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Effects of high and low glycemic load meals on energy intake, satiety and hunger in obese Hispanic American youth

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Abstract

Some short-term pediatric studies have suggested beneficial effects of low glycemic load (LGL) meals on feelings of hunger and on energy intake. No systematic studies of the effects of LGL diets have been conducted in obese US Hispanic children even though Hispanic children have a particularly high prevalence of obesity and thus stand to benefit from successful interventions.

Objective—To examine the effects of LGL and high-GL (HGL) meals on appetitive responses and *ad libitum* energy intake of obese Hispanic youth.

Methods—88 obese Hispanic youth ages 7-15y were randomly assigned to consume meals designed to be either LGL (n=45) or HGL (n=43). Following the morning test meal, subjects serially reported hunger, fullness, and satiety using a visual analog scale and provided samples for analysis of serum insulin and plasma glucose. Participants were then fed another test meal and given a snack platter from which to eat *ad libitum*. Energy, macronutrients, and glycemic load (GL) of consumed foods were calculated for each meal.

Results—Subjects in the HGL group had significantly higher insulin ($p=0.0005$) and glucose ($p=0.0001$) responses to the breakfast meal compared to the LGL group. However, there were no significant between-group differences in the total energy consumed from the snack platter (1303 vs. 1368 kcal, $p=0.5$), or in the subjective feelings of hunger ($p=0.3$), fullness ($p=0.5$) or satiety ($p=0.3$) between the two groups.

Conclusions—Our study provides no evidence that, for obese Hispanic youth, changing the GL of the diet affects short-term hunger, fullness, satiety, or energy intake.

Keywords

obese; Hispanic youth; glycemic load; food intake; hunger; satiety; fullness

Introduction

The prevalence of obesity and its complications is high among Hispanic American children and adolescents. In the most recent NHANES data, 23.2% of Hispanic American children

and adolescents were reported as having BMI $\geq 95^{\text{th}}$ percentile for age and sex, an 80% higher prevalence compared to non-Hispanic White children and adolescents (1). Obese Hispanic Americans have also been shown to have a high prevalence of obesity associated co-morbidities, including hyperinsulinemia and impaired glucose tolerance (2,3).

A low glycemic index (LGI) or low glycemic load (LGL) diet has been suggested as an alternate dietary intervention for the treatment of obesity that could also help prevent the development of impaired glucose homeostasis (4-6). The glycemic index (GI) of a test food is defined as the glucose area under the curve (AUC) measured for 2 hours after consumption of 50 g of carbohydrate from the test food divided by the AUC after consumption of 50 g of carbohydrate from a standard food, either white bread or glucose (7). Glycemic load (GL) is the product of glycemic index and the carbohydrate content in the food item. Hence GL is a measure of carbohydrate bioavailability that takes into account the quantity consumed.

Consumption of LGL foods are expected to result in a reduced postprandial rise of insulin (8), thus altering availability of metabolic fuels after a meal (4,5,9). After a high-glycemic index (HGI) / high glycemic load (HGL) meal, blood glucose and insulin levels initially rise much higher than after a LGI/LGL meal (5). High levels of insulin result in stimulation of cellular nutrient uptake, inhibition of hepatic glucose production, and suppression of lipolysis (4,6). Subsequent declines in blood glucose concentration induced by the relative hyperinsulinemia of a HGI diet have been proposed to result in excessive hunger and overeating (4). HGI diets have therefore been hypothesized to promote excessive weight gain (4,5).

Some short-term pediatric studies have shown beneficial effects of LGI /LGL compared to HGI/HGL meals on hunger, satiety, and voluntary food intake (5,10). Because of its salutary effects on postprandial hyperinsulinemia, a LGL diet might be hypothesized to be especially useful in obese Hispanic youth for whom the prevalence of hyperinsulinemia, insulin resistance and type 2 diabetes is particularly high (2,11-15). However, there are no published data examining the effects of LGL meals on appetitive responses in Hispanic children.

The objective of this study was to compare the subjective, hormonal, and metabolic responses of obese Hispanic youth to consumption of LGL and HGL meals under controlled, standardized conditions. We hypothesized that compared to the group fed HGL meals, the LGL group would have significantly lower glucose and lower insulin responses, the LGL group would report less hunger before their meals and greater satiety after their meals, and, as a result, the LGL group would consume less energy from a post-meal *ad libitum* snack platter.

