Plasma ω-3 fatty acids in pregnancy are inversely associated with postpartum weight retention in a multiethnic Asian cohort

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ABSTRACT

Background: Studies have demonstrated associations between polyunsaturated fatty acids (PUFAs) and adiposity. It is unclear whether PUFAs in pregnancy have an effect on maternal weight retention after childbirth, which can contribute to long-term obesity.

Objective: We examined the association of maternal plasma PUFAs in pregnancy with 18-mo postpartum weight retention (PPWR) in a multiethnic Asian cohort.

Design: We studied pregnant women (n = 653) recruited between June 2009 and September 2010 from a prospective cohort. At 26–28 wk of gestation, plasma phosphatidylcholine PUFA concentrations were measured and determined as percentages of total fatty acids (FAs). PPWR was calculated based on the difference between measured weight at the first antenatal clinic visit and at 18 mo postpartum.

Results: The median retained weight of women was 0.90 kg (IQR: −1.40, 3.25) at 18 mo postpartum. Of 653 women, 544 women (83.3%) had PPWR of <5 kg and 109 (16.7%) had PPWR of ≥5 kg. In adjusted linear regression models, higher plasma eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA), and total ω-3 (n=3) PUFA concentrations were associated with lower PPWR: EPA: β = −0.62 kg/1% increase of total FAs (95% CI: −1.18, −0.05); DHA: β = −0.24 kg/1% increase (95% CI: −0.45, −0.02); total ω-3 PUFAs: β = −0.20 kg/1% increase (95% CI: −0.36, −0.03), whereas a higher ratio of plasma ω-6-to-ω-3 PUFAs was associated with a higher PPWR [β = 0.21 kg/unit increase (95% CI: 0.05, 0.36)].

Conclusions: Higher plasma percentages of ω-3 PUFAs and a lower ratio of ω-6-to-ω-3 PUFAs in the late-second trimester of pregnancy are associated with less weight retention at 18 mo postpartum. This may offer an alternative strategy to assist postpartum weight reduction by increasing EPA and DHA status together with a decreased ratio of ω-6-to-ω-3 PUFA through diet or fish-oil supplementation during pregnancy.

INTRODUCTION

Obesity prevalence continues to increase, and obesity is a growing burden in women of childbearing age (1). Many women attribute substantial weight gain and fat deposition to childbearing (2). Pregnancy is a life stage that can potentially affect

REFERENCES

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2. Supplemental Tables 1 and 2 are available from the “Online Supporting Material” link in the online posting of the article and from the same link in the online table of contents at http://ajcn.nutrition.org.

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future weight gain trajectory (1, 2). An increase in body weight after pregnancy or postpartum weight retention (PPWR)\(^{20}\) has been reported as a risk factor predisposing women to obesity and related long-term adverse health outcomes (1).

PPWR is referred to the average weight change from preconception until a time point after delivery (1, 3). It includes the weight gain during gestation (preconception through gestation), early postpartum weight loss (delivery to 6 wk postpartum), and later postpartum weight changes (after 6 wk until weight before next pregnancy) (1, 3). Retaining weight of \(\geq 5\) kg above preconception weight at 1–2 y postpartum is considered substantial PPWR (1, 3). PPWR appears to be more physiologically harmful than weight gain during other life periods because the retained body fat is preferentially deposited in central rather than in peripheral sites, thus increasing risk for development of metabolic and cardiovascular disease (1).

Recently, there is growing interest in the role of \(\omega-3\) and \(\omega-6\) PUFAs in adiposity development. Both short-chain [i.e., \(\alpha\)-linolenic acid (ALA)] and long-chain \(\omega-3\) PUFAs [i.e., EPA and DHA] have been shown to reduce adiposity in animal feeding studies (4, 5), but evidence from human studies has been less consistent. Although some observational studies have reported that \(\omega-3\) PUFA levels are inversely associated with body weight or fat mass (6, 7), other studies conducted among Canadian Inuit and Cree Indian populations have shown \(\omega-3\) PUFA levels to be associated with increased abdominal obesity (8, 9). Dietary supplementation studies have also provided conflicting findings, with some studies reporting weight and fat loss after \(\omega-3\) PUFA supplementation (10–12) and others reporting no effect on body weight and fat (13–16). Although \(\omega-6\) PUFAs [i.e., linoleic acid (LA) and arachidonic acid (AA)] have been shown to stimulate adipogenesis in animal studies (17, 18), there is no clear link with obesity in human epidemiologic studies. There has been a large increase in the ratio of \(\omega-6\)-to-\(\omega-3\) PUFAs in the diet from an estimated 1:1 earlier in human evolution to 16:1 or even higher today (19). An increased ratio of \(\omega-6\)-to-\(\omega-3\) PUFAs has been associated with increased risk of obesity in humans (19).

