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Dietary Patterns Exhibit Sex-Specific Associations with Adiposity and Metabolic Risk in a Cross-Sectional Study in Urban Mexican Adolescents

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Abstract

Background: Studies in Western nations have shown associations of certain dietary patterns with obesity and metabolic risk in youth. Little is known about these relations in newly industrialized countries where obesity prevalence is surpassing those of developed countries.

Objective: We sought to characterize dietary patterns in a cross-sectional study in 224 adolescents aged 8–14 y in Mexico and to investigate associations of the dietary patterns with adiposity and metabolic risk.

Methods: We used principal components analysis to derive dietary patterns from food-frequency questionnaire data. By using linear regression models that accounted for mother's marital status, education, and smoking habits and child's age and physical activity, we examined associations of the dietary patterns with adiposity [body mass index *z* score, waist circumference, the sum and ratio of the subscapular and triceps skinfold thicknesses, blood pressure, serum fasting glucose and a C-peptide–based measure of insulin resistance (CP-IR), lipid profile, and a metabolic syndrome risk *z* score (MetS *z* score)].

Results: We identified a ''prudent'' dietary pattern characterized by high intakes of vegetables, fruit, fish, chicken, and legumes and a ''transitioning'' dietary pattern, which comprises processed meats, Mexican foods, and sweetened beverages. Each unit increase in the prudent pattern factor score corresponded with 0.33 ng/mL (95% CI: 0.09, 0.57 ng/mL) lower C-peptide, 0.08 units (95% CI: 0.02, 0.13 units) lower CP-IR, and a 0.14 unit (0.00, 0.27 unit) lower MetS *z* score in boys. In girls, the transitioning pattern corresponded with higher subscapular + triceps skinfold thickness (per 1-unit increase in the factor score: 2.46 mm; 95% CI: 0.10, 4.81 mm). These results did not change after accounting for pubertal status.

Conclusions: A prudent dietary pattern was protective against metabolic risk in adolescent boys, whereas a transitioning dietary pattern corresponded with higher adiposity among adolescent girls. Given that adolescence is a key developmental period for long-term health, efforts to elucidate dietary determinants of metabolic risk during this life stage may have long-term benefits. *J Nutr* 2017;147:1977–85.

Keywords: prudent dietary pattern, transitioning dietary pattern, adolescents, metabolic risk, nutritional epidemiology

Introduction

Childhood obesity is an emerging public health concern in Latin America (1). In the past 3 decades, many countries in this region experienced a marked increase in pediatric overweight and obesity (1), with a sharper increase in prevalence than that of developed countries (2). Mexico is at the forefront of the epidemic, with an increase in the prevalence of adolescent

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Abbreviations used: CP-IR, C-peptide–based measure of insulin resistance; DBP, diastolic blood pressure; ELEMENT, Early Life Exposure in Mexico to Environmental Toxicants; ICC, intraclass correlation; MetS *z* score, metabolic syndrome risk *z* score; PCA, principal components analysis; SBP, systolic blood pressure.

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overweight and obesity from 30% in 2012 (3) to nearly 35% in 2016 (4). These statistics may be attributable, at least in part, to the fact that this country has recently undergone considerable economic growth, a process that coincides with a shift in intake of traditional diets rich in whole grains, fiber, fruit, and vegetables, toward a Western diet characterized by high intakes of saturated fat, refined carbohydrates, and processed foods (5).

Until recently, the field of nutritional epidemiology has focused on quantifying the effects of single foods or nutrients on health outcomes. However, given that we eat meals composed of multiple foods and nutrients, the single-food and -nutrient approach may be confounded by overall eating patterns and diet quality, making it challenging to untangle the impact of specific food components in observational studies (6). Therefore, the use of dietary patterns, in which foods and their nutrients are represented by latent variables that provide a more realistic depiction of dietary intake, may provide better insights into diet-disease relations (6). Studies in children and adolescents in developed countries have shown associations of certain dietary patterns—namely, the Western dietary pattern—with higher BMI, hyperlipidemia, and insulin resistance (7, 8). However, little is known about these relations in emerging economies, which may be at greater risk of development of obesity and obesity-related disease due to rapid changes in the food environment and ease of access to unhealthy processed foods. To date, only 3 published studies to our knowledge have examined the associations of dietary patterns in relation to obesity, metabolic risk, or both among youth in Latin America (9–11), 2 of which were based in the same cohort of Colombian schoolchildren (9, 10). In the Colombian population, a snacking dietary pattern that comprised candy, chocolate bars, cookies, ice cream, packaged fried snacks, and soda was associated with higher adiposity at ages 5–12 y (9) as well as greater weight gain during 2.5 y of follow-up (10). The third study, which was carried out in 7- to 18-y-olds in Mexico, identified a Western dietary pattern characterized by high intakes of soft drinks, tortillas, and snacks and a low intake of fresh fruit, which was associated with insulin resistance (11). Considering that lifestyle modifications, including those aimed at changing dietary habits, have the potential to ameliorate metabolic disease risk (12), characterizing population-specific dietary patterns and elucidating their relation with obesity and metabolic risk factors are key first steps to identifying potential avenues for prevention.

The goals of the present study are 2-fold. First, we sought to characterize dietary patterns in a cohort of peripubertal youth in Mexico City. Second, we sought to investigate the associations of the dietary patterns of interest with adiposity and biomarkers of metabolic risk.

