Associations of dietary sugar and glycemic index with adiposity and insulin dynamics in overweight Latino youth[1–3]

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ABSTRACT

Background: Few studies have examined the relation between dietary carbohydrate quality, adiposity, and insulin dynamics in children.

Objective: The objective of the study was to determine which aspects of dietary carbohydrate, specifically dietary sugar, fiber, glycemic index, or glycemic load, are associated with adiposity and insulin dynamics in overweight Latino children.

Design: We examined 120 overweight Latino children (10–17 y old) with a family history of type 2 diabetes. Dietary intake was determined by repeated 24-h diet recalls. Adiposity was assessed by using total-body dual-energy X-ray absorptiometry. Insulin dynamics [insulin sensitivity (SI), acute insulin response, and disposition index (an index of β-cell function)] were measured by using a frequently sampled intravenous-glucose-tolerance test.

Results: After adjustment for covariates, total sugar (g/d) was positively correlated with body mass index (BMI; in kg/m^2), BMI z scores, and total fat mass ($r = 0.20$, $r = 0.22$, and $r = 21$, respectively; $P < 0.05$) and negatively correlated with SI and disposition index ($r = -0.29$ and $r = -0.24$, respectively; $P < 0.05$). Dietary fiber, glycemic index, and glycemic load were not significantly correlated with adiposity or insulin dynamics before or after control for covariates. Regression analyses showed that total sugar intake explained an additional 3.4%, 4.6%, and 2.4% of the variance in BMI, BMI z scores, and total fat mass, respectively, and an additional 5.6% and 4.8% of the variance in SI and disposition index ($P < 0.05$), respectively, after control for covariates.

Conclusion: In this cohort, total sugar intake, rather than glycemic index or glycemic load, was associated with higher adiposity measures, lower SI, and lower measures of insulin secretion. *Am J Clin Nutr* 2007;86:1331–8.

KEY WORDS Insulin sensitivity, disposition index, adiposity, Latinos, children, overweight, diet, sugar, glycemic index, carbohydrate

INTRODUCTION

In the Latino population, the rise in pediatric obesity has been closely paralleled by a rise in incidence rates of type 2 diabetes (1). Although total carbohydrate intake encompasses a wide range of food groups, including grains, cereals, fruit, vegetables, and sweets, it appears that foods that are higher in sugar content or those that have a higher glycemic index (GI) and glycemic load (GL) tend to be the more controversial carbohydrate contributors to obesity and related diseases. The 2000 dietary guidelines for sugar distinguished added sugars—ie, sugars that are added to foods or beverages at the table or used as ingredients in processed or prepared foods such as cakes, cookies, and soft drinks—from other types of carbohydrates (2). Added sugar intake, particularly sugar-sweetened beverage intake, has increased steadily as documented by both food supply data and nationwide food consumption survey data (3–5). Many studies have shown that higher intakes of sugar-sweetened beverages are related to greater adiposity in youth (6–9).

Although most investigators agree that excess sugar consumption adversely affects health, there has been renewed attention regarding carbohydrate quality and metabolic disease risk. Carbohydrates are often classified in terms of GI and GL to enable better assessment of their effect on glucose metabolism. GI refers to the quantitative assessment of foods that is based on the postprandial blood glucose response compared with a reference food (white bread or glucose) (10). GL is calculated by multiplying the GI by the amount of carbohydrate consumed (11). Low-GI diets have been associated with less ecreased adiposity in both adults and children (12–14). Randomized interventions with adolescents showed that low-GI diets resulted in significantly lower body mass index (BMI; in kg/m^2), body weight, and fat mass than did standard reduced-fat diets (13, 14).

The risk of type 2 diabetes has also been linked to high intakes of sugar (15, 16), low intakes of dietary fiber (17, 18) and high-GI or high-GL diets (or diets that are high in both GI and GL) (17, 19). However, few studies have examined the association of these dietary variables with insulin dynamics [insulin sensitivity (SI), acute insulin response (AIR), and disposition index (DI; an index of β-cell function)] (20–22), and even fewer have looked

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at this association in a pediatric sample (13, 16). We have previously shown in a longitudinal study of Latino children [the Study of Latino Adolescents at Risk for Diabetes (SOLAR)] that higher intakes of total sugar (which included added and naturally occurring sugars) were associated with lower AIR and DI (the product of AIR and SI) (23), but no comparative analysis of the influence of GI or GL was performed. To our knowledge, no study has compared the effects of dietary sugar, added sugar, fiber, GI, and GL on insulin dynamics in an adolescent population. Therefore, we aimed to examine the association between total sugar, added sugar, fiber intake, GI, and GL on adiposity parameters and insulin dynamics in overweight Latino adolescents. On the basis of our previous findings in this particular population (16), we hypothesized that sugar and fiber intakes would be related to adiposity and insulin dynamics more than would GI or GL.

