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Diet During Pregnancy and Risk of Preeclampsia or Gestational Hypertension

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Abstract

PURPOSE: We sought to examine associations of first-trimester intake of calcium, n-3 and n-6 fatty acids, trans fatty acids, magnesium, folate, and vitamins C, D, and E with preeclampsia (PE) and gestational hypertension (GH).

METHODS: We studied associations of diet with PE or GH among 1718 women in the prospective cohort study Project Viva, using logistic regression and adjusting for maternal age, prepregnancy body mass index, first trimester systolic blood pressure, race/ethnicity, education, and parity. We assessed first-trimester diet using a validated semiquantitative food frequency questionnaire.

RESULTS: A total of 59 (3%) women developed PE, and 119 developed (7%) GH. We found a somewhat-lower risk of PE associated with higher intake of the elongated n-3 fatty acids docosahexaenoic and eicosapentaenoic acids (odds ratio [OR] 0.84, 95% confidence interval [95% CI]: 0.69–1.03 per 100 mg/day), fish (OR 0.91, 95% CI 0.75–1.09 per serving/day), and the ratio of docosahexaenoic + eicosapentaenoic to arachadonic acid (OR 0.82, 95% CI 0.66–1.01). We did not observe a lower risk of GH or PE with a greater intake of calcium; vitamin C, D, or E; milk; magnesium; folate; or with lower intake of n-6 or trans fatty acids.

CONCLUSIONS: Our results support a potential benefit for elongated n-3 fatty acids in preventing preeclampsia.

Keywords

Antioxidants; Calcium; Diet; Hypertension—Pregnancy-Induced; n-3 Fatty Acids; Nutrition; Preeclampsia; Pregnancy

INTRODUCTION

Hypertensive disorders of pregnancy, including preeclampsia (PE) and gestational hypertension (GH), are associated with substantial morbidity and mortality for both mother and child. Most established risk factors for PE or GH, including maternal age, race/ethnicity, parity, and previous hypertension or PE, are not modifiable (1). During the past two decades,

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a number of studies have examined whether maternal diet during pregnancy might influence risk for PE or GH.

A systematic review of randomized clinical trials demonstrated a reduction in the relative risk for the development of high blood pressure (0.58, 95% confidence interval [95% CI]: 0.22–0.97) and PE (0.35, 95% CI 0.20–0.60) with supplementation of at least 1 g/day of calcium during pregnancy (2). However, the largest trial, the Calcium for Preeclampsia Prevention trial, did not find a benefit of calcium supplementation on PE or GH (3). Observational studies have suggested potential benefits of other nutrients, including n-3 fatty acids or fish oils (4-6), magnesium (7), and antioxidant vitamins (8), and a potential risk of trans fatty acids (9), based upon either dietary intake or levels of biomarkers among women who develop PE. However, not all observational studies and randomized trials have shown any effect of these nutrients (10-17). In particular, recent trials have not supported a benefit of vitamins C and E (18-20).

Both randomized trials and observational studies are valuable in studying nutrient-outcome associations. Although experimental trials can minimize confounding and demonstrate causality, observational studies can provide information about a range of nutrient intake and about nutrients derived from foods as well as from supplements. The purpose of this observational study was to examine associations of maternal intake of milk, fish, calcium, n-3 and n-6 fatty acids, trans fatty acids, magnesium, folate, and vitamins C, D, and E from both foods and supplements with development of PE or GH.

MATERIALS AND METHODS

Population and Study Design

Study subjects were participants in Project Viva, a prospective observational cohort study of gestational diet and other behaviors, pregnancy outcomes, and offspring health. We recruited women attending their initial prenatal visit at one of eight urban and suburban obstetrical offices in a multi-specialty group practice in eastern Massachusetts from 1999 to 2002. Recruitment and retention details have been summarized previously (21,22). All mothers provided informed consent, and all procedures were in accordance with the ethical standards for human experimentation established by the Declaration of Helsinki (23). Institutional review boards of participating institutions approved the study.

Of 2128 live births in Project Viva, we excluded participants with outpatient medical records not available for review (n = 45), women who did not complete the first-trimester dietary questionnaire (n = 339), women with preexisting chronic hypertension who did not develop PE (n = 24), or women with missing covariate information (n = 2), leaving 1718 (81%) participants available for analysis. Included participants were somewhat more likely to be white (72% vs. 66%) and college graduates (70% vs. 65%) but were similar to the overall cohort in age (93% vs. 93% ages 20–40 years), parity (51% vs. 52% parous), and prevalence of PE (4% vs. 4%) and GH (7% vs. 7%).

