Energy-Adjusted Dietary Inflammatory Index in pregnancy and maternal cardiometabolic health: findings from the ROLO study

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BACKGROUND: Excessive inflammation during pregnancy has been linked to adverse long-term health outcomes for both mothers and their children. One such outcome is maternal cardiometabolic dysfunction. The Energy-Adjusted Dietary Inflammatory Index is a method of scoring the overall inflammatory potential of the diet. Research on how the inflammatory potential of the maternal diet during pregnancy affects maternal cardiometabolic factors is limited.

OBJECTIVE: We investigated if the maternal Energy-Adjusted Dietary Inflammatory Index was associated with maternal cardiometabolic factors during pregnancy.

STUDY DESIGN: This is a secondary analysis of 518 individuals who participated in the ROLO (Randomized cOntrol trial of a LOw glycemic index diet in pregnancy) study. Maternal Energy-Adjusted Dietary Inflammatory Index scores were calculated in early (12–14 weeks' gestation) and late pregnancy (34 weeks' gestation) using data collected from 3-day food diaries. Body mass index, blood pressure, fasting lipid profiles, glucose levels, and HOMA1-IR were obtained in early and late pregnancy. Multiple linear regression examined associations between early-pregnancy Energy-Adjusted Dietary Inflammatory Index and early and late maternal cardiometabolic markers. In addition, the relationship between late-pregnancy Energy-Adjusted Dietary Inflammatory Index and late cardiometabolic factors was explored. Regression models were adjusted for maternal ethnicity, maternal age at delivery, education level, smoking status, and original randomized control trial group allocation. In regression models examining late-pregnancy Energy-Adjusted Dietary Inflammatory Index with late lipids, change in lipid level from early to late pregnancy was also adjusted for.

RESULTS: Women's mean (standard deviation) age at delivery was 32.8 (±4.01) years, with median (interquartile range) body mass index of 24.45 (23.34–28.20) kg/m^2. Mean (standard deviation) Energy-Adjusted Dietary Inflammatory Index was 0.59 (±1.60) in early pregnancy and 0.67 (±1.59) in late pregnancy. In adjusted linear regression analysis, first-trimester maternal Energy-Adjusted Dietary Inflammatory Index was positively associated with maternal body mass index (*B*=0.007; 95% confidence interval, 0.003–0.011; *P*=.001), early-pregnancy cardiometabolic markers including total cholesterol (*B*=0.155; 95% confidence interval, 0.061–0.249; *P*=.001), triglycerides (*B*=0.043; 95% confidence interval, 0.005–0.080; *P*=.03), low-density lipoproteins (*B*=0.129; 95% confidence interval, 0.049–0.209; *P*=.002), and diastolic blood pressure (*B*=0.538; 95% confidence interval, 0.070–1.006; *P*=.02), and late-pregnancy cardiometabolic markers including total cholesterol (*B*=0.127; 95% confidence interval, 0.012–0.243; *P*=.01) and low-density lipoproteins (*B*=0.110; 95% confidence interval, 0.010–0.209; *P*=.03). In the third trimester, Energy-Adjusted Dietary Inflammatory Index was associated with late-pregnancy diastolic blood pressure (*B*=0.624; 95% confidence interval, 0.103–1.145; *P*=.02), HOMA1-IR (*B*=0.030; 95% confidence interval, 0.005–0.054; *P*=.02), and

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J.R.H. owns a controlling interest in Connecting Health Innovations LLC (CHI), a company that has licensed the right to his invention of the Dietary Inflammatory Index from the University of South Carolina to develop computer and smartphone applications for patient counseling and dietary intervention in clinical settings. N.S. is an employee of CHI. The subject matter of this paper will not have any direct bearing on that work, nor has that activity exerted any influence on this project.

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glucose (B=0.003; 95% confidence interval, 0.003−0.034; P=.03). No associations were observed between third-trimester Energy-Adjusted Dietary Inflammatory Index and late-pregnancy lipid profiles.

CONCLUSION: Maternal diets with a higher Energy-Adjusted Dietary Inflammatory Index, which were low in anti-inflammatory foods and rich in proinflammatory foods, were associated with increased levels of cardiometabolic health risk factors in pregnancy. Promoting dietary intakes that have a lower inflammatory potential may support more favorable maternal cardiometabolic profiles during pregnancy.

