

The relation of sugar intake to β cell function in overweight Latino children¹⁻³

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ABSTRACT

Background: Few studies have investigated the association between sugar intake and insulin dynamics in children, and none have examined this association in overweight Latino youth.

Objective: We aimed to examine the relation between dietary components, especially sugar intake, and insulin dynamics in overweight Latino youth.

Design: We examined 63 overweight Latino children aged 9–13 y. Dietary intake was determined by 3-d records, and body composition was measured with dual-energy X-ray absorptiometry. Insulin sensitivity (S_I), acute insulin response (AIR), and disposition index (an index of β cell function) were measured by using a frequently sampled intravenous-glucose-tolerance test and minimal modeling. Hierarchical regression analysis ascertained the potential independent relation between insulin dynamics and dietary components.

Results: The relation between macronutrient intake and any variable related to insulin dynamics was not significant. However, higher total sugar intake, although not related to S_I , was significantly associated with lower AIR ($\beta = -0.296$, $P = 0.045$) and lower β cell function ($\beta = -0.421$, $P = 0.043$), independent of the covariates age, sex, body composition, Tanner stage, and energy intake. Sugar-sweetened beverage intakes trended toward inverse association with lower AIR ($\beta = -0.219$, $P = 0.072$) and β cell function ($\beta = -0.298$, $P = 0.077$).

Conclusions: In overweight Latino children, higher intakes of sugar and sugar-sweetened beverages were associated with lower AIR and disposition index, which suggested that these children already have early signs of poor β cell function. These results emphasize the need for early nutritional interventions to reduce daily sugar intake in overweight Latino children and potentially reduce their risk for type 2 diabetes. *Am J Clin Nutr* 2005;82:1004–10.

KEY WORDS Latino adolescents, overweight, obesity, sugar, sugary beverages, β cells, disposition index, type 2 diabetes

INTRODUCTION

Latino children in the United States are more likely to be overweight than are non-Latino white children. In 2000, 43.8% of Latinos aged 12–19 y were at risk of overweight [body mass index (BMI; in kg/m^2) \geq 85th percentile for age and sex], and 23.4% were overweight (BMI \geq 95th percentile for age and sex) (1). The increase in obesity parallels the increase in the incidence of the prediabetic state and of type 2 diabetes in overweight adolescents. These combined epidemics are highly prevalent

among certain ethnic groups, including Latinos (2). If left untreated, these risk factors could have disastrous consequences for minority health and the health care costs of future generations.

On the basis of findings in previous studies in adults, the progression of type 2 diabetes is linked to increased adiposity, insulin resistance (3), and the inability of β cells to compensate adequately for insulin resistance (4). Although, to date, very limited research has examined the etiology of type 2 diabetes in youth, the pathophysiology is likely similar to that described in adults. We previously showed that Latino children are more likely to be insulin resistant than are white non-Latino children, independent of adiposity (5). In a cohort of 150 overweight Latino children, 28% had evidence of the prediabetic state or impaired glucose tolerance (6). It is interesting that the disposition index (DI, a proxy measure of β cell function) was the only variable associated with impaired glucose tolerance; ie, subjects with impaired glucose tolerance had significantly lower DI values than did subjects with normal glucose tolerance (5). These results suggest that children in this high-risk population show signs of β cell deterioration, which could lead to the progression of type 2 diabetes.

Dietary factors, specifically sugar intake, may play a prominent role in insulin dynamics and disease risk. In epidemiologic studies, higher sugar intake and sweetened soft-drink consumption were positively associated with increased risk of type 2 diabetes in women (7). McKeown et al (8) found that total dietary fiber, cereal fiber, fruit fiber, and whole-grain intakes were inversely associated with the homeostasis model assessment of insulin resistance. Because these studies were conducted in white adults and because they used fasting glucose or insulin values (or

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² Supported by a Mentor-Based Postdoctoral Fellowship 2005 from the American Diabetes Association (to JND), grant no. R01 DK 59211 from the National Institutes of Health, and grant no. MO1 RR 00043 from the General Clinical Research Center, National Center for Research Resources.

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Received June 7, 2005.

