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Exercise prevents the augmentation of postprandial lipaemia attributable to a low-fat high-carbohydrate diet

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There is concern that replacement of dietary fat with carbohydrate may not reduce the overall risk of CHD because this replacement strategy elevates postprandial plasma triacylglycerol (TAG) concentrations. The present study was designed to test the hypothesis that daily exercise can offset the augmented postprandial lipaemia associated with a short-term high-carbohydrate diet. Nine healthy, normolipidaemic men aged 33 (SD 4) years consumed a test meal (g/kg body mass; 1·2 fat, 1·1 carbohydrate, 0·2 protein) on three occasions: after 3 d on a typical Western diet (46, 38 and 16 % energy from carbohydrate, fat and protein respectively); after 3 d on an isoenergetic high-carbohydrate diet (corresponding values: 70, 15 and 15 % energy); after 3 d on the same high-carbohydrate diet with 30 min moderate exercise daily. Fasting plasma TAG concentration was higher after the high-carbohydrate diet (1·15 (SE 0·16) mmol/l) than after the Western diet (0·83 (SE 0·10) mmol/l; $P = 0·03$). Similarly, postprandial lipaemia (6 h total area under plasma TAG concentration *v.* time curve) was higher after the high-carbohydrate diet (12·54 (SE 2·07) mmol/l·h) than after the Western diet (9·30 (SE 1·30) mmol/l·h; $P = 0·004$). The addition of exercise to the high-carbohydrate diet significantly reduced postprandial lipaemia (9·95 (SE 1·94) mmol/l·h; $P = 0·01$ when compared with the high-carbohydrate diet) but not fasting TAG concentration (1·02 (SE 0·24) mmol/l). In conclusion, daily exercise prevented the augmentation of postprandial lipaemia attributable to the short-term high-carbohydrate diet and, thus, exercise may be a powerful adjunct to dietary change.

High-carbohydrate diet: Moderate exercise: Plasma triacylglycerol: Postprandial lipaemia

There is divergence of opinion on the optimal macronutrient recommendation for protection against CHD. Whereas the reduction of dietary saturated fat forms a well-accepted tenet, the controversy focuses on which macronutrient can be the best replacement. The two main contenders are unsaturated fat and complex carbohydrates. Replacement of saturated fat with unsaturated fat has been shown to have effects on lipoprotein metabolism which would be expected to reduce the risk of CHD (Mensink & Katan, 1992). However, distrust of the strategy to replace fat with fat (Connor & Connor, 1997) has shifted the focus of recommendations to diets low in fat and high in carbohydrates (Krauss *et al.* 2000).

Replacement of dietary fat with carbohydrate has been widely recommended for CHD prevention, mainly because this replacement strategy reduces plasma total and LDL-cholesterol concentrations (Clevidence *et al.* 1992). However, low-fat high-carbohydrate diets also increase fasting plasma triacylglycerol (TAG) and decrease HDL-cholesterol concentrations (Mensink & Katan, 1992). According to epidemiological evidence, the latter alterations may be expected to increase the risk of CHD (Hokanson & Austin, 1996).

As well as increasing fasting plasma TAG concentration, high-carbohydrate diets also increase postprandial lipaemia (Jeppesen *et al.* 1997; Koutsari *et al.* 2000). This effect appears to be important for two main reasons. First, an exaggerated plasma TAG response, especially at late time points, has been recognised as an independent risk marker for CHD (Patsch *et al.* 1992). Second, the magnitude of postprandial lipaemia shows an inverse association with fasting HDL-cholesterol concentration (Patsch *et al.* 1983) and a positive association with the preponderance of small and dense LDL particles (Karpe *et al.* 1993) that are potentially atherogenic (Griffin, 1999). A high plasma TAG response probably enhances the opportunity for exchange of

Abbreviations: AUC, 6 h area under plasma or serum concentration *v.* time curve; FFA, free fatty acids; TAG, triacylglycerols.

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HDL- and LDL-cholesteryl esters for TAG from TAG-rich lipoproteins, resulting in the formation of cholesterol-depleted HDL and small dense LDL particles (Miesenböck & Patsch, 1992). Thus, one may speculate that exaggeration of postprandial lipaemia can, at least partly, explain the reduction in HDL-cholesterol concentration (Katan, 1998) and shift in LDL particle mass from predominantly larger to predominantly smaller LDL particles (Dreon *et al.* 1999) which have been reported in healthy volunteers in response to a high-carbohydrate diet.

