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Vegetable and lean proteins -based and processed meat and refined grains -based dietary patterns in early childhood are associated with pubertal timing in a sex-specific manner: a prospective study of children from Mexico City

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Abstract

Childhood diet has been implicated in timing of sexual maturation. A key limitation of published studies is the focus on individual foods rather than patterns. We hypothesized that dietary patterns characterized by fruits and vegetables during early childhood (age 3 y) would be associated with delayed pubertal timing, while energy-dense and meat-based dietary patterns would relate to earlier puberty. The study population included 496 participants of a Mexico City birth cohort. The exposures of interest were dietary patterns derived from principal component analysis of dietary data collected via a semi-quantitative food frequency questionnaire when the children were 3 y of age, and the outcomes were physician-assessed Tanner stages for pubic hair, breast (girls), genitalia and testicular volume (boys) between 9 and 18 y, and initiation of menarche (girls). In regression analyses, we estimated adjusted hazard ratios (HR) and 95% confidence intervals (CI) for having reached Tanner stage 4 or initiation of menarche in girls, and testicular volume ≥15 mL in boys. Among girls, those in the highest vs. lowest tertile of *vegetables and lean proteins* pattern had a 35% (95% CI 3% to 67%) lower adjusted probability of having reached breast stage 4. Among boys, the *processed meat and refined grain* pattern score was associated with more advanced testicular development (adjusted HR = 3.58 [0.62 to 6.53] for testicular volume ≥15 mL for highest vs. lowest tertile). Early childhood dietary patterns may play a role in the tempo of sexual maturation, which could ultimately carry implications for chronic disease susceptibility.

Keywords

meat; vegetables; menarche; puberty; pediatrics

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1. INTRODUCTION

The timing of puberty is a determinant of chronic disease risk,[1] and is associated with diet during childhood [2, 3]. For example, higher frequency of red meat intake at ages 5–12 y (years) has been related to earlier menarche among Colombian girls [4], and higher premenarcheal sugar-sweetened beverages (SSB) intake has been associated with earlier menarche in a US study [5]. In contrast, isoflavones and vegetable protein have been related to delayed age at menarche [6, 7]. The number of investigations among boys remains limited and mostly cross-sectional, although their findings also support a role of childhood diet in the timing of puberty. To illustrate, one recent cross-sectional investigation among Chinese children found that boys with higher dietary quality scores had lower prevalence of spermatarche or voice break [8].

Since multiple types of foods and nutrients are hypothesized to affect the timing of puberty, the consideration of dietary patterns could enhance our understanding of the role of childhood diet on sexual maturation. A dietary pattern approach is useful because it takes into account the fact that certain dietary factors may be highly correlated; and these foods and/or nutrients in the foods could potentially interact to affect pubertal timing. Examining dietary patterns is also beneficial from an interpretation standpoint because foods are not consumed in isolation but rather as part of underlying patterns. Only one study has analyzed dietary patterns in relation to puberty [9]. In this cross-sectional analysis among 787 Korean adolescents, study authors identified 4 dietary patterns using principal components analysis and found that higher consumption of a “shellfish and processed meat” dietary pattern was associated with more advanced genital development in boys and breast development in girls. Nonetheless, the cross-sectional design was a major limitation given that diet may have changed as the result of pubertal initiation (i.e. reverse causation bias) [10].

Another nuance of the investigation on childhood diet and sexual maturation is the timing of dietary assessment. Some authors have posited that earlier childhood diet is more strongly related to timing of sexual maturation than childhood diet measured closer in time to the initiation of puberty [11]. This could potentially have to do with a developmental milestone that occurs in early childhood, the adiposity rebound (AR). The AR is a transition around 5–6 y of age when BMI begins to increase after reaching a nadir, and its timing correlates with timing of puberty. For example, one New Zealand study among girls reported a 0.9 year earlier menarche among those with early AR versus late AR [12]. Similarly, a retrospective study among Swedish males found that those in the earliest tertile of AR had a peak height velocity 7 months earlier on average than those in the late tertile [13]. Furthermore, there is accumulating evidence that timing of the adiposity rebound may be responsive to early dietary factors [14, 15]. For example, diet between the ages of 2.5 and 3.5 y has been related to the timing of the AR in Latino children living in the US [15].

The Early Life Exposures in Mexico to ENvironmental Toxicants (ELEMENT) study from Mexico City provided us with the opportunity to examine relationships between childhood dietary patterns and sexual maturation. As a part of this prospective study, diet was measured annually by food frequencies between the ages of 1 to 5 y. We focused on dietary patterns at 3 y of age, a potentially critical window of exposure before AR. Based on findings from a

subsample of children in the ELEMENT study [16], we hypothesized that we would identify at least two dietary patterns, one aligning with a prudent pattern (characterized by high fruit and vegetable intake, whole grains, and lean proteins), while another would more closely follow a transitioning pattern (characterized by more meat, processed foods, and energy-dense foods). We hypothesized the prudent pattern would be associated with later pubertal development while a dietary pattern more in line with a transitioning diet would be associated with earlier development. Our objectives were thus two-fold: to identify dietary patterns using principal components analysis and then to relate those patterns with sexual maturation markers, using a time-to-event model strategy.

