# Long-term effects of ad libitum low-fat, high-carbohydrate diets on body weight and serum lipids in overweight subjects with metabolic syndrome[1–3]

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**ABSTRACT**

**Background:** Overweight individuals with metabolic syndrome are at increased risk of type 2 diabetes and coronary vascular disease. Weight gain and features of the syndrome may be ameliorated by dietary intervention.

**Objective:** We investigated the effects of replacing one-quarter of daily fat intake by complex or simple carbohydrate on body weight and intermediary metabolism.

**Design:** Forty-six subjects with ≥3 metabolic syndrome risk factors were randomly assigned to receive a control diet; a low-fat, complex carbohydrate diet (LF-CC); or a low-fat, simple carbohydrate diet (LF-SC) for 6 mo. Thirty-nine subjects completed the trial. About 60% of daily dietary intake was provided free of charge through a grocery store. Energy intake was ad libitum. Body weight, body mass index (BMI), blood pressure, and blood lipids were measured at months 0, 2, 4, and 6.

**Results:** There was a significant diet × time interaction on body weight and BMI ($P < 0.001$). Weight loss was greatest with the LF-CC diet [change in body weight: control diet, 1.03 kg (NS); LF-CC diet, −4.25 kg ($P < 0.01$); LF-SC diet, −0.28 kg (NS)]. Total cholesterol decreased by 0.33 mmol/L, 0.63 mmol/L, and 0.06 mmol/L in subjects consuming the control, LF-CC, and LF-SC diets, respectively (difference between the LF-CC and LF-SC groups: $P < 0.05$). There were no significant changes in LDL cholesterol, whereas HDL cholesterol decreased over time in all 3 groups ($P < 0.0001$). Triacylglycerol concentrations were higher in the LF-SC group than in the other 2 groups ($P < 0.05$).

**Conclusions:** A low-fat, high-polysaccharide diet in overweight individuals with abnormal intermediary metabolism led to moderate weight loss and some improvement in serum cholesterol. Increasing simple carbohydrates did not promote weight gain, but nor was there improvement in body weight or lipid profile. *Am J Clin Nutr* 2002;75:11–20.

**KEY WORDS** Metabolic syndrome, obesity, weight loss, low-fat diet, complex carbohydrate, simple sugars, blood lipids, CARMEN trial

## INTRODUCTION

Low-fat, high-carbohydrate diets have long been advocated to promote weight loss in the obese (1–3). High-carbohydrate diets are less energy dense, provide more bulk per kilojoule, are more satiating, and are more rapidly oxidized and less readily stored than are high-fat diets (4–8). These features help reduce food intake and, in compliant subjects, aid in weight loss.

Recently, however, the use of high-carbohydrate diets has been questioned. First, the results obtained in short-term, rigorously controlled trials of highly compliant subjects (6–10) are often not replicated when low-fat advice is transferred to larger, longer-term community trials (11–13). Second, high-carbohydrate diets may have adverse effects on circulating lipids. Although the serum-cholesterol-lowering effects of replacing total, or more specifically saturated, fat by carbohydrate are well established, evidence now shows a concomitant reduction in HDL cholesterol and an increase in triacylglycerol, both of which are adverse factors for cardiovascular disease (CVD) risk (14, 15). Whether these changes in HDL cholesterol and triacylglycerol are maintained long term during weight loss is less well understood, as is the clinical significance of these changes if accompanied by reductions in serum LDL concentrations and blood pressure (16). Third, an association may exist between a high sugar intake and the prevalence of overweight and obesity. Until the recently published CARMEN (Carbohydrate Ratio Manipulation in European National Diets) trial (17), no community intervention trials had specifically investigated the role of simple carbohydrates in the etiology of obesity, and several

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questions regarding a high sugar intake, increase in energy density, and potential weight gain have been raised.

The present study investigated the effect of 2 low-fat, high-carbohydrate diets on weight loss and intermediary metabolism in overweight subjects recruited as part of the CARMEN multicenter trial (17). Subjects at high risk of CVD were selected according to risk factors for the metabolic syndrome. Resistance to insulin-mediated glucose disposal occurs in many (18) but not all (19) overweight individuals. Although progression to type 2 diabetes may not follow, hyperinsulinemia is often associated with moderately raised blood glucose, blood pressure, and mild lipid disorders. This metabolic syndrome cluster is relatively common in older, overweight, sedentary individuals (20). We hypothesized that, if long-term compliance could be achieved, an ad libitum low-fat, high-carbohydrate diet would reduce body weight and fatness and, in turn, improve lipid and other indexes of intermediary metabolism associated with coronary disease risk.

## SUBJECTS AND METHODS

### Subjects

Overweight subjects were recruited through television and newspaper advertisements placed in the Cambridge, United Kingdom, area. One hundred twenty potential subjects were invited to attend a screening session in which body weight, height, waist circumference, blood pressure, and blood biochemistry indexes were measured to determine eligibility for the present trial. Subjects currently dieting were excluded, as were those planning to begin a weight control program within the next 8 mo. Fifty subjects were recruited on the basis of having ≥3 risk factors for metabolic syndrome. These risk factors included age >38 y, overweight [body mass index (BMI; in kg/m$^2$) of 27–40], central obesity as assessed by the waist-to-hip ratio (women, >0.8; men, >0.9), a family history of type 2 diabetes, fasting plasma glucose of 5.5–6.9 mmol/L, HDL cholesterol <1.0 mmol/L, triacylglycerol >2.0 mmol/L, and diastolic blood pressure of 85–100 mm Hg. Thirteen of these subjects were also part of the multicenter CARMEN trial, in which 398 overweight individuals recruited simply on the basis of body weight participated in an identical 6-mo intervention. The results of the CARMEN trial are published elsewhere (17).

Four subjects were excluded for noncompliance during a 1-mo run-in period; the remaining 46 subjects were randomly assigned to either the control diet ($n$ = 15); the low-fat, high-complex-carbohydrate (LF-CC) diet ($n$ = 16); or the low-fat, high-simple-carbohydrate (LF-SC) diet ($n$ = 15) group. Ethical approval for the study was obtained from the Dunn Nutrition Unit and Cambridge Local Research ethics committees and all subjects gave written, informed consent before participating in the screening procedure.

