum Monovette tube...
(Sarstedt) were obtained half an hour after completion of the meal and then hourly for 6 h. During the postprandial period, only water was consumed; this was provided ad libitum in the first trial and then replicated in subsequent trials.

Sample analyses

Blood collected in the EDTA-coated Monovette tubes was separated within 15 min of collection and aliquots of plasma were stored at −20°C before being analyzed. Samples were analyzed for HDL cholesterol (fasting samples only) by a man-ganese chloride and phosphotungstic acid precipitation method (Boehringer Mannheim GmbH, UK Limited, Lewes, United Kingdom). We also analyzed samples for total cholesterol (fasting samples only), triacylglycerol, and glucose (all Boehringer Mannheim GmbH, UK Limited) and fatty acids (Wako Chemicals, Neuss, Germany) by enzymatic colorimetric methods with a centrifugal analyzer (Cobas-Bio, Roche, Basel, Switzerland). Plasma glucagon concentrations were determined by radioimmunoassay. Blood collected in the serum Monovette tubes was allowed to clot for 60 min before separation. Serum was stored at −70°C before analysis for insulin by radioimmunoassay (COAT-A-COUNT Insulin, Diagnostic Products Corporation, Los Angeles). All samples for one subject were analyzed in the same run. Quality-control sera (Roche, Boehringer Mannheim, and Nycomed Pharma AS, Billingstad, Norway) were used to ensure accuracy. Within-batch CVs were 1.3% for triacylglycerol, 0.8% for glucose, 1.1% for total cholesterol, 1.8% for HDL cholesterol, 0.9% for fatty acids, 3.9% for insulin, and 6.9% for glucagon. Apolipoprotein (apo) E phenotypes were determined by isoelectric focusing with Western blot techniques in a human genetics laboratory.

Statistical analyses

The 6-h area under the plasma triacylglycerol concentration versus time curve calculated by using the trapezium rule was defined as the total lipemic response, with the area normalized to the baseline concentration defined as the incremental lipemic response. Similarly, the total and incremental glycemic and insulinemic responses and the total fatty acid response were calculated as the 6-h areas under the respective plasma or serum insulinemic responses and the total fatty acid response were calculated by using the trapezium rule was defined as the total lipemic response, with the area normalized to the baseline concentration defined as the incremental lipemic response. Both the total and incremental lipemic responses did not differ among the trials. Nine subjects had the most common apo E phenotype, E3,3, whereas one subject had the E3,2 phenotype and one had the E4,3 phenotype.

### Postprandial plasma and serum concentrations

The plasma triacylglycerol responses to the test meal in the 3 trials are shown in Figure 2 and the total and incremental lipemic responses are shown in Figure 2. Both the total and incremental lipemic responses were lower in the exercise trial than in the control trial (P < 0.05). Although the total lipemic response was not significantly attenuated by intake restriction (P = 0.17), there was a tendency for the incremental lipemic response to be lower in the exercise than in the control trial (P = 0.08). The cumulative triacylglycerol response was 2.01 ± 0.12 mmol/L · h, which represented a 41% decrease in triacylglycerol response in the exercise trial compared with the control trial. The incremental triacylglycerol responses did not differ among the trials.

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response to be lower in the intake restriction trial than in the control trial \( (P = 0.07) \). Numerically, the reduction in the total lipemic response due to exercise was almost 3 times as great as the reduction due to intake restriction \( (2.14 \text{ compared with } 0.79 \text{ mmol} \cdot \text{h}^{-1} \cdot \text{L}^{-1}, \text{ respectively}) \), but the reductions in the incremental lipemic response were more similar for the 2 interventions \( (1.03 \text{ and } 0.68 \text{ mmol} \cdot \text{h}^{-1} \cdot \text{L}^{-1} \text{ for exercise and intake restriction, respectively}) \). Plasma glucose, serum insulin, and plasma fatty acid concentrations after the test meal are shown in Figure 3, and the summary responses are given in Table 2. The total glycemic response tended to be higher in the exercise trial than in the control trial \( (P = 0.07) \), but the incremental glycemic response did not differ among the trials. The total and incremental insulimic responses were lower in the exercise trial than in both the control and intake restriction trials \( (P < 0.05) \). The total fatty acid response was higher in the exercise trial than in the control trial \( (P < 0.05) \). In the exercise trial, the total fatty acid response also tended to be higher than in the intake restriction trial \( (P = 0.07) \). There were no significant correlations between fasting triacylglycerol concentrations or indexes of body composition and the differences among the trials in either total or incremental lipemic response.

