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Targeting Dietary Fat or Glycemic Load in the Treatment of Obesity and Type 2 Diabetes: A Randomized Controlled Trial

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Abstract

Aims—To compare the effects of lifestyle modification programs that prescribe low-glycemic load (GL) vs. low-fat diets in a randomized trial.

Methods—Seventy-nine obese adults with type 2 diabetes received low-fat or low-GL dietary instruction, delivered in 40-week lifestyle modification programs with identical goals for calorie intake and physical activity. Changes in weight, HbA_{1c}, and other metabolic parameters were compared at weeks 20 and 40.

Results—Weight loss did not differ between groups at week 20 (low-fat: −5.7 ± 3.7%, low-GL: −6.7 ± 4.4%, $p = .26$) or week 40 (low-fat: −4.5 ± 7.5%, low-GL: −6.4 ± 8.2%, $p = .28$). Adjusting for changes in antidiabetic medications, subjects on the low-GL diet had larger reductions in HbA_{1c} than those on the low-fat diet at week 20 (low-fat: −0.3 ± 0.6%, low-GL: −0.7 ± 0.6%, p = .01), and week 40 (low-fat: −0.1 ± 1.2%, low-GL: −0.8 ± 1.3%, $p = .01$). Groups did not differ significantly on any other metabolic outcomes ($p \geq .06$).

Conclusions—Results suggest that targeting GL, rather than dietary fat, in a low-calorie diet can significantly enhance the effect of weight loss on HbA_{1c} in patients with type 2 diabetes.

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Conflict of Interest
The authors have a competing interest to declare.

Keywords

Glycemic Load; Dietary Fat; Obesity; Type 2 Diabetes; Lifestyle Modification; Diet

Introduction

Current guidelines recommend a program of diet, exercise, and behavior therapy (i.e., lifestyle modification) for the treatment of obesity [1]. Such programs typically instruct patients to consume a low-calorie diet that provides ~50-60% of energy from carbohydrate, ≤ 30% from fat, and the remainder from protein. Although the abundance of carbohydrate may be of concern to persons with diabetes and their care providers, numerous studies have shown that diabetic participants in standard lifestyle modification programs (which typically prescribe a low-fat, high-carbohydrate diet) achieve significant mean improvements in blood glucose and glycated hemoglobin (HbA_{1c}) [2-3].

Dietary approaches to weight loss and diabetes control may target glycemic index (GI) or glycemic load (GL) as an alternative to standard low-fat diet prescriptions. The findings regarding the health effects of these alternative approaches, however, are mixed. Some laboratory tests have found foods higher in GI or GL to be related to poorer short-term metabolic outcomes, as well as greater hunger, less satiety, and greater subsequent food intake [4-6]. Others have produced null findings [7-8] or inverse associations [9].

Results of individual clinical trials and epidemiological studies also have produced equivocal findings regarding the relationship of GI and GL to metabolic parameters [10-13], cardiovascular disease risk factors [13-15], and weight loss [16-19]. A recent meta-analysis of randomized controlled trials found a statistically significant, though modest, difference favoring low-GI/GL diets over higher-GI/GL alternatives for weight loss in non-diabetic participants [20]. An earlier meta-analysis found that subjects with type 2 diabetes achieved a 6% greater reduction in glycated proteins (HbA_{1c} and fructosamine) on low-GI vs. high-GI diets [21]. These studies were typically short-term and were not intended to induce weight loss through lifestyle modification.

The present study compared the effects of low-fat and low-GL diets, delivered in the context of a lifestyle modification program for weight loss in participants with type 2 diabetes mellitus. Co-primary outcomes were changes in weight and HbA_{1c}, and secondary outcomes included changes in several additional metabolic parameters and dietary intake variables.

Methods

Trial Design and Study Setting

This was a single-site parallel-group study with balanced (1:1) randomization. Given the nature of the interventions, blinding to treatment condition was not possible. The study was conducted at the University of Pennsylvania Center for Weight and Eating Disorders in Philadelphia, PA, from September 2006 to July 2009.