Key words: cardiometabolic factors, Energy-Adjusted Dietary Inflammatory Index, inflammation, maternal health, nutrition, pregnancy

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Why was this study conducted?

This study investigates associations between the inflammatory potential of the diet and markers of maternal cardiometabolic health in early and late pregnancy using the Energy-Adjusted Dietary Inflammatory Index (E-DII).

Key findings

First-trimester maternal E-DII was positively associated with early-pregnancy maternal body mass index and cardiometabolic markers including total cholesterol, triglycerides, low-density lipoproteins, and diastolic blood pressure. Maternal E-DII in early pregnancy was also positively associated with cardiometabolic markers in late pregnancy, including total cholesterol and glucose. Third-trimester maternal E-DII was not associated with lipid profiles or glucose, but was positively associated with late-pregnancy diastolic blood pressure and late-pregnancy HOMA1-IR.

What does this add to what is known?

Our findings suggest that E-DII may be a useful tool for assisting pregnant persons in improving their dietary intake during pregnancy by reducing the inflammatory potential of the maternal diet and thus maternal cardiometabolic dysfunction.

Introduction

Excessive inflammation during pregnancy has been linked to adverse maternal and child outcomes, including hypertensive disorders of pregnancy,[1] impaired fetal growth,[2] preterm birth, low birthweight, and maternal insulin resistance.[3] Associations have been observed between dietary intake, inflammation, and cardiovascular markers in nonpregnant cohorts.[4−6] This has led to interest in whether dietary inflammatory potential during pregnancy can modify an individual's risk of cardiometabolic disease in later life. A narrative review of pregnant and nonpregnant adults concluded that adequate evidence exists to demonstrate that a proinflammatory diet is associated with increased risk of noncommunicable diseases such as cardiovascular disease (CVD) and certain cancers.[7]

The Dietary Inflammatory Index (DII) was developed to quantify the inflammatory potential of an individual's diet, measured through inflammatory markers such as Interleukin-1 and C-reactive protein (CRP).[8−10] It has been previously validated for use in pregnant cohorts,[11,12] and includes 45 food parameters such as flavonoids, caffeine, macronutrients, and micronutrients. The greater the DII score, the more proinflammatory the diet. The Energy-Adjusted Dietary Inflammatory Index (E-DII) adjusts for energy intake and has an improved predictive ability relative to that of unadjusted DII.[9,13]

Given that E-DII provides an inflammatory profile of the diet and that pregnancy is an inflammatory state, E-DII may provide understanding on how to improve dietary intakes and consequently promote optimal cardiometabolic health. Chronic systemic inflammation has been implicated in the pathophysiology of later CVD and type 2 diabetes mellitus within a pregnant population.[14] Diet is a potentially modifiable factor that could have a significant effect on maternal cardiometabolic health during pregnancy. We investigated if the inflammatory potential of maternal diet in early and late pregnancy, as measured by E-DII scores, shows correlation with maternal cardiometabolic health.

Materials and Methods

Population

This is a secondary analysis of 518 women who participated in the ROLO (Randomized cOntrol trial of a LOw glycemic index diet in pregnancy) study. The ROLO study took place in Dublin, Ireland from 2007 to 2011,[15] and had institutional ethical approval (Research Ethics Committee of the National Maternity Hospital [GEN/279/12]). In the primary study, secundigravidas (n=800) who previously delivered a macrosomic infant (≥4000 g) were randomized to either a low glycemic index (GI) diet or usual care. Individuals recruited were aged >18 years, at <18 weeks' gestation, with singleton pregnancy, not taking any medication, and with no history of gestational diabetes mellitus (GDM). The study results were previously published.[15,16]

Exposure

Dietary assessment. Dietary intakes were assessed at 12 to 14 weeks' gestation (before low GI dietary intervention) and at 34 weeks using 3-day food diaries. Individuals were asked to record the type and quantity of all foods and drinks, either by weight or by household measure, consumed over 3 consecutive days, including 1 weekend day. Food diaries were analyzed using the nutritional analysis software NetWISP, version 3.0 (Tinuviel Software, Llanfechell,

