Context: Conventional dietary approaches for the treatment of obesity have generally yielded disappointing results.

Objective: To examine the effects of a low–glycemic index (GI) diet compared with a standard reduced-fat diet in the management of pediatric obesity.


Setting: Academic medical center.

Participants: One hundred seven obese but otherwise healthy children.

Main Outcome Measures: Changes in body mass index (BMI [calculated as weight in kilograms divided by the square of height in meters]) and body weight from first to last clinic visit.

Results: A total of 64 patients received the low glycemic index diet and 43 received the reduced-fat diet for 4.3 vs 4.2 months’ mean duration of follow-up, with 3.3 vs 3.3 mean number of visits, respectively. Body mass index (−1.53 kg/m² [95% confidence interval, −1.94 to −1.12] vs −0.06 kg/m² [−0.56 to +0.44], P < .001) and body weight (−2.03 kg [95% confidence interval −3.19 to −0.88] vs +1.31 kg [−0.11 to +2.72], P < .001) decreased more in the low-GI group compared with the reduced-fat group. In multivariate models, these differences remained significant (P < .01) after adjustment for age, sex, ethnicity, BMI or baseline weight, participation in behavioral modification sessions, and treatment duration. Significantly more patients in the low-GI group experienced a decrease in BMI of at least 3 kg/m² (11 kg/m² [17.2%] vs 1 kg/m² [2.3%], P = .03).

Conclusions: A low-GI diet seems to be a promising alternative to standard dietary treatment for obesity in children. Long-term randomized controlled trials of a low-GI diet in the prevention and treatment of obesity are needed.


EXCESSIVE BODY weight is arguably the most prevalent medical problem in the United States today. Approximately 25% of children and more than 50% of adults are considered overweight according to data from the most recent National Health and Nutrition Examination Survey. Overweight and obesity in childhood contribute to a range of immediate and long-term problems, including diabetes mellitus, dyslipidemia, hypertension, sleep apnea, musculoskeletal problems, gastrointestinal disease, and psychosocial difficulties. Overweight children, especially those older than 7 years, are at increased risk for obesity and cardiovascular disease in adulthood.

The standard approach to the treatment of obesity involves the reduction of dietary fat, the most energy-dense nutrient. The US Department of Agriculture, the American Heart Association (Dallas, Tex), and the American Diabetes Association (Alexandria, Va) currently recommend reduced-fat diets in the prevention and treatment of obesity. However, weight loss on reduced-fat diets is characteristically modest and transient. Moreover, prevalence rates of overweight and obesity have risen dramatically in recent years, despite decreases in dietary fat as a percentage of total energy to near recommended levels.

Recently, a low–glycemic index (GI) diet has been proposed as a novel treatment for obesity. Glycemic index refers to the relative rise in blood glucose occurring after consumption of a food containing a standard amount of carbohydrate. Most refined grain products and potatoes have a high GI, whereas nonstarchy vegetables, legumes, and fruits generally have...
PARTICIPANTS AND METHODS

STUDY DESIGN

During the period between September 1, 1997, and August 31, 1998, children attending the Optimal Weight for Life Program at Children’s Hospital, Boston, Mass, for treatment of obesity were assigned by the program administrator, based on schedule availability, to 1 of 2 teams, each composed of a subspecialty-trained pediatrician, a dietitian, and at times a pediatric nurse practitioner. One team prescribed a low-GI diet, the other a reduced-fat diet. Except for specific dietary recommendations, each team provided similar diagnostic evaluation and treatment. To estimate the effects of dietary treatment on body fatness, we retrospectively examined the changes in body mass index (BMI [calculated as weight in kilograms divided by the square of height in meters]) and body weight from the participant’s initial visit to last visit before December 31, 1998, according to dietary treatment assignment. The mean changes were adjusted for potential confounding variables as described below.

PARTICIPANTS

A total of 190 patients (excluding those with Cushing syndrome, hypothyroidism, hypothalamic disease, diabetes mellitus or an obesity-associated genetic syndrome, or those concurrently following a very low-energy diet) were evaluated during the study period. We further excluded 83 individuals for lack of follow-up (<1 month) and/or incomplete data, leaving a cohort of 107. Descriptive characteristics of this cohort are presented in Table 1.

