

# Effects of an ad libitum low-glycemic load diet on cardiovascular disease risk factors in obese young adults<sup>1-3</sup>

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## ABSTRACT

**Background:** The optimal nutritional approach for the prevention of cardiovascular disease among obese persons remains a topic of intense controversy. Available approaches range from conventional low-fat to very-low-carbohydrate diets.

**Objective:** The aim of this pilot study was to evaluate the efficacy of an ad libitum low-glycemic load diet, without strict limitation on carbohydrate intake, as an alternative to a conventional low-fat diet.

**Design:** A randomized controlled trial compared 2 dietary treatments in obese young adults ( $n = 23$ ) over 12 mo. The experimental treatment emphasized ad libitum consumption of low-glycemic-index foods, with 45–50% of energy from carbohydrates and 30–35% from fat. The conventional treatment was restricted in energy (250–500 kcal/d deficit) and fat (<30% of energy), with 55–60% of energy from carbohydrate. We compared changes in study outcomes by repeated-measures analysis of log-transformed data and expressed the results as mean percentage change.

**Results:** Body weight decreased significantly over a 6-mo intensive intervention in both the experimental and conventional diet groups (–8.4% and –7.8%, respectively) and remained below baseline at 12 mo (–7.8% and –6.1%, respectively). The experimental diet group showed a significantly greater mean decline in plasma triacylglycerols than did the conventional diet group (–37.2% and –19.1%, respectively;  $P = 0.005$ ). Mean plasminogen activator inhibitor 1 concentrations decreased (–39.0%) in the experimental diet group but increased (33.1%) in the conventional diet group ( $P = 0.004$ ). Changes in cholesterol concentrations, blood pressure, and insulin sensitivity did not differ significantly between the groups.

**Conclusion:** An ad libitum low-glycemic load diet may be more efficacious than a conventional, energy-restricted, low-fat diet in reducing cardiovascular disease risk. *Am J Clin Nutr* 2005;81: 976–82.

**KEY WORDS** Obesity, glycemic index, glycemic load, dietary composition, weight-reducing diet, cholesterol, triacylglycerol, plasminogen activator inhibitor 1, PAI-1, young adults

## INTRODUCTION

The alarming prevalence of obesity and the associated risk of cardiovascular disease (CVD) have been well documented (1) and extensively publicized in the United States. As a result, millions of obese adults are following weight-loss diets. Recently, Atkins-type very-low-carbohydrate diets have rapidly grown in popularity (2), although low-fat diets remain the cornerstone of conventional treatment based on clinical practice recommendations (3, 4). Whereas a few studies have suggested

that carbohydrate-restricted diets may have significantly greater benefits than do low-fat diets in reducing CVD risk (5, 6), there is widespread concern regarding the safety and long-term efficacy of severe carbohydrate restriction (7, 8).

A low-glycemic load (GL) diet, containing unrestricted amounts of carbohydrate from low-glycemic index (GI) foods, represents an alternative to low-fat diets on the one hand and to low-carbohydrate diets on the other. The GI is defined as the incremental area under the blood glucose response curve after consumption of 50 g of available carbohydrate from a test food, divided by the area under the curve after consumption of 50 g of carbohydrate from a reference food (ie, glucose or white bread) (9). The GL is the arithmetic product of the amount of carbohydrate consumed and the GI (10) and thus describes the overall effects of both quantity and source of carbohydrate on postprandial glycemia (11). Risk of CVD has been inversely associated with dietary GI or GL in some (12–15) but not all (16) epidemiologic studies. Moreover, whereas several short-term intervention studies have described beneficial effects of low-GI diets on blood lipids in overweight adults (17–20) and on the capacity for fibrinolysis in diabetic patients (21, 22), the long-term efficacy of low-GL diets in reducing CVD risk has not previously been evaluated (23).

The aim of this pilot study was to evaluate the efficacy of an experimental ad libitum low-GL diet. We hypothesized that the experimental diet would have a more beneficial effect on CVD risk factors than would a conventional, energy-restricted, low-fat diet over a 12-mo intervention.