Participants

Men and women, ages 18-65 years, with a diagnosis of type 2 diabetes and a BMI of 27 to 45 kg/m^2 (maximum weight of 136 kg) were eligible to participate. Individuals with type 1 diabetes, uncontrolled hypertension (> 160/100 mm Hg) or thyroid disease, unstable angina, malignant arrhythmias, myocardial infarction in the past year, cancer (active or in remission < 5 years), clinically significant psychosocial impairment, or any history of cerebrovascular, renal, hepatic, or protein-wasting diseases were excluded. Additionally, women who were

pregnant or lactating were excluded from the trial. The research was approved by the Institutional Review Board at the University of Pennsylvania School of Medicine and is registered with ClinicalTrials.gov (NCT00729196). Participants were not charged for treatment, and they were not paid for their participation in the trial.

Seventy-nine individuals (63 women, 16 men) were enrolled in six cohorts and randomly assigned, using a computer-generated scheme, to a lifestyle modification program that included a low-fat or low-GL diet prescription. Twenty-nine participants did not complete treatment (See Figure 1 for participant flow). Attrition did not differ between groups ($p = .54$).

Interventions

For both the low-fat and low-GL conditions, the dietary prescription was delivered as a component of a lifestyle modification program, which also included an exercise prescription and group-based behavior therapy sessions. Behavior therapy sessions included four to eight participants, lasted approximately 90 minutes each, and were held weekly for 20 weeks and biweekly for 20 additional weeks. Both conditions were taught the same set of behavioral and cognitive skills (e.g., self-monitoring, stimulus control, problem-solving, challenging dysfunctional thoughts). Goals for energy intake were 5024-6280 kJ/d and 6280-7536 kJ/d for participants who weighed < 113.4 kg and ≥ 113.4 kg, respectively. Participants were provided with a calorie-counting guide [22] to assist in meeting energy intake goals. The physical activity prescription was identical in the two conditions; participants initially were instructed to complete at least 50 minutes of moderate-intensity activity (e.g., brisk walking) per week, and to increase to at least 175 minutes per week over the first 20 weeks of treatment. Interventionists had doctoral- or masters-level training in clinical psychology. (Masters-level clinicians were supervised by a licensed psychologist with extensive experience in lifestyle modification for weight loss.) Within each randomization cohort, the same interventionist provided treatment to the low-fat and low-GL conditions.

Low-fat condition—Participants randomized to the low-fat condition received instruction on identifying sources of dietary fat and were encouraged to model their diet on a “Low-Fat Pyramid” (similar to the Food Guide Pyramid). They were prescribed a goal of consuming ≤ 30% of energy from fat (i.e., 40-50 g/d and 50-60 g/d for participants in the 5024-6280 kJ/d and 6280-7536 kJ/d ranges, respectively). Participants recorded calorie and fat gram intake in their daily self-monitoring logs. Low-fat recipes were distributed and low-fat items and meals were sampled in session. Participants also received a low-fat eating plan (i.e., menus, recipes, and grocery lists) for 2 weeks' worth of meals and snacks at an average of ~6280 kJ and 30 grams of fat per day.

Low-GL condition—Participants in this condition received instruction on the glycemic effects of food. They were given a “Low-GL Pyramid” and were encouraged to structure their diets accordingly. Rather than computing GL values for the foods they consumed, they were taught several guidelines for identifying low-, moderate-, and high-GL items [23]. Participants in this condition were prescribed goals of consuming ≤ 3 and ≤ 1 serving per day of moderate-GL and high-GL items, respectively. They recorded servings of moderate- and high-GL foods, as well as calorie intake, in their daily self-monitoring logs. Participants received recipes and sampled foods that were consistent with the dietary goals of the low-GL condition. In addition, participants were given a low-GL eating plan that provided an average of ~6280 kJ, 3 servings of moderate-GL foods, and < 1 serving of high-GL foods per day over 2 weeks.

Outcome Measures

Study outcomes were assessed at baseline, week 20, and week 40. (In addition, weight was measured, for feedback purposes, at each lifestyle modification session.)

Anthropometric measures—Weight was measured on a calibrated electronic scale (Tanita BEB-800, Tokyo, Japan) with participants in light clothing and no shoes. Height was measured (at baseline) with a wall-mounted stadiometer. Waist circumference was measured at the umbilicus with the participant standing and the tape measure parallel to the floor. Blood pressure was measured with an automated sphygmomanometer (Dinamap Pro 100, GE Healthcare, Waukesha, WI), after participants were seated for at least 5 minutes. Height, waist circumference, and blood pressure were each measured in duplicate and recorded as the mean of the two values.