We performed study visits after the mothers' prenatal clinical appointments at study recruitment (median 10.4 weeks of gestation) and at midpregnancy (26–28 weeks). Using a combination of questionnaires and interviews, we collected information about maternal race/ethnicity, age, education, parity, education, smoking during pregnancy, household income, medical history, height, prepregnancy weight, and history of pregnancy complications. We obtained results of maternal glucose tolerance testing from the clinical medical record and categorized women as having normal glucose tolerance, impaired glucose tolerance, or gestational diabetes (24). We also obtained from the clinical record the systolic blood pressure recorded at the initial first trimester obstetric clinical visit.

Dietary Assessment

Participants completed semiquantitative food frequency questionnaires (SFFQ) at study enrollment. The SFFQ asked about the average frequency of consumption "during this pregnancy," that is, since the last menstrual period, of more than140 specified foods, as well as additional questions about beverages and supplements. The Project Viva SFFQ was modified for use in pregnancy from a well-validated instrument used in the Nurses' Health Study and other large cohorts, which has been used to study a number of diet-disease relationships, including all nutrients we studied in the present analysis (25,26). Additionally, we previously calibrated the questionnaire for use during pregnancy and found expected correlations with maternal blood levels of elongated n-3 and trans fatty acids and some antioxidants (27). In that study, dietary intake of the parent n-3 fatty acid alphalinolenic acid was not associated with erythrocyte levels. We did not calibrate the SFFQ for calcium, vitamin D, or n-6 fatty acids. To obtain estimates of nutrients, we used the Harvard nutrient composition database, which is based primarily on U.S. Department of Agriculture publications and is continually supplemented by other published sources and personal communications from laboratories and manufacturers, as previously described (22,28). We energy adjusted all estimates of nutrient intake using the nutrient residual method (29). Nutrients of primary interest for this analysis included calcium; the elongated n-3 fatty acids docosahexaeonic acid and eicosapentaenoic acid (DHA+EPA); the parent n-3 fatty acid alpha linolenic acid (ALA); the elongated n-6 fatty acid arachidonic acid (AA); the parent n-6 fatty acid linolenic acid (LA); total trans fatty acids; magnesium; folate; and vitamins C and E. We calculated ratios of elongated (DHA+EPA:AA) and total (ALA:LA) n-3:n-6 fatty acids. We also evaluated consumption of milk, a primary source of calcium, and fish, a primary source of elongated n-3 fatty acids. We report total daily intake (foods + supplements) of each nutrient, and additionally evaluated intake from foods alone for calcium; vitamins C, D, and E; magnesium; and folate.

Definition of Outcomes

We reviewed outpatient charts for blood pressure and urine protein results. We additionally reviewed inpatient hospital charts only for women who had a diagnosis or discharge code indicating PE or GH and who did not already meet criteria for the same diagnosis based upon our review of outpatient charts.

We classified women as having chronic hypertension if they were taking antihypertensive medication or if they had two elevated clinically measured blood pressure values (systolic >140mm Hg or diastolic >90mm Hg) before 20 weeks of gestation. We defined PE and GH according to the recommendations of the National High Blood Pressure Education Program (30). We categorized a woman as having GH if she did not have chronic hypertension and developed elevated systolic (>140 mm Hg) or diastolic (>90 mm Hg) blood pressure on two or more occasions after 20 weeks of gestation. We categorized a woman as having PE if she did not have chronic hypertension but developed increased blood pressure and proteinuria (dipstick value of 1+ on two or more occasions or ≥2+ once) > 4 hours but ≤7 days apart, or if she had chronic hypertension and developed proteinuria after 20 weeks of gestation.

Statistical Analysis

We calculated mean levels of nutrients for women with normal blood pressure, PE, and GH. We determined associations of foods and nutrients with the outcomes using multivariable logistic regression. We included as covariates only those participant characteristics that independently predicted the outcomes, namely maternal age, prepregnancy body mass index, first-trimester systolic blood pressure, race/ethnicity, education, and parity. Inclusion of other factors, including total energy intake, maternal smoking, income, history of PE or GH in previous pregnancy, gestational weight gain, and gestational diabetes mellitus in a past or the current pregnancy, did not change effect estimates for nutrients by >10%, and so we did not

retain these variables in our analyses. We performed all analyses using SAS Version 8.2 (SAS Institute, Cary, NC).

