Postprandial Lipemia After Short-Term Variation in Dietary Fat and Carbohydrate

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Replacement of dietary fat with carbohydrate may not reduce the overall risk of coronary heart disease (CHD), because this elevates plasma triacylglycerol (TAG) concentrations. The lipoproteinemic effects of a high-carbohydrate diet are likely to be more marked shortly after the initiation of such a diet than after longer periods of intervention during which adaptive processes may counteract the initial effects. Therefore, we studied the postprandial responses to a standard meal after 3-day dietary intervention periods. An additional objective was to establish a model for future study of the mechanisms involved. Nine normolipidemic men consumed the meal (1.2 g fat, 1.1 g carbohydrate, and 0.2 g protein per 1 kg body mass) after 3 days on a high-carbohydrate diet (68% ± 3% energy from carbohydrate, mean ± SD) and also after 3 days on an isoenergetic high-fat diet (66% ± 5% energy). Venous blood samples were obtained from fasted subjects and for 6 hours after the meal. In the fasted state, TAG was higher after the high-carbohydrate diet (1.18 ± 0.18 *v* 0.62 ± 0.09 mmol/L, mean ± SEM, $P = .02$) and high-density lipoprotein (HDL) cholesterol was lower (1.01 ± 0.08 *v* 1.10 ± 0.09 mmol/L, $P = .002$). The area under the plasma TAG concentration versus time curve was 42% ± 7% higher after the high-carbohydrate diet ($P = .003$). After the high-carbohydrate diet, the postprandial insulin response did not differ between trials, but glucose and 3-hydroxybutyrate responses were lower ($P = .009$ and $P = .02$, respectively) and the lactate response was higher ($P = .001$). Plasma nonesterified fatty acids (NEFAs) were lower after the high-carbohydrate diet in the fasted state and for 4 hours postprandially, but were higher thereafter (interaction of time × trial, $P = .001$). These results indicate that compared with a high-fat diet, the plasma TAG response to a standard high-fat meal is markedly higher after a few days on a high-carbohydrate diet, with major differences in the associated metabolic milieu. The magnitude of these changes and the rapidity with which they developed suggest that this model may be attractive for future studies of the underlying mechanisms.

A REDUCTION in the intake of dietary fat has been widely recommended as one means to prevent or treat coronary heart disease (CHD). In the United Kingdom, for example, a desirable level likely to decrease the incidence of diet-related chronic disease has been set at 33% of energy from fat, a reduction of nearly 7% compared with the figures for 1996.[1] Most recommendations have specified that the fat eliminated from the diet be replaced by carbohydrates so that energy intake is maintained at approximately the same level. Recently, however, there has been concern that low-fat, high-carbohydrate diets may not confer the anticipated benefits for cardiovascular disease risk[2] because such diets increase plasma concentrations of triacylglycerol (TAG) and reduce high-density lipoprotein (HDL) cholesterol.[3] Based on population studies, both changes would be expected to increase—rather than decrease—the risk of CHD,[4] potentially offsetting any benefit from associated decreases in low-density lipoprotein cholesterol.

Consequently, it is important that the links between dietary change and lipoprotein metabolism are understood and their implications for the atherosclerotic process are explored. Such investigations are most appropriately conducted in the postprandial state for 2 reasons: first, because this state is particularly critical and dynamic,[5] and second, because of accumulating evidence that high postprandial TAG concentrations are a strong and independent predictor of early atherosclerosis[6] and coronary artery disease.[7]

Studies in healthy women[8] and in patients with non–insulin-dependent diabetes mellitus[9] have shown that postprandial lipemia may be increased, rather than decreased, during the consumption of a high-carbohydrate diet. However, in both of these studies, the test meals were not standard but reflected the composition of the background diets that were compared and therefore contained a different amount of carbohydrate and fat. Since the addition of carbohydrate to a fixed fat load significantly accentuates the degree of postprandial lipemia,[10] these studies do not distinguish the acute effects on postprandial lipemia of a single test meal from the chronic effects of the preceding diet.