Biochemical measures—Blood samples were collected after an overnight fast and were analyzed at the Clinical and Translational Research Center (CTRC) at the University of Pennsylvania School of Medicine using standard procedures for: HbA_{1c}; glucose; insulin; c-peptide; total, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) cholesterol; triglycerides; and high-sensitivity C-reactive protein. Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated from fasting glucose and insulin levels [24]. Medication use was tracked throughout the study and changes in antidiabetic medications were quantified as follows: new medication or increased dosage from baseline (+1); no change in medications or dosages from baseline (0); or discontinued medication or decreased dosage from baseline (−1).

Dietary measures—Participants were instructed to keep 3-day food records (2 weekdays and 1 weekend day) prior to each assessment visit. A research dietitian at the CTRC Bionutrition Core reviewed records for plausibility, queried participants when appropriate, and analyzed records using Nutrition Data Systems for Research software [25].

Statistical Analyses

Descriptive statistics were generated to identify the distribution of scores and the need for transformation. Unless noted otherwise, the distribution of all variables was sufficiently normal. Groups were compared at baseline using t-tests for continuous data and chi-square tests for categorical data. The co-primary outcome variables were changes in weight and HbA_{1c}, which were compared between groups at weeks 20 and 40. Weight change was defined as a percentage of baseline body weight.

Hierarchical linear modeling (HLM) with full maximum likelihood estimation was used to investigate changes in weight and HbA_{1c}, as well as other metabolic parameters and dietary intake variables over time. This approach allows for participants with at least two longitudinal observations to be included in the analysis, thereby allowing participants with partial data to contribute to the analysis. Unconditional models were fitted to test for linear and non-linear (quadratic) patterns in the outcomes, and to determine whether slopes should be treated as random effects. Covariates (i.e., treatment group, age, and gender) were then added to these models to evaluate their effect. The model of HbA_{1c} change also included change in diabetes medication (increase, decrease, or no change) as a time-varying covariate. Continuous predictors were mean-centered (around the group mean) before entry into the analysis. Modeled means and standard errors were used to evaluate differences in outcomes between treatment groups at 20 and 40 weeks. SPSS version 17 [26] was used to complete all analyses.

Results

Baseline demographic, clinical, and dietary intake characteristics for participants in the low-fat and low-GL conditions are shown in Table 1. Groups did not differ significantly on any variables. Values for continuously distributed data are presented as group mean ± standard error.

Weight Change

As shown in Figure 2 (Panel A), mean reductions in body weight were not significantly different between groups. Participants in the low-fat and low-GL groups lost 5.7 ± 0.6% and 6.7 ± 0.7% of initial weight, respectively, at week 20 ($p = .26$). At week 40, reductions were 4.5 ± 1.2% and 6.4 ± 1.3%, respectively ($p = .28$).

Glycemic Control

Comparisons of between-groups changes in HbA_{1c}, controlling for changes that participants' physicians made to their diabetes medications, are shown in Figure 1 (Panel B). At week 20, participants in the low-fat and low-GL groups achieved reductions in HbA_{1c} of 0.3 ± 0.1% and 0.7 ± 0.1%, respectively ($p = .01$). The difference between groups was greater at week 40, with participants in the low-fat and low-GL groups achieving reductions of 0.1 ± 0.2% and 0.8 ± 0.2%, respectively ($p = .01$). As shown in Figure 2, the percentage of participants who increased, decreased, or did not change the intensity of their diabetes medication regimen did not differ between groups at week 20 ($p = .51$) or week 40 ($p = .70$).

Metabolic Outcomes

Changes in metabolic markers also were compared at weeks 20 and 40. As shown in Table 2, changes in systolic and diastolic blood pressure were marginally more favorable among participants in the low-fat vs. low-GL group at weeks 20 and 40 ($p = .06$ to $.08$). There were no between-groups differences in changes in fasting glucose, insulin, HOMA-IR, c-peptide, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, or high-sensitivity C-reactive protein at week 20 or week 40 ($p \geq .15$).

