Inverse relation between dietary fiber intake and visceral adiposity in overweight Latino youth[1–3]

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ABSTRACT

Background: To date, no studies have assessed the longitudinal changes of dietary intake on metabolic risk factors in Latino youth.

Objective: We assessed the relation between changes in dietary intake, specifically sugar and fiber intakes, with changes in adiposity and risk factors for type 2 diabetes in a longitudinal analysis of overweight Latino youth.

Design: Overweight Latino youth ($n = 85$; aged 11–17 y) underwent the following measures over 2 y [mean ($\pm$SD) time difference was 1.5 ± 0.5 y]: dietary intake by 2-d diet recalls, body composition by dual-energy X-ray absorptiometry and magnetic resonance imaging, and glucose and insulin indexes by oral- and intravenous-glucose-tolerance tests. Partial correlations and repeated-measures analysis of covariance assessed the relation between changes in dietary intake with changes in adiposity and glucose and insulin indexes, independent of the following a priori covariates: sex, Tanner stage, time between visits, and baseline dietary and metabolic variables of interest.

Results: Increases in total dietary fiber (g/1000 kcal) and insoluble fiber (g/1000 kcal) were associated with decreases in visceral adipose tissue (VAT) ($r = -0.29$, $P = 0.02$, and $r = -0.27$, $P = 0.03$, for total dietary and insoluble fiber, respectively), independent of baseline covariates and change in subcutaneous abdominal adipose tissue. Participants who had decreased total dietary fiber (mean decrease of $3 \text{ g} \cdot 1000 \text{ kcal}^{-1} \cdot \text{d}^{-1}$) had significant increases in VAT compared with participants who had increased total dietary fiber (21% compared with -4%; $P = 0.02$). No other changes in dietary variables were related to changes in adiposity or metabolic variables.

Conclusion: Small reductions in dietary fiber intake over 1–2 y can have profound effects on increasing visceral adiposity in a high-risk Latino youth population. *Am J Clin Nutr* 2009;90:1160–6.

INTRODUCTION

Latino youth are more likely to be at risk of overweight than are white youth, with 40.5% of Latino boys and 37.1% of Latino girls (12–19 y of age) being overweight ($\geq$85th Centers for Disease Control and Prevention percentile) compared with 34.5% of white boys and 31.7% of whites girls (1, 2). Our group has shown that overweight Latino children (8–14 y of age) in the Los Angeles area are insulin resistant and >30% have pre-diabetes (3, 4), the metabolic syndrome (5), or both. The early onset of insulin resistance, as seen in Latino youth and the inability of β cells to compensate adequately to the high degree of insulin resistance may be factors that contribute to the pathogenesis of type 2 diabetes in this ethnic group (6–8).

In the past decade, the decline in carbohydrate quality has been identified as a likely culprit leading to increased adiposity and metabolic disorders among children and adults (9). Specifically, the consumption of added sugars, which are sugars that are eaten separately at the table or used as ingredients in processed or prepared foods (ie, cakes, cookies, and soft drinks), has increased steadily as documented by both food supply data and nationwide food consumption survey data (10–12). Many studies have shown that higher intakes of total sugar and sugar-sweetened beverages are related to increased adiposity in youth (13–16). In a cross-sectional analyses, we have shown that increased intake of added sugar was the only variable associated with increased total fat mass and decreased insulin secretion and β cell function in Latino youth (10–17 y of age) (17, 18). We have also recently shown that, in a 16-wk nutrition and exercise randomized controlled trial with overweight Latino adolescents, participants who decreased their added sugar intake had a 33% improvement in insulin secretion (19).

