Maternal diet in pregnancy is associated with differences in child body mass index trajectories from birth to adolescence

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ABSTRACT

Background: Nutrition in pregnancy and accelerated childhood growth are important predictors of obesity risk. Yet, it is unknown which dietary patterns in pregnancy are associated with accelerated growth and whether there are specific periods from birth to adolescence that are most sensitive to these associations.

Objectives: To examine the extent to which 3 dietary indices in pregnancy [Dietary Inflammatory Index (DII), Alternate Healthy Eating Index for Pregnancy (AHEI-P), and Mediterranean Diet Score (MDS)] are associated with child BMI z-score (BMI-z) trajectories from birth to adolescence.

Methods: We examined 1459 mother-child dyads from Project Viva that had FFQ data in pregnancy and ≥3 child BMI-z measurements between birth and adolescence. We used linear spline mixed-effects models to examine whether BMI-z growth rates and BMI z-scores differed by quartile of each dietary index from birth to 1 mo, 1–6 mo, 6 mo to 3 y, 3–10 y, and >10 y.

Results: The means ± SDs for DII (range, −9 to +8 units), AHEI-P (range, 0–90 points), and MDS (range, 0–9 points) were −2.6 ± 1.4 units, 61 ± 10 points, and 4.6 ± 2.0 points, respectively. In adjusted models, children of women in the highest (vs. lowest) DII quartile had higher BMI-z growth rates between 3–10 y (β, 0.03 SD units/y; 95% CI: 0.00–0.06) and higher BMI z-scores from 7 y through 10 y. Children of women with low adherence to a Mediterranean diet had higher BMI z-scores from 3 y through 15 y. Associations of AHEI-P with growth rates and BMI z-scores from birth through adolescence were null.

Conclusions: A higher DII and a lower MDS in pregnancy, but not AHEI-P results, are associated with higher BMI-z trajectories during distinct growth periods from birth through adolescence. Identifying the specific dietary patterns in pregnancy associated with rapid weight gain in children could inform strategies to reduce child obesity. *Am J Clin Nutr* 2021;00:1–10.

Keywords: dietary pattern, Mediterranean diet, dietary inflammatory, index, AHEI, growth, body, 27 mass index, child BMI, trajectories, early childhood, adolescence

Introduction

The prevalence of obesity among children and adolescents in the United States, which is 18.5% (National Health and Nutrition Examination Survey 2015–2016), remains higher than the Healthy People 2020 goal of 14.5% (1, 2). Childhood overweight continues to be a major public health concern, as children with overweight and obesity are more likely to have lower self-esteem and poorer school achievement, to continue to suffer from overweight (OW) and obesity (OB) as adults, and to develop type 2 diabetes and metabolic syndrome (3–5). Accelerated growth, specifically in early childhood, is a risk factor for later OW and OB (6–8). Therefore, identifying early modifiable determinants of growth trajectories in children is crucial to developing preventive strategies that may mitigate these adverse health outcomes.

Few longitudinal studies have examined relationships between maternal dietary patterns in pregnancy and child growth trajectories through adolescence. We and others have published data on the associations of maternal diet with offspring growth, OB, and cardiometabolic health. However, these studies have restricted outcome measures to 1 point in time (9–13) or to a short follow-up period (14–17). Data from these studies do not allow us to consider dynamic growth changes that occur across childhood

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Abbreviations used: AHEI-P, Alternate Healthy Eating Index for Pregnancy; BMI-z, BMI z-score; DII, Dietary Inflammatory Index; GA, gestational age; MDS, Mediterranean Diet Score; MED, Mediterranean Diet; OB, obesity; OW, overweight; ppBMI, pre-pregnancy BMI.

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and adolescence, and might lead to an incomplete understanding of the associations between diet during pregnancy and childhood growth.

Another limitation of existing studies is the heterogeneity in methodologies used to classify dietary patterns. Several studies have used a posteriori–defined dietary patterns derived using principal component analysis (11), latent class analysis (14), or reduced rank regressions (17, 18). These dietary patterns are derived specifically for the population being studied and are therefore not generalizable across populations. Given their known relationship with important health outcomes and the availability of already established nutritional guidelines which could inform preventive strategies, further research is needed to understand the role of specific a priori dietary indices applied to diets consumed in pregnancy on offspring growth trajectories.

To address these knowledge gaps, we examined the associations of 3 dietary indices based on pregnancy diets [Dietary Inflammatory Index (DII), Mediterranean Diet Score (MDS), and Alternate Healthy Eating Index-for Pregnancy (AHEI-P)] with offspring growth trajectories during specific periods from birth through adolescence. We hypothesized that a higher DII (indicating a pro-inflammatory diet), lower MDS, and lower AHEI-P would be associated with a *1*) more rapid BMI z-score (BMI-z) gain; and *2*) higher BMI-z. We also hypothesized that these associations would be stronger during childhood and adolescence than in infancy, given the role of an obesogenic environment in the programming of child obesity (19).

Methods

Study design and participants

Children were participants in Project Viva, an ongoing, prospective cohort study of prenatal and perinatal influences on maternal and child health (20). From April 1999 to July 2002, Project Viva enrolled pregnant women at their initial prenatal visits at 8 obstetrical offices of Atrius Harvard Vanguard Medical Associates, a multispecialty group practice in eastern Massachusetts. Study procedures for this cohort have been described previously (20). Of 2128 live, singleton births, we included 1459 (68.6%) mother-child dyads with available FFQ data in pregnancy and ≥3 child BMI-z measurements from birth to adolescence (median, 3 y; range, 0–18.3 y) without missing covariate data (**Figure 1**).