SUBJECTS, RESEARCH METHODS, AND PROCEDURES

Participants

The design, data collection procedures, and findings of the University of Southern California longitudinal SOLAR cohort have been described in detail elsewhere (19, 22). Although this is a longitudinal cohort that we intend to follow throughout puberty (ie, up to age 18 y), the present study is a cross-sectional analysis of 120 participants ($\bar{x} \pm$ SD age:14.1 $\pm$ 1.7 y old) with available dietary data in years 3–6 of SOLAR. This analysis was different from the previous dietary analyses conducted in this cohort (16) because it captured older children in later maturation stages and was much larger in sample size. Physical characteristics [ie, age, weight, height, BMI, BMI percentile, and body composition (ie, total fat mass and total lean tissue)] and fasting and 2-h glucose data were available for the entire sample, whereas insulin dynamics data were available for only 73% of the sample. At study entry, children were required to meet the following inclusion criteria: *1*) age 8–13 y; *2*) BMI $\geq$ 85th percentile for age and sex according to the guidelines of the Centers for Disease Control and Prevention (23); *3*) Latino ancestry (all 4 grandparents of Latino origin as determined by parental self-report), and *4*) history of type 2 diabetes in $\geq$1 parent, sibling, or grandparent, as determined by parental self-report. Subjects were ineligible if they were taking medications known to affect body composition, had syndromes or diseases known to affect body composition or fat distribution, or had had any major illness at any time. None of the 120 participants included in the present analysis were diabetic when the clinical and dietary data were collected. All procedures were carried out at the University of Southern California General Clinical Research Center.

Written informed consent and assent were obtained from both the parents and the child before testing commenced. The Institutional Review Board of the University of Southern California approved this study.

Body composition

Height and weight were measured to the nearest 0.1 kg and 0.1 cm by using a beam medical scale and a wall-mounted stadiometer, respectively; the average of 2 measurements was used for analysis. BMI and BMI percentiles for age and sex were determined by using EPII 2000 software (version 1.1; Centers for Disease Control and Prevention, Atlanta, GA). Body composition (total fat mass and total lean tissue mass) was assessed with a total-body dual-energy X-ray absorptiometry (DXA) scan conducted with a QDR 4500W scanner (Hologic, Bedford, MA). Tanner stage based on breast stage and pubic hair was assessed during a history and physical examination conducted by a licensed pediatric health care provider with the use of established guidelines (24).

Insulin and glucose dynamics

After an overnight fast, a 2-h oral-glucose-tolerance test (OGTT) was conducted by using a dose of 1.75 g glucose/kg body wt (to a maximum of 75 g). Blood was sampled and assayed for glucose and insulin at −5 min (fasting state) and 120 min (2 h) relative to glucose ingestion. The OGTT results were used to determine normal glucose tolerance (2-h glucose $<$ 140 mg/dL) or impaired glucose tolerance (2-h glucose $\geq$ 140 and $<$ 200 mg/dL), criteria proposed by the American Diabetes Association (18).

Within 1 mo after the OGTT, nondiabetic children were asked to return to the General Clinical Research Center for an overnight visit, during which a frequently sampled intravenous-glucose-tolerance test (FSIVGTT) was performed; this test determines insulin dynamics—ie, SI, AIR, and the DI. The children were served dinner and an evening snack; only water and noncaloric and noncaffeinated beverages were allowed between 2000 and the time of the testing on the following morning, when the FSIVGTT was performed. A topical anesthetic (EMLA cream; AstraZeneca, Wilmington, DE) was applied to the antecubital area of both arms, and, 1 h later, a flexible intravenous catheter was inserted into both arms. Two fasting blood samples, at −15 and −5 min, were obtained for measurement of basal glucose and insulin concentrations. At time zero, glucose (25% dextrose; 0.3 g/kg body wt) was administered intravenously. Blood samples were then collected at 2, 4, 8, 19, 22, 30, 40, 50, 70, 100, and 180 min. Insulin (0.02 units/kg body wt) was injected intravenously at 20 min [Humulin R (regular insulin for human participants); Eli Lilly and Company, Indianapolis, IN]. Plasma glucose and insulin concentrations were measured, and values were entered into MINMOD MILLENNIUM 2003 software (version 5.16; Richard N Bergman, University of Southern California, Los Angeles, CA) for assessment of SI, AIR, and DI.