A few studies have investigated maternal PUFAs in relation to infant body weight at birth, but none examined the association with the body weight of mothers (20). In this study, we examined the associations of maternal plasma PUFA concentrations during pregnancy with PPWR. We hypothesized that higher maternal plasma concentrations of \(\omega-3\) PUFAs and a lower ratio of \(\omega-6\)-to-\(\omega-3\) PUFAs in the late-second trimester of pregnancy would be associated with decreased 18-mo PPWR.

METHODS

Study design and participants

Data were drawn from the GUSTO (Growing Up in Singapore Towards healthy Outcomes) prospective cohort study (clinicaltrials.gov, NCT01174875) (21). This study was conducted according to the guidelines laid down in the Helsinki Declaration. Ethical approval was obtained from the Domain Specific Review Board of Singapore National Health Care Group (reference D/09/021) and the Centralised Institutional Review Board of SingHealth (reference 2009/280/D).

Pregnant women attending antenatal care (<14 wk of gestation) from June 2009 to September 2010 in KK Women’s and Children’s Hospital or National University Hospital were recruited. These women were aged \(\geq 18\) y and had homogeneous parental ethnic groups (Chinese, Malay, or Indian). Women who became pregnant again before 18 mo postpartum were excluded from this analysis. Informed, written consent was obtained from all women.

Data collection

Detailed interviews and measurements were conducted in the clinics at recruitment and at 26–28 wk of gestation. Data on socioeconomic status, educational attainment, obstetric history, smoking status, physical activity, and fish-oil supplementation were collected. Smoking exposure was defined as current smoking or exposure to second-hand smoke at home and/or at work on a daily basis. Physical activity during pregnancy was assessed by using a structured interviewer-administered questionnaire that was designed based on 3 types of activities: light-moderate–, moderate–, and vigorous-intensity activities. Examples for each type of activity were provided to help women recall their activities or exercise in the past 6 mo. The total score of physical activity was computed from the summation of the duration (in minutes) and frequency (days) of these activities, which was expressed in metabolic equivalent task–minutes per week (22, 23).

Data on mode of infant feeding were collected through interviewer-administered questionnaires at 3 and 6 mo postpartum. At each interview, women were asked to classify the types of infant feeding, i.e., 1) exclusive or predominant (only breast milk and water were given), 2) partial (a mixture of breast milk and formula milk was given), or 3) no breastfeeding (only formula milk was given). In this analysis, we defined breastfeeding as those women who fed their infants via method 1 for the entire first 6 mo postpartum, formula feeding was defined as those women who fed their infants via method 3 for the entire first 6 mo postpartum, and mixed breastfeeding was defined as those women who fed their infants via method 2 or those who did not meet the criteria for breastfeeding and formula feeding in the first 6 mo postpartum (24).

Dietary assessments

A 24-h dietary recall was administered face-to-face by trained clinical staff at 26–28 wk of gestation using the 5-stage, multiple-pass interviewing technique (25). Standardized household measuring utensils and food pictures of various portion sizes were used to assist women in quantifying their food and beverage intakes. Total daily energy intake was assessed by using nutrient analysis software (Dietplan; Forestfield Software) with a food composition database of locally available foods (26). For food items not found in the database, nutrient information was obtained from either food labels or the USDA national nutrient database (27).