## SUBJECTS AND METHODS

### Screening and enrollment

The protocol was approved by the institutional review board at Children's Hospital Boston, and written informed consent was obtained from each subject. Inclusion criteria included: age between 18 and 35 y, body mass index (BMI; in  $\text{kg}/\text{m}^2$ ) >27, body

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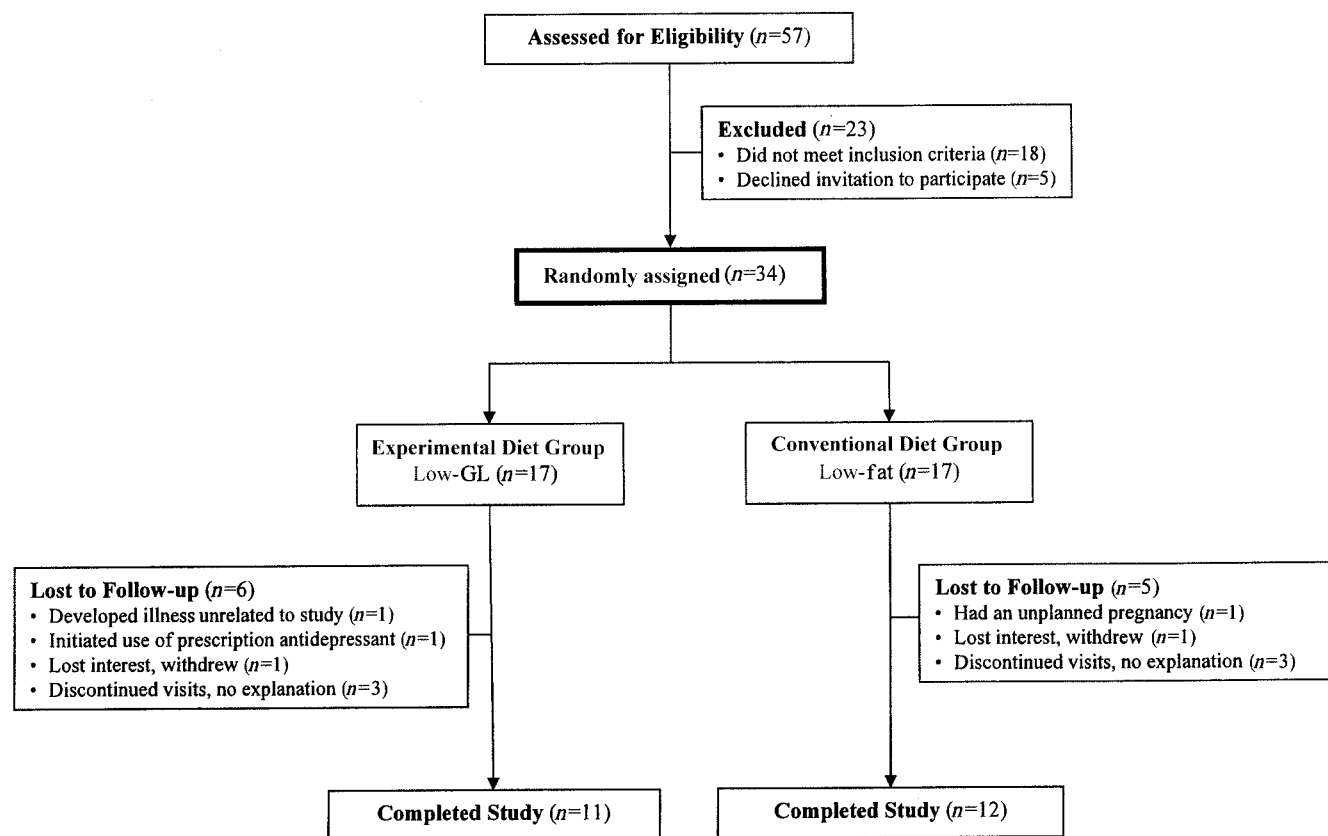


FIGURE 1. Enrollment, random assignment, and follow-up of subjects.

weight < 136 kg (300 lb), and absence of major medical illness as assessed by physical examination and laboratory screening tests (ie, kidney and liver enzymes, thyrotropin, glycosylated hemoglobin, fasting plasma glucose, and urinalysis). After being screened for eligibility, 34 obese young adults (30 females and 4 males) were enrolled in the study. Of these, 22 females and 1 male completed assessments at the end of the 12-mo intervention (Figure 1).

### Study design

Subjects were randomly assigned to the experimental (low-GL diet) or conventional (low-fat diet) treatment group between August 2001 and July 2002. The study comprised a 6-mo intensive intervention (12 dietary counseling sessions) and a 6-mo follow-up (2 dietary counseling sessions). The duration of each counseling session was 1 h. Study outcomes were measured at 0, 6, and 12 mo.