It is, therefore, important that the effects of high-carbohydrate diets are investigated in the postprandial state, and that ways are found to introduce such diets without adversely affecting plasma TAG concentrations. An increase in physical activity may be effective in this regard. Both exercise conditioning (Weintraub *et al.* 1989) and a single exercise session (Tsetsonis *et al.* 1997) have been shown to reduce postprandial lipaemia, at least when subjects consume Western diets.

The present study was designed to test the hypothesis that daily exercise offsets the augmentation of postprandial lipaemia associated with a high-carbohydrate diet. We elected to employ a short-term intervention in order to investigate the influence of exercise on plasma TAG responses during the initial period of dietary change. This period represents a dynamic phase during which lipoproteinaemic changes are accentuated as the body responds to the change in dietary macronutrient composition (Koutsari *et al.* 2000). Exercise was of moderate intensity for 30 min daily, as recommended by the Centers for Disease Control and Prevention and the American College of Sports Medicine (Pate *et al.* 1995).

Materials and methods

Subjects

The study was approved by Loughborough University's Ethical Advisory Committee, and all subjects gave their informed consent. Nine healthy men aged 33 (SD 4) years, with BMI 24·4 (SD 1·2) kg/m^2 and maximal O_2 uptake 54 (SD 7) ml/kg per min participated. Their habitual diets were assessed by the weighed food inventory method over two weekdays and one weekend day. None were vegetarian or had fat intake contributing <25 or >45 % energy intake. Subjects' habitual diets provided 10·6 (SD 2·0) MJ/d, with 50 (SD 5) % energy as carbohydrate, 34 (SD 6) % energy as fat and 16 (SD 2) % energy as protein. As measured after an overnight fast on recruitment, whilst subjects were following their habitual lifestyle, plasma concentrations (mmol/l) of TAG, total and HDL-cholesterol were 1·10 (SD 0·47), 4·49 (SD 1·02) and 1·17 (SD 0·18) respectively. Subjects were non-smokers and none had any physician-diagnosed cardiovascular or metabolic disease or was taking drugs known to influence lipid or carbohydrate metabolism. All subjects were recreationally active in moderate-intensity exercise (e.g. fast walking, easy cycling) at least three times per week. Four of them also engaged in more strenuous exercise, again about three times per week.

Study design

Each subject consumed a standard high-fat mixed meal after three different interventions. These interventions comprised: (1) 3 d on a typical Western diet; (2) 3 d on a high-carbohydrate diet; (3) 3 d on the same high-carbohydrate diet with the addition of one 30 min session of moderate-intensity exercise each day. The order of the interventions was counterbalanced. During the 3 d leading up to the first intervention period subjects consumed a standardised prescribed diet (46, 38 and 16 % energy derived from carbohydrate, fat and protein respectively) and refrained from alcohol consumption and from exercise. There was a 10 d washout period between interventions, during the first 7 d of which subjects resumed their usual physical activity and dietary habits. During the last 3 d of this period, i.e. leading up to the next intervention, they consumed the same standardised prescribed diet as before the first intervention, again refraining from alcohol consumption and exercise. This standardisation procedure has been shown to give good reproducibility of the postprandial TAG response (Gill & Hardman, 1998).

Preliminary exercise tests

Two preliminary exercise tests were conducted. In the first, the steady-state relationship between submaximal O_2 uptake and treadmill speed was established. In the second, maximal O_2 uptake was determined during uphill running or walking at a constant speed using a modified version of the protocol devised by Taylor *et al.* (1955). The treadmill speed that elicited 60 % maximal O_2 uptake was then interpolated, on an individual basis.

Experimental diets

The typical Western diet provided 46 % energy as carbohydrate, 38 % energy as fat and 16 % energy as protein (Ministry of Agriculture, Fisheries and Food, 1997). Corresponding values (% energy) for the isoenergetic high-carbohydrate diet were 70, 15 and 15. The energy values of the diets matched, on an individual basis, the subjects' habitual energy intake. They were based on normal foods, excluding alcohol, and their energy and macronutrient contents are shown in Table 1. The sources of carbohydrates were cereals, pasta, rice, fruits, breads, vegetables, honey, jam and biscuits. The sources of fat were commercial margarines, oils, nuts, dairy products and meats. The food items were provided for the subjects, together with detailed instructions about methods of preparation and cooking. Subjects prepared three meals and one or two snacks each day, to a detailed menu, weighing each item. The importance of following the diets 'to the gram' was explained to the subjects, who recorded each item consumed.

Exercise sessions

During the high-carbohydrate diet plus exercise intervention the subjects ran or walked on the treadmill in the laboratory at 60 % maximal O_2 uptake for 30 min each afternoon.

