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Prenatal dietary patterns in relation to adolescent offspring adiposity and adipokines in a Mexico City cohort

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Abstract

Maternal diet during pregnancy has been associated with obesity among offspring. The extent to which trimester-specific dietary patterns are associated with markers of adiposity during adolescence remains unclear. We examined associations between prenatal diet patterns with adolescent offspring measures of adiposity and adipokines in 384 mother–adolescent dyads from the Mexico City ELEMENT cohort. Trimester-specific diet patterns were derived from principal component analysis of food frequency questionnaire data. Adolescent anthropometry and serum leptin and adiponectin were measured at 10–17 years. Three maternal diet patterns were identified: Prudent Diet (PD), high in fish and vegetables, the High Meat and Fat Diet (HMFD), high in pork and processed meats, and the Transitioning Mexican Diet (TMD), high in corn tortillas and sugar-sweetened beverages. Multiple linear regression was used to estimate sex-stratified associations among quartiles of diet patterns with adiposity and adipokines, adjusting for maternal marital status, education, and parity. First trimester TMD was associated with greater anthropometric measures and higher leptin in females, while third trimester HMFD was associated higher body fat percentage, triceps thickness, waist circumference, and leptin, but lower adiponectin among males. Contrary to expectation, there were positive associations between the trimester 1 PD pattern and anthropometric measurements in females, and for trimester 2 HMFD and TMD patterns with adipokines among males. Findings suggest maternal diet patterns may influence offspring adiposity markers during adolescence in a sex-specific manner.

Introduction

Developmental programing research suggests that the *in utero* nutritional environment is an important determinant of offspring adiposity. The Dutch Famine study demonstrated that maternal malnutrition during the early stages of gestation was associated with offspring obesity in adulthood.[1] Other human studies link overnutrition during pregnancy with higher adiposity; to illustrate, higher animal protein intake during pregnancy has been associated with higher body mass index (BMI) in offspring at age 20.[2] Other studies have related a prenatal diet higher in carbohydrates and sugar to higher BMI in early childhood.[3]

However, there are multiple limitations to the current body of literature. First, existing research on the links between the *in utero* dietary environment and offspring metabolic health has primarily focused on offspring BMI.[2] BMI is an imperfect surrogate of adiposity and metabolic risk[4,5]; thus, other body composition markers and adiposity-related hormones are needed to uncover mechanisms and later disease risk. For example, adipokines such as leptin and adiponectin are a family of hormones secreted by fat tissue that play a role in obesity development and the relationship between obesity and other metabolic conditions.[6] These two adipokines are only produced by adipose tissue.[7] In particular, leptin has been shown to suppress appetite, while adiponectin may protect against insulin resistance.[8] Considering the role of these hormones is imperative as they provide insight into the development of cardiovascular disease during adolescence and may be more sensitive to changes during puberty.[9]

Second, few studies have examined the role of the *in utero* dietary environment on offspring metabolic health during adolescence, a time of multiple developmental changes. Adolescence is a sensitive period for the development of obesity-related diseases.[10]

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Specifically, puberty is characteristic of changes in adipose tissue distribution, placing some adolescents at a greater risk for cardiovascular disease than others.[11] Moreover, studies have demonstrated sex differences in measures of total body fat (i.e., BMI and body fat percentage), peripheral and central fat during the progression of puberty.[11]

Third, previous research on offspring adiposity and prenatal diet has primarily focused on individual macronutrients[12] and micronutrients,[13] or pre-specified dietary quality indices, such as glycemic index[14] or the Mediterranean diet.[15] Data-driven techniques that identify naturally occurring diet patterns may offer a more comprehensive picture of prenatal diet. Further, the examination of diet patterns during each trimester allows for the possibility that diet may change throughout pregnancy.

To address gaps in the current literature, the present study aimed to investigate whether maternal trimester-specific dietary patterns were associated with measures of adiposity and adipokine levels of offspring during adolescence. Based on a *priori* knowledge, we hypothesized that healthier maternal dietary patterns would be associated with lower leptin, higher adiponectin, and lower adiposity. We further hypothesized that associations between maternal dietary patterns and adipokines would be sex-specific.

Methods

Study sample

The analytic sample included 384 mother–adolescent dyads from two sequential cohorts of the Early Life Exposures in Mexico to ENvironmental Toxicants (ELEMENT) study.[16] Mothers were recruited during their first trimester of pregnancy between 1997 and 2003 from clinics in Mexico City. Research staff administered food frequency questionnaires (FFQs) and a sociodemographic questionnaire. This study is a secondary analysis of data from the original randomized controlled trial that began in 1994.[17]

Data were available for the majority of the dyads (353) across all three trimesters. Since not all women enrolled in the study during the first trimester, the largest sample size available was during trimester 2 ($n = 384$; see Fig. 1 for more details).