### Experimental protocol

All subjects completed a 1-mo run-in period during which they consumed a control diet (containing 40% fat) to accustom them to the study grocery store system before the intervention. Subjects were then randomly assigned to either the control diet, the LF-CC diet, or the LF-SC diet groups and were provided with appropriate foods for the 6-mo intervention period. Subjects continued to live at home and came to the study grocery store on 1 or 2 occasions per week to collect foods and discuss their energy and macronutrient intakes with the program dietitian. The program was designed to provide 60–70% of each subject's dietary intake. No fresh foods (eg, fruit and vegetables) were provided. Participants were excluded from the program if they failed to attend the study grocery story for a total of >28 d during the 6 mo. During short holiday breaks away from the unit, subjects were asked to maintain the diet of their group if possible by supplying their own foods. However, no dietary monitoring of these periods was possible. Subjects attended a research clinic monthly, at which time they were weighed while fasting and lightly clad. Every 2 mo, waist circumference (as recommended by a 1998 National Institutes of Health evidence report; 21) and blood pressure were measured and a fasting blood sample collected for measurement of serum concentrations of glucose, total cholesterol, LDL cholesterol, HDL cholesterol, triacylglycerol, and fatty acids.

### Assessment of dietary intake

Total dietary intake was estimated from 7-d and 3-d weighed-food records collected on 5 occasions during the study. Reported food intake was recorded before the run-in period (3-d record), during the 1-mo run-in period (7-d record), and during months 1 (3-d record), 4 (3-d record), and 6 (7-d record) of the intervention. The dietitian provided subjects with food scales accurate to 1 g and diet record booklets and explained in detail the weighed intake method. Subjects were asked to weigh all foods eaten both within and outside the home. The energy and macronutrient contents of the reported foods were calculated from the weight of food consumed by using both information obtained from the food packaging and that provided by standard UK food-composition tables (22) in which foods were coded according to the nearest appropriate food type or brand. A cutoff of 1.2 × basal metabolic rate (BMR) (23) was calculated from the age, height, weight, and sex of each subject to establish the reliability of the estimates of energy intake throughout the study. Reported energy intake of <1.2 × BMR was assumed to be unreliable and unrepresentative of habitual food intake.

### Intervention diets

The study was designed to provide the participants with ≥60% of their total energy intake from the study grocery store; the remainder of energy intake was provided by the subjects' home diet. The goals of the study were to *1*) maintain fat intake in the control group at habitual amounts (≈35–40% of energy), *2*) reduce fat intake by 10% of total energy in both low-fat groups, *3*) to alter the ratio of simple to complex carbohydrate to 1:2 in the LF-CC group, and *4*) to alter the ratio of simple to complex carbohydrate to 2:1 in the LF-SC group. Subjects were not encouraged to actively reduce their intake, but to eat ad libitum while maintaining the integrity of the macronutrient composition of their allocated diet at all times. Only prepackaged and prepared foods were provided during the study. Subjects were encouraged to consume fresh fruit and vegetables but were required to provide this portion of the diet themselves.

### Study grocery store system

Food was provided free of charge to the study subjects from a study grocery store near the research clinic. Subjects were asked to attend the store at least once per week and were encouraged to attend more frequently if possible. A wide variety of prepackaged foods (**Appendix A**), selected on the basis of their fat or

**TABLE 1**
Baseline characteristics of the overweight subjects with risk factors for metabolic syndrome who completed the 6-mo dietary intervention[1]

| Variable | Control group ($n$ = 1 M, 10 F) | LF-CC group ($n$ = 5 M, 9 F) | LF-SC group ($n$ = 6 M, 8 F) |
|---|---|---|---|
| Family history of diabetes ($n$) | 4 | 6 | 5 |
| Age (y) | 48.6 ± 4.4[2] | 44.2 ± 5.5 | 45.9 ± 5.0 |
| Height (m) | 1.66 ± 0.08 | 1.68 ± 0.10 | 1.70 ± 0.11 |
| Weight (kg) | 91.4 ± 9.2 | 91.2 ± 9.5 | 89.3 ± 15.7 |
| BMI (kg/m$^2$) | 33.1 ± 3.3 | 32.3 ± 3.6 | 30.9 ± 3.0 |
| Waist circumference (m) | 0.98 ± 0.14 | 1.03 ± 0.10 | 1.00 ± 0.11 |
| Men | 1.02 ± 0.00 | 1.08 ± 0.06 | 1.09 ± 0.06 |
| Women | 0.98 ± 0.15 | 1.01 ± 0.11 | 0.93 ± 0.09 |
| SBP (mm Hg) | 132 ± 14 | 136 ± 17 | 138 ± 22 |
| DBP (mm Hg) | 87 ± 10 | 86 ± 13 | 84 ± 13 |
| Total cholesterol (mmol/L) | 6.2 ± 1.0 | 5.7 ± 1.0 | 5.9 ± 1.4 |
| HDL cholesterol (mmol/L) | 1.4 ± 0.3 | 1.3 ± 0.2 | 1.1 ± 0.3 |
| LDL cholesterol (mmol/L) | 4.1 ± 0.9 | 3.7 ± 0.7 | 3.8 ± 0.8 |
| TC:HDL | 4.6 ± 1.4 | 4.6 ± 1.0 | 5.3 ± 1.9 |
| Triacylglycerol (mmol/L) | 2.1 ± 1.1 | 1.9 ± 1.3 | 2.3 ± 1.3 |
| Fasting glucose (mmol/L) | 5.5 ± 0.6 | 5.9 ± 0.7 | 5.6 ± 0.5 |

[1] LF-CC, low-fat, high-complex-carbohydrate; LF-SC, low-fat, high-simple-carbohydrate; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol. There were no significant differences between groups at baseline.
[2] $\bar{x}$ ± SD.

carbohydrate (simple and complex) content, was available. Foods were displayed on shelves, in refrigerators, and in freezer compartments and were clearly labeled according to diet group. Subjects were free to choose only from the selection of foods allocated to their diet group. After the foods were selected, the bar code of each item was scanned and the macronutrient composition of the selection determined. Researchers checked the macronutrient composition of each item as it was scanned to ensure that incorrect items had not been selected. Subjects were then provided with a printout of the individual foods selected. Before their return to the store, the subjects were asked to weigh all individual items that remained uneaten. The energy and macronutrient content of the foods eaten for that period and for the entire intervention were then calculated and the results discussed with the individual subject.