Table 1. Composition of the typical Western and high-carbohydrate (CHO) diets

(Mean values or mean values and standard deviations for nine male subjects)

	Western diet		High-CHO diet	
	Mean	SD	Mean	SD
Energy (MJ)	10·9	1·2	10·9	1·2
Carbohydrate (% energy)	46		70	
Sugars	23		36	
Starch	23		34	
Fat (% energy)	38		15	
Saturated	16		6	
Monounsaturated	12		4	
Polyunsaturated	9		4	
Protein (% energy)	16		15	
Cholesterol (mg)	260	29	159	25
Fibre (g)	12	1	12	1

Expired air samples were collected every 15 min using Douglas bags. The samples were analysed for O_2 and CO_2 using a paramagnetic O_2 analyser and an infra-red analyser respectively (Series 1400; Servomex, Crowborough, East Sussex, UK). Expired air volumes were measured using a dry gas meter (Harvard Apparatus, Edenbridge, Kent, UK) and corrected to standard temperature and pressure (dry). O_2 uptake and CO_2 production were calculated using the Haldane transformation to calculate the volume of air inspired from direct measurements of the volume expired. Heart rate was monitored using short-range telemetry (SPORT-TESTER; Polar Electro, Kempele, Finland) and ratings of perceived exertion using the Borg (1982) scale. Only activities of daily living were permitted during the two non-exercise interventions.

Test meal protocol

Subjects arrived at the laboratory after a 12 h fast, at approximately 08.00 hours. A cannula was introduced into a forearm or antecubital vein and the subject rested quietly in a supine position for 10 min, after which a baseline blood sample was obtained. The test meal was then consumed over a median of 11 (range 9–14) min. The meal comprised cereal, coconut, nuts, chocolate, fruit and whipping cream (g/kg body mass; 1·2 fat, 1·1 carbohydrate, 0·2 protein). For these subjects this meal equated to 95 (SD 8) g fat, 86 (SD 7) g carbohydrate, 14 (SD 1) g protein and 5·14 (SD 0·14) MJ energy, 69 % of which was derived from fat. Further blood samples were obtained (with subjects in the supine position) 15, 30, 45, 60 and 90 min after completion of the meal, and then hourly until 6 h. The cannula was kept patent by flushing with non-heparinised saline (9 g NaCl/l). Expired air samples were also collected for 6 min periods in the fasted state and postprandially (every hour) using Douglas bags, for measurement of O_2 uptake and CO_2 production, as described earlier. Subjects rested or worked quietly during the observation period and consumed only water. The water was provided *ad libitum* on the first test and quantities replicated during subsequent tests.

Diet analysis

Weighed food inventories were analysed for energy and major nutrients using a computerised version (Comp-Eat 5·0; Nutrition Systems, London, UK) of UK food composition tables (Holland *et al.* 1991).

Analytical methods

Blood samples were collected into pre-cooled 9 ml potassium EDTA Monovettes (Sarstedt, Leicester, Leics., UK) and kept on ice until centrifugation within 15 min. At most time points a separate sample was collected into a 4·5 ml plain Monovette for serum preparation. Portions of plasma and serum were stored at −20°C until analysed for plasma total cholesterol, HDL-cholesterol (fasted and 6 h samples only), glucose, lactate (Roche Diagnostics Ltd, Lewes, West Sussex, UK), free fatty acids (FFA; Wako Chemicals GmbH, Neuss, Germany; and serum 3-hydroxybutyrate (Sigma Diagnostics, Poole, Dorset, UK) by enzymic colorimetric methods using an automated analyser (Cobas Mira Plus; Roche Diagnostic Systems, Welwyn Garden City, Herts., UK). Using the same analyser, the concentrations of TAG in plasma were measured enzymically, with correction for free glycerol (Humphreys *et al.* 1990). Serum was stored at −70°C until analysed for insulin using a solid-phase ^{125}I radioimmunassay (Coat-A-Count Insulin; Diagnostic Products Corporation, Los Angeles, CA, USA). Radioactivity was measured using an automated gamma counting system (Cobra II; Packard Instrument Company Inc., Downers Grove, IL, USA). All samples from each subject were analysed in the same batch. Accuracy and precision were maintained using quality-control sera (Roche Diagnostics Ltd). Within-batch CV (%) were 0·7 for cholesterol, 1·8 for HDL-cholesterol, 1·2 for TAG, 0·8 for glucose, 1·3 for lactate, 0·9 for FFA, 1·4 for 3-hydroxybutyrate and 4·2 for insulin.