United Kingdom). This software used the food composition database from the sixth edition of McCance and Widdowson's food composition tables.[17]

Energy-Adjusted Dietary Inflammatory Index. Maternal E-DII scores were calculated using the energy density method[9] from 3-day food diaries for early (12−14 weeks) and late (34 weeks) pregnancy by researchers at the University of South Carolina. A total of 28 dietary parameters were available from the ROLO food diaries, including macronutrients, vitamins, and caffeine.[9] Dietary information was converted and quantified per 1000-kcal values and linked to a regionally representative database of mean (standard deviation) intakes for each dietary parameter. A z-score was created for each dietary parameter by subtracting the adjusted global daily mean intake from the adjusted participant intake.[13] This value was divided by its standard deviation, then converted to a proportion and centered on 0 by multiplying by 2 and subtracting 1. This value is multiplied by the score of the inflammatory effect of the corresponding food component and summed across parameters to yield the overall E-DII score. The E-DII score ranges from −8.87 to 7.98, representing a diet with minimum and maximum inflammatory potential, respectively. The median E-DII score is 0.23.[9] A higher E-DII score indicates a more proinflammatory diet, and a lower value represents a more antiinflammatory diet.

Outcomes

Anthropometric measurements. Weight and height were measured at the first antenatal visit (12−14 weeks). Participants were weighed in light clothing without shoes using seca stand-on scales (seca GmbH & Co. KG, Hamburg, Germany) to the nearest 0.1 kg, and height was measured to the nearest 0.1 cm using a wall-mounted stadiometer. Body mass index (BMI) was calculated using height squared divided by weight. Maternal BMI was categorized per the World Health Organization (WHO) classification system.[18] BMI in the first trimester was assumed to reflect prepregnancy BMI.

Blood samples. Fasting blood samples were taken at approximately 14 weeks' gestation (early pregnancy) and 34 weeks' gestation (late pregnancy). The lipase/GPO-PAP (glycerol phosphate oxidase-p-aminophenazone) method was used to measure high-density lipoprotein (HDL). Serum total cholesterol was measured using Roche cholesterol oxidase method, and triglyceride concentrations were measured using direct HDL Roche third-generation method, on the cobas c 702 module of the Roche cobas 8000 analyzer per the manufacturer's instructions (Roche Diagnostics GmbH, Penzberg, Germany). These methods are standardized against the isotope dilution−mass spectrometry (ID-MS) methods. The Friedewald equation was used to estimate low-density lipoprotein (LDL) concentration. Multianalyte profiling was performed on the Luminex MAGPIX system (Luminex Corporation, Austin, TX). Blood insulin levels were determined using the Human Endocrine Panel, and maternal insulin resistance was calculated using the homeostatic model assessment (HOMA) index; HOMA score is equal to (fasting insulin μU/mL × fasting glucose mmol/L) divided by 22.5. The techniques and the laboratory that completed these analyses were calibrated in accordance with the National Institute of Standards in Technology reference materials.[19]

Blood pressure. Auscultatory blood pressure (BP) (mm Hg) was measured by a trained healthcare professional at booking (early; 12−14 weeks) and at 34 weeks' gestation (late) using a calibrated mobile Trimline aneroid sphygmomanometer (Trimline Medical Products Corporation, Somerville, NJ). Participants were seated, with their arm placed at heart level. Mean arterial pressure (MAP) was calculated by doubling the diastolic BP value, adding the sum to the systolic BP value, and dividing this figure by 3.

Confounders

Maternal age at delivery was recorded. Maternal education level, smoking status, and ethnicity were self-reported at the first antenatal appointment. In the 4-year period (2007−2011) of the primary ROLO study, blood samples taken early in the study were stored for longer before analysis than the samples taken later in the study. Some minor differences were observed in total cholesterol and HDL results between blood samples stored for longer and those stored for shorter periods. Therefore, adjustment was made in the total cholesterol and HDL regression models to account for length of storage time before analysis.

Statistical analysis

Variables were assessed for normality using the Kolmogorov−Smirnov test and visual inspection of histograms. Mean and standard deviation are reported for normally distributed variables and median and interquartile range (25th−75th percentile) for nonnormally distributed data. These variables were $\log^{10}$-transformed before analysis. Paired sample *t*-tests were performed to examine change in E-DII and in maternal lipids from early to late pregnancy.