It has been suggested that carbohydrate-induced hypertriacylglycerolemia is a short-term adaptive response to an increase in dietary carbohydrate and that this effect diminishes after a couple of weeks.[11,12] On the other hand, results from experimental[13] and epidemiologic[14] studies show that this can be a long-lasting phenomenon. Either way, the lipoproteinemic changes are likely to be more marked shortly after the initiation of a high-carbohydrate diet than after longer periods of intervention during which adaptive processes may counterbalance the initial effects. The mechanisms underlying carbohydrate-induced hypertriacylglycerolemia may thus be best revealed through the study of the initial responses to a switch to a carbohydrate-rich diet.

The present study was therefore designed to investigate the effects of short-term manipulation of the fat and carbohydrate content of isoenergetic diets on the metabolic responses to a standard test meal. An additional objective was to establish a short-term dietary intervention model for future investigations of the hypertriacylglycerolemic effect of a carbohydrate-rich diet and, importantly, its prevention. Diets with distinctly

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different composition were studied to provide a clear picture of differences in postprandial events.

SUBJECTS AND METHODS

Subjects

The study was approved by the University's Ethical Advisory Committee, and all subjects provided informed consent. Nine normolipidemic men aged 26.7 ± 3.8 years (mean ± SD) with a body mass index of 25.1 ± 1.8 kg/m^2 participated. To exclude individuals with extreme dietary practices, only subjects with a fat intake constituting between 25% and 45% of energy intake (assessed by the weighed-food inventory method over 2 weekdays and 1 weekend day) were accepted. Analysis of these inventories showed that the subjects' normal diet provided 11.7 ± 1.7 MJ/d, with 35% ± 9% of energy as fat (13% ± 5% as saturated, 13% ± 4% as monounsaturated, and 8% ± 4% as polyunsaturated), 50% ± 9% as carbohydrate (20% ± 6% as simple and 30% ± 9% as complex), and 15% ± 3% as protein. As measured after an overnight fast upon recruitment while subjects were following their habitual life-style, plasma concentrations of total cholesterol, HDL cholesterol, and TAG were 4.18 ± 0.94, 1.08 ± 0.19, and 0.93 ± 0.29 mmol/L, respectively. All subjects were nonsmokers and none were vegetarian. None had any physician-diagnosed cardiovascular or metabolic disease or were using medications known to influence lipid or carbohydrate metabolism. Seven subjects were recreationally active in moderate exercise (eg, fast walking or easy cycling) 3 times per week. Two also engaged in more strenuous exercise, again about 3 times per week. Five subjects possessed the apolipoprotein E3/3 phenotype, 2 E3/2, and 2 E4/3.

Study Design and Experimental Diets

Each subject undertook 2 oral fat tolerance tests with an interval of 2 weeks. For 3 days before each test, they followed 1 of 2 prescribed diets in a randomized balanced design. The diets were isoenergetic and their energy value approximated, on an individual basis, the subjects' previously determined energy intake. One diet was high in carbohydrate and the other was high in fat; protein intakes were similar. The diets were based on normal foods, excluding alcohol, and their energy and macronutrient contents are shown in Table 1. The sources of carbohydrate were fruits, vegetables, breads, cereals, pasta, honey, jam, and biscuits. The sources of fat were commercial margarines, oils, nuts, avocado pears, dairy products, and meats. Three meals and two snacks were consumed each day. Subjects prepared these themselves, from a menu agreed upon with the experimenters, and weighed each item. During the 3 days before their first experimental diet, the diet was unrestricted but subjects weighed and recorded all foods consumed. This food intake was then replicated during the 3 days preceding the second experimental diet. Subjects refrained from exercise for 6 days prior to each oral fat tolerance test, ie, during the 3-day diet intervention period and for 3 days beforehand. They also refrained from alcohol intake on all 6 days.