### Statistical analyses

Statistical analyses were performed with use of SAS (version 8; SAS Institute Inc, Cary, NC). Repeated-measures analyses (generalized linear mixed procedure) were used to investigate the changes over time in anthropometric, lipid, and other metabolic variables; the macronutrient composition and energy intake of foods taken from the study grocery store; and self-reported intake of macronutrients and energy. All subjects were included in the analyses until the point at which they withdrew or were excluded from the trial. A mixed-model approach was used in which all missing values resulting from drop out and exclusion were assumed to be missing at random. The interaction between time and diet was included in the model to test whether changes over time differed between diet groups. For anthropometric, lipid, and other metabolic variables, baseline (month 0) was used as a covariate. For the macronutrient composition and energy intake of foods from the study store and self-reported total macronutrient and energy intakes, the run-in measure was used as a covariate.

When the interaction between diet and time was significant, the mean difference between the 3 diets differed with time. The 3 diets were then modeled separately to investigate changes over time. It is inappropriate to model dietary effects separately and hence these are not reported. The significant interaction indicates that there was a difference between the diets in the patterns over time and the separate models indicate what those patterns were. When the interaction between diet and time was not significant, the mean difference did not vary with time and the 3 time courses could be considered parallel. The interaction term was removed from the model and became part of the error term. A second model without the interaction was then used to investigate the main effects of diet and time. The effect of weight loss of >3% of body weight was analyzed independent of diet group between the beginning and the end of the intervention by using a paired $t$ test. Spearman's correlation was used to identify relations between weight loss and other outcome measures for all subjects, independent of diet group. Significance was set at $P < 0.05$.

## RESULTS

Of the 46 men and women randomly assigned to treatment groups, 3 participants withdrew when informed of their dietary treatment, all having been assigned to the control diet. An additional 4 subjects either withdrew or were excluded for noncompliance during the 6-mo intervention (control, $n$ = 1; LF-CC, $n$ = 2; LF-SC, $n$ = 1). Thus, 39 subjects completed the entire study. When calculated as a percentage of the 50 subjects originally recruited, the rate of drop-out was 22%. Baseline characteristics of the 39 subjects who completed the trial are shown in **Table 1**. There were no significant differences in age, body weight, BMI, waist circumference, blood pressure, fasting glucose concentrations, or lipid profile between the 3 diet groups at baseline.

### Intake of foods selected from the study grocery store

The subjects' monthly energy intake and the macronutrient composition (as a percentage of energy intake) of the food selected from the study grocery store are shown in **Table 2**. About 60% of predicted energy requirements (estimated as 1.4 × BMR) were provided by the study store during the intervention. There

**TABLE 2**
Energy and macronutrient intake of food selected from the study grocery store during the 1-mo run-in and 6-mo dietary intervention[1]

| | Energy intake | | | Energy intake as a percentage of requirements: all diet groups[2] | Fat intake | | | Complex carbohydrate intake | | | Simple carbohydrate intake | | | Simple:complex carbohydrate | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Control group | LF-CC group | LF-SC group | | Control group | LF-CC group | LF-SC group | Control group | LF-CC group | LF-SC group | Control group | LF-CC group | LF-SC group | Control group | LF-CC group | LF-SC group |
| | *kJ/d* | | | | *% of energy* | | | *% of energy* | | | *% of energy* | | | | | |
| Run-in ($n = 46$) | 4200 | 4814 | 5006 | 47.5 | 39.5 | 36.0 | 36.5 | 19.2 | 21.2 | 22.6 | 18.6 | 21.7 | 18.8 | 0.97 | 1.02 | 0.83 |
| Month 1 ($n = 43$) | 2412 | 2486 | 2934 | 26.5 | 38.8 | 25.3 | 23.2 | 21.4 | 33.8 | 23.2 | 16.3 | 13.5 | 29.9 | 0.76 | 0.40 | 1.29 |
| Month 2 ($n = 43$) | 4358 | 4175 | 4687 | 44.6 | 36.6 | 23.2 | 20.3 | 23.4 | 33.3 | 23.8 | 16.6 | 13.9 | 32.8 | 0.71 | 0.42 | 1.38 |
| Month 3 ($n = 42$) | 6703 | 5303 | 6917 | 64.2 | 35.4 | 25.5 | 19.6 | 22.9 | 33.0 | 23.5 | 19.3 | 13.2 | 33.8 | 0.84 | 0.40 | 1.44 |
| Month 4 ($n = 42$) | 7166 | 6426 | 7739 | 71.5 | 33.5 | 24.6 | 19.1 | 22.6 | 34.2 | 23.4 | 20.2 | 13.8 | 35.1 | 0.89 | 0.40 | 1.50 |
| Month 5 ($n = 40$) | 7270 | 6763 | 8203 | 75.0 | 33.7 | 24.1 | 19.6 | 23.2 | 32.8 | 24.0 | 21.9 | 13.6 | 34.8 | 0.94 | 0.41 | 1.45 |
| Month 6 ($n = 39$) | 8077 | 7186 | 7734 | 75.5 | 34.7 | 24.7 | 19.3 | 24.2 | 33.4 | 22.6 | 19.9 | 13.7 | 36.1 | 0.82 | 0.41 | 1.60 |
| Mean (months 1–6) | 5997 ± 2161[3] | 5389 ± 1794 | 6369 ± 2099 | 59.6 ± 19.9 | 35.5 ± 2.00[a] | 24.6 ± 0.84[b] | 20.2 ± 1.53[b] | 23.0 ± 0.93[a] | 33.4 ± 0.52[b] | 23.4 ± 0.49[a] | 19.0 ± 2.18[a] | 13.6 ± 0.25[b] | 33.8 ± 2.20[c] | 0.83± 0.08[a] | 0.41± 0.01[b] | 1.44± 0.11[c] |
| Significant effects | | | | | | | | | | | | | | | | |
| Time × diet | | NS | | — | | NS | | | NS | | | $P < 0.05$ | | | $P < 0.05$ | |
| Diet | | NS | | — | | $P < 0.0001$ | | | $P < 0.0001$ | | — | — | — | — | — | — |
| Time | | $P < 0.0001$ | | — | | $P < 0.0001$ | | | NS | | $P < 0.05$ | NS | $P < 0.01$ | NS | NS | $P < 0.01$ |

[1] LF-CC, low-fat, high-complex-carbohydrate; LF-SC, low-fat, high-simple-carbohydrate. Means within a row with different superscript letters are significantly different, $P < 0.05$.
[2] Average energy intake from the study grocery store calculated as a percentage of predicted requirements (1.4 × basal metabolic rate).
[3] $\bar{x} \pm$ SD.