### Experimental diet

The experimental diet prescription was not energy restricted. Rather, we used an ad libitum approach based on previous research that suggested greater satiety and decreased voluntary energy intake among subjects consuming low-GL diets (24). Subjects were counseled to consume carbohydrate-containing foods with a relatively low GI (eg, nonstarchy vegetables, fruit, legumes, nuts, and dairy products; 24), to consume carbohydrate with protein and healthful fat at every meal and snack, and to eat

to satiety and snack when hungry. A low-GL food pyramid provided the basis for nutrition education (25). The target macronutrient composition was 45–50% of energy from carbohydrate, 30–35% of energy from fat, and the remainder from protein.

### Conventional diet

The conventional diet prescription was based on current recommendations for weight loss and CVD risk reduction, with emphasis on restricting energy intake by reducing dietary fat (3). Meal plans were based on an exchange system (26) designed to elicit an energy deficit of 250–500 kcal/d. Energy requirements were estimated by using the Harris-Benedict equation (27), multiplied by 1.5 to account for physical activity and adjusted for baseline dietary intake. The American Diabetes Association's diabetes food pyramid provided the basis for nutrition education (28). The target macronutrient composition was 55–60% of energy from carbohydrate, <30% of energy from fat, and the remainder from protein.

### Behavioral therapy and physical activity recommendations

Both groups received the same behavioral therapy and physical activity recommendations. Behavioral therapy focused on enhancing self-efficacy for lifestyle change by using social cognitive theory as a conceptual framework (29). Fostering behavioral capability (ie, knowledge and skill) and self-control was the primary objective during the dietary counseling sessions. Patient expectations (ie, anticipated outcomes), expectancies (ie, values

ascribed to outcomes), and perceptions of environmental influences were among the topics of discussion. To operationalize the self-control construct, the study dietitian encouraged patients to set goals around eating behaviors, to self-monitor goal attainment, and to explore solutions to problems. Physical activity recommendations were consistent with public health guidelines (30).

### Process evaluation

The intervention process was evaluated on the basis of attendance at the dietary counseling sessions and adherence to respective diet prescriptions. All subjects received extensive instruction in keeping food diaries. Three-dimensional food models, plates, bowls, glasses, and measuring cups and spoons were used to educate subjects regarding accurate appraisal of portion sizes. The diaries were reviewed with each subject at the time of receipt to provide clarification, as necessary, on recorded foods and beverages. FOOD PROCESSOR PLUS software (version 8.2; ESHA Research, Salem, OR) was used to quantify intakes of fat, carbohydrate, protein, and fiber. The GI of individual carbohydrate-containing foods was assigned according to published values based on a glucose reference (31). Daily GL was calculated by multiplying the total amount of dietary carbohydrate (in g) by the weighted GI for each food and then adjusted for energy intake:

$$\text{weighted GI} = \sum (\text{GI for food item} \\ \times \text{proportion of total carbohydrate contributed by item}) \quad (1)$$

and

$$\text{GL} = (\text{weighted GI} \times \text{grams of carbohydrate})/1000 \text{ kcal} \quad (2)$$

To ensure that treatments were delivered according to established procedures, the study dietitian completed a tracking form and progress note immediately after each session. Seven-day food diaries were obtained at baseline (month 0), during the intensive intervention period (3 and 6 mo), and at the end of follow-up (12 mo) for evaluation of process outcomes. In addition, patients were encouraged to keep food diaries throughout the intervention as a self-monitoring strategy. Patients were not given explicit information regarding the target contributions of each macronutrient to total energy intake. Rather, the study dietitian reviewed the diaries after each counseling session and provided practical advice, as necessary, to foster eating behaviors consistent with the diet prescriptions. The project director met with the study dietitian on a regular basis to review food diaries, tracking forms, and progress notes.

### Assessment of study outcomes

Weight and height were assessed by using an electronic scale (model 6702; Scale-Tronix, White Plains, NY) and a wall-mounted stadiometer (Holtain Limited, Crymych, United Kingdom), respectively. Body composition was measured by dual-energy X-ray absorptiometry (DXA) with the use of Hologic instrumentation (model QDR 4500; Hologic Inc, Bedford, MA). Blood pressure was determined by using an automated system

(Dinamapp, Tampa, FL) while the subject sat quietly. A blood sample was drawn by venipuncture after a 12-h overnight fast.