**DISCUSSION**

The main finding of this study was that, in this group of postmenopausal women, the exercise-induced reduction in postprandial lipemia was almost 3 times as great as the reduction due to a similar diet-induced energy deficit. In addition, the effect of exercise on lipid and carbohydrate metabolism was qualitatively, as well as quantitatively, different from the effect of intake restriction. Exercise, but not intake restriction, reduced triacylglycerol concentrations in the fasting state and reduced postprandial insulin concentrations. In the fasting state, plasma fatty acid concentrations were significantly elevated by exercise only, and only exercise resulted in elevated postprandial fatty acid concentrations. Thus, it is clear that the effect of exercise on postprandial lipid metabolism was different from the effect of an energy deficit induced by dietary restriction.

The energy deficit incurred in the exercise trial was slightly greater than that induced by energy-intake restriction. However, the effect of exercise was different in nature from the effect of intake restriction and was so much greater that the small difference between the 2 energy deficits does not affect the interpretation of the results. The effect of exercise on postprandial lipemia is closely related to the energy expended during exercise \( (10, 11) \). Thus, the slightly different deficits between the 2 interventions would result in only slightly different effects on lipemia if the mechanisms by which both interventions acted were the same.

Exercise, but not intake restriction, reduced triacylglycerol concentrations in the fasting state. This reduction could have been due to increased clearance of triacylglycerol from the circulation into peripheral tissues or to reduced hepatic very-low-density-lipoprotein (VLDL) secretion. Previous research supports both an up-regulation of muscle LPL activity \( (23) \) and, in rats, reduced triacylglycerol secretion \( (24) \) after exercise training. However, from our current data it is not possible to ascertain the relative importance of the possible mechanisms, although it is clear that the effect was not mediated by energy deficit.

In the fasting state, fatty acid concentrations were significantly elevated in the exercise trial relative to the control trial and were numerically, although not significantly, elevated in the intake restriction trial. In the exercise trial, low muscle glycogen concentrations may have mediated the increased plasma fatty acid concentrations \( (25) \). Alternatively, elevated growth hormone concentrations during exercise \( (26) \) and in response to intake restriction \( (27) \) may have contributed to the elevated fatty acid concentrations in the exercise trial. This reduction could have been due to increased clearance of triacylglycerol from the circulation into peripheral tissues or to reduced hepatic very-low-density-lipoprotein (VLDL) secretion. Previous research supports both an up-regulation of muscle LPL activity \( (23) \) and, in rats, reduced triacylglycerol secretion \( (24) \) after exercise training. However, from our current data it is not possible to ascertain the relative importance of the possible mechanisms, although it is clear that the effect was not mediated by energy deficit.
concentrations in the 2 interventions because the lipolytic effect of this hormone is delayed and acts over several hours (28).

Exercise reduced both the total and incremental lipemic responses relative to the control trial, and the total response was also attenuated relative to intake restriction. As in the fasting state, this reduction could have been due to increased hydrolysis of circulating triacylglycerol mediated by increased activity of LPL or to reduced secretion of VLDL by the liver. Again, from the present data it is not possible to determine the relative importance of these 2 mechanisms. Theoretically, it is also possible that the appearance of chylomicrons into the circulation was delayed in the exercise trial. This is unlikely, however, because peak triacylglycerol concentrations occurred at a similar time in all 3 trials and any effects of exercise on gut function are unlikely to have persisted until the following day.

Intake restriction did not reduce the total lipemic response to the test meal relative to the control trial, but the incremental lipemic response tended to be attenuated (\( P = 0.07 \)). The finding that intake restriction moderated only the postprandial rise in plasma triacylglycerol and did not influence fasting triacylglycerol concentrations is consistent with the literature, which suggests that energy intake restriction promotes an increased postprandial rise in adipose tissue LPL activity and not an increase in activity in the fasting state (14–16).