Pearson's correlations examined associations between E-DII and maternal cardiometabolic markers. Adjusted and nonadjusted linear regression analysis was performed to determine associations among early-pregnancy maternal E-DII and early-pregnancy BMI, early- and late-pregnancy lipid profile, glucose, HOMA1-IR, BP, and MAP. Adjusted regression models controlled for maternal ethnicity, maternal age at delivery, maternal education level, original randomized controlled trial (RCT) group allocation, and smoking history. Regression models with HDL and total cholesterol as the outcome variable were adjusted for stored time before analysis. Multiple linear regression analyses were completed to assess associations among late-pregnancy E-DII and late-pregnancy lipids, glucose, HOMA1-IR, BP, and MAP. Models were controlled for the same confounders as for early pregnancy. Another regression model was completed, which

TABLE 1
Maternal characteristics of the ROLO pregnancy study cohort, Dublin, Ireland, from 2007 to 2011

Characteristics	n (%)	Mean (median)	SD (IQR)
Maternal age at delivery (y)	518	32.75	4.01
Maternal BMI (kg/m^2)	516	(24.45)	(23.34–28.20)
Birth history			
Elective cesarean delivery	58 (12.3)		
Emergency cesarean delivery	33 (7)		
Preterm (<37 wk)	6 (1.2)		
BMI category[a]			
Healthy, n (%)	231 (44.8)	—	—
Overweight, n (%)	198 (38.4)	—	—
Obese, n (%)	85 (16.5)	—	—
Ethnicity			
White Irish, n (%)	454 (86.7)	—	—
Smoking status			
Nonsmoker, n (%)	504 (97.3)	—	—
Cardiometabolic factors			
Early HOMA1-IR	372	(2.40)	(1.43–3.88)
Late HOMA1-IR	344	(3.06)	(1.80–5.10)
Early total cholesterol (mmol/L)	229	4.52	1.20
Late total cholesterol (mmol/L)	233	5.94	1.57
Early triglycerides (mmol/L)	229	1.11	0.45
Late triglycerides (mmol/L)	233	1.80	0.70
Early LDL (mmol/L)	229	3.29	0.97
Late LDL (mmol/L)	233	4.26	1.24
Early HDL (mmol/L)	229	0.72	0.33
Late HDL (mmol/L)	233	0.87	0.40
Early glucose (mmol/L)	505	4.49	0.35
Late glucose (mmol/L)	485	(4.40)	(4.2–4.7)
Early systolic BP (booking) (mm Hg)	326	111.85	11.11
Early diastolic BP (booking) (mm Hg)	326	66.80	6.45
Late systolic BP (week 34) (mm Hg)	332	112.66	10.38
Late diastolic BP (week 34) (mm Hg)	332	67.58	7.30
Early MAP (booking) (mm Hg)	326	81.81	6.74
Late MAP (week 34) (mm Hg)	420	82.58	7.01
E-DII			
E-DII early pregnancy (trimester 1)	518	0.59	1.60
E-DII late pregnancy (trimester 3)	518	0.67	1.59

Results presented as mean and SD for normally distributed data, median and IQR (25th–75th centile) for nonparametric data, and number (percentage) for categorical data.

BMI, body mass index; *BP*, blood pressure; *E-DII*, Energy-Adjusted Dietary Inflammatory Index; *HDL*, high-density lipoprotein; *IQR*, interquartile range; *LDL*, low-density lipoprotein; *MAP*, mean arterial pressure; *ROLO*, Randomised cOntrol trial of a LOw glycaemic index diet in pregnancy vs no dietary intervention to prevent recurrence of fetal macrosomia.

[a]World Health Organization classification of BMI.

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included early-pregnancy lipid levels within the model to control for change in lipid values from the first to the third trimester. In all analyses, the confidence interval (CI) was set at 95%, and results were considered statistically significant if $P \leq .05$. Statistical analysis was completed using IBM SPSS Statistics, version 26.0 (IBM Corp, Armonk, NY).