In contrast, dietary fiber appears to play a protective role against excess adiposity and metabolic disorders in both adults (20–22) and children (23, 24). Although the healthy reputation of dietary fiber continues to grow, national data consistently show that children consume less than one-half of the recommended amount of dietary fiber, which is 14 g fiber $\cdot 1000 \text{ cal}^{-1} \cdot \text{d}^{-1}$ (24, 25). One study showed that increased dietary fiber intake, primarily through increased whole-grain consumption, was associated with lower body mass and greater insulin sensitivity among adolescents (23). We have also shown in a cross-sectional analysis of Latino youth that total dietary fiber, specifically soluble dietary fiber, was associated with lower waist circumference and the metabolic syndrome. We have also shown that, in a 16-wk nutrition and exercise randomized controlled trial, participants who

increased their fiber intake had a 10% reduction in visceral adipose tissue (VAT) (19).

To our knowledge, no study has investigated observational, longitudinal changes in dietary variables, specifically sugar and fiber intakes, with changes in adiposity and metabolic disorders in high-risk Latino youth. Therefore, the purpose of this study was to examine changes in dietary variables over 1 or 2 y with changes in adiposity variables and glucose and insulin indexes in overweight Latino youth. On the basis of our cross-sectional and intervention findings, we hypothesized that reductions in sugar intake and increases in dietary fiber intake would be related to reductions in obesity and related metabolic disorders.

SUBJECTS AND METHODS

Participants

The design, data collection procedures, and findings of the University of Southern California longitudinal SOLAR (Study of Latino Adolescents at Risk for Diabetes) cohort, which began in the year 2000, have been described in detail elsewhere (3, 4). Although cross-sectional dietary analyses have previously been conducted in this cohort (17, 18), the present study is the first to examine longitudinal dietary changes. Eighty-five participants with 2 complete annual visits (48.2% having 1 y between visits and 51.8% with 2 y between visits of dietary intake, adiposity measures, and glucose and insulin indexes) are included in this analysis. At study entry, children were required to meet the following inclusion criteria: *1*) 8–13 y of age, *2*) body mass index (BMI; in kg/m^2) ≥85th percentile for age and sex based on guidelines from 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*) family history of type 2 diabetes in ≥1 parent, sibling, or grandparent 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 any major illness since birth. The Institutional Review Board of the University of Southern California, Health Sciences Campus, approved this study. Informed written consent and assent were obtained from both parents and child before testing started.

Anthropometry and body composition

A licensed pediatric health care provider conducted a detailed medical history examination and determined Tanner staging according to breast development in girls and hair stage in boys (26, 27). Weight was measured to the nearest 0.1 kg with the use of a beam medical scale, and height was measured to the nearest 0.1 cm with the use of a wall-mounted stadiometer. BMI and BMI percentiles were determined (28). Whole-body fat and soft lean tissue were measured by dual-energy X-ray absorptiometry with the use of a Hologic QDR 4500W (Hologic, Bedford, MA). Subcutaneous abdominal adipose tissue (SAAT) and VAT were determined by magnetic resonance imaging, which was performed at the Los Angeles County, University of Southern California Imaging Science Center. A single-slice axial TR 400/16 view of the abdomen at the level of the umbilicus was analyzed for cross-sectional area of adipose tissue. The scan lasted ≈2 min,

and a General Electric 1.5 Signa LX-Echospeed devise with a General Electric 1.5-Tesla magnet was used (Waukesha, WI).

Insulin and glucose indexes

After an overnight fast, a 2-h oral-glucose-tolerance test (OGTT) was conducted with a dose of 1.75 g glucose/kg body weight (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. Fasting and 2-h glucose concentrations were used to determine normal or impaired glucose tolerance as defined by the American Diabetes Association (29). Two-hour insulin area under the curve and incremental insulin area under the curve (IAUC) were calculated from the OGTT data.

