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Journal

Journal of the American Heart Association, 13(24)

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Publication Date

2024-12-17

DOI

10.1161/JAHA.124.035391

Peer reviewed

Journal of the American Heart Association

ORIGINAL RESEARCH

Dietary Inflammatory Index Is Differentially Associated With Cardiometabolic Health After Pregnancy on the Basis of Adverse Pregnancy Outcome Exposure

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BACKGROUND: Inflammatory diets may influence risk of cardiovascular disease. Subsequent cardiovascular disease is also influenced by adverse pregnancy outcomes (APOs) such as preterm birth, small-for-gestational-age birth, gestational diabetes, and hypertensive disorders of pregnancy. However, the associations between inflammatory diet, APOs, and cardiometabolic health remain unclear.

METHODS AND RESULTS: We used data from the nuMoM2b (Nulliparous Pregnancy Outcomes Study Monitoring Mothers-to-Be) HHS (Heart Health Study) to assess the relationship between dietary quality and cardiometabolic health. We calculated Energy-Adjusted Dietary Inflammatory Index scores representing the inflammatory burden in a person's diet. We used linear regression to determine the association between Energy-Adjusted Dietary Inflammatory Index score and cardiometabolic outcomes. We performed stratified analyses for outcomes with a significant interaction between Energy-Adjusted Dietary Inflammatory Index and APO. Data were available from 3249 participants at a median of 3.1 years after delivery. Higher Energy-Adjusted Dietary Inflammatory Index scores were associated with higher body mass index (B=0.29 kg/m^2 [95% CI, 0.16–0.42]), waist circumference (0.66 cm [95% CI, 0.39–0.93]), diastolic blood pressure (0.26 mm Hg [95% CI, 0.09–0.44]), mean arterial pressure (0.23 mm Hg [95% CI, 0.06–0.40]), triglycerides (2.11 mg/dL [95% CI, 1.05–3.18]), creatinine (2.78 mg/dL [95% CI, 1.13–4.44]), insulin (exp[B]=1.04 [95% CI, 1.03–1.05]) and C-reactive protein (exp[B]=1.07 [95% CI, 1.04–1.10]), and lower high-density lipoprotein cholesterol (−0.41 mg/dL [95% CI, −0.66 to −0.37]) (all P<0.01). Significant interactions with APO (P<0.05) were identified for body mass index and waist circumference, with stratified analysis revealing stronger associations for individuals with APOs.

CONCLUSIONS: A more proinflammatory diet was associated with worse cardiometabolic health measures, and these relationships differed by a person's APO history. Further investigation should establish how dietary modifications after pregnancy may potentially mitigate cardiovascular disease risk.

Key Words: lifestyle behaviors ■ pregnancy ■ prevention

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This manuscript was sent to Tiffany M. Powell-Wiley, MD, MPH, Associate Editor, for review by expert referees, editorial decision, and final disposition.

Supplemental Material is available at https://www.ahajournals.org/doi/suppl/10.1161/JAHA.124.035391

For Sources of Funding and Disclosures, see page 8.

JAHA is available at: www.ahajournals.org/journal/jaha

CLINICAL PERSPECTIVE

What Is New?

- A more inflammatory diet was associated with worse cardiometabolic health in the 2 to 7 years after a first pregnancy.
- Associations between inflammatory diet and cardiometabolic health were significant for all participants but stronger for those who experienced an adverse pregnancy outcome.

What Are the Clinical Implications?

- Inflammatory diets, diets high in processed foods and low nutritional density, may contribute to worse cardiometabolic health in the reproductive years.
- Clinicians should consider providing additional dietary education to individuals who experienced an adverse pregnancy outcome to potentially mitigate the development of cardiovascular disease.