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\(^{20}\)Abbreviations used: AA, arachidonic acid; ALA, \(\alpha\)-linolenic acid; GDM, gestational diabetes mellitus; GUSTO, Growing Up in Singapore Towards healthy Outcomes; GWG, gestational weight gain; LA, linoleic acid; PPWR, postpartum weight retention.
Anthropometric measurements

Maternal height was measured to the nearest 0.1 cm by using a Seca 213 Portable Stadiometer (SECA) at 26–28 wk of gestation. Self-reported prepregnancy weight and measured weight at the first antenatal visit (±14 wk of gestation) were collected. BMI (in kg/m²) was calculated as weight divided by the square of height. Because maternal BMI at the first antenatal visit was strongly correlated with prepregnancy BMI ($r = 0.96; P < 0.001$) and not subject to recall bias, it was used for analyses in this study. Serial measurements of maternal weight throughout pregnancy were collected from the medical records. Linear mixed-effects model with the best linear unbiased predictor was used to estimate the linear trajectory of gestational weight gain (GWG) per week between 15 and 35 wk of gestation for each individual (28). Total GWG was not computed because not all women had weight data close to their delivery (within 4 wk of delivery). Maternal weights at ±14 wk of gestation and 18 mo postpartum were measured to the nearest 0.1 kg by using an electronic weighing scale (SECA). PPWR was calculated as the difference between measured weight at ±14 wk of gestation and measured weight at 18 mo postpartum.

Plasma glucose and plasma phosphatidylcholine FA analyses

At 26–28 wk of gestation, maternal fasting blood samples were collected for plasma glucose and PUFA analyses. At the same visit, women underwent a 75-g oral glucose-tolerance test for the diagnosis of gestational diabetes mellitus (GDM). Plasma glucose concentrations at 0 and 120 min after the oral glucose load were measured by colorimetry [ADVIA 2400 Chemistry system (Siemens Medical Solutions Diagnostics) and Beckman LX20 Pro analyzer (Beckman Coulter)]. GDM was diagnosed according to the 1999 WHO criteria: ≥7.0 mmol/L for fasting glucose and/or ≥7.8 mmol/L for 2-h postglucose (29).

Analysis of plasma phosphatidylcholine FAs has been described elsewhere (30). Briefly, lipid extraction was carried out with chloroform/methanol (Fisher Scientific), and phosphatidylcholine was separated by solid-phase extraction. After purification and extraction, phosphatidylcholine FA methyl esters were separated by gas chromatography (BPX-70 column mounted on a Hewlett-Packard HP6890) and detected by flame ionization. Plasma phosphatidylcholine concentrations of FAs were expressed as percentages of total FAs. For all FAs identified in plasma phosphatidylcholine, inter- and intraassay variation coefficients were <6% and <3%, respectively. In this study, we examined the percentages of ALA, EPA, DHA, LA, AA, total ω-3 PUFAs, total ω-6 PUFAs, and the ratio of ω-6-to-ω-3 PUFAs.

Statistical analysis

Categorical data are presented as frequencies and percentages, whereas continuous data are presented as means ± SDs. Comparisons between maternal characteristics and PPWR were performed by using Pearson’s chi-square test for categorical variables and independent t test for continuous variables. Multiple regression analysis was performed to examine the association between individual maternal plasma phosphatidylcholine PUFAs and PPWR in continuous form. Binary logistic regression analysis was performed to examine the association between individual maternal plasma phosphatidylcholine PUFAs and PPWR in categorical form. Normal and substantial PPWR were defined as <5 and ≥5 kg, respectively.

In the main adjusted model, we controlled for maternal age, education, ethnicity, parity, GDM, physical activity, total energy intake, smoking exposure during pregnancy, and early-pregnancy BMI, which were selected a priori based on literature review (1–3). We additionally controlled for GWG per week and mode of infant feeding in the main adjusted model. These 2 factors were not included in our main adjusted model because they may be in the causal pathway between maternal PUFAs (measured at midgestation) and PPWR, which could result in overadjustment, but additional analyses were conducted to examine for any potential mediating effect. To examine the contributing role of fish-oil supplementation on maternal PUFA status in relation to PPWR, we further adjusted for maternal fish-oil supplementation.

PPWR was computed based on the change score (post minus baseline score), which has the advantage of being more intuitive to interpret than an absolute value. The use of a change score as a dependent variable in regression analysis without adjusting for baseline score as a covariate is equivalent to assuming that the coefficient of regressing the post score (dependent variable) on the baseline score is 1 (31). This assumption of coefficient = 1 is unrealistic because a higher baseline score tends to associate with a lower change score. Analysis of the change score with adjustment for the baseline score as a covariate removes this unrealistic assumption (31). Hence, baseline BMI, which serves a function similar to baseline weight, was adjusted.