Accepted for publication August 12, 2005.

both) and less precise determinations of body composition with anthropometric values as outcome measures, it is difficult to generalize these findings. To date, few studies have investigated the association between insulin dynamics and dietary components in children, and, to our knowledge, none examined this association with sugar intake in an overweight Latino youth population. Thus, the purpose of the current study was to examine the relation between total sugar intake and insulin dynamics, as ascertained by using a frequently sampled intravenous-glucose-tolerance test (IGTT), in overweight Latino youth. We hypothesized that sugar intake would be negatively associated with insulin sensitivity (S_I) and positively associated with acute insulin response (AIR) and disposition index in this population.

SUBJECTS AND METHODS

Subjects

The current study was conducted in a subcohort of overweight Latino children drawn from the larger cohort recruited in the greater Los Angeles, CA, area to the University of Southern California (USC) SOLAR Diabetes Project through clinics, word of mouth, and local newspaper and radio advertisements. This subcohort consisted of all children for whom dietary intake data were collected. Children were required to meet the following inclusion criteria: age 8–13 y; BMI \geq 85th percentile for age and sex according to the Centers for Disease Control and Prevention (9); Latino ancestry (all 4 grandparents were of Latino origin as determined by self-report); and history of type 2 diabetes in at least one parent, sibling, or grandparent. None of the children were taking medications known to affect body composition, currently had syndromes or diseases known to affect body composition or fat distribution, or had had any major illness since birth. Findings from subsets of this study population have been previously reported (5, 6, 10, 11), but no analysis of dietary intake and its relation with insulin dynamics has been published.

Written informed consent was obtained from both the parents and the children before testing began. The Institutional Review Board of USC approved this study.

Protocol

Children arrived at the USC General Clinical Research Center (GCRC) after an overnight fast. After weight and height were measured, a detailed medical history was obtained, and a physical examination was performed by a physician (including Tanner staging based on breast stage in girls and pubic hair stage in boys; 12, 13). A 2-h oral-glucose-tolerance test (OGTT) was then conducted. Children who met the above inclusion criteria and who did not have type 1 or type 2 diabetes according to the guidelines of the American Diabetes Association; 2) were invited to return for further testing during an inpatient GCRC visit.

Children who completed the outpatient visit and met the inclusion criteria were admitted to the GCRC in the late afternoon for an overnight visit. After admission, total body composition was measured by using dual-energy X-ray absorptiometry (DXA). 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 a frequently sampled IGTT was performed to measure insulin dynamics. All testing was completed by 1200.

Detailed methods

Height to the nearest 0.1 cm and weight to the nearest 0.1 kg were measured by using a beam medical scale and wall-mounted stadiometer, respectively, and the average of 2 measurements was used for analysis. BMI and BMI percentiles for age and sex were ascertained by using EPI-INFO 2000 software (version 1.1; Centers for Disease Control and Prevention, Atlanta, GA). The 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 before (fasting state) and 120 min after (2-h) glucose ingestion. For this study, OGTT data were used only for the measurement of impaired and normal glucose tolerances. Body composition was assessed with a total-body DXA scan, which was performed by a certified Radiological Technologist with a Hologic QDR 4500W (Hologic, Bedford, MA).

Insulin-modified frequently sampled intravenous-glucose-tolerance test

After an overnight fast, a topical anesthetic (EMLA cream; AstroZeneca, Wilmington, DE) was applied to the antecubital area of both arms at \approx 0730; 1 h later, a flexible intravenous catheter was inserted into one of the arms. Two fasting blood samples, at -15 and -5 min, were obtained for measurement of basal glucose and insulin values. At time zero, glucose (25% dextrose; 0.3 g/kg body wt) was administered intravenously. Blood samples were then collected at the following times: 2, 4, 8, 19, 22, 30, 40, 50, 70, 100, and 180 min. Insulin [0.02 units/kg body wt; Humulin R (regular insulin for human subjects), Eli Lilly and Company, Indianapolis, IN] was injected intravenously at 20 min. Plasma was analyzed for glucose and insulin concentrations, and values were entered into the MINMOD MILLENIUM 2003 computer program (version 5.16; RN Bergman, USC, Los Angeles, CA) for determination of insulin sensitivity (S_I), the AIR (the insulin area under the curve above basal for the first 10 min of the frequently sampled IGTT), and the DI (the product of AIR and S_I , a measure of pancreatic β cell function).