Results

Table 1 includes the cohort demographics (n=518); 86.7% of individuals were White Irish, with mean age of 32.8 years at delivery and median BMI of 24.5 kg/m^2. In this cohort, 38.4% had overweight and 16.5% had obesity according to the WHO BMI classification[20] (Table 1). In total, 17.5% (n=91) of women had cesarean delivery. Six preterm deliveries at <37 weeks occurred, with none <32 weeks. In the full cohort (n=759), 4 women in the intervention arm had a primary postpartum hemorrhage of >500 mL, as opposed to 5 in the control group. Mean (SD) pregnancy E-DII scores were 0.59 (±1.60) and 0.67 (±1.59) in early and late pregnancy, respectively. Mean E-DII did not significantly change from early to late pregnancy (P=.23) (Table 2).

Early-pregnancy Energy-Adjusted Dietary Inflammatory Index score and cardiometabolic markers

Maternal lipid levels increased from early to late pregnancy (Table 3). In adjusted linear regression analysis, early pregnancy E-DII was positively associated with early maternal BMI (B=0.007; 95% CI, 0.003–0.011; P=.001), early total cholesterol (B=0.155; 95% CI, 0.061–0.245; P=.001), early triglycerides (B=0.043; 95% CI, 0.005–0.080; P=.03), early LDL (B=0.129; 95% CI, 0.049–0.209; P=.002), and early diastolic BP (B=0.538; 95% CI, 0.070–1.006; P=.02). Early pregnancy E-DII was also positively associated with late-pregnancy total cholesterol (B=0.127; 95% CI, 0.012–0.243; P=.01) and late LDL (B=0.116; 95% CI, 0.011–0.221; P=.03) (Table 4). In adjusted analysis, no associations were observed between

TABLE 2
Change in Energy-Adjusted Dietary Inflammatory Index from early to late pregnancy, ROLO pregnancy study cohort, Dublin, Ireland, from 2007 to 2011

E-DII	Mean	SD	Effect size	*P* value
E-DII trimester 1, time point 1	0.5890	1.597	0.052	.22
E-DII trimester 3, time point 2	0.672	1.594		

Paired sample *t* test was used to test if there was significant difference in E-DII score between trimester 1 and trimester 3. Statistical significance was set at $P \leq .05$.

E-DII, Energy-Adjusted Dietary Inflammatory Index.

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TABLE 3
Change in maternal lipid levels from early to late pregnancy, ROLO pregnancy study cohort, Dublin, Ireland, from 2007 to 2011

Maternal Lipids	n	Mean	SD	Effect size	*P* value
Early total cholesterol (mmol/L)	209	4.597	1.172	1.030	<.001
Late total cholesterol (mmol/L)	209	5.951	1.617		
Early triglycerides (mmol/L)	209	1.100	0.445	1.195	<.001
Late triglycerides (mmol/L)	209	1.823	0.731		
Early LDL cholesterol (mmol/L)	209	3.271	0.944	0.874	<.001
Late LDL cholesterol (mmol/L)	209	4.244	1.261		
Early HDL cholesterol (mmol/L)	209	0.726	0.335	0.991	<.001
Late HDL cholesterol (mmol/L)	209	0.892	0.408		

Paired sample *t* test was used to test for change in maternal lipid levels over pregnancy. Effect size over time is shown. Statistical significance was set at $P \leq .05$.

HDL, high-density lipoprotein; *LDL*, low-density lipoprotein.

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early-pregnancy E-DII and early or late HOMA1-IR (Table 4).

Late-pregnancy Energy-Adjusted Dietary Inflammatory Index score and cardiometabolic markers

In late pregnancy, E-DII was associated with late-pregnancy diastolic BP (B=0.624; 95% CI, 0.103−1.145; P=.02), late HOMA1-IR (B=0.030; 95% CI, 0.005−0.054; P=.02), and late glucose (B=0.003; 95% CI, 0.000−0.006; P=.03) (Table 5). No associations were observed between late-pregnancy E-DII and late-pregnancy lipid profiles. Similarly, no associations were observed between third-trimester E-DII score and late lipid profiles when models were adjusted for increase in lipid levels from early to late pregnancy.