Dietary Intake

Changes in dietary intake are presented in Table 3. Reductions in energy from fat were significantly greater among those in the low-fat group at weeks 20 and 40 ($p \leq .01$). By contrast, those in the low-GL group had significantly greater reductions in energy from carbohydrate ($p \leq .01$), and significantly greater reductions in dietary GI ($p \leq .003$) and GL ($p \leq .03$), at both time points. Changes on other measured dietary variables did not differ significantly between groups at week 20 or 40.

Discussion

This study found significantly larger reductions in HbA_{1c} in overweight and obese patients with type 2 diabetes who were prescribed a low-GL diet, versus an isoenergetic low-fat diet, as part of a lifestyle modification program. The advantage for diabetes control was observed despite lack of significant differences in body weight, fasting glucose, and dietary fiber between the two groups. This finding suggests that the quality and quantity of carbohydrate consumed play an important role in broad glycemic exposure among patients with type 2 diabetes, even in a state of negative energy balance necessary for concomitant weight loss.

The effect of reducing GI or GL on glycemic control in persons with diabetes has been summarized in several meta-analyses [21,27-28]. However, the studies included in those analyses differed notably from the present trial. In the Cochrane review by Thomas and

Elliot, for instance, only 5 of the 11 included studies tested diets for at least 12 weeks [28]. Of those, only two were of participants with type 2 diabetes; they were both cross-over studies with a total of only 22 subjects between them. The meta-analysis found that low-GI or low-GL diets were associated with reductions in HbA_{1c} that were approximately 0.3% to 0.5% greater than with higher-GI or GL alternatives [28]. In the present study, we found that the advantage for the low-GL diet was 0.4% at the midpoint of treatment (i.e., week 20) and 0.7% at the conclusion. The widening gap between groups was more attributable to an increase in HbA_{1c} (from week 20 to 40) among those in the low-fat group, than to a continued reduction in the low-GL group.

A recent 12-month randomized controlled comparison of low-GI, high-GI, and low-carbohydrate diets in patients with type 2 diabetes found no differences among groups in HbA_{1c} [29]. Furthermore, HbA_{1c} was *higher* in all groups at study's end than at baseline. Similarly, another trial found no significant change in HbA_{1c} at 1 year among patients with type 2 diabetes who were assigned to follow a low-carbohydrate (and by extension, low-GL) or low-fat diet [30]. Both of those studies, however, offered far less frequent and intense clinical contacts than the level of treatment provided in the present study.

Studies of lifestyle modification in participants with type 2 diabetes typically show that changes in HbA_{1c} track with changes in weight in this population [31]. Thus, diminished improvements in HbA_{1c} are expected when patients with type 2 diabetes reach a weight loss plateau or begin to regain weight. The low-calorie diet that is typically prescribed, however, is a low-fat diet. Without explicit instruction to choose low-GI carbohydrates, participants who follow a low-fat (i.e., high-carbohydrate) diet may be inadvertently following an eating plan that puts them at risk for increasing glycemic exposure when they cease to create an energy deficit. That participants in our low-GL group maintained their HbA_{1c} reduction in full, in the absence of additional weight loss in the second half of the intervention, suggests that they continued to adhere to the principles of low-GL eating, even as treatment became less frequent.

In contrast to the significant HbA_{1c} advantage for the low-GL diet, the between-groups differences in weight loss (~ 1 and 2 kg at weeks 20 and 40, respectively) were not statistically significant and were consistent with those reported previously. A meta-analysis of six randomized controlled trials found that low-GI/GL diets induced a mean weight loss that was 1 kg greater than that achieved with higher-GI/GL alternatives in healthy participants [20]. We had hypothesized a significant difference in weight loss, in part, due to findings that the reduction in metabolic rate after a 10% weight loss was significantly but modestly smaller with a low-GL diet, compared with a low-fat diet [32]. Additionally, feeding laboratory-based findings that lower-GL meals are associated with greater satiety and less energy intake at subsequent meals suggested that consumption of low-GL foods would facilitate adherence to a low-calorie diet [4-6]. Whether those mechanisms were engaged in the present study is unknown. However, the study is underpowered to detect changes in body weight that might result from relatively subtle effects on metabolic rate, hunger, or satiety, especially when calorie prescriptions were identical in both groups.