**TABLE 3**
Reported 7-d and 3-d energy and macronutrient intakes preintervention, during the 1 month run-in, and during the 6-mo dietary intervention[1]

| | Energy intake | | | PAL | | | Fat intake | | | Complex carbohydrate intake | | | Simple carbohydrate intake | | | Simple:complex carbohydrate | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Control group | LF-CC group | LF-SC group | Control group | LF-CC group | LF-SC group | Control group | LF-CC group | LF-SC group | Control group | LF-CC group | LF-SC group | Control group | LF-CC group | LF-SC group | Control group | LF-CC group | LF-SC group |
| | *kJ/d* | | | | | | *% of energy* | | | *% of energy* | | | *% of energy* | | | | | |
| Preintervention (3-d) ($n = 46$) | 8352 | 7368 | 8242 | 1.20 | 1.03 | 1.13 | 32.0 | 29.8 | 35.8 | 26.9 | 31.2 | 26.6 | 21.7 | 18.9 | 16.6 | 0.81 | 0.61 | 0.62 |
| Run-in (7-d) ($n = 46$) | 8366 | 9430 | 9365 | 1.21 | 1.29 | 1.31 | 32.6 | 32.6 | 33.9 | 28.2 | 26.8 | 27.3 | 21.2 | 20.9 | 18.9 | 0.75 | 0.78 | 0.69 |
| Month 1 (3-d) ($n = 43$) | 7860 | 7829 | 8167 | 1.14 | 1.08 | 1.13 | 30.8 | 24.5 | 22.4 | 27.8 | 35.0 | 30.8 | 20.7 | 17.6 | 24.7 | 0.74 | 0.50 | 0.80 |
| Month 3 (3-d) ($n = 42$) | 8478 | 8240 | 9962 | 1.18 | 1.15 | 1.39 | 30.6 | 22.4 | 21.1 | 29.6 | 35.8 | 27.6 | 20.1 | 20.8 | 29.0 | 0.68 | 0.58 | 1.05 |
| Month 4 (3-d) ($n = 42$) | 7467 | 9050 | 10392 | 1.07 | 1.27 | 1.44 | 31.4 | 23.5 | 21.3 | 28.9 | 36.8 | 29.0 | 21.6 | 16.2 | 29.5 | 0.75 | 0.44 | 1.02 |
| Month 6 (7-d) ($n = 39$) | 8281 | 7316 | 9790 | 1.17 | 1.07 | 1.33 | 31.2 | 26.0 | 19.6 | 27.9 | 34.2 | 26.4 | 19.9 | 15.7 | 32.3 | 0.71 | 0.46 | 1.22 |
| Mean (months 1–6) | 8022 ± 1868[a,2] | 8108 ± 2689[b] | 9578 ± 2600[c] | 1.14 ± 0.20 | 1.14 ± 0.31 | 1.32 ± 0.38 | 31.0 ± 1.48[a] | 24.1 ± 5.36[b] | 21.1 ± 3.11[b] | 28.6 ± 3.44[a] | 35.5 ± 3.89[b] | 28.5 ± 5.10[a] | 20.6 ± 3.04[a] | 17.6 ± 8.05[b] | 28.9 ± 8.48[c] | 0.72 ± 0.12 | 0.50 ± 0.21 | 1.02 ± 0.46 |
| Significant effects | | | | | | | | | | | | | | | | | | |
| Time × diet | | $P < 0.01$ | | | — | | | NS | | | NS | | | $P < 0.05$ | | | — | |
| Diet | — | — | — | | — | | | $P < 0.0001$ | | | $P < 0.0001$ | | — | — | — | | — | |
| Time | NS | NS | $P < 0.0002$ | | — | | | NS | | | NS | | NS | NS | $P < 0.05$ | | — | |

[1] LF-CC, low-fat, high-complex-carbohydrate; LF-SC, low-fat, high-simple-carbohydrate; PAL, physical activity level (reported energy intake/predicted basal metabolic rate). Means within a row with different superscript letters are significantly different, $P < 0.05$.
[2] $\bar{x} \pm$ SD.

were no significant diet × time interactions among treatments for energy intake, fat intake, or complex carbohydrate intake. For energy intake, when the interaction term was removed, there was no significant difference between diet groups, but energy intake increased in all diet groups over time (time effect, $P < 0.0001$). Fat intake was significantly lower in both LF groups than in the control group (diet effect, $P < 0.0001$). Complex carbohydrate intake was significantly higher in the LF-CC group than in either the control group or the LF-SC group (diet effect, $P < 0.0001$). Simple carbohydrate intake was higher in the LF-SC group than in either the control group or the LF-CC group and changed significantly over time among the 3 diet groups (diet × time interaction, $P < 0.05$). Only in the LF-SC diet group did simple carbohydrate intake increase over time (time effect, $P < 0.01$). These differences between diet groups suggest that food provision from a controlled source was a successful means of altering the macronutrient composition of the diet.

### Reported intake

Analyses of self-reported energy and macronutrient intakes are shown in **Table 3**. Physical activity level (reported energy intake/predicted BMR) values during the intervention were 1.14, 1.14, and 1.32 for the control, LF-CC, and LF-SC diet groups, respectively. Although the reporting of energy intake differed significantly among the 3 diet groups over time (diet × time interaction, $P < 0.01$), only in the LF-SC group were the results above the 1.2 × BMR cutoff, a consequence of improvements in reporting energy intake in that group as the study progressed (time effect, $P < 0.0002$).

Evidence of underreporting of energy intake in all 3 diet groups requires the reported macronutrient intakes to be viewed with caution. There were no significant diet × time interactions for reported fat or complex carbohydrate intake and hence no significant differences over time among dietary treatments. Removal of the interaction term from the model showed reported fat intake to be significantly lower in both LF groups than in the control group (diet effect, $P < 0.0001$). Reported complex carbohydrate intake was significantly higher in the LF-CC group than in either the control group or the LF-SC group (diet effect, $P < 0.0001$). Reported simple carbohydrate intake changed significantly over time among the 3 diet groups (diet × time interaction, $P < 0.05$) and increased from the run-in to the end of intervention in the LF-SC group only (time effect, $P < 0.05$).

