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A Randomized Clinical Trial Comparing Low-Glycemic Index versus ADA Dietary Education among Individuals with Type 2 Diabetes

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Abstract

Objective—To compare the effects of a low-glycemic index (GI) diet to the American Diabetes Association (ADA) diet on glycosylated hemoglobin (HbA1c) among individuals with type 2 diabetes.

Subjects/Methods—Forty individuals with poorly controlled type 2 diabetes were randomized to either a low-GI or an ADA diet. The intervention, consisting of eight educational sessions (monthly for the first six months and then at months 8 and 10), focused on either a low-GI or an ADA diet. Data on demographic, diet, physical activity, psychosocial factors, and diabetes medication use were assessed at baseline, and 6- and 12-months. Generalized linear mixed models were used to compare the two groups on HbA1c, diabetic medication use, blood lipids, weight, diet, and physical activity.

Results—Participants (53% female; mean age= 53.5 years) were predominantly white with mean body mass index of 35.8 kg/m^2. While both interventions achieved similar reductions in mean HbA1c at 6 months and at 12 months, the low-GI diet group was less likely to add or increase dosage of diabetic medications (odd ratio=0.26, p=0.01). Improvements in HDL cholesterol, triglycerides, and weight loss were similar among groups.

Conclusions—Compared to the ADA diet, the low-GI diet achieved equivalent control of HbA1c using less diabetic medication. Despite its limited size, this trial suggests that low-GI diet is a viable alternative to ADA diet. Findings should be evaluated in a larger randomized controlled trial.

Keywords

Glycemic index; carbohydrates; diabetes mellitus; type 2; randomized clinical trial

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INTRODUCTION

Diabetes can be associated with serious complications. Microvascular complications such as retinopathy, nephropathy, and neuropathy are believed to result from chronically elevated blood glucose levels [1–4]. Additionally, recent evidence suggests a clear effect of glycemic control on macrovascular complications such as coronary heart disease and stroke, which are the primary causes of death in persons with diabetes [5]. These devastating complications are, to a large extent, preventable through the improvement of glycemic control [6,7].

Dietary management is the cornerstone of care for diabetes, and carbohydrate intake has the greatest influence on blood glucose. Based on the American Diabetes Association (ADA) recommendations [8–10], carbohydrates should provide 45 to 65 percent of total energy intake. The ADA diet which emphasizes carbohydrate counting (grams of carbohydrate) and even distribution (timing) of daily carbohydrate intake is currently recommended for patients with diabetes as the mainstay of glycemic control.

Carbohydrate types and their glycemic responses have been classified by the glycemic index. The glycemic index (GI) of a food is defined as the glucose response during a 2-hour period following consumption of 50g of carbohydrate from the specific test food, divided by the glucose response after consumption of 50g of carbohydrate from a control food, which generally is either white bread or glucose [11]. Glycemic load (GL) is a calculation of the GI value of a food multiplied by its total available carbohydrate content.

A recent meta-analysis of randomized clinical trials (RCT) [12] suggests that low-GI diet has a moderate effect on improving short-term glycemic control in diabetic patients [12]. However, in most of the reviewed RCTs, patients were fed experimental diets. Therefore, the feasibility of the low-GI diet in the clinical setting remains unknown. In addition, there is no evidence that long-term consumption of a low-GI diet will contribute to improved glycemic control in people with diabetes [9,13]. Diabetic care usually requires both medication and optimal dietary management, with the latter decreasing the dependence on diabetic medications for control of HbA1c. Diabetic medication changes should be assessed when a co-therapy, like dietary counseling, is administered.

The objective of the current study was to examine the efficacy of low-GI dietary education compared to the ADA dietary education on glycemic control, diabetic medication change, blood lipids, blood pressure, body weight, and dietary GI score for patients with type 2 diabetes.

MATERIALS/SUBJECTS AND METHODS

Study Subjects

The study population was recruited for the Diabetic Educational Eating Plan (DEEP) Study (ClinicalTrials.gov Identifier: NCT00473811) from a primary care setting at the University Campus of the University of Massachusetts Memorial Healthcare Center (UMMHC). Detailed study methodology was described elsewhere [14]. Briefly, subjects were randomized to either a low-GI diet or the standard ADA diet. Study eligibility included: a diagnosis of type 2 diabetes documented in the patient's medical chart; HbA1c level ≥ 7 (an indication of poor glycemic control [15]); ≥ 21 years old; with a telephone in home or easy access to one; ability to understand and provide informed consent; and willingness to be randomized to either of the two study arms. Exclusion criteria included: pregnancy or planning to become pregnant during the study; plans to move out of the area within the 12-month study period; and documented acute coronary event (myocardial infarction or unstable angina) within the previous 6 months.

Eleven primary care physicians were contacted to assist with recruitment and eight physicians agreed to participate. These physicians identified a total of 154 of their patients as study candidates. Patients were sent a study invitation letter signed by their primary care physician and the principal investigator. Initial telephone contact was be made by a research assistant two weeks after the letter was mailed unless the research assistant was called by the patient declining recruitment into the study. This information was included in the invitation letter. Of 154 potentially eligible patients, 17 (11%) responded to the letter and refused to receive telephone contact. An additional nine subjects learned about the study from flyers or messages through the intranet service at the UMMHC, and agreement for their participation was obtained from their primary care physicians. Of all study candidates, 40 patients were determined to be eligible through telephone interviews by a research assistant, followed by a HbA1c screening, and were then enrolled in the study. Of 40 patients enrolled, only 3 (7.5%) were responding from flyers, and 37 (92.5%) from the physician lists.