Table 1. Composition of the Experimental Diets

Component	High-Carbohydrate Diet	High-Fat Diet
Energy (MJ)	12.2 ± 2.1	11.9 ± 2.1
Carbohydrate (%)	68 ± 3	18 ± 4
Simple	35 ± 5	6 ± 2
Complex	33 ± 4	12 ± 2
Fat (%)	18 ± 3	66 ± 5
Saturated	8 ± 1	24 ± 3
Monounsaturated	6 ± 1	22 ± 2
Polyunsaturated	3 ± 1	17 ± 2
Cholesterol (mg)	200 ± 89	352 ± 74
Protein (%)	14 ± 1	16 ± 3
Fiber (g)	22 ± 8	18 ± 5

NOTE. Results are the mean ± SD (n = 9 men). Each diet was consumed for 3 days.

Oral Fat Tolerance Tests

Subjects arrived at the laboratory after a 12-hour fast at approximately 8 AM. A cannula was introduced into a forearm or antecubital vein and the subject rested quietly in the supine position for 10 minutes, after which a baseline blood sample was obtained. The test meal was then consumed over a mean of 11.4 minutes (both trials). The meal consisted of cereal, coconut, nuts, chocolate, fruit, and whipping cream and was provided according to body mass (per 1 kg body mass: 1.2 g fat, 1.1 g carbohydrate, and 0.2 g protein). For these subjects, this meant 96 ± 3 g (mean ± SD) fat, 93 ± 3 g carbohydrate, 14 ± 1 g protein, and 5.54 ± 0.19 MJ energy, 69% of which originated from fat. Further blood samples were obtained (with subjects in the supine position) 15, 30, 45, 60, and 90 minutes after completion of the meal, and then hourly until 6 hours. The cannula was kept patent by flushing with nonheparinized saline (9 g/L). Expired-air samples were collected for 6-minute periods immediately before each postprandial blood sample using Douglas bag techniques for measurement of oxygen uptake and carbon dioxide production. Subjects rested or worked quietly during the observation period and consumed only water. This was provided ad libitum on the first test and replicated during the second test.

Diet Analysis

Weighed-food inventories were analyzed for energy and major nutrients using a computerized version (Comp-Eat 5.0; Nutrition Systems, London, UK) of UK food composition tables.[15]

Analytical Methods

Blood samples were collected into precooled Potassium-EDTA Monovettes (Sarstedt, Leicester, UK) and kept on ice until centrifugation within 15 minutes. At most time points, a separate sample was collected into a plain Monovette for serum preparation. Portions of plasma and serum were stored at −20°C until analysis for total cholesterol, HDL cholesterol (fasted and 6-hour samples only), TAG, glucose, lactate (Boehringer Mannheim, Lewes, UK), nonesterified fatty acids ([NEFAs] Wako, Neuss, Germany) (all plasma), and serum 3-hydroxybutyrate (Sigma Diagnostics, St Louis, MO) by enzymatic colorimetric methods using an automated analyzer (Cobas Mira Plus; Roche, Basel, Switzerland). Serum was stored at −70°C until analysis for insulin using a solid-phase ^{125}I radioimmunoassay (Coat-A-Count Insulin; Diagnostic Products, Los Angeles, CA). Radioactivity was measured using an automated gamma counting system (Cobra II; Packard Instrument, Downers Grove, IL). All samples from any one subject were analyzed in the same batch. Accuracy and precision were maintained using quality-control sera (Boehringer Mannheim and Roche). The within-batch coefficient of variation was 0.7% for cholesterol, 1.8% for HDL cholesterol, 1.2% for TAG, 0.8% for glucose, 1.3% for lactate, 0.9% for NEFA, 1.4% for 3-hydroxybutyrate, and 4.2% for insulin. Phenotypes of apolipoprotein E were determined by isoelectric focusing using Western blot techniques. The hemoglobin concentration and hematocrit were measured for the estimation of differences in plasma volume between tests.[16]