### Body weight

The effects of diet on body weight, BMI, and waist circumference during the 6-mo intervention are shown in **Figure 1**. Between 0 and 6 mo, body weight changed by 1.03, −4.25, and −0.28 kg in the control, LF-CC, and LF-SC groups, respectively. Changes over time were significantly different among the 3 groups (diet× time interaction, $P < 0.001$). When each diet group was modeled separately, there was significant weight loss over time only in the LF-CC group (time effect, $P < 0.01$). The effect of dietary treatment on body weight was reflected by a similar change in BMI (diet × time interaction, $P < 0.001$). Only in the LF-CC group did BMI decrease significantly over the 6-mo intervention (time effect, $P < 0.01$). Weight loss was not accompanied by significant changes in abdominal obesity: there were no significant changes in waist circumference over time among the diet groups, over time in any individual diet group, or between the 3 diet groups.

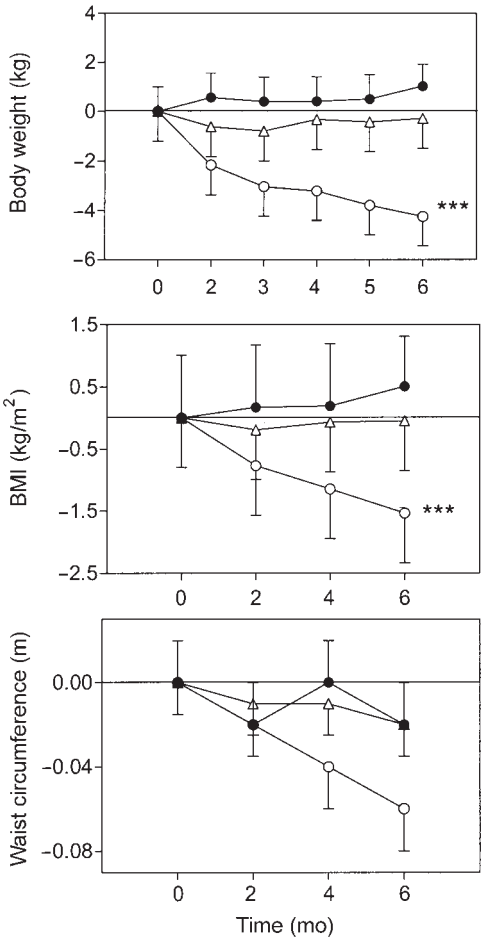


**FIGURE 1.** Mean (±SEM) changes relative to baseline (month 0) in body weight, BMI, and waist circumference during the 6-mo intervention in subjects consuming the control diet (●); low-fat, high-complex-carbohydrate diet (LF-CC; ○); and low-fat high-simple-carbohydrate diet (LF-SC; △). There was a significant diet × time effect for body weight and BMI, $P < 0.001$. ***Significant time effect, $P < 0.01$.

### Lipids and blood pressure

Total cholesterol decreased by −0.33, −0.63, and −0.06 mmol/L in the control, LF-CC, and LF-SC groups, respectively, by the end of the intervention (**Figure 2**). There was no significant diet × time interaction and hence no evidence that changes in total cholesterol over time were different among the 3 treatment groups. Removal of the interaction term showed that total cholesterol was lower in the LF-CC group than in the LF-SC group (diet effect, $P < 0.05$). Total cholesterol decreased by 11.1% of baseline by the end of intervention in the LF-CC group.

There was no significant change over time among the dietary groups for LDL or HDL cholesterol, nor was there a significant difference between the 3 diet groups. LDL cholesterol decreased by 11.4% of baseline in the LF-CC group, but this change was not significant because of the small sample size and individual variability. There was no significant effect of dietary treatment on HDL cholesterol, but HDL cholesterol decreased in all groups over the 6 mo of the intervention (time effect, $P < 0.0001$). The ratio of total to HDL cholesterol (data not shown) was not significantly affected by diet treatment but, influenced by the decrease in HDL, increased significantly over time across all treatments (time effect, $P < 0.01$).

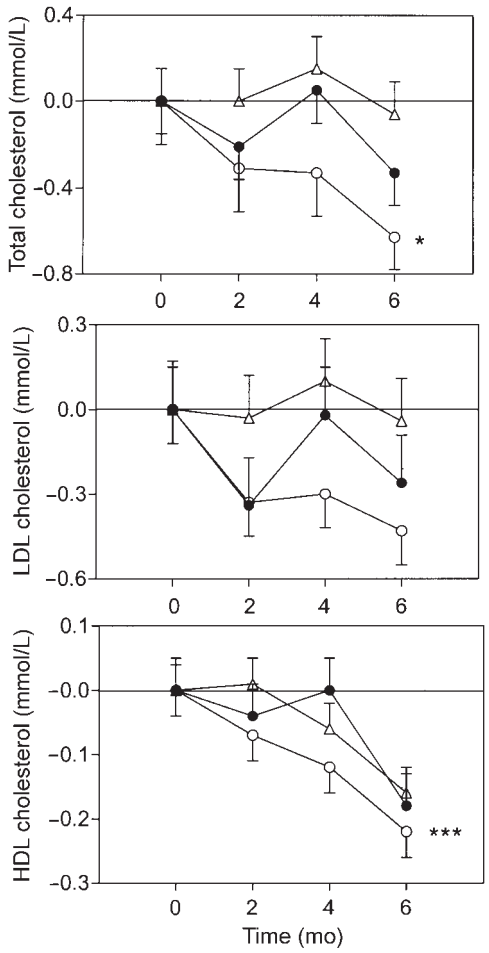


**FIGURE 2.** Mean (±SEM) changes relative to baseline (month 0) in the lipoprotein profile during the 6-mo intervention in subjects consuming the control diet (●); low-fat, high-complex-carbohydrate diet (LF-CC; ○); and low-fat high-simple-carbohydrate diet (LF-SC; △). There were no significant differences in changes over time between the 3 diet groups. *Significantly different from the LF-SC group, $P < 0.05$ (diet effect). ***Significantly different from baseline in all 3 groups, $P < 0.0001$ (time effect).