2. METHODS AND MATERIALS

2.1 Study Population

The study population includes child participants from two of three sequentially-enrolled cohorts of the Early Life Exposure in Mexico to Environmental Toxicants (ELEMENT) study [17, 18]. Between 1997 and 2004, mother/child dyads were recruited from prenatal clinics of the Mexican Social Security Institute in Mexico City as a part of three separate studies, which we call cohorts 2A and 2B (usually considered as a single cohort due to similarities in research goals and study design), and 3. Cohorts 2A (birth years 1998 to 2001; n=212 child participants) and 2B (birth years 1999 to 2000; n=433 child participants) were observational studies designed to evaluate the role of lead exposure on maternal and child outcomes, while cohort 3 (birth years 2000 to 2001; n=393 child participants) was a randomized trial designed to test the reduction of plasma lead concentration through calcium supplementation. During early childhood visits in each of the studies, which occurred at 1 y of age and approximately every 6 months thereafter until 5 y of age, trained interviewers administered surveys to the mothers concerning the children's diets. Beginning in 2015, a subset of 550 participants from the original birth cohorts 2A, 2B, and 3 participated in a follow-up study on sexual maturation. These cohorts were selected for follow-up because the participants in them were of peri-pubertal ages. Our analytic sample comprises 496 children with diet information taken during the early childhood visits in addition to physician assessment of Tanner stages during the sexual maturation visit (Figure 1; sample sizes were slightly different for menarche and testicular volume outcomes). These children did not differ from the entire sample of 550 participants according to any of the maternal or sociodemographic characteristics listed in Table 2. The institutional review boards at the Mexico National Institute of Public Health and the University of Michigan approved research protocols, and informed consent was obtained for all participants.

2.2 Exposure: Dietary patterns

During the early childhood visits at ages 1 to 5 y, trained interviewers administered a 116-item semi-quantitative food frequency questionnaire (FFQ) to the mothers concerning usual consumption habits of their children. The questionnaire asked mothers to recall how often in the past 3 months her child typically consumed one serving of a standard portion size of each food item. Response options ranged from never to 6 times per day. We converted these raw response values (1–9) into servings/day (ranging from 0.03 to 6.0). In accordance with our research focus on the pre-adiposity rebound time frame, we used the dietary

information from the 3 y visit. In the instance a participant did not have a 3 y visit, we used data from the 4 y visit (n=38 participants) or the 5 y visit (n=1 participant).

2.2 Outcome: Sexual maturation

During the sexual maturation visit, trained physicians performed Tanner staging [19] (breast and pubic hair for girls, genitalia and pubic hair for boys) and assessed testicular volume using a Prader orchidometer (boys only). In addition, girls were asked whether they had started menstruating, and if so, the age that this first occurred. Our pubertal outcomes were classified as time to event outcomes, i.e. how long it took for the participant to attain a particular Tanner stage or to experience menarche. Most participants had already reached the first stages of puberty (Tanner stage >1 or testicular volume > 3 mL), so our outcome of interest was the time to reach the latter stages of puberty. For boys, this meant reaching Tanner pubic hair or genital stage 4, or testicular volume 15 mL. For girls, this meant reaching Tanner pubic hair or breast stage 4. Because we could not determine the exact date for entering particular Tanner stages, the observation time was a time interval, from birth to the follow up visit. For girls, we also evaluated whether or not they had experienced menarche.

2.3 Covariates

At the enrollment visit, mothers completed sociodemographic questionnaires which inquired about the mother's age, previous pregnancies, education, and marital status. Mother's age was categorized as <25 y, 25 to <30 y, 30 to <35 y, and ≥30 y. Number of previous pregnancies was defined as 0 or 1, 2, or ≥3. Education, considered a proxy for socioeconomic status, was categorized as <9 y, 9 to <12 y, 12 y, or ≥12 y. Marital status was defined as married/civil union, or single, separated, divorced, widowed. During the early postnatal visits, mothers were asked if they were still breastfeeding. At the first visit in which the mother reported she had stopped breastfeeding, she was asked to report the duration of breastfeeding in weeks. Duration of breastfeeding was categorized into quartiles. Original study cohort was categorized as cohort 2A, 2B, and 3. Weight measurements were taken using a digital scale (in kg, BAME Model 420; Catalogo Medico) and height measurements were taken using a calibrated stadiometer (in cm, BAME Model 420; Catalogo Medico) at the sexual maturation follow-up visits by trained research staff. Body mass index (BMI; kg/m²) was calculated and standardized into age and sex-specific z scores based on the World Health Organization reference [20].