Each eligible patient participated in the study for one year. At the baseline visit, participants provided consent; and a fasting blood sample, blood pressure, and anthropometric measures were taken. These measures were also assessed at the 6-month and 12-month visits. A questionnaire packet assessing demographic information, diet and physical activity, psychosocial variables, and medication use were completed at baseline, 6-month and 12-month visits. Each patient received $20 at both the 6-month and the 12-month visit. The study protocol was approved by the University of Massachusetts Medical School Institutional Review Board for use on human subjects in medical research.

Study Treatment

Intervention Format—The intervention consisted of monthly visits for 6 months with two visits in the following 6 months. An outline of the intervention content is listed in Table 1.

Study Diets—A comparison of the two diets is listed in Table 2. Briefly, the ADA diet includes carbohydrate counting, and entails following the Medical Nutrition Therapy Guidelines from the ADA [8,9,13]. The ADA recommends that total daily carbohydrate should be based on the participant's estimated caloric needs, with a goal of consuming an average of 55% total energy from carbohydrate sources. By contrast, the low-GI diet patients were educated on how to choose predominantly low-GI foods with efforts to tailor the integration of GI foods to the patient's lifestyle and taste preferences through substitutions, additions, and directed changes. Two registered dietitians were trained, and each delivered either low-GI or ADA sessions.

Sample Size Estimation

We estimated sample size based on calculation in terms of feasibility outcomes (i.e., estimating proportions such as overall retention and retention per condition). The proposed sample size was chosen in order to obtain precise estimates (narrow confidence intervals). Ten patients per arm give fairly wide confidence intervals (i.e., low precision); however, increasing to 20 patients per arm yields a relatively large decrease in tolerance (from 0.20 to 0.15), i.e., increase in precision.

In our pilot work baseline HbA1c was 8.0 (standard deviation (SD)=0.95) [23]. Taking a conservative approach, we used the SD upper 75% confidence limit (1.16). We assumed the group mean of HbA1c be 8.00% in ADA group, and 7.50% in the low-GI ADA group at 12 months. Staying conservative, we chose >90% power. Under these assumptions, a trial with 115 subjects per group would be required to achieve >90% power to detect a difference of 0.5% in HAb1c at 12 months between the randomized groups. Conservatively assuming 20% dropout at 12 months, 144 patients per group would be need to be enrolled (total N=288) for

a full-powered study using HbA1c as the primary outcome. Therefore, we are under powered to detect the difference of HbA1c. The present was designed to provide pilot data to justify and inform a larger randomized trial.

Randomization

Randomization was carried out after informed consent and baseline data collection was conducted by a research assistant. In order to achieve HbA1c balance between the two arms, randomization was blocked on HbA1c for two groups. One group included participants with HbA1c of <8, and the other group included participants with HbA1c ≥ 8. Within each of the two HbA1c groups, subjects were randomized to the two arms in randomly permuted blocks of size 3 and 6 using the ralloc program in Stata [16].

Measurements

Body weight and height were measured without shoes and wearing light clothing, using a balance beam scale and statiometer. Body mass index (BMI) was calculated as weight (kg)/ height (in meters) squared, at each study point. Waist circumference also was measured. Blood pressure measurements were taken using a Dinamap XL automated blood pressure monitor (Critikon, Arlington, Tex.).

At each time point, using a questionnaire designed specifically for this purpose, patients recorded use of all oral and injected hypoglycemic medications, with attention to changes at follow-up visits. A pharmacist reviewed questionnaires to ensure completeness and adequacy of details. Depressive symptoms were assessed using the Center for Epidemiological Studies-Depression Scale (CES-D) [17,18]. Diabetes-specific emotional distress was assessed by the Problem Areas in Diabetes (PAID) [19]. Each item is scored 0 to 4 ("Not a problem" to "Serious Problem"). The sum of the 20 items is multiplied by 1.25 to yield a final score of 0–100, with higher scores indicating higher emotional distress.

A 12-hour fasting blood sample was collected between 7 and 10 am. HbA1c and blood lipids (including total cholesterol, HDL, LDL, and triglycerides) were measured in the UMMHC Hospital Laboratory. All assays met the standardization criteria of the CDC-NHLBI Lipid Standardization Program.

The 7-day dietary recall (7DDR), which is similar to a food frequency questionnaire (FFQ) in format but focuses the subject on the previous week's diet only, was used for dietary assessment at each visit [20]. Nutrient scores, such as total energy in kcal and carbohydrate intake, GI (referent=white bread), GL, and percentage of energy from fat, protein and carbohydrate, were computed from the data collected from the 7DDR, as has been previously reported by our group [14,21–23]. The 7DDR also included a brief validated physical activity assessment described elsewhere [14,23–25].

Statistical analyses

Descriptive statistics of participants' characteristics were calculated by condition. Between-group differences were evaluated using two-group mean t-test for continuous variables or Fisher's exact test for categorical variables. All major outcomes, including HbA1c, and blood lipids approximately followed a normal distribution and were analyzed with their original values. To correct extreme skewness values of triglyceride (skewness: 1.22 and Kurtosis: 4.23), triglyceride values were transformed using the natural logarithm of raw values.