RESULTS

Among the 1718 participants in this study, 59 (3%) developed PE, and 119 developed (7%) GH. Half (49%) were nulliparous. Consistent with other studies, women with PE were more likely to be nulliparous, age < 20 or > 40 years, black, unmarried, less educated, overweight, have higher first-trimester systolic blood pressure, and have a prior history or PE or GH (Table 1). Women who developed GH were more likely to be nulliparous, white, less educated, overweight, have a higher first trimester systolic blood pressure, and have prior PE or GH (Table 1). Gestational diabetes mellitus was more common among women who Developed PE or GH (Table 1).

In Table 2, we list mean daily intake of foods and nutrients (from foods and supplements) among women who developed PE or GH and those who did not. The great majority of women (92%) took prenatal or multivitamins in the first trimester of pregnancy. Women with PE had somewhat lower consumption of DHA+EPA n-3 fatty acids, vitamin C, vitamin E, folate, and magnesium, than women with normal blood pressure. Women with GH had somewhat higher intake of vitamins C, D, and E. Otherwise, intake did not appear to differ substantially between groups.

In Table 3, we present results from multivariable logistic regression analyses, evaluating the effects of foods and energy-adjusted nutrients on risk of PE and GH. Estimates are adjusted for maternal age, race/ethnicity, prepregnancy body mass index, first trimester blood pressure, education, and parity. We did not observe any reduction in risk for PE or GH associated with higher intake of milk or of calcium and vitamin D total (Table 3) or from foods alone (data not shown).

We found a somewhat-lower risk of PE associated with higher intake of the elongated n-3 fatty acids DHA+EPA (odds ratio [OR] 0.84, 95% 95% CI 0.69–1.03 per 100 mg/day), fish (OR 0.91, 95% CI 0.75–1.09 per serving/day), and the ratio of DHA+EPA to arachidonic acid (OR 0.82, 95% CI 0.66–1.01). Intake of n-6 or trans fatty acids, or of the parent n-3 fatty acid ALA, was not associated with risk for PE or GH. Greater intake of magnesium was related to a slightly lower estimated risk for PE, although confidence intervals were broad.

We also observed an increased odds of GH among women with higher total vitamin C, D, or E intake, which is contrary to the hypothesized direction. Intake of these vitamins from foods alone was not associated with GH or PE (data not shown). Exclusion of a few individuals with high intake from supplements eliminated the association of vitamin E, but not vitamin C, with GH (data not shown). Intake of folate either total (Table 3) or from foods alone (data not shown) was not associated with either outcome.

DISCUSSION

In this prospective study, we found no evidence that intake of calcium, folate, or antioxidant vitamins reduced risk for PE or GH or that intake of n-6 or trans fatty acids increased risks. We observed a somewhat reduced risk for PE associated with intake of elongated n-3 fatty acids and fish. The prevalence of and risk factors for GH and PE in our population were similar to those reported in previous studies (30).

The hypothesis that fish oil might be protective against hypertensive disorders of pregnancy dates to observations in the 1980s that elongated n-3 fatty acids result in increased vasodilation and decreased platelet aggregation (31). Women who develop preeclampsia have been found

to have lower levels biochemical markers of n-3 fatty acid intake (4,6). Observational studies of fish intake and randomized trials of fish oil supplementation generally have not supported a protective effect (11,13,16). A more recent study, however, found a U-shaped association, with greater risk of hypertensive disorders among women with the lowest and highest intake of n-3 fatty acids, primarily from cod-liver oil (32). Our results suggest that further investigation into potential benefits of moderate n-3 fatty acid intake might be warranted.

Evidence for an association of calcium intake with risk for PE has been inconsistent. Data from some clinical trials suggest that calcium supplementation may reduce risk primarily among women with low baseline calcium intake or at high risk of hypertensive disorders. Mean calcium intake in the study population was adequate (> 900 mg/day) (2), and we saw no increased risk of PE or GH among those with low as compared with high calcium intake within this range of generally adequate intake. Similarly, intake of vitamins D, E, C, and folate were also realtively high; it is posssible that we might have observed a benefit of these nutrients when compared with much lower intakes. Although magnesium is used as a treatment for established hypertension in pregnancy, dietary or supplemental intake of magnesium has also not been proven protective against the development of hypertensive disorders (13,30).