Blood samples from the OGTT were separated for plasma and immediately transported on ice to Los Angeles County-USC Medical Center Core Laboratory, where glucose was analyzed on a Dimension Clinical Chemistry system using the Hexokinase method (Dade Behring, Deerfield, IL). Blood samples from the frequently sampled IGTT were immediately centrifuged for 10 min at 2500 rpm and 8–10°C to obtain plasma, and aliquots were frozen at -70 °C until assayed. Glucose was assayed in duplicate by using the glucose oxidase method and a Yellow Springs Instrument 2700 Analyzer (Yellow Springs Instrument, Yellow Springs, OH). Insulin was assayed in duplicate by using a specific human insulin enzyme-linked immunosorbent assay kit (Linco, St Charles, MO).

Dietary intake

Dietary composition was determined by 3-d diet records, obtained on 2 weekdays and 1 weekend day. Records were given to children \approx 1–2 wk before the inpatient GCRC visit. Either a Registered Dietitian or a dietary technician clarified all records by using 2-dimensional life-size food models. Diet records were analyzed with the NUTRITIONIST PRO system (version 2.2.16; Axxya Systems, Indianapolis, IN). A total of 85 subjects completed the diet records, and these records were screened carefully



TABLE 1

Sample characteristics

	Total (n = 63)	Males (n = 32)	Females (n = 31)
Age (y) ¹	11.4 ± 1.7 ²	11.6 ± 1.6	11.2 ± 1.8
Height (cm)	149.4 ± 10.4	151.7 ± 8.7	147.2 ± 11.5
Weight (kg)	63.6 ± 16.2	64.1 ± 14.4	63.1 ± 18.2
BMI (kg/m ²)	28.1 ± 4.6	27.7 ± 4.6	28.5 ± 4.8
Fat mass (kg) ³	24.3 ± 8.6	23.3 ± 7.9	25.4 ± 9.3
Total lean tissue mass (kg) ³	37.1 ± 9.7	39.0 ± 9.9	35.2 ± 9.3
Percentage body fat (%)	37.8 ± 6.4	36.1 ± 6.9	40.2 ± 5.3
Tanner stage ⁴ [n (%)]			
1	23 (36.5)	16 (50.0)	7 (22.6)
2	17 (26.6)	10 (30.3)	7 (22.6)
3	5 (7.8)	1 (3.0)	4 (12.9)
4	13 (20.3)	3 (9.1)	10 (32.3)
5	5 (7.8)	2 (6.1)	3 (9.7)
Insulin sensitivity ³ [$\times 10^{-4}$ min ⁻¹ /(μ U/mL)]	1.96 ± 1.14	2.04 ± 1.13	1.87 ± 1.16
Acute insulin response ³ (μ U/mL)	1818.0 ± 1341.9	1920.3 ± 1513.3	1712.4 ± 1166.8
Disposition index ³ ($\times 10^{-4}$ min ⁻¹)	2623.9 ± 1225.4	2852.0 ± 1348.1	2388.6 ± 1054.8

¹ *t* Tests were used to compare differences in continuous variables between males and females. No significant differences were found.

² $\bar{x} \pm$ SD (all such values).

³ Statistical comparisons on nonnormally distributed variables were performed by using log-transformed data, but data are shown as nontransformed values for ease of interpretation.

⁴ The overall Tanner stage was significantly different between males and females, $P < 0.05$ (chi-square test).

by using several criteria for inclusion in the main analysis: reported energy intake within 2 SD from the predicted energy requirements derived from the new dietary reference intakes (14) based on age, sex, and body weight and assuming a low level of physical activity on any of the 3 d (1 subject excluded); the absence of dietary data for any of the 3 d (9 subjects excluded); the reporting of <2 meals on any of the 3 d (7 subjects excluded); or the reporting of no beverages (including water) on any of the 3 d (5 subjects excluded). After the application of these criteria, 22 children were excluded, which resulted in a final sample of 63. A complete nutrient analysis, including macronutrients and micronutrients (eg, fiber or calcium) and Food Guide Pyramid servings per day were generated by using the NUTRITIONIST PRO system. Sugar carbohydrates were defined as any amount (in grams) of glucose, sucrose, fructose, lactose, or galactose found in food products. Nonsugar carbohydrates were calculated by subtracting sugar carbohydrates (g/d) from the total carbohydrate (g/d) amount. Sugar-sweetened beverages included any beverage containing added sugar—ie, partial fruit juice [eg, Kool-Aid (Kraft General Foods, Glenview, IL) or Hi-C punch (Coca-Cola Co, Atlanta, GA)], soda, sweetened tea or coffee, sports drinks (eg, Gatorade; Quaker Oats Co, Chicago, IL), and chocolate milk. All dietary analysis was based on the average of the 3 d.