At least two studies, published after the launch of the present one, found that insulin secretion rates – as assessed by insulin concentration 30 minutes after oral glucose consumption – moderated the effect of a low-GL diet on weight loss. Pittas et al. compared weight loss achieved with isocaloric low- and high-GL provided diets in healthy overweight participants [33]. Although there was no effect of diet on weight loss for those with low insulin secretion, the low-GL diet produced a significantly greater reduction in weight for those with a high insulin response. Similarly, Ebbeling et al. found no main effect of diet (low-GL vs. low-fat) on weight loss among obese young adults [34]. However, a significant

effect was apparent among those with high baseline insulin secretion; the low-GL and low-fat diets produced weight losses of 5.8 kg and 1.2 kg, respectively, at 18 months. In both of the studies described above, participants were overweight or obese, but with no history of type 2 diabetes. It is unclear whether a similar moderating effect would have been found in the present study had we conducted an oral glucose tolerance test at baseline.

Several previous studies that examined the effects of low-GL diets provided diet-consistent food to participants. We instead opted to instruct participants in how to reduce the GL of diets they selected autonomously. This approach has the advantage of external validity, as long-term food provision is not practical for the long-term management of obesity and type 2 diabetes. The disadvantage of providing instruction, rather than food, is that adherence to the diet is likely less complete. We note, however, that between-groups differences in dietary intake (as measured by analysis of 3-day food records) were consistent with expectations based on the dietary instruction that each group received. That is, participants in the low-fat group reduced fat intake to a greater extent than did those in the low-GL group, who in turn, reduced carbohydrate intake, GI, and GL to a greater degree than their low-fat counterparts.

Aside from the significant advantage of the low-GL diet for reducing HbA_{1c}, there were no other significant differences between groups in other metabolic outcomes, including fasting glucose. This finding highlights the contribution of postprandial glycemic exposure to overall diabetes control. Both systolic and diastolic blood pressure improved to a marginally greater degree in the low-fat group at weeks 20 and 40, compared to the low-GL group. Examination of group means revealed a modest increase in blood pressure among participants in the low-GL group. This finding is difficult to interpret. We did not control for changes in antihypertensive medications, sodium intake, or other factors that may explain the observed differences in blood pressure.

A weakness of this study is the attrition rate of 36.7%. We suspect that dissatisfaction with treatment (including with initial weight loss) contributed to attrition, particularly among those who dropped out before week 20 (24.1%) or were lost to follow-up (16.5%). In addition, the lack of financial compensation provided little incentive for participants who achieved unsatisfactory results to return for assessments. Our greatest protection against the effects of attrition was the use of mixed models for analyses. This approach is considered superior to alternative methods of handing missing data such as including only participants with complete data or assigning a pre-specified score to the missing data. A major strength of the trial includes the long duration and large sample size relative to most previous comparably designed studies. An additional strength was the incorporation of each diet into a standard-format lifestyle modification program with identical energy intake goals, exercise prescriptions, and behavioral interventions in both conditions.

The current position of the American Diabetes Association [35] is that “the use of the glycemic index and glycemic load may provide a modest additional benefit for glycemic control over that observed when total carbohydrate is considered alone” (Evidence grade B: supportive evidence from well-conducted cohort studies.) The present study was not designed to address this issue directly, as the comparison diet was a higher-GL low-fat diet. Future investigations should compare low- and high-GI/GL diets that control for total carbohydrate intake.

This study demonstrated that prescribing a calorie-restricted low-GL diet to overweight and obese adults with type 2 diabetes resulted in greater glycemic control than was achieved with an isoenergetic low-fat prescription. The advantage of the low-GL diet for improving HbA_{1c} was apparently not attributable to weight change or calorie-restriction, as these were

equivalent between groups. These results add to a growing literature on the benefits of following a low-GL diet for diabetes control.

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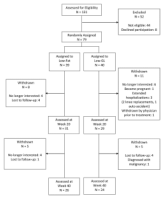

Figure 1.
Participant flow through the trial.