Missing values for maternal education ($n = 7$), gestational diabetes ($n = 35$), GWG per week ($n = 64$), physical activity ($n = 10$), smoking exposure ($n = 2$), total daily energy intake ($n = 7$), fish-oil supplement intake ($n = 57$), and mode of feeding ($n = 38$) were imputed 100 times by using multiple imputation analyses by chained equation (32). The results of the 100 analyses were pooled by using Rubin’s rule (33). A sensitivity analysis was performed by including only women with a complete dataset for all covariates ($n = 508$). All point estimates were presented with 95% CIs. All statistical analyses were performed by using IBM SPSS statistics, version 19, or Stata-Corp Stata Statistical Software, release 13.

RESULTS

Participant characteristics

Of 1152 enrolled women with singleton pregnancies, 126 women (10.9%) became pregnant again before 18 mo postpartum, leaving 1026 women (89.1%) who were eligible for this study. Of those, 920 women had early-pregnancy weight data, and 821 had an adequate volume of plasma for analysis of phosphatidylcholine PUFAs. At 18 mo postpartum, 168 women were lost to follow-up or missed their 18-mo visit. A final sample of 653 women (63.6%) was included in the present analysis (Figure 1). In comparison to excluded women ($n = 373, 36.4%$), those included were found to be older ($P < 0.001$), be multiparous ($P = 0.001$), be less likely to have been exposed to cigarette smoke during pregnancy ($P = 0.005$), and practice breastfeeding or mixed feeding ($P = 0.011$). No statistically significant differences in maternal characteristics were observed.
for education, ethnicity, GDM, early-pregnancy BMI, GWG per week, physical activity, total daily energy intake, and fish-oil supplement intake between included and excluded women.

Table 1 shows the characteristics of women categorized by normal and substantial PPWR. The median PPWR for all women (n = 653) was 0.90 kg (IQR: −1.40, 3.25 kg); 544 (83.3%) women had normal PPWR (median: 0.30 kg; IQR: −1.20, 1.95 kg), and 109 (16.7%) had substantial PPWR (median: 7.00 kg; IQR: 5.63, 8.80 kg). Women with substantial PPWR were younger (P < 0.001), were more likely to belong to the Malay or Indian ethnic group (P = 0.002), were primiparous (P < 0.001), were less likely to have GDM (P < 0.001), had higher early-pregnancy BMI (P = 0.003), had higher GWG per week (P < 0.001), and had a lower tendency to take fish-oil supplements during pregnancy (P = 0.012). There were no differences between the groups with regard to education levels, physical activity levels, smoking exposure, total daily energy intake, and mode of feeding. In comparison with women with normal PPWR, women with substantial PPWR had lower plasma phosphatidylcholine percentages of EPA (P < 0.001), DHA (P = 0.020), total ω-3 PUFAs (P = 0.001), and AA (P = 0.024), but they had a higher plasma phosphatidylcholine ratio of ω-6-to-ω-3 PUFAs (P = 0.012). Plasma phosphatidylcholine percentages of ALA, LA, and total ω-6 PUFAs were not different between the 2 groups of women.

**Plasma phosphatidylcholine PUFAs concentrations and PPWR**

Table 2 shows the linear regression models of individual maternal plasma phosphatidylcholine concentration of PUFAs with 18-mo PPWR. After adjustment for confounders (model 2), plasma phosphatidylcholine EPA, DHA, and total ω-3 PUFAs during pregnancy were inversely associated with PPWR [EPA: β= −0.62 kg (95% CI: −1.18, −0.05 kg); DHA: β= −0.24 kg (95% CI: −0.45, −0.02 kg); total ω-3 PUFAs: β= −0.20 kg (95% CI: −0.36, −0.03 kg)], whereas the plasma phosphatidylcholine ratio of ω-6-to-ω-3 PUFAs was positively associated with PPWR (β= 0.21 kg; 95% CI: 0.05, 0.36). No associations were seen for ALA or individual and total ω-6 PUFAs. When adjustments for GWG per week and mode of feeding were conducted in the additional analyses (model 3), the degree of associations remained similar. When further adjustment was made for fish-oil supplementation (model 4), there was generally an attenuation of the associations between maternal plasma phosphatidylcholine PUFAs and 18-mo PPWR. Effect sizes of PPWR were reduced by 20–25% for plasma phosphatidylcholine EPA, DHA, total ω-3 PUFAs, and ratio of ω-6-to-ω-3 PUFAs, indicating fish-oil supplementation contributed to approximately one-quarter of plasma phosphatidylcholine ω-3 PUFAs.