STANDARD TREATMENT COMPONENTS

All patients received a comprehensive medical evaluation (medical history, physical examination, and laboratory investigation), dietary counseling, and lifestyle counseling (recommendations were based on decreasing physical inactivity and increasing physical activity). Counseling sessions included the child and at least 1 parent, when possible, according to established practice. Specific goals were individualized, with consideration given to the patient’s developmental level and readiness to change. Follow-up appointments were generally recommended to occur on a monthly basis for the first 4 months, and then as needed.

In addition, problem-focused behavior therapy was provided by the program psychologist on an individual basis when referred by a team member. Within these sessions, a particular nutritional or physical activity goal was identified as a primary treatment target. A behavioral program was then developed, using positive reinforcement for meeting the specified goal. Specifics of treatment were adapted according to the patient’s age and developmental stage.

DIETARY TREATMENTS

One team prescribed a standard balanced, hypoenergetic reduced-fat diet because of research demonstrating improvements in adiposity on this diet when combined with behavioral modification and exercise. The diet followed US Department of Agriculture recommendations for intake of specific food types, as depicted by the Food Guide Pyramid. Particular emphasis was placed on limiting intake of high-fat, high-sugar, and energy-dense foods, and increasing intake of grain products, vegetables, and fruit.

RESULTS

Characteristics of the cohort are described in Table 1 according to dietary treatment group. Mean age, length of follow-up, number of visits, and sex were similar between the 2 treatment groups. Baseline BMI and body weight were slightly greater in the reduced-fat group compared with the low-GI group, but the difference was not statistically significant. Ethnicity differed in the study cohort primarily owing to different follow-up rates of the 190 patients before exclusion for lack of follow-up, white subjects comprised 71% of 118 individuals assigned to the low-GI group vs 67% of 72 individuals assigned to the reduced-fat group. Patients in the low-GI group were somewhat more likely (P = .09) to receive a referral for behavioral therapy than patients in the reduced-fat group. No adverse events were reported during the study period.

The Figure depicts the mean change in BMI from the participant’s first to last clinic visit according to dietary treatment and baseline BMI tertile. For each BMI tertile, the low-GI group had a significantly larger decrease in BMI than the reduced-fat group (P < .02 for each comparison). Compared with the reduced-fat group, a larger percentage of patients in the low-GI group experienced a decrease in BMI of at least −3 kg/m² (11 participants [17.2%] vs 1 participant [2.3%], P = .03). As presented in Table 2, the overall mean change in BMI for the low-GI group was −1.53 kg/m², compared with −0.06 kg/m² for the reduced-fat group (P < .001). This difference remained statistically significant (−1.15 kg/m² vs 0.03 kg/m², P < .01) after adjusting for age, sex, ethnicity, length of follow-up, baseline BMI, and behavioral therapy referral. Results were similar for change in body weight after adjustment (−1.16 kg vs 1.44 kg, P < .01).

We also obtained similar results when restricting the analyses to those subjects who did not receive behavioral treatment (mean BMI change after adjustment as

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Recommendations were tailored on an individual basis to incorporate an energy restriction of approximately 1042 kJ (250 kcal) to 2084 kJ (500 kcal) per day compared with usual energy intake. Specific macronutrient goals were 55% to 60% carbohydrate, 15% to 20% protein, and 25% to 30% fat.

The other team prescribed a low-GI diet because of preliminary research suggesting a physiologic mechanism relating GI to body weight regulation.16,17 The low-GI diet was designed to obtain the lowest glycemic response possible while providing adequate dietary carbohydrates, satisfying all nutritional recommendations for children, and maintaining palatability. This diet differed from the standard diet not just in the GI of the component carbohydrates foods, but also in the macronutrient ratio. Emphasis was placed on food selection, not energy restriction: patients were instructed to eat to satiety and snack when hungry. Specifically, patients were told to combine low-GI carbohydrate, protein, and fat at every meal and snack. A “Low-GI Pyramid,”17 modeled after the Food Guide Pyramid, was used as a teaching tool. This modified pyramid placed vegetables, legumes, and fruits at the base, lean proteins and dairy products on the second level, whole-grain products on the third level, and refined grain products, potatoes, and concentrated sugars at the top. Specific macronutrient goals were 45% to 50% carbohydrate, 20% to 25% protein, and 30% to 35% fat.