Calculations and statistics

Plasma and serum concentrations measured in the fasted state were compared using one-way ANOVA for repeated measures. Differences among interventions in postprandial concentrations over time were compared using two-way ANOVA for repeated measures to investigate interactions. In addition, summary measures (Matthews *et al.* 1990) of the postprandial responses were calculated as the total areas under plasma or serum concentration *v.* time curves (AUC) using the trapezoidal rule. Incremental AUC were also calculated for plasma TAG responses as the total AUC minus the fasting value extrapolated over 6 h. Summary measures were compared among interventions using one-way ANOVA for repeated measures. Data were checked for normality before statistical analyses were performed. When data were not normally distributed (plasma TAG concentrations and total AUC for TAG), they were transformed to their natural logarithm. Normal distribution of transformed values was confirmed before statistical testing. If significant differences were identified by one-way ANOVA, Tukey *post hoc* comparisons were performed to determine where these differences occurred. Whole-body carbohydrate and

fat utilisation rates and energy expenditure were calculated using indirect calorimetry, without measurement of urinary N excretion (Frayn, 1983). LDL-cholesterol concentration in the fasted state was estimated using the Friedewald formula (Friedewald *et al.* 1972). $P < 0{\cdot}05$ was adopted as significant throughout. Statistical procedures were performed using Statistica for Windows, version 5.0 (Statistica, Tulsa, OK, USA).

Results

Experimental diets and exercise sessions

Both experimental diets were well tolerated, aside from a common complaint by the subjects of feeling excessively full during the high-carbohydrate diet. Compliance, assessed by food inventories and detailed discussions with subjects, was high.

During the high-carbohydrate diet plus exercise intervention, eight subjects ran on the flat at 2·6 (SD 0·4) m/s and one subject walked at 2·1 m/s at 4 % uphill gradient. The average O_2 uptake during exercise for the 3 d was 33·0 (SD 5·0) ml/kg per min and represented 61 (SD 3) % of maximal O_2 uptake. Average values for heart rate and RER were 145 (SD 19) beat/min and 0·94 (SD 0·03) respectively. Ratings of perceived exertion were 12 (SD 1) corresponding to the description 'fairly hard'. Gross energy expenditure per session was 1·58 (SD 0·36) MJ, 81 (SD 10) % from carbohydrate and 19 (SD 10) % from fat. On average, 60 (SD 35) g carbohydrate and 7 (SD 4) g fat were oxidised daily during exercise.

Plasma and serum concentrations in the fasted state

Fasting plasma concentrations of TAG, total, HDL- and LDL-cholesterol, FFA, glucose, lactate and serum concentrations of insulin and 3-hydroxybutyrate are presented in Table 2. Triacylglycerol concentration was significantly higher after the high-carbohydrate diet than after the Western diet ($P = 0{\cdot}03$). The addition of exercise to the high-carbohydrate diet did not significantly reduce TAG concentration ($P = 0{\cdot}19$ when compared with the high-carbohydrate diet). LDL-cholesterol concentrations were significantly lower after both the high-carbohydrate and the high-carbohydrate plus exercise interventions than after the Western diet ($P < 0{\cdot}05$ in both cases). There were no significant differences among interventions in any of the other variables.

Postprandial responses

Total and incremental plasma TAG concentrations after the test meal are shown in Fig. 1, with summary measures (AUC) of these responses in Table 3. The total AUC was 35 % greater after the high-carbohydrate diet than after the Western diet ($P < 0{\cdot}01$). The addition of daily exercise to the high-carbohydrate diet significantly reduced this response ($P = 0{\cdot}01$ when compared with the high-carbohydrate diet), almost to the level observed after the Western diet. The pattern of change over time in total TAG concentration differed among interventions (interaction of intervention×time, $P < 0{\cdot}01$). Peak concentration occurred later after the high-carbohydrate diet, both with and without exercise (5 (SE 1) h for both interventions), than after the Western diet (3 (SE 1) h; $P < 0{\cdot}01$). The incremental AUC did not differ between the diet-only interventions, but the addition of daily exercise to the high-carbohydrate diet significantly reduced the incremental AUC compared with the high-CHO diet alone ($P < 0{\cdot}05$). The pattern of change over time in incremental TAG concentrations differed among interventions (interaction of intervention×time, $P < 0{\cdot}01$).