### Laboratory analyses

Plasma lipid concentrations were measured in a laboratory certified by the Centers for Disease Control and Prevention—National Heart, Lung, and Blood Institute Lipid Standardization Program. Total cholesterol, HDL cholesterol, and triacylglycerols were measured by using a Hitachi 911 analyzer (Roche Diagnostics, Indianapolis, IN), and LDL cholesterol was measured by using a homogenous enzymatic assay (Genzyme Corp, Cambridge, MA) (32). Plasma concentrations of plasminogen activator inhibitor 1 (PAI-1) were measured by using an enzyme-linked immunosorbent assay (ELISA; Diagnostica Stago, Parsippany, NJ). Plasma glucose and serum insulin concentrations were measured by using a Hitachi 917 analyzer (Roche Diagnostics) and an Elecsys 2010 system (Roche Diagnostics), respectively. With the use of glucose (mg/dL) and insulin ( $\mu\text{U}/\text{mL}$ ) concentrations, we calculated the quantitative insulin sensitivity check index:  $1/(\text{insulin} + \log \text{glucose})$  (33).

### Statistical analysis

We analyzed dietary data and study outcomes by repeated-measures analysis of variance. We tested each variable for change over time (0, 6, and 12 mo) and for a difference in time course between the 2 groups (experimental and conventional diet) by assessing the main effect of time and the group  $\times$  time interaction, respectively. To avoid the increased risk of type I inferential error from multiple comparisons, we limited statistical testing of time trends to the overall change (from 0 to 6 to 12 mo), with the exception of 2 planned comparisons for process measures, ie, GL and dietary fat. We accounted for within-subject correlation by using a banded covariance structure, which allowed a lower correlation between the 0- and 12-mo observations than between the 0- and 6-mo or the 6- and 12-mo observations. Statistical significance was defined as  $P < 0.05$ .

The primary analysis included data from only the 23 subjects who completed the study. Secondary analyses included available data from all 34 randomly assigned subjects. Study outcomes were log transformed for analysis, and results are expressed as percentage change. Dietary data were analyzed without transformation. We used SAS software (release 9.0; SAS Institute Inc, Cary, NC) for all computations.

## RESULTS

### Subjects

Baseline data for the subjects who completed the study ( $n = 23$ ; 67.6% of those randomly assigned to a treatment group) are presented in **Table 1**. There were no significant differences in baseline measures between diet groups. The male who completed the study was in the conventional diet group.

### Process data

Attendance at the 14 dietary counseling sessions approximated 100% for the 23 subjects who completed the study; 2 subjects missed just one visit each. Nutrient intake data derived from the food diaries are presented in **Table 2**. At baseline, we found no significant differences between groups with respect to

**TABLE 1**

Baseline characteristics of the experimental (low-glycemic load) and conventional (low-fat) diet groups<sup>1</sup>

Variable	Group	
	Experimental diet (n = 11)	Conventional diet (n = 12)
Age (y)	29.8 ± 1.7	27.2 ± 1.3
Weight (kg)	93.3 ± 5.3	83.2 ± 3.3
Height (cm)	165.6 ± 2.1	163.3 ± 2.1
Total cholesterol (mg/dL)	191.2 ± 9.4	186.0 ± 9.0
LDL cholesterol (mg/dL)	113.1 ± 6.1	109.4 ± 7.6
HDL cholesterol (mg/dL)	49.0 ± 2.9	53.8 ± 2.7
Triacylglycerols (mg/dL)	133 ± 17	109 ± 15
PAI-1 (ng/mL)	58.4 ± 4.9	47.5 ± 7.8
Systolic blood pressure (mmHg)	106 ± 2	105 ± 4
Diastolic blood pressure (mmHg)	64 ± 3	63 ± 2
Insulin sensitivity index	0.34 ± 0.01	0.35 ± 0.01

<sup>1</sup> All values are  $\bar{x} \pm \text{SEM}$ . PAI-1, plasminogen activator inhibitor 1. There were no significant differences between groups (independent-sample *t* test).

the nutrients of interest. GL decreased significantly in the experimental diet group (0–6 mo,  $P < 0.001$ ; 0–12 mo,  $P < 0.001$ ) and did not change in the conventional diet group. Dietary fat decreased significantly in the conventional diet group (0–6 mo,  $P < 0.001$ ; 0–12 mo,  $P = 0.004$ ) and increased nonsignificantly in the experimental diet group.