2.4 Statistical Analysis

First, we used principal component analysis (PCA) to identify dietary patterns as previously described [16]. Briefly, we grouped similar food items into categories based on nutritional similarity and cultural relevance (see Supplemental Table S1), and computed total-energy adjusted food group intakes using the residual method [21]. We next performed principal component analysis of the food groups and rotated with orthogonal transformation to obtain uncorrelated factors. We determined the number of factors to retain based on visual inspection of the Scree plot, eigenvalues >1, and interpretability. We considered food groups with factor loadings >0.30 or <-0.30 to provide meaningful interpretation of the pattern. Dietary pattern scores for each participant were computed by multiplying the factor loadings

by the frequency of consumption in each group and then summing (provided in the PCA-R package). Dietary pattern factor scores were parameterized into tertiles for each dietary pattern, separately by sex.

To identify potential confounders, we examined means $\pm$ SD dietary pattern factor scores according to categories of maternal and study characteristics. For ordinal characteristics, we conducted a test for linear trend by including in the model a continuous variable representing the ordinal levels of the characteristic. For nominal characteristics, we utilized a type-III Wald test.

For the main analysis, we performed time-to-event analysis using interval-censored Cox regression models, performed separately for each outcome. In these models, the outcome was the time interval from the child's birthdate to the date of the follow-up visit in which the child had experienced the event (i.e. reaching stage 4 or menarche). If the event of interest had not occurred by the follow up visit, the observation was treated as "censored" in the statistical models. These censored observations still contributed person-time for the estimation of effect estimates. Indeed, the treatment of censored observations is an important feature of time to event analyses as simply excluding censored observations would result in biased estimates [22].

The exposures in these time-to-event models were tertiles of each dietary pattern factor scores. We computed hazard ratios (HR) and 95% confidence intervals (CI), comparing the second and third tertiles to the first tertile of dietary pattern factor scores. In adjusted analysis, we included continuous variables for maternal education, parity, duration of breastfeeding, and an indicator for cohort. Additional adjustment for calcium treatment status (since cohort 3 was a randomized trial) did not alter findings and was thus excluded for sake of parsimony. We also evaluated whether excluding participants who had diet measures at age 5 y but not at 3 y of age altered the estimates. Finally, we further adjusted for BMI-for-age z scores at sexual maturation visit to evaluate the extent to which the associations of interest were independent of excess adiposity. All statistical analyses were performed using R statistical software.

3. RESULTS

Forty-eight percent were male. The overall mean $\pm$ SD and median (range) age of the participants at the sexual maturation follow-up was 14.5 ± 2.1 y and 14.3 (9.8 to 16.5) (Supplemental Table S2; means $\pm$ SD ages for subcohorts 2A, 2B, and 3 were 16.5 ± 0.66 , 16.4 ± 0.90 , 12.6 ± 1.05 for girls and 16.6 ± 0.71 , 16.4 ± 0.83 , and 12.8 ± 1.02 for boys). The prevalence of overweight/obesity at follow-up was 37%. At this time, 61% of girls were in breast stage 4, 47% had pubic hair stage 4, and 88% had experienced menarche (refer to Supplemental Table S3 for concordance between the measures). The median age (IQR) at menarche was 12.0 (11.1 to 12.8) y. Among boys, 63% had a Tanner genital stage 4, 44% had a pubic hair stage 4, and 83% had testicular volume >15 mL at the follow-up visit (refer to Supplemental Table S3 for concordance between the measures).

We identified 5 dietary patterns that together explained 30.3% of the variance (Table 1). The first, called *vegetables and lean proteins* was characterized by a high intake of vegetables, legumes, chicken, and organ meat. The *maize products and sugar-sweetened beverage (SSB)* pattern included a high intake of SSB, maize and chili products, and low intake of chicken and milk. The *processed meats and refined grain* pattern was marked by high intake of refined grain, processed meats and eggs, and low intake of whole grains and milk. The *fruit and yogurt* pattern had a high intake of fruit, natural juice, and yogurt, and low intake of fried Mexican foods and chips. Finally, the *whole grain and fat* pattern was characterized by high intake of pureed vegetable soup, fish, avocado, whole grains, sweet condiments, and unsaturated spreads.

Several maternal/study characteristics were correlated with dietary pattern scores (Table 2). Mother's number of previous pregnancies was positively associated with the *maize products and SSB* pattern scores and inversely associated with *fruit and yogurt* pattern scores. Mother's education was inversely associated with the *maize products and SSB* pattern scores; in contrast, mother's education was positively associated with *fruit and yogurt* pattern scores as well as the *whole grain and fat* pattern scores. Duration of breastfeeding was inversely associated with the *maize products and SSB* pattern, although it was of marginal statistical significance. Study cohort was associated with each dietary pattern, albeit in different directions. Children in cohort 3 (started in 2001) had higher *maize products and SSB* pattern scores than children in cohorts 2A and 2B, but lower scores in all other patterns.