Fasting triacylglycerol concentrations were higher in the LF-SC group than in the other 2 diet groups (diet effect, $P < 0.05$; **Figure 3**). Blood pressure was highly variable throughout the trial in all diet groups. There was no significant diet × time interaction for systolic or diastolic blood pressure and no evidence that changes over time were significantly different. However, systolic blood pressure was higher in the control group than in either low-fat group (diet effect, $P < 0.01$), and diastolic blood pressure was higher in the control group than in the LF-CC group (diet effect, $P < 0.05$).

### Effects of weight loss

The effects of weight change on the metabolic syndrome variables analyzed as weight loss of < or ≥ 3% of baseline body weight but independent of diet group are shown in **Table 4**. BMI, waist circumference, total cholesterol, and triacylglycerol decreased significantly during the 6-mo intervention only in subjects who lost ≥3% body weight ($P < 0.05$). HDL cholesterol, LDL cholesterol, and systolic blood pressure also tended to decrease more in subjects who lost ≥3% of their baseline body weight (NS). Spearman correlations showed that weight loss tended to be correlated with a decrease in all measured variables but significantly correlated with the change in BMI, waist circumference, and triacylglycerol only (**Table 5**).

## DISCUSSION

This study of low-fat, high-carbohydrate diets in overweight subjects with characteristics of metabolic syndrome showed that further weight gain is prevented, and modest weight loss may be achieved, with a diet in which one-quarter of dietary fat is replaced by simple or complex carbohydrates. The most weight was lost and total cholesterol concentrations tended to decrease when complex carbohydrates were substituted for fat in the diet. It is possible that the 11–12% decrease in cholesterol induced by the high-polysaccharide diet could affect CVD outcome (24). Subjects consuming the high-sugar diet did not lose significant amounts of weight, but also did not gain weight despite the

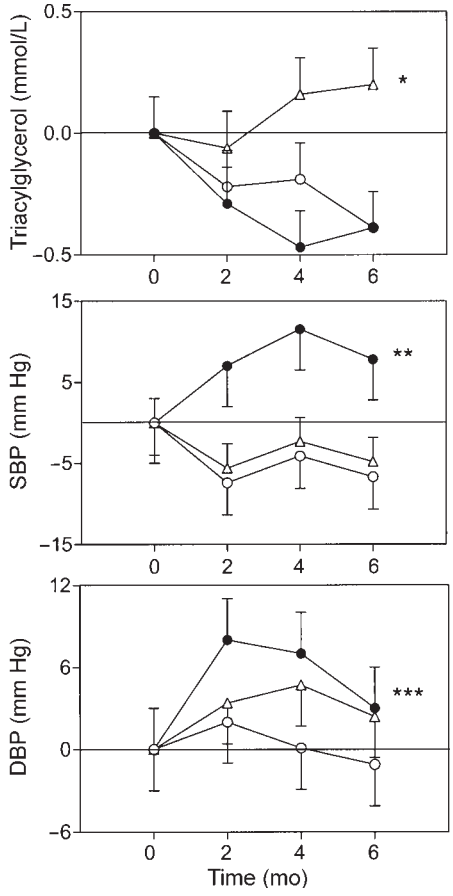


**FIGURE 3.** Mean (±SEM) changes relative to baseline (month 0) in triacylglycerol, systolic blood pressure (SBP), and diastolic blood pressure (DBP) pressure during the 6-mo intervention in subjects consuming the control diet (●); low-fat, high-complex-carbohydrate diet (LF-CC; ○); and low-fat high-simple-carbohydrate diet (LF-SC; △). There were no significant differences in changes over time between the 3 diet groups. *Significantly higher than in the other 2 treatment groups, $P < 0.05$ (diet effect). **Significantly higher than in both LF groups, $P < 0.01$ (diet effect). ***Significantly higher in the control group than in the LF-CC group, $P < 0.05$ (diet effect).

**TABLE 4**
The effect of weight loss of ≥3% of body weight during the 6-mo intervention on changes (Δ) in anthropometric, lipid, and blood pressure variables[1]

| Change in variable | <3% Weight loss ($n$ = 23) | ≥3% Weight loss ($n$ = 16) |
|---|---|---|
| Δ BMI (kg/m$^2$) | 0.40 ± 0.17 | −1.70 ± 0.20[2] |
| Δ Waist circumference (m) | −0.01 ± 0.01 | −0.06 ± 0.01[3] |
| Δ Total cholesterol (mmol/L) | −0.10 ± 0.16 | −0.69 ± 0.19[4] |
| Δ Triacylglycerol (mmol/L) | 0.09 ± 0.19 | −0.55 ± 0.22[4] |
| Δ HDL cholesterol (mmol/L) | −0.16 ± 0.05 | −0.21 ± 0.06 |
| Δ LDL cholesterol (mmol/L) | −0.17 ± 0.17 | −0.38 ± 0.20 |
| Δ SBP (mm Hg) | −0.15 ± 3.3 | −5.00 ± 3.69 |
| Δ DBP (mm Hg) | 1.45 ± 1.88 | 0.94 ± 2.10 |

[1] $\bar{x}$ ± SEM. Change was calculated as month 6 − month 1. SBP, systolic blood pressure; DBP, diastolic blood pressure.
[2–4] Significantly different from <3% weight loss: [2] $P < 0.0001$, [3] $P < 0.01$, [4] $P < 0.05$.

ad libitum nature of the diet; hence, total and LDL-cholesterol concentrations remained unchanged.

The influence of diet on lipid profiles in men and women with mild dyslipidemia, and in some cases associated glucose intolerance, is an important issue. Metabolic syndrome is relatively widespread throughout Western populations and is of considerable public health concern. It has been argued that the reduction in CVD risk achieved by lowering total or LDL-cholesterol concentrations through the substitution of carbohydrate for fat is negated by adverse changes in HDL and triacylglycerol concentrations (14, 15). Certainly, studies have shown associations between increased carbohydrate intake and decreased HDL cholesterol (25–27). In our intervention, in which an increase in dietary carbohydrates resulted in weight loss, the small improvements in total and LDL cholesterol did not appear to be offset by worsening of the HDL-cholesterol and triacylglycerol lipid fractions. HDL cholesterol did decline over the time course of the intervention, but the decrease occurred across all dietary treatments, including the control diet, possibly as a consequence of the metabolic characteristics of the men and women recruited for this trial. When the increase in carbohydrate content of the diet was not accompanied by significant weight loss and there was no improvement in total or LDL cholesterol, the decline in HDL cholesterol was associated with an increase in circulating triacylglycerol concentrations. This may be of some concern.