Statistical analysis

Data were analyzed by using SPSS software (version 11.0; SPSS Inc, Chicago, IL). Variables that were not normally distributed—ie S_1 , DI, AIR, body composition (fat mass and total lean tissue mass), and total fat intake—were log transformed. Independent-sample *t* tests and analysis of covariance were used to assess differences in physical characteristics, insulin dynamics, and dietary intakes between males and females. Hierarchical multiple regression analyses were employed to examine the extent to which various dietary factors, particularly

nonsugar and sugar carbohydrates, predicted the dependent variables S_1 , AIR, and DI. 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 insulin dynamics that was due to total sugar intake. First, sex, age, Tanner stage, fat mass, and total lean tissue mass were entered into the model. In addition, S_1 was entered in the analysis with AIR as the dependent variable. Next, energy (kcal/d) was entered, and then the food components, including macronutrients (carbohydrates, protein, and fat), micronutrients (calcium and fiber), Food Guide Pyramid servings (grain, meat, dairy, and fruit and vegetable servings/d), energy density (energy intake divided by total grams of either food or beverage), nonsugar and sugar carbohydrates, and sugar-sweetened beverages, were entered separately. Accepted statistical significance was $P < 0.05$.

RESULTS

Background characteristics for each sex, including physical variables and insulin dynamics, are shown in **Table 1**. Mean Tanner stage was the only physical characteristic that differed significantly between males and females. There were no sex differences in insulin dynamics. Dietary intakes for each sex are shown in **Table 2**. In addition, there were no significant differences in dietary intake between males and females. Thus, we pooled data across sex for further analysis.

Hierarchical multiple regression found that macronutrients (ie, carbohydrate, protein, and fat, expressed in g/d), micronutrients (ie, calcium and fiber, expressed in g/d), energy density, and all Food Guide Pyramid servings a day were not significantly associated with any of the insulin dynamic variables. However, when the subtypes of carbohydrates were examined, sugar intake (g/d) was the only dietary component significantly related to insulin dynamics, independent of sex, age, body composition, Tanner stage, and total energy intake. Sugar carbohydrate intake

TABLE 2Dietary composition of the study sample¹

Nutrient composition ²	Total (n = 63)	Males (n = 32)	Females (n = 31)
Energy (kcal/d)	1934.5 ± 490.4	2039.7 ± 494.1	1825.9 ± 469.8
Protein			
(g/d)	78.3 ± 21.0	82.6 ± 22.5	73.9 ± 18.8
(% of kcal/d)	16.5 ± 3.3	16.4 ± 3.2	16.5 ± 3.5
Carbohydrate			
(g/d)	251.4 ± 67.8	263.0 ± 68.8	240.6 ± 66.0
(% of kcal/d)	52.2 ± 5.5	51.6 ± 5.7	52.8 ± 5.3
Total dietary fat			
(g/d)	71.8 ± 24.3	77.4 ± 24.1	66.0 ± 23.5
(% of kcal/d)	33.0 ± 5.4	33.9 ± 5.1	32.03 ± 5.5
Dietary fiber (g/d)	15.7 ± 5.7	17.0 ± 6.3	14.5 ± 4.7
Sugar carbohydrates			
(g/d) ³	103.4 ± 40.3	109.5 ± 37.8	98.2 ± 42.6
(% of kcal/d)	21.2 ± 5.4	21.3 ± 4.5	21.1 ± 6.3
Nonsugar carbohydrates			
(g/d) ⁴	148.0 ± 36.5	153.5 ± 39.1	142.4 ± 33.3
(% of kcal/d)	30.6 ± 7.5	30.0 ± 7.6	31.2 ± 7.3
Sugar-sweetened beverages			
(servings/d) ⁵	2.3 ± 1.3	2.6 ± 1.3	2.0 ± 1.3
(mL/d)	552 ± 312	654 ± 312	480 ± 312