Among girls, there was an inverse linear association between the *vegetables and lean proteins* pattern score and breast stage 4 (Table 3). After adjustment for mother's previous pregnancies, maternal education, duration of breastfeeding and cohort, those in the highest tertile of scores had a 35% lower probability of having reached breast stage 4 at the follow-up visit (95% CI 3% to 67%; P, trend=0.03). In contrast, there was a marginally statistically significant positive association between the *whole grain and fat* pattern and having reached breast stage 4 (Hazard ratio for Tertile 3 compared to Tertile 1: 1.65 with 95% CI 0.85 to 2.45; P, trend=0.07). This relation was slightly attenuated after adjustment for potential confounders. There were no dose-response relations between any of the dietary patterns and pubic hair or menarche.

Among boys, there was a positive association between the *processed meats and refined grains* pattern scores and testicular volume 15 mL (Table 4). After adjustment for mother's previous pregnancies, maternal education, duration of breastfeeding and cohort, those in the highest tertile of scores had a 3.58 times higher probability of having reached testicular volume 15 than boys in the lowest tertile of scores (95% CI 0.62 to 6.53; P, trend=0.02). In crude analysis, there was no association between the *maize products and SSB* pattern scores and testicular volume. However, after adjustment for potential confounders, there was a marginally statistically significant inverse association between the *maize products and SSB pattern* and testicular volume. Boys in the highest tertile of scores had 0.61 times the probability of having reached testicular volume 15 than boys in the lowest tertile (95% CI 0.20 to 1.02; P, trend=0.07). We did not find any associations between the dietary pattern and genital stage or pubic hair stage. In sensitivity analyses, excluding participants without

diet assessments at 3 y of age did not substantially alter findings. Similarly, including BMI-for-age z scores in the fully adjusted models did not attenuate the estimates.

4. DISCUSSION

In this prospective study of Mexican children, we identified 5 major dietary patterns during the pre-adiposity rebound time period: *vegetables and lean proteins*, *maize products and SSB*, *processed meats and refined grain*, *fruit and yogurt*, and *whole grain and fat*. Our findings were mostly consistent with our hypotheses, as two of the identified dietary patterns were associated with pubertal measures in the expected direction. Specifically, we found that a higher *vegetable and lean proteins* pattern, which aligns with the prudent dietary pattern identified by Perng et al.[16], was related to delayed breast development among girls. In contrast, a higher *processed meat and refined grain* pattern, which is more in line with a transitioning dietary pattern, was related to advanced testicular development among boys. The patterns *maize products and SSB*, *fruit and yogurt*, and *whole grain and fat* were not statistically significantly associated with any of the sexual maturation markers.

The finding that a dietary pattern rich in vegetables, legumes, and lean meats was associated with delays in breast development is consistent with the literature examining individual foods and/or macronutrients in relation to sexual maturation and growth. For example, a study among 112 German children that higher vegetable protein intake at 3–4 y and 5–6 y of age corresponded with later pubertal growth spurt, peak height velocity, and age at menarche/voice break [6]. Another study among 67 US girls showed that higher consumption of vegetable protein at age 3 to 5 y was related to a later age at menarche [23]. Studies examining intake of particular components found in vegetable sources including fiber[24] and phytoestrogens- isoflavones[7] and enterolactone [25]- have also supported delays in the age at menarche or breast development among girls. Another potential pathway, which would have different public health implications, is through cadmium exposure. Vegetables are a source of dietary cadmium in this study population [26], with 33% of the dietary cadmium exposure was estimated to come from vegetables. In some studies, cadmium exposure has been related to delays in sexual maturation [27].

The finding that the *processed meat and refined grain* pattern was associated with earlier testicular development in boys has not been previously described in prospective studies. The association could potentially be explained by the processed meat rather than the high consumption of refined grain and eggs. For example, the cross-sectional Korean study found that adherence to the dietary pattern “shellfish and processed meat” was related to earlier self-reported genital development among boys [9]. In addition, a Chinese study found that boys who had already experienced spermatarche consumed more red meat and dairy products than those who did not yet have it [28]. It is also possible that the findings could be explained by the animal protein supplied by the processed meat as well as the eggs. In support, a German study including boys found that higher consumption of animal protein was associated with earlier pubertal growth spurt and earlier voice break [6]. In contrast to the findings on meat and animal protein, studies examining carbohydrate and/or fiber intake have not been associated with pubertal measures (albeit most of the studies were among girls) [3, 7].

Of note, there were also a few non-significant associations worth mentioning. Among boys, there was a suggestive association between the *maize products and SSB* pattern and later testicular development. This pattern was characterized by high consumption of maize-based products such as tortillas and sugar-sweetened beverages and lower consumption of milk. This relationship could be driven by the fact that this dietary pattern was inversely related to milk consumption; which has been associated with advanced sexual maturation in boys [28]. Another potential explanation is that high intake of sugar-sweetened beverages was related to weight gain [29] which in turn led to delayed sexual maturation among boys with the highest weight gain [30].