Comment

Principal findings

In early pregnancy, E-DII was positively associated with maternal cardiometabolic biomarkers, namely, early-pregnancy total cholesterol, triglycerides, and LDL, and late LDL and diastolic BP. Early-pregnancy E-DII was also associated with maternal BMI. In late pregnancy, 1-unit increase in maternal E-DII score was associated with 0.62 mm Hg higher diastolic BP. Late-pregnancy E-DII was also associated with late-pregnancy HOMA1-IR and late-pregnancy glucose. E-DII in this cohort was on average proinflammatory and did not significantly change from early to late pregnancy.

Results and clinical implications

Pregnancy can be considered a vascular stress test for future cardiovascular complications.[21,22] The development of pregnancy complications has been shown to predict a woman's risk of premature CVD, which is a leading cause of morbidity and mortality worldwide.[22,23] During pregnancy, individuals undergo substantial changes in lipid and glucose levels, thus ensuring an adequate supply of nutrients to the fetus.[24,25] We observed this increase in lipid levels, with mean total cholesterol, triglycerides, HDL, and LDL showing significant increases from early to late pregnancy.

In recent years, studies have examined associations between dietary patterns and perinatal outcomes such as preeclampsia, GDM and preterm birth[26]; however, few have examined how the inflammatory potential of the diet during pregnancy affects maternal lipid profile.[27,28] Given that maternal cardiometabolic lipid markers during pregnancy have been linked with later maternal cardiometabolic health, investigating factors that influence cardiometabolic markers during pregnancy has the potential to inform interventions in pregnancy that may reduce associated risks for long-term health by moderating the increase in lipid markers.[21] In our analysis, most of the associations observed were between E-DII and early- rather than late-pregnancy lipids. This suggests that the inflammatory potential of the diet in relation to lipids may be more influential in early pregnancy. In a Brazilian prospective cohort study (n=299) investigating prepregnancy dietary patterns and lipid levels, diets high in proinflammatory foods were associated with higher total cholesterol, triglycerides, and LDL throughout pregnancy. Conversely, diets high in fruit, vegetables, and fish were associated with higher HDL cholesterol levels.[29] Evidence suggests that higher-than-expected increase in maternal cholesterol levels during pregnancy is

TABLE 4
Associations between early-pregnancy Energy-Adjusted Dietary Inflammatory Index and early- and late-pregnancy maternal cardiometabolic factors, ROLO pregnancy study cohort, Dublin, Ireland, from 2007 to 2011

Cardiometabolic factors	*B*	95% CI		*P* value	Adjusted R^2
		Lower	Upper		
Maternal BMI (kg/m^2) Log^10	0.007	0.003	0.011	.001	0.046
Early total cholesterol (mmol/L)[a]	0.155	0.061	0.249	.001	0.155
Late total cholesterol (mmol/L)[a]	0.127	0.012	0.243	.01	0.294
Early triglycerides (mmol/L)	0.042	0.005	0.080	.03	0.021
Late triglycerides (mmol/L)	0.044	−0.014	0.103	.14	0.015
Early LDL (mmol/L)	0.129	0.049	0.209	.002	0.050
Late LDL (mmol/)	0.116	0.011	0.221	.03	0.069
Early glucose (mmol/L)	0.014	−0.007	0.036	.19	0.003
Late glucose (mmol/L)	0.003	0.000	0.006	.051	0.010
Early HDL (mmol/L)[a]	0.010	−0.010	0.030	.34	0.481
Late HDL (mmol/L)[a]	0.009	−0.012	0.031	.40	0.589
Early HOMA1-IR Log^10	0.010	−0.013	0.003	.48	0.002
Late HOMA1-IR Log^10	0.009	−0.014	0.033	.44	0.001
Early systolic BP (mm Hg)	0.356	−0.451	1.162	.39	−0.009
Early diastolic BP (mm Hg)	0.538	0.070	1.006	.02	0.006
Late systolic BP (mm Hg)	0.330	−0.401	1.061	.38	−0.014
Late diastolic BP (mm Hg)	0.288	−0.230	0.807	.27	−0.007
Early MAP — booking (mm Hg)	0.477	−0.013	0.967	.06	−0.002
Late MAP — 34 wk (mm Hg)	0.304	−0.204	0.811	.24	−0.011

Multiple linear regression: models adjusted for maternal age at delivery, maternal smoking, original randomized controlled trial allocation group, maternal education, and maternal ethnicity. Nonnormally distributed independent variables were log^10-transformed before analysis. Statistical significance was set at $P \leq .05$.