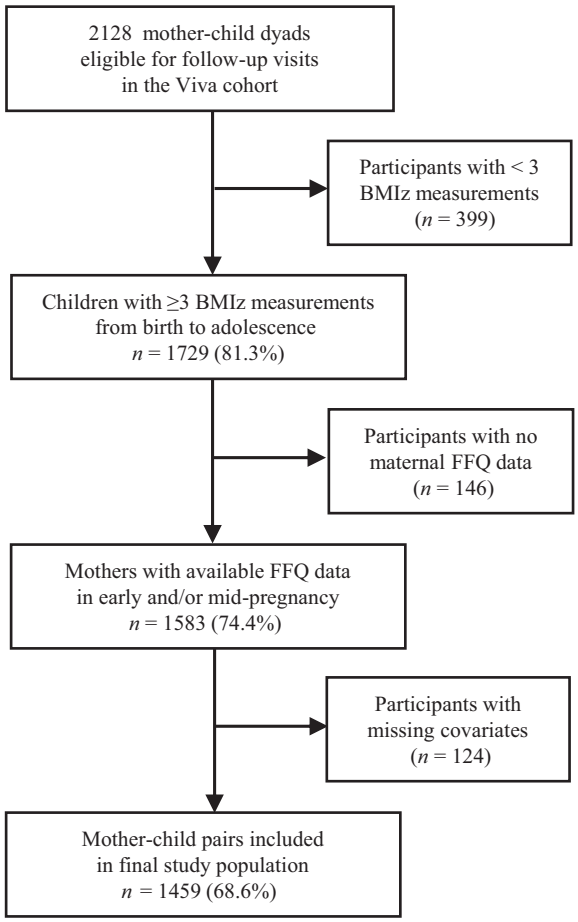

FIGURE 1 Flow diagram of participants (n = 1459) for this analysis from the Project Viva, a cohort recruited from the Boston, Massachusetts, area in 1999–2002. Abbreviation: BMI-z, BMI z-score.

Dietary assessments in pregnancy

Mothers completed semi-quantitative FFQs at the first (median 9.9 weeks of gestation) and second (median 27.9 weeks of gestation) in-person study visits. The first FFQ assessed diet intake since the last menstrual period and the second FFQ assessed intake during the previous 3 mo. The 166-item FFQ was slightly modified for use in pregnancy from the extensively validated Willett FFQ used in the Nurses' Health Study and other large cohort studies, and has been validated in pregnancy (21, 22). We used the Harvard nutrient composition database, which is based primarily on USDA publications, to obtain estimates of nutrients (23).

Dietary Inflammatory Index.

Given the role of dietary and systemic inflammation in early life on later obesity outcomes in children (24, 25), we explored the role of dietary inflammation in pregnancy on offspring growth trajectories. The DII is a validated literature-based and population-based measure that has been developed to characterize and quantify the cumulative inflammatory potential of an individual's diet (26). The DII is not a dietary pattern, but an assessment of the pro- or anti-inflammatory potential of any diet. A detailed procedure for DII estimations in this cohort has been described previously (27). The DII includes various foods and nutrients, such as SFA, MUFA, omega (n)-3, and n-6 PUFA, and several antioxidants, such as vitamin B6, vitamin B12, folic acid, and selenium. The DII ranges from −9 to +8 units, and a higher (i.e., more positive) DII score indicates a more proinflammatory diet, whereas a more negative score represents a more anti-inflammatory diet.

Mediterranean Diet Score.

To evaluate adherence to the Mediterranean diet (MED) during pregnancy, we used the MDS, modified from a large cohort study in adults (28). Women whose intake was above a prespecified threshold, based on recommendations for pregnant women (29),

for each of the components presumed to fit the MED (vegetables, fruits, fish, dairy products, legumes, whole-grain products, nuts, and MUFA) and below a threshold for components not fitting the MED (red and processed meat) were assigned a value of 1. Otherwise, they were assigned a value of 0. After summing up scores of each component, the final score range was 0–9, with higher scores indicating closer adherence to a Mediterranean-type diet.

Alternate Healthy Eating Index for Pregnancy.

As previously detailed (22), we used the AHEI-P to measure diet quality on a 90-point scale based on recommendations from the USDA's Dietary Guidelines for Americans, with each of the following 9 components contributing a maximum of 10 points if participants met the recommendations fully: vegetables, fruit, ratio of white to red meat, fiber, trans fat, ratio of PUFA to SFA, and folate, calcium, and iron from foods. We excluded the nuts component because, at that time, some women avoided nuts during pregnancy out of concern for allergen sensitization, and we added 3 components to reflect food-based intake of nutrients important during pregnancy: folate, iron, and calcium. These modifications of the AHEI resulted in the AHEI-P. We summed all component scores to obtain a total diet quality score ranging from 0 (worst) to 90 (best). We excluded the alcohol component from the original AHEI and the MDS because it is not recommended during pregnancy.

Child anthropometrics

During research visits in infancy (median, 0.5 y; range, 0.4–0.8 y), early childhood (median, 3.2 y; range, 2.8–6.2 y), mid-childhood (median, 7.7 y; range, 6.6–10.7 y), and early adolescence (median, 12.9 y; range, 11.9–16.6 y), trained research assistants measured weight and length/height using standardized protocols (20). We also obtained additional anthropometric data from medical records from well-child visits. Because pediatric clinics used the paper-and-pencil method to measure length from birth to 2 y, we applied a corrective algorithm to the clinical lengths to account for the systematic overestimation of lengths resulting from this technique (30). Using both research and clinical measures, we calculated BMIs (in kg/m^2) and derived age- and sex-specific z-scores (BMI-z) using the WHO child growth standards (31).

Covariates

Maternal variables.

Mothers reported their age, height, pre-pregnancy weight, education, race/ethnicity, parity, household income, and smoking status via interviews and questionnaires at enrollment. For the race/ethnicity variable, research assistants asked mothers the question, "which of the following best describes your race or ethnicity?" Mothers had a choice of 1 or more of the following mutually exclusive racial/ethnic groups: Hispanic or Latina; White or Caucasian; Black or African American; Asian or Pacific Islander; American Indian or Alaskan Native; and other (please specify). For the participants who chose "other" race/ethnicity, we compared the specified responses to the US census definition for the other 5 races and ethnicities and reclassified them where appropriate. If a participant chose more than 1 racial/ethnic group, we classified them in the "other" category. We calculated pre-pregnancy BMI (ppBMI) and categorized it per WHO guidelines as follows: underweight (BMI $<$ 18.5), normal (BMI 18.5 to $<$25), OW (25 to $<$30), and OB ($\geq$30).