Among girls, higher *whole grain and fat* pattern scores were related to earlier breast development. This pattern was composed of a few types of fat that have been implicated in timing of sexual maturation, including polyunsaturated fat (e.g. from fatty fish)[11], and monounsaturated fat (e.g. from avocados) [31]. Nonetheless, even within the specific types of dietary fat, the directions of these associations are quite inconsistent and challenging to interpret [3]. This pattern also includes higher consumption frequency of high-fat dairy, and dairy has been related to earlier sexual maturation among girls [32].

It is of note that the associations among girls were only evident for breast development and not for pubic hair or menarche, whereas for boys the relations were only evident for testicular volume and not for genital or pubic hair Tanner stages. It is unclear if this has to do with measurement issues or if there is a biological explanation. For girls, it is possible that Tanner breast staging provided a more accurate pubertal staging measure than age at menarche because it was physician-assessed. Furthermore, the girls in this study may have been outside the ideal age range to accurately recall their menarcheal age. On the other hand, breast stages can be overestimated among heavier girls, meaning that higher breast stages may be a stronger proxy for adiposity than pubertal progression. Nonetheless, this explanation is not supported by the fact that adjusting for BMI-for-age z scores did not alter the estimates. Among boys, although all measures were physician-assessed, testicular volume assessment is the most objective and considered the gold standard for detecting the initial stages of puberty [33]. Aside from measurement error, it is possible that the various markers of puberty could be influenced differentially by the same dietary exposures, since the hormonal pathways responsible for their development are interrelated but not identical [34].

The study has several strengths. First is the prospective nature of the data, which allows us to temporally separate the consumption patterns from the occurrence of puberty. Another is the use of physician-assessed pubertal measures for the Tanner staging and testicular volume. Principal Components Analysis is often used to identify dietary patterns in nutrition research [16, 35], although this is one of the first studies to consider the role of childhood dietary patterns on timing of sexual maturation. The study population was also a strength because very few studies have characterized dietary patterns in Mexican populations, and relatively little is known about dietary determinants of puberty in Central America in general. Further, the FFQ was developed and validated in the Mexican population from multiple 24-hour recalls [36]. There are also limitations. This was a relatively small sample population as our sample size was based on data availability rather than power calculations; thus, we may have

been underpowered to detect associations. We did not have consistent measurements of BMI during the early childhood time period, which would have allowed us to examine whether the timing of adiposity rebound mediated the association. In addition, because a majority of the adolescents were not assessed for sexual maturation markers during early adolescence, we were not able to evaluate whether early childhood dietary pattern scores were related differently to timing of the initiation of puberty versus reaching the latter stages of puberty. Further, dietary data collected from food frequencies is subject to measurement error and potentially recall bias, if the reported diets differed systematically according to the pubertal outcomes. Finally, the FFQ was validated in an adult rather than pediatric population.

In summary, we found that higher scores in the *vegetables and lean proteins* and *processed meat and refined grain* dietary patterns prior to the adiposity rebound were related to timing of sexual maturation in a sex-specific manner among Mexican youth. Specifically, a higher *vegetables and lean proteins* pattern score was associated with delays in breast development among girls while higher *processed meats and refined grain* pattern scores correlated with advanced testicular development among boys. These findings are generally in line with current nutritional guidelines that favor diets high in fruits, vegetables, and lean proteins over highly processed and energy-dense diets. Nonetheless, the potential link between higher early childhood consumption of red/processed meat and earlier testicular development among boys warrants replication in other populations. In addition, further investigation into the particular components that could be driving associations with vegetables and processed meat would aid in the formation of nutritional and/or policy recommendations concerning these foods.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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LIST OF ABBREVIATIONS

Y	years
CI	confidence interval
AR	adiposity rebound
ELEMENT	Early Life Exposure in Mexico to ENvironmental Toxicants
FFQ	food frequency questionnaire
PCA	principal components analysis
HR	hazard ratio

SD	standard deviation
IQR	interquartile range
SSB	sugar-sweetened beverages

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Figure 1.
Selection of subjects for the study

Table 1.