During a follow-up visit of the adolescent offspring in 2015–2017, adiposity measures and fasting leptin and adiponectin levels were collected. At the follow-up visit, adolescents ranged from 10 to 17 years old. Of the 554 adolescents measured at the follow-up visit, 379 had corresponding maternal FFQ data for at least one trimester of pregnancy. Figure 1 illustrates how this subcohort relates to the overall ELEMENT cohort, and Supplemental Figure S1 shows the overall timeline of the study. The Mexico National Institute of Public Health and the University of Michigan Human Subjects Committee approved all research protocols and procedures, and all participants provided informed consent.

Exposure assessment of maternal dietary patterns

Maternal prenatal diet was assessed each trimester via a semi-quantitative FFQ with 106 items validated for use among Mexican Spanish-speaking women of reproductive age.[18,19] The FFQ asked how often each standard portion size of a food item was consumed with nine possible responses: <1 time/month or never, 1–3 times/month, 1 time/week, 2–4 times/week, 5–6 times/week, 1 time/day, 2–3 times/day, 4–5 times/day, and ≥6 times/day.

Outcome assessment of offspring anthropometry and adipokines

Measures of adolescent adiposity included waist circumference (WC), triceps skinfold thickness (TS), body fat percentage (BF %), and BMI. Participants wore a clinical examination gown and were asked to remove hair ornaments, shoes, and socks according to the ELEMENT study protocol.[16] Research assistants measured height to the nearest 0.5 cm with a Tanita stadiometer (Model WB-3000 m), weight to the nearest kg (InBody 270, Biospace, California, USA), WC to the nearest 0.1 cm at the iliac crest using a non-stretchable measuring tape (QM2000 QuickMedical; SECA model 201, Hamburg, Germany), and TS in mm (Lange calipers; Beta Technology, CA, USA) using standard anthropometry procedures.[20] BF% was estimated using bioelectrical impedance equipment (InBody 270, Biospace, CA, USA). Staff obtained duplicate measures for height, WC, and TS, and the average of two measurement values was used for analysis. Adolescent weight and height were used to calculate BMI (kg/m^2) and standardized as a z score for sex and age using the World Health Organization (WHO) growth reference.[20]

Fasting serum samples were collected from adolescents, frozen at −80°C, and shipped on dry ice to the University of Michigan Diabetes Research and Training Center Chemistry Lab (Ann Arbor, MI). Leptin and adiponectin levels were measured using RIA (Millipore).

Covariates

Mothers reported sociodemographic characteristics at enrollment, including age at pregnancy, parity, education, and marital status. Maternal age was operationalized into four categories: 14–23, 23–26, 26–30, and 30–44 years old. Parity was categorized as 0 or 1, 2, ≥3. Maternal education was separated into the following categories: did not complete secondary (<9 years), completed some high school (9 to <12 years), completed high school (12 years), higher education (>12 years). Marital status was coded as a dichotomous variable: married or civil union and single, separated, divorced or widowed. Covariates were determined using a *priori* knowledge.[21]

Statistical analysis

Principal component analysis (PCA) was used to identify dietary patterns in each trimester. Individual food items from the FFQ were put into 40 nutritionally similar food groups based on a nutrient profile.[22] Raw response frequencies of consumption values (1–9) were converted to servings per day and then adjusted for total energy intake using the residual method.[23] Next, PCA was performed and rotated orthogonally to obtain uncorrelated factors. The number of factors to keep was determined based on interpretability and visual inspection of scree plots and eigenvalues >1. Food groups with factor loadings >+0.30 or <−0.30 were considered relevant for the pattern. Scores were calculated by multiplying the factor loadings by the frequency of consumption in each group and then summing. Diet patterns were named in accordance with a prior publication for consistency, but factor loadings and food pattern scores are slightly different (due to differences in the analytic sample sizes).[24] Diet pattern scores were categorized into quartiles for analysis. Mothers received a score for each diet pattern, and scores were uncorrelated with one another.

We examined crude associations between maternal characteristics and the exposure (dietary patterns) as well as outcomes (leptin, adiponectin, and the adiposity measures). We estimated

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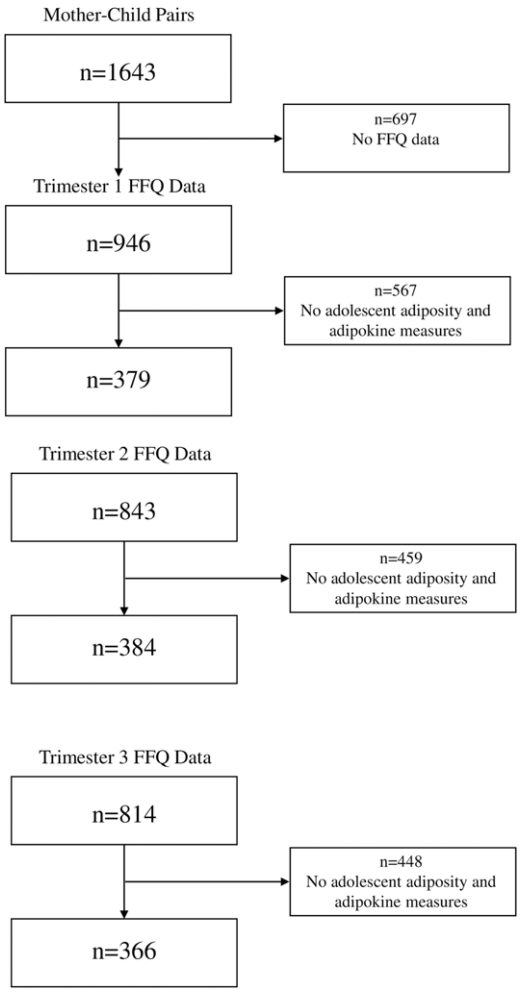