Child variables.

Research assistants abstracted from delivery medical records information on perinatal characteristics, including child's sex, birth weight, and delivery date. We calculated the gestational age (GA) at birth based on established methods (32). We determined sex-specific birthweight-for-GA z-scores using US reference data (33).

Ethics approval

Mothers provided written informed consent at enrollment and at each postnatal follow-up visit, and children provided assent at the mid-childhood and adolescent visits. The Institutional Review Boards of Harvard Pilgrim Health Care Institute and the Brigham and Women's Hospital approved the project, in line with ethical standards established by the Declaration of Helsinki.

Statistical analysis

All statistical analyses were performed using Stata/SE version 15.1 (StataCorp.). We used linear spline mixed-effects models to examine the associations of dietary quality indices with BMI z-scores from birth to adolescence. Briefly, this method estimates the BMI-z trajectory using a series of linear slopes that are connected by knot points to determine growth rates during specific periods (34). To determine knot points, we first used the best-fitting fractional polynomials for BMI z-scores over time to visually estimate the approximate age at which a BMI z-score trajectory changed in magnitude or direction (**Supplemental Figure 1**). Fractional polynomials allow for more flexible trajectory shapes than local polynomial or Lowess curves, and their use to visualize and select knot positions for linear splines is well documented (35). We placed knot points also to coincide with developmental milestones and sensitive periods of obesity development. We used the Bayesian Information Criterion to determine the optimum number (4 knots) and location of knots (at ages 1 mo, 6 mo, 3 y, and 10 y) (32, 36), resulting in 5 linear slopes at the following intervals: birth to 1 mo, $>$1–6 mo, $>$6 mo to 3 y, $>$3–10 y, and $>$10 y (**Supplemental Table 1**). Subsequently, we estimated average BMI-z growth rates for each growth period by including each age interval as a predictor in the linear spline model. We estimated the average BMI-z at 7 individual time points (birth, 1 mo, 6 mo, 3 y, 7 y, 10 y, and 15 y) using the margins command in Stata.

To determine whether BMI-z growth rates within each of the specified periods differed by maternal diet, we included each dietary index (in quartiles) in separate models as a fixed effect, and also included each as an interaction with each of the 5 linear slopes in the linear spline models. We used the average

first- and second-trimester dietary indices because these were highly correlated ($r > 0.64$; $P < 0.001$ for all indices) and because we have previously shown in this cohort that overall means of foods and nutrients from foods did not change appreciably from the first to second trimester (37). As a sensitivity analysis, we repeated our main analysis with the dietary measures from the first and second trimesters separately. We also specified random effects for the intercept and for each of the 5 linear slopes using an unstructured covariance matrix, to account for repeated BMI-z observations in each child and to reflect the heterogeneity in the data. In all models, we adjusted for the following confounders: maternal age, education, race/ethnicity, parity, income, smoking, and ppBMI. We analyzed the interaction terms of each dietary index with each of the 5 linear BMI-z slopes; these estimates describe whether the association of each maternal dietary index with the BMI-z becomes stronger/weaker with increasing age. We also used the linear spline models to estimate adjusted differences (and 95% CIs) in predicted BMI z-scores between quartiles of dietary indices at birth, 1 mo, 6 mo, 3 y, 7 y, 10 y, and 15 y using the margins command in Stata.

Finally, we examined interactions by child sex and ppBMI category (OW/OB vs. normal). We also conducted a sensitivity analysis excluding preterm infants ($n = 97$) and excluding mothers with preexisting conditions (chronic hypertension or type 1 or 2 diabetes; $n = 33$).

Results

Participant characteristics

Characteristics of the dyads are shown in **Table 1** by quartiles of MDS. Compared with the 669 women excluded from the analysis, the 1459 included women were older (mean ± SD, 32.6 ± 4.7 y vs. 30.2 ± 5.9 y, respectively; $P < 0.001$); were less likely to have obesity (14% vs. 20%, respectively; $P < 0.001$) and smoke in pregnancy (11% vs. 17%, respectively; $P < 0.001$); and more likely to have a college education (72% vs. 48%, respectively; $P < 0.001$), report White race/ethnicity (73% vs. 52%, respectively; $P < 0.001$), and have a household income >$70,000 (63% vs. 54%, respectively; $P = 0.001$). Included women also had lower DII (mean ± SD, −2.6 ± 1.4 units vs. −2.4 ± 1.5 units, respectively; $P = 0.001$), higher AHEI-P (mean ± SD, 61 ± 10 points vs. 60 ± 10 points, respectively; $P = 0.04$), and higher MDS scores (mean ± SD, 4.6 ± 2 points vs. 4.2 ± 2 points, respectively; $P < 0.001$) compared to excluded women. Among the included participants, the mean ± SD MDS was 4.6 ± 2 points, the DII was −0.26 ± 1.4 units, and the AHEI-P was 61 ± 10 points. The MDS correlated strongly with the DII (Pearson $r = -0.69$; $P < 0.001$) and AHEI-P ($r = 0.72$; $P < 0.001$). The AHEI-P also was strongly correlated with the DII ($r = -0.71$; $P < 0.001$). Women with the highest versus lowest adherence to the Mediterranean diet were older, less likely to have obesity or smoke during pregnancy, and were more likely to have a college education and a household income >$70,000. They also had the highest AHEI-P and lowest DII scores in pregnancy (Table 1). Similar trends in characteristics were observed among women by AHEI-P and DII quartiles (**Supplemental Tables 2** and **3**). There were no significant differences in characteristics among children of women in different quartiles of MDS and AHEI-P scores, though infants of mothers with the highest DII scores were more likely to have a lower birthweight-for-GA z-score.