MEASUREMENT OF OUTCOME VARIABLES

The primary outcome variables were change in BMI and change in body weight. Height was measured to the nearest 0.1 cm with a wall-mounted stadiometer (Holtrain Ltd, Crymych, England) and weight was measured above: −1.47 kg/m² for low-GI diet [n = 31] and 0.20 kg/m² for reduced-fat diet [n = 28], P < .001) and to white subjects (−1.73 kg/m² for low-GI diet [n = 54] and −0.29 kg/m² for reduced-fat diet [n = 23], P < .01). Finally, we found no statistically significant interactions between dietary treatment and any of the covariates (data not shown).

Dietary treatment for obesity among adults is largely unsuccessful. Approximately 25% of men and 40% of women in the United States are currently trying to lose weight.20 Discouragingly, efforts by the vast majority of these individuals will fail to achieve lasting weight loss.10,30,31 Reduced-fat and/or energy-restricted diets generally produce a maximum 10% decrease in body weight among those individuals remaining in treatment programs. Much of this weight is regained within 12 months, with a virtually complete relapse after 5 years. Clinical studies of obesity treatment in children have yielded mixed results.27,32 This situation has prompted calls from experts and official agencies for the development of innovative treatment strategies.33 The results of this study suggest that a low-GI diet may be one such approach.

Children receiving the standard reduced-fat diet showed no change in adjusted BMI during the course of the study, representing a modest improvement over the increase in BMI that would be expected with increasing age.34 By contrast, children receiving the low-GI diet showed an adjusted decrease in BMI of 1.15 kg/m². This result is especially interesting in that the low-GI diet involved no restriction of total energy or specific macronutrient consumption. Instead, patients in this treatment group were encouraged to eat to satiety and snack when hungry. Furthermore, the magnitude of the effect seen here may have been limited by factors inherent to an urban pediatric obesity clinic, including a heterogeneous patient population, inadequate insurance coverage for obesity management,35 and poverty.

To our knowledge, the vast majority of pediatric obesity studies to date have tested different behavioral modification techniques or multimodality programs (behavioral therapy, diet, and physical activity together).27,32 Surprisingly few studies involving children have examined the effects of dietary composition on weight loss per se, while controlling for other interventions. In our study, by contrast, specific dietary prescriptions differed between groups, whereas dietary counseling methods (parent and child), behavioral modification techniques,24 ancillary recommendations (to increase physical activity and decrease inactivity), and treatment intensity did not.

Regarding possible underlying mechanisms, a low-GI diet may facilitate weight loss by lowering insulin levels. High-GI diets stimulate more insulin secretion than iso-
Acutely, high insulin levels would tend to promote up-dial insulin levels and greater c-peptide secretion.\cite{36,37} Energetic, low-GI diets, as evidenced by higher postpran-dial insulin levels and greater c-peptide secretion.\cite{36,37} The take of nutrients into liver, muscle, and adipose tissue; inhibit hepatic release of glucose; and suppress lipolysis. After the nutrients of a high-GI meal have been absorbed from the digestive tract, the body may have difficulty accessing stored metabolic fuels, leading to excessive hunger and overeating.\cite{16} Chronically, hyperinsulinemia would tend to direct nutrients from oxidation to storage. Several, though not all, epidemiological studies have shown that individuals with the highest fasting or stimulated insulin levels at baseline gain the most weight prospectively.\cite{38,40} Moreover, insulin treatment of type 2 diabetes,\cite{41} and intensive insulin treatment of type 1 diabetes\cite{42} predictably results in weight gain. In animal studies, a high-GI diet was found to increase fatty acid synthetase activity, adipocyte size, glucose incorporation into total lipids,\cite{43,44} and insulin resistance\cite{45,46} compared with a low-GI diet.

Several issues relating to study design and interpretation should be addressed. First, participants were not formally randomized to treatment group. Thus, the presence of confounding influences, such as selection bias and provider effects, cannot be excluded. Second, dietary change was not monitored following intervention (as, for example, with diet records). Therefore, the degree to which noncompliance affected outcome is not known. Third, mean follow-up time was relatively short (4.3 months); long-term evaluation of the dietary treatments is beyond the scope of this study. Fourth, the target macronutrient composition of the low-GI diet differed from that of the reduced-fat diet, in an attempt to obtain the lowest possible glycemic response. Therefore, the effects of this diet can not be attributed solely to GI. In light of these qualifications, our findings should be viewed as preliminary.