Nonstandard Abbreviations and Acronyms

APO	adverse pregnancy outcome
DII	Dietary Inflammatory Index
E-DII	Energy-Adjusted Dietary Inflammatory Index
HHS	Heart Health Study
nuMoM2b	Nulliparous Pregnancy Outcomes Study Monitoring Mothers-to-Be

Individuals who experience adverse pregnancy outcomes (APOs), such as preterm birth, small-for-gestational-age birth, hypertensive disorders of pregnancy, or gestational diabetes, are at elevated risk for cardiovascular disease (CVD). Specifically, experiencing gestational hypertension, preeclampsia, preterm birth, and gestational diabetes is associated with 1.7, 2.7, 1.6, and 1.7 odds, respectively, of developing CVD in the decade after pregnancy.[1] People who experience an APO are significantly more likely to subsequently manifest hypertension, dyslipidemia, metabolic syndrome, coronary artery disease, kidney disease, and stroke in the years after experiencing an APO.[2–7] APOs collectively occur in ≈20% of pregnancies.[8,9] Thus, the years after pregnancy represent a key point in the life span to intervene to improve cardiometabolic health and prevent cardiovascular disease. To improve cardiometabolic health and prevent cardiovascular disease for those who experience APOs, health behavior intervention in the years after pregnancy may be beneficial.[10,11]

Diet is a key modifiable target to prevent CVD and promote cardiometabolic health.[12–14] While diet likely affects cardiometabolic health and disease risk through multiple actions, one key mechanism may be through a reduction in inflammation, as illustrated by the beneficial effects of the anti-inflammatory Mediterranean diet.[14–17] Inflammation is a key contributing mechanism to risk for CVD and is also increased in pregnant individuals who have an APO.[18–20] One method to quantify the inflammatory potential of a person's diet is the Dietary Inflammatory Index (DII) score or the closely related Energy-Adjusted Dietary Inflammatory Index (E-DII). The latter is a derivative of the DII and further adjusts for caloric intake.[21,22] Higher scores on the DII or E-DII, which indicate a more proinflammatory diet, have been associated with a higher risk of CVD, metabolic syndrome, and the development of APOs, specifically gestational diabetes and preeclampsia.[23–28] Thus, dietary intervention after pregnancy may be a promising avenue to mitigate CVD risk. We sought to determine whether inflammatory potential of diet, as measured by the E-DII score, is associated with key markers of cardiometabolic health and whether the strength of the associations differs by APO status in the years after pregnancy. We hypothesized that those with higher E-DII scores would have worse cardiometabolic health markers and that the associations would be magnified in those who had an APO.

METHODS

We conducted a secondary analysis of data from participants in the nuMoM2b-HHS (Nulliparous Pregnancy Outcomes Study Monitoring Mothers-to-Be Heart Health Study), the methods of which have been previously described.[29,30] Data are available from the Eunice Kennedy Shriver National Institute of Child Health and Human Development Data and Specimen Hub data repository. In brief, nuMoM2b HHS focused on investigating cardiometabolic health in the initial years after a first pregnancy (index pregnancy). Participants attended a clinical study visit about 3 (range, 2–7; interquartile range, 2.5–3.6) years after the index pregnancy. At the clinical study visit, participants completed a blood draw, clinical and anthropomorphic measurements, and questionnaires including the Block Food Frequency Questionnaire to assess current dietary intake. All study procedures were approved by each site's local institutional review board. Participants provided written informed consent. For this analysis, participants who attended the follow-up study visit 2 to 7 years after the index delivery and who had clinical

data about cardiometabolic health and answers to the Block Food Frequency Questionnaire collected were considered eligible. Those with diabetes or hypertension before the index pregnancy, missing dietary data, or missing adverse pregnancy outcome data were excluded.