The uptake of triacylglycerol from circulating chylomicrons and VLDL is a 2-stage process. Lipoprotein-triacylglycerol is first hydrolyzed by the action of LPL on the capillary endothelium, which liberates fatty acids. These fatty acids then move into adipocytes, where they are entrapped and reesterified. In the postprandial state, \( \approx 25–54\% \) of LPL-derived fatty acids are entrapped into adipocytes and the remainder spill over into the circulation (29). Indeed, the majority of plasma fatty acids in the postprandial state may be derived from the action of LPL (29).

In the exercise trial, postprandial plasma fatty acid concentrations were higher than in the control trial (\( P < 0.05 \)) and tended to be higher than in the intake restriction trial (\( P = 0.07 \)). This suggests greater spillover of fatty acids into the circulation in the exercise trial. This could have been due to increased action of adipose tissue LPL without a corresponding increase in esterification within adipocytes, or to reduced reesterification with similar LPL activity. Both possibilities are plausible and they are not mutually exclusive, but if the former mechanism were working exclusively we might expect elevated fatty acids in the intake restriction trial compared with the control trial because it is likely that adipose tissue LPL activity was increased in the intake restriction trial, and this was not seen. The postprandial insulin response was lower in the exercise trial than in the control and intake restriction trials. Because insulin is a potent stimulator of reesterification of fatty acids in adipocytes (30), it is not unreasonable to speculate that uptake of fatty acids into adipocytes was reduced in the exercise trial. Thus, after exercise, deposition of circulating triacylglycerol into adipose tissue might be reduced, with liberated fatty acids directed to muscle and the liver for oxidation. If true, this might have implications for obesity and the regulation of body fatness. This also suggests that the elevated fatty acid concentrations in the exercise trial compared with the control and intake restriction trials are entirely appropriate, in stark contrast to the inappropriately elevated fatty acid concentrations associated with the insulin resistance syndrome (31).

The total glucose response was somewhat higher in the exercise trial than the control trial (\( P = 0.07 \), which might have
been a consequence of the elevated fatty acid concentrations reducing the uptake of glucose into muscle via the glucose–fatty acid cycle (32). Thus, one might argue that the present data indicate a reduction in insulin sensitivity in the exercise trial. However, both the total and incremental insulin responses to the test meal were lower in the exercise trial than in control trials, and the incremental glycemic responses did not differ among trials. This suggests improved insulin sensitivity in the exercise trial; a lower insulin concentration was needed to control the rise in plasma glucose concentrations. The latter interpretation is probably more valid, because a slightly reduced uptake of glucose into muscle when insulin concentrations are markedly reduced is not inconsistent with improved insulin sensitivity.

It is clear that prior moderate exercise and a dietary energy deficit of similar magnitude did not have comparable effects on fasting and postprandial lipid metabolism. However, it is still possible that the effect of exercise was a consequence of energy deficit, but at a tissue rather than at a whole-body level. A diet-induced energy deficit shifts metabolism toward fasting. To compensate, fatty acids are released from adipose tissue, thereby reducing adipose tissue stores, and liver glycogen is converted to glucose and released into the circulation, thereby reducing liver glycogen stores (33). However, the increased energy requirements of exercise are met by muscle glycogen, intramuscular triacylglycerol (34), and, speculatively, liver triacylglycerol in addition to circulating fatty acids and liver glycogen. Increased oxidation of liver triacylglycerol might occur through increased oxidation of VLDL in skeletal muscle or increased hepatic oxidation of fatty acids during exercise. Thus, it is likely that the sites and substrates in which the energy deficit was incurred differed markedly between the exercise and energy-intake-restriction trials, and this might have mediated the different effects that the 2 interventions had on fasting and postprandial metabolism.

A high capacity to metabolize triacylglycerol, which is revealed by low postprandial lipemia, is an important determinant of elevated plasma HDL concentrations, particularly in the HDL2 subfraction (35). HDL concentrations can increase (36) or decrease (37) with diet-induced weight loss, but exercise can elevate HDL concentrations with or without a loss of body fat (38, 39). This supports the data from the present study which suggests that, over the long term, energy deficit may improve triacylglycerol metabolism but the mechanisms by which exercise enhances triacylglycerol metabolic capacity are independent of, or at least additive to, the effects of a whole-body energy deficit.

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