Methods

Setting and Participants

Participants were enrolled in an obesity intervention study, and had completed 12-weeks of the comprehensive program. The intervention was a community-based multidisciplinary weight loss program that consisted of dietary instructions, physical activity, and parenting sessions. The model of delivery included weekly individual and group counseling sessions.

Participants were recruited through advertising at community facilities in Washington, DC. Latino children ages 7 to 15 years with BMI $\geq 95^{\text{th}}$ percentile for age and sex, who were otherwise healthy were eligible. Latino ethnicity was determined by parents, who identified themselves, their spouses and both sets of grandparents with the Hispanic or Latino cultural

group. All participants had a physical examination with assessment of pubertal development at recruitment. Exclusion criteria were any known medical condition or use of medications that would interfere with study objectives or procedures.

The study was approved by the Children's National Medical Center (CNMC) Institutional Review Board. Written consent was obtained from parents of participating children and signed assent from the children and adolescents.

Meal Study Design

A randomized parallel meal study design was adapted from Ludwig et al (5) which required one 24-hour admission for each participant. Participants were randomly assigned to consume a series of three meals designed to be either LGL or HGL.

Participants were instructed not to eat after 2:00 pm on the day of admission and were admitted to the CNMC General Clinical Research Center (GCRC) between 5:30 and 6:00 PM.

Dinner (the first randomized meal) was served at 6:15 PM and completed by 7:00 PM (Table 1). During all test meals, no distractions, television or talking was permitted. After dinner, the subject could drink water but no food or other beverages. Participants were then familiarized with a "hunger and satiety assessment tool," which consisted of a series of visual analog scales asking "how hungry do you feel right now?", "how full do you feel right now?", and "how much food could you eat right now?" The scales were based on previously used tools (16-19). Participants were required to be in bed by 9:30 PM.

Participants were awakened the following morning between 6:00 and 6:15 AM. They were asked to void and change into a hospital gown and undergarments. Height was measured using a wall-mounted stadiometer (SECA 216, Hanover, MD). Weight was measured on a digital scale (Healthometer, Bridgeview, IL). Body mass index (BMI) was calculated as kg/m². Total body fat mass was assessed by air displacement plethysmography (Life Measurement Inc., Concord, Ca) as previously described (20).

A peripheral indwelling venous catheter was placed at 7 AM and fasting baseline serum insulin and plasma glucose were measured. Participants then completed the hunger and satiety assessment tool and were given either an LGL or HGL breakfast (meal 2) between 7:30 and 8:30AM depending on their random assignment. The participants were instructed to complete their breakfast within 15 minutes. The hunger and satiety assessment tool was completed every 30 minutes for the next 5 hours until lunch. Blood samples were drawn at 30, 60, 120, and 300 minutes for serum insulin and plasma glucose. Five hours after breakfast, the intravenous catheter was removed, and the third test meal was given for lunch, which was either LGL or HGL depending on the assigned group. Thirty minutes after the test lunch was consumed, a snack platter was placed in the participant's room. The decision to have the snack platters in the participant's rooms at a set time instead of waiting for the participants to request the platters as in some previous pediatric studies (5,10) was based on the younger age and ethnicity of our study subjects. Due to the relatively unassertive nature and unquestioning respect for authority, or *respeto*, (21) considered a core value in many Latino families, it was felt that the subjects would be unlikely to request additional food in an unfamiliar hospital environment. Subjects were instructed to eat as much as they liked from the platter if they felt hungry. The platter contained identical items for both groups (Table 1). Participants could request additional servings of the food items. Five hours post-lunch, participants completed their final hunger and satiety assessment tool and were discharged from the study.

Composition of Study Meals

All food and beverage items planned for each meal were weighed on a digital food scale (Tanita 1140, Arlington Heights, IL) to the nearest gram to achieve four specific and standardized goals: (1) total energy intake, (2) distribution of energy across the test meals, (3) distribution of macronutrient energy within each meal, and (4) the randomized test GL. The total energy for the three meals was designed to provide 1.2 times each participant's resting energy expenditure, estimated using the Harris Benedict energy equations (22). Total energy intake was distributed, approximately 35% at meal 1 (dinner), 30% at meal 2 (breakfast), and 35% at meal 3 (lunch). For each meal served (except for the *ad libitum* snack platter), the goal was to provide meal GL of less than 50 (g/1000 Kcal) for the LGL group and above 80 (g/1000 Kcal) for the HGL group. The macronutrient composition for the LGL diet was 45-50% of LGI carbohydrates, 20-25% protein, and 30-35% fat. The composition of the HGL diet was 55-60% carbohydrates, 15-20% protein and 25-30% fat. Participants were asked to consume all the food provided for each test meal.