Mean HbA1c and lipid values, by visit and study group, were estimated using SAS PROC MIXED [26]. The dependent variable was the HbA1c or each lipid measure at each point. The independent variables were: time of visit (baseline, 6-month or 12-month), treatment group

(low-GI or ADA diet group), and an interaction term between time and treatment group as fixed effects, with subject treated as random effect. The effects of body weight, waist circumference, dietary intake, physical activity, and psychological measures by visit and study group were estimated in a similar manner.

There were two subjects (10%) in each group who missed one year HbA1c, and one subject in the low-GI group who missed the six month HbA1c measure. We imputed HbA1c missing values using baseline value carried forward as Ware recommended [27] and HbA1c were re-analyzed.

Based on self-reports of diabetic medication use, a simple medication-change code was created at 6 and 12 months (see Table 5). Using a multinomial logistic regression model, we first analyzed medication association with study group, age, gender, BMI, HbA1c, time (6-month versus 12-month). Since the direction and magnitude of associations for both discontinuation or lower dose (−1) versus no change (0), adding new drug or increase dose (+1) versus no change (0) are the same range, we pooled the data to fit an ordinal logistic regression model for medication change, assuming proportional odds ratios across the categories.

All analyses were performed using SAS (version 9.13; SAS Institute Inc., Cary, NC).

RESULTS

Participants were between the ages of 33 and 77 years old [mean: 53.5 (SD=8.4)]. Ninety-five percent were overweight or obese with a mean BMI of 35.8 kg/m^2 (see Table 3). Fifty-three percent were female, and 53% had a bachelor's degree or higher education. Fifty-five percent were employed full time. Participants were predominantly white (85%) and married or living with partner (70%). Ninety percent (n=36) were taking medication for diabetes. Detailed diabetes medication use information at baseline was reported elsewhere [14]. Briefly, the most common oral medication was metformin (73%), followed by glyburide (38%). Ten subjects (25%) used insulin in addition to oral hypoglycemic agents. The two groups resembled each other statistically in most baseline characteristics, except age where subjects in the ADA group were slightly younger.

Table 4 presents HbA1c values as well as other physiological, dietary, and physical activity, and psychosocial variables over time. Three p-values are presented: 1) for time and group interaction, which is the test of treatment effect; 2) for time, which is the test whether the measure was changed over time; and 3) from t-test of the difference of two group means. These were obtained from mixed model fitting physiological, dietary intake, physical activity, or psychological variables as dependent variable, time measurement, treatment group, and interaction between time and group term as fixed effect, and subject as a random effect.

Mean HbA1c was significantly decreased during the study for both groups ($p<0.001$), especially between baseline and 6 months (8.1% to 7.4% for the ADA group, and 8.7% to 8.0% for the low-GI group), but were attenuated at 12 months. However, there is no treatment effect evident ($p=0.88$), and no difference between two groups on HbA1c at any time point. Results were similar after using baseline value carried forward for subjects with missing values.

Total cholesterol levels were significantly decreased ($p=0.03$) for both groups, but there was no significant difference between two groups. HDL and triglycerides were unchanged, nor was there a difference between the two groups. However, there was a difference in LDL cholesterol levels between two groups at 12 months ($p=0.048$). At 12 months, LDL cholesterol levels were significantly lower in low-GI group than the ADA group with LDL cholesterol levels decreasing by 17 mg/dl in the ADA group and increasing by 1.3 mg/dl in the low-GI group.

Diastolic blood pressure decreased significantly (p=0.01), and was significantly lower in the low-GI group compared to the ADA diet group (p=0.03) at 6 months. At 12 months, there was no significant difference in diastolic blood pressure between two groups, although both groups showed improvement from baseline values. Systolic blood pressure remained unchanged in both groups.

Although patients stated at baseline that they were not following a low or modified carbohydrate diet [14], both groups consumed a very low percentage of calories from carbohydrates (37% in the ADA group versus 36% in the low-GI group at baseline), and very high percentage of calories from saturated fat (15% versus 14%, at baseline), which improved modestly in the low-GI group at 6 months (15.5% in the ADA group versus 12.7% in the low-GI group, p=0.03), and in both groups at 12 months (14.2% versus 13.2%, p=0.41).

Differences in dietary glycemic index between the two groups did not approach statistical significance until 12 months (80 in the ADA group versus 76 in the low-GI group, p=0.07). However, compared to the ADA group, GL was significantly lower in the low-GI group at 6 months (97 versus 141, p=0.02). Interestingly, at 12 months, total carbohydrate intake increased in the ADA group, but decreased in the low-GI group. Daily caloric intake dropped by 624 kcal in low-GI group at 6 months, and remained 325 kcal lower than baseline at 12 months. Caloric intake increased minimally in the ADA group. Depression and PAID scores slightly decreased within each group but did not reach statistical significance. These scores were also not significantly different between two groups at any time point.