Cardiometabolic Health Data

Cardiometabolic measures of interest, ascertained at the HHS visit, were body mass index (BMI), waist circumference, systolic blood pressure, diastolic blood pressure (DBP), mean arterial pressure (MAP), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides, hemoglobin A1c, insulin, glucose, creatinine, and hs-CRP (high-sensitivity C-reactive protein). These markers reflect the new American Heart Association framework for Cardiovascular–Kidney–Metabolic Health.[31] Procedures for obtaining these data have been previously described.[30] Briefly, during the study visit, BMI was calculated on the basis of height and weight. Waist circumference measurements were taken twice and averaged. Three blood pressure readings were obtained by an automated device (OMRON HEM-907XL); readings were averaged for analyses. MAP was calculated on the basis of the following formula: MAP=DBP+1/3(systolic blood pressure–DBP). Blood samples were obtained at the study visit to measure HDL-C, LDL-C (calculated by Friedewald equation), triglycerides, insulin, glucose, and hs-CRP. Samples were stored at –70 °C until assayed and measured in batch analyses. Participants with nonfasting data were not included in analyses for HDL-C, LDL-C, triglycerides, hemoglobin $A1c_a$, glucose, insulin, creatinine, and hs-CRP but were included for BMI, blood pressure, and waist circumference analyses.

Dietary Data

Participants completed the Block Food Frequency Questionnaire at the time of the nuMoM2b HHS study visits. The questionnaire assessed average dietary intake over the past year and is a well validated and established metric to assess individual nutrient intake over time.[32–34] NutriQuest analyzed the food frequency questionnaires from the study visit to obtain daily average intake of macro- and micronutrients (eg, calories, caffeine, vitamin B, fiber, vitamin A, tea, polyunsaturated fatty acids). Macro- and micronutrient data were used to calculate the E-DII.[21] The specific method for calculating the E-DII score has been described.[22,35] Participants' intake of micro- and macronutrients was first converted to intake per 1000 kcal densities. Participants' intakes were then subtracted from the corresponding global average and divided by the global standard deviation to create a *Z* score. *Z* scores were centered on 0 and then multiplied by the "article effect score" for each food parameter. The article effect score characterizes the relationship between each food parameter and systemic inflammation on the basis of data from nearly 2000 research articles. The products of these calculations for each of the food parameters are then added to derive the overall E-DII score. Scores can range from about –8 to +8, with negative scores representing a more anti-inflammatory diet and positive scores representing a more inflammatory diet.

Adverse Pregnancy Outcomes

The APOs of interest for this analysis were gestational diabetes, hypertensive disorders of pregnancy, preterm birth, and small-for-gestational-age birth. Each of these outcomes has been associated with adverse cardiometabolic health after pregnancy.[5,8,36] APO status was determined by nuMoM2b investigators at the time of the index pregnancy. Whether an additional pregnancy occurred between the index pregnancy and the HHS study visit was not assessed in this analysis. Gestational diabetes and hypertensive disorders of pregnancy were adjudicated by the nuMoM2b investigators on the basis of data collected by trained medical record abstractors.[29] Preterm birth was defined as birth occurring before 37 weeks 0 days of gestation. Small-for-gestational-age birth was defined as a birth weight at or below the fifth percentile for gestational age at birth according to the Alexander growth curve. A participant was considered to have had an APO if ≥1 of these occurred in the index pregnancy.

Statistical Analysis

Descriptive statistics were calculated for maternal demographic characteristics. Continuous variables were described with means±SD. Categorical variables were described with frequencies and percentages. Before conducting linear regressions, assumptions of linearity, independence, and normality were checked. Insulin and hs-CRP were log transformed. Additionally, we examined if any outliers were present in the data or if any independent variables were highly correlated. Linear regression models were used to examine the association between the E-DII score (ie, the independent variable) and each cardiometabolic outcome (ie, BMI, waist circumference, systolic blood pressure, DBP, MAP, HDL-C, LDL-C, triglycerides, hemoglobin A1c, insulin, glucose, creatinine, and hs-CRP). Covariates of interest were maternal age at the HHS study visit, education (college graduate versus non–college graduate), insurance status (Medicaid versus all other), smoking status at the time of the HHS study visit (never versus ever), and history of breastfeeding after the index pregnancy (never versus ever). Race and ethnicity (non-Hispanic