Extensive in vitro data as well as some in vivo studies have suggested a pathologic role for oxidative stress as a cause of PE and, thus, antioxidants as a possible therapy (33). Whereas one trial among women at high risk for PE demonstrated a >50% risk reduction from supplementation with the antioxidant vitamins C and E (34), more recent trials have not substantiated this finding (18-20). In the current study, we saw some evidence for a direct association of vitamin C and E intake with GH, attributable to intake from supplements only. This association may have been by chance given that the direction was opposite to that hypothesized.

The strengths of the present study include prospective evaluation of both food and nutrient intake during the first trimester of pregnancy with a SFFQ that has been validated for a number of nutrients, including n-3 and trans fatty acids and antioxidants. Additionally, we obtained detailed information on a number of other maternal factors that have been previously shown to be important risk factors for PE or GH. Although we used clinically measured blood pressure and urine values, we applied research criteria to outcome definitions. Even though our overall sample size was relatively large, we might have had insufficient power to detect true associations given the small number of women who developed either outcome in this cohort of women at average risk. It is possible that we did not detect a true association between nutrients and PE or GH because most women took vitamins and had generally adequate intake of all nutrients studied. It is also possible that our SFFQ did not accurately assess dietary intake, especially for nutrients such as ALA and magnesium for which it has not been validated. The study population was generally well educated and relatively older and, thus, results may not be generalizable to other populations.

In conclusion, results from the present study do not support the premise that maternal intake of calcium or vitamins C and E during pregnancy can prevent hypertensive disorders of pregnancy, although they do suggest a potential benefit for elongated n-3 fatty acids in preventing preeclampsia. Future studies targeting women at high risk, or following further elucidation of underlying pathophysiologic mechanisms, may indicate a role for dietary interventions to prevent GH or PE.

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Selected Abbreviations and Acronyms

PE, preeclampsia; GH, gestational hypertension; 95% CI, 95% confidence interval; SFFQ, semiquantitative food frequency questionnaire; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; ALA, alpha linolenic acid; AA, arachidonic acid; LA, linoleic acid; OR, odds ratio.

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TABLE 1

Characteristics of 1718 participants in Project Viva, according to whether they developed preeclampsia, gestational hypertension, or neither (normal blood pressure) during pregnancy

Participant characteristics	Women with normal blood pressure, *n* = 1540 (90%)	Women with preeclampsia, *n* = 59 (3%)	Women with gestational hypertension, *n* = 119 (7%)
	Percent[a]		
Age (years)			
<20	3	7	3
20–<30	25	29	28
30–<40	68	58	66
⩾40	4	7	3
Race/ethnicity			
White	72	59	77
Black	12	29	9
Hispanic	6	7	7
Other	10	5	7
Nulliparous	47	73	66
Married/cohabiting	93	86	94
College graduate	69	58	76
Income			
<$40,000	12	15	8
$40,000–<70,000	22	20	23
⩾$70,000	59	59	63
Missing/don't know	7	5	6
Previous pregnancy history[b]			
Preeclampsia	4	11	14
Gestational hypertension	9	23	37
Gestational diabetes mellitus	3	9	5
Prepregnancy body mass index (kg/m^2)			
<24.9	67	37	48
25.0–29.9	21	27	29
⩾30	12	36	23
Smoked in pregnancy	11	19	12
First trimester systolic blood pressure			
<110 mm Hg	35	19	11
110–130 mm Hg	63	73	81
>130 mm Hg	2	8	8
Glucose status (current pregnancy)			
Normal	84	69	75
Impaired glucose tolerance	12	14	13
Gestational diabetes	4	17	12

[a] Percentages may not total 100% because of rounding.

[b] Among the 1174 (68%) women with a previous pregnancy.