Calculations and Statistics

Plasma and serum concentrations measured in the fasted state were compared using Student's *t* test for correlated means. Differences in postprandial responses between trials were compared using repeated-measures ANOVA to investigate interactions between trials and time. The significance of differences between cell means was evaluated using the Tukey post hoc test. In addition, summary measures of the

postprandial responses were derived.[17] Total postprandial responses were calculated as the area under the plasma or serum concentration versus time curve using the trapezoidal rule. The incremental postprandial TAG response was calculated as the total area under the plasma concentration versus time curve, minus the fasting value extrapolated over 6 hours. Summary measures were compared between trials using Student's *t* test for correlated data. Relationships were evaluated using Pearson's product moment correlation coefficient. Data were checked for normality before the analyses were performed. A 5% level of significance was used throughout, and the results are expressed as the mean ± SEM, unless otherwise stated. Statistical procedures were performed using Statistica for Windows 1995, version 5.0 (StatSoft, Tulsa, OK).

RESULTS

Diet Interventions

Both experimental diets were well tolerated and compliance, assessed by food inventories and detailed discussions with the subjects, was good. Plasma volume differences between trials were small (relative to high-carbohydrate trial, −0.1% ± 0.6%, mean ± SD), so the concentrations are presented without correction.

Plasma and Serum Concentrations in the Fasted State

Plasma TAG, plasma lactate, and serum insulin were higher after the high-carbohydrate diet, whereas HDL cholesterol and NEFA were significantly lower (Table 2). No significant differences were found for glucose or total cholesterol. The concentration of 3-hydroxybutyrate was lower after the high-carbohydrate diet ($P = .06$).

Postprandial Responses

The total lipemic response was higher after the high-carbohydrate diet versus the high-fat diet, and the pattern of change in TAG concentration differed between trials (interaction of trial × time, $P < .001$). The peak concentration occurred later after the high-carbohydrate diet (3 to 4 hours postprandially, as compared with 1 to 2 hours), and at 6 hours, it was still significantly higher than the fasting value ($P < .001$). The total lipemic response was closely related to the fasting TAG concentration in the high-carbohydrate trial ($r = .97$, $P < .01$) but not in the high-fat trial ($r = .61$, NS). The incremental lipemic response was higher after the high-carbohydrate diet, and the pattern of change over time also differed between trials (interaction of time × trial, $P < .001$) (Fig 1).

Table 2. Fasting Concentrations of Plasma Total and HDL Cholesterol, TAG, NEFA, 3-Hydroxybutyrate, Insulin, Glucose, and Lactate After High-Carbohydrate and High-Fat Diets

Parameter	High-Carbohydrate Diet	High-Fat Diet	P*
Total cholesterol (mmol/L)	3.98 ± 0.25	3.81 ± 0.25	.14
HDL cholesterol (mmol/L)	1.01 ± 0.08	1.10 ± 0.09	.002
TAG (mmol/L)	1.18 ± 0.18	0.62 ± 0.09	.02
NEFA (mmol/L)	0.21 ± 0.03	0.30 ± 0.04	.003
3-Hydroxybutyrate (mmol/L)	0.02 ± 0.01	0.07 ± 0.02	.06
Insulin (μIU/mL)	10.9 ± 0.8	9.2 ± 0.6	.03
Glucose (mmol/L)	5.13 ± 0.13	5.26 ± 0.13	.15
Lactate (mmol/L)	1.08 ± 0.09	0.73 ± 0.03	.004

NOTE. Results are the mean ± SEM (n = 9 men). Each diet was consumed for 3 days.

*Significance of differences between trials by Student's *t* test for correlated means.