Within 1 mo after the OGTT visit, nondiabetic children were asked to come back to the General Clinical Research Center for an overnight visit when a frequently sampled intravenous glucose tolerance test (FSIVGTT) was performed. At time 0, glucose (25% dextrose, 0.3 g/kg body weight) was administered intravenously, and insulin [0.02 U/kg body wt; Humulin R (regular insulin for human injection); Eli Lilly, Indianapolis, IN] was injected intravenously at 20 min. A total of 13 blood samples were collected. Plasma collected during the FSIVGTT was measured for glucose and insulin, and values were entered into the MINMOD MILLENIUM 2003 computer program (version 5.16; Richard N Bergman, University of Southern California, Los Angeles, CA) for assessment of insulin sensitivity, acute insulin response, and disposition index (an index of β cell function).

Assays

Blood samples from all time points taken during the OGTT and FSIVGTT were centrifuged (10 min, 2500 rpm, 8–10°C) immediately to obtain plasma, and aliquots were frozen at –70°C until assayed. Glucose from the OGTT was analyzed on a Dimension Clinical Chemistry system with the use of an in vitro Hexokinase method (Dade Behring, Deerfield, IL). Glucose from the FSIVGTT 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 immunoabsorbent assay kit (Linco, St Charles, MO).

Dietary intake

Dietary intake was assessed from two 24-h diet recalls (2 weekdays) by using the multiple pass technique at each annual visit. One recall was administered in person by a bilingual dietary technician during the outpatient visit with the use of 3-dimensional food models. The second recall was administered by 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, glycemic index (GI), and glycemic load (GL) (using both the standard glucose and the white bread reference). The NDS-R program also calculates the servings of the food and beverage group, based on the Dietary Guidelines for Americans 2005 (30) or the Food and Drug Administration (31).

The dietary data were carefully screened for plausibility through a multiple-step process. Data from 90 participants were first screened by evaluating the participants' comments; 5 subjects were excluded because they reported either being sick ($n = 4$) or had braces tightened ($n = 1$). All 85 remaining participants had 2 viable recalls for both yearly visits, and the average of 2 recalls per year was used for dietary analysis. The dietary data were also examined for plausibility of caloric intake by assessing the distribution of the residuals of the linear regression of caloric intake by body weight at baseline. All participants had a residual that was <2 SDs from the mean. Thus, all 85 participants were included in the present analyses.

Statistical procedures

For the preliminary analyses, paired t tests and chi-square tests were used to assess differences in physical, metabolic, and dietary characteristics between yearly visits. Partial correlations were performed to assess the relation between changes in dietary variables (ie, energy, macronutrients, total and added sugars, sugar, fructose, dietary fiber, insoluble fiber, soluble fiber, GI, GL, and food and beverage servings per day) with changes in adiposity variables (ie, BMI percentiles, BMI z scores, total body fat, VAT, and SAAT) and changes in glucose and insulin indexes (ie, fasting and 2-h glucose and insulin, insulin sensitivity, acute insulin response, disposition index, glucose IAUC, and insulin IAUC). Covariates included in the partial correlations were sex, Tanner stage, time between visits, baseline dietary variable and health outcome of interest, baseline and follow-up body composition (for insulin indexes), or SAAT (for VAT). Partial scatter plots were produced of the significant relations between changes in dietary variables and changes in adiposity or glucose and insulin indexes, adjusting for the covariates listed above.

When partial correlations were significant, then repeated-measures analysis of covariance was conducted for variables, adjusting for covariates. Subjects were divided into categories according to whether they decreased sugar intake, they increased fiber intake, or they decreased sugar intake and increased fiber intake of any magnitude relative to caloric intake. The between-subject factor was the change in dietary variable category (increase compare with decrease), the within-subject variable was the health outcome of interest (ie, adiposity variable and glucose and insulin indexes), and the time variable was baseline compared with follow-up. In all models, the following a priori covariates were included: sex, Tanner stage, time between visits, baseline value of dietary variable of interest, and baseline value of health outcome of interest. Baseline and follow-up SAAT values were included a priori in all VAT models, and baseline and follow-up VAT values were included in all SAAT models. Data were analyzed with SPSS software (version 13.0; SPSS Inc, Chicago, IL) with significance level set at $P < 0.05$.