Methods

Study population. This study included participants from 2 of 3 cohorts within the Early Life Exposure in Mexico to Environmental Toxicants (ELEMENT) Project, an ongoing study in pregnant women and their offspring in Mexico City, Mexico. Between 1997 and 2004, women were recruited from public maternity hospitals serving a homogenous low- to middle-income population in Mexico City during the first trimester of pregnancy (13, 14). In 2011–2012, we recontacted a subset of the offspring ($n = 250$), who were then 8–14 y of age, based on a convenience sample of participants for whom we had an adequate volume of archived prenatal biospecimens (maternal urine and cord blood).

At the in-person research visits that took place in 2011 (hereafter referred to as the peripubertal visit), the participants provided an 8-h fasting blood sample and completed anthropometric assessment and interviewer-administered questionnaires, including an FFQ. The analytic sample included 224 children who had complete data on dietary intake, anthropometric measures, and metabolic biomarkers. These children were not different from the 250 children who attended the peripubertal visit with respect to sociodemographic and family characteristics (**Supplemental Table 1**). The institutional review boards of the Mexico National Institute of Public Health and the University of Michigan approved the research protocols. Maternal informed consent and child assent were provided for all participants.

Dietary assessment. At the peripubertal research visit, research staff administered an age-specific semiquantitative FFQ to the children. The FFQ was adapted from the 2006 Mexican Health and Nutrition Survey (15), which queried the frequency of consumption of 109 food items during the past 7 d. Participants reported the frequency of consumption of standard portions (e.g., 1 natural unit, cup, slice, piece, etc.) of each food ranging from "Never" to "≥6 times per day." Children were assisted by their caregivers when necessary to improve the accuracy and precision of self-reported intakes.

Before statistical analyses, we consolidated the 109 food items into 35 culturally relevant food groups (**Supplemental Table 2**). We then estimated total daily energy intake by using the USDA Food-Composition Database (16) and adjusted each food group by total energy intake by using the residual method (17).

Adiposity. Trained research staff measured the children's weight (kilograms) on a digital scale (BAME Model 420; Catálogo Médico), height (centimeters) by using a calibrated stadiometer (BAME Model 420; Catálogo Médico), waist circumference (centimeters) by using a nonstretchable measuring tape (QM2000; QuickMedical), and the subscapular and triceps skinfold thicknesses (millimeters) by using calibrated skin calipers (Lange; Beta Technology) (18). All anthropometric assessments were carried out in duplicate. Because the correlations between the repeated measures were high (Pearson's R^2 >0.98), we used the average of the 2 values for each indicator in the analysis.

We used weight and height to calculate BMI as an indicator of body size and overall adiposity (19) and standardized it as an age- and sex-specific z score by using the WHO growth reference (20). We used waist circumference as a proxy for central visceral adiposity (19), and the sum (subscapular and triceps) and the ratio (subscapular:triceps) of the skinfold thicknesses as markers of total and central subcutaneous adiposity (21), respectively.

Blood pressure. Research staff measured systolic blood pressure (SBP) and diastolic blood pressure (DBP) 5 times (millimeters of mercury) in the seated position with an automated blood pressure monitor (BPM-200 Medical Devices Blood Pressure Monitor, BpTRU). Because the intraclass correlations (ICCs) between the measurements were high ($ICC_{SBP} = 0.95$, $ICC_{DBP} = 0.89$), we used the average of the values for the analysis and focused on SBP instead of DBP because it is more accurately measured in children and is a better predictor of future health outcomes (22).

Metabolic biomarkers. With the use of fasting blood collected at the peripubertal visit, we measured serum glucose enzymatically and serum C-peptide by using an automated chemiluminescence immunoassay (Immulite 1000; Siemens Medical Solutions). We also quantified serum total cholesterol, TGs, and HDL cholesterol by using a biochemical analyzer (Cobas Mira Plus; Roche Diagnostics) and calculated LDL cholesterol as total cholesterol – HDL cholesterol – (TGs/5).

In addition to the individual biomarkers, we derived 2 indexes: a C-peptide–based measure of insulin resistance (CP-IR) and a metabolic syndrome risk z score (MetS z score). We calculated CP-IR on the basis of the following equation: (fasting serum C-peptide × fasting serum glucose)/405 (23). For the MetS z score, we calculated the sum of 5 age- and sex-specific internal z scores for waist circumference, fasting glucose, fasting C-peptide, TGs:HDL cholesterol, and (SBP + DBP)/2. This score is a modification (i.e., use of C-peptide rather than insulin) of a score proposed by Viitasalo et al. (24) in a study that examined correlations among the metabolic biomarkers in children and adults and established its association with incident type 2 diabetes and cardiovascular disease.

We used this score because there is currently no consensus definition for metabolic syndrome in children and adolescents (25).