The foods offered were intended to contain many similar items for the LGL and HGL groups as well as some items that differed greatly in GI. The same menu was served for dinner and lunch (Table 1). The *ad libitum* snack platter consisted of commonly available snack foods such as fruits, vegetables, cookies and chips (Table 1). Any left-over food was weighed. Dietary analysis was performed using Nutritionist Pro software (version 4.2, Axxya System, Stafford, TX). GI and GL were calculated using the International Table of Glycemic Index and Load for glucose (23).

Blood Analysis

Serum insulin concentrations were measured by a solid-phase, 2-site chemiluminescent immunometric assay (Immulin 2000 Analyzer, Diagnostic Products, Los Angeles, CA). Plasma glucose was collected in tubes containing a glycolytic inhibitor and measured by the hospital laboratory using the hexokinase-glucose-6-phosphate dehydrogenase method (Dade Behring Inc, Deerfield, IL).

Statistical Methods

A sample size of 88 children was calculated to provide 80% statistical power to detect a 200 kcal (0.4 standard deviation) difference in energy consumption between the two dietary groups with the 2-tailed type 1 error set at $p=.05$.

Analysis was performed using SAS (SAS 9.1 version, SAS Institute Inc, Cary, NC) and Stata® (version 10, StataCorp, College Station, TX). The group difference in energy consumption from the *ad libitum* snack platter was the primary outcome variable used to assess the effects of the dietary treatment modalities. The secondary outcome variables were differences in insulin and glucose response to the test breakfast meal and the differences in subjective feelings of hunger, satiety and fullness following the test breakfast meal. The differences between treatment groups were assessed using longitudinal analysis controlling for baseline values. Incremental areas under the glycemic and insulin response curves were calculated using the pkcollapse method in Stata®.

Results

A total of 88 participants were enrolled, 45 in the LGL and 43 in the HGL group (Table 2). The groups were similar in age, sex, pubertal stage, body composition, and degree of insulin resistance. All enrolled participants completed the study.

Table 3 provides the energy, the GI and GL for the test meals consumed by subjects. There were no significant between-group differences in the total energy consumed at any of the 3 meals, either in absolute terms or as a percentage of the subject-specific energy quantities offered at each meal (Table 3). As intended, the meals consumed by the LGL group, had significantly lower GI and GL than the meals consumed by the HGL group.

Energy consumption during the *ad libitum* snack platter is shown in Table 4. There were no significant between-group differences in the total energy consumed from the *ad libitum* snack platter. A trend in higher protein consumption was observed in the LGL group (Table 4).

Participant's ratings of hunger, fullness and satiety are shown in Figure 1. There were no significant differences in the reported hunger rating (34.2 mm, CI 30.4 to 38.1 mm for LGL vs. 31.2 mm, CI 27.3 to 35.2 mm for HGL, $p=0.3$), fullness rating (31.3 mm, CI 27.0 to 35.5 mm for LGL vs. 29.3 mm, CI 24.9 to 33.7 mm for HGL, $p=0.5$), or satiety rating (36.9 mm, CI 33.1 to 40.6 mm for LGL vs. 33.9 mm, CI 30.1 to 37.8 mm for HGL, $p=0.3$), between the two dietary groups in the time interval between meal 2 (breakfast) and meal 3 (lunch). Additionally, there were no significant differences at any point in the study or after the *ad libitum* snack platter in the reported hunger ($\beta=-2.3$, $p=0.5$), fullness ($\beta=-0.4$, $p=0.3$), or satiety ($\beta=-4.3$, $p=0.2$) ratings between the two dietary groups.

Insulin and glucose responses following the breakfast meal are shown in Figure 2. Participants in the HGL group had significantly higher glucose response to their breakfast test meal compared to the LGL group (28548 mg·min/dL, CI 27771 to 29324 mg·min/dL vs. 26297 mg·min/dL, CI 25678 to 26915 mg·min/dL, $p=0.0001$), indicating that the intended difference in glycemic profile for the LGL and HGL groups was achieved. The mean plasma glucose concentration nadirs after the HGL and LGL test meals were not significantly different (6.3 mg/dL below baseline vs. 4.5 mg/dL below baseline; $p=0.3$). Insulin response was significantly higher in the HGL group than the LGL group after the breakfast test meal (17331 μ IU·min/mL, CI 14198 to 20776 μ IU·min/mL vs. 10021 μ IU·min/mL, CI 7726 to 12615 μ IU·min/mL, $p=0.0001$).