Figure 1. Trimester 1 data were available for 379 mother–adolescent dyads. Eight dyads from trimester 1 did not have follow-up data for trimester 2, and 22 did not have follow-up data for trimester 3. Data from 384 dyads were available in trimester 2 and 13 of these dyads lacked trimester 1 data. Trimester 3 data were available for 366 dyads, 9 of which did not have trimester 1 data.

Spearman correlations between adiposity measures and adipokines. Adolescent anthropometric variables and adipokines were analyzed in relation to maternal prenatal diet pattern quartiles using multiple linear regression. All analyses were sex-stratified. Quartile 1 (Q1) was used as the reference group, and the analysis was adjusted for maternal marital status, education, and parity. In sensitivity analysis, we further adjusted for pubertal status (menarche status for girls and testicular volume for boys, measured as described elsewhere[25]) and total energy intake. All analyses were conducted using R Software (version 3.5.0; Boston, MA). A 2-sided α level of 0.05 was considered statistically significant.

Results

The mean ± SD age of adolescents was 13.7 ± 1.9 years, and 49.3% of the sample was female. Average adolescent leptin and adiponectin levels were 24.3 ± 18.2 ng/ml and 11.7 ± 3.9 ng/ml. Average triceps thickness was 18.5 ± 6.9 mm, WC was 78.8 ± 11.7 cm, BMI-Z-score was 0.6 ± 1.3, and BF % was 26.8 ± 9.8%. The mean ± SD age of mothers at delivery was 26.6 ± 5.6 years. There were no significant associations between maternal demographic characteristics and adolescent anthropometry measures. Maternal characteristics were not associated with adipokines, except higher maternal parity was associated with lower leptin levels (Supplemental Table S1).

Three prenatal maternal dietary patterns for trimester 1 were derived using PCA (Table 1). The Prudent Diet (PD) pattern was high in fish, tomatoes, potatoes, fruit, cruciferous vegetables, yellow vegetables, leafy vegetables, other vegetables, legumes, and soup and accounted for 7% variance. The High Meat and Fat Diet (HMFD) was high in beef, pork, processed meat, chili, chips, spread, and Mexican foods, low in milk, and accounted for 5% variance. The third pattern, the Transitioning Mexican Diet (TMD), was high in chilis, corn tortillas, sugar beverages, coffee, low in beef, spreads, and high-fat dairy and accounted for 5% variance. Similar patterns were observed in trimesters 2 and 3 (Supplemental Tables S2 and S3, respectively). Across pregnancy, there was moderately high correlation for the PD (rho between 0.4 and 0.6), but the other diet patterns were not as well-correlated over time. The three diets account for 17% variance of the maternal diets, which is similar to the variance explained by diet patterns in other studies (e.g., 15%[26], 21%[27] and 30%[28]).

Associations between maternal sociodemographic characteristics and maternal diet patterns are shown in Table 2. Maternal age was positively associated with the PD pattern, and education was positively associated with the PD and TMD patterns.

Spearman correlations between the adipokines and anthropometric measures were statistically significant (Supplement Table S4). Leptin was most highly correlated with BF% ($r = 0.88$), and adiponectin was negatively correlated with WC ($r = -0.32$).

PD pattern

There was a non-linear positive association between the trimester 1 PD pattern and WC in adjusted models (Table 3) among females only. To illustrate, females in Q2 had a 0.76 cm larger WC (95% CI: 0.32, 1.21) than the reference group, and quartile 4 (Q4) had a 0.26 cm larger WC (95% CI: −0.18, 0.70). In addition, PD was associated with BMIZ-score in females, with Q2 having a 0.58 greater BMIZ-score than the reference group (95% CI: 0.16, 0.99).

HMFD pattern

In trimester 2, there was a positive association between the HMFD pattern and male adiponectin, such that those in quartile 4 had 2.3 ng/mL higher adiponectin levels than those in quartile 1 (95% CI: 0.7, 3.9; Supplemental Table S6).

In trimester 3, there were positive associations between the HMFD pattern with body fat percentage, triceps thickness, and WC among males. To illustrate, those in quartile 4 versus quartile 1 had 0.7 (95% CI: 0.2, 1.1; p for trend = 0.05), 0.7 (95% CI: 0.2, 1.2; p for trend = 0.02), and 0.6 (95% CI: 0.1,1.0; p for trend = 0.21) higher body fat percentage, triceps thickness, and WC, respectively (Table 4). There was a positive association between the HMFD pattern and leptin among males, such that those in quartile 4 versus quartile 1 had 5.99 higher ng/mL leptin (95% CI: 0.07, 11.9; p for trend = 0.06) (Table 5). Finally, there was a non-linear inverse association with adiponectin; those in quartile 2 had −1.7 lower ng/mL adiponectin than those in quartile 1 (95% CI: −3.4, −0.02) (Table 5).