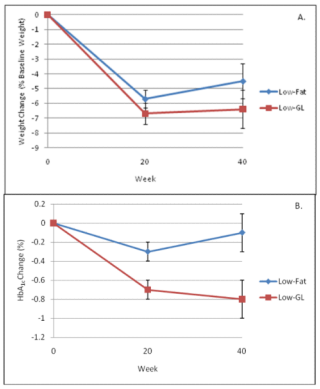

Figure 2.

Mixed model estimates showing effects of low-fat and low-GL diets on weight change (Panel A) and HbA_{1c} (Panel B). Differences in weight loss were not statistically significant at week 20 or week 40. However, the changes in HbA_{1c} were significantly different at both times.

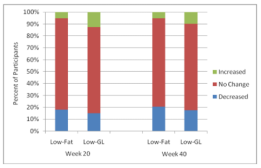

Figure 3.

Comparisons of the percentage of participants in each condition who increased, did not change, or decreased the number or dosage of medications used to treat diabetes during the course of the study. Chi-square analyses showed no differences between groups at week 20 or week 40.

Table 1

Baseline characteristics of participants.

	Low-Fat (n = 39)	Low-GL (n = 40)	p
Female sex – n (%)	31 (79.5%)	32 (80.0%)	.96
Ethnicity – n (%)			.60
Caucasian	16 (41.0%)	17 (42.5%)	
African American	17 (43.6%)	21 (52.5%)	
Other/Not Reported	6 (15.4%)	2 (5.0%)	
Age (y)	52.5 ± 1.3	52.8 ± 1.4	.87
Weight (kg)	99.1 ± 2.3	102.3 ± 2.7	.37
BMI (kg/m^2)	35.8 ± 0.7	36.7 ± 0.8	.44
HbA_{1c} (%)	7.0 ± 0.2	6.6 ± 0.2	.20
Waist circumference (cm)	111.6 ± 1.6	114.2 ± 1.7	.25
Glucose (mg/dl)	124.8 ± 7.3	112.2 ± 6.4	.20
Insulin (mIU/L)	13.2 ± 1.5	15.8 ± 1.7	.26
HOMA	4.2 ± 0.6	4.4 ± 0.5	.83
Total cholesterol (mg/dl)	171.2 ± 5.1	183.9 ± 5.6	.10
LDL cholesterol (mg/dl)	90.7 ± 4.4	99.9 ± 5.1	.18
HDL cholesterol (mg/dl)	54.3 ± 2.2	56.7 ± 2.7	.49
Triglycerides (mg/dl)	131.9 ± 10.0	141.8 ± 15.8	.60
Systolic blood pressure (mm Hg)	128.6 ± 2.3	130.1 ± 2.4	.66
Diastolic blood pressure (mm Hg)	71.2 ± 1.5	71.4 ± 1.6	.93
High sensitivity C-reactive protein (mg/L)	7.5 ± 1.8	8.0 ± 1.3	.83
c-peptide (ng/mL)	2.8 ± 0.3	3.3 ± 0.2	.15
Total energy (kJ)	7812.2 ± 404.0	8403.7 ± 416.6	.33
Energy from fat (% kJ)	37.0 ± 1.0	38.2 ± 1.1	.44
Energy from carbohydrate (% kJ)	46.0 ± 1.1	44.0 ± 1.2	.22
Energy from protein (% kJ)	17.6 ± 0.6	18.8 ± 1.1	.35
Total sugar (g)	82.4 ± 6.2	78.4 ± 5.6	.65
Added sugar (g)	57.7 ± 5.8	52.2 ± 5.4	.51
Fiber (g)	16.1 ± 1.2	16.4 ± 1.1	.86
Dietary glycemic index	63.4 ± 0.7	63.6 ± 0.8	.81
Dietary glycemic load	129.3 ± 8.0	129.0 ± 7.2	.98

For continuous variables, cells contain group means ± standard error. P-values are for the between-groups difference.

Table 2

Changes in Metabolic Outcomes at Weeks 20 and 40.