Glucose from the OGTT was analyzed on a Dimension Clinical Chemistry system by using an in vitro Hexokinase method (Dade Behring, Deerfield, IL). Blood samples from the FSIVGTT were centrifuged [10 min, 2500 RPM, 8–10 °C (model RT6000B; Dupont, Wilmington, DE)] immediately to obtain plasma, and aliquots were frozen at −70 °C until they were assayed. Glucose was assayed in duplicate on an analyzer (model 2700; Yellow Springs Instrument, Yellow Springs, OH) by using the glucose oxidase method. Insulin was assayed in duplicate by using a specific human insulin enzyme-linked immunosorbent assay kit (Linco, St Charles, MO).

Dietary intake

Dietary intake was assessed from two 24-h diet recalls (2 weekdays) using the multiple-pass technique. One recall that included the use of 3-dimensional food models was administered in person by a bilingual dietary technician during the outpatient visit. The second recall was administered via telephone by the

same technician in the week after the visit. Nutrition data were analyzed by using NDS-R software (version 5.0_35; Nutrition Data System for Research, University of Minnesota, Minneapolis, MN). The NDS-R program calculates key dietary variables for this analysis, including total sugars, dietary fiber, added sugar, GI, and GL (using both the standard glucose and the white bread reference). Added sugars are defined as those sugars or syrups added to foods during food preparation or commercial food processing, such as high-fructose corn syrup (HFCS); they do not include naturally occurring sugars such as lactose and fructose (25). GI and GL values were assigned to >18 000 foods in the NDS-R database by using information from a range of currently published reports; the primary source was the 2002 international table of glycemic index and glycemic load values (11), a compilation of studies comprising >750 foods. GI data were selected from the published reports when a study met the following criteria: North American foods, healthy subjects, and a 2-h glucose response. For database foods that were not a match to foods in published reports, GI was either estimated from similar foods or calculated from available carbohydrate amounts and the GI of ingredients within the food.

The dietary data were carefully screened for plausibility through a multiple-step process. Data were first screened by evaluating the participants' comments—ie, recalls in which participants reported being sick or having eaten differently than normal were excluded. Of the 123 participants, 114 had 2 viable recalls without exclusionary comments and 15 had 1 viable recall without exclusionary comments. The clinical and dietary data of those who had 1 recall were compared with the data of those who had 2 recalls by using independent *t* tests; there were no significant differences in clinical characteristics or dietary variables between groups. Subsequently, the dietary data were examined for plausibility of caloric intake by assessing the distribution of the residuals of the linear regression of caloric intake by body weight. Three participants were excluded because of a residual that was >2 SDs from the mean. Thus, 120 participants were included in the present analyses.

Statistical analysis

Dependent variables that were not normally distributed—ie, SI and AIR—were log transformed. For the preliminary analyses, independent *t* tests and chi-square tests were used to assess differences in physical, clinical, and dietary characteristics between the sexes and between the subsamples used. Simple Pearson bivariate correlations were used to assess the simple relations of total carbohydrate, total sugar, added sugar, dietary fiber, GI, and GL to adiposity variables, glucose values, and insulin dynamics. Partial correlations were performed to assess the same correlations independent of sex, Tanner stage, body composition (for glucose and insulin dynamics), energy (kcal/d), and noncarbohydrate macronutrient intake (g/d; ie, total fat and protein intake). Partial scatter plots were produced of the significant relations between dietary variables and adiposity or insulin dynamics (or both), after adjustment for the covariates listed above. Simple and partial correlations were also run to assess the relation of more specific types of sugar and fiber intake—ie, fructose, glucose, sucrose, galactose, lactose, maltose, soluble and insoluble fiber—to adiposity and insulin dynamics. However, all of the sucrose absorbed in the body that arrives at the liver via the portal circulation is free fructose and glucose. To account for this hydrolysis process and to accurately assess the effect that fructose has on adiposity and metabolic variables, we also ran the analyses using 50% of sucrose plus 100% fructose content. In addition, because the NDS-R software does not have the ability to separate out HFCS from fructose content, simple and partial correlations were run to assess the relation of foods and beverages high in free dietary fructose (ie, fruit and 100% fruit juices) and foods high in HFCS (ie, soda, sweetened beverages, desserts, and candies) to adiposity and insulin dynamics.

Hierarchical multiple regression analyses were used to examine the extent to which various dietary factors—in particular, aspects of dietary carbohydrate—predicted the dependent variables of interest. Thus, the sequential process of hierarchical regression allowed us to determine the change in explained variation after each equation and to identify the unique variance in adiposity and insulin dynamics that was due to the various dietary variables. Sex, Tanner stage, and body composition were entered into the model; then energy content and noncarbohydrate macronutrient intake were entered; and, finally, the dietary intake variable of interest was entered. All analyses were performed by using SPSS software (version 11.0; SPSS Inc, Chicago, IL) with significance level set at $P < 0.05$.