TMD pattern

The trimester 1 prenatal TMD pattern was positively associated with fat distribution among female offspring (Table 3). To

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Table 1. Trimester 1 principal component loadings of foods for selected principal components

	Prudent Diet	High Meat and Fat Diet	Transitioning Mexican Diet[a]
Milk	−0.246	−0.532*	−0.141
High-fat dairy	0.218	0.080	−0.318*
Yogurt	−0.022	−0.263	−0.160
Ice cream	−0.038	0.188	−0.090
Fruit	0.368*	−0.216	0.007
Egg	0.031	0.018	−0.056
Chicken	0.100	−0.003	−0.150
Beef	0.077	0.309*	−0.320*
Pork	−0.084	0.446*	−0.001
Processed meat	−0.058	0.492*	−0.156
Organ meat	0.259	0.000	−0.067
Fish	0.364*	−0.015	−0.267
Tomato	0.398*	−0.020	0.188
Potato	0.337*	0.103	−0.215
Avocado	0.287	0.001	0.070
Cruciferous vegetables	0.407*	0.041	−0.108
Yellow vegetables	0.551*	−0.119	−0.207
Leafy vegetables	0.589*	−0.061	−0.081
Legumes	0.471*	0.150	0.097
Corncob	0.268	0.139	−0.091
Other vegetables	0.624*	−0.131	−0.011
Soup	0.331*	0.078	−0.139
Chili	0.104	0.387*	0.382*
Corn flour drink (Atole)	−0.005	0.005	−0.251
Corn tortilla	−0.074	0.083	0.656*
Refined grain	−0.062	0.169	−0.139
Whole grain	0.196	−0.161	−0.171
Chips	−0.147	0.443*	−0.078
Dessert	−0.254	0.064	−0.007
Jam	0.085	0.042	−0.287
Sugar beverages	−0.038	−0.034	0.471*
Diet soda	0.057	0.002	0.100
Coffee	−0.050	0.170	0.355*
Wine	0.073	0.216	0.141
Unsaturated oil	0.145	0.136	0.146
Butter	0.050	0.282	−0.287
Spread	0.039	0.362*	−0.322*
Crisco	0.056	0.089	−0.008
Mexican foods	−0.073	0.478*	0.098
Natural juice	0.158	−0.185	−0.171
Percent variance explained	7	5	5

*Foods considered meaningful if loadings were greater than an absolute value of 0.3.
[a]Factor loadings and diet pattern scores multiplied by (−1) for interpretability (i.e., to load positively with tortillas and sugar-sweetened beverages).

illustrate, Q4 of the TMD pattern was significantly greater than the reference for body fat percentage (0.48; 95% CI: 0.11, 0.85; p for trend = 0.09), triceps thickness (0.51 mm; 95% CI: 0.07, 0.95; p for trend = 0.15), and WC (0.52 cm; 95% CI: 0.05, 0.98; p for trend = 0.32). There was also a positive association between trimester 1 TMD pattern and leptin levels in females only (Table 6). To highlight, compared to female adolescents in the lowest quartile (Q1) of the prenatal maternal TMD pattern, the highest quartile (Q4) of the TMD pattern had 10.9 ng/mL higher leptin (95% CI: 3.8, 18.0; p for trend = 0.01).

In trimester 2, the TMD pattern was associated with lower leptin among male offspring alone (p for trend 0.03; Supplemental Table S6), but it was not associated with other adiposity indicators.

In sensitivity analyses, there were no marked differences with respect to the direction, magnitude, or precision in the estimates after adjusting for pubertal status and total energy intake.

Discussion

In this cohort of Mexican adolescents, we observed a positive association between a trimester 1 TMD pattern and body fat percentage, triceps skinfolds, WC, and leptin levels in the female adolescent offspring. We also found positive associations between a trimester 3 HMFD pattern with anthropometric and adipokine adiposity indicators among boys only. There were also some associations in the unexpected direction, including positive associations between a trimester 1 PD pattern and female WC and BMIZ-score.

The trimester 1 TMD was associated with higher leptin levels in females and higher adiposity (body fat percentage, WC, and triceps skinfolds). This dietary pattern was high in sugar-sweetened beverages, and of the dietary patterns identified, it was most similar to a Western diet pattern. From this perspective, the study findings align with some previous childhood studies. For example, previous research in a Singaporean population found a positive association between higher maternal sugar intake and infancy and early childhood BMI.[3] Similarly, a previous study among a Dutch population found a positive association between maternal intake of sugar-sweetened beverages and childhood BMI and fat mass.[29] Further, an animal study found that mice fed a diet rich in fats, sugar, and salt (a "Western" diet) during pregnancy gave birth to pups that went on to gain more weight and have more adipose tissue compared to the pups born from mothers eating a control diet.[30]