¹ All values are $\bar{x} \pm$ SD.² *t* Tests found no significance differences between males and females in any dietary intake variables.³ Sugar carbohydrates are defined as carbohydrates containing glucose, sucrose, fructose, lactose, or galactose.⁴ Nonsugar carbohydrates were calculated by subtracting sugar carbohydrates (g/d) from the total carbohydrate (g/d) amount.⁵ Sugar-sweetened beverages included any beverage containing added sugar: partial fruit juice (eg, Kool-Aid or Hi-C punch), soda, sweetened tea or coffee, sports drinks (eg, Gatorade), and chocolate milk.

(g/d) explained 5.9% of the variance in AIR ($\beta = -0.296$, $P = 0.045$) (Table 3) and 12.0% of the variance in DI ($\beta = -0.421$, $P = 0.043$) (Table 4). In other words, higher total sugar intake was associated with lower AIR and decreased β cell function. Total sugar intake was not associated with S_I . Nonsugar carbohydrate intake was not associated with S_I , AIR, or DI. When sugar carbohydrate intake was further examined in representative components, sugar-sweetened beverages made up 40% of total sugar intake. Thus, sugar-sweetened beverages were entered into the regression model in place of nonsugar and sugar carbohydrate intakes, so that it could be assessed whether the variation could be explained by sugar-sweetened beverage consumption rather than by total sugar intake. There was a trend for sugar-sweetened beverages (servings/d) to explain 2.4% of the variance in AIR ($\beta = -0.219$, $P = 0.07$) (Table 3), independent of sex, age, body composition, Tanner stage, and total energy intake. There was also a trend for sugar-sweetened beverages to explain 4.6% of the variance in DI ($\beta = -0.298$, $P = 0.08$) (Table 4).

Sex differences in the association between sugar carbohydrate intake (g/d) and DI are shown in Figure 1. There was a significant sugar carbohydrate effect for DI ($P = 0.043$). The sex effect and the sex \times sugar carbohydrate interaction were not significant for DI (both: $P > 0.05$). Thus, the males and females were analyzed together, and the relation shows that the greater the total sugar intake, the lower the DI or β cell function. The sex differences between sugar-sweetened beverages (servings/d) and DI are also depicted in scatter-plot form for ease of interpretation (Figure 2). There was a trend for a sugar-sweetened beverage effect for DI ($P = 0.077$). The sex effect and the sex \times sugar-sweetened beverage interaction for DI were not significant ($P >$

0.05). The relation shows that the greater the sugar-sweetened beverage intake, the lower the DI or β cell function. Similar results were seen with scatter plots depicting the relation between the intake of sugar carbohydrate or sugar-sweetened beverages and AIR (data not shown).

DISCUSSION

We have previously shown that the Latino children are more likely to be insulin resistant than are white children, independent of differences in adiposity (5). Greater insulin resistance increases the secretory demands placed on β cells, which has been shown to lead to progressive β cell dysfunction in some populations (15). Over time, it is this deterioration of β cells in susceptible persons, such as Latinos, that may be a factor contributing to the pathogenesis of type 2 diabetes (5, 15). Thus, it is logical to extend these initial findings to ascertain the potential association of dietary components with increased insulin secretion and β cell function. The disposition index, the product of S_I and insulin secretion, has been identified as a measure of the overall compensatory response of β cells to insulin resistance (16). A higher DI represents overcompensation (high insulin secretion relative to the degree of insulin resistance), whereas lower values represent an inability of the pancreas to secrete enough insulin at the level of insulin resistance. We originally hypothesized that our subjects, all of whom are at risk of type 2 diabetes because of family history, ethnicity, and overweight status, would have high insulin secretion levels—ie, high AIR and DI—in a compensatory response to high intakes of sugar. However, contrary to our hypothesis, regression analysis found that subjects with high sugar intakes had significantly lower

TABLE 3Multiple regression of sugar and nonsugar carbohydrates and sugar-sweetened beverages on log acute insulin response¹