Covariates. Upon enrollment, mothers reported their age in years (categorized as 15–24, 25–34, and 35–44 y in bivariate analyses), smoking habits during pregnancy (yes or no), parity (0, 1–2, or ≥3 previous births), delivery mode of index birth (cesarean or vaginal delivery), marital status (married or cohabiting compared with single), and educational level (<10, 10–12, or ≥13 y) via an interviewer-administered questionnaire. We also measured the women's height to the nearest 0.1 cm. At the peripubertal visit, the child (with proxy assistance from the accompanying caregiver when necessary) completed a validated interviewer-administered questionnaire that queried the amount of time he or she spent engaged in moderate-to-vigorous physical activity each week (26). For the analysis, we categorized physical activity as quartiles of total hours per week. A pediatrician assessed each child to determine Tanner stage on a scale of 1 (no development) to 5 (full development) for genital (boys), breast (girls), and pubic hair (both) development. We then dichotomized pubertal status as prepubertal or pubertal: boys were classified as pubertal if they received an assessment of Tanner stage >1 for genital or pubic hair development, and girls were classified as pubertal if they received an assessment of Tanner stage >1 for breast or pubic hair development (27). Because stature is a direct determinant of blood pressure in children (28), we also considered age- and sex-standardized height *z* score (20) as a covariate in models where blood pressure was the outcome of interest.

Data analysis. First, we created dietary patterns by using food frequencies from the FFQ. To do this, we consolidated the 109 food items into 35 food groups on the basis of their nutritional properties (Supplemental Table 2). By using principal components analysis (PCA; PROC FACTOR in SAS), we then consolidated the food groups into principal components ("factors") and rotated them orthogonally to maintain noncorrelation and to facilitate interpretability. PROC FACTOR extracts as many factors as there are original variables—that is, the 35 food groups were converted into 35 factors, each of which represents a unique dietary pattern parameterized as a continuous, normally distributed score that can be interpreted as the extent to which an individual's diet resembles the combination of food groups within a given factor. We considered food groups with factor loadings ≥|0.30| to be a key contributor to a dietary pattern. This cutoff is similar to those used in other studies (29, 30) and was used for interpretation only because we did not exclude food groups with factor loadings <|0.30| from PCA. Of the 35 factors, we retained the first 2 based on the scree plot and the standard criterion of eigenvalues >1 (31).

Next, we examined the associations of the 2 factor scores with sociodemographic and perinatal characteristics to identify potential confounders to the relation between diet and metabolic health. To assess the significance of these associations, we used a test for linear trend for ordinal variables (e.g., where an indicator for an ordinal variable is entered into the model continuously) and the Wald test for binary variables.

Next, for multivariable analysis, we examined associations of each continuous factor score with the health outcomes separately for boys and girls because we found evidence of an interaction with sex (P-interaction < 0.05). In model 1, we accounted for mother's marital status, education, and smoking habits during pregnancy and the child's age at the time of the peripubertal research visit. In model 2, we further adjusted for the child's physical activity. Finally, in model 3, we accounted for pubertal status, which could be a mediator because nutrition can influence pubertal development (32, 33), which, in turn, could influence physiology and metabolic health (34). Across the models, we evaluated the impact of covariate adjustment by comparing change in the direction, magnitude, and precision (according to 95% CIs) of the estimates.

Finally, we performed some sensitivity and post hoc analyses. First, we tested for an interaction between the 2 factor scores and pubertal status. We found no evidence of effect modification by puberty (all P-interaction > 0.10); thus, results are presented for prepubertal and pubertal children combined. Second, we examined the impact of adjustment for maternal age at enrollment, parity, and delivery method. Including these variables did not change the results, so they were not included in the final models. Finally, because stunted children may exhibit inherently different physiology than their nonstunted counterparts (35), we excluded $n = 7$ children with height *z* scores <−2 SDs. The exclusion of these participants did not alter the results, so we included all of the children in the analysis. We performed all analyses by using SAS 9.4.

Results

The median age of the participants was 10.1 y (range: 8.1–14.7 y), and 48.2% ($n = 107$) were boys. Mean ± SD values of the adiposity indicators, metabolic outcomes, and blood pressure are reported in **Table 1**. By using dietary data from the FFQ, we identified 2 major dietary patterns, which accounted for 15.1% of total variability in the original food groups (**Supplemental Table 3**). Factor 1, which explained 9.2% of variability, was a "prudent" dietary pattern characterized by high intakes of vegetables, fruit, fish, legumes, and chicken. Factor 2, which accounted for 5.9% of variability, was characterized by high intakes of processed meats, Mexican foods [some of which were fried (e.g., fried tacos), *atole* (a traditional corn- and masa-based drink), and sugar-sweetened beverages] and low intakes of whole grains, pureed vegetable soups, and sweetened and whole milk. We refer to factor 2 as the "transitioning" dietary pattern because it likely reflects the shift in methods of food preparation from traditional Mexican (e.g., boiled, baked, unsweetened) toward Western methods of preparation (e.g., fried, processed, sweetened).

In bivariate analyses (**Table 2**), we observed that children of married women and those who smoked during pregnancy had a lower prudent dietary pattern score, whereas those whose mothers attained a higher educational level had a higher transitioning

TABLE 1 Descriptive statistics for adiposity indicators, blood pressure, and metabolic biomarkers in 224 Mexican adolescents aged 8–14 y[1]