In trimester 3, a maternal HMFD pattern was associated with higher levels of adiposity in male offspring. In this trimester, the HMFD pattern loaded highly on high-fat dairy, chicken, processed meat, spread, and low on tortillas and sugar-sweetened beverages. This association also has biologic plausibility, especially given that the late third trimester of intra-uterine life is a period of fat

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Table 2. Associations between maternal sociodemographic characteristics and maternal prenatal diet patterns scores

	Sample size (*n*)	Prudent diet, Mean (SD)	High Meat and Fat Diet, Mean (SD)	Transitioning Mexican, Mean (SD)
Maternal age, years				
14–23	95	−0.34 (0.92)	−0.05 (1.10)	0.085 (0.990)
23–26	77	−0.09 (1.09)	−0.08 (1.01)	−0.00 (1.05)
26–30	101	−0.02 (0.93)	−0.031 (1.084)	−0.15 (0.85)
30–44	106	0.13 (1.05)	−0.256 (0.942)	0.08 (1.10)
P[a]		0.001*	0.20	0.74
Parity				
0 or 1 child	138	−0.12 (1.03)	−0.11 (1.10)	0.02 (0.94)
2 children	134	−0.06 (0.92)	−0.13 (1.06)	−0.04 (0.98)
3 or more children	107	−0.03 (1.08)	−0.08 (0.92)	0.03 (1.11)
P[a]		0.49	0.83	0.98
Maternal education, years				
Did not complete secondary (<9)	46	−0.17 (0.93)	0.036 (1.05)	0.40 (1.07)
Completed some high school (9 to <12)	155	−0.26 (0.95)	−0.15 (1.05)	0.10 (0.98)
Completed high school (12)	127	0.11 (1.12)	−0.10 (1.03)	−0.10 (1.00)
Higher education (>12)	51	0.010 (0.84)	−0.13 (1.01)	−0.31 (0.91)
P[a]		0.007*	0.69	0.001*
Marital status				
Married or civil union	338	−0.08 (1.03)	−0.33 (1.25)	0.06 (1.00)
Single, separated, divorced, or widowed	41	−0.08 (1.00)	−0.08 (1.01)	−0.00 (1.00)
P[a]		0.99	0.15	0.70

*$P < 0.05$.
[a]*P* values calculated from one-way ANOVA.

accumulation for the offspring.[31] Further, a meta-regression analysis of animal model studies found that maternal high-fat diet was associated with increased adiposity during adulthood in male offspring,[32] aligning with our study finding. A separate animal study revealed that exposure to a maternal high-fat diet rich in monounsaturated fatty acids programed male offspring fatty acid metabolism and predisposed to greater adiposity in adulthood.[33]

To our knowledge, no other studies have examined the links between prenatal dietary patterns and offspring adiposity during adolescence. Yet, results from human epidemiologic studies generally support our first and third trimester findings in that poor diet quality throughout gestation has been associated with increased neonatal offspring adiposity,[34] (although sex-specific findings were not reported). A separate US study reported inverse associations between maternal diet quality during pregnancy and offspring adiposity in early postnatal life.[35]

Some of the statistically significant associations were not in the hypothesized directions. First, the finding that higher trimester 1 PD was non-linearly associated with higher adiposity among females was not in line with expectations, given that the PD pattern was high in fruits and vegetables. A few previous studies have found that a healthier prenatal maternal diet was associated with a lower prevalence of overweight and obesity in adolescents.[36,37] In contrast, higher adherence to a Healthy Eating Index during pregnancy was associated with lower adiponectin levels in male offspring at 4–7 years old.[38] Other studies report null associations between pregnancy diet and adolescent adiposity after accounting for confounders.[36] Thus, it is important to note that our study results may be impacted by unmeasured confounding such as sociodemographic and lifestyle factors specific to the present Mexico City cohort. One possible non-causal explanation for our results is that the PD pattern is associated with higher economic stability in the home, which could be associated with higher weight gain. We have previously shown that higher economic stability is associated with larger, more regular meals, potentially leading to higher overall caloric intake in the Mexican working-class context.[39] Nonetheless, results remained the same when adjusted for energy intake in our analysis. A few other associations were also not in the expected direction, namely the trimester 2 associations among boys between HMFD and TMD diet patterns with adiponectin and leptin, respectively. The possibility of chance findings should not be discounted, given that statistically significant associations were not observed across the anthropometric indicators.