The mechanisms by which carbohydrates may affect the lipid profile are not well understood. VLDL cholesterol production may increase in response to dietary carbohydrate, leading to an increase in triacylglycerols and a subsequent decrease in HDL cholesterol. Certainly, changes in triacylglycerol concentrations appear to alter the phospholipid, free cholesterol, and cholesterol ester components of HDL (28). The effect of specific carbohydrate types per se is difficult to discern from our study because of the differential weight loss between individuals in the 2 carbohydrate groups. Our results suggest, however, that when moderate weight loss is achieved, there are no adverse changes in the lipid profile in overweight, metabolically compromised men and women. Interestingly, HDL cholesterol decreased in both high-carbohydrate groups and weight loss did not ameliorate this decline. It is difficult to attribute this decrease simply to dietary change because HDL-cholesterol concentrations also decreased in the subjects consuming the control diet.

Weight changes in the current trial are similar to those previously reported in the multicenter CARMEN trial (17). In this larger trial, an average of 1.7 and 2.6 kg body weight was lost with the LF-SC and LF-CC diets relative to the control diet, compared with 1.3 and 5.3 kg in the current intervention. In the CARMEN trial, there were no significant changes, either beneficial or adverse, in any of the circulating lipids measured (17). Greater weight loss with the LF-CC diet was consistent across both trials. In the current trial it seems likely that weight loss rather than macronutrient composition per se was driving the change in cholesterol.

Weight loss or gain is a consequence of a change in energy balance, ie, energy consumed relative to energy expended. Macronutrient composition merely drives appetite and hence the energy intake side of the equation. Data from the weighed-food records in our trial showed total energy intake to be 8022, 8108, and 9578 kJ/d in the control, LF-CC, and LF-SC groups, respectively. Because diet records are reliable only as a tool by which to rank intake and not as an absolute measure (23), it is necessary to be cautious when interpreting these results. As would be predicted from the amount of weight lost in the 2 carbohydrate groups, reported energy intake was much higher in the high-sugar group. The successful pattern of weight loss in the complex carbohydrate group, however, cannot be explained by any differential in reported energy intake between this group and the control group. Subjects in the high-sugar group found it difficult to incorporate the very high sugar component into their diet, and encouragement by the dietitians resulted in supplementation of, rather than substitution for, both fat and complex carbohydrates. A high sugar intake has been proposed as a causal factor in the etiology of obesity. The results of epidemiologic studies, however, oppose this view (29) and are supported by our current trial. Despite a considerable increase in sugar intake, there was no evidence of weight gain in the LF-SC group.

The provision of food from the study grocery store was intended to increase long-term motivation and appeared successful. The aim was to replace 10% of dietary fat with complex or simple carbohydrates, because low-fat, low-energy diets result in a negative energy and fat balance and weight loss if the study is well controlled and the integrity of the diet maintained (5–8). A low-fat, low-energy diet induces satiety at a lower energy intake than does a high-fat, high-energy diet (30–32). Although in well-controlled interventions compliance with a low-fat diet will aid weight loss, little success is achieved in trials in which individuals eat freely at home.

**TABLE 5**
Spearman correlations between weight loss and changes (Δ) in anthropometric, lipid, and blood pressure variables[1]

| Change in variable | Correlation coefficient | $P$ |
|---|---|---|
| Δ BMI (kg/m$^2$) | 0.99 | <0.0001 |
| Δ Waist circumference (m) | 0.42 | <0.05 |
| Δ Total cholesterol (mmol/L) | 0.29 | 0.073 |
| Δ LDL cholesterol (mmol/L) | 0.03 | 0.854 |
| Δ HDL cholesterol (mmol/L) | 0.05 | 0.781 |
| Δ Triacylglycerol (mmol/L) | 0.36 | <0.05 |
| Δ SBP (mm Hg) | 0.21 | 0.215 |
| Δ DBP (mm Hg) | 0.16 | 0.339 |

[1] Change was calculated as month 6 − month 1. SBP, systolic blood pressure; DBP, diastolic blood pressure.

The results of our current trial are mixed, and we must conclude that, even in men and women in whom motivation and compliance should be high, only modest weight loss can be achieved with an ad libitum low-fat, high-carbohydrate diet. It seems likely that the policy of reducing dietary fat may be more successful in preventing further weight gain rather than in treating overweight individuals. It is perhaps reassuring, however, to see that if some weight is lost, there is little evidence of worsening of the lipid profile with a high-carbohydrate diet in this high-risk group. Recommendation of a high-polysaccharide, high-fiber diet appears to be most appropriate and supports current public health practice. If total and LDL cholesterol are considered the primary and most important intermediary measures of CVD risk (24), then a reduction of >10% must be considered clinically significant. Whether these small improvements can be achieved in the public health arena, where motivation may be low, dietary compliance is poorly monitored, and there is little assurance of dietary change, remains in question.

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**APPENDIX A**

Foods available from the study grocery store for the control; low-fat, high-complex-carbohydrate (LF-CC); and low-fat, high-simple-carbohydrate (LF-SC) diets