Table 5 presents summary data on diabetic medication change during the study. In the ADA group, two subjects decreased medication use and four subjects added medication or increased dose at 6 months. From 6 to 12 months, four participants added medication or increased dose. In the low-GI group, three subjects decreased medication use and one increased it at 6 months. While from 6 to 12 months, one participant decreased and two added medication or dose. Results from a ordinal logistic regression model for medication change showed that participants in the low-GI group had much lower likelihood of switching to a new drug or increasing dosage of diabetes medication (odds ratio (OR)=0.26, p=0.01). Also, higher BMI (OR=1.12, p=0.01), male gender (OR=2.83, p=0.08) and higher HbA1c (OR=1.53, p=0.02) predicted higher likelihood of switching to a new drug or increasing doses.

Average class attendance differed between groups, with an average attendance of 6.57 (SD=1.91) for ADA group and 4.50 (SD=1.90) for the low-GI group (p=0.002). Participants completed a questionnaire at the end of the study to assess the acceptability of the study. Both groups of participants liked the diet they were prescribed (100% in the GI versus 88% in the ADA group; p=0.49). Additionally, all participants in the low-GI group reported the intervention was helpful versus 77% in the ADA group (p=0.11). Thirty-five percent of ADA group versus 23% of low-GI group reported that it was difficult for them to maintain the new diet (p=0.69). All participants in the low-GI group and 71% of those in the ADA group reported enjoying eating unfamiliar foods (p=0.05). There were no diet-related adverse events reported in either group during the study.

DISCUSSION

Compared to the ADA diet, the low-GI diet led to a reduction in the use of diabetic medication while achieving equivalent control of HbA1c and blood lipids. Despite its limited size, this trial suggests that the low-GI diet may be an alternative to the conventional ADA diet. This finding should be evaluated in a larger randomized controlled trial.

Both ADA and low-GI diets resulted in significant and comparable improvements in HbA1c and lipid profiles. The ADA diet has changed over the years, and now incorporates some low-

GI concepts, without specifically recommending it as an alternative. Because both groups had a low carbohydrate diet at baseline, the addition of "healthy" foods to both diets likely had a beneficial effect in both groups. The low-GI diet is directed at quality of carbohydrate rather than quantity. Since the group was already consuming a low carbohydrate diet, we did not observe a reduction in percentage of calories from carbohydrate in the low-GI group but instead saw a shift in the balance of the rest of the diet with an overall reduction in calories from saturated fat. The ADA group increased their total carbohydrate intake, while the low-GI group decreased grams and changed quality of carbohydrate, thereby decreasing GL. Both groups were able to achieve improvement on HbA1c via these methods.

It is notable that the low-GI group was able to achieve dietary changes (specifically a lower GL score) from the materials and tools provided in the study, despite an inability to attend as many group sessions as the ADA group. Changes in the food environment (with an increasing emphasis on whole grains, fruit and vegetables, nuts and legumes) provide an opportunity to simplify the concepts of the low-GI diet. Patients in either group who could not attend sessions were provided written materials and/or brief telephone counseling. The low-GI group achieved equal or better results on glycemic control, debunking the myth that the low-GI diet is more difficult to understand than the ADA diet, a finding that is confirmed by our satisfaction survey.

A 2003 meta-analysis by Brand-Miller of RCTs of low-GI diet among individuals with diabetes, suggested that choosing low-GI foods in place of high-GI foods has a beneficial effect on glycemic control [12]. However, the effect was modest. One main limitation is that some studies had interventions lasting only 2–5 weeks, making HbA1c an inappropriate measure to use, partly explaining the lack of change seen in some of the studies. Of the nine studies with type 2 diabetics, five measured HbA1c. However, only one kept participants on the diet long enough (12 weeks) [28] for meaningful HbA1c changes to be seen [1,3]. The study with the 12-week intervention period [28] found HbA1c levels 0.9% lower in the low-GI group than in the high-GI group. These are clinically meaningful reductions [4,29]. In the present study, at 6 months, HbA1c change was −0.67% for the ADA and −0.75% for the low-GI diet group, taking into account that a reduction of 0.5% in HbA1c would be seen by most clinicians as a clinically meaningful contribution to achieving target glucose control [29].

Medication therapy is an important part of diabetes prevention and management. In the meta-analysis by Brand-Miller and colleagues [12] change in diabetes medication change was not evaluated. Based on ADA recommendation [9,10], diabetic patients are advised to have their HbA1c checked every three months, providing an opportunity for adjustment of diabetes medication. Our data indicate that diet was a greater part of glycemic control in the low-GI arm of the study than in the ADA arm. Our study extends the literature by examining diabetes medication change patterns to gauge the influence of a dietary intervention upon medication use. Medication changes should be assessed when a co-therapy, like dietary counseling, is administered.

Weight loss was not significant during the study in either group, despite reduction in calories in the low-GI group. Studies have demonstrated that with improved glycemic control, patients with poorly controlled diabetes usually tend to gain weight, in part due to resolution of glucosuria. In obese patients with type 2 diabetes reducing caloric intake improves glycemic control, often more rapidly than does weight loss [30,31]. In non-diabetic obese children and adolescents, one study also found no association between weight loss and HbA1c change in that population during a 12-week weight loss program [32].

There are several strengths to the present study. First, this was a randomized clinical trial to test whether a low GI-diet education improves glycemic control in free-living patients with uncontrolled diabetes. Second, this study was of sufficient duration to detect intervention-

related changes in HbA1c. Third, we collected and examined change in diabetic medication use as an outcome variable. Finally, we evaluated acceptability at the end of the study to provide information on satisfaction with the diets and their outcomes.