Sex differences in our associations are potentially due to variation in body composition and hormones impacting adipokine and anthropometric variables across sexes. For example, higher subcutaneous fat levels in females compared to males have been related to higher leptin levels, possibly due to greater mRNA expression of

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Table 3. Sex-stratified adjusted associations[a] between trimester 1 diet patterns and offspring adolescent anthropometric measures

		Body fat percentage		Triceps skinfolds, mm		Waist circumference, cm		BMIZ-score	
	n	Females	Males	Females	Males	Females	Males	Females	Males
Prudent Diet									
Q1	95	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Q2	95	0.45 (0.08, 0.81)*	0.27 (−0.18, 0.72)	0.41 (−0.02, 0.85)	0.23 (−0.24, 0.70)	0.76 (0.32, 1.21)*	0.20 (−0.27, 0.67)	0.58 (0.16, 0.99)*	0.05 (−0.40, 0.49)
Q3	94	0.02 (−0.34, 0.37)	0.03 (−0.43, 0.49)	0.03 (−0.40, 0.45)	−0.09 (−0.56, 0.38)	0.28 (−0.15, 0.72)	0.06 (−0.41, 0.54)	−0.05 (−0.46, 0.36)	−0.11 (−0.56, 0.34)
Q4	95	0.13 (−0.23, 0.49)	0.46 (−0.01, 0.92)	−0.07 (−0.49, 0.36)	0.34 (−0.14, 0.82)	0.26 (−0.18, 0.70)	0.31 (−0.17, 0.79)	0.09 (−0.32, 0.51)	0.15 (−0.31, 0.61)
P[b]		0.07	0.62	0.55	0.48	0.73	0.84	0.35	0.92
High Meat and Fat Diet									
Q1	95	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Q2	95	0.15 (−0.21, 0.51)	−0.11 (−0.58, 0.35)	0.31 (−0.11, 0.74)	0.41 (−0.07, 0.89)	0.13 (−0.31, 0.58)	0.21 (−0.27, 0.69)	0.29 (−0.13, 0.71)	−0.02 (−0.48, 0,43)
Q3	94	0.25 (−0.12, 0.62)	−0.21 (−0.67, 0.24)	0.35 (−0.09, 0.79)	0.01 (−0.46, 0.48)	0.26 (−0.20, 0.72)	0.05 (−0.41, 0.52)	0.12 (−0.32, 0.55)	−0.10 (−0.54, 0.35)
Q4	95	0.13 (−0.25, 0.51)	−0.23 (−0.66, 0.21)	0.39 (−0.06, 0.84)	−0.05 (−0.50, 0.39)	0.04 (−0.43, 0.52)	−0.09 (−0.55, 0.35)	0.13 (−0.31, 0.58)	−0.29 (−0.71, 0.13)
P[b]		0.42	0.79	0.26	0.52	0.77	0.94	0.4	0.60
Transitioning Mexican Diet									
Q1	95	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Q2	95	0.18 (−0.18, 0.55)	−0.15 (−0.61, 0.31)	0.21 (−0.22, 0.65)	0.13 (−0.34, 0.61)	0.37 (−0.09, 0.82)	0.10 (−0.37, 0.57)	0.14 (−0.29, 0.56)	0.02 (−0.43, 0.47)
Q3	94	0.28 (−0.09, 0.64)	−0.09 (−0.54, 0.37)	0.19 (−0.25, 0.62)	0.26 (−0.21, 0.73)	0.24 (−0.22, 0.69)	0.33 (−0.13, 0.80)	0.22 (−0.21, 0.65)	0.26 (−0.18, 0.70)
Q4	95	0.48 (0.11, 0.85)*	−0.20 (−0.66, 0.26)	0.51 (0.07, 0.95)*	0.03 (−0.45, 0.51)	0.52 (0.05. 0.98)*	0.11 (−0.37, 0.59)	0.40 (−0.04, 0.83)	0.04 (−0.42, 0.49)
P[b]		0.09	0.91	0.15	0.59	0.32	0.9	0.11	0.90

*Denotes $P < 0.05$.
[a]From linear regression models adjusted for marital status, maternal education, and maternal parity.
[b]*P* for trend estimated by including a continuous ordinal variable representing quartiles of dietary pattern adherence into the linear regression models.

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Table 4. Sex-stratified adjusted associations[a] between trimester 3 maternal prenatal diet patterns and offspring adolescent anthropometric measures