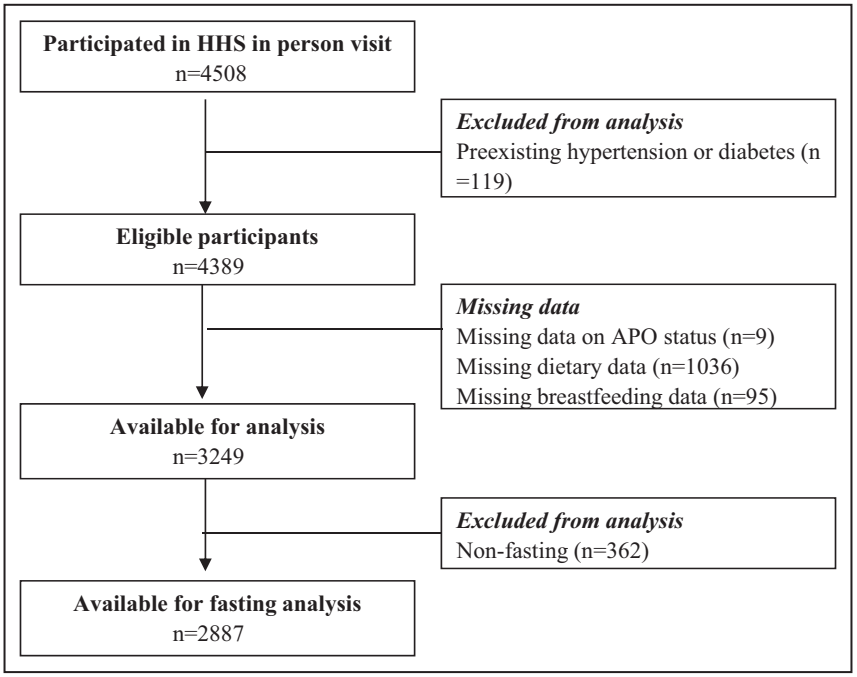

Figure 1. Flow diagram of participant inclusion.
Reasons for exclusion and number are reported on the right-hand side. Retained participants at each step are reported on the left-hand side. APO indicates adverse pregnancy outcome; and HHS, Heart Health Study.

White versus all others), understood as a social construct, was also used as a covariate. Additionally, 43 participants reported taking hypertension, cholesterol, or diabetes medications. We performed a sensitivity analysis with participants taking medications removed from the analyses to determine if medications may have influenced our results. Imputation for missing data was not performed, and any participants with missing variables were excluded. We used χ^2, *t* tests, and Mann–Whitney *U* test to assess for differences in demographic factors and cardiometabolic outcomes between those with dietary data and without dietary data. In the regression models, we assessed for effect modification between E-DII and APO status using an interaction term. When evidence of an interaction ($P<0.05$) existed, we conducted analyses stratified by APO status (yes or no). All analyses were conducted in R 4.3.1 (R Foundation for Statistical Computing).

RESULTS

From an available cohort of 4508 participants who attended a follow-up study visit 2 to 7 years after the index delivery, participants with diabetes or hypertension before the index pregnancy (n=119), missing dietary data (n=1036), missing adverse pregnancy outcome data (n=9), or missing breastfeeding data (n=95) were excluded, yielding a final sample size of 3249. A total of 362 participants were nonfasting or had missing data on fasting status and were excluded from cholesterol, hemoglobin A_{1C}, glucose, insulin, creatinine, and hs-CRP analyses (Figure 1). Participants

Table 1. Demographic Characteristics

Demographics	Analytic sample (n=3249)	
	No.	%
Completed college	1860	57
Ever smoker	840	26
Government insurance	721	22
Breastfed ever	2915	87
Race or ethnicity		
Non-Hispanic White	2145	66
Non-Hispanic Black	311	10
Hispanic	536	16
Asian	109	3
Other*	144	4
Experienced APO	1046	32
Gestational diabetes	129	4
Hypertensive disorders of pregnancy	749	23
Preterm birth	237	7
Small for gestational age	131	4
	Mean	SD
Age	30.95	5.47

APO indicates adverse pregnancy outcome.
*Other includes American Indian, Alaska Native, Native Hawaiian, other Pacific Islander and multiracial categories.