Average growth rates and BMI-z

The average annual BMI-z growth rates within each growth period are shown in **Supplemental Table 4**, and the average BMI z-scores at birth, 1 mo, 6 mo, 3 y, 7 y, 10 y, and 15 y are shown in **Table 2**. The overall BMI-z trajectory is shown in **Supplemental Figure 2**. After a period of growth deceleration in the newborn period, we found that the most rapid relative BMI-z gain occurred during infancy, between 1 and 6 mo (mean, 1.68 SD units/y; SEM, 0.08). Thereafter, the BMI-z continued to accelerate in early childhood at a lower rate (mean, 0.11 SD units/y; SEM, 0.01), followed by a slow deceleration in mid-childhood and adolescence. The mean BMI-z was 0.35 SD units (SEM, 0.03; 64th percentile) at birth, nadired at 1 mo, and reached its peak at 3 y (mean, 0.68 SD units; SEM, 0.03; 75th percentile).

Associations of maternal dietary indices with child BMI-z growth rates and BMI z-scores

Dietary Inflammatory Index.

During the period between 3 and 10 y, children who were exposed to the highest (Q4) versus lowest quartile (Q1) of DII in utero had a faster BMI-z growth rate (β, 0.03 SD units/y; 95% CI: 0.00–0.06; **Table 3**; **Figure 2**A), resulting in higher BMI z-scores in mid-childhood [7 y; 0.17 SD units (95% CI: 0.01–0.33); 10 y, 0.26 SD units (95% CI: 0.06–0.47); **Table 4**; Figure 2A].

Mediterranean Diet Score.

Adherence to the Mediterranean diet in pregnancy was not associated with the offspring BMI-z growth rate, though there was a significant trend towards a faster growth rate between 3 and 10 y with lower maternal adherence ($P = 0.04$). Children of women consuming the lowest (Q1) versus highest (Q4) quartile of MDS in utero had higher BMI z-scores in early and mid-childhood and in adolescence [3 y, 0.18 SD units (95% CI: 0.01–0.35); 7 y, 0.25 SD units (95% CI: 0.08–0.42); 10 y, 0.30 SD units (95% CI: 0.09–0.52); 15 y, 0.23 SD units (95% CI: 0.02–0.43); Table 4; Figure 2B].

Alternate healthy eating index for pregnancy.

There were no evident associations between the AHEI-P dietary pattern in pregnancy and offspring growth rates in any time period (Table 3). Additionally, exposure to the AHEI-P in utero was not associated with the BMI-z at any evaluated time point from birth to adolescence (Table 4; Figure 2C).

Sensitivity and effect modification analyses

The association of pregnancy DII with child BMI-z growth rates between 3–10 y and BMI z-scores did not differ by child sex (P-interaction > 0.20). The associations of pregnancy DII with child BMI-z growth rates between 3–10 y differed by ppBMI category (P-interaction = 0.03). Children of

TABLE 1 Characteristics of study participants by quartile of Mediterranean Diet Score in Project Viva, a cohort recruited from the Boston, Massachusetts, area in 1999–2002

	n	Total	Q1, *n* = 239	Q2, *n* = 475	Q3, *n* = 252	Q4, *n* = 493	*P* value
Maternal characteristics							
MDS, points	1459	4.6 ± 2	1.6 (0.6)	3.5 (0.5)	5 (0)	6.9 (0.9)	<0.001
DII, units	1459	−2.6 ± 1.4	−1 (1.5)	−2.2 (1.1)	−3.0 (0.9)	−3.7 (0.7)	<0.001
AHEI-P, points	1459	61 ± 10	50 (7)	57 (7)	63 (7)	69 (8)	<0.001
Age, y	1459	32.6 ± 4.7	31. 3 ± 4.9	32.2 ± 4.8	32.9 ± 4.5	33.4 ± 4.4	<0.001
Pre-pregnancy BMI, kg/m^2	1459	24.6 ± 5.2	25.6 ± 5.9	25.1 ± 5.5	24.4 ± 5.1	23.9 ± 4.6	<0.001
Pre-pregnancy BMI category, *n* (%)	1459	—	—	—	—	—	0.007
Underweight, < 18.5	—	48 (3)	3 (1)	17 (4)	7 (3)	21 (4)	
Normal, 18.5 to <25	—	890 (61)	137 (57)	269 (57)	157 (62)	327 (66)	
Overweight, 25 to <30	—	319 (22)	54 (23)	116 (24)	53 (21)	96 (20)	
Obese, ≥30	—	202 (14)	45 (19)	73 (15)	35 (14)	49 (10)	
Education, *n* (%)	1459	—	—	—	—	—	<0.001
Not a college graduate	—	406 (28)	104 (44)	155 (33)	67 (27)	80 (16)	
College graduate	—	1053 (72)	135 (56)	320 (67)	185 (73)	413 (84)	
Race/ethnicity, *n* (%)	1459	—	—	—	—	—	0.08
White	—	1062 (73)	161 (67)	343 (72)	192 (76)	366 (74)	
Black	—	176 (12)	35 (15)	59 (12)	26 (10)	56 (11)	
Hispanic	—	88 (6)	24 (10)	27 (6)	12 (5)	25 (5)	
Asian	—	79 (5)	6 (3)	28 (6)	13 (5)	32 (7)	
Other	—	54 (4)	13 (5)	18 (4)	9 (4)	14 (3)	
Nulliparous, *n* (%)	1459	—	—	—	—	—	0.06
No		750 (51)	147 (57)	251 (53)	130 (52)	232 (47)	
Yes		709 (49)	102 (43)	224 (47)	122 (48)	261 (53)	
Household income >$70,000/y, *n* (%)	1459	—	—	—	—	—	<0.001
No	—	537 (37)	111 (46)	192 (40)	81 (32)	153 (31)	
Yes	—	922 (63)	128 (54)	283 (60)	171 (68)	340 (69)	
Pregnancy smoking status, *n* (%)	1459	—	—	—	—	—	<0.001
Never	—	998 (68)	157 (66)	323 (68)	167 (66)	351 (71)	
Former	—	309 (21)	42 (17)	93 (20)	54 (22)	120 (24)	
Smoked during pregnancy	—	152 (11)	40 (17)	59 (12)	31 (12)	22 (5)	
Chronic hypertension	1432	—	—	—	—	—	0.50
No	—	1410 (98)	232 (99)	454 (98)	246 (98)	478 (98)	
Yes	—	22 (2)	1 (1)	8 (2)	5 (2)	8 (2)	
Type 1 or 2 diabetes	1459	—	—	—	—	—	
No	—	1448 (99)	237 (99)	471 (99)	249 (99)	491 (99)	0.68
Yes	—	11 (1)	2 (1)	4 (1)	3 (1)	2 (1)	
Child characteristics							
Sex, *n* (%)	1459	—	—	—	—	—	
Male	—	730 (50)	108 (45)	243 (51)	137 (54)	242 (49)	0.21
Birthweight, g	1459	3484 ± 570	3523 ± 600	3448 ± 588	3460 ± 613	3511 ± 509	0.21
BWGA z-score, SD units	1458	0.2 ± 0.93	0.31 ± 0.96	0.17 ± 0.92	0.17 ± 0.95	0.21 ± 0.94	0.24
Gestational age, weeks	1459	39.5 ± 1.9	39.5 ± 2	39.4 ± 2.1	39.5 ± 2	39.7 ± 1.6	0.06