### Outcomes

Study outcomes are presented in **Table 3**. Body weight decreased significantly over the 6-mo intensive intervention in the experimental and conventional diet groups, and it remained below baseline at 12 mo. Mean weight loss did not differ significantly between the groups, and there were no significant differences between the experimental and conventional diet groups in the mean percentage change in fat mass (–16.5 compared with –15.7;  $P = 0.97$ ) and lean mass (–1.1 compared with –1.5;  $P = 0.92$ ). Nevertheless, the experimental diet group showed greater mean declines in plasma triacylglycerols. Mean changes in plasma PAI-1 concentrations also differed between the groups, decreasing in the experimental diet group and increasing in the conventional diet group. Decreases in total cholesterol and increases in HDL cholesterol were marginally nonsignificant and did not differ significantly between groups. There were no significant changes in LDL cholesterol or blood pressure in either group throughout the study. The insulin sensitivity index increased significantly in both groups. Results were materially unchanged in the secondary analyses that included data from all randomly assigned subjects (data not presented).

### DISCUSSION

In light of widespread concern regarding the high toll of the obesity epidemic on human suffering (34) and health care costs (35), development of effective weight-management strategies is a public health priority (36). Debate about the appropriate diets for promoting weight loss and decreasing CVD risk has focused largely on the metabolic effect of dietary carbohydrate and fat (37–41). Obesity has become increasingly prevalent over the last 2 decades (42), and the contribution of carbohydrate to total

energy intake has increased in tandem with a reduction in the contribution of fat (43). The increase in carbohydrate intake can be largely attributed to consumption of high-GI foods (44). Taken together, these observations suggest that both the quantity and the source of carbohydrate may be important considerations.

To our knowledge, the randomized controlled trial presented herein is the first long-term study comparing a low-GL diet, with emphasis on consumption of low-GI sources of carbohydrate, with a low-fat diet for decreasing CVD risk in obese young adults. We hypothesized that less hunger or greater satiety in response to an ad libitum low-GL diet may facilitate a decrease in energy intake (45), without the need for externally imposed energy restriction. Our hypothesis is supported, in that mean weight loss among persons following the ad libitum low-GL diet and mean weight loss among persons following the energy-restricted low-fat diet did not differ significantly during the intensive 6-mo intervention (–8.4% and –7.8%, respectively), and there was no significant weight rebound during the follow-up. Moreover, we previously observed greater decreases in BMI and fat mass among adolescents in response to a low-GL diet than in response to a low-fat diet (46). Differences in the response to an energy-restricted diet between adolescents and adults, particularly women, may partially explain the varied patterns of weight loss between our 2 studies. Adolescents have a strong desire for autonomy and seem to resist the use of an exchange system that imposes energy restriction; for this reason, the flexibility of an ad libitum approach may be especially beneficial in this age group. In contrast, many young women are accustomed to following conventional energy-restricted diets, which may limit the likelihood of seeing a group effect over a 12-mo period in this patient population. Additional studies are needed to examine group effects with longer-term follow-up. Nevertheless, our findings compare favorably with those of studies evaluating the effect of severe carbohydrate restriction on weight loss (5, 6). Foster et al (5) observed decreases in body weight of 4.4% and 2.5% at 12 mo among patients prescribed carbohydrate-restricted and low-fat diets, respectively. In a similar 12-mo study, Stern et al (6) observed decreases of 3.5% and 2.4%.

A low-GL diet, such as that used in the present study, may represent an optimal compromise between low-fat diets at one end of the spectrum and carbohydrate-restricted diets at the other. Although changes in body weight did not differ between the 2 groups, the metabolic benefits of a low-GL diet in decreasing CVD risk may be significantly greater than those achieved with either of the more restrictive approaches. The low-fat diet had a significantly less favorable effect on circulating triacylglycerol and PAI-1 concentrations than did the low-GL diet. Indeed, low-fat diets typically have a high carbohydrate content, which causes postprandial hyperglycemia and hyperinsulinemia (47). In turn, these episodes may enhance hepatic triacylglycerol production or reduce peripheral clearance (39, 48) and also promote the synthesis and secretion of PAI-1 (49) via plausible physiologic and molecular mechanisms. Attention has been directed toward controlling triacylglycerol and PAI-1 concentrations in light of the direct associations between these variables and cardiovascular events (50, 51). Whereas very-low-carbohydrate diets have beneficial effects on triacylglycerol concentrations (perhaps as a result of their low GL), the sustainability of such highly restrictive diets over the long term is questionable (5, 6). A low-GL diet,