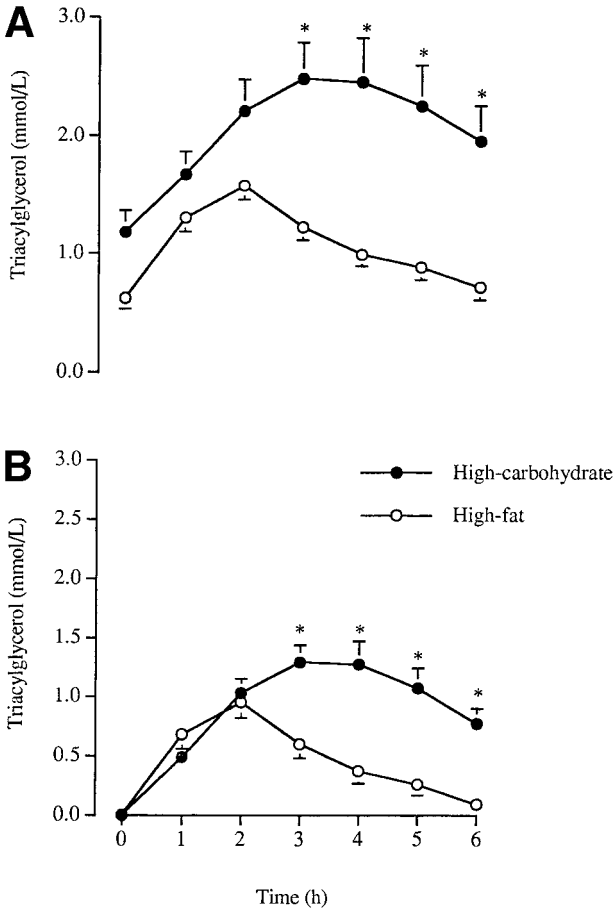

Fig 1. (A) Total and (B) incremental plasma concentrations of TAG during oral fat tolerance tests after 3 days on a high-carbohydrate diet and after 3 days on a high-fat diet (mean ± SEM; n = 9 men). *Significantly different *v* corresponding value in the other trial, $P < .05$.

The postprandial responses of insulin, NEFA, 3-hydroxybutyrate, glucose, and lactate are shown in Fig 2 and the summary measures of these responses in Table 3. The serum insulin response did not differ between trials, nor did the change in concentration over time. The plasma glucose response was lower ($P = .009$) after the high-carbohydrate diet, and the plasma lactate response was higher ($P = .001$). The change in plasma glucose and lactate over time did not differ between trials. The pattern of change in NEFA over time was clearly different (interaction of trial × time, $P < .001$); the concentration was lower after the high-carbohydrate diet in the fasted state and for 4 hours postprandially, but higher thereafter. The 3-hydroxybutyrate response to the high-fat meal was significantly lower ($P = .02$) after the high-carbohydrate diet, with a much smaller increase between 2 and 6 hours, versus the high-fat diet (interaction of trial × time, $P < .001$). The mean postprandial value for the respiratory exchange ratio was higher