Values are expressed as *n* (%) for categorical data (compared using the chi-square test) and mean ± SD for normally distributed data (compared using the ANOVA test). Abbreviations: AHEI-P, Alternate Healthy Eating Index for Pregnancy; BWGA: birthweight for sex and gestational age; DII, Dietary Inflammatory Index; MDS, Mediterranean Diet Score; Q, quartile.

normal-weight mothers with in-utero exposure to the highest versus lowest quartile of DII experienced a higher growth rate between 3–10 y (0.04 SD units/y; 95% CI: 0.01–0.08) in adjusted analyses. However, there were no significant associations between pregnancy DII and child BMI-z growth rates in children of mothers with OW/OB in unadjusted models. The overall growth trajectories by quartile of DII were similar because the associations of maternal DII with BMI z-scores from childhood through early adolescence did not vary by ppBMI category (*P*-interaction > 0.20).

The associations of pregnancy MDS with child BMI z-scores did not differ by child sex (*P*-interaction > 0.20). The associations of MDS with child BMI z-scores in early childhood differed weakly by ppBMI status (*P*-interaction = 0.07), where exposure to maternal OW/OB status and low MDS in utero versus exposure to a normal maternal weight and high MDS was associated with a slightly higher BMI-z [OW/OB–MDS Q1 vs.

TABLE 2 Predicted average child BMI z-scores (SD units) at various ages from birth to adolescence among 1459 children in Project Viva

Age	Mean	SEM	Percentile
Birth	0.35	0.03	64
1 mo	−0.30	0.03	38
6 mo	0.41	0.03	66
3 y	0.68	0.03	75
7 y	0.59	0.03	72
10 y	0.52	0.04	70
15 y	0.48	0.04	68

Linear spline mixed-effects models were used to estimate mean BMI-z (SD units) and SEM at each age among 1459 children in Project Viva with 25,016 BMI-z observations. BMI percentiles were then derived from z-scores. The BMI-z values are based on WHO age- and sex-standardized BMI scores. Abbreviation: BMI-z, BMI z-score.

normal BMI–MDS Q4 (ref) at 3 y, 0.32 SD units; 95% CI: −0.02 to 0.66].

We did not conduct interaction analyses with AHEI-P, because the primary associations were null. Our sensitivity analyses showed no overall changes in the primary outcomes when mothers with chronic hypertension or preexisting diabetes were excluded from the analytical sample. Results were also similar when preterm infants were excluded from the analytical sample. However, in this subgroup of only term infants, there were associations between maternal MDS and growth rates from 1–6 mo and BMI-z at birth. Aside for some attenuation in the associations of second-trimester MDS scores with BMI z-scores in early adolescence, results were overall similar when using the first and second FFQs separately.

Discussion

In this prospective cohort study, we showed that specific dietary indices in pregnancy were associated with period-specific growth rates and BMI z-scores from birth to adolescence. Specifically, children with intrauterine exposure to high maternal dietary inflammation and low adherence to a Mediterranean-type diet experienced faster growth rates from early childhood to mid-childhood, resulting in higher BMI z-scores during the follow-up period. However, maternal adherence to the AHEI-P was not predictive of offspring BMI z-scores or growth rates in this cohort.

We observed a rapid deceleration in BMI z-scores in the first month of life, followed by rapid BMI-z gain in infancy, then moderately fast growth in early childhood, and finally a slight deceleration in later childhood and adolescence. The period of rapid relative decreases in BMI z-scores observed after birth was consistent with findings from reference studies in Europe and the United States comparing child growth to WHO standards (38). Rolland-Cachera et al. (38) showed that when exclusively breastfed infants were analyzed separately, their growth patterns were closer to the WHO standards, suggesting that early feeding practices may explain the differences in BMI-z growth patterns observed in the first few months of life. The majority of infants in the Viva Cohort were not exclusively breastfed in the first 4 mo

TABLE 3 Associations of maternal dietary indices in pregnancy with rate of BMI z-score change in the newborn period, infancy, infancy to early childhood, early- to mid-childhood, and mid-childhood to adolescence periods