RESULTS

Physical and clinical characteristics are shown in **Table 1**. Insulin dynamic information via FSIVGTT was available for only 88 (73%) of the 120 participants. Dietary characteristics are presented in **Table 2**. Independent *t* tests found no significant differences in physical, clinical, or dietary characteristics between the entire sample and the subsample used for insulin dynamics analyses. Independent *t* tests and chi-square tests found very few differences in physical, clinical, and dietary characteristics between males and females. The only variables that differed significantly between males and females were total lean tissue mass (53.5 ± 10.8 and 45.1 ± 7.6 kg, respectively), fasting glucose (91.7 ± 6.7 and 88.9 ± 7.6 μU/mL, respectively), and Tanner staging (3.2 ± 1.4 and 4.1 ± 0.9, respectively). Given that very few sex differences existed in physical or clinical characteristics, sex was used as a covariate in subsequent analyses.

Using simple Pearson bivariate correlations, we found that no dietary variables were significantly correlated with adiposity,

TABLE 1
Subject characteristics[1]

	Value
Boys [*n* (%)]	68 (57)
Age (y)	14.1 ± 1.7[2]
Tanner stage	3.6 ± 1.3
BMI (kg/m^2)	31.4 ± 4.7
BMI *z* scores	2.1 ± 0.4
Total fat (kg)	30.2 ± 9.8
Total lean tissue mass (kg)	49.9 ± 10.4
Fasting glucose (μU/mL)	90.4 ± 7.3
2-h glucose (μU/mL)	118.9 ± 19.3
Insulin sensitivity [×10^{-4} min^{-1}/(μU/mL)][3]	1.63 ± 0.76
Acute insulin response (μU/mL × 10 min)[3]	1461.1 ± 853.2
Disposition index (×10^{-4}/min^{-1})[3]	2059.0 ± 944.3

[1] $n = 120$ except where indicated otherwise.
[2] $\bar{x}$ ± SD (all such values).
[3] $n = 88$.

TABLE 2
Dietary variables[1]

	Value
Energy (kcal)	1825 ± 554
Fat	
(g/d)	66.4 ± 25.3
(% kcal)	32.2 ± 6.3
Carbohydrate	
(g/d)	242.1 ± 83.1
(% kcal)	53.0 ± 8.3
Protein	
(g/d)	70.4 ± 21.4
(% kcal)	16.1 ± 4.6
Total sugars	
(g/d)	113.5 ± 51.9
(% kcal)	24.8 ± 7.6
Added sugars	
(g/d)	69.5 ± 46.0
(% kcal)	15.0 ± 7.9
Dietary fiber	
(g/d)	14.2 ± 6.1
(g/1000 kcal)	8.0 ± 3.0
Glycemic index[2]	59.8 ± 4.9
Glycemic load[2]	137.3 ± 51.6

[1] All values are $\bar{x}$ ± SD. $n = 120$.
[2] Glucose reference.

glucose values, or insulin dynamics. However, after adjustment for covariates (ie, sex, Tanner, body composition, energy intake, and noncarbohydrate macronutrients), total dietary sugar was the only dietary variable significantly correlated with adiposity variables and insulin dynamics. After adjustment for covariates, total sugar intake (g/d) was positively correlated with BMI and BMI z scores ($r = 0.20$, $P = 0.034$; $r = 0.22$, $P = 0.018$, respectively) (data not shown). Total sugar intake (g/d) was also positively correlated with total fat mass (kg) ($r = 0.21$, $P = 0.025$) (**Figure 1**). Total sugar intake was inversely correlated with SI and DI, independent of covariates ($r = -0.289$, $P = 0.010$; $r = -0.244$, $P = 0.039$, respectively) (**Figure 2**). There was also a trend for total sugar intake to be inversely correlated with AIR ($r = -0.220$, $P = 0.054$) (data not shown). There was no significant correlation between the total carbohydrate intake, specific types of sugar intake (ie, fructose, glucose, sucrose, galactose, lactose, and maltose), or fiber intake (ie, insoluble, soluble) and adiposity variables, glucose values, or insulin dynamics, with or without control for covariates. However, after adjustment for the derived fructose content (50% of sucrose + 100% of fructose), there was a significant positive correlation between derived fructose and total fat mass ($r = 0.202$, $P = 0.032$) and an inverse correlation to SI ($r = -0.260$, $P = 0.021$) and DI ($r = -0.231$, $P = 0.042$), independent of covariates. When we examined food servings, there was a significant positive correlation between sweetened beverage intake and total lean tissue mass and between 100%