**TABLE 2**  
Dietary data

Variable	Group		<i>P</i> <sup>1</sup>		
	Experimental diet ( <i>n</i> = 11)	Conventional diet ( <i>n</i> = 12)	Group	Time	Group × time interaction
Glycemic load (g/1000 kcal)			<0.001	<0.001	<0.001
Baseline <sup>3</sup>	77.2 ± 5.7 <sup>2</sup>	77.8 ± 2.2			
Interim <sup>4</sup>	54.4 ± 2.0	78.4 ± 1.4			
12 mo	53.0 ± 2.7	77.1 ± 2.4			
Glycemic index			0.006	<0.001	0.004
Baseline	56.2 ± 1.2	56.6 ± 1.0			
Interim	46.2 ± 1.6	52.8 ± 0.9			
12 mo	46.3 ± 2.0	52.9 ± 1.1			
Carbohydrate (% of energy)			<0.001	0.43	<0.001
Baseline	52.7 ± 2.2	54.8 ± 1.4			
Interim	47.2 ± 1.6	59.4 ± 0.8			
12 mo	45.5 ± 1.1	58.3 ± 1.9			
Total fat (% of energy)			<0.001	0.03	0.006
Baseline	32.6 ± 1.6	30.0 ± 1.1			
Interim	33.0 ± 1.2	23.4 ± 0.9			
12 mo	35.4 ± 1.2	24.3 ± 2.0			
Saturated fat (% of energy)			0.04	<0.001	0.18
Baseline	11.3 ± 0.8	10.7 ± 0.7			
Interim	9.1 ± 0.7	7.5 ± 0.3			
12 mo	10.6 ± 0.9	7.6 ± 0.7			
Protein (% of energy)			0.17	<0.001	0.07
Baseline	15.7 ± 1.0	16.1 ± 0.9			
Interim	21.1 ± 1.1	18.7 ± 0.4			
12 mo	20.5 ± 0.9	18.1 ± 1.0			
Fiber (g/1000 kcal)			0.22	<0.001	0.53
Baseline	9.6 ± 1.0	8.2 ± 0.5			
Interim	14.9 ± 1.3	12.6 ± 1.1			
12 mo	13.5 ± 1.1	12.8 ± 1.2			
Energy (kcal)			0.84	<0.001	0.76
Baseline	1860 ± 72	1802 ± 116			
Interim	1391 ± 79	1409 ± 46			
12 mo	1494 ± 82	1472 ± 85			

<sup>1</sup> Testing for overall difference in level between experimental and conventional diet groups (main effect of group), change over time (main effect of time), and difference in time course between groups (group × time interaction). Repeated-measures ANOVA was used to account for within-subject correlations.

<sup>2</sup>  $\bar{x} \pm \text{SEM}$  (all such values).

<sup>3</sup> Testing for equal means in experimental and conventional diet groups by independent-sample *t* test found no significant baseline differences for any of the listed nutrients.

<sup>4</sup> Means of data collected at 3 and 6 mo.

containing moderate amounts of carbohydrate and fat, offers a potentially more flexible approach. In contrast to very-low-carbohydrate diets, the reduction in GL in the experimental group was achieved by a relatively small decrease in carbohydrate intake that was accompanied by a substantial reduction in GI. Nevertheless, the mean decrease in triacylglycerol concentration in the experimental group over 12 mo (37.2%) compares favorably with decreases of 17.0% (5) and 28.6% (6) in previous studies of very-low-carbohydrate diets. Data from metabolic studies, epidemiologic investigations, and clinical trials lend support to the efficacy of eating patterns that are consistent with a low-GL diet (38), including consumption of vegetables, fruit, and whole grains as primary sources of carbohydrate. Moreover, our findings extend data from previous short-term studies showing beneficial reductions in triacylglycerol concentrations with low-GI diets (52–55).