RESULTS

Eighty-five Latino adolescents (48 males; 11–17 y of age) had complete dietary, adiposity, and glucose and insulin data at 2 annual visits [mean ($\pm$SD) difference in time between visits: 1.5 ± 0.5 y]. The physical and metabolic characteristics for baseline and follow-up are shown in **Table 1**. With the use of paired t tests and chi-square tests, a significant difference was observed

TABLE 1
Physical and metabolic characteristics of Latino children ($n = 85$)[1]

	Baseline	Follow-up	P value[2]
Sex (M/F)	48/37	—	<0.001
Age (y)	14.2 ± 1.6[3]	15.8 ± 1.6	<0.001
Tanner stage (n)			
1	8	2	<0.001
2	5	2	
3	20	12	
4	18	19	
5	34	50	
BMI percentile	96.7 ± 3.6	95.4 ± 5.5	0.002
BMI z score	2.0 ± 0.04	1.9 ± 0.5	0.003
Total fat (kg)	28.7 ± 9.6	29.6 ± 10.2	0.266
Total lean (kg)	50.2 ± 1.2	54.0 ± 1.2	<0.001
VAT (cm^2)	36.6 ± 21.5	40.0 ± 24.6	0.05
SAAT (cm^2)	389.6 ± 140.9	402.7 ± 168.3	0.09
Fasting glucose (mg/dL)	88.6 ± 6.5	91.0 ± 7.1	0.003
2-h Glucose (mg/dL)	114.8 ± 18.8	118.6 ± 22.8	0.197
Fasting insulin (μU/mL)	15.6 ± 9.2	14.1 ± 8.0	0.157
2-h Insulin (μU/mL)	154.5 ± 134.7	137.8 ± 125.1	0.270
Glucose IAUC (nmol $\cdot$ min$^{-1} \cdot$ L^{-1})	74.6 ± 34.1	69.6 ± 34.3	0.234
Insulin IAUC (nmol $\cdot$ min$^{-1} \cdot$ L^{-1})	299.6 ± 212.4	257.8 ± 225.3	0.08
SI ($\times 10^{-4} \cdot$ min$^{-1} \cdot \mu$U$^{-1} \cdot$ mL^{-1})	1.8 ± 1.0	1.7 ± 1.0	0.537
AIR (μU/mL $\times$ 10 min)	1475.0 ± 968.8	1473.7 ± 997.1	0.93
DI ($\times 10^{-4}$/min)	2066.9 ± 939.2	2090.0 ± 1570.9	0.896

[1] VAT, visceral adipose tissue; SAAT, subcutaneous abdominal adipose tissue; IAUC, incremental area under the curve; SI, insulin sensitivity; AIR, acute insulin response; DI, disposition index.
[2] Calculated by using paired t tests and chi-square tests (for sex and Tanner stage only).
[3] Mean $\pm$ SD (all such values).

in Tanner stage, BMI percentile, BMI z score, total lean, VAT, and fasting glucose between the 2 visits ($P \leq 0.05$).

Nutrient and food characteristics at baseline and follow-up are shown in **Table 2**. A significant increase was observed in whole-grain intake (servings per day) between visits ($P = 0.04$). Partial correlations showed that decreases in dietary fiber (g/1000 kcal) were associated with increases in VAT, independent of sex, Tanner stage, baseline fiber and VAT values, and baseline and follow-up SAAT values ($r = -0.29$, $P = 0.02$; **Figure 1**A). Partial correlations showed that decreases in insoluble fiber (g/1000 kcal) were also associated with increases in VAT, independent of the above co-variates ($r = -0.27$, $P = 0.03$; Figure 1B). Changes in carbohydrate intake and soluble dietary fiber intake were not related to changes in any health outcomes ($P > 0.20$). No other changes in dietary variables (including energy, protein, fat, all sugar variables, GL, GI, or food and beverage groups) were significantly related to changes in adiposity variables or changes in glucose and insulin indexes.