	Boys ($n = 108$)	Girls ($n = 116$)
Adiposity		
BMI, kg/m^2	19.2 ± 3.2	19.8 ± 4.0
BMI *z* score[2]	0.86 ± 1.23	0.88 ± 1.28
Waist circumference, cm	69.9 ± 10.3	72.4 ± 11.4
SS + TR, mm	25.6 ± 11.1	31.3 ± 12.2
SS:TR	0.73 ± 0.22	0.77 ± 0.23
Blood pressure, mm Hg		
SBP	104.6 ± 10.2	101.6 ± 10.2
DBP	65.8 ± 7.4	65.6 ± 7.6
Metabolic biomarkers		
Serum fasting glucose, mg/dL	87.8 ± 8.1	86.2 ± 10.9
Serum C-peptide, ng/mL	1.7 ± 1.2	1.9 ± 1.3
CP-IR[3]	0.36 ± 0.42	0.42 ± 0.40
Serum total cholesterol, mg/dL	152 ± 28.9	157 ± 28.2
Serum TGs, mg/dL	79.0 ± 38.2	96.4 ± 47.9
Serum LDL cholesterol, mg/dL	76.3 ± 23.7	80.6 ± 22.4
Serum HDL cholesterol, mg/dL	59.8 ± 11.2	57.6 ± 11.4
MetS *z* score[4]	0.04 ± 0.66	0.01 ± 0.59

[1] Values are means ± SDs. CP-IR, C-peptide–based insulin resistance index; DBP, diastolic blood pressure; MetS *z* score, metabolic syndrome risk *z* score; SBP, systolic blood pressure; SS, subscapular skinfold thickness; TR, triceps skinfold thickness.
[2] Standardized according to age- and sex-specific values from the WHO growth reference for children aged 5–19 y.
[3] Calculated as (fasting serum C-peptide × fasting serum glucose)/405.
[4] Calculated as the average of 5 internally standardized *z* scores for waist circumference, fasting glucose, C-peptide, TG-to-HDL-cholesterol ratio, and the average of SBP and DBP.

TABLE 2 Relations of dietary factor scores with characteristics of 224 ELEMENT adolescents[1]

		Prudent dietary pattern		Transitioning dietary pattern	
	n	Factor 1 score	*P*	Factor 2 score	*P*
Maternal characteristics at the time of enrollment					
Age at enrollment, y			0.61		0.31
15–24	93	−0.02 ± 0.87		0.09 ± 1.01	
25–34	102	−0.02 ± 1.08		−0.07 ± 1.06	
35–44	29	0.12 ± 1.11		−0.06 ± 0.72	
Marital status			0.01		0.12
Married	163	−0.10 ± 0.93		0.06 ± 1.03	
Single	61	0.27 ± 1.13		−0.17 ± 0.90	
Maternal education, y			0.38		0.07
<10	79	−0.13 ± 0.91		−0.19 ± 0.80	
10–12	113	0.11 ± 1.06		0.11 ± 1.13	
≥13	32	−0.06 ± 0.96		0.11 ± 0.92	
Parity			0.87		0.65
0	82	0.01 ± 1.08		−0.01 ± 0.92	
1–2	126	−0.01 ± 0.01		0.04 ± 1.06	
≥3	16	−0.02 ± 0.02		−0.30 ± 0.93	
Smoking during pregnancy			0.007		0.72
Yes	5	−1.19 ± 0.51		−0.16 ± 1.11	
No	219	0.03 ± 0.99		0.00 ± 0.99	
Delivery method			0.95		0.86
Cesarean	93	−0.01 ± 1.11		−0.01 ± 0.93	
Vaginal	130	−0.01 ± 0.90		0.02 ± 1.05	
Child characteristics at the peripubertal visit					
Sex			0.07		0.03
Male	107	−0.13 ± 0.90		0.16 ± 1.05	
Female	117	0.12 ± 1.07		−0.14 ± 0.93	
Child's age, y			0.36		0.93
<10	107	0.01 ± 0.90		0.00 ± 1.06	
10–12	66	0.13 ± 1.08		−0.03 ± 0.79	
>12	51	−0.19 ± 1.07		0.03 ± 1.12	
Weight status (BMI), kg/m^2			0.87		0.44
Underweight, <18.5	16	0.07 ± 0.73		−0.36 ± 0.76	
Normal weight, 18.5–24.9	91	−0.04 ± 0.98		0.00 ± 1.11	
Overweight, 25–29.9	75	0.02 ± 1.15		0.09 ± 0.92	
Obese, ≥30	42	0.02 ± 0.88		−0.03 ± 0.95	
Physical activity, h/wk			0.86		0.02
Q1 (median: 16)	33	−0.05 ± 0.78		−0.17 ± 0.80	
Q2 (median: 18)	60	0.14 ± 1.01		−0.17 ± 0.88	
Q3 (median: 19)	60	−0.26 ± 0.79		−0.02 ± 1.00	
Q4 (median: 22)	71	0.12 ± 1.19		0.24 ± 1.14	
Male puberty indicators[2]					
Pubic hair			0.82		0.20
Prepubertal	82	−0.12 ± 0.87		0.23 ± 1.09	
Pubertal	22	−0.17 ± 1.06		−0.09 ± 0.90	
Testicles			0.81		0.39
Prepubertal	50	−0.12 ± 0.77		0.25 ± 1.24	
Pubertal	53	−0.16 ± 1.03		0.07 ± 0.87	
Female puberty indicators[2]					
Pubic hair			0.38		0.58
Prepubertal	84	0.17 ± 0.96		−0.17 ± 0.82	
Pubertal	33	−0.02 ± 1.32		0.07 ± 1.20	
Breast			0.43		0.32
Prepubertal	75	0.18 ± 0.77		−0.21 ± 1.24	
Pubertal	42	0.01 ± 1.03		−0.03 ± 0.87	

[1] Values are means ± SDs unless otherwise indicated. *P* values represent a test for linear trend where an ordinal indicator is entered into the model as a continuous variable, with the exception of binary variables (Wald test). ELEMENT, Early Life Exposure in Mexico to Environmental Toxicants; Q, quartile.