Nevertheless, we believe that these findings are relevant because the magnitude of the observed effect is large and remained significant after adjustment for a variety of potentially confounding factors; the low-GI diet was tested against the current standard of care; and the data are consistent with a plausible physiologic mechanism. Moreover, the study reflects experience of a major, clinical pediatric obesity program, not a specialized research protocol employing carefully selected subjects and costly interventions. Rather, our study confronted a variety of problems inherent to the outpatient treatment of childhood obesity today, including variable motivation and compliance (eg,

### Table 1. Cohort Characteristics According to Dietary Treatment*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Low-GI (n = 64)</th>
<th>Reduced-Fat (n = 43)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>10.6 ± 4.0</td>
<td>10.2 ± 3.1</td>
<td>.54</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>32.5 ± 7.3</td>
<td>34.5 ± 7.2</td>
<td>.15</td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>73.9 ± 30.5</td>
<td>79.2 ± 31.9</td>
<td>.39</td>
</tr>
<tr>
<td>Length of follow-up, mo</td>
<td>4.3 ± 2.9</td>
<td>4.3 ± 3.5</td>
<td>.97</td>
</tr>
<tr>
<td>No. of visits</td>
<td>3.3 ± 1.3</td>
<td>3.4 ± 1.9</td>
<td>.78</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>30 (61.2)</td>
<td>19 (38.8)</td>
<td>.78</td>
</tr>
<tr>
<td>Female</td>
<td>34 (58.6)</td>
<td>24 (41.4)</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black/Hispanic</td>
<td>10 (33.3)</td>
<td>20 (66.7)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>White</td>
<td>54 (70.1)</td>
<td>23 (29.9)</td>
<td></td>
</tr>
<tr>
<td>Behavioral therapy†</td>
<td>33 (68.8)</td>
<td>15 (31.2)</td>
<td>.09</td>
</tr>
</tbody>
</table>

* Sex, ethnicity, and behavioral therapy values are expressed as number (percentage); all other values are means ± SD. BMI indicates body mass index; GI, glycemic index. Age, BMI, and body weight correspond with baseline data. P values correspond with t tests for means or χ² analysis for percentages. †Values pertain to the number of patients in each group who received behavioral therapy referral.

### Table 2. Changes in BMI and Body Weight According to Dietary Treatment*

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Dietary Treatment</th>
<th>No. of Patients</th>
<th>Unadjusted Means (95% CI)</th>
<th>P</th>
<th>Adjusted Means† (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI change, kg/m²</td>
<td>Low-GI</td>
<td>64</td>
<td>-1.53 (95% CI)</td>
<td>&lt;.001</td>
<td>-1.15 (95% CI)</td>
<td>.001</td>
</tr>
<tr>
<td></td>
<td>Reduced-fat</td>
<td>43</td>
<td>-0.06 (95% CI)</td>
<td></td>
<td>0.06 (95% CI)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(-1.94 to -1.12)</td>
<td></td>
<td>(-1.69 to -0.60)</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>(-0.56 to 0.44)</td>
<td></td>
<td>(0.51 to 0.57)</td>
<td></td>
</tr>
<tr>
<td>Weight change, kg</td>
<td>Low-GI</td>
<td>64</td>
<td>-2.03 (95% CI)</td>
<td>&lt;.001</td>
<td>-1.16 (95% CI)</td>
<td>.007</td>
</tr>
<tr>
<td></td>
<td>Reduced-fat</td>
<td>43</td>
<td>1.31 (95% CI)</td>
<td></td>
<td>-2.64 (95% CI)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(-3.19 to -0.88)</td>
<td></td>
<td>(-2.91 to 0.33)</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>(-0.11 to 2.72)</td>
<td></td>
<td>1.44</td>
<td></td>
</tr>
</tbody>
</table>

* BMI indicates body mass index; GI, glycemic index; and CI, confidence interval. All values are means (confidence intervals), unless otherwise indicated. †Means are adjusted for age, sex, ethnicity, duration of follow-up, behavioral therapy referral, and baseline BMI.
Accepted for publication March 27, 2000.

This study was supported by grants from the Children's Hospital League, Boston, Mass, and the Charles H. Hood Foundation, Boston, Mass. Dr Lenders was supported by grant T32 DK07703, and Dr Ludwig was supported by grant 1K08 DK02440 from the National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, Md.

We wish to thank Drs Richard Malley, MD, Joseph Majzoub, MD, Hans Ritichard, PhD, and Robyn Mehlenbeck, PhD, for advice and encouragement. This work would not have been possible without the contributions of Colleen Kochman, RN, PNP, Linda Olsen, RD, MS, Mary Jane Ott, RN, PNP, and Lillie Santiago to patient care.

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