	R ²	Standardized β^3	P value for β
Model 1 ⁴	0.58		
Sex		-0.077	NS
Age		-0.112	NS
Total lean tissue mass (kg) ⁵		0.063	NS
Total fat mass (kg) ⁵		-0.111	NS
Tanner stage		-0.172	NS
S ₁		-0.873	0.001
Model 2	0.59		
Sex		-0.057	NS
Age		-0.142	NS
Total lean tissue mass (kg)		0.081	NS
Total fat mass (kg)		-0.099	NS
Tanner stage		-0.184	NS
S ₁		-0.853	0.001
Energy		0.071	NS
Model 3 ⁴	0.60		
Sex		-0.038	NS
Age		-0.136	NS
Total lean tissue mass (kg)		0.080	NS
Total fat mass (kg)		-0.131	NS
Tanner stage		-0.209	NS
S ₁		-0.839	0.001
Energy		0.132	NS
Nonsugar carbohydrate (g/d)		0.222	NS
Sugar carbohydrate (g/d)		-0.296	0.045
Model 4 ⁶	0.60		
Sex		-0.075	NS
Age		-0.100	NS
Total lean tissue mass (kg)		0.045	NS
Total fat mass (kg)		-0.853	NS
Tanner stage		-0.201	NS
S ₁		-0.895	0.001
Energy		0.205	NS
Sugar-sweetened beverages (servings/d)		-0.219	0.072

¹ *n* = 63. S₁, insulin sensitivity.² Partial correlation coefficients for each model are included in the multiple regression analysis.³ Regression coefficients (β) for multiple regression analysis of dietary intake on log acute insulin response.⁴ The partial correlation coefficients are significant, *P* < 0.05.⁵ Statistical analyses were performed by using the log-transformed variable.⁶ For the partial correlation coefficient, *P* for trend = 0.07.

insulin secretion and worse β cell function than did those with lower sugar intakes (Table 3 and 4). These results suggest that high levels of sugar intake may already be contributing to an exacerbation of β cell secretion.

The relation between dietary components and S₁ or insulin resistance is still unclear. Many adult studies have shown that diets that are low in glycemic index and high in dietary fiber intake are associated with increased S₁ (8, 17, 18). Glycemic index has also been positively associated with the risk of type 2 diabetes in women (19) and men (20). However, these studies used fasting samples of glucose or insulin (or both) and a different expression of the carbohydrate variables, so these results are not directly applicable to the current study. In contrast, an 8-wk

TABLE 4Multiple regression of sugar and nonsugar carbohydrates and sugar-sweetened beverages on log disposition index¹

	R ²	Standardized β^3	P for β
Model 1 ⁴	0.08		
Sex		-0.112	NS
Age		-0.169	NS
Total lean tissue mass (kg) ⁵		0.101	NS
Total fat mass (kg) ⁵		-0.134	NS
Tanner stage		-0.231	NS
Model 2	0.08		
Sex		-0.081	NS
Age		-0.207	NS
Total lean tissue mass (kg)		0.120	NS
Total fat mass (kg)		-0.132	NS
Tanner stage		-0.258	NS
Energy		0.104	NS
Model 3 ⁴	0.18		
Sex		-0.054	NS
Age		-0.193	NS
Total lean tissue mass (kg)		0.113	NS
Total fat mass (kg)		-0.188	NS
Tanner stage		-0.298	NS
Energy		0.188	NS
Nonsugar carbohydrate (g/d)		0.317	NS
Sugar carbohydrate (g/d)		-0.421	0.043
Model 4 ⁶	0.21		
Sex		-0.105	NS
Age		-0.166	NS
Total lean tissue mass (kg)		0.087	NS
Total fat mass (kg)		-0.122	NS
Tanner stage		-0.267	NS
Energy		0.297	0.092
Sugar-sweetened beverages (servings/d)		-0.298	0.077

¹ *n* = 63.² Partial correlation coefficients for each model are included in the multiple regression analysis.³ Regression coefficients (β) for multiple regression analysis of dietary intake on log disposition index.⁴ The partial correlation coefficients are significant, *P* < 0.05.⁵ Statistical analyses were performed by using the log-transformed variable.⁶ The partial correlation coefficient, *P* for trend = 0.06.

intensive feeding study using the frequently sampled IGTT method found that dietary fiber intake had no effect on any of the insulin values (ie, S₁, AIR, or DI) in 20 elderly women (21). Another study that used frequently sampled IGTT measurements found total fat intake to be inversely related to S₁ in adults (*n* = 1173), but this association was not significant after adjustments for BMI and waist-hip ratio.