Several issues pertaining to study design warrant consideration. Strengths of the study include the use of treatments of equal intensity in both experimental and control diet groups, which would eliminate this factor as a source of confounding; excellent attendance at counseling sessions among those who completed the study; a longer follow-up than in previous studies of GL or GI and CVD risk factors (17, 18, 20–22); and careful attention to process evaluation. Moreover, changes in dietary fiber, a frequently cited confounder in evaluations of GI or GL (56, 57), were similar between groups. Limitations include the self-reporting of dietary intakes for process evaluation, reliance on published GI values for calculating dietary GL (31), and a small, predominately female sample from which there was some attrition. Underreporting of dietary intake is a well-recognized phenomenon in all outpatient studies aiming to assess the effects of

**TABLE 3**  
Study outcomes<sup>1</sup>


Variable	Group		P <sup>2</sup>		
	Experimental diet (n = 11)	Conventional diet (n = 12)	Group	Time	Group × time interaction
Weight			0.18	<0.001	0.89
Interim <sup>3</sup>	−8.4 (−11.4, −5.3)	−7.8 (−10.7, −4.9)			
12 mo	−7.8 (−13.0, −2.2)	−6.1 (−11.2, −0.7)			
Total cholesterol			0.90	0.06	0.22
Interim	−9.9 (−16.7, −2.5)	−2.1 (−9.2, 5.5)			
12 mo	−8.5 (−17.4, 1.5)	−6.2 (−15.0, 3.5)			
LDL cholesterol			0.85	0.17	0.59
Interim	−9.1 (−18.6, 1.4)	−2.6 (−12.3, 8.2)			
12 mo	−9.7 (−21.6, 3.9)	−7.4 (−19.1, 6.0)			
HDL cholesterol			0.41	0.08	0.20
Interim	2.3 (−6.0, 11.3)	−0.3 (−8.1, 8.2)			
12 mo	12.2 (2.9, 22.3)	1.1 (−6.9, 9.8)			
Triacylglycerols			0.96	<0.001	0.005
Interim	−35.4 (−44.6, −24.7)	−7.1 (−19.8, 7.6)			
12 mo	−37.2 (−47.7, −24.5)	−19.1 (−32.2, −3.6)			
PAI-1			0.78	0.11	0.004
Interim	−58.3 (−74.7, −31.3)	30.4 (−19.2, 110.4)			
12 mo	−39.0 (−70.2, 24.9)	33.1 (−32.9, 164.3)			
Systolic blood pressure			0.78	0.81	0.99
Interim	−0.9 (−5.9, 4.2)	−0.5 (−5.3, 4.4)			
12 mo	0.2 (−4.7, 5.3)	0.6 (−4.1, 5.5)			
Diastolic blood pressure			0.84	0.72	0.82
Interim	−2.0 (−7.2, 3.4)	0.3 (−4.8, 5.6)			
12 mo	−0.3 (−6.2, 6.0)	1.4 (−4.4, 7.6)			
Insulin sensitivity index			0.32	<0.001	0.94
Interim	6.4 (1.5, 11.5)	5.8 (1.1, 10.7)			
12 mo	10.4 (3.6, 17.6)	8.7 (2.3, 15.5)			

<sup>1</sup> Mean change in log-transformed variable at 6 and 12 mo (b), retransformed to percentage change [ $100\% \times (\exp(b) - 1)$ ], with 95% confidence limits. Repeated-measures ANOVA was used to account for within-subject correlations.

<sup>2</sup> Testing for overall difference in level between experimental and conventional groups (main effect of group), change over time (main effect of time, 2 df), and difference in time course between groups (group × time interaction, 2 df).

<sup>3</sup> Data collected at 6 mo.

diet composition on weight loss, although adjustment of other dietary variables for energy intake may partially correct for underreporting. When calculating GL from self-report data, we relied on published GI values (31), many which were derived from studies conducted in countries where foods may differ from those consumed in the United States. An attrition rate of 32.4%, although considered problematic in terms of drawing unbiased conclusions (58), is similar to rates observed in previous long-term dietary intervention studies (5, 6).

In conclusion, a low-GL diet containing moderate amounts of carbohydrate from low-GI sources may be more efficacious than a conventional low-fat diet in reducing CVD risk. The greater benefits in response to an ad libitum diet, compared with an energy-restricted diet, are particularly noteworthy. This pilot study provides a rationale for conducting long-term, larger-scale studies comparing the effects of low-GL, low-fat, and very-low-carbohydrate diets on CVD risk among obese persons. 

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All authors contributed to the interpretation of results. CBE and DSL designed the study, provided oversight, and wrote the manuscript. MML was responsible for dietary counseling. KBS and LGS conducted the process

evaluations to assess adherence to diet prescriptions. HAF provided consultation on statistical analysis of the data. None of the authors had any personal or financial conflict of interest.

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