		Body fat percentage		Triceps skinfolds, mm		Waist circumference, cm		BMIZ-score	
	n	Females	Males	Females	Males	Females	Males	Females	Males
Prudent Diet									
Q1	95	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Q2	95	−0.15 (−0.54, 0.23)	−0.15 (−0.60, 0.30)	−0.23 (−0.69, 0.22)	−0.14 (−0.60, 0.32)	−0.16 (−0.64, 0.31)	−0.22 (−0.68, 0.24)	−0.15 (−0.60, 0.30)	−0.29 (−0.74, 0.15)
Q3	94	0.25 (−0.13, 0.63)	−0.29 (−0.75, 0.16)	0.07 (−0.38, 0.51)	0.24 (−0.23, 0.71)	0.22 (−0.25, 0.68)	0.03 (−0.44, 0.50)	0.31 (−0.14, 0.75)	−0.07 (−0.53, 0.38)
Q4	95	0.15 (−0.23, 0.54)	0.18 (−0.27, 0.64)	−0.07 (−0.52, 0.38)	0.26 (−0.20, 0.73)	0.30 (−0.17, 0.77)	0.16 (−0.30, 0.63)	−0.06 (−0.51, 0.39)	−0.00 (−0.45, 0.45)
P[b]		0.13	0.96	0.63	0.26	0.33	0.91	0.65	0.89
High Meat and Fat Diet									
Q1	95	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Q2	95	0.07 (−0.30, 0.44)	0.37 (−0.08, 0.83)	0.23 (−0.20, 0.67)	0.16 (−0.31, 0.63)	0.07 (−0.39, 0.52)	0.30 (−0.17, 0.77)	0.19 (−0.24, 0.63)	0.29 (−0.16, 0.75)
Q3	94	0.06 (−0.33, 0.44)	0.52 (0.08, 0.97)*	−0.03 (−0.48, 0.41)	0.39 (−0.06, 0.85)	0.07 (−0.40, 0.55)	0.45 (−0.01, 0.91)	0.39 (−0.06, 0.84)	0.40 (−0.04, 0.84)
Q4	95	−0.21 (−0.59, 0.18)	0.68 (0.23, 1.14)*	−0.12 (−0.57, 0.33)	0.68 (0.21, 1.15)*	−0.32 (−0.79, 0.15)	0.56 (0.08, 1.03)*	−0.01 (−0.46, 0.43)	0.44 (−0.02, 0.90)
		0.19	0.05*	0.51	0.02*	0.55	0.21	0.68	0.29
Transitioning Mexican Diet									
Q1	95	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Q2	95	−0.07 (−0.45, 0.30)	−0.21 (−0.66, 0.25)	−0.04 (−0.48, 0.40)	0.23 (−0.23, 0.69)	−0.11 (−0.58, 0.35)	−0.07 (−0.54, 0.39)	0.06 (−0.38, 0.50)	−0.15 (−0.60, 0.30)
Q3	94	−0.15 (−0.53, 0.24)	−0.26 (−0.72, 0.20)	−0.03 (−0.48, 0.42)	−0.30 (−0.76, 0.17)	−0.10 (−0.57, 0.37)	−0.29 (−0.76, 0.18)	−0.17 (−0.61, 0.28)	−0.39 (−0.85, 0.06)
Q4	95	−0.22 (−0.59, 0.14)	−0.06 (−0.53, 0.42)	−0.25 (−0.68, 0.18)	0.25 (−0.23, 0.72)	−0.39 (−0.84, 0.06)	0.15 (−0.33, 0.63)	−0.38 (−0.81, 0.04)	−0.10 (−0.56, 0.36)
		0.16	0.98	0.43	0.57	0.39	0.99	0.17	0.80

*Denotes $P < 0.05$.
[a]From linear regression models adjusted for marital status, maternal education, and maternal parity.
[b]*P* for trend estimated by including a continuous ordinal variable representing quartiles of dietary pattern adherence into the linear regression models.

Table 5. Adjusted associations between trimester 3 maternal diet patterns and offspring adolescent leptin and adiponectin levels by sex

		Leptin (ng/ml)[a]		Adiponectin (ng/mL)[a]	
	n	Females	Males	Females	Males
Prudent Diet					
Q1	95	Reference	Reference	Reference	Reference
Q2	95	−3.94 (−11.42, 3.54)	−2.62 (−8.29, 3.04)	2.14 (0.48, 3.81)*	0.40 (−1.25, 2.06)
Q3	94	3.85 (−3.42, 11.12)	−0.99 (−6.77, 4.79)	0.58 (−1.0, 2.2)	0.02 (−1.67, 1.71)
Q4	95	2.16 (−5.18, 9.50)	3.57 (−2.24, 9.37)	1.32 (−0.31, 3.0)	0.57 (−1.12, 2.27)
P[b]		0.4	0.21	0.51	0.94
High Meat and Fat Diet					
Q1	95	Reference	Reference	Reference	Reference
Q2	95	−0.36 (−7.51, 6.80)	2.99 (−0.88, 8.87)	−0.31 (−1.91, 1.29)	−1.72 (−3.42, −0.02)*
Q3	94	−0.02 (−7.54, 7.50)	5.15 (−0.55, 10.84)	0.22 (−1.46, 1.90)	−0.78 (−2.43, 0.87)
Q4	95	−1.72 (−9.07, 5.64)	5.99 (0.07, 11.90)*	0.39 (−1.26, 2.03)	−0.13 (−1.84, 1.58)
P[b]		0.64	0.06	0.55	0.97
Transitioning Mexican Diet					
Q1	95	Reference	Reference	Reference	Reference
Q2	95	−2.75 (−9.91, 4.41)	−0.28 (−6.09, 5.52)	0.30 (−1.32, 1.92)	−0.29 (−1.96, 1.39)
Q3	94	−5.51 (−12.84, 1.82)	−2.13 (−8.02, 3.76)	0.50 (−1.16, 2.15)	0.64 (−1.06, 2.33)
Q4	95	−6.66 (−13.76, 0.43)	0.24 (−5.75, 6.23)	0.17 (−1.43, 1.77)	0.85 (−0.87, 2.58)
P[b]		0.18	0.36	0.61	0.72

*Denotes $P < 0.05$.
[a]From linear regression models adjusted for marital status, maternal education, and maternal parity.
[b]*P* trend estimated by including a continuous ordinal variable representing quartiles of dietary pattern adherence into the linear regression models.