This present study also has limitations. First, there were differential attendance rates between groups. Attendance was lower in the low-GI, which may have adversely affected understanding and achievement of a lower-GI diet. The mean age in low-GI was higher than for ADA, which may have created differential barriers to attendance. In addition, the low- GI dietary intervention may be perceived as complicated; however, our data on acceptability of the diets do not support this as a possibility. Second, at baseline 77.5% of participants had hyperlipidemia, and all but one of these participants was taking lipid-lowering medications [14]. However, we did not assess lipid-lowering medication change, preventing us from evaluating the impact of the intervention on changes in the use of these types of medications. Finally, we did not assess self-reported reasons for medication changes.

CONCLUSIONS

Compared to ADA diet, the low-GI diet achieved equivalent control of HbA1c using less diabetic medication. We conclude that the low-GI diet is a viable alternative to the standard ADA diet. Findings should be evaluated in a larger randomized controlled trial.

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Table 1

Outline of the intervention components and content, the Diabetic Educational Eating Plan (DEEP) study, Worcester, Massachusetts, 2005–2007.

Session Number	Content Focus for a Low GI Diet or ADA Diet	Format
1 **Month 1**	**Introduction to the program.** Discuss program goals; Discuss participants' outcome expectations and identify participants' motivations to stay in the program; Discuss previous history of dietary changes and challenges experienced; Learn facts related to a Low GI diet or ADA diet (i.e., substitutions, fiber, and carbohydrate counting); and Discuss the importance of goal setting and self-monitoring. A sample a low GI or ADA diet meal will be demonstrated in our study kitchen with participants to facilitate self-efficacy for dietary change through taste-testing and observation/discussion of meal preparation. Training manual with food selections of GI or carbohydrate contents provided to each patient.	Group 2 hour class with registered dietitian
2 **Month 2**	**Goal Setting.** Patient-centered counseling including review of self-monitoring with emphasis on reinforcing success and encouraging problem-solving with regard to challenges; Further instruction in goal setting skills (short and long-term goals) and discussion of their importance for change; Discussion of participant's short-term program goals (which foods they choose to begin to change); and Enlisting social support for dietary change and study participation.	Individual 30 min. with registered dietitian
3 **Month 3**	**Label Reading and Influences on Our Food Choices.** Shopping tour to learn how to read and understand nutrition labels; Review of self-monitoring efforts; Managing prompts (e.g., socials, holidays) for undesirable eating behaviors; Discussion of successes among group participants and group problem-solving regarding challenges and impediments; Further discussion of goal setting and action planning strategies; and Encouragement of continued self-monitoring and personal evaluation of challenges and use of problem-solving strategies to overcome challenges.	Group 60 min. shopping tour, 30 minute group discussion
4 **Month 4**	**Managing Blood Sugars and Problem Solving.** Patient-centered counseling; Review of self-monitoring efforts with emphasis on reinforcement of successes and problem-solving concerning challenges; Management of low and high blood sugars; Heart healthy eating tips; and Continued practice of goals and action planning skills. Participants will be given the opportunity to sample a low GI or ADA diet meal in the study kitchen.	Group Visit 60 min with registered dietitian
5 **Month 5**	**Portion Size, Tips and Techniques.** Patient-centered counseling; Review of self-monitoring efforts with emphasis on reinforcement of successes and problem-solving concerning challenges; Learn accurate portion size estimation; Discussion of low GI or low CHO foods to eat at various meals throughout the day; and Discussion of recipe modification strategies. Participants will be given the opportunity to sample a low GI or ADA diet meal in the study kitchen.	Group Visit 60 min with registered dietitian
6 **Month 6**	**Dining Out.** Patient-centered counseling; Review of self-monitoring efforts with emphasis on reinforcement of successes and problem-solving concerning challenges; Increase awareness of challenges when eating away from home; and Discuss management of challenges and selection of healthy choices at restaurants. Participants will be given the opportunity to sample a low GI or ADA diet meal in the study kitchen.	Group Visit 60 min with registered dietitian
7 **Month 8**	**Reinforcement.** Patient-centered counseling; Review of self-monitoring efforts with emphasis on reinforcement of successes and problem-solving concerning challenges; and Setting of new goals and discussion of potential challenges. Participants will be given the opportunity to sample a low GI or ADA diet meal in the study kitchen.	Group Visit 60 min with registered dietitian
8 **Month 10**	**Reinforcement.** Patient-centered counseling; Review of self-monitoring efforts with emphasis on reinforcement of successes and problem-solving concerning challenges; and Discuss long-term adherence, resources. Participants will be given the opportunity to sample a low GI or ADA diet meal in the study kitchen.	Group Visit 60 min with registered dietitian

Table 2

Comparison of the American Diabetes Association (ADA) and a Low GI Diet, the Diabetic Educational Eating Plan (DEEP) study, Worcester, Massachusetts, 2005–2007.