In the repeated-measures analyses of covariance, there was a significant time-by-total fiber intake category (increase compared with decrease) interaction for VAT, controlling for sex, Tanner stage, baseline fiber intake, baseline VAT, and baseline and follow-up SAAT values. Those participants who decreased dietary fiber ($n = 46$) from year 1 to year 2 (mean decrease of 3 g · 1000 kcal^{-1} · d^{-1}) had a significant increase in VAT compared with participants who increased dietary fiber ($n = 39$; mean increase of 3 g · 1000 kcal^{-1} · d^{-1}) (21% compared with -4%; $P = 0.02$) (**Figure 2**). Although the partial correlation showed that insoluble fiber was inversely associated with VAT, the insoluble fiber category (increase compared with decrease) interaction for VAT was not significant.

DISCUSSION

To our knowledge, this is the first study to assess observational, longitudinal changes in dietary intake with changes in adiposity variables or glucose and insulin indexes in overweight Latino youth. Changes in energy intake, macronutrients, sugar variables, and all food and beverage servings were not related to any changes in health outcomes. The only relations found between changes in diet and changes in metabolic health were an inverse relation between total and insoluble fiber intakes and visceral adiposity.

Contrary to our hypothesis, decreases in sugar variables (including total sugar, added sugar, fructose, and sugary beverages) were not related to improvements in insulin secretion. We have previously shown that in cross-sectional analyses of this cohort, high intakes of total and added sugar and of sugary beverages were the only dietary components associated with total body fat and poor β cell function (17, 18). We have also recently shown in a 16-wk nutrition and exercise randomized controlled trial with overweight Latino adolescents that those participants who decreased their added sugar intake (mean decrease of 47 g/d, equivalent to 1 soda) had a 33% improvement in insulin secretion, independent of intervention group (19). Another potential explanation is that total and added sugar intakes were consistently high at both visits ($\approx$24% and 14% of kilocalories, respectively, for total and added sugar), and there were no significant differences in sugar intake between visits. In addition, these children were older and at more advanced pubertal stages in this longitudinal analysis, and they were already extremely insulin resistant and exhibiting early signs of β cell dysfunction. Thus, the negative effect of sugar intake on insulin secretion and β cell function may have occurred earlier in this population, and the lack of any change over time masked the possibility of seeing any effects on metabolic outcomes.

Although dietary fiber intake is relatively low in this population ($\approx$9 g · 1000 kcal^{-1} · d^{-1}) and did not significantly change between visits, small decreases (ie, 3 g · 1000 kcal^{-1} · d^{-1}) were associated with a 21% increase in visceral adiposity.

TABLE 2
Nutrient characteristics of Latino children ($n = 85$)[1]

	Baseline	Follow-up	P value
Nutrients			
Energy (kcal)	1769.9 ± 577.7	1845.8 ± 630.0	0.359
Protein (% of kcal)	15.3 ± 3.8	16.3 ± 4.3	0.10
Fat (% of kcal)	32.4 ± 5.9	32.7 ± 7.4	0.82
Carbohydrates (% of kcal)	53.0 ± 9.5	50.3 ± 11.3	0.09
Total sugar (% of kcal)	24.2 ± 7.3	23.5 ± 7.7	0.55
Added sugar (% of kcal)	14.3 ± 7.4	14.7 ± 8.2	0.74
Dietary fiber (g/1000 kcal)	8.5 ± 3.0	8.3 ± 3.6	0.70
Insoluble fiber (g/1000 kcal)	5.7 ± 2.4	5.7 ± 2.8	0.93
Soluble fiber (g/1000 kcal)	2.6 ± 0.8	2.4 ± 1.0	0.06
Glycemic index	59.1 ± 5.5	58.6 ± 5.6	0.48
Glycemic load	132.5 ± 51.0	131.8 ± 53.5	0.92
Food groups			
Meat (servings/d)	3.3 ± 2.3	3.7 ± 2.7	0.35
Dairy (servings/d)	2.0 ± 1.2	2.0 ± 1.5	0.82
Vegetables (servings/d)[2]	1.7 ± 1.3	2.1 ± 1.6	0.07
Fruit (servings/d)[3]	0.9 ± 0.9	0.9 ± 1.1	0.85
Whole grains (servings/d)	0.9 ± 1.2	1.2 ± 1.6	0.04
Refined grains (servings/d)	4.9 ± 2.4	4.6 ± 2.4	0.26
Sugar-sweetened beverages (servings/d)	1.3 ± (1.2	1.3 ± 1.5	0.89