Postprandial concentrations of insulin, FFA, 3-hydroxybutyrate, glucose and lactate are shown in Fig. 2 and summary measures (total AUC) are presented in Table 3. None of the AUC differed among interventions, except 3-hydroxybutyrate. The AUC for 3-hydroxybutyrate was significantly lower after the high-carbohydrate diet than after the Western diet ($P = 0{\cdot}03$), with a much smaller rise between 2 and 6 h postprandially (interaction of intervention×time, $P < 0{\cdot}01$). The addition of exercise to the high-carbohydrate diet did not have a significant effect on

Table 2. Plasma or serum concentrations (mmol/l) measured in the fasted state for healthy men after 3 d on a Western diet, a high-carbohydrate (CHO) diet or a high-CHO diet+30 min daily exercise (Ex)†

(Mean values with their standard errors for nine subjects)

	Western diet		High-CHO diet		High-CHO diet+Ex		Statistical significance of differences among interventions (one-way ANOVA): *P*
	Mean	SE	Mean	SE	Mean	SE	
Triacylglycerol	0·83	0·10	1·15*	0·16	1·02	0·24	0·03
Total cholesterol	4·15	0·27	4·04	0·27	3·95	0·26	0·08
HDL-cholesterol	1·05	0·08	0·95	0·04	0·98	0·06	0·28
LDL-cholesterol‡	2·75	0·25	2·56*	0·2	2·54*	0·25	0·01
Free fatty acids	0·26	0·04	0·24	0·05	0·31	0·04	0·37
3-Hydroxybutyrate	0·04	0·01	0·04	0·01	0·04	0·01	0·86
Insulin (μl/ml)	8·1	0·8	8·4	1·3	7·7	0·90	0·74
Glucose	5·35	0·17	5·29	0·16	5·22	0·13	0·17
Lactate	0·76	0·07	0·91	0·10	0·81	0·06	0·17

Mean values were significantly different from those for the Western diet (Tukey *post hoc* test): * $P < 0{\cdot}05$.
† For details of subjects, diets and procedures, see Table 1 and p. 199.
‡ Estimated using the Friedewald formula (Friedewald *et al.* 1972).

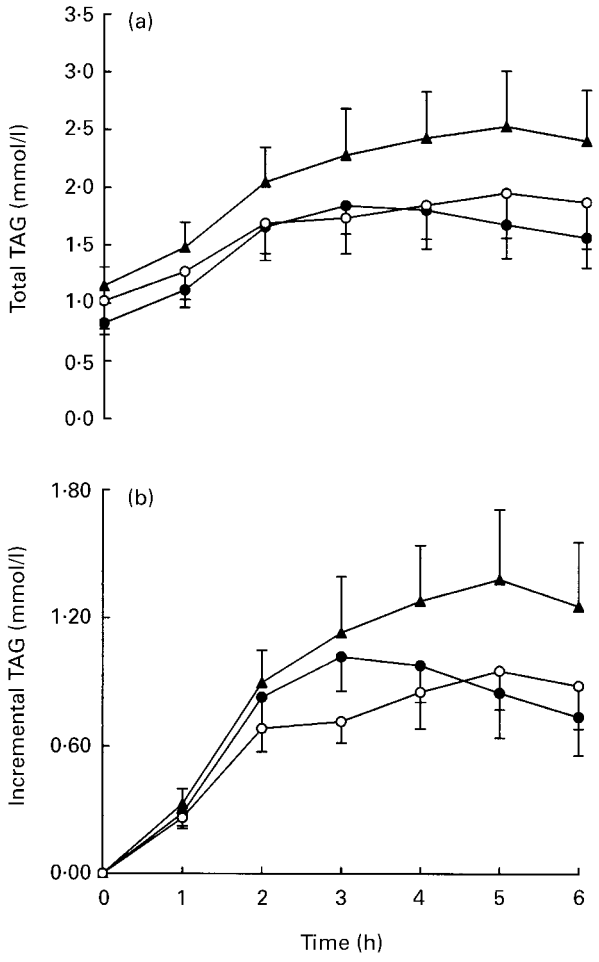

Fig. 1. Total (a) and incremental (b) plasma triacylglycerol (TAG) concentrations in the fasted state (0 h) and for 6 h following consumption of a high-fat mixed meal after 3 d on a Western diet (●), after 3 d on a high-carbohydrate (CHO) diet (▲) and after 3 d on the same high-CHO diet with 30 min daily of moderate exercise (○). For details of subjects, diets and procedures, see Table 1 and p. 199. Values are means with their standard errors represented by vertical bars for nine subjects. Statistical analyses based on areas under concentration *v.* time curves are given in Table 3.

the AUC for 3-hydroxybutyrate. The change in plasma concentrations of glucose over time differed among interventions (interaction of intervention×time, $P < 0{\cdot}01$). Neither total nor HDL-cholesterol changed over the postprandial period for any of the interventions.

Indirect calorimetry

Fasting and postprandial RER values are presented in Fig. 2. Neither the high-carbohydrate diet, nor the high-carbohydrate diet plus exercise, significantly affected fasting or postprandial energy expenditure or substrate oxidation.