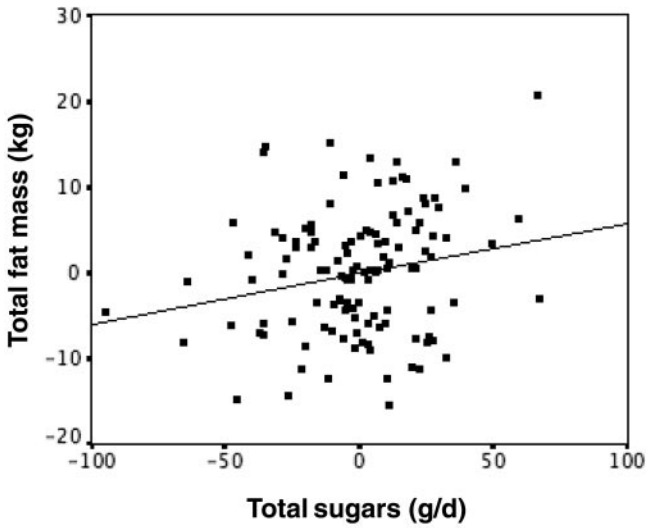

FIGURE 1. $n = 120$; Partial correlation of total sugar intake (g/d) with total body fat (kg) after adjustment for sex, Tanner stage, energy intake per day, noncarbohydrate macronutrient intake per day, and total fat-free mass ($r = 0.21$, $P = 0.025$).

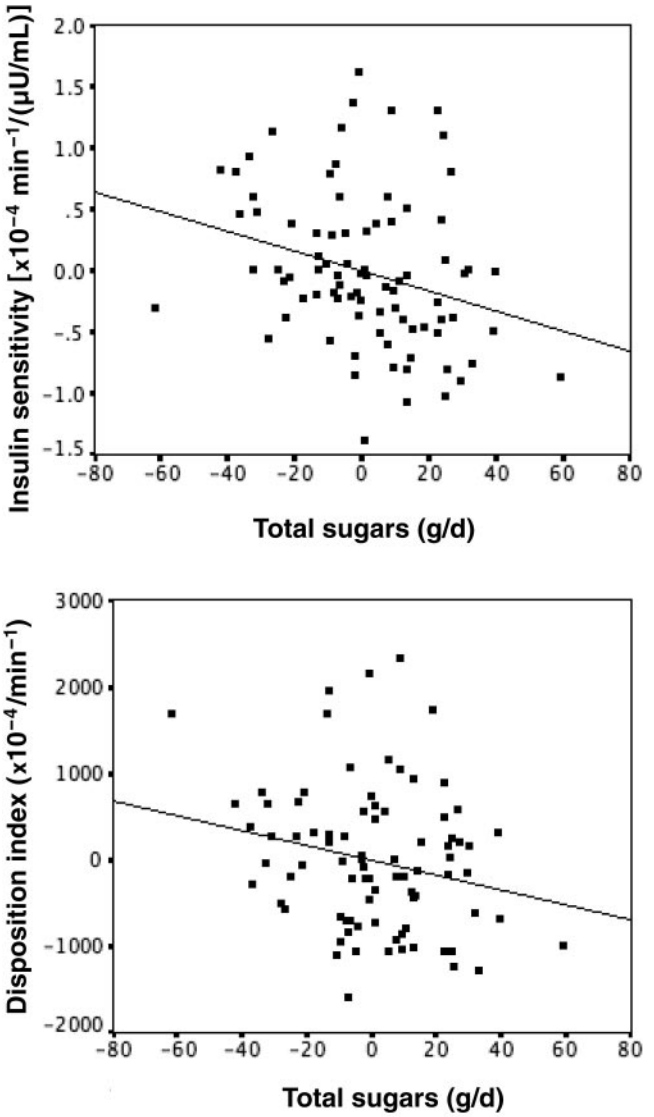

FIGURE 2. Top: partial correlation of total sugar intake (g/d) with insulin sensitivity after adjustment for sex, Tanner stage, energy intake per day, noncarbohydrate macronutrient intake per day, total fat mass, and total fat-free mass ($r = -0.29$, $P = 0.010$). $n = 88$. Bottom: partial correlation of total sugar intake (g/d) with disposition index after adjustment for sex, Tanner stage, energy intake per day, noncarbohydrate macronutrient intake per day, total fat mass, and total fat-free mass ($r = -0.24$, $P = 0.039$). $n = 88$.

fruit juice and total fat mass. However, after control for covariates, there was no significant correlation between any of the food or beverage components and adiposity or insulin dynamics. In addition, neither GI nor GL (using both the standard glucose and the white bread reference) was significantly correlated with adiposity variables, glucose values, or insulin dynamics, with or without adjustment for covariates.

After significant correlations were found with total sugar intake, hierarchical multiple regression analyses were performed to examine the extent to which total sugar intake predicted variances in adiposity variables and insulin dynamics. Once sex, Tanner stage, energy, and noncarbohydrate macronutrient were entered into the model, total sugar intake explained an additional 3.4% of the variance in BMI and an additional 4.6% of the variance in BMI *z* scores (data not shown). Total sugar intake explained an additional 2.4% of the variance in total body fat after control for covariates (**Table 3**), whereas total sugar intake explained an additional 5.6% of the variance in SI (**Table 4**) and an additional 4.8% of the variance in DI (**Table 5**) after control for covariates.