Adverse events

no adverse events were reported.

Discussion

To our knowledge, the present investigation is the first to report the short-term effects of LGL meals in obese Latino children and adolescents. The purpose of this study was to examine the metabolic, hormonal, and appetite responses of obese Latino youth to a LGL or a HGL meal. The HGL and LGL meals in our study were carefully constructed to be comparable in palatability, and to consist of food items with which participants were familiar.

Contrary to our hypotheses, we found no significant differences between the LGL and HGL groups in the energy consumed from the post-lunch *ad libitum* snack platter. Neither the percentage of energy consumed from the total offered at each of the three randomized meals, nor the cumulative energy consumption over the entire study were significantly different between the two groups. We also found no significant differences in the reported perception of hunger, fullness, or satiety between the two dietary groups. Several adult (4) and two pediatric (5,10) studies reported decreased hunger, increased satiety, and decreased voluntary intake in response to LGL/LGL meals. Ludwig et al (5) reported significantly higher ratings of hunger and greater *ad libitum* energy intake after a HGI meal in a

randomized crossover study comparing HGI and LGI meals in 12 adolescent boys. Ball et al (10) reported a 48-minute prolongation of satiety after a LGI versus a HGI supplement in a similar crossover study of 16 adolescents, but found no differences in hunger ratings or changes in actual energy intake. Such crossover meal studies have the advantages of a within-subjects design that controls for many extraneous factors that may complicate human studies. However, because the actual characteristics of the foods or supplements consumed during both conditions are experienced by subjects, such studies may be affected by carryover effects. In addition, it is conceivable that subjects in the crossover studies are more likely than in parallel designs to compare the items consumed under the two conditions and respond to the demand characteristics of the experiment so as to seek to confirm what they believed were the investigator's hypotheses. The present study, which used a randomized, between-subjects design, would be anticipated to be less susceptible to such confounding.

Other possible explanations for the negative findings observed in the present study and the positive findings of other pediatric investigations (5,10) include differences in study population and other design considerations. All our study participants considered themselves Hispanic, whereas predominantly Caucasian youth participated in the previous studies. It is conceivable that the degree of baseline insulin resistance was greater in our cohort and could have blunted the impact of differences in meal GL. Diet-phenotype interactions have been reported in some earlier studies (24,25). The participants in our study were also somewhat younger, with a mean age of 12 years compared to a mean age of 16 years in Ludwig et al (5) and 14 years in Ball et al (10). Another distinction between the current study and the two previous pediatric studies lies in the presentation of the *ad libitum* platters to the participants. In the earlier studies, adolescents had to request a platter, an approach intended to give them the opportunity to pay attention to their internal hunger cues. In the current study, the platters were left in the participants' rooms 30-minutes post-lunch without requiring a request for food. It is conceivable that participants may have consumed what was given to them without necessarily feeling hungry. The composition of the *ad libitum* snack platter was also different from the two earlier pediatric studies. It consisted of food items considered highly palatable and familiar to the study participants. One possible explanation for our findings is that our toxic food environment may so dominate eating behavior that altering factors such as the GI of prior meals may have little effect for those exposed to the highly palatable foods found in many households.

Finally, although the current study was adequately powered to find the previously reported (5) differences in energy intake, it was not powered to detect small energy differences. Appendix Table 1 shows the energy differences that would have been detectable between the two groups in the present study of 88 subjects at 80%, 90%, and 95% power. The LGL group consumed only 5% less energy than the HGL group in contrast to the 81% greater energy consumption in the HGI group reported by Ludwig et al (5). Based on the large standard deviation for energy intake from the *ad libitum* snack platter, a sample size of 1906 participants would have been required to detect the observed 5% difference in intake with 80% power (26). Large standard deviations in parallel-design studies of this nature reflect the large inter-individuals differences in metabolic requirements and feeding behavior among a heterogeneous population.

Consistent with findings from other laboratory studies (10,17), the obese youth in the present investigation consumed large quantities of food from the post-lunch snack platter despite consuming adequate energy at lunch for metabolic needs, demonstrating that deficiencies in meal intake regulation are common in obese children. In addition to the physiologic factors regulating the desire to eat, however, it is important to note that hunger and satiety may be affected by learned behaviors (27). It is possible that obese study

participants have also learned to override their internal hunger and satiety cues. It has been suggested that the desire to eat is regulated more by external cues than by actual hunger, particularly in obese individuals (27).