[1] All values are means ± SDs.
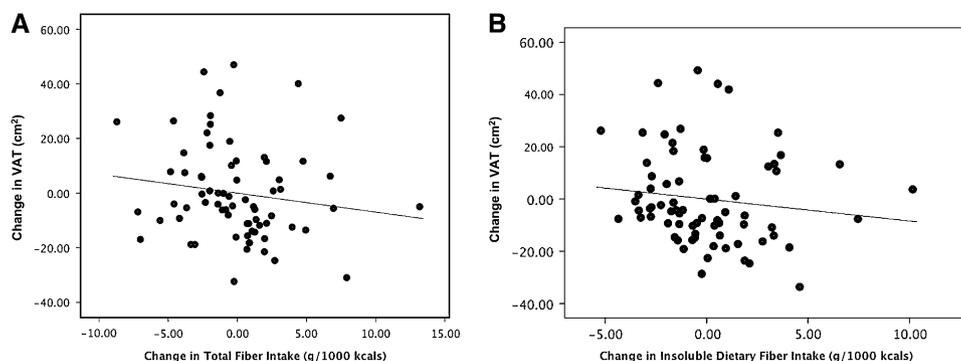[2] Fried vegetables not included.
[3] Juice not included.

FIGURE 1. Partial correlations were performed on 85 overweight Latino children. Changes in total (A) and insoluble (B) fiber intakes (g/1000 kcal) were inversely associated with changes in visceral adipose tissue (VAT) ($r = -0.29$, $P = 0.02$, and $r = 0-0.27$, $P = 0.03$, for total and insoluble fiber intakes, respectively), adjusted for sex, Tanner stage, baseline VAT, total or insoluble fiber intake, and baseline and year 2 subcutaneous abdominal adipose tissue.

We have previously shown in a cross-sectional analysis of this cohort (10–17 y of age) that soluble fiber intake was inversely associated with waist circumference and metabolic syndrome (32). We have also recently shown, in the same 16-wk nutrition and exercise intervention mentioned above, that participants who had quite small increases in total fiber intake ($\approx$5 g/d), regardless of intervention group, had significant reductions in VAT (19). These results differ slightly from our intervention findings, because participants in this study who decreased fiber intake had significant increases in visceral adiposity, whereas participants in the intervention who increased fiber intake had significant reductions in visceral adiposity; however, the magnitude of fiber and visceral fat changes was very similar. Both studies suggest that modest increases in dietary fiber intake, the equivalent of 1/2 cup beans/d or 1 whole-wheat tortilla/d, could substantially lower visceral adiposity. Note that this finding was independent of change in energy intake and that there were no significant differences in energy intake across years between participants who increased fiber intake compared with participants who decreased fiber intake (64.2 ± 798.6 kcal/d compared with 86.8 ± 728.1 kcal/d; $P = 0.89$).

It has been well documented that visceral fat is directly linked to metabolic disorders in animals, adults, and children (33–36). One explanation is that visceral fat results in hepatic insulin resistance via a "portal" effect of free fatty acids released from the omental fat (33). The influx of free fatty acids into the liver then leads to increased hepatic glucose production (34, 35) and decreased hepatic insulin clearance, which then leads to insulin resistance and hyperinsulinemia. We have previously shown that visceral fat in Latino youth is strongly and positively associated with fasting insulin and acute insulin response and negatively associated with insulin sensitivity (36).