DISCUSSION

We previously showed that Latino youth are more insulin resistant than are their non-Latino white counterparts, independent of adiposity (26). Furthermore, our group has established that 32% of overweight Latino children are prediabetic as defined by impaired fasting or 2-h glucose tolerance (27, 28). The early onset of insulin resistance seen in Latino youth and the inability of β cells to compensate adequately for the high degree of insulin resistance may be factors contributing to the pathogenesis of type 2 diabetes in this ethnic group (26, 29, 30). Dietary carbohydrate quality may contribute to this greater risk. Thus, in the present study, we investigated the relations between carbohydrate quality and both adiposity and insulin dynamics.

A debate continues about which aspect of carbohydrate quality is most related to obesity and the risk of type 2 diabetes. A growing body of evidence indicates that a high intake of added sugars, specifically those in sugar-sweetened beverages, contributes to the pediatric obesity epidemic (6–8, 31). Research in adults has also shown that higher intakes of dietary fiber are linked to less adiposity (22, 32, 33). High GI and GL have also been linked to higher incidence of obesity (14, 34). However, most of the research has been conducted by using less precise measures of adiposity, ie, BMI and waist circumference. Very few studies have evaluated the association of carbohydrate intake with more precise measures of body composition. In a study of 849 Danish children aged 10 y or 16 y, dietary GI and GL were positively associated with body fatness as measured by the sum of 4 skinfold thicknesses in the 16-y-old boys, whereas no associations were found among girls or younger boys (35). In the present study, high intakes of total sugar were positively correlated with increased BMI and BMI *z* scores and with an increase in total fat mass as measured by DXA, after adjustment for physical characteristics, energy, and noncarbohydrate macronutrients.

Few studies have assessed the association between dietary variables, specifically the various aspects of carbohydrate quality, and insulin dynamics, and most of those studies were conducted in adults. For example, diets low in GI and high in dietary fiber are associated with a lower insulin area under the curve (21, 22). In the Insulin Resistance Atherosclerosis Study, dietary fiber was positively associated with SI and DI and inversely associated with fasting insulin after adjustment for lifestyle factors, BMI, and energy intake. Digestible carbohydrates and GL were also associated with SI and insulin secretion, but this relation disappeared after adjustment for energy intake (32). In children, 2 studies have shown that diets high in carbohydrate and low in dietary fat are associated with lower SI, as measured by

TABLE 3
Hierarchical multiple regression of total sugar intake on total fat mass[1]

	R^2	R^2 change	Unstandardized $\beta \pm$ SE[2]	*P* for β
Model 1[3]	0.453	0.453		
Sex			−4.696 ± 0.77	<0.001
Tanner stage			15.466 ± 1.99	<0.001
Total fat-free mass (kg)			0.933 ± 0.10	<0.001
Model 2	0.461	0.008		
Sex			−4.615 ± 0.80	<0.001
Tanner stage			15.395 ± 2.02	<0.001
Total fat-free mass (kg)			0.934 ± 0.10	<0.001
Energy (kg)			2.968E−03 ± 0.003	0.306
Dietary protein (g/d)			−2.874E−02 ± 0.04	0.509
Dietary fat (g/d)			−6.265E−02 ± 0.06	0.307
Model 3[3]	0.485	0.024		
Sex			−4.670 ± 0.78	<0.001
Tanner stage			15.179 ± 1.98	<0.001
Total fat-free mass (kg)			0.922 ± 0.10	<0.001
Energy (kcal)			−6.081E−03 ± 0.01	0.215
Dietary protein (g/d)			1.174E−02 ± 0.05	0.800
Dietary fat (g/d)			3.227E−02 ± 0.07	0.659
Total dietary sugar (g/d)			5.882E−02 ± 0.03	0.025

[1] $n = 120$. Hierarchical multiple regression analysis was used to examine the extent to which total sugar predicted total fat mass.
[2] Regression coefficients for multiple regression analysis of independent variables on total fat mass.
[3] The partial correlation coefficients are significant, $P < 0.05$.