	ADA Study Diet	Low-GI Study Diet
Carbohydrate (CHO)	All CHO foods are treated the same.	CHO foods are ranked according to GI
	Patient provided with daily CHO gram goals. The total amount of carbohydrate needed daily is based on a person's calorie needs- carbohydrate should comprise an average of 55% percent of daily energy intake.	
		Patient provided with goals to reduce daily dietary GI score to 55 from baseline levels, established via food records.
Protein	Lean proteins providing 15–20% of total calories.	
Fat	Saturated (<10% of caloric intake) and trans-fatty acids restricted. <7% recommended for those individuals with LDL cholesterol >100mg/dl. Dietary cholesterol <300mg/day, <200mg/day for those with LDL cholesterol >100 mg/dl.	
Glycemic factors (i.e.; acidity, fat, soluble fiber)	Patients encouraged to reach a goal of 25–35 grams of total fiber per day. No other factors are addressed.	Patients encouraged to use all GI factors as appropriate for modification of recipes and food choices.
Recipe modification	Patients provided with ADA recipes and suggestions.	Patients provided with low GI substitutes for high-GI foods, recipes provided.
Label reading	Patients taught to read total carbohydrate content (subtracting fiber from total CHO if more than 5 grams per serving), or utilize the diabetic exchange lists with serving size and CHO gram goals in mind.	Patients taught to read ingredients and choose predominantly low GI foods.
Physical Activity	Patients encouraged to exercise 30 minutes per day most days of the week	

Table 3

Selected participants' characteristics of the ADA and low-GI group at baseline (N=40), the Diabetic Educational Eating Plan (DEEP) study, Worcester, Massachusetts, 2005–2007.

	Total	ADA	Low-GI	P-value*
Categorical Variables	**Count (%)**	**Count (%)**	**Count (%)**	
Gender				
Male	19 (47.50)	11 (52.38)	8 (42.11)	0.55
Female	21 (52.50)	10 (47.62)	11 (57.89)	
Education				
High school or less	9 (22.50)	6 (28.57)	3 (15.79)	0.26
Some college/associate's degree	10 (25.00)	3 (14.29)	7 (36.84)	
Bachelor's degree or more	21 (52.50)	12 (57.14)	9 (47.37)	
Ethnicity				
White	34 (85.00)	16 (76.19)	18 (94.74)	0.19
Other	6 (15.0)	5 (23.81)	1 (5.26)	
Marital status				
Married or living with partner	28 (70.00)	16 (76.19)	12 (63.19)	0.49
Other	12 (30.00)	5 (23.81)	7 (36.84)	
Work status				
Full-time	22 (55.00)	12 (57.14)	10 (52.63)	0.91
Part-time	7 (17.50)	4 (19.05)	3 (15.79)	
Retired/Unemployed/Other	11 (27.50)	5 (23.81)	6 (31.58)	
In general, would you say your health is:				
Excellent				
Very good	7 (17.95)	3 (14.29)	4 (22.22)	0.40
Good	26 (66.67)	16 (76.19)	10 (55.56)	
Fair	6 (15.38)	2 (9.52)	4 (22.22)	
Poor				
Continuous Variables	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	
Age (years)	53.53 (8.40)	51.0 (8.25)	56.31 (7.85)	0.04
Body mass index	35.78 (7.00)	35.95 (6.75)	35.58 (7.46)	0.87
Duration since diagnosis(years)	9.32 (9.66)	6.62 (6.47)	12.65 (11.93)	0.07

* p-value was from two group t test for continuous variables or Fisher's exact test for categorical variables.

Table 4

Physiological, dietary, and physical activity, and psychosocial variables over time by study group, the Diabetic Educational Eating Plan (DEEP) study, Worcester, Massachusetts, 2005–2007.