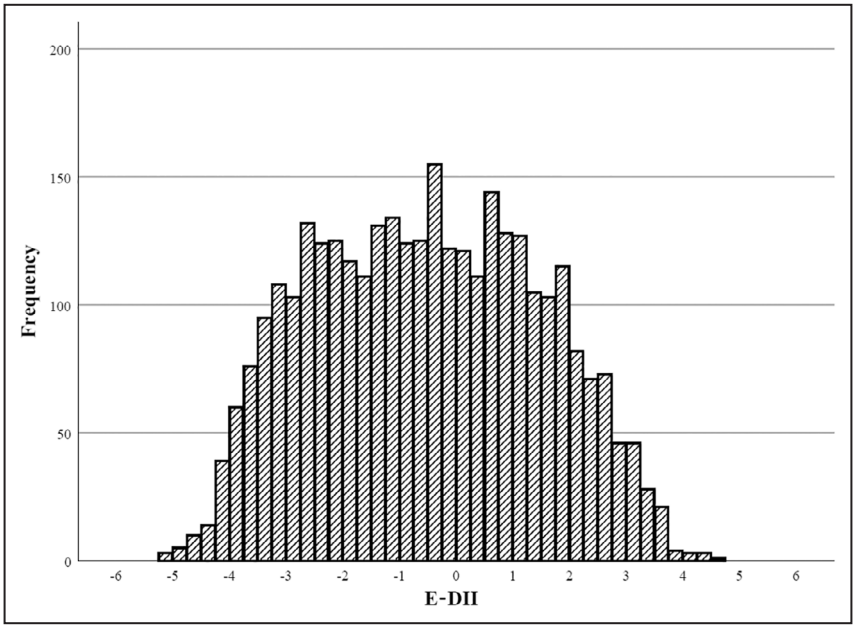

Figure 2. Distribution of E-DII score.
E-DII scores were normally distributed. Positive scores reflect a more pro-inflammatory diet, while negative scores reflect a more anti-inflammatory diet. Scores around 0 represent a neutral diet in regard to inflammatory potential. E-DII indicates Energy-Adjusted Dietary Inflammatory Index.

were on average aged 31 years and had their follow-up visit 3.1 years after their index delivery. Of participants with available dietary data, 66% were non-Hispanic White, 57% had at least a college education, 22% had Medicaid insurance, 26% were ever smokers, and 87% ever breastfed. Complete demographic characteristics are presented in Table 1. Participants who did not complete the dietary survey were more likely to be non-White, have less than a college education, smoke, and have Medicaid insurance. The APO rate did not differ between those who provided dietary data and those who did not (Table S1).

The average E-DII score was −0.53 (±1.99), representing a neutral inflammatory diet. The distribution of E-DII scores is displayed in Figure 2. Data on means±SD and sample sizes for each of the cardiometabolic outcomes are presented in Table 2. Participants on average had an overweight BMI (27.2 kg/m^2), were normotensive (111/72 mm Hg), had normal blood glucose (89 mg/dL), and had near optimal cholesterol levels (HDL-C, 56 mg/dL; LDL-C, 108 mg/dL).

All data used in regression models met the assumptions of linearity, normality, and independence. In adjusted linear regression models (Table 3), a 1-point higher E-DII score was associated with a higher BMI (0.29 kg/m^2 [95% CI, 0.16–0.42]; P=0.02), waist circumference (0.66 cm [95% CI, 0.39, 0.93]; P<0.001), DBP (0.26 mm Hg [95% CI, 0.09–0.44]; P=0.04), MAP (0.23 mm Hg [95% CI, 0.06–0.40]; P=0.007) triglycerides (2.11 mg/dL [95% CI, 1.05–3.18]; P<0.001), creatinine (2.78 mg/dL [95% CI, 1.13–4.44]; P=0.001), lower HDL-C (−0.41 mg/dL [95% CI, −0.66 to −0.15]; P=0.002), and 4% higher insulin (exp(B)=1.04 [95% CI, 1.03–1.05]; <0.001), and 7% higher hs-CRP (exp(B)=1.07 [95% CI, 1.04–1.10]; P<0.001). In a sensitivity analysis