Factor loadings for 5 dietary patterns in Mexican children at 3 y of age

	Factor 1 <i>Vegetables and lean protein</i>	Factor 2 <i>Maize products and SSB</i>	Factor 3 <i>Processed meats and refined grains</i>	Factor 4 <i>Fruit and yogurt</i>	Factor 5 <i>Whole grain and fat</i>
Food groups					
Other vegetables	0.72[/]	0.02	0.04	0.00	0.06
Dark yellow vegetables	0.70	−0.06	0.01	0.01	0.11
Potato	0.61	−0.04	0.19	−0.23	0.04
Green leafy vegetables	0.58	0.07	−0.22	0.00	0.23
Legumes	0.48	0.26	0.07	0.12	0.17
Chicken	0.35	−0.30	0.25	0.11	−0.14
Organ meat	0.31	0.02	−0.01	0.25	−0.01
Tomato	0.30	0.10	0.02	0.22	0.12
Milk	−0.19	−0.63	−0.38	−0.23	−0.19
Chili products	0.08	0.54	0.15	−0.19	0.00
Maize-based products	0.07	0.51	−0.23	−0.07	−0.25
Sugar-sweetened bev.	−0.11	0.49	−0.03	−0.05	−0.06
Refined grains	0.09	0.14	0.55	0.20	−0.19
Processed meats	0.05	−0.04	0.53	−0.08	0.17
Eggs	0.01	−0.09	0.48	0.05	0.08
Whole grains	0.13	0.05	−0.30	0.19	0.43
Low-fat dairy	−0.08	−0.13	0.06	0.51	0.05
Fruit	0.26	0.29	0.05	0.47	0.18
Natural juices	0.13	0.03	0.06	0.45	0.16
Fried Mexican foods	−0.02	0.28	0.17	−0.35	0.18
Chips	−0.02	0.15	0.27	−0.32	−0.14
Unsaturated spreads	−0.15	−0.03	0.29	−0.10	0.53
High-fat dairy	0.07	−0.10	0.06	−0.13	0.50
Fish	0.24	−0.01	−0.02	0.02	0.48
Pureed vegetable soups	0.24	−0.08	−0.03	0.12	0.42
Sweet condiments	−0.01	0.03	0.10	0.20	0.37
Avocado	0.20	0.15	−0.13	0.00	0.33
Beef and pork	0.17	0.04	0.25	−0.28	0.15
Sweets and desserts	0.02	0.10	0.29	0.24	−0.05
Vegetable oil	0.00	−0.02	0.02	0.16	−0.02
Atole	0.09	0.24	−0.19	0.05	0.01
% Variance explained	8.5%	5.9%	5.5%	5.5%	4.9%

[/] Factor loading

Table 2.

Associations of maternal and sociodemographic characteristics with dietary pattern factor scores at 3 y of age among 496 Mexican children

Maternal sociodemographic characteristics		N	Dietary pattern factor score, means ± SD			
			Vegetables and lean protein	Maize products andSSB ²	Processed meats and refined grains	Fruit and yogurt
Mother's age at baseline, y						
<25	201	-0.20 ± 0.93 ¹	-0.28 ± 0.81	-0.26 ± 1.06	-0.25 ± 1.01	-0.22 ± 0.99
25 to <30	168	-0.09 ± 0.92	-0.49 ± 0.72	-0.28 ± 0.85	-0.21 ± 0.81	-0.19 ± 0.77
30 to <35	77	-0.02 ± 1.02	-0.37 ± 0.69	-0.26 ± 0.92	-0.07 ± 1.02	0.00 ± 0.90
35	48	-0.15 ± 0.88	-0.35 ± 0.77	-0.57 ± 0.83	-0.08 ± 0.76	0.00 ± 0.90
P, trend ³		0.30	0.35	0.13	0.12	0.20
Mother's number of previous pregnancies						
0 or 1	191	-0.05 ± 0.93	-0.46 ± 0.73	-0.32 ± 0.95	-0.14 ± 0.92	-0.14 ± 0.89
2	175	-0.24 ± 0.91	-0.43 ± 0.78	-0.27 ± 0.98	-0.09 ± 0.90	-0.11 ± 0.93
3	129	-0.08 ± 0.98	-0.16 ± 0.75	-0.28 ± 0.93	-0.40 ± 0.95	-0.13 ± 0.92
P, trend		0.61	0.001	0.70	0.03	0.87
Mother's education, y						
Did not complete secondary (<9)	57	-0.18 ± 0.88	-0.14 ± 0.81	-0.11 ± 0.91	-0.28 ± 0.89	-0.31 ± 0.93
Completed some high school (9 to <12)	197	-0.10 ± 0.99	-0.34 ± 0.72	-0.30 ± 0.99	-0.29 ± 0.89	-0.20 ± 0.92
Completed high school (12)	174	-0.15 ± 0.90	-0.39 ± 0.81	-0.36 ± 0.98	-0.08 ± 0.93	-0.07 ± 0.90
Higher education (>12)	67	-0.11 ± 0.95	-0.64 ± 0.65	-0.26 ± 0.80	-0.09 ± 0.94	0.09 ± 0.86
P, trend		0.88	<0.0001	0.37	0.04	0.005
Mother's marital status, %						
Married or civil union	443	-0.15 ± 0.93	-0.37 ± 0.77	-0.28 ± 0.97	-0.18 ± 0.93	-0.11 ± 0.92
Single, separated, divorced, or widowed	53	0.08 ± 0.99	-0.44 ± 0.70	-0.42 ± 0.86	-0.28 ± 0.89	-0.33 ± 0.84
P value		0.09	0.52	0.33	0.45	0.09
Duration of breastfeeding, quartiles						
Q1, 0 to 3 weeks	127	-0.20 ± 0.95	-0.43 ± 0.74	-0.34 ± 0.88	-0.24 ± 0.96	-0.24 ± 0.99
Q2, 4 to 7 weeks	142	-0.06 ± 0.97	-0.43 ± 0.78	-0.33 ± 0.88	-0.16 ± 0.84	0.05 ± 0.86
Q3, 8 to 12 weeks	135	-0.11 ± 0.95	-0.34 ± 0.72	-0.37 ± 1.05	-0.22 ± 0.89	-0.19 ± 0.86
Q4, 13 to 34 weeks	91	-0.16 ± 0.85	-0.25 ± 0.81	-0.07 ± 1.00	-0.12 ± 1.04	-0.17 ± 0.92
P, trend		0.80	0.07	0.10	0.50	0.96