Variable	Baseline Mean (SE)	6 Months Mean (SE)	12 months Mean (SE)	* p-value for intervention effect (p-value for time)
PHYSIOLOGICAL VARIABLE				
HbA1c (%)				0.88 (<0.001)
ADA	8.10 (0.28)	7.43 (0.28)	7.67 (0.28)	
Low-GI	8.74 (0.29)	7.99 (0.29)	8.39 (0.30)	
p-value†	0.11	0.16	0.08	
Total Cholesterol				0.13 (0.03)
ADA	168.10 (9.06)	157.33 (9.06)	149.71 (9.35)	
Low-GI	175.58 (9.53)	160.52 (9.79)	173.63 (0.06)	
p-value†	0.57	0.82	0.09	
LDL Cholesterol (mg/dl)				0.05 (0.12)
ADA	88.95 (7.52)	81.86 (7.51)	71.49 (7.81)	
Low-GI	93.16 (8.07)	87.10 (8.31)	94.50 (8.32)	
p-value†	0.70	0.64	0.048	
HDL Cholesterol (mg/dl)				0.68 (0.13)
ADA	42.95 (2.26)	42.19 (2.26)	44.29 (2.30)	
Low-GI	45.42 (2.38)	46.25 (2.42)	47.53 (2.43)	
p-value†	0.45	0.22	0.34	
Natural Logarithm of Triglycerides				0.43 (0.10)
ADA	5.0484 (0.14)	4.98 (0.14)	4.93 (0.15)	
Low-GI	4.99 (0.15)	4.78 (0.16)	4.90 (0.16)	
p-value†	0.79	0.37	0.90	
Diastolic Blood Pressure (mmHg)				0.24 (0.01)
ADA	79.32 (1.85)	77.90 (1.81)	74.22 (1.88)	
Low-GI	77.10 (1.91)	71.75 (2.05)	72.94 (2.16)	
p-value†	0.41	0.03	0.66	
Systolic Blood Pressure (mmHg)				0.20 (0.69)
ADA	130.78 (2.46)	132.76 (2.41)	128.92 (2.52)	
Low-GI	131.32 (2.54)	126.41 (2.75)	129.54 (2.91)	
p-value†	0.88	0.09	0.87	
Weight (lbs)				0.89 (0.01)
ADA	225.81 (10.33)	222.52 (10.33)	224.05 (10.34)	
Low-GI	211.58 (10.86)	207.46 (10.88)	208.66 (10.89)	
p-value†	0.35	0.32	0.31	
Waist Circumference (inches)				0.44 (0.07)
ADA	44.53 (1.33)	44.52 (1.33)	45.98 (1.34)	
Low-GI	43.65 (1.40)	43.77 (1.42)	44.13 (1.46)	
p-value†	0.65	0.70	0.36	
DIETARY VARIABLE				
Glycemic index (white bread=100)				0.86 (0.13)
ADA	82.03 (1.31)	80.56 (1.28)	80.36 (1.40)	
Low-GI	79.35 (1.36)	77.93 (1.42)	76.64 (1.46)	
p-value†	0.16	0.18	0.07	
Glycemic load				0.04 (0.29)
ADA	128.41 (12.22)	140.68 (11.91)	147.98 (13.31)	
Low-GI	146.08 (12.56)	97.18 (13.31)	119.77 (13.75)	
p-value†	0.32	0.02	0.15	
Caloric intake (kcal/day)				0.02 (0.08)
ADA	1656.29 (141.98)	1724.96 (138.69)	1779.52 (153.17)	
Low-GI	1998.69 (146.67)	1374.51 (154.58)	1674.03 (159.02)	
p-value†	0.10	0.10	0.63	
Total carbohydrate (g/day)				0.04 (0.17)
ADA	153.54 (12.89)	163.90 (12.57)	165.78 (14.00)	
Low-GI	173.10 (13.26)	118.67 (14.04)	153.37 (14.49)	
p-value†	0.29	0.02	0.54	
Carbohydrate percentage				0.81 (0.59)
ADA	37.38 (2.02)	38.66 (1.98)	38.47 (2.14)	
Low-GI	35.66 (2.11)	35.93 (2.20)	37.78 (2.24)	
p-value†	0.56	0.36	0.82	
Fat percentage				0.91 (0.25)

Variable	Baseline Mean (SE)	6 Months Mean (SE)	12 months Mean (SE)	* p-value for intervention effect (p-value for time)
ADA	45.00 (2.04)	43.14 (2.00)	42.75 (2.17)	
Low-GI	44.25 (2.13)	41.42 (2.22)	42.32 (2.27)	
p-value†	0.80	0.57	0.89	
Protein percentage				
ADA	18.45 (1.04)	19.04 (1.01)	19.75 (1.11)	0.098(0.23)
Low-GI	21.25 (1.07)	23.35 (1.13)	20.12 (1.16)	
p-value†	0.06	0.006	0.82	
Saturated fat percentage				0.17 (0.25)
ADA	15.10 (0.83)	15.52 (0.82)	14.21 (0.87)	
Low-GI	14.07 (0.88)	12.73 (0.91)	13.15 (0.92)	
p-value†	0.40	0.03	0.41	
Dietary fiber (g/day)				0.19 (0.32)
ADA	11.52 (1.11)	12.10 (1.08)	12.11 (1.18)	
Low-GI	11.83 (1.15)	9.43 (1.20)	12.18 (1.23)	
p-value†	0.85	0.10	0.97	
Leisure time physical activity (min/week)				0.81 (0.14)
ADA	121.06 (36.88)	183.89 (33.30)	112.51(36.88)	
Low-GI	165.89 (35.58)	188.11 (35.60)	119.85 (40.06)	
p-value†	0.39	0.93	0.89	
PSYCHOSOCIAL FACTOR				0.60 (0.47)
CES-D depression score				
ADA	10.00 (2.62)	11.67 (2.62)	9.04 (2.76)	
Low-GI	11.21 (2.76)	9.61 (2.94)	8.42 (3.05)	
p-value†	0.75	0.60	0.88	
PAID (Problem Areas in Diabetes) score				0.59 (0.38)
ADA	19.81 (3.53)	19.19 (3.53)	19.03 (3.67)	
Low-GI	22.42 (3.71)	18.55 (3.86)	20.66 (3.95)	
p-value†	0.61	0.90	0.76	

* p-values were from mixed model fitting physiological, dietary intake, physical activity, or psychological variables as dependent variable, time measurement, treatment group, and interaction between time and group term as fixed effect, and subject as a random effect. Treatment effect is defined as difference in changes in measures between time points: the interaction term between time and group.

† p-value was from t-test for Ho: group means between ADA and low-GI groups are equal.