	Mean differences in BMI-z growth rates (SD units/y)				
Maternal Dietary Index	Birth to 1 mo	1 to 6 mo	6 mo to 3 y	3 to 10 y	10 to 18 y
DII (units)					
Q1 (<−3.6)	Ref	Ref	Ref	Ref	Ref
Q2 (−3.6 to −2.8)	0.21 (−2.50 to 2.92)	0.15 (−0.28 to 0.58)	−0.02 (−0.08 to 0.04)	0.01 (−0.01 to 0.04)	−0.01 (−0.04 to 0.02)
Q3 (−2.8 to −1.9)	−1.11 (−3.81 to 1.59)	0.15 (−0.28 to 0.57)	−0.00 (−0.07 to 0.06)	0.00 (−0.02 to 0.03)	0.00 (−0.02 to 0.03)
Q4 (>−1.9)	−0.49 (−3.36 to 2.38)	0.43 (−0.02 to 0.88)	0.00 (−0.06 to 0.07)	0.03 (0.00 to 0.06)	−0.03 (−0.06 to 0.00)
P-trend[1]	0.53	0.08	0.88	0.08	0.21
MDS (points)					
Q4 (≥6)	Ref	Ref	Ref	Ref	Ref
Q3 (5)	1.40 (−1.44 to 4.25)	0.11 (−0.34 to 0.55)	−0.02 (−0.08 to 0.05)	0.00 (−0.03 to 0.03)	0.01 (−0.02 to 0.04)
Q2 (3 to 4)	−1.03 (−3.43 to 1.37)	0.27 (−0.11 to 0.66)	0.00 (−0.05 to 0.06)	0.03 (0.00–0.05)	−0.02 (−0.05 to 0.00)
Q1 (<3)	−2.58 (−5.56 to 0.39)	0.31 (−0.16 to 0.78)	0.05 (−0.02 to 0.12)	0.02 (−0.01 to 0.05)	−0.02 (−0.05 to 0.02)
P-trend[1]	0.07	0.11	0.21	0.04	0.09
AHEI-P (points)					
Q4 (>67)	Ref	Ref	Ref	Ref	Ref
Q3 (61 to 67)	−0.60 (−3.29 to 2.09)	0.01 (−0.42 to 0.43)	0.03 (−0.03 to 0.09)	−0.00 (−0.03 to 0.02)	−0.01 (−0.04 to 0.02)
Q2 (54 to 61)	−1.18 (−3.91 to 1.55)	0.19 (−0.24 to 0.62)	−0.00 (−0.07 to 0.06)	0.01 (−0.02 to 0.04)	0.00 (−0.02 to 0.03)
Q1 (<54)	−0.45 (−3.31 to 2.40)	0.20 (−0.26 to 0.65)	−0.03 (−0.09 to 0.04)	0.00 (−0.03 to 0.03)	−0.02 (−0.05 to 0.01)
P-trend[1]	0.65	0.29	0.31	0.64	0.33

Data are from participants in Project Viva, a cohort recruited in the Boston, Massachusetts, area in 1999–2002. Children were classified as being in the newborn period from birth to 1 mo, being in infancy from 1 to 6 mo, being in infancy to early childhood from 6 mo to 3 y, being in early to mid-childhood from 3 to 10 y, and being in mid-childhood to adolescence from 10 to 18 y. Linear spline mixed-effects models were used to estimate mean differences in BMI-z growth rates (units/y) by quartiles of 3 dietary indices during 5 growth periods among 1459 participants. We included the maternal dietary index as a fixed effect and as an interaction term with each of the 5 splines of age and adjusted for maternal age at enrollment, education, parity, race/ethnicity, pregnancy smoking status, household income, and pre-pregnancy BMI. The reference category for DII exposure was the lowest quartile of inflammation or Q1, while the reference category for MDS and AHEI-P was the highest quartile of adherence or Q4. The BMI-z values are based on WHO age- and sex-standardized BMI scores. Abbreviations: AHEI-P, Alternate Healthy Eating Index for Pregnancy; BMI-z, BMI z-score; DII, Dietary Inflammatory Index; MDS, Mediterranean Diet Score; Q, quartile; Ref, reference.

[1] *P*-trends were obtained from linear spline mixed-effects models as defined above, using each 4-category dietary index as a continuous variable.