Maternal sociodemographic characteristics		Dietary pattern factor score, means $\pm$ SD				
		N	Vegetables and lean protein	Maize products andSSB ²	Processed meats and refined grains	Fruit and yogurt
Initial study cohort the child was enrolled						
2A (started in 1997)	129	0.04 $\pm$ 1.02	-0.52 $\pm$ 0.76	0.12 $\pm$ 0.92	0.00 $\pm$ 1.00	-0.06 $\pm$ 1.00
2B (started in 1999)	115	0.06 $\pm$ 1.01	-0.51 $\pm$ 0.79	0.01 $\pm$ 1.03	0.12 $\pm$ 1.00	0.03 $\pm$ 0.90
3 (started in 2001)	252	-0.30 $\pm$ 0.82	-0.24 $\pm$ 0.72	-0.65 $\pm$ 0.80	-0.43 $\pm$ 0.78	-0.24 $\pm$ 0.86
P value		0.0001	0.0002	<0.0001	<0.0001	.02

¹ Means $\pm$ SD

² SSB= sugar sweetened beverages

³ For ordinal characteristics, we conducted a test for linear trend by running a linear regression model with dietary pattern score as the outcome and a continuous variable representing the ordinal levels of the characteristic. For nominal characteristics, we utilized a type-III Wald test.

Table 3.

Prospective associations of dietary patterns at 3 y of age with sexual maturation markers among girls in Mexico City

Dietary patterns	Breast stage 4 n=258		Pubic hair 4 n=258		Age at menarche n=263	
	Unadjusted HR (95% CI) ¹	Adjusted HR (95% CI) ²	Unadjusted HR (95% CI) ¹	Adjusted HR (95% CI) ²	Unadjusted HR (95% CI) ¹	Adjusted HR (95% CI) ²
<i>Vegetables and lean proteins</i>						
Tertile 1	1.00	1.00	1.00	1.00	1.00	1.00
Tertile 2	0.77(0.39,1.15)	0.79(0.38,1.21)	1.37(0.69,2.05)	1.41(0.70,2.12)	0.69(0.31,1.07)	0.67(0.30,1.04)
Tertile 3	0.65(0.33,0.97)	0.60(0.28,0.92)	1.69(0.86,2.51)	1.57(0.79,2.36)	0.74(0.32,1.15)	0.72(0.31,1.14)
P for trend ³	0.05	0.03	0.08	0.11	0.21	0.21
<i>Maize products and SSB</i>						
Tertile 1	1.00	1.00	1.00	1.00	1.00	1.00
Tertile 2	0.82(0.43,1.21)	0.85(0.41,1.28)	0.60(0.31,0.89)	0.63(0.31,0.94)	0.37(0.14,0.60)	0.36(0.14,0.59)
Tertile 3	0.98(0.51,1.45)	1.00(0.48,1.53)	0.95(0.50,1.40)	1.02(0.50,1.53)	0.82(0.33,1.31)	0.81(0.30,1.32)
P for trend	0.94	0.99	0.66	0.97	0.89	0.82
<i>Processed meats and refined grains</i>						
Tertile 1	1.00	1.00	1.00	1.00	1.00	1.00
Tertile 2	0.75(0.36,1.15)	0.79(0.36,1.22)	1.28(0.59,1.97)	1.31(0.58,2.03)	0.75(0.38,1.12)	0.73(0.35,1.11)
Tertile 3	0.79(0.39,1.19)	0.86(0.37,1.35)	0.98(0.46,1.49)	1.07(0.48,1.66)	0.84(0.39,1.28)	0.78(0.25,1.31)
P for trend	0.40	0.63	0.67	0.93	0.21	0.25
<i>Fruit and yogurt</i>						
Tertile 1	1.00	1.00	1.00	1.00	1.00	1.00
Tertile 2	1.07(0.56,1.59)	1.08(0.54,1.61)	0.84(0.41,1.26)	0.75(0.36,1.14)	0.83(0.42,1.25)	0.79(0.38,1.20)
Tertile 3	1.43(0.75,2.11)	1.37(0.65,2.08)	1.09(0.57,1.61)	0.98(0.49,1.48)	1.08(0.47,1.68)	1.02(0.38,1.66)
P for trend	0.18	0.27	0.63	0.95	0.91	0.87
<i>Whole grain and fat</i>						
Tertile 1	1.00	1.00	1.00	1.00	1.00	1.00
Tertile 2	1.58(0.80,2.37)	1.54(0.73,2.34)	1.44(0.72,2.16)	1.40(0.69,2.12)	1.30(0.60,2.01)	1.41(0.63,2.19)
Tertile 3	1.65(0.85,2.45)	1.68(0.82,2.34)	1.48(0.77,2.19)	1.41(0.72,2.09)	0.79(0.34,1.23)	0.78(0.33,1.24)
P for trend	0.07	0.08	0.14	0.22	0.43	0.43