Table 2. Descriptive Data for Cardiometabolic Outcomes

Variable	No.	Mean	SD
BMI, kg/m^2	3233	27.28	7.22
Waist circumference, cm	3235	84.88	14.90
SBP, mm Hg	3239	111.33	10.45
DBP, mm Hg	3239	71.86	9.19
MAP, mm Hg	3239	85.00	9.05
HDL-C, mg/dL	2881	56.24	13.28
LDL-C, mg/dL	2881	108.25	33.99
Triglycerides, mg/dL	2881	92.52	53.31
Creatinine, mg/dL	2872	145.65	86.80
Glucose, mg/dL	2877	89.95	14.53
Hemoglobin A1c	2860	0.35	0.15
	No.	Median	IQR
hs-CRP, mg/L	2823	0.14	0.06–0.45
Insulin, mIU/L	2883	5.21	3.93–8.60

BMI indicates body mass index; DBP, diastolic blood pressure; HDL-C, high density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; IQR, interquartile range; LDL-C, low density lipoprotein cholesterol; MBP, mean arterial pressure; and SBP, systolic blood pressure.

Table 3. Adjusted Linear Regression Models for Associations Between E-DII and Cardiometabolic Outcomes

Variable	Coefficient	95% CI	Standard error	*P* value	Interaction *P* value
BMI	0.29	0.16 to 0.42	0.07	0.02	0.04
Waist circumference	0.66	0.39 to 0.93	0.14	<0.001	0.04
SBP	0.17	−0.03 to 0.37	0.10	0.20	0.89
DBP	0.26	0.09 to 0.44	0.09	0.04	0.71
MAP	0.23	0.06 to 0.40	0.09	0.007	0.84
HDL-C	−0.41	−0.66 to −0.15	0.13	0.002	0.14
LDL-C	−0.28	−0.95 to −0.40	0.35	0.43	0.52
Triglycerides	2.11	1.05 to 3.18	0.54	<0.001	0.24
Creatinine	2.78	1.13 to 4.44	0.84	0.001	0.21
Log insulin	0.04	0.03–0.05	0.01	<0.001	0.65
Glucose	−0.09	−0.38 to 0.20	0.15	0.53	0.50
Hemoglobin A1c	−0.001	−0.004 to 0.37	0.005	0.68	0.47
Log hs-CRP	0.07	0.04 to 0.10	0.02	<0.001	0.38

Analyses adjusted for maternal age, self-reported race, education, government insurance, smoking status, and breastfeeding. BMI indicates body mass index; DBP, diastolic blood pressure; E-DII, Energy-Adjusted Dietary Inflammatory Index; HDL-C, high density lipoprotein cholesterol; hs-CRP, high sensitivity C-reactive protein; LDL-C, low density lipoprotein cholesterol; MAP, mean arterial pressure; and SBP, systolic blood pressure.

with those on medications removed from the analysis, results were similar (Table S2).

Statistically significant interactions were present related to APO status in the association between E-DII and BMI (*P*=0.04) and waist circumference (*P*=0.04). (Table 3). In the APO group, a 1-unit higher E-DII score was associated with a higher BMI (0.45 [95% CI, 0.21, 0.69]; *P*<0.001) and waist circumference (0.96 [95% CI, 0.47–1.45]; *P*<0.001). (Table 4). In the non-APO group, a 1-unit higher E-DII score was associated with a higher BMI (0.24 [95% CI, 0.09–0.39]; *P*=0.002) and waist circumference (0.60 [95% CI, 0.29–0.91]; *P*<0.001) (Table 4).

DISCUSSION

In this analysis, we sought to determine whether a more inflammatory diet, as measured by the E-DII, was associated with cardiometabolic health markers and whether identified associations differed according to the occurrence of an APO in the index pregnancy. Consistent with our hypothesis, higher E-DII scores were associated with worse cardiometabolic health markers (ie, higher BMI, waist circumference, DBP, MAP, triglycerides, insulin, creatinine, and hs-CRP, and lower HDL-C). Interaction effects were identified for BMI, and waist circumference, with associations stronger for those who had APOs compared with those who had not had APOs. Our data suggest that diet may influence cardiometabolic health after pregnancy, particularly for those who experience APOs and may be a fruitful intervention target to reduce CVD.