Several possible mechanisms should be considered on how dietary fiber, specifically insoluble fiber, might affect visceral adiposity. Insoluble fiber has been shown to increase fecal bulk and to shorten transit time (37), therefore allowing less time for digestion and absorption of nutrients (38). This decreased nutrient absorption, or energy reduction, could directly affect total fat mass and ultimately visceral fat accumulation. A second potential mechanism is that dietary fiber directly affects glucose and insulin regulation (39). In a study of 2909 healthy black and white young adults, Ludwig et al (40) found that dietary fiber intake was a significant predictor of fasting insulin concentrations, body weight, waist-to-hip ratio, and 2-h insulin after a glucose challenge. These results suggest that dietary fiber lowers glycemic and insulinemic response to a meal. Insulin concentrations can in turn influence appetite as well as lipid metabolism. Another possible explanation is that foods high in fiber, specifically fruit and vegetables, are often high in phytoestrogens, ie, isoflavones and lignans, which have been inversely associated with central adiposity (41), dyslipidemia (42), and hyperglycemia and insulin resistance (43). Together, these mechanisms support our findings that increasing fiber intake can result in reductions in visceral adiposity.

Although we did not find a significant relation between changes in food and beverage servings per day with changes in any health outcomes, it is important to examine which foods or beverages changed in those participants who either increased or decreased fiber intake. Fiber increasers consumed significantly more nonfried vegetables, more fruit and vegetables combined, and more legumes between visits (increases of 1.3, 2, and 0.5 servings/d, respectively) compared with fiber decreasers. These findings suggest that fairly modest increases in fruit and vegetable intake of 1 or 2 servings/d and bean intake of 0.5 servings (equivalent to 1/4 cup) could have profound effects on
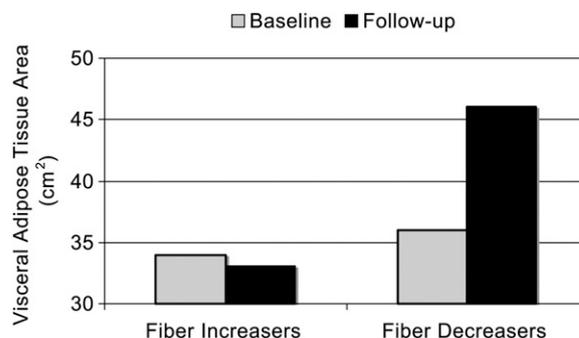

FIGURE 2. Repeated-measures ANCOVA was performed on data from 85 overweight Latino children. Participants who had decreased dietary fiber ($n = 46$) from year 1 to year 2 (mean decrease of 3 g · 1000 kcal^{-1} · d^{-1}) had a significant increase in visceral adipose tissue compared with participants who had increased dietary fiber ($n = 39$; mean increase of 3 g · 1000 kcal^{-1} · d^{-1}) (21% compared with -4%; $P = 0.02$). Models were adjusted for sex, Tanner stage, time between visits, baseline visceral adipose tissue, energy, baseline fiber intake, and baseline subcutaneous abdominal adipose tissue.

lowering visceral adiposity and subsequent related metabolic disorders.

There are a few limitations of the current study. This study is limited by the use of two 24-h diet recalls, which rely solely on the participants' self-report 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, using well-trained diet technicians, screening for participants' comments, and assessing plausibility of caloric intake by body weight. 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, we would not see individual dietary factors affecting adiposity. However, we did find an association between dietary fiber and visceral adiposity, independent of large variations in adiposity.

In conclusion, this is the first study to show that modest decreases in dietary fiber over 1 or 2 y can have dramatic effects on increasing visceral adiposity in overweight Latino youth. Public health messages and interventions focusing on improving the quality of carbohydrate intake, particularly increasing the intake of total and insoluble dietary fiber, for reducing obesity and related metabolic disorders are warranted in high-risk pediatric populations.

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