**Table 3** shows the logistic regression models of individual maternal plasma phosphatidylcholine PUFAs concentrations with the risk of retaining substantial postpartum weight. As indicated in model 2, women with higher plasma phosphatidylcholine EPA, DHA, and total ω-3 PUFAs concentrations during pregnancy had a lower likelihood of retaining ≥5 kg weight at 18 mo postpartum after confounders adjustment [EPA: OR = 0.48 (95% CI: 0.26, 0.91); DHA: OR = 0.84 (95% CI: 0.71, 0.99); total ω-3 PUFAs: OR = 0.84 (95% CI: 0.75, 0.98)]. The associations of plasma phosphatidylcholine AA and the ratio of ω-6 to ω-3 with the risk of retaining substantial postpartum weight were attenuated after adjustment for confounders. Plasma phosphatidylcholine concentrations of ALA, LA, and total ω-6 PUFAs were not significantly associated with PPWR in the adjusted models.

In sensitivity analyses based on women with a complete dataset (n = 508), results remained similar for the outcomes by using PPWR as a continuous ([Supplemental Table 1]) or categorical variable ([Supplemental Table 2]) in relation to plasma phosphatidylcholine ω-3 PUFAs concentrations.

**DISCUSSION**

In this multiethnic cohort, 1 in 6 women (16.7%) was found to have substantial weight retention (≥5 kg) at 18 mo postpartum with significantly higher rates found in Malay and Indian women than in Chinese women. We showed that higher maternal plasma phosphatidylcholine percentages of ω-3 PUFAs and a lower plasma phosphatidylcholine ratio of ω-6-to-ω-3 PUFAs at 26–28 wk of gestation were significantly associated with lower 18-mo PPWR after adjusting for demographic and health covariates. This association was largely driven by EPA and DHA rather than ALA. ω-6 PUFAs, such as LA, AA, and total ω-6 PUFAs, were not related to PPWR. GWG per week and mode of infant feeding in the first 6 mo postpartum did not seem to mediate the associations. Overall, our data suggest that increased maternal plasma long-chain ω-3 PUFAs in pregnancy may play a role in reducing weight retention during the postpartum period.

Our findings are consistent with existing epidemiologic studies (6, 34) and nutritional intervention trials that use EPA and DHA supplementation (10–12) in nonpregnant populations, showing that increased plasma EPA and DHA concentrations were associated with decreased body weight. A recent study involving...
Higher plasma concentration of total and body fat (34). Another cross-sectional study revealed that a DHA (3-PUFA) supplementation (e.g., fish or fish oil) on weight loss have also been observed in energy-restricted overweight or obese individuals (10, 11). These findings suggest that long-chain ω-3 PUFAs have a role in weight regulation. In contrast, some intervention studies indicate no relation between ω-3 PUFA supplementation and obesity (14, 15). This, however, may be attributable to study power, confounding factors, ω-3 PUFA dose, proportions of EPA and DHA used, and interindividually variations in the responses to fish-oil supplements.

Multiple mechanisms have been proposed to explain the effects of long-chain ω-3 PUFAs on obesity. In animal models, EPA and DHA have been shown to counteract obesity through suppression of hepatic lipogenesis (35), stimulation of fat oxidation (36), and enhancement of energy expenditure (37), which in turn could suppress fat synthesis and deposition. EPA and DHA may also reduce adiposity by improving gut health through a reduction of oxidative stress and inflammation (38). An in vitro study found that EPA has a greater anti-inflammatory
TABLE 2
Maternal plasma phosphatidylcholine PUFAs in pregnancy and PPWR at 18 mo (n = 653)