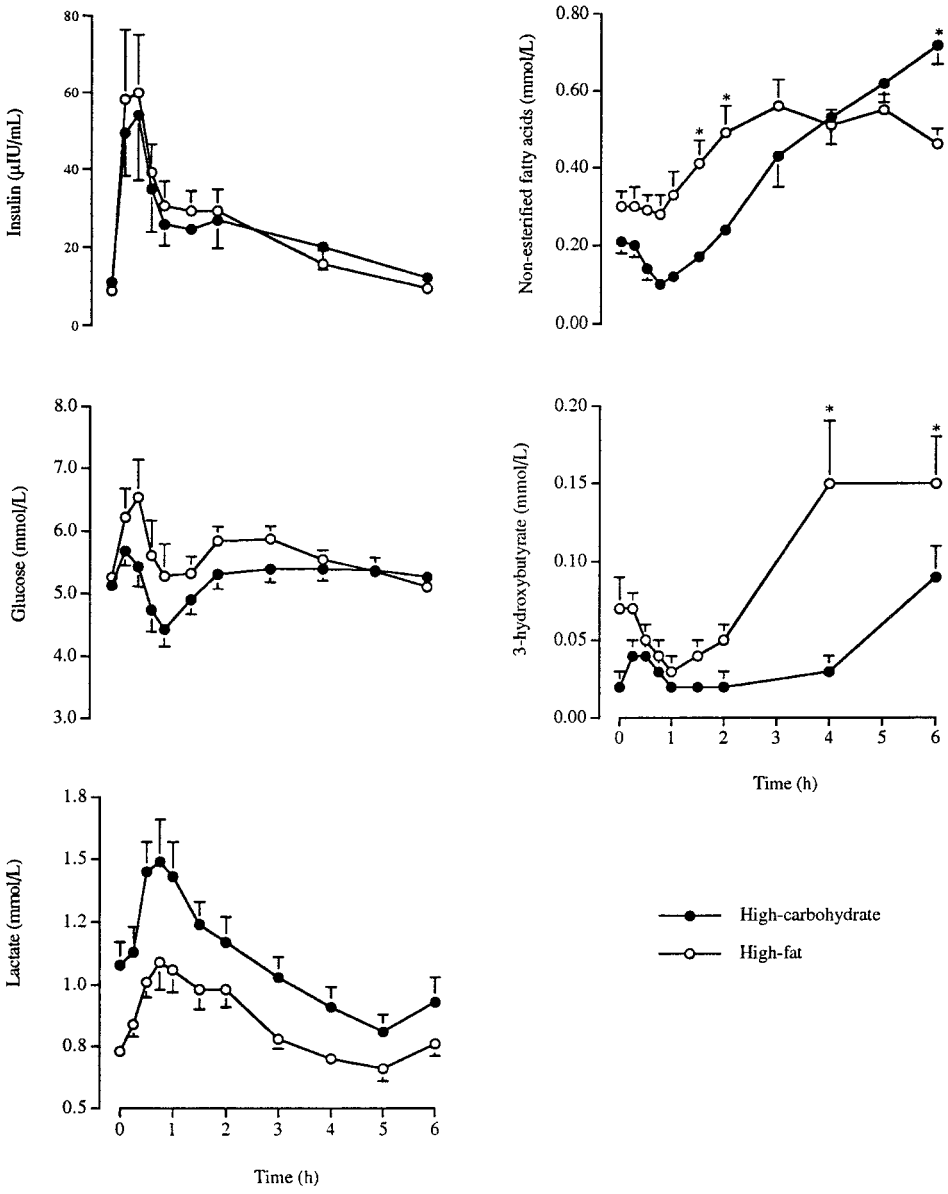

Fig 2. Postprandial concentrations of plasma NEFA, glucose, lactate, serum insulin, and 3-hydroxylbutyrate after 3 days on a high-carbohydrate diet and after 3 days on a high-fat diet (mean ± SEM; n = 9 men). *Significantly different *v* corresponding value in the other trial, $P < .05$.

after the high-carbohydrate diet (0.82 ± 0.01 *v* 0.76 ± 0.01, $P = .003$).

Neither total nor HDL cholesterol changed over the observation period in either trial. In the 6-hour blood samples, plasma total cholesterol did not differ significantly between trials but, as in the fasted state, HDL cholesterol was lower after the high-carbohydrate diet (1.00 ± 0.08 *v* 1.10 ± 0.09 mmol/L, $P = .002$).

DISCUSSION

This study was designed to investigate the effects of manipulation of the fat and carbohydrate content of isoenergetic diets on the metabolic responses to a standard test meal. It was performed during the initial, dynamic period of dietary change when effects on lipoprotein metabolism become manifest. Our findings confirm that low-fat, high-carbohydrate diets increase fasting[3] and postprandial[8,9] plasma TAG, with a complementary decrease in HDL cholesterol. However, in both of the postpran-

Table 3. Six-Hour Area Under the Plasma or Serum Concentration Versus Time Curve for TAG, NEFA, 3-Hydroxybutyrate, Insulin, Glucose, and Lactate After High-Carbohydrate and High-Fat Diets