[2] Puberty was defined as Tanner stage 2–5 (vs. 1) for breast (girls), testicular (boys), and pubic hair (both) development.

dietary pattern score. These associations may be a reflection of the nutrition transition, in which individuals of higher socioeconomic status tend to consume diets and lead lifestyles that more closely resemble those of persons in industrialized countries than do their less affluent counterparts (36). Compared with boys, girls had higher prudent and lower transitioning dietary pattern scores. Finally, children who engaged in more physical activity (hours per week) had a higher score for the transitioning dietary pattern.

Table 3 shows sex-specific results from the multivariable analysis for the adiposity outcomes. Although the results in this table are generally null, the transitioning dietary pattern was associated with higher subscapular and triceps skinfold thicknesses in girls after adjustment for weekly physical activity in model 2 (per 1-unit increase in the transitioning pattern score: 2.46 mm; 95% CI: 0.10, 4.81 mm) and pubertal status in model 3 (per 1-unit increase in the transitioning pattern score: 2.38 mm; 95% CI: 0.03, 4.74 mm).

Table 4 shows sex-specific associations of the 2 dietary patterns with the metabolic biomarkers and blood pressure. Among boys, we found an inverse association of the prudent dietary pattern score with C-peptide, CP-IR, and MetS *z* score across all 3 models. For example, in the confounder-adjusted model (model 1), each 1-unit increase in the prudent pattern score corresponded with 0.33 ng/mL (95% CI: 0.08, 0.58 ng/mL) lower C-peptide, a 0.08 unit (95% CI: 0.02, 0.14 units) lower CP-IR, and a 0.14 unit (95% CI: 0.00, 0.27 units) lower MetS *z* score. Further adjustment for the child's weekly physical activity and pubertal status did not materially alter these findings.

In girls, the prudent dietary pattern was associated with higher blood pressure, with an ~1.8 mm Hg higher SBP per 1 unit of the dietary pattern score across all models (Table 4). Adjustment for height *z* score attenuated the estimate toward the null—model 1: 1.44 mm Hg (95% CI: −0.16, 3.04 mm Hg) per unit of factor 1; model 2: 1.44 mm Hg (95% CI: −0.15, 3.04 mm Hg) per 1 unit of the prudent dietary pattern score; and model 3: 1.42 mm Hg (−0.18, 3.01 mm Hg) per unit of the prudent dietary pattern score. We did not find associations of either dietary pattern with serum lipid concentrations in boys or girls; thus, we show these results separately in **Table 5**.

Discussion

In this study in Mexican youth aged 8–14 y, we characterized 2 major dietary patterns and investigated their associations with adiposity, blood pressure, and biomarkers of metabolic risk. The first dietary pattern, which we refer to as the prudent dietary pattern because it consisted of vegetables, fruit, fish, legumes, and chicken, was associated with a more favorable glycemic profile and a lower MetS *z* score in boys. The second dietary pattern, which we named the transitioning dietary pattern because it was characterized by a high consumption of processed meats, Mexican foods, and sweetened beverages and low intakes of whole grains, pureed vegetable soups, whole milk, and sweetened milk, was associated with higher subcutaneous adiposity in girls.

Dietary patterns. To date, the majority of published studies on dietary patterns in relation to obesity, metabolic risk, or both in Mexican populations have been carried out in adults (11, 37–39), and some have identified a dietary pattern similar to our factor 1 (11, 38). Of particular relevance to the present study are results from a cross-sectional analysis carried out in another population of similarly aged Mexican adolescents (7–18 y) of medium socioeconomic status who were residing in Cuernavaca and Toluca, 2 urban cities in central Mexico within 65 km of Mexico City (11). In this study, Romero-Polvo et al. (11) identified a dietary pattern comprising fresh vegetables and fruit, tomato juice, potatoes, legumes, and unsweetened tea and coffee.

TABLE 3 Sex-specific associations of dietary patterns with adiposity among 224 Mexican adolescents[1]