Discussion

The present study compared the effects of a Western diet and an isoenergetic low-fat high-carbohydrate diet on postprandial TAG responses to a standard test meal. More importantly, it also investigated whether participation in daily moderate-intensity exercise can oppose the augmentation of postprandial lipaemia attributable to a high-carbohydrate diet.

In agreement with other reports (Jeppesen *et al.* 1997), the high-carbohydrate diet exaggerated postprandial lipaemia compared with the Western diet. Indeed, our findings probably underestimate this effect because, after the high-carbohydrate diet, TAG concentrations were still very high at the end of our 6 h observation period. However, in line with our hypothesis, this carbohydrate-induced increase in lipaemia was negated by daily moderate-intensity exercise for 30 min. Previous studies have shown that such exercise reduces postprandial lipaemia (Weintraub *et al.* 1989; Tsetsonis *et al.* 1997) but, without exception, in these studies the subjects were on Western diets. To the best of our knowledge, the present data are the first to demonstrate that the TAG-lowering effect of moderate exercise is sufficiently potent to offset the augmentation of postprandial lipaemia which develops when subjects change from a Western diet to a low-fat high-carbohydrate diet. Interestingly, we did not observe any significant effect on fasting

Table 3. The 6 h areas under the plasma or serum concentration *v.* time curves (AUC; mmol/l·h) after consumption of a high-fat mixed meal after 3 d on a Western diet, a high-carbohydrate (CHO) diet or a high-CHO diet+30 min daily exercise (Ex)‡

(Mean values with their standard errors for nine subjects)

	Western diet		High-CHO diet		High-CHO diet+Ex		Statistical significance of differences among interventions (one-way ANOVA): *P*
	Mean	SE	Mean	SE	Mean	SE	
Total AUC for triacylglycerol	9·30	1·30	12·54**	2·07	9·95†	1·94	0·004
Incremental AUC for triacylglycerol§	4·33	0·77	5·65	1·18	3·84†	0·59	0·048
Free fatty acids	2·07	0·18	1·74	0·17	1·97	0·15	0·20
3-Hydroxybutyrate	0·42	0·07	0·26*	0·07	0·31	0·07	0·004
Insulin (μl/ml·h)	126	15	135	16	116	16	0·18
Glucose	34·1	0·9	33·2	1·0	33·4	1·1	0·28
Lactate	5·51	0·26	5·78	0·45	5·70	0·44	0·67

Mean values were significantly different from those for the Western diet (Tukey *post hoc* test): * $P < 0{\cdot}05$, ** $P < 0{\cdot}01$.
Mean values were significantly different from those for high-CHO diet (Tukey *post hoc* test): † $P < 0{\cdot}05$.
‡ For details of subjects, diets and procedures, see Table 1 and p. 199.
§ Total AUC minus fasting value extrapolated over 6 h.