Parameter	High-Carbohydrate Diet	High-Fat Diet	*P**
Total TAG (mmol/L · h)	12.63 ± 1.64	6.64 ± 0.50	.003
Incremental TAG (mmol/L · h)	5.54 ± 0.64	2.92 ± 0.47	.002
NEFA (mmol/L · h)	2.40 ± 0.20	2.80 ± 0.21	.12
3-Hydroxybutyrate (mmol/L · h)	0.23 ± 0.06	0.62 ± 0.11	.02
Insulin (μIU/mL · h)	144 ± 34	143 ± 25	.99
Glucose (mmol/L · h)	31.5 ± 1.0	33.6 ± 1.1	.009
Lactate (mmol/L · h)	6.40 ± 0.43	4.96 ± 0.22	.01

NOTE. Results are the mean ± SEM (n = 9 men). Each diet was consumed for 3 days.

*Significance of differences between trials by Student's *t* test for correlated means.

dial studies just cited, the test meals were not standardized but reflected the composition of the background diet and thus contained different amounts of fat and carbohydrate. Therefore, the differences in the postprandial responses to the test meals encompassed both the acute effects of the nutrients in the meals and the chronic effects of the preceding diets. In the present study, we used the same test meal after each diet intervention, and the differences in the responses to the meal therefore reflect the effects of the antecedent diets per se.

Our major new finding is that postprandial lipemia in healthy men was 42% higher after a short-term high-carbohydrate diet versus a high-fat diet, with differences in the TAG concentration between trials of up to 0.9 mmol/L. This finding, alongside the marked differences in the associated postprandial metabolic milieu, suggests that our 3-day dietary model may be attractive for future investigations of the mechanisms of carbohydrate-induced hypertriacylglycerolemia and of interventions that might attenuate this.

In the fasted state, diet-induced differences in very–low-density lipoprotein (VLDL) secretion may have contributed to the 0.6-mmol/L difference in the plasma TAG concentration between trials. Increased carbohydrate consumption stimulates hepatic synthesis and secretion of VLDL-TAG,[18] generating an expansion of the fasting TAG pool.[8,19] This may represent compositional changes in VLDL, with the production of larger particles, but since we did not measure VLDL apolipoprotein B levels, we have no evidence for this possibility. Increased de novo lipogenesis usually occurs during high-carbohydrate feeding but is not likely to have been the main determinant of increased VLDL synthesis, since the contribution of newly synthesized fatty acids to the total mass of VLDL-TAG probably never exceeds 17% to 20%.[20] Even so, this may spare the oxidation of fatty acids, channeling them into esterification and the secretory pathway,[20] which is in line with the 4-fold decrease in hepatic fatty acid oxidation that has been reported after a high-carbohydrate diet, alongside a 50% increase in the VLDL secretion rate.[18] In addition, suppressive effects of the high-fat diet on VLDL secretion may have contributed to the difference in the fasting TAG concentration between trials. Studies in rat hepatocytes have found that a high-fat diet decreases VLDL production, partly because the increased oxidation of fatty acids decreases their availability for VLDL-TAG synthesis.[21,22] Both mechanisms, ie, suppression of hepatic fatty acid oxidation after a high-carbohydrate diet and/or enhancement after a high-fat diet, would be consistent with the difference we observed between trials in plasma 3-hydroxybutyrate—a marker for hepatic fatty acid oxidation (Fig 2).

In the fasted state, the removal of VLDL may also have been influenced by the experimental diets, amplifying the differences in the TAG concentration between trials. The clearance rate of an artificial lipid emulsion has been reported to be higher after 4 days on a high-fat diet (65% energy as fat) versus the same period on a high-carbohydrate diet (80% energy as carbohydrate).[23] In line with these findings, it has recently been reported[24] that VLDL-TAG clearance in humans in the fasted state was reduced after a low-fat, high-carbohydrate diet.

In the postprandial state, we found an exaggerated and protracted lipemic response after the high-carbohydrate diet. This will have been due partly to the expanded fasting TAG pool, because chylomicrons compete with VLDL for the same rate-limiting enzyme (lipoprotein lipase) in the hydrolysis of their core TAG.[25] However, this is unlikely to be the sole explanation for the difference in postprandial lipemia.