Table 6. Adjusted associations between trimester 1 diet patterns and offspring adolescent leptin and adiponectin levels by sex

		Leptin (ng/ml)[a]		Adiponectin (ng/mL)[a]	
	n	Females	Males	Females	Males
Prudent Diet					
Q1	95	Reference	Reference	Reference	Reference
Q2	95	6.62 (−0.57, 13.81)	1.09 (−4.61, 6.79)	−1.38 (−2.98, 0.22)	−0.52 (−2.15, 1.12)
Q3	94	−1.22 (−8.21, 5.77)	−2.32 (−8.02, 3.38)	−0.22 (−1.77, 1.34)	−0.01 (−1.64, 1.63)
Q4	95	−0.51 (−7.55, 6.53)	5.30 (−0.53, 11.13)	−0.27 (−1.84, 1.29)	0.46 (−1.22, 2.14)
P[b]		0.46	0.23	0.66	0.82
High Meat and Fat Diet					
Q1	95	Reference	Reference	Reference	Reference
Q2	95	5.22 (−1.79, 12.23)	0.84 (−5.07, 6.74)	−0.45 (−2.00, 1.11)	−1.04 (−2.70, 0.61)
Q3	94	1.57 (−5.68, 8.82)	−0.63 (−6.38, 5.12)	−0.57 (−2.18, 1.03)	−0.28 (−1.90, 1.33)
Q4	95	0.49 (−6.98, 7.96)	−0.96 (−6.45, 4.54)	−1.13 (−2.79, 0.52)	0.31 (−1.24, 1.85)
P[b]		0.56	0.39	0.38	0.85
Transitioning Mexican Diet					
Q1	95	Reference	Reference	Reference	Reference
Q2	95	−2.42 (−9.35, 4.51)	−0.42 (−6.24, 5.40)	−0.05 (−1.63, 1.53)	−0.67 (−2.31, 0.97)
Q3	94	1.97 (−4.99, 8.94)	−1.35 (−7.08, 4.37)	−1.55 (−3.13, 0.04)	−0.57 (−2.18, 1.05)
Q4	95	10.89 (3.78, 18.00)*	−1.06 (−6.98, 4.86)	−0.56 (−2.18, 1.05)	−0.39 (−2.06, 1.28)
P[b]		0.01*	0.39	0.4	0.89

*Denotes $P < 0.05$.
[a]From linear regression models adjusted for marital status, maternal education, and maternal parity.
[b]*P* trend estimated by including a continuous ordinal variable representing quartiles of dietary pattern adherence into the linear regression models.

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leptin in subcutaneous fat compared to visceral fat.[40] In addition, sex steroid differences could play a role. Higher testosterone levels have been associated with lower leptin levels, irrespective of body fat percentage.[41]

Many of the relationships between diet patterns and adipokines were null, which is also in line with a few other studies.[21,42,43] To illustrate, when adipokine levels were measured in 9 year olds, no associations between prenatal maternal calorie, protein, total fat, saturated fat, fiber, sugar-sweetened beverage consumption, and offspring adipokine levels were found.[42] Similarly, there were no associations between blood cord adipokine levels and maternal adherence to the Mediterranean diet.[21] Additionally, another study found no association between child adiposity or leptin levels and a higher pro-inflammatory prenatal maternal diet.[43] It is possible that only one measurement of adipokines during adolescence was not adequate to detect true underlying differences in metabolism.

The present study had various strengths and limitations. The longitudinal study design was one strength that allowed for inter-generational analysis. We also assessed pregnancy diet multiple times, which allowed us to consider the possibility that associations could vary by trimester. It was interesting to note that the non-PD patterns were not well-correlated over time, suggesting that women's diets changed over pregnancy, as reported previously.[44] Other strengths included the relatively large sample size and objective measurements. The fact that the participants are from Mexico City means that the results may not be generalizable to other populations. The FFQ is subject to measurement error and could lead to bias, notably if measurement error differed with respect to maternal weight status. Finally, specific unmeasured confounders such as maternal physical activity during pregnancy could have influenced the results.

In conclusion, a trimester 1 TMD pattern was associated with higher leptin and adiposity among female offspring, while a trimester 3 High Meat and Fat Diet was associated with higher adiposity indicators among males within this Mexican cohort. There were also a few associations in the unexpected direction, including for the trimester 1 Prudent diet pattern with female WC and BMIZ-score, and trimester 2 Transitioning Mexican and High Meat and Fat diets with male adipokines. These findings point to the potential role of pregnancy diet patterns on the long-term anthropometry and metabolic health of offspring within the Mexican population.

Supplementary materials. For supplementary material for this article, please visit https://doi.org/10.1017/S2040174422000678

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Conflicts of interest. The authors have no conflicts of interest to disclose.

Ethical standards. The study was approved by the Mexico National Institute of Public Health (INSP) and the University of Michigan Human Subjects Committee in accordance with relevant national and international guidelines for medical research.

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