TABLE 4
Hierarchical multiple regression of total sugar intake on insulin sensitivity[1]

	R^2	R^2 change	Unstandardized $\beta \pm$ SE[2]	P for β
Model 1[3]	0.280	0.280		
Sex			−0.140 ± 0.08	0.081
Tanner stage			4.62E−02 ± 0.03	0.096
Total fat-free mass (kg)			−8.94E−03 ± 0.004	0.032
Total fat mass (kg)			−6.21E−03 ± 0.003	0.032
Model 2	0.327	0.047		
Sex			−0.14 ± 0.08	0.072
Tanner stage			5.30E−02 ± 0.03	0.056
Total fat-free mass (kg)			−9.30E−03 ± 0.004	0.026
Total fat mass (kg)			−6.17E−03 ± 0.003	0.032
Energy (kg)			1.16E−04 ± 0.00	0.234
Dietary protein (g/d)			1.25E−03 ± 0.001	0.345
Dietary fat (g/d)			−2.17E−03 ± 0.002	0.267
Model 3[3]	0.384	0.056		
Sex			−0.18 ± 0.08	0.021
Tanner stage			6.54E−02 ± 0.03	0.017
Total fat-free mass (kg)			−1.10E−02 ± 0.004	0.007
Total fat mass (kg)			−4.28E−03 ± 0.003	0.133
Energy (kcal)			4.33E−04 ± 0.00	0.006
Dietary protein (g/d)			1.41E−04 ± 0.001	0.916
Dietary fat (g/d)			−5.39E−03 ± 0.002	0.018
Total dietary sugar (g/d)			−2.14E−03 ± 0.001	0.010

[1] $n = 88$. Statistical analysis was performed by using log-transformed insulin sensitivity; hierarchical multiple regression analyses were used to examine the extent to which total sugar predicted insulin sensitivity.
[2] Regression coefficients for multiple regression analysis of independent variables on insulin sensitivity.
[3] The partial correlation coefficients are significant, $P < 0.05$.

euglycemic-hyperinsulinemic clamp or FSIVGTT (36, 37); however, the type or quality of carbohydrate was not examined.

We previously showed that intakes of sugar and sugar-sweetened beverages were associated with lower AIR and DI in Latino children, independent of adiposity and energy and macronutrient intakes (16). However, we did not find an association of sugar intake with SI, nor did we previously examine the relation between GI or GL and insulin dynamics. In the present study, we extended the previous analysis to include a larger (120 rather than 63 participants) and older (14- rather than 11-y-old) group of children. Consistent with the previous analysis, our results show that total sugar was inversely related to AIR and DI. However, in this larger sample, total sugar intake was also inversely related to SI. In the present study, we did not see an association between type of sugar (ie, glucose, fructose, galactose, or lactose) and adiposity or insulin dynamics with or without control for covariates. However, given that half of sucrose is broken down to fructose during ingestion, the derived fructose content (50% sucrose + 100% fructose) was positively correlated with total fat mass and inversely correlated with SI and DI after control for covariates. Although we would expect that this relation is somewhat driven by the HFCS content, it is not possible to specifically test this hypothesis, given the limitation of the dietary software. However, we were able to examine the relation of foods and beverages high in free dietary fructose (ie, fruit and 100% fruit juices) and high in HFCS (ie, soda, sweetened beverages, desserts, and candies) to adiposity and insulin dynamics. Yet, there was no association between any of the foods and beverages high in either free fructose or HFCS with adiposity and insulin dynamics after control for covariates. These results suggest that, whereas both overall sugar consumption and derived fructose consumption are associated with excess adiposity and reduced SI and β-cell function in this population, we cannot yet tease out which foods and beverages contribute to this association.

There is a great deal of confusion about the interpretation of GI and GL. In the present study, as in the study conducted by Liese et al (32), the GI was relatively low and the variability relatively small. For example, the mean GI was 60 ± 5. Other epidemiologic and intervention studies that have shown a positive association between GI or GL (or both) and adiposity or insulin resistance measures have generally been centered around high-GI (ie ≥ 70) diets (17, 34, 38). Thus, in our population, the GI values may not have been high enough or have exhibited enough variation to show a significant relation with adiposity or insulin dynamics. In addition, with lower GIs and small variations from the mean, the GL is primarily determined by the total amount of carbohydrates. Similar to numerous other studies (17–19, 32), we did not see an association of total carbohydrate intake with adiposity or insulin dynamics in our study, which may explain why no significant association was found for GL.

Both dietary sugar and fiber intakes play a role in determining the GI and GL; however, in the present study, fiber intake is relatively low (14.7 g/d) and accounts for a small percentage of the daily caloric intake (8.0 g/1000 kcal). In contrast, sugar intake is relatively high (114.0 g/d) and accounts for a much larger percentage of the daily intake (25%), which is nearly 50% of the overall carbohydrate intake. National epidemiologic data show a similar intake for dietary fiber but a much lower intake for dietary sugar (39). The excessive amount of sugar intake in this population could explain why sugar intake was the only dietary variable associated with higher adiposity, lower SI, and lower measures of insulin secretion. These results suggest that children with

TABLE 5
Hierarchical multiple regression of total sugar intake on disposition index[1]