	Associations of each dietary pattern factor score with adiposity indicators			
	BMI *z* score	Waist circumference, cm	SS + TR, mm	SS:TR
Boys (*n* = 108)				
Factor 1: prudent dietary pattern				
Model 1	−0.18 (−0.45, 0.08)	−1.32 (−3.43, 0.79)	−1.67 (−4.08, 0.72)	−0.05 (−0.09, 0.00)
Model 2	−0.18 (−0.45, 0.09)	−1.27 (−3.36, 0.81)	−1.65 (−4.05, 0.74)	−0.05 (−0.09, 0.00)
Model 3	−0.17 (−0.44, 0.09)	−1.22 (−3.32, 0.89)	−1.54 (−3.96, 0.88)	−0.04 (−0.09, 0.00)
Factor 2: transitioning dietary pattern				
Model 1	−0.11 (−0.33, 0.11)	−0.98 (−2.73, 0.76)	−1.13 (−3.11, 0.85)	0.00 (−0.04, 0.04)
Model 2	−0.11 (−0.34, 0.11)	−0.76 (−2.50, 0.99)	−1.02 (−3.03, 0.98)	0.00 (−0.04, 0.04)
Model 3	−0.11 (−0.34, 0.11)	−0.73 (−2.50, 1.04)	−1.01 (−3.05, 1.02)	0.00 (−0.04, 0.04)
Girls (*n* = 116)				
Factor 1: prudent dietary pattern				
Model 1	0.05 (−0.17, 0.27)	0.52 (−1.35, 2.39)	0.08 (−1.99, 2.15)	−0.03 (−0.07, 0.01)
Model 2	0.05 (−0.17, 0.27)	0.51 (−1.35, 2.38)	0.07 (−1.99, 2.14)	−0.03 (−0.07, 0.01)
Model 3	0.04 (−0.18, 0.26)	0.47 (−1.39, 2.32)	0.04 (−2.02, 2.10)	−0.03 (−0.07, 0.01)
Factor 2: transitioning dietary pattern				
Model 1	0.21 (−0.05, 0.46)	1.54 (−0.59, 3.66)	2.29 (−0.05, 4.63)	0.00 (−0.05, 0.04)
Model 2	0.21 (−0.05, 0.46)	1.65 (−0.49, 3.80)	2.46 (0.10, 4.81)*	−0.01 (−0.05, 0.04)
Model 3	0.20 (−0.06, 0.45)	1.55 (−0.59, 3.69)	2.38 (0.03, 4.74)*	−0.01 (−0.05, 0.04)

[1] Values are βs (95% CIs) for each outcome per 1-unit increase in the dietary pattern factor score. Model 1 adjusted for mother's marital status, education, and smoking habits during pregnancy and for child's age; model 2 adjusted as for model 1 and for child's physical activity (hours per week); model 3 adjusted as for model 2 and for child's pubertal status (prepubertal vs. pubertal). *$P < 0.05$. SS, subscapular skinfold thickness; TR, triceps skinfold thickness.

TABLE 4 Sex-specific associations of dietary patterns with metabolic biomarkers among 224 Mexican adolescents[1]

	Associations of each dietary pattern factor score with metabolic biomarkers				
	Serum fasting glucose, mg/dL	Serum C-peptide, ng/mL	CP-IR[2]	SBP, mm Hg	MetS *z* score[3]
Boys (*n* = 108)					
Factor 1: prudent dietary pattern					
Model 1	−1.66 (−3.41, 0.09)	−0.33 (−0.58, −0.08)*	−0.08 (−0.14, −0.02)*	−1.05 (−3.20, 1.10)	−0.14 (−0.27, 0.00)*
Model 2	−1.65 (−3.39, 0.10)	−0.33 (−0.57, −0.09)*	−0.08 (−0.13, −0.02)*	−1.04 (−3.18, 1.11)	−0.14 (−0.27, 0.00)*
Model 3	−1.71 (−3.46, 0.03)	−0.31 (−0.53, −0.08)*	−0.07 (−0.13, −0.02)*	−0.90 (−3.07, 1.27)	−0.13 (−0.26, 0.01)
Factor 2: transitioning dietary pattern					
Model 1	−0.67 (−2.13, 0.79)	−0.16 (−0.37, 0.04)	−0.04 (−0.09, 0.01)	−1.14 (−2.91, 0.63)	−0.11 (−0.22, 0.01)
Model 2	−0.61 (−2.09, 0.87)	−0.15 (−0.36, 0.06)	−0.04 (−0.08, 0.01)	−1.08 (−2.87, 0.72)	−0.10 (−0.21, 0.01)
Model 3	−0.64 (−2.13, 0.84)	−0.10 (−0.30, 0.09)	−0.02 (−0.07, 0.02)	−0.96 (−2.77, 0.86)	−0.09 (−0.20, 0.03)
Girls (*n* = 116)					
Factor 1: prudent dietary pattern					
Model 1	0.83 (−1.05, 2.70)	0.02 (−0.21, 0.24)	0.01 (−0.06, 0.07)	1.80 (0.12, 3.48)*	0.06 (−0.04, 0.16)
Model 2	0.84 (−1.01, 2.70)	0.02 (−0.21, 0.24)	0.01 (−0.06, 0.08)	1.79 (0.13, 3.46)*	0.06 (−0.04, 0.16)
Model 3	0.81 (−1.05, 2.67)	0.02 (−0.21, 0.25)	0.01 (−0.06, 0.08)	1.79 (0.11, 3.46)*	0.06 (−0.04, 0.16)
Factor 2: transitioning dietary pattern					
Model 1	0.33 (−1.82, 2.48)	0.04 (−0.23, 0.30)	0.01 (−0.07, 0.09)	0.21 (−1.75, 2.17)	0.05 (−0.07, 0.17)
Model 2	0.06 (−2.09, 2.21)	0.00 (−0.26, 0.26)	0.00 (−0.08, 0.08)	0.42 (−1.55, 2.38)	0.04 (−0.08, 0.16)
Model 3	0.02 (−2.14, 2.18)	0.00 (−0.26, 0.26)	0.00 (−0.08, 0.08)	0.40 (−1.59, 2.38)	0.04 (−0.08, 0.16)

[1] Values are βs (95% CIs) for each outcome per 1-unit increase in the dietary pattern factor score. Model 1 adjusted for mother's marital status, education, and smoking habits during pregnancy and for child's age; model 2 adjusted as for model 1 and for child's physical activity (hours per week); model 3 adjusted as for model 2 and for child's pubertal status (prepubertal vs. pubertal). *$P < 0.05$. CP-IR, C-peptide–based measure of insulin resistance; MetS *z* score, metabolic syndrome risk *z* score; SBP, systolic blood pressure.
[2] Calculated as (fasting serum C-peptide × fasting serum glucose)/405.
[3] Calculated as the average of 5 internally standardized *z* scores for waist circumference, fasting glucose, C-peptide, TG-to-HDL-cholesterol ratio, and the average of SBP and diastolic blood pressure.