The addition of simple carbohydrates to a high-fat meal accentuates the lipemic response.[10] One of several mechanisms could be that carbohydrate ingestion enhances the hepatic glycolytic rate, inhibiting the oxidation of fatty acids and diverting them toward the secretory pathway.[18,26] These responses to the carbohydrate in our meal could have been potentiated after a high-carbohydrate diet, priming the liver to secrete more VLDL-TAG postprandially—as well as in the fasted state. This, in turn, would increase the total TAG response. The high TAG concentrations during the late postprandial period (Fig 1) after the high-carbohydrate diet are consistent with this explanation, as are the low 3-hydroxybutyrate concentration and the high NEFA concentration which may have served as a substrate for hepatic TAG synthesis (Fig 2).

Plasma NEFA concentrations were higher after the high-fat diet during the early part of the observation period (Fig 2). In the postprandial state, the release of NEFA into the circulation is determined by the activity of intracellular hormone-sensitive lipase and the extent to which lipoprotein lipase–derived fatty acids are not entrapped in tissues.[27] Therefore, the accentuated NEFA response we observed after the high-fat diet could reflect an impairment of the normal postprandial suppression of hormone-sensitive lipase by insulin and/or a greater ''spillover'' of fatty acids from the action of lipoprotein lipase.

In the fasted state, glucose homeostasis was well maintained after the high-fat diet. However, the postprandial plasma glucose response was much greater after the high-fat diet, despite a similar insulin response. This probably reflects an impaired uptake of glucose by muscle due to the greater availability for oxidation of plasma fatty acids,[28] and the lower postprandial respiratory exchange ratios after the high-fat diet provide evidence for this.

The high plasma TAG concentration after the carbohydrate-rich diet may enhance the opportunity for the reciprocal transfer of cholesterol and TAG between HDL and TAG-rich lipoproteins, explaining our observation that HDL cholesterol was lower. However, the lower HDL cholesterol concentration after a high-carbohydrate diet could also simply reflect a lower requirement for HDL-mediated cholesterol removal at a lower intake of dietary fat.[29] It is interesting that the 3-day intervention was enough to provoke the expected decrease of HDL cholesterol by the carbohydrate-rich diet. As the modulation of HDL cholesterol is likely to be slower than the changes in TAG after initiation of a carbohydrate-rich diet, this supports the validity of our dietary intervention model.

In conclusion, the short-term consumption of a high-carbohydrate diet reduced plasma fasting HDL cholesterol, increased fasting TAG, and elevated the postprandial TAG response to a standard meal as compared with an isoenergetic high-fat diet. These findings complement earlier studies[8,9,30] using test meals with the same macronutrient composition as the diet interventions. However, to provoke clear metabolic changes, we used extreme dietary interventions. Our high-fat diet was very high in fat and associated with metabolic changes

observed in a state of insulin resistance, ie, higher blood glucose and plasma NEFA concentrations. Moreover, the nature of the foods consumed during the high-carbohydrate intervention (50% sugars, high–glycemic index complex carbohydrates) would be expected to stimulate hypertriacylglycerolemia more than diets containing, for example, fewer sugars and more legumes. For these reasons, our findings should not be interpreted as providing guidance on dietary recommendations.

The potential atherogenicity of carbohydrate-induced hypertriacylglycerolemia and concomitant decreases in HDL cholesterol are the subject of current debate.[2,11,31-34] These discussions have been based thus far on observations of fasting plasma lipids. However, postprandial plasma TAG in the late postprandial phase (6 hours after fat intake) may constitute an independent risk marker for CHD.[7] Interestingly, we observed augmented late postprandial TAG concentrations after the carbohydrate-rich diet. However, it is not known whether this carbohydrate-induced postprandial elevation of TAG is likely to confer a level of atherogenic risk similar to that evident in individuals consuming diets higher in fat.[7] The study of the early phase after a change to a high-carbohydrate diet may help to reveal the mechanisms underlying carbohydrate-induced hypertriacylglycerolemia.

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