Variable	Low-Fat (n = 39)	Low-GL (n = 40)	p
Waist Circumference (cm)			
Week 20	−6.2 ± 0.7	−6.6 ± 0.7	.662
Week 40	−6.4 ± 1.4	−7.1 ± 1.4	.721
Glucose (mg/dl)			
Week 20	−7.9 ± 4.4	−10.8 ± 4.6	.624
Week 40	−10.4 ± 8.2	−17.0 ± 8.5	.570
Insulin (mIU/L)			
Week 20	−3.2 ± 1.2	−3.6 ± 1.2	.801
Week 40	−4.6 ± 2.2	−5.5 ± 2.3	.770
HOMA			
Week 20	−1.2 ± 0.4	−1.2 ± 0.4	.979
Week 40	−1.7 ± 0.7	−1.8 ± 0.7	.969
Systolic Blood Pressure (mmHg)			
Week 20	−4.2 ± 1.9	0.6 ± 2.0	.068
Week 40	−4.5 ± 3.6	4.6 ± 3.7	.078
Diastolic Blood Pressure (mmHg)			
Week 20	−2.7 ± 1.1	0.2 ± 1.2	.059
Week 40	−3.0 ± 2.1	2.8 ± 2.2	.057
Triglycerides (mg/dl)			
Week 20	−17.2 ± 8.0	−29.9 ± 8.3	.246
Week 40	−3.0 ± 15.0	−29.3 ± 15.6	.223
Total Cholesterol (mg/dl)			
Week 20	−9.4 ± 4.0	−17.3 ± 4.2	.147
Week 40	2.7 ± 7.5	−10.8 ± 7.8	.209
LDL Cholesterol (mg/dl)			
Week 20	−1.3 ± 3.3	−6.4 ± 3.5	.256
Week 40	3.1 ± 6.2	−5.5 ± 6.3	.328
HDL Cholesterol (mg/dl)			
Week 20	−3.9 ± 1.0	−4.8 ± 1.1	.544
Week 40	0.0 ± 1.9	−1.3 ± 2.0	.641
High Sensitivity C-Reactive Protein (mg/L)			
Week 20	−3.3 ± 1.2	−2.8 ± 1.2	.744
Week 40	−3.3 ± 2.2	−2.6 ± 2.3	.828
C-Peptide (ng/mL)			
Week 20	−0.1 ± 0.2	−0.2 ± 0.2	.739
Week 40	−0.4 ± 0.3	−0.6 ± 0.3	.581

Cells contain group means ± standard error. P-values are for the between-groups difference.

Table 3

Changes in Dietary Intake at Weeks 20 and 40

Variable	Low-Fat (n = 39)	Low-GL (n = 40)	p
Total energy (kJ/d)			
Week 20	−2223.2 ± 316.5	−2745.7 ± 319.5	.208
Week 40	−800.1 ± 562.3	−1810.0 ± 586.2	.215
Energy from fat (% kJ)			
Week 20	−7.4 ± 0.9	−4.3 ± 0.9	.007
Week 40	−4.1 ± 1.5	1.6 ± 1.6	.010
Energy from carbohydrate (% kJ)			
Week 20	5.6 ± 1.0	2.0 ± 1.1	.007
Week 40	3.8 ± 1.7	−2.7 ± 1.8	.010
Energy from protein (% kJ)			
Week 20	2.3 ± 0.9	2.5 ± 0.9	.903
Week 40	1.3 ± 1.7	1.6 ± 1.7	.917
Total Sugar (g)			
Week 20	−12.1 ± 4.5	−17.0 ± 4.5	.417
Week 40	4.2 ± 8.3	−8.0 ± 8.6	.308
Added Sugar (g)			
Week 20	−17.0 ± 4.0	−22.3 ± 4.1	.311
Week 40	−2.3 ± 7.2	−14.9 ± 7.5	.229
Fiber (g)			
Week 20	0.0 ±1.1	0.6 ± 1.1	.676
Week 40	0.2 ± 2.1	1.6 ± 2.2	.648
Dietary glycemic index			
Week 20	−2.6 ± 0.9	−6.5 ± 1.0	.002
Week 40	1.3 ± 1.7	−6.2 ± 1.7	.003
Dietary glycemic load			
Week 20	−33.0 ± 5.7	−49.9 ± 5.7	.027
Week 40	−8.0 ± 10.2	−40.4 ± 10.6	.031

Cells contain group means ± standard error. P-values are for the between-groups difference.