We noted that food groups within this dietary pattern were fairly consistent with our prudent dietary pattern, with the exception that potatoes and tea and coffee were not key food items in this dietary pattern in ELEMENT. This discrepancy likely reflects natural variations in diet between study populations, and thus emphasizes the importance of characterizing population-specific dietary patterns.

The transitioning dietary pattern was composed of processed meats, Mexican foods (some of which were fried; e.g., fried tacos), *atole*, and sweetened beverages [which were previously found to be associated with obesity risk in this population (40)] and low intakes of whole grains, pureed vegetable soups, and sweetened and whole milk. Although, to our knowledge, this dietary pattern has not been reported in the literature, aspects of it—specifically, high intakes of Mexican refined-grain–based products and sweetened beverages—resemble the Western dietary pattern characterized by Romero-Polvo et al. (11). We hypothesize that this pattern reflects dietary habits of a setting undergoing the nutrition transition, because it includes several traditional Mexican foods (e.g., *atole*, tacos, quesadillas, corn cakes), but with Western methods of preparation.

Associations of the prudent dietary pattern with health outcomes. The prudent dietary pattern was associated with a more favorable glycemic profile (lower fasting glucose, C-peptide, and CP-IR) and lower metabolic risk (lower MetS *z* score) in boys. Our results align with the finding of Romero-Polvo et al. (11) that a prudent dietary pattern corresponded with lower fasting glucose among Mexican adolescents. Similarly, cross-sectional investigations in US (41) and Greek (42) adults reported that compliance with a diet high in fresh vegetables, fruit, legumes, and fish is inversely associated with odds of metabolic syndrome, as well as metabolic syndrome components including central adiposity, hyperglycemia, dyslipidemia, and elevated blood pressure. Together, these findings support the growing body of evidence that indicates that food groups within the prudent dietary pattern, namely fruit and vegetables and whole grains, may be protective against obesity-related disease via their vitamin and mineral, phytochemical, and fiber contents (43–45). A potential explanation for why we observed an association of this dietary pattern with glycemia and metabolic risk in boys but not girls could be that boys undergo puberty later than girls (46). Given that the pubertal transition is a time of rapid physiologic change, including a temporary increase in insulin resistance (47), and high interindividual variability in metabolism, it is possible that the slower pubertal onset in male participants enabled us to detect a relation between diet and metabolic health biomarkers.

Of note, the prudent dietary pattern was associated with higher blood pressure in girls. However, accounting for height, which is a determinant of blood pressure in children (28), attenuated the association, suggesting that this finding was driven by a positive relation between the prudent dietary pattern score and linear growth. Although we are not aware of any studies that have specifically examined the relation between a prudent dietary pattern and linear growth or height, our finding makes sense in light of the fact that key food groups within this dietary pattern—namely, dark leafy greens, chicken, legumes, and fish—contain important micronutrients (e.g., iron, calcium, and B vitamins in the leafy greens and legumes) and macronutrients (e.g., lean protein from the chicken and fish) that are necessary for linear growth (48).

Associations of the transitioning dietary pattern and health outcomes. The transitioning dietary pattern was associated with higher subcutaneous adiposity (according to the sum of the subscapular and triceps skinfold thicknesses) in girls. We

TABLE 5 Sex-specific associations of dietary patterns with lipid profile among 224 Mexican adolescents[1]

	Associations of each dietary pattern factor score with serum lipid concentrations, mg/dL			
	Total cholesterol	TGs	LDL cholesterol	HDL cholesterol
Boys (n = 108)				
Factor 1: prudent dietary pattern				
Model 1	−1.61 (−7.83, 4.61)	3.50 (−4.89, 11.90)	−3.77 (−8.77, 1.22)	1.45 (−0.99, 3.91)
Model 2	−1.58 (−7.80, 4.64)	3.60 (−4.77, 11.97)	−3.72 (−8.72, 1.26)	1.43 (−1.01, 3.87)
Model 3	−1.88 (−8.05, 4.29)	4.01 (−4.51, 12.52)	−3.80 (−8.75, 1.15)	1.12 (−1.29, 3.53)
Factor 2: transitioning dietary pattern				
Model 1	−2.74 (−7.85, 2.36)	−5.65 (−12.50, 1.21)	−0.75 (−4.91, 3.40)	−0.86 (−2.88, 1.16)
Model 2	−2.63 (−7.81, 2.55)	−5.32 (−12.26, 1.63)	−0.53 (−4.74, 3.68)	−1.03 (−3.08, 1.00)
Model 3	−3.53 (−8.66, 1.61)	−5.06 (−12.16, 2.03)	−1.07 (−5.26, 3.12)	−1.44 (−3.45, 0.56)
Girls (n = 116)				
Factor 1: prudent dietary pattern				
Model 1	0.26 (−4.19, 4.70)	1.63 (−6.58, 9.84)	−0.35 (−3.91, 3.20)	0.28 (−1.69, 2.26)
Model 2	0.27 (−4.18, 4.71)	1.67 (−6.52, 9.86)	−0.35 (−3.91, 3.20)	0.29 (−1.69, 2.26)
Model 3	0.37 (−4.09, 4.83)	1.63 (−6.52, 9.79)	−0.29 (−3.86, 3.28)	0.33 (−1.65, 2.31)
Factor 2: transitioning dietary pattern				
Model 1	−3.91 (−8.94, 1.13)	−1.18 (−10.57, 8.21)	−2.18 (−6.23, 1.87)	−1.50 (−3.74, 0.75)
Model 2	−4.19 (−9.27, 0.89)	−1.78 (−11.25, 7.69)	−2.20 (−6.30, 1.89)	−1.63 (−3.90, 0.63)
Model 3	−4.04 (−9.15, 1.07)	−2.09 (−11.53, 7.36)	−2.07 (−6.19, 2.04)	−1.55 (−3.82, 0.72)