TABLE 2

Maternal first trimester diet, assessed by semiquantitative food frequency questionnaire, among women with normal blood pressure, preeclampsia or gestational hypertension

	Women with normal blood pressure *n* = 1540 (90%)	**Women with preeclampsia *n* = 59 (3%)**	**Women with gestational hypertension *n* = 119 (7%)**
Maternal first trimester diet: Daily intake[a]		**Mean (SD)**	
Total energy (kcal)	2051 (670)	2220 (811)	2048 (673)
Calcium (mg)[b]	1309 (416)	1270 (443)	1317 (440)
Vitamin D (IU)[b]	496 (210)	466 (183)	542 (262)
Milk intake (servings)	1.2 (1.0)	1.3 (1.4)	1.1 (1.0)
DHA + EPA (mg)	184 (316)	146 (144)	185 (222)
ALA (mg)	934 (385)	983 (428)	937 (355)
Total n−3 fatty acids (mg)	1131 (530)	1142 (453)	1137 (419)
Fish (servings)	0.25 (0.24)	0.22 (0.19)	0.27 (0.29)
AA (mg)	93 (33)	93 (31)	93 (32)
LA (mg)	11748 (3112)	11690 (3297)	11779 (3021)
Total n−6 fatty acids (mg)	11857 (3121)	11802 (3308)	11889 (3029)
Trans fatty acids (mg)	2140 (707)	2170 (748)	2180 (615)
n−3:n−6 ratio	0.09 (0.03)	0.10 (0.02)	0.10 (0.02)
DHA + EPA:AA ratio	1.96 (3.68)	1.50 (1.23)	1.86 (1.55)
Vitamin C (mg)[b]	269 (241)	256 (154)	378 (765)
Vitamin E (mg)[b]	31 (60)	29 (46)	41 (92)
Magnesium (mg)[b]	342 (73)	324 (64)	341 (62)
Folate (mcg)[b]	952 (467)	854 (297)	1019 (486)

[a] All estimates of nutrient intake were adjusted for total energy intake using the nutrient residual method (29).

[b] Intake from foods + supplements.

TABLE 3

Adjusted[a] odds of developing preeclampsia or gestational hypertension by maternal first-trimester diet

Maternal first-trimester diet: Daily intake	Preeclampsia vs. normal	Gestational hypertension vs. normal
	Odds ratio (95% confidence interval)	
Calcium (per 300 mg)[b]	1.03 (0.84–1.27)	0.99 (0.85–1.15)
Vitamin D (per 100 IU)[b]	0.99 (0.87–1.13)	1.11 (1.01–1.21)
Milk intake (per serving)	1.25 (1.00–1.57)	0.93 (0.76–1.12)
DHA + EPA (per 100 mg)	0.84 (0.69–1.03)	1.01 (0.95–1.08)
ALA (per g)	1.35 (0.66–2.74)	1.14 (0.68–1.92)
Total n−3 fatty acids (per g)	1.01 (0.55–1.85)	1.13 (0.79–1.61)
Fish (per weekly serving)	0.91 (0.75–1.09)	1.04 (0.94–1.15)
AA (per 100 mg)	0.99 (0.91–1.08)	1.03 (0.97–1.10)
LA (per g)	0.99 (0.91–1.08)	1.01 (0.95–1.08)
Total n−6 fatty acids (per g)	0.99 (0.91–1.08)	1.01 (0.95–1.08)
Trans fatty acids (per g)	0.90 (0.62–1.32)	1.00 (0.76–1.33)
n−3:n−6 ratio	0.99 (0.89–1.11)	1.02 (0.96–1.08)
DHA + EPA:AA ratio	0.82 (0.66–1.01)	0.99 (0.93–1.07)
Vitamin C (per 100 mg)[b]	1.00 (0.85–1.18)	1.07 (1.02–1.11)
Vitamin E (per 100 mg)[b]	1.04 (0.63–1.72)	1.28 (1.01–1.60)
Magnesium (per 100 mg)[b]	0.81 (0.53–1.25)	0.96 (0.71–1.29)
Folate (per 100 mcg)[b]	0.96 (0.89–1.03)	1.02 (0.99–1.06)

[a] Estimates are adjusted for maternal age (<20, 20−<40, 40+ years), prepregnancy body mass index (continuous), first-trimester systolic blood pressure (continuous), race/ethnicity (black, Hispanic, white, other), education (college graduate, < college graduate), and parity (0, 1+). Nutrients are adjusted for total energy intake. Each row represents results from a separate model.

[b] Intake from foods + supplements.