Few studies have examined the relation between dietary patterns and insulin dynamics in children (22–24). One study found that a high ratio of dietary fat to carbohydrate is correlated with lower S₁ as measured by euglycemic-hyperinsulinemic clamp in normal-weight African American and white children (22). Similarly, another study found that children fed a high-carbohydrate, low-fat diet had greater S₁, as measured with the frequently sampled IGTT, than did those fed a low-carbohydrate, high-fat diet (24). Both studies found that low fat and high carbohydrate intakes were associated with greater S₁, but the effect of the type of carbohydrate was not examined. We previously reported that higher dietary fat intakes in African American children (aged

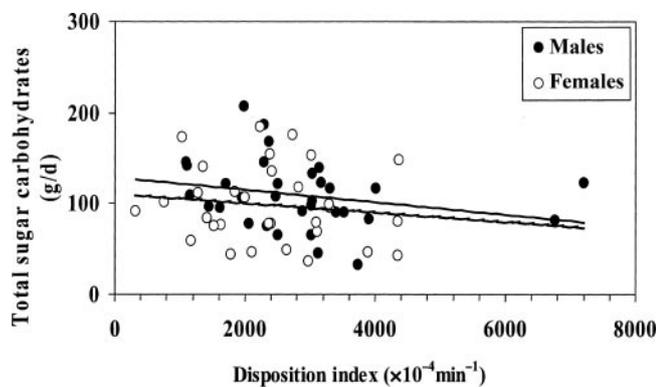


FIGURE 1. Sex differences in the association between sugar carbohydrate intake (g/d) and disposition index (DI). There was a significant sugar carbohydrate effect for DI ($P = 0.043$). The sex effect and the sex \times sugar carbohydrate interaction for DI were not significant (both: $P > 0.05$). The relation shows that the greater the total sugar intake, the lower the DI or β cell function ($R^2 = 0.18$, $P = 0.014$). Statistical comparisons were made by using the log-transformed DI, but data are shown as nontransformed values for ease of interpretation.

7–14 y) were associated with lower S_{I1} , with the use of frequently sampled IGTT measurements and independent of body fat, sex, and Tanner stage (25). In another study conducted by our group, we also found that carbohydrate intakes were positively associated with S_{I1} in African American and white children (23). This finding extended previous results and showed both that the number of servings of fruit per day was positively associated with S_{I1} and that the number of servings of vegetables per day was negatively associated with AIR. In the current study of Latino youth, we did not detect any significant relations between dietary variables and S_{I1} , nor was there an association of food servings (ie, fruit, vegetable, dairy, meat, or grain) per day, dietary fiber, total fat, or monounsaturated, polyunsaturated, or saturated fat with any of the insulin dynamic measurements.

High sugar intake in this population, which accounts for >40% of the total carbohydrate intake, prompted us to look specifically at the association between the type of carbohydrate consumed and insulin dynamics. When carbohydrates were divided into nonsugar and sugar carbohydrates, sugar intake was

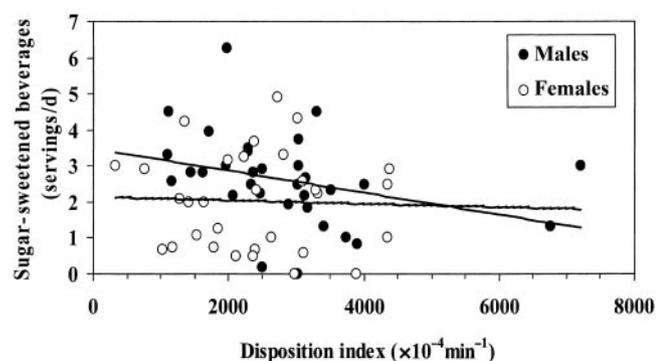


FIGURE 2. Sex differences in the association between sugar-sweetened beverage consumption (servings/d) and disposition index (DI). There was a trend for a sugar-sweetened beverage effect for DI ($P = 0.077$). The sex effect and the sex \times sugar-sweetened beverage interaction for DI were not significant (both: $P > 0.05$). The relation shows that the greater the sugar-sweetened beverage intake, the lower the DI or β cell function ($R^2 = 0.21$, $P = 0.06$). Statistical comparisons were made by using the log-transformed DI, but data are shown as nontransformed values for ease of interpretation.