Study strengths and limitations

The strengths of this study include the relatively large sample size, the broad age range of participants that may increase its generalizability, and the well-controlled study design. However the study was carried out only among obese Hispanic youth and was not powered to detect smaller differences in energy consumption. The sample size however was adequate to detect differences in the appetitive responses between the two dietary groups. Table 1 in the appendix shows the detectable differences for hunger, fullness, and satiety ratings at 80%, 90%, and 95% power. This indicates that the study was capable of detecting quite small differences in hunger, fullness, and satiety ratings between the two dietary groups and would not miss very modest differences in these outcomes. Therefore, it is unlikely that the study's failure to detect such differences was due to inadequate power.

Conclusions

In summary, obese Hispanic youth consuming LGL meals had significantly lower glucose and insulin responses compared to the HGL group. However, there were no significant differences in total energy consumed, or in reports of hunger, satiety or fullness between the two dietary groups. We conclude that these data do not support the hypothesis that a LGL diet suppresses hunger or increases satiety in obese Hispanic children and adolescents. We hypothesize that the effects of a toxic food environment with exposure to a variety of high fat, high energy palatable foods could potentially overwhelm any biological effects of a LGL diet, hence emphasizing the importance of policies advocating for healthy food environments particularly among underserved minority children.

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Figure 1. Mean ($\pm$ SEM) hunger, fullness, and satiety ratings between meal 2 (breakfast) and meal 3 (lunch) controlling for baseline levels. Hunger, fullness, and satiety determined by a 100 mm visual analog scale administered every 30 minutes between breakfast and lunch (16-19). Plot symbols: $\bullet$, Low GL; $\circ$, High GL. P values for differences between the two dietary groups: hunger rating ($p=0.29$); fullness rating ($p=0.53$); satiety rating ($p=0.29$).

Figure 2.
 Mean ($\pm$ SEM) change (Δ) from baseline and IAUC for glucose and insulin concentrations 0-300 min after breakfast test meals.
 Plot symbols: $\bullet$, Low GL; $\circ$, High GL.
 *** p=0.001.

Table 1**Content of Meals Served***

	Low Glycemic Load (LGL)	High Glycemic Load (HGL)
Breakfast	2% milk Yogurt Cheese slices Roasted turkey slices Mayonnaise Margarine Scrambled egg Grapes Low-carbohydrate style 100% whole wheat bread Peanuts Strawberries Water	2% milk Yogurt Cheese slices Roasted turkey slices Mayonnaise Margarine Scrambled egg Grapes Mandarin oranges canned (no juice) White bread Cornflakes cereal Cookie, vanilla wafer Fruit juice
Dinner and Lunch	Roasted turkey slices Cheese slices Mayonnaise Yogurt Low-carbohydrate style 100% whole wheat bread Grapes Mandarin oranges canned (no juice) Peanuts Diet gelatin sugar free Water	Roasted turkey slices Cheese slices Mayonnaise Yogurt White bread Grapes Mandarin oranges, canned (no juice) Cranberry juice Cookie, vanilla wafer
Snack Platter (same food items for both groups) [†]	Oreo cookies, chocolate chip cookies; potato chips; corn chips; pretzels; fresh fruits; canned fruits; fruit rolls, pudding; rice cakes; fresh carrots; grape tomatoes; crackers; cheese crackers; granola bars; fudge cookies	

* The macronutrient composition for the LGL diet was 45-50% of low glycemic index carbohydrates, 20-25% protein, and 30-35% fat. The composition of the HGL diet was 55-60% carbohydrates, 15-20% protein and 25-30% fat.

[†] Approximately 3000 calories was offered on the platter (see methods for energy load of meals).