| Food category | Control diet | LF-CC diet | LF-SC diet |
|---|---|---|---|
| Beverages | Orange juice<br>Whole-orange cordial<br>Blackcurrant cordial<br>Cola drink[1]<br>Orange soft drink[1]<br>Lemonade[1] | Orange drink, no added sugar<br>Orange cordial, no added sugar<br>Blackcurrant cordial, no added sugar<br>Diet cola drink[1]<br>Diet orange soft drink[1]<br>Diet lemonade[1]<br>Apple cordial, no added sugar | Orange juice<br>Whole-orange soft drink<br>Blackcurrant soft drink<br>Cola drink[1]<br>Orange soft drink[1]<br>Lemonade[1] |
| Crackers and cookies | Cream cracker, savory<br>Reduced-sugar cookies<br>Sweetmeal cookies<br>Plain chocolate cookies | Cream cracker, savory<br>Highbake water crackers<br>Crisp bread<br>Reduced-fat cookies<br>Thin arrowroot cookies<br>Low-fat oat cookies | Savory cracker<br>Reduced-fat cookies<br>Fruit cookies<br>Fig cookies<br>Low-fat oat cookies |
| Cakes | Genoa fruit cake | — | Jam sponge cake |
| Cereals | Muesli<br>Wheat cereal[3] | Muesli, no added sugar<br>Bran cereal[4] | High-sugar wheat cereal 1[2]<br>High-sugar wheat cereal 2[5] |
| Cheese | Red Leicester cheese<br>Matured Cheddar cheese<br>Half-fat Cheddar cheese<br>Light cream cheese | Half-fat cottage cheese<br>Half-fat cottage cheese with pineapple<br>Extra light soft cheese | Half-fat cottage cheese<br>Half-fat cottage cheese with pineapple<br>Extra light soft cheese |
| Fish | Cod fillet fish fingers<br>Canned tuna steak, brine | Cod fillets | Cod fillets |
| Fresh meat | Extra lean sausages<br>Port loin chops<br>Boneless chicken breast<br>Unsmoked, rindless bacon | Extra lean sausages<br>Pork loin chops<br>Boneless chicken breast<br>Unsmoked, rindless bacon | Extra lean sausages<br>Pork loin chops<br>Boneless chicken breast<br>Unsmoked, rindless bacon |
| Preserves | Raspberry and blackcurrant jams<br>Finecut marmalade | Reduced-sugar strawberry and blackcurrant jams<br>Reduced-sugar marmalade | Raspberry and blackcurrant jams<br>Finecut marmalade<br>Honey |
| Main meals, ready made | Leek and mushroom bake<br>Chicken and cashew with egg rice<br>Crispy Peking duck<br>Traditional cumberland pie<br>Traditional shepherd's pie<br>Chow mein[7]<br>Mini spare ribs<br>Vegetarian spring rolls<br>Macaroni and cheese<br>Lasagne, vegetarian<br>Cheddar cheese pancakes | Low-fat chicken tikka masala<br>Vegetable and pasta medley[6]<br>Vegetable lasagne[6]<br>Chicken supreme with rice[6]<br>Chicken and broccoli pasta bake[6]<br>Tagliatelle carbonara[6]<br>Chicken and prawn curry[7]<br>Tikka masala curry[7] | Low-fat chicken tikka masala<br>Vegetable and pasta medley[6]<br>Vegetable lasagne[6]<br>Chicken supreme with rice[6]<br>Chicken and broccoli pasta bake[6]<br>Tagliatelle carbonara[6]<br>Chicken and prawn curry[7]<br>Tikka masala curry[7] |
| Canned foods | Baked beans<br>Chopped tomatoes | Chopped tomatoes<br>Red kidney beans<br>Reduced-sugar baked beans | Pear halves<br>Pineapple rings<br>Peach slices |
| Pizza and pasta | Pizza, pepperoni | Spaghetti, dried<br>Lasagne, dried<br>Tagliatelli, dried<br>Fusilli tricolor, dried<br>Pizza, quatro formaggio<br>Pizzeria marinara | Pizza, quatro formaggio<br>Pizzeria marinara |
| Potatoes | Microwave, crinkle-cut French fries<br>Frozen oven French fries | Microwave, crinkle-cut French fries<br>Frozen oven French fries | Microwave, crinkle-cut French fries<br>Frozen oven French fries |
| Desserts, ready made | Deep-filled cherry pie<br>Bramley apple pie<br>Treacle tart | Rhubarb crumble<br>Apple and blueberry crumble<br>Puff pastry apple tart | Pavlova, raspberry<br>Rhubarb crumble<br>Crème caramel |

*(Continued)*

**APPENDIX A** (*Continued*)

| | | | |
|---|---|---|---|
| | Apricot and peach tart<br>Diet chocolate mousse<br>Strawberry low-fat fool<br>Strawberry trifle<br>Jam roly poly with custard<br>Italian tartufo dessert<br>Italian tiramisu dessert<br>Fruit yogurt<br>Muesli yogurt | Short-grain pudding rice<br>Vanilla ice cream[6] | Real fruit lemon sorbet<br>Mangoes, dried<br>Apricots, dried<br>Fruits of the forest yogurt<br>Duet diet strawberry yogurt<br>Diet virtually fat-free yogurt<br>Vanilla ice cream[6] |
| Confectionery | Chocolate and toffee bar[8]<br>Chocolate and malt bar[8] | Fruit muesli bar<br>Coconut low-fat chocolate bar<br>Honey low-fat chocolate bar | Fruit sweets (various)<br>Coconut low-fat chocolate bar<br>Honey low-fat chocolate bar |
| Sauces | Mayonnaise, thick and creamy<br>Tomato ketchup<br>Italian pasta sauce | Tomato ketchup, reduced sugar | Tomato ketchup<br>Italian pasta sauce |
| Soups | Cream of tomato soup | Reduced-energy tomato soup<br>Reduced-energy minestrone<br>Chicken noodle soup | Reduced-energy tomato soup<br>Reduced-energy minestrone<br>Chicken noodle soup |
| Spreads | Monounsaturated extra-light spread<br>Polyunsaturated extra-light spread | very-low-fat spread<br>extra-low-fat spread | Very low-fat spread<br>Extra low-fat spread |
| Dairy products | Cream alternative<br>Whole milk<br>Semiskim milk | Cream alternative<br>Skim milk<br>Semiskim milk | Cream alternative<br>Skim milk<br>Semiskim milk |
| Sugar | | Sweetener, powder<br>Sweetener, tabs | White sugar |

[1]Coca-Cola International, London.
[2]Frosted Shreddies; Kellogg Co Ltd, Manchester, United Kingdom.
[3]Shredded Wheat; Kellogg Co Ltd.
[4]All Bran; Kellogg Co Ltd.
[5]Frosties; Kellogg Co Ltd.
[6]Weight Watchers; HJ Heinz & Co, Middlesex, United Kingdom.
[7]Lean Cuisine; Nestle UK Ltd, Surrey, United Kingdom.
[8]Mars Bar, Milky Way; Mars UK Ltd, Norfolk, United Kingdom.