inversely associated with insulin secretion. We also noted that almost half of this sugar intake is derived from sugar-sweetened beverages; that is, the average child consumes ≈ 2.5 servings/d (ie, ≈ 550 mL/d). Studies have found that sugar-sweetened beverages are associated with obesity in children (26, 27) and with type 2 diabetes in adults (28). In the current study, sugar-sweetened beverage intakes were also inversely associated to insulin secretion, which suggests that sugary beverage consumption was the driving force behind the inverse relation between total sugar intakes and insulin secretion. These results imply that a reduction in sugar-sweetened beverage consumption could potentially exert positive effects on insulin dynamics, although this hypothesis remains to be tested in intervention studies.

Several limitations of our study should be considered. This study was cross-sectional and thus cause-and-effect relations of sugar intake and insulin dynamics are precluded. Intervention studies are needed to ascertain the magnitude and direction of the effect of sugar intake on insulin action throughout childhood. The current study is limited by the use of self-reported 3-d diet records, which rely solely on the subjects' self-reporting and are often prone to errors, such as underrecording or underreporting. We took several steps to improve the accuracy of the records, such as having a dietitian or dietary technologist meet individually with each subject to clarify entries; using large, colorful, 2-dimensional models to depict accurate portion identification; and creating stringent exclusion criteria with respect to overreporting and incomplete or unusual diet records. Another limitation is that this study did not control for physical activity, which has been found to improve insulin dynamics (29), specifically S_{I1} (30, 31). In contrast, we showed in previous reports from this cohort that physical activity has a minimal effect on insulin dynamics after control for body composition and other confounders (11). However, we intend in future studies to measure physical activity levels and to assess whether the relation between those levels and dietary variables, specifically sugar intake, is still associated with insulin dynamics after control for physical activity.

Another limitation of this study is that we did not examine the association of glycemic index with insulin dynamics. Yet the consumption of sugar-sweetened beverages, which would have similar glycemic index values, appeared to be driving the inverse relation between total sugar intake and insulin secretion. The homogeneity of the habitual diet in this population could also be considered a limitation, mainly because certain nutrients and food groups that were previously associated with insulin dynamics—eg, dietary fiber, fruit, vegetables, and whole grains—were present in such small amounts that a significant association was not found. However, it is precisely this homogeneity, specifically the excessive intake of total sugars and sugar-sweetened beverages, that appears to be negatively affecting insulin secretion and that is quite possibly the underlying factor contributing to the risk of type 2 diabetes in this high-risk population. Finally, the current study employed a relatively small sample of children ($n = 63$). The limitations of the small sample size are somewhat offset by the use of precise measurements of body composition (DXA) and insulin action (frequently sampled IGTT), a progressive statistical method, and an understudied high-risk population.

In summary, this is the first study to examine the association between sugar intake and insulin action in overweight Latino

children. We noted an inverse association between sugar carbohydrate intake and sugar-sweetened beverages with β cell compensation. Modest reductions in this sugar intake could potentially preserve β cell function and prevent metabolic disorders in these children. Public health strategies to prevent obesity and type 2 diabetes, in particular dietary interventions, should focus on reducing simple sugar consumption in this population. 

We are grateful to the project coordinator, Quintilia Avila, and to the nurses and nutrition staff at the University of Southern California—General Clinical Research Center. We are also grateful to the children and their families for making this study possible.

JND was responsible for the dietary data entry and analysis, statistical analysis, and manuscript preparation; EEV assisted with dietary entry and manuscript preparation; MJW was the physician responsible for all medical procedures and tests in this study (study physician of this project) and assisted with evaluation and interpretation of the data and manuscript preparation; GDCB assisted with dietary analysis and data entry and manuscript preparation; MLC assisted with evaluation and interpretation of the data, minimal modeling of insulin sensitivity, and manuscript preparation; GQS assisted with evaluation and interpretation of the data and manuscript preparation; MIG was the Principal Investigator for this project and oversaw and managed all aspects of this study. None of the authors had any financial or personal conflict of interest.

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