Table 2

Characteristics of Participants

	Low Glycemic Load (n=45)	High Glycemic Load (n=43)
Age (y) *	12.3±2.4	12.0±2.0
Sex (% female)	51	44
Tanner Pubertal Stage (%)		
1	31.1	23.3
2-3	33.3	37.2
4-5	35.6	39.5
BMI (kg/m ²) *†	29.9±6.3	29.2±4.9
BMI z-score *	2.1±0.4	2.1±0.4
Body fat percent *	42.6±5.9	41.0±7.1
Waist z-score *	1.4±0.7	1.4 ±0.6
HOMA-IR index *±	3.6±2.9	4.2±2.8

* mean ±SD

† BMI, body mass index

± HOMA-IR, Homeostatic model assessment for insulin resistance calculated as the product of fasting plasma glucose concentration (mmol/L) and fasting serum insulin concentration (μU/L) divided by 22.5.(28)

Table 3

Energy and glycemic characteristics of test meals consumed by participants

	Low Glycemic Load (n=45)	High- Glycemic Load (n=43)	p-value
	Mean (95% CI)	Mean (95% CI)	
Meal 1 (Dinner)			
Energy consumed (kcal)	583 (523 - 643)	624 (567 - 681)	0.33
Energy consumed of total offered (%)	88.5 (83.6 – 93.5)	89.2 (83.8 – 94.7)	0.86
Meal glycemic index *	36.5 (35.4 – 37.5)	58.5 (56.8 – 60.2)	0.0001
Meal glycemic load (g/1000 kcal) [†]	40.4 (38.5 – 42.3)	80.8 (77.5 – 84.0)	0.0001
Meal 2 (Breakfast)			
Energy consumed (kcal)	526 (475 – 577)	520 (466 – 575)	0.88
Energy consumed of total offered (%)	90.7 (85.5 – 95.9)	86.6 (80.8 – 92.4)	0.29
Meal glycemic index *	36.0 (34.0 – 38.0)	60.7 (59.4 – 62.0)	0.0001
Meal glycemic load (g/1000 kcal) [†]	40.2 (37.4 – 43.0)	87.4 (81.9 – 92.9)	0.0001
Meal 3 (Lunch)			
Energy consumed (kcal)	626 (572 – 680)	660 (611 – 710)	0.34
Energy consumed of total offered (%)	93.6 (89.7 – 97.4)	93.4 (89.6 – 97.2)	0.96
Meal glycemic index *	35.7 (34.6 – 36.8)	59.3 (58.2 – 60.3)	0.0001
Meal glycemic load (g/1000 kcal) [†]	39.4 (37.4 – 41.4)	81.4 (78.8 – 84.1)	0.0001

* Glycemic index assigned according to published values of glucose reference. Meal glycemic index is the sum of the weighted values for each food item.

[†] Glycemic load is the product of daily glycemic index and total carbohydrate intake adjusted for energy intake.

Table 4Energy and dietary composition of *Ad libitum* snack platter (post lunch) consumed by participants

	Low Glycemic Load (n=45)	High Glycemic Load (n=43)	p-value
	Mean (95% CI)	Mean (95% CI)	
Energy consumed (kcal)	1303 (1143 – 1463)	1368 (1217 – 1518)	0.56
Energy consumed of total offered (%)	55.4 (48.5 – 62.2)	57.1 (51.2 – 63.0)	0.70
Macronutrient composition			
Energy from protein (%)	7.0 (6.1 – 7.8)	5.8 (5.1 – 6.6)	0.053
Energy from carbohydrate (%)	60.2 (58.1 – 62.4)	59.6 (56.9– 62.3)	0.72
Energy from fat (%)	33.7 (31.8 – 35.7)	35.5 (32.8 – 38.3)	0.28
Meal glycemic index *	59.8 (57.9 – 61.9)	61.9 (60.6 – 63.2)	0.08
Meal glycemic load (g/1000 kcal) [†]	89.7 (86.4 – 93.0)	92.2 (87.8 – 96.6)	0.36

* Glycemic index assigned according to published values of glucose reference. Meal glycemic index is the sum of the weighted values for each food item.

[†] Glycemic load is the product of daily glycemic index and total carbohydrate intake adjusted for energy intake.

Appendix Table 1

Detectable difference at various levels of Statistical Power, 2-tailed type 1 error of 5% *

Difference expressed as	sd	≥80% Power	≥90% Power	≥95% Power
Energy Consumed (kcal)	505	303	354	404
Glucose AUC (mg.min/dL)	2,601	1,561	1,821	2,080
Insulin AUC (uIU.min/mL)	10,486	6,292	7,340	8,389
Hunger Rating (mm)	21.5	6.5	7.5	8.6
Fullness Rating (mm)	15.3	4.6	5.4	6.1
Satiety Rating (mm)	15.4	4.6	5.4	6.1

* Based on 10 repeated assessments correlated at 70% within a patient.