BMI, body mass index; *BP*, blood pressure; *CI*, confidence interval; *HDL*, high-density lipoprotein; *LDL*, low-density lipoprotein; *MAP*, mean arterial pressure.

[a] Total cholesterol and HDL models adjusted for length of time of sample storage before analysis.

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associated with increased risk of dyslipidemia and CVD postpartum.[30] In an RCT of nonpregnant adults, those consuming diets low in inflammatory foods for 3 months showed a significant reduction in CRP and lipid levels, with a dose-response effect.[27]

In our cohort, 55% of women had overweight or obesity at their first antenatal appointment. This high proportion is consistent with international data.[31] There is strong evidence that inflammation is an important link between overweight and obesity and CVD.[12] Women who have a high BMI have increased risk of pregnancy complications and have higher baseline inflammation.[32] We found that E-DII was positively associated with BMI taken at the first antenatal appointment. This association remained after adjustment for potential confounders, suggesting that lowering the inflammatory potential of the maternal diet, particularly in the first trimester of pregnancy, may benefit BMI. However, more research is needed in intervention studies.

High BP in pregnancy is associated with significant adverse outcomes for the mother, including long-term maternal CVD.[33–36] It has been suggested that inflammation plays a role in BP control.[37–39] A UK pregnancy cohort investigated the association between hypertensive disorders of pregnancy and subsequent diagnosis of 12 various CVDs. Persons with hypertension during pregnancy, when compared with those without hypertension, had increased risk of stroke, cardiac atherosclerotic events, heart failure, cardiovascular mortality, and chronic hypertension as soon as 1 year after pregnancy.[40] Like previous findings,[41] our results showed that early-pregnancy E-DII score was positively associated with early-pregnancy diastolic BP, and late-pregnancy E-DII was positively associated with late-pregnancy diastolic BP. Increased diastolic BP during pregnancy has been associated with preeclampsia[42] which has been shown to be a predictor for future cardiometabolic dysfunction.[40] Incorporating anti-inflammatory foods within the diet during pregnancy may have the potential to improve maternal BP, specifically diastolic BP, and reduce risk of future CVD. However, no associations were observed between maternal E-DII and systolic BP or MAP in either trimester.

In our analysis, third-trimester E-DII score was associated with an increase in late-pregnancy HOMA1-IR and late glucose levels. Maternal inflammation, measured by concentration of TNFα, is associated with increased maternal insulin resistance.[43] A recent prospective study found that increased HOMA1-IR during pregnancy was a risk factor for GDM.[44] In addition to this, insulin resistance has also been linked to preeclampsia.[45] It is important to note that although significant, the increase in late E-DII score was associated with a very small increase of 0.030 units in HOMA1-IR and of 0.003 mmol/L in glucose concentration; future research is needed to investigate the significance of these increases from a clinical perspective.

The Dietary Approaches to Stop Hypertension (DASH) diet has recently emerged as a potential option for pregnant women to optimize maternal body

TABLE 5
Associations between late-pregnancy Energy-Adjusted Dietary Inflammatory Index and late-pregnancy maternal cardiometabolic factors, ROLO pregnancy study cohort, Dublin, Ireland, from 2007 to 2011

E-DII, trimester 3

	B	95% CI		*P* value	Adjusted R^2 value
		Lower	Upper		
Late total cholesterol (mmol/L)[a]	0.065	−0.053	0.183	.28	0.283
Late triglycerides (mmol/L)	0.011	−0.048	0.071	.71	0.006
Late LDL (mmol/L)	0.050	−0.057	0.157	.36	0.023
Late HDL (mmol/L)[a]	0.019	−0.003	0.040	.09	0.593
Late glucose (mmol/L) Log^10	0.003	0.000	0.006	.03	0.011
Late HOMA1-IR Log^10	0.030	0.005	0.054	.012	0.018
Late systolic BP (mm Hg)	0.255	−0.485	0.995	.50	−0.015
Late diastolic BP (mm Hg)	0.624	0.103	1.145	.02	0.006
Late MAP − 34 wk (mm Hg)	0.500	−0.013	1.012	.06	−0.004

Multiple linear regression: models adjusted for maternal age at delivery, maternal smoking, randomized controlled trial group, maternal education, and ethnicity. Nonnormally distributed independent variables were log^{10}-transformed before analysis. Statistical significance was set at $P \leq .05$.