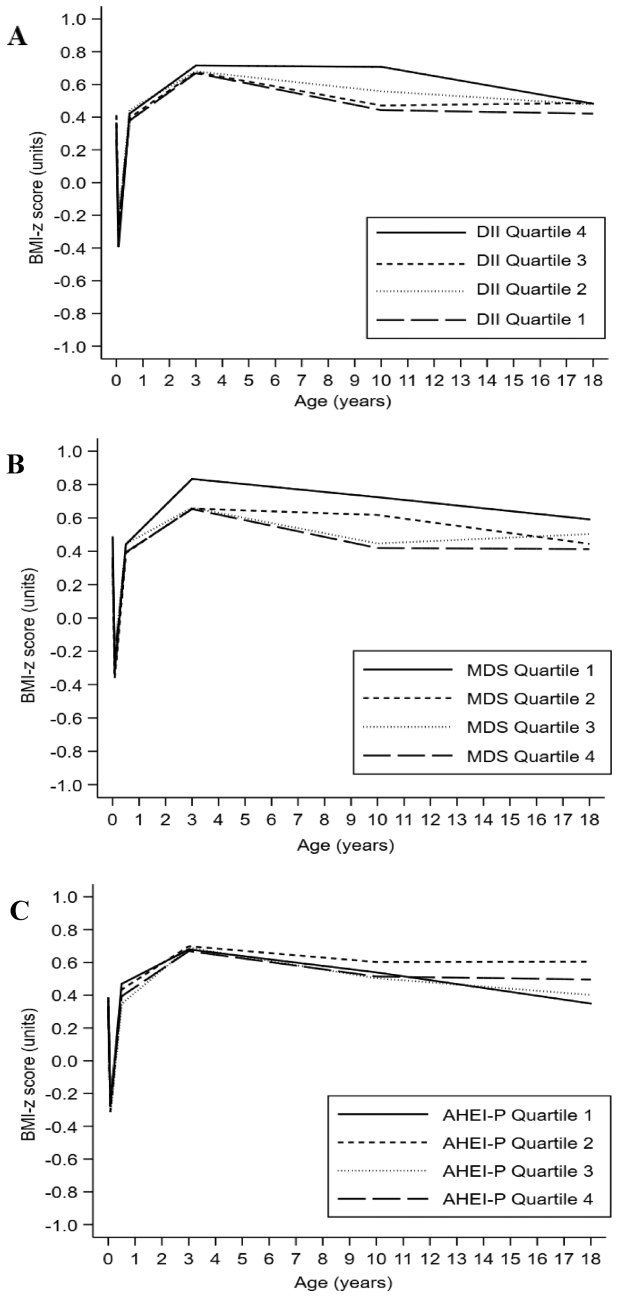

FIGURE 2 Predicted trajectories of child BMI-z from birth through adolescence from adjusted linear splines mixed-effect models according to quartiles of (A) maternal DII in pregnancy, (B) MDS in pregnancy, and (C) AHEI-P among 1459 participants (25,016 BMI-z observations) in Project Viva, a cohort recruited from the Boston, Massachusetts, area in 1999–2002. Abbreviations: AHEI-P, Alternate Healthy Eating Index for Pregnancy; BMI-z, BMI z-score; DII, Dietary Inflammatory Index; MDS, Mediterranean Diet Score.

of life, unlike children in the WHO reference study, who were primarily breastfed during this period.

Our findings are consistent with and expand upon those of other groups that have explored only a specific dietary pattern or explored outcomes at only 1 time point. Other studies have similarly found no associations of the AHEI-P or MDS scores in pregnancy with birth weight (15), but significant associations of DII scores with birth weight (39) and of DII and MDS scores with BMI z-scores in mid-childhood (9, 12). We additionally report that MDS scores in pregnancy were associated with higher BMI z-scores in adolescence, which is novel. Our findings suggest that there are specific developmental periods when the diet during pregnancy may influence offspring growth. Leveraging data from longitudinal cohort studies that can simultaneously examine associations at several time points can shed light into dynamic relationships that may exist between pregnancy diets and offspring growth patterns.

We identified only 4 studies (14–17) that have shown associations of a priori and a posteriori maternal dietary patterns with BMI-z trajectories in early childhood. However, these studies have been limited to short follow-up periods (e.g., until age 4.5 y) and have not been able to assess whether there are specific, distinct periods between birth and adolescence, when rates of weight gain are more susceptible to the programming effect of nutrition in pregnancy.

Our findings suggest that the influence of the pregnancy diet on child BMI z-scores and growth velocity may not be apparent until later in childhood (3–10 y). It has been previously speculated that the maternal diet during pregnancy may induce epigenetic alterations in the fetus that might be unmasked by an obesogenic environment later in life or may influence obesogenic feeding behaviors and brain reward pathways that manifest in later childhood, when children begin to make more independent food choices (40, 41). Thus, child diet is likely a mediator on the causal pathway from prenatal diet to child growth. Future research should quantify the mediating effects of child dietary patterns throughout childhood and adolescence in the associations of maternal diet with child growth trajectories.

We found that AHEI-P scores in pregnancy were not associated with offspring growth trajectories, while DII and MDS indices were. These results are consistent with prior studies that found no associations between the maternal AHEI-P score and birth weight or offspring OW/OB risk (15, 22, 42). These differences might reflect the mechanisms underlying the effects of the maternal diet on offspring health. Higher adherence to the MED and a lower DII score both specifically reflect higher intakes of anti-inflammatory n-3 PUFA, MUFA, and antioxidants, which have been shown in rodent and nonhuman primate models to regulate hedonic pathways in the brain and attenuate inflammation- and oxidative stress–mediated metabolic dysfunction in the offspring (43–47). However, the AHEI-P did not specifically include an assessment of intakes of nuts and seeds, the MUFA/SFA ratio, or the ratio of (white and red) meat to fish, which are critical components of the MDS. Although the AHEI-P score was correlated with the DII and MDS scores in this cohort, the lack of findings with the AHEI-P may be due to differences in the nutrients and foods that are evaluated in the AHEI-P versus in the MDS and DII. Given their role in metabolic programming, future studies should evaluate the role of other dietary patterns that include specific assessments of these important nutrients and foods on offspring adiposity and growth.

Our study has several strengths. The prospective, longitudinal design and the relatively large sample size decrease the chance of recall bias, increase the reliability of our data, and improve the overall precision for predicting risk. To prevent attrition bias due to missing covariate data, we included only participants without missing covariates. To reduce selection bias, we included children with measurements at clinical visits; therefore, we were

TABLE 4 Associations of maternal dietary indices in pregnancy with predicted BMI z-scores in infancy, childhood, and adolescence among participants in Project Viva, a cohort recruited from the Boston, Massachusetts, area in 1999–2002