Our findings are consistent with previous reports from epidemiological data sets for the general population (National Health and Nutrition Examination Survey, Nurses' Health Study, and Health Professionals Follow-Up Study) which have also shown that higher inflammatory diet scores are associated with higher hs-CRP, triglycerides, and BMI and lower HDL-C.[37,38] Beyond individual markers of cardiometabolic health, meta-analyses have shown that higher inflammatory diet scores are associated with up to 41% higher

Table 4. Stratified Results by APO Status for Associations Between E-DII and Cardiometabolic Outcomes

Variable	Coefficient	95% CI	Standard error	*P* value
	APO			
BMI	0.38	0.14–0.63	0.12	0.002
Waist circumference	0.82	0.32–1.32	0.26	<0.001
	No APO			
BMI	0.21	0.06–0.36	0.08	0.007
Waist circumference	0.51	0.20–0.83	0.13	<0.001

Analyses adjusted for maternal age, self-reported race, education, government insurance smoking, and breastfeeding status. APO indicates adverse pregnancy outcome; BMI, Body mass index; and E-DII, Energy-Adjusted Dietary Inflammatory Index.

risk for CVD and 31% higher risk for cardiovascular death.[24,39,40] One proposed mechanism by which diet contributes to CVD is by contributing to chronic inflammation.[21,22] Low-quality diets such as diets high in processed foods, added sugar, and saturated fats have been associated with chronic, low-grade systemic inflammation.[14,41] Conversely, anti-inflammatory diets like the Mediterranean diet have been proposed to lower risk for CVD by lowering chronic inflammation.[42,43] While further work is needed to elucidate the potential mechanisms by which inflammatory diet may be associated with cardiometabolic health after pregnancy, our results warrant further investigation.

We hypothesized that the stronger association between diet and cardiometabolic health observed in those who experienced an APO was related to shared inflammatory pathologies.[20,44] People who develop APOs have higher systemic inflammatory levels across pregnancy compared with those without APOs.[20,45] We posited that an inflammatory diet after pregnancy may extend the inflammatory effects from the APO and exacerbate risk for poor cardiometabolic health. However, we did not find any differences in hs-CRP related to APO status. Further studies are warranted to explore if an anti-inflammatory diet improves cardiometabolic health for those who have experienced an APO and the potential mechanisms.

Pregnancy and the postpartum period represent a unique period in the life course when women and families are highly motivated to implement behavioral change.[46,47] These periods also represent a point in the life course when interventions might be successfully delivered to prevent the development of CVD.[10,48] Postpartum interventions have largely targeted weight loss.[49] In the Blood Pressure Postpartum (BP^2) intervention trial, among people with a history of hypertensive disorders of pregnancy, those adhering to national diet and physical activity recommendations (ie, 5 servings of vegetables and 2 servings of fruit per day and 150 minutes of physical activity per week) had a lower BMI, waist circumference, and blood pressure compared with those who did not meet recommendations.[50] Similarly, multiple randomized trials have shown that lifestyle modification is an effective intervention to reduce BMI, lower triglycerides, and prevent progression to type 2 diabetes in those with a history of gestational diabetes.[11,51–54]

Future work is required to understand whether potential changes in dietary patterns over time may be associated with the development of poor cardiometabolic health. For context, moving from a diet dominated by fast food or the Standard American Diet to a Mediterranean type of diet would potentially decrease someone's E-DII score by 6 points.[55,56] Nevertheless, although the association was stronger in those with an APO history for 2 outcomes, associations were still present in those without an APO history. In total, these data suggest an anti-inflammatory diet might be particularly beneficial in those with APO, given their higher rates of CVD and stronger association of inflammatory diet with CVD, but that anti-inflammatory diets might hold benefit for all. Our results come from an observational study, and causation or directionality cannot be determined. Intervention studies are needed to determine if an anti-inflammatory diet indeed influences CVD in this population and the length of time adherence to an anti-inflammatory diet is needed to achieve the desired effect.