From interval-censored Cox proportional hazards models where outcome is occurrence of each pubertal marker by the time of the interview date and exposures are indicator variables for dietary pattern score tertiles. HR= hazard ratio

From models that included adjustment for maternal education (years), parity, and duration of breastfeeding (weeks) as continuous variables and adjustment for cohort as an indicator variable

The P for trend was obtained by including in the Cox regression a continuous variable representing the tertile of the dietary pattern

Table 4.

Prospective associations of dietary patterns at 3 y of age with sexual maturation markers among boys in Mexico City

Dietary patterns	Genital stage 4 n=238			Pubic hair 4 n=238			Testicular volume >15 mL n=230		
	Unadjusted HR (95% CI) ¹	Adjusted HR (95% CI) ²	Unadjusted HR (95% CI) ¹	Adjusted HR (95% CI) ²	Unadjusted HR (95% CI) ¹	Adjusted HR (95% CI) ²	Unadjusted HR (95% CI) ¹	Adjusted HR (95% CI) ²	
Vegetables and lean proteins									
Tertile 1	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	
Tertile 2	1.02(0.54,1.50)	1.15(0.58,1.72)	0.82(0.37, 1.27)	0.85(0.38,1.32)	0.68(0.30,1.06)	0.73(0.30,1.16)			
Tertile 3	0.84(0.44,1.22)	0.84(0.43,1.26)	0.85(0.41, 1.30)	0.82(0.38,1.25)	0.75(0.30,1.20)	0.76(0.26,1.25)			
P for trend ³	0.40	0.47	0.57	0.43	0.29	0.33			
Maize products and SSB									
Tertile 1	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	
Tertile 2	0.75(0.41,1.09)	0.74(0.38,1.10)	0.85(0.42, 1.28)	0.84(0.41,1.27)	1.02(0.40,1.64)	0.97(0.32,1.62)			
Tertile 3	0.84(0.45,1.22)	0.88(0.44,1.32)	1.01(0.47, 1.56)	1.13(0.50,1.76)	0.71(0.30,1.11)	0.61(0.20,1.02)			
P for trend	0.35	0.53	0.96	0.82	0.13	0.07			
Processed meats and refined grain									
Tertile 1	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	
Tertile 2	1.01(0.53,1.48)	0.96(0.48,1.44)	0.95(0.40, 1.50)	0.86(0.35,1.37)	1.39(0.65,2.13)	1.48(0.64,2.31)			
Tertile 3	1.07(0.59,1.56)	1.04(0.51,1.56)	0.98(0.46, 1.51)	0.99(0.44,1.54)	3.49(0.90,6.07)	3.58(0.62,6.53)			
P for trend	0.65	0.95	0.97	0.96	0.02	0.02			
Fruit and yogurt									
Tertile 1	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	
Tertile 2	1.35(0.71,2.00)	1.29(0.64,1.94)	0.86(0.39, 1.33)	0.85(0.37,1.32)	0.75(0.33, 1.16)	0.64(0.27,1.02)			
Tertile 3	0.87(0.47,1.28)	0.80(0.40,1.20)	0.71(0.33, 1.09)	0.71(0.32,1.09)	0.82(0.33, 1.30)	0.72(0.26,1.18)			
P for trend	0.52	0.29	0.17	0.17	0.43	0.14			
Whole grain and fat									
Tertile 1	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	
Tertile 2	1.99(0.99, 3.00)	1.89(0.89,2.90)	1.20(0.53, 1.87)	1.19(0.50,1.87)	1.30(0.57,2.03)	1.32(0.56,2.07)			
Tertile 3	1.10(0.59,1.61)	0.99(0.50,1.49)	0.75(0.35, 1.15)	0.70(0.31,1.09)	0.75(0.30,1.19)	0.71(0.25,1.18)			
P for trend	0.64	0.99	0.22	0.14	0.46	0.45			

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From interval-censored Cox proportional hazards models where outcome is occurrence of each pubertal marker by the time of the interview date and exposures are indicator variables for dietary pattern score tertiles. HR=hazard ratio
2 From models that included adjustment for maternal education (years), parity, and duration of breastfeeding (weeks) as continuous variables and adjustment for cohort as an indicator variable
3 The P for trend was obtained by including in the Cox regression a continuous variable representing the tertile of the dietary pattern