[1] Values are βs (95% CIs) for each outcome per 1-unit increase in the dietary pattern factor score. Model 1 adjusted for mother's marital status, education, and smoking habits during pregnancy and for child's age; model 2 adjusted as for model 1 and for child's physical activity (hours per week); model 3 adjusted as for model 2 and for child's pubertal status (prepubertal vs. pubertal).

also noted that, in general, both dietary patterns were associated with higher adiposity in girls (e.g., positive associations with BMI z score, waist circumference, and subscapular and triceps skinfold thicknesses) but lower adiposity in boys, even after multivariable adjustment. These findings may reflect differences in body composition between males and females during puberty [i.e., girls tend to gain fat mass, whereas boys accrue fat-free mass (49)] or, possibly, residual sex-specific confounding. Nevertheless, these differences are noteworthy given that previous studies that examined associations of diet with adiposity and metabolic health did so for boys and girls together (9–11), which may not be appropriate given that within the age range of our study population, girls are likely to be further along in the pubertal transition than boys (46) and thus are also more likely to exhibit puberty-related metabolic changes (34). Additional studies in other populations of similarly aged youth are required to confirm our findings.

Strengths and limitations. This study has several strengths. First, we used PCA to characterize dietary patterns, which is a data-driven way to assess eating habits and diet quality (6), as opposed to focusing on individual foods or nutrients. Second, in addition to measures of adiposity, we were able to measure several metabolic biomarkers, which are not only difficult to obtain from pediatric populations but also provide valuable insight into metabolic disturbances that could occur independently of excess fat mass. Third, we examined the relations of interest in a population of youth currently undergoing puberty, which may be a vulnerable period for the development of obesity and obesity-related metabolic disturbances (50) and thus identification of risk factors during this life stage could unveil avenues for effective intervention.

This study also has several limitations. First, we noted a lack of significance for many of the diet–metabolic biomarker associations despite the fact that the direction and magnitude of the relations were consistent. This could be due to the relatively small sample size or, possibly, that the effect of diet on metabolic health may manifest with age over time. Future studies in large samples that explore prospective associations between dietary patterns and metabolic health beyond adolescence are warranted. Second, the cross-sectional study design precludes inference on temporality or causality. Third, the prudent and transitioning dietary patterns explained ~15% of variance in the original food groups; thus, a large proportion of dietary habits may not have been captured. Nevertheless, the percentage variance explained by these 2 dietary patterns is similar in magnitude to what has been reported in other studies (~15–20%) (11, 51–53). Fourth, because we obtained dietary information via FFQ, there may have been recall bias in the reporting of food intake due to memory errors or between-participant variability in the extent to which the dietary reporting was completed by the child or the caregiver. In addition, because the instrument we used inquired about food intake during the past 7 d, the responses captured may not be completely representative of long-term dietary habits. Fifth, although the FFQ was validated for women living in Mexico City (54), it has not yet been validated in children or adolescents. However, a study in 300 schoolchildren in Mexico City found similar dietary preferences between the children and their mothers (55), thus providing support for the use of the FFQ in the present study population. Sixth, although we cannot rule out the possibility of false-positive findings, we do not foresee this to be an issue given that we only examined associations of 2 dietary patterns with a set of correlated outcomes. Moreover, the goal of this study was to examine and compare the direction, magnitude, and significance of the associations, rather than to focus on significance. Finally, our results may not be generalizable to the Mexican population as a whole given that ELEMENT comprises urban adolescents in a specific region in central Mexico.

Conclusions. In conclusion, we identified 2 major dietary patterns in this population of Mexican adolescents: a prudent dietary pattern and a transitioning dietary pattern. The prudent pattern was associated with a more favorable glycemic profile and lower metabolic risk in boys, whereas the transitioning pattern corresponded with higher adiposity in girls. Although longitudinal studies are needed to ascertain temporality between these dietary patterns and the health outcomes, our results add to the growing body of literature that indicates that higher intakes of fruit and vegetables, whole grains, legumes, and lean protein are likely beneficial for metabolic health, whereas the consumption of fried foods, refined carbohydrates, and sugar-sweetened beverages may lead to excess adiposity. Given that adolescence is a key developmental period for metabolic and behavioral health (56), efforts to improve nutrition and encourage healthy habits during this life stage may have lifelong benefits.

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