BP, blood pressure; *CI*, confidence interval; *E-DII*, Energy-Adjusted Dietary Inflammatory Index; *HDL*, high-density lipoprotein; *LDL*, low-density lipoprotein; *MAP*, mean arterial pressure.

[a]Total cholesterol and HDL models adjusted for length of time of sample storage before analysis.

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weight,[46] BP,[47,48] inflammation,[49] and insulin resistance.[50] Interestingly, a higher DASH score has been associated with lower inflammatory markers.[51] Elements of the DASH diet align with advice for achieving a low E-DII, including consumption of fruit, vegetables, whole grains, and low-fat dairy while reducing intake of saturated fat, cholesterol, sodium, and red and processed meat.[52] A Mediterranean diet that consists mainly of plant foods, animal foods, and olive oil has been shown to minimize inflammation and benefit maternal and offspring cardiovascular health.[53−55] A prospective cohort study reported a significant inverse relationship between Mediterranean diets and risk of hypertension among women with a history of GDM.[56] In another study, Mediterranean diet score was moderately negatively correlated (ie, r≈−0.50 to −0.70) with the DII.[57] Furthermore, it has been reported that Mediterranean or less inflammatory diets were associated with lower total, CVD, and coronary heart disease mortality.[58] Adopting a healthy dietary pattern in pregnancy may reduce E-DII. A study that examined the impact of a low-GI diet and healthy-eating advice during pregnancy reported significantly lowered E-DII scores in their cohort.[28]

Research implications

Further exploration is required to ascertain whether diets with low E-DII scores can be achieved while meeting dietary recommendations for pregnancy.[28] The findings of our study suggest a need to further analyze the benefits of lowering the inflammatory potential of the diet throughout pregnancy to improve both short- and long-term maternal cardiometabolic health. Our findings suggest that reducing the inflammatory potential of the maternal diet may optimize maternal cardiometabolic markers during pregnancy, with potential to improve postpartum cardiovascular health. Longitudinal data that provide follow-up and monitoring of cardiometabolic risk factors after pregnancy are warranted to determine long-term impact. Because maternal inflammation is also an important determinant of offspring health outcomes,[28,59,60] implications are even more far-reaching.

Strengths and limitations

This study has various strengths. E-DII was used for this analysis, and was available from 3-day food diaries for early and late pregnancy. Our study included a broad range of cardiometabolic risk factor variables and a variety of potential confounders. This study is not without limitations. Sample bias may have been present given the homogeneous nature of this cohort. All mothers were on their second pregnancy, were healthy at study recruitment, and had a median BMI of 24 kg/m^2 at their first antenatal appointment. The sample was predominantly White Irish, and most had attained third-level education or above. Therefore, the sample is not representative of the general population, which may affect the applicability of the findings to the broader population. In the current research, dietary data and E-DII were used at 2 time points to represent early- and late-pregnancy maternal diet. The inclusion of midpregnancy dietary data and E-DII would have strengthened our findings because it would have allowed a more thorough assessment of maternal diet throughout pregnancy. Future research investigating diet throughout gestation, along with before and after pregnancy, may provide further insights into the dynamic longitudinal relationship between dietary intake and maternal lipids during pregnancy. The E-DII is not without limitations. It is calculated on the basis of the effect of individual nutrients on intermediate biomarkers, but does not take into account the effect of overall dietary intake on those biomarkers or assess how each individual component of the score would affect health.

Conclusions

Our findings suggest that diets high in proinflammatory foods and nutrients

during pregnancy were associated with some cardiometabolic risk factors. These findings highlight the potential benefits of consuming diets with overall lower inflammatory potential during pregnancy to improve maternal cardiovascular health. However, more intervention research is needed. ■

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Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.xagr.2023.100214.

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