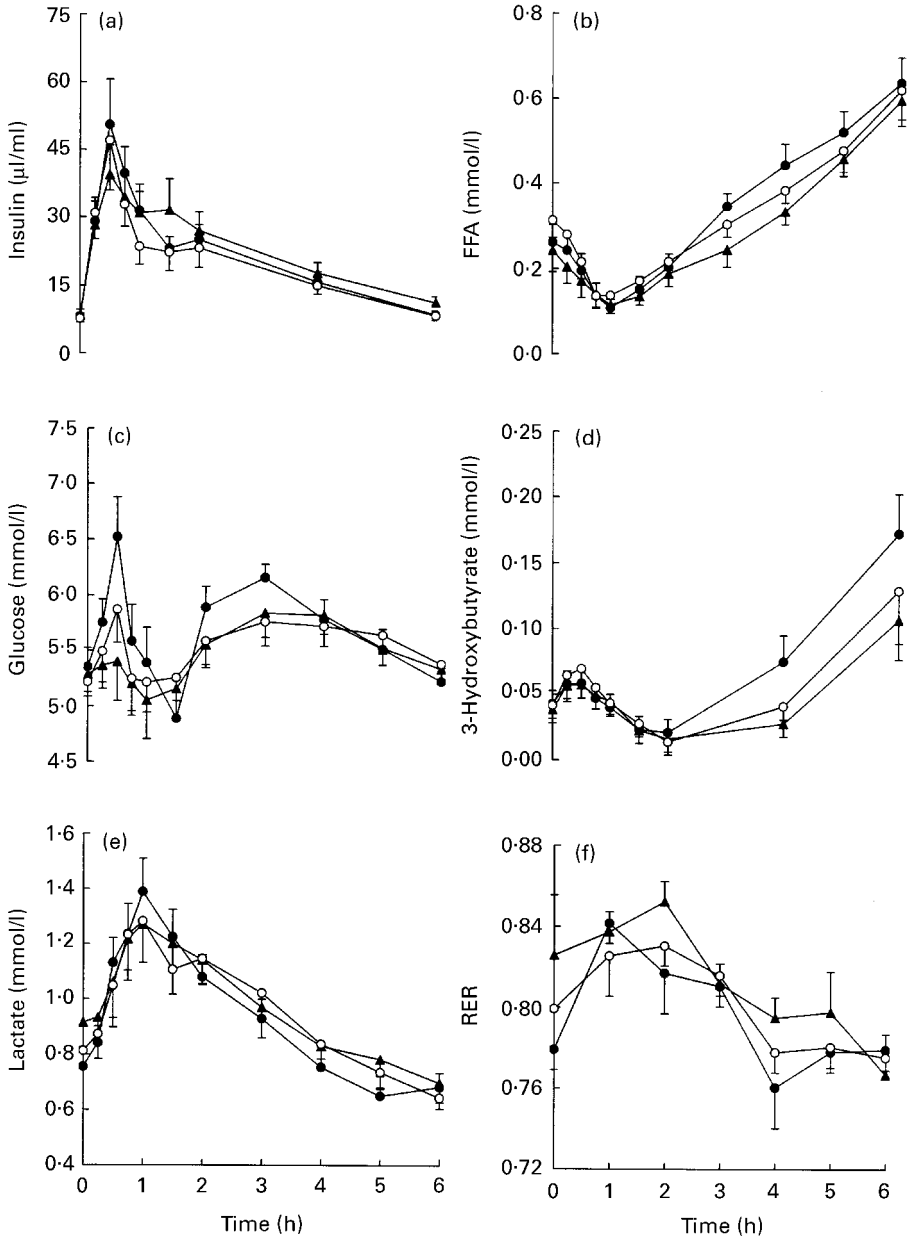

Fig. 2. Plasma insulin (a) free fatty acids (FFA; b), glucose (c), 3-hydroxybutyrate concentrations (d) and lactate (e), and RER values (f) in the fasted state (0 h) and for 6 h following consumption of a high-fat, mixed meal after 3 d on a Western diet (●), after 3 d on a high-carbohydrate (CHO) diet (▲) and after 3 d on the same high-CHO diet with 30 min daily of moderate exercise (○). For details of subjects, diets and procedures, see Table 1 and p. 199. Values are means with their standard errors represented by vertical bars for nine subjects. Statistical analyses based on areas under concentration *v.* time curves are given in Table 3.

TAG concentrations of adding exercise to the high-carbohydrate diet. By contrast, when the TAG metabolic capacity was challenged in the postprandial state the normalising effect of exercise was clearly revealed, showing the importance of studying human subjects in the fed state. Another interesting observation was that adding daily exercise to the high-carbohydrate diet reduced postprandial TAG concentrations, but did not lead to an earlier time to reach peak concentration (Fig. 1).

The higher plasma TAG concentrations observed in the fasted state probably contributed to the carbohydrate-induced exaggeration in postprandial lipaemia (Björkegren *et al.* 1996). High-carbohydrate diets most probably accelerate VLDL-TAG secretion by suppressing hepatic oxidation of FFA, presumably diverting a greater proportion of the FFA towards hepatic TAG synthesis (Sidossis & Mittendorfer, 1999). In the present study the lower postprandial 3-hydroxybutyrate response after the high-carbohydrate diet than after the Western diet (Table 3 and Fig. 2) may indicate a suppressive effect of increased dietary carbohydrate on hepatic fatty acid oxidation. Postprandial lipaemia might also be augmented after the high-carbohydrate diet through reduced hydrolysis of circulating TAG by lipoprotein lipase. At a whole-body level, both a

reduction in clearance of VLDL-TAG (Parks *et al.* 1999) and lower plasma heparin-releasable lipoprotein lipase activity (Campos *et al.* 1995) have been reported after high-carbohydrate diets. These alterations would lead to increases in plasma TAG concentrations, especially in the postprandial state when the number of circulating TAG-rich lipoproteins increases dramatically.