Table 5

Subjects with medication changes during the study

Group	Subject ID	Medication category	Baseline	Change at 6 months	Change Code* at 6 moths	Change at 12 months	Change Code* at 12 moths
LOW-GI GROUP	115	Sulfonylurea	+	+	0	−	−1
		Biguanide	+	+		+	
		Thiazolidinedione	+	+		−	−1
		Insulin	+	+		→	1
		GLP-1	−	−		−	
		Medication change summary code*			0		1
		HBA1c	12	12		13.2	
	402	Sulfonylurea	+	←	−1	+	0
		Biguanide	+	+		+	
		Thiazolidinedione	−	−		−	
		Insulin	−	−		−	
		GLP-1	−	−		−	
		Medication change summary code*			−1		0
		HBA1c	8	7		7.1	
	607†	Sulfonylurea	+	+	0	+	0
		Biguanide	+	+		+	
		Thiazolidinedione	−	−		−	
		Insulin	−	−		−	
		GLP-1	−	−		−	
		Medication change summary code*			0		0
		HBA1c	8	8		7.3	
	898	Sulfonylurea	+	→	1	−	−1
		Biguanide	−	−		−	
		Thiazolidinedione	−	+	1	−	−1
		Insulin	−	−		−	
		GLP-1	−	−		−	
		Medication change summary code*			1		−1
		HBA1c	8	8		7.9	
	10001	Sulfonylurea	+	+		+	
		Biguanide	+	←	−1	+	0
		Thiazolidinedione	−	−		−	
		Insulin	−	−		−	
		GLP-1	−	−		−	
		Medication change summary code*			−1		0
		HBA1c	8	7		6.8	
	10010	Sulfonylurea	−	−	0	−	
		Biguanide	+	+		+	
		Thiazolidinedione	+	−		−	
		Insulin	+	←	−1	+	
		GLP-1	−	+		→	1
		Medication change summary code*			−1		1
		HBA1c	7	8		8.3	
ADA GROUP	76†	Sulfonylurea	+	+	0	−	0
		Biguanide	+	+		−	
		Thiazolidinedione	+	+		−	
		Insulin	+	+		+	
		GLP-1	−	−		−	

Group	Subject ID	Medication category	Baseline	Change at 6 months	Change Code* at 6 moths	Change at 12 months	Change Code* at 12 moths
		Medication change summary code*			0		0
		HBA1c	9	9		9	
	204	Sulfonylurea	−	−	0	−	
		Biguanide	−	−	0	+	1
		Thiazolidinedione	−	−		−	
		Insulin	+	+		+	
		GLP-1	−	−		−	
		Medication change summary code*			0		1
		HBA1c	9	7		7.8	
	595	Sulfonylurea	+	−		−	
		Biguanide	+	+		+	
		Thiazolidinedione	−	−		−	
		Insulin	+	→	1	→	1
		GLP-1	−	+		+	
		Medication change summary code*			1		1
		HBA1c	7	8		7.4	
	876	Sulfonylurea	−	−		−	
		Biguanide	+	+		+	
		Thiazolidinedione	−	−	0	−	
		Insulin	+	←	−1	+	0
		GLP-1	−	−		−	
		Medication change summary code*			−1		0
		HBA1c	7	7		6.5	
	968	Sulfonylurea	+	+	0	+	
		Biguanide	+	+		+	
		Thiazolidinedione	−	−		−	
		Insulin	−	−		−	
		GLP-1	−	−	0	+	1
		Medication change summary code*			0		1
		HBA1c	8	7		7.2	
	991†	Sulfonylurea	−	−	0	−	0
		Biguanide	+	+		+	
		Thiazolidinedione	−	−		−	
		Insulin	−	−		−	
		GLP-1	−	−		−	
		Medication change summary code*			0		0
		HBA1c	7	6		6.5	
	1069	Sulfonylurea	−	−	0	−	
		Biguanide	+	+		+	
		Thiazolidinedione	−	−		−	
		Insulin	+	←	−1	+	0
		GLP-1	−	−		−	
		Medication change summary code*			−1		0
		HBA1c	8	6		6.3	
	1090	Sulfonylurea	+	+	0	+	
		Biguanide	+	+		+	
		Thiazolidinedione	+	+		−	−1
		Insulin	−	−		+	1
		GLP-1	−	−		−	
		Medication change summary code*			0		1

Group	Subject ID	Medication category	Baseline	Change at 6 months	Change Code* at 6 moths	Change at 12 months	Change Code* at 12 moths
		HBA1c	8	7		7.1	
	1143	Sulfonylurea	–	–	0	–	
		Biguanide	+	+		+	
		Thiazolidinedione	–	–		–	
		Insulin	–	–		–	
		GLP-1	–	+	1	+	0
		Medication change summary code*			1		0
		HBA1c	9	8		8	
	10003	Sulfonylurea	–	–	0	–	
		Biguanide	+	+		+	
		Thiazolidinedione	+	+		+	
		Insulin	+	→	1	+	0
		GLP-1	–	–		–	
		Medication change summary code*			1		0
		HBA1c	9	7		8.5	
	10004	Sulfonylurea	+	+	0	+	
		Biguanide	+	+		+	
		Thiazolidinedione	+	–	–1	–	0
		Insulin	–	+	1	+	0
		GLP-1	–	–		–	
		Medication change summary code*			1		0
		HBA1c	10	9		9	
	10013	Sulfonylurea	–	–	0	–	
		Biguanide	–	+	1	+	0
		Thiazolidinedione	–	–		–	
		Insulin	–	–		–	
		GLP-1	–	–		–	
		Medication change summary code*			1		0
		HBA1c	7	6		6.3	

+ **KEY:** present/no change since last report

– not present

→ increased dose

← decreased dose

*: "–1" if any medication was discontinued or decreased dosage, "0" if no change, "+1" if dose was increased or a drug was added. In addition for summary score, we assumed any change in insulin presides over all other changes.

† change was in the same medication category coded as 0.