Maternal Dietary Index	Mean difference in predicted BMI-z (SD units)						
	Birth	1 mo	6 mo	3 y	7 y	10 y	15 y
DII (units)							
Q1 (<−3.6)	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Q2 (−3.6 to −2.8)	−0.02 (−0.19 to 0.15)	0.00 (−0.17 to 0.18)	0.07 (−0.08 to 0.21)	0.01 (−0.14 to 0.16)	0.07 (−0.08 to 0.22)	0.12 (−0.08 to 0.31)	0.08 (−0.11 to 0.26)
Q3 (−2.8 to −1.9)	0.05 (−0.13 to 0.21)	−0.04 (−0.22 to 0.13)	0.02 (−0.13 to 0.16)	0.01 (−0.15 to 0.16)	0.02 (−0.13 to 0.17)	0.03 (−0.17 to 0.22)	0.05 (−0.13 to 0.23)
Q4 (>−1.9)	−0.10 (−0.28 to 0.08)	−0.14 (−0.32 to 0.05)	0.04 (−0.11 to 0.20)	0.05 (−0.12 to 0.21)	0.17 (0.01–0.33)	0.26 (0.06–0.47)	0.14 (−0.06 to 0.33)
P-trend[1]	0.47	0.14	0.73	0.62	0.09	0.04	0.23
MDS (points)							
Q4 (≥6)	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Q3 (5)	−0.11 (−0.29 to 0.07)	0.01 (−0.18 to 0.19)	0.05 (−0.10 to 0.20)	0.01 (−0.15 to 0.17)	0.02 (−0.14 to 0.18)	0.03 (−0.18 to 0.24)	0.07 (−0.13 to 0.26)
Q2 (3 to 4)	−0.04 (−0.19 to 0.11)	−0.12 (−0.28 to 0.04)	−0.00 (−0.13 to 0.13)	0.00 (−0.13 to 0.14)	0.11 (−0.02 to 0.25)	0.20 (0.03–0.37)	0.09 (−0.07 to 0.26)
Q1 (<3)	0.12 (−0.07 to 0.31)	−0.08 (−0.28 to 0.11)	0.05 (−0.11 to 0.21)	0.18 (0.01–0.35)	0.25 (0.08–0.42)	0.30 (0.09–0.52)	0.23 (0.02–0.43)
P-trend[1]	0.38	0.16	0.76	0.12	0.003	0.002	0.04
AHEI-P (points)							
Q4 (>67)	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Q3 (61 to 67)	−0.00 (−0.17 to 0.17)	−0.05 (−0.22 to 0.13)	−0.05 (−0.19 to 0.10)	0.02 (−0.13 to 0.17)	0.00 (−0.15 to 0.15)	−0.01 (−0.20 to 0.18)	−0.06 (−0.25 to 0.12)
Q2 (54 to 61)	0.06 (−0.12 to 0.23)	−0.04 (−0.22 to 0.14)	0.04 (−0.11 to 0.19)	0.03 (−0.12 to 0.18)	0.06 (−0.09 to 0.22)	0.09 (−0.11 to 0.28)	0.10 (−0.08 to 0.28)
Q1 (<54)	0.03 (−0.15 to 0.21)	−0.01 (−0.19 to 0.18)	0.08 (−0.08 to 0.23)	0.01 (−0.15 to 0.17)	0.02 (−0.14 to 0.18)	0.03 (−0.18 to 0.23)	−0.08 (−0.28 to 0.11)
P-trend[1]	0.60	0.96	0.21	0.86	0.62	0.57	0.91

Linear splines mixed-effects models and the postestimation margins command in Stata were used to estimate mean differences in BMI-z (units) by quartiles of 3 dietary indices at specific ages among 1459 participants. We included the maternal dietary index as a fixed effect and as an interaction term with each of the 5 splines of age and adjusted for maternal age at enrollment, education, parity, race/ethnicity, pregnancy smoking status, household income, and pre-pregnancy BMI. The reference category for DII exposure was the lowest quartile of inflammation or Q1, while the reference category for MDS and AHEI-P was the highest quartile of adherence or Q4. The BMI-z values are based on WHO age- and sex-standardized BMI scores. Abbreviations: AHEI-P, Alternate Healthy Eating Index for Pregnancy; BMI-z, BMI z-score; DII, Dietary Inflammatory Index; MDS, Mediterranean Diet Score; Q, quartile; Ref, reference.

[1] *P*-trends were obtained from linear spline mixed-effects models as defined above, using each 4-category dietary index as a continuous variable.

not dependent on active participation. We evaluated multiple dietary indices and focused on those that are known to be associated with disease risk (48–50) and that can be generalized to other populations. We assessed several potential confounders and conducted sensitivity and interaction analyses to assess the robustness of our findings.

Our study did have some limitations. We cannot exclude the possibility of residual confounding, given the observational nature of this study. However, we did adjust for multiple important potential confounders, including maternal ppBMI, which is a major prenatal determinant of child growth and the obesity risk. Although breastfeeding may influence growth trajectories in childhood, we did not adjust for this factor, because it is not a confounder, and it may be on the causal pathway of the associations between the prenatal diet and child growth trajectory (39). Therefore, additional research is needed to evaluate the extent to which breastfeeding mediates these associations. Furthermore, future studies should evaluate other child factors, such as physical activity and screen time, and model them longitudinally to span the entire growth trajectory to accurately explore any interaction effects. Our sensitivity analyses that excluded infants born preterm might have introduced a collider-stratification bias, because prematurity may be on the causal pathway between maternal diet and child growth. However, we observed no appreciable changes to our results after excluding those infants born preterm. Additionally, the Project Viva cohort is predominantly White and is characterized by generally higher socioeconomic statuses and lower rates of obesity, limiting generalizability to other cohorts. Furthermore, our results may have been biased towards the null since the study population was generally healthier, with a better dietary profile compared to excluded participants. We may have also been underpowered to detect significant associations in children of women entering pregnancy with OW/OB. Finally, we used different measurement methods for weight and height during research and clinic visits, perhaps biasing results towards the null, though we did apply a corrective algorithm to the clinical lengths.

In conclusion, dietary indices in pregnancy that reflect the anti-inflammatory potential of foods and nutrients were associated with rates of weight gain, specifically in childhood, and BMI-z trajectories from childhood through adolescence. Because the childhood period is sensitive to the programming effect of diet in pregnancy and is strongly predictive of later obesity risks, future intervention studies should explore the role of anti-inflammatory diets in pregnancy on offspring growth, feeding behaviors, and cardiometabolic health.

The authors' responsibilities were as follows—EO: designed the original research study for which the data used in this analysis were collected; CM-D, SLR-S, IMA, SS, EO: developed the concept and design; CM-D, SLR-S, IMA, NS, JRH: acquired or analyzed the data; CM-D: drafted the manuscript and had primary responsibility for the final content; and all authors: contributed to the interpretation of the findings and read and approved the final manuscript.

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Data Availability

Data described in the manuscript, code book, and analytic code will be made available upon request pending application and approval.

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