The addition of exercise to the high-carbohydrate diet prevented the exaggeration in postprandial lipaemia observed with the high-carbohydrate diet alone. Earlier studies showed that regular exercise ameliorated increases in fasting TAG concentrations due to high-carbohydrate diets (Ullrich & Albrink, 1986; Thompson *et al.* 1988). Although we did not find significant effects of exercise in the fasted state, it may be that the moderate reductions in fasting TAG concentrations observed in the majority of subjects contributed to lower responses during the postprandial state. Exercise could have ameliorated the carbohydrate-induced augmentation of postprandial lipaemia by affecting VLDL-TAG production. As mentioned earlier, high-carbohydrate diets increase VLDL-TAG secretion by inhibiting hepatic FFA oxidation (Sidossis & Mittendorfer, 1999). By contrast, studies in rats have shown that exercise training decreases VLDL-TAG production, even during long-term carbohydrate feeding (Zavaroni *et al.* 1981). One explanation may be that exercise training alters the hepatic partitioning of fatty acids between esterification and oxidation (Fukuda *et al.* 1991). In line with this thinking, the reduction in the postprandial 3-hydroxybutyrate response by the high-carbohydrate diet was attenuated by daily exercise, although this difference was not statistically significant (Table 3 and Fig. 2). Exercise could have also opposed the suppressive effects of high-carbohydrate diets on lipoprotein lipase activity (Campos *et al.* 1995). Both a single exercise session (Annuzzi *et al.* 1987; Kantor *et al.* 1987) and exercise training (Thompson *et al.* 1988) increase plasma heparin-releasable lipoprotein lipase activity and the capacity for intravenous fat clearance. These exercise-induced changes may have affected postprandial TAG metabolism when exercise was combined with the high-carbohydrate diet.

Insulin can affect postprandial lipaemia due to direct effects on VLDL-TAG secretion (Lewis *et al.* 1995), as well as through effects on lipoprotein lipase activity (Farese *et al.* 1991). However, the serum insulin response to the test meal did not differ among interventions (Table 3 and Fig. 2), so that these effects may not have been responsible for the differences we observed in postprandial TAG responses (unless there were important changes in insulin sensitivity). As expected (Mensink & Katan, 1992), the high-carbohydrate diet reduced LDL-cholesterol concentration, although the addition of exercise did not reduce this concentration further. Interestingly, the interventions we employed did not alter HDL-cholesterol significantly, despite the fact that both high-carbohydrate diets (Katan, 1998) and exercise (Durstine & Haskell, 1994) have been reported to influence this variable. It has been suggested that HDL-cholesterol concentration is strongly determined by TAG metabolic capacity (Miesenböck & Patsch, 1992). It may be, therefore, that the initial effect of high-carbohydrate diets or exercise is to alter postprandial TAG metabolism, and that changes in HDL-cholesterol level develop later.

In the present study we employed an isoenergetic high-carbohydrate diet in order to investigate specifically the effect of altering diet composition on postprandial lipaemia. It should be noted that under *ad libitum* food intake conditions, carbohydrate-induced hypertriacylglycerolaemia is often avoided by lower energy intake and associated weight loss (Kasim-Karakas *et al.* 2000). Under these conditions, simultaneous daily exercise would probably have an even greater impact on postprandial TAG metabolism than that observed in the present study. Our model cannot show whether the prevention of carbohydrate-induced augmentation of postprandial lipaemia was due to the effect of the last session of exercise *per se* or to the accumulated effects of the three exercise sessions, or both. However, the first possibility seems unlikely because a single exercise session of longer duration and greater energy expenditure did not significantly decrease postprandial lipaemia (Tsetsonis & Hardman, 1996).

A case has been made for replacing saturated fat in the diet with unsaturated fatty acids as a strategy to reduce the risk of CHD (Mensink & Katan, 1992; Kris-Etherton, 1999). This strategy would reduce LDL-cholesterol without the adverse effects on TAG and HDL-cholesterol concentrations seen when fat is replaced with carbohydrate. Our findings show that a further attractive alternative may be the adoption of a physically-active lifestyle alongside a low-fat high-carbohydrate diet. This strategy might be particularly effective when the diet comprises predominantly complex carbohydrate, as this form of carbohydrate does not detrimentally affect postprandial lipaemia (Rivellese *et al.* 1994). However, we employed a short-term intervention model and so have no evidence on the long-term efficacy of exercise in this regard, a topic which justifies further investigation.

In conclusion, daily exercise prevented the augmentation of postprandial lipaemia resulting from the short-term consumption of a high-carbohydrate diet. Moreover, we employed a diet in which 70 % energy came from carbohydrate, a more extreme dietary change than that recommended for the population at large. Nonetheless, a modest amount of exercise, attainable by most individuals was sufficient to negate carbohydrate-induced hypertriacylglycerolaemia. As this process is often considered one of the most deleterious effects of high-carbohydrate diets, our findings show that exercise may be a powerful adjunct to dietary change.

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