	R^2	R^2 change	Unstandardized $\beta \pm$ SE[2]	P for β
Model 1[3]	0.183	0.183		
Sex			−705.30 ± 368.80	0.059
Tanner stage			−16.58 ± 132.02	0.900
Total fat-free mass (kg)			−30.81 ± 19.70	0.122
Total fat mass (kg)			−5.23 ± 13.72	0.704
Model 2	0.200	0.017		
Sex			−727.19 ± 377.77	0.058
Tanner stage			−1.59 ± 134.30	0.991
Total fat-free mass (kg)			−32.08 ± 20.15	0.116
Total fat mass (kg)			−4.78 ± 13.93	0.732
Energy (kg)			0.24 ± 0.48	0.619
Dietary protein (g/d)			2.36 ± 0.48	0.718
Dietary fat (g/d)			−2.45 ± 9.54	0.798
Model 3[3]	0.247	0.048		
Sex			−880.64 ± 375.32	0.022
Tanner stage			50.03 ± 133.19	0.708
Total fat-free mass (kg)			−39.10 ± 19.93	0.053
Total fat mass (kg)			3.07 ± 14.06	0.828
Energy (kcal)			1.56 ± 0.76	0.044
Dietary protein (g/d)			−2.27 ± 6.69	0.735
Dietary fat (g/d)			−15.83 ± 11.13	0.159
Total dietary sugar (g/d)			−8.89 ± 4.05	0.031

[1] $n = 88$. Hierarchical multiple regression analyses were used to examine the extent to which total sugar predicted the disposition index.
[2] Regression coefficients for multiple regression analysis of independent variables on the disposition index.
[3] The partial correlation coefficients were significant, $P < 0.05$.

high intakes of sugar already have early signs of poor β-cell function. We recently found, in a small sample of overweight Latino adolescents, that, after a 12-wk modified carbohydrate nutrition intervention, participants with greater reductions in added-sugar intake had significantly greater improvements in insulin secretion (40). It appears, at least in that population, that sugar intake plays a more integral role in increasing pediatric obesity and risk of type 2 diabetes than does the combination of the carbohydrate foods, as reflected in the GI and GL values. In addition, the concept of GI and GL can be quite confusing to the general public, and even more so in relation to children. A focus on a simpler message, such as reducing sugar intake or increasing dietary fiber (or both), may be a more effective dietary approach to reduce adiposity and type 2 diabetes risk, especially in a pediatric population.

Several limitations of our study should be noted. This study was cross-sectional, and thus cause-and-effect relations of sugar intake on adiposity and insulin dynamics cannot be made. The current study is limited by the use of two 24-h diet recalls; diet recalls rely solely on the participants' self-reporting and are often prone to errors. However, several steps were taken to ensure the accuracy of dietary data, such as using the multiple-pass method, involving well-trained diet technicians, screening for participants' comments, and assessing the plausibility of caloric intake by body weight. In addition, the recalls reflect dietary intakes only on weekdays, and weekend eating is not captured. Nevertheless, one would expect weekend eating to capture unhealthier dietary intakes, which would potentially strengthen our results. Another limitation is that the current study included a relatively small sample of children ($n = 120$), but this limitation is somewhat offset by the use of precise measures of body composition (DXA), insulin dynamics (FSIVGTT), and control for various covariates. This particular sample is extremely homogeneous—ie, overweight Latino children with a family history of type 2 diabetes—and it would be conceivable that, in an already overweight population, individual dietary factors may not have much of an additional effect on adiposity. Considering that we did find an association of sugar intake with adiposity, we can speculate that this association would have been even stronger in a population that included both normal-weight and overweight subjects. However, the homogeneity of the sample in the present study gives us the unique opportunity to assess factors influencing the progression of type 2 diabetes, independent of large variations in adiposity, in a population greatly at risk.

In addition, the NDS-R program is unable to separate out the HFCS content from fructose content. NDS-R also counts sucrose as pure table sugar, and it does not account for the ingestion process, in which sucrose is broken down into fructose and glucose for absorption. This is a limitation, given that the fructose component of dietary sugars has been implicated in the many adverse metabolic effects of dietary sugars, including greater adiposity and insulin resistance (41). However, we attempted to address this limitation by assessing the relation of foods high in free dietary fructose (ie, fruit and 100% fruit juices) and foods high in HFCS (ie, soda, sweetened beverages, desserts, and candies) with adiposity variables and insulin dynamics.

In conclusion, this study is one of the first to show that sugar intake is positively associated with total fat intake and inversely associated with insulin dynamics. These results indicate that modest reductions in sugar intake could have a large effect in reducing obesity and type 2 diabetes risk factors in high-risk

pediatric populations. Novel interventions that focus on reducing simple sugar consumption are warranted in this population.

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