<table>
<thead>
<tr>
<th>PUFAs, %</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β (95% CI)</td>
<td>P</td>
<td>β (95% CI)</td>
<td>P</td>
</tr>
<tr>
<td>ALA</td>
<td>0.32 (–1.95, 2.60)</td>
<td>0.781</td>
<td>0.19 (–2.00, 2.39)</td>
<td>0.863</td>
</tr>
<tr>
<td>EPA</td>
<td>–0.99 (–1.55, –0.43)</td>
<td>0.001</td>
<td>–0.62 (–1.18, –0.05)</td>
<td>0.032</td>
</tr>
<tr>
<td>DHA</td>
<td>–0.26 (–0.48, –0.04)</td>
<td>0.021</td>
<td>–0.24 (–0.45, –0.02)</td>
<td>0.030</td>
</tr>
<tr>
<td>Total ω-3</td>
<td>–0.25 (–0.42, –0.08)</td>
<td>0.004</td>
<td>–0.20 (–0.36, –0.03)</td>
<td>0.022</td>
</tr>
<tr>
<td>LA</td>
<td>0.05 (–0.04, 0.15)</td>
<td>0.265</td>
<td>0.02 (–0.07, 0.11)</td>
<td>0.698</td>
</tr>
<tr>
<td>AA</td>
<td>–0.14 (–0.32, 0.05)</td>
<td>0.145</td>
<td>–0.03 (–0.22, 0.16)</td>
<td>0.763</td>
</tr>
<tr>
<td>Total ω-6</td>
<td>0.06 (–0.03, 0.16)</td>
<td>0.198</td>
<td>0.05 (–0.04, 0.14)</td>
<td>0.289</td>
</tr>
<tr>
<td>Ratio of ω-6 to ω-3</td>
<td>0.24 (0.08, 0.40)</td>
<td>0.003</td>
<td>0.21 (0.05, 0.36)</td>
<td>0.011</td>
</tr>
</tbody>
</table>

1 Data were analyzed by using multiple linear regressions. Model 1 was unadjusted; model 2 was adjusted for maternal age, education, ethnicity, parity, gestational diabetes, early-pregnancy BMI, physical activity, smoking exposure, and total energy intake; model 3 was adjusted as for model 2 and additionally for gestational weight gain per week and mode of infant feeding; model 4 was adjusted as for model 2 and additionally for fish-oil supplementation. AA, arachidonic acid; ALA, α-linolenic acid; LA, linoleic acid; PPWR, postpartum weight retention.

An increased ratio of ω-6 to ω-3 in our current diet, primarily because of a shift toward high use of vegetable oils, has been implicated in causing greater adipose tissue accumulation and as a potential contributor to the rise in obesity prevalence (19). A longitudinal study in 534 normal-weight women showed that a high ratio of ω-6-to-ω-3 PUFAs in red blood cell membrane phospholipids was associated with an increased risk of weight gain (7). Although a positive association between the plasma phosphatidylcholine ratio of ω-6-to-ω-3 PUFAs and PPWR was observed in our study, this is most likely due to the high concentrations of ω-3 PUFAs because no associations were noted between plasma phosphatidylcholine ω-6 PUFAs and PPWR.

We recognized and considered the following limitations. The measured weight at the first antenatal clinic visit was used as the baseline to compute PPWR, which could underestimate PPWR. However, the first-trimester weight has been shown to be a sufficiently accurate measurement to represent prepregnancy weight, where weight change is minimal at this stage (41). Weight at the end of pregnancy was not available, which restricted our ability to confirm whether the association observed was mediated by gestational weight gain.

TABLE 3
Maternal plasma phosphatidylcholine PUFAs in pregnancy and risk of PPWR ≥5 kg at 18 mo (n = 653)