Strengths and Limitations

While the nuMoM2b study is geographically and racially and ethnically diverse, participants who were non-White, had less than a college education, and had public insurance were less likely to complete the dietary questionnaire and also had worse cardiometabolic health, contributing to potential bias in the reported findings. Worse E-DII scores are associated with lower education, public insurance, smoking, younger age, and non-White race or ethnicity, so we can hypothesize that had the individuals without dietary data been included, they likely would have had worse E-DII scores and indeed had worse cardiometabolic health outcome data, and our findings would be similar. The accuracy of E-DII scores is dependent on participants precisely recalling dietary intake; however, the Block Food Frequency Questionnaire is a well-validated and established tool to evaluate dietary intake, and errors in recall should bias toward the null. The dietary assessment reflects the past year of dietary behaviors, but we cannot determine how long individuals participated in the reported dietary behavior. We also cannot rule out that poor cardiometabolic health indices caused participants to modify their diet to become more proinflammatory, although we believe that is unlikely given typical clinical guidance and that it is much more likely, given the known associations between diet and cardiometabolic health, that the diet preceded the worse cardiometabolic state. We adjusted for relevant covariates; however, given the observational nature of the study, residual confounding bias is a limitation. Data about whether a participant had experienced a subsequent pregnancy between the index pregnancy and HHS study visit or the outcome of such a pregnancy were not available. Additional pregnancies may change APO exposure and cardiometabolic health outcomes. Despite these limitations, the study also benefits from its prospective design, deeply phenotyped exposures and outcomes, and diverse population.

CONCLUSIONS

A more inflammatory diet after the first pregnancy is associated with worse cardiometabolic health, and this association is stronger for those with a history of

APOs. Future work is required to understand whether dietary modifications with anti-inflammatory diets might reduce the risk of poor cardiometabolic health after pregnancy, especially for individuals with a history of ≥1 APOs. Intervention studies are needed to determine the effect of diet on cardiometabolic health, to identify the optimal timing of this intervention, and to determine the mechanism by which diet is related to cardiometabolic health in this high-risk population.

ARTICLE INFORMATION

Received March 7, 2024; accepted October 29, 2024.

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Sources of Funding

The nuMoM2b project was supported by grant funding from the Eunice Kennedy Shriver National Institute of Child Health and Human Development: U10 HD063036; U10 HD063072; U10 HD063047; U10 HD063037; U10 HD063041; U10 HD063020; U10 HD063046; U10 HD063048; and U10 HD063053. In addition, support was provided by Clinical and Translational Science Institutes: UL1TR001108 and UL1TR000153. The nuMoM2b Heart Health Study was supported by cooperative agreement funding from the National Heart, Lung, and Blood Institute and the Eunice Kennedy Shriver National Institute of Child Health and Human Development: U10-HL119991; U10-HL119989; U10-HL120034; U10-HL119990; U10-HL120006; U10-HL119992; U10-HL120019; U10-HL119993; U10-HL120018, and U01HL145358; with supplemental support to NHLBI U10-HL119991 from the Office of Research on Women's Health and the Office of Disease Prevention and to National Heart, Lung, and Blood Institute U01HL145358 from the Office of Research on Women's Health; and the National Center for Advancing Translational Sciences through UL-1-TR000124, UL-1-TR000153, UL-1-TR000439, and UL-1-TR001108; and the Barbra Streisand Women's Cardiovascular Research and Education Program, and the Erika J. Glazer Women's Heart Research Initiative, Cedars-Sinai Medical Center, Los Angeles.

Disclosures

Dr Wirth is a cocreator of the DII and E-DII measures. He is an employee Connecting Health Innovations LLC (CHI), a company that has licensed the rights of the DII. The remaining authors have no disclosures to report.

Supplemental Material

Tables S1–S2

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