<table>
<thead>
<tr>
<th>PUFAs, %</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>P</td>
<td>OR (95% CI)</td>
<td>P</td>
</tr>
<tr>
<td>ALA</td>
<td>1.42 (0.35, 5.82)</td>
<td>0.624</td>
<td>1.58 (0.34, 7.34)</td>
<td>0.563</td>
</tr>
<tr>
<td>EPA</td>
<td>0.37 (0.20, 0.67)</td>
<td>0.001</td>
<td>0.48 (0.26, 0.91)</td>
<td>0.025</td>
</tr>
<tr>
<td>DHA</td>
<td>0.84 (0.72, 0.97)</td>
<td>0.020</td>
<td>0.84 (0.71, 0.99)</td>
<td>0.039</td>
</tr>
<tr>
<td>Total ω-3</td>
<td>0.83 (0.74, 0.94)</td>
<td>0.003</td>
<td>0.85 (0.75, 0.98)</td>
<td>0.020</td>
</tr>
<tr>
<td>LA</td>
<td>1.03 (0.97, 1.10)</td>
<td>0.317</td>
<td>1.01 (0.94, 1.08)</td>
<td>0.867</td>
</tr>
<tr>
<td>AA</td>
<td>0.86 (0.75, 0.98)</td>
<td>0.023</td>
<td>0.87 (0.74, 1.03)</td>
<td>0.089</td>
</tr>
<tr>
<td>Total ω-6</td>
<td>1.00 (0.94, 1.07)</td>
<td>0.923</td>
<td>0.98 (0.92, 1.05)</td>
<td>0.564</td>
</tr>
<tr>
<td>Ratio of ω-6 to ω-3</td>
<td>1.13 (1.05, 1.24)</td>
<td>0.013</td>
<td>1.10 (0.98, 1.22)</td>
<td>0.096</td>
</tr>
</tbody>
</table>

1 Data were analyzed by using binary logistic regressions. Model 1 was unadjusted; model 2 was adjusted for maternal age, education, ethnicity, parity, gestational diabetes, early-pregnancy BMI, physical activity, smoking exposure, and total energy intake; model 3 was adjusted as for model 2 and additionally for gestational weight gain per week and mode of infant feeding; model 4 was adjusted as for model 2 and additionally for fish-oil supplementation. AA, arachidonic acid; ALA, α-linolenic acid; LA, linoleic acid; PPWR, postpartum weight retention.
through excessive GWG. However, our current findings should remain valid because the adjustment for GWG per week did not attenuate the association between maternal plasma phosphatidylcholine PUFAs and PPWR. Maternal body composition was not measured, so changes in fat mass and lean mass could not be examined. Plasma phosphatidylcholine PUFAs were measured at a single point in time during pregnancy without assessment of any change over time. No measurements of body weight and no diet assessments were made in the 18-mo follow-up period. Some differences in characteristics (i.e., age, parity, smoking exposure, and mode of feeding) were noted between included and excluded women, which could introduce a potential selection bias that affected generalizability of the results to a wider population. However, we controlled for these variables in the statistical analysis. Additionally, our study recruited Asian participants, which may limit the generalizability of our findings to populations of other ethnic groups.

The present findings should be interpreted cautiously. By recognizing that plasma concentrations of FAs are useful biomarkers to reflect habitual dietary intake (42), it is tempting to speculate that increased intakes of ω-3 PUFAs (e.g., from fish or fish oil) during pregnancy may help control weight retention after giving birth. However, we cannot exclude the possibility that lower plasma or dietary ω-3 PUFAs and a higher PPWR may both be secondary to other independent factors, such as poor-quality diet or lack of health awareness in women. Nevertheless, our analysis included covariates such as education that should reflect these factors. The measurements of ω-3 and ω-6 PUFAs were made in plasma phosphatidylcholine, which is not as good a long-term marker of PUFA status as erythrocyte membrane phospholipids. This may result in exposure misclassification and lead to underestimation of the true associations. Although various potential confounding factors were considered and adjusted for in our analyses, there might be unadjusted or residual confounding factors that remain.

In summary, this study is the first demonstration to our knowledge that higher maternal plasma phosphatidylcholine percentages of EPA, DHA, and total ω-3 PUFAs and a lower ratio of ω-6-to-ω-3 PUFAs in the late-second trimester of pregnancy are associated with less weight retention at 18 mo postpartum. At present, recommendations to reduce PPWR are mainly based on dietary energy restriction and increased physical activity (43, 44), which are less likely to be complied with by many women. Our results may offer an alternative strategy to assist postpartum weight reduction by increasing EPA and DHA status together with a decreased ratio of ω-6-to-ω-3 PUFAs through diet or fish-oil supplementation during pregnancy. However, more nutritional intervention trials are required to confirm the effects of maternal dietary and plasma PUFAs in pregnancy as well as in the postpartum period on later weight regulation and metabolic outcomes.

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