Directly Quantitated Dietary (n-3) Fatty Acid Intakes of Pregnant Canadian Women Are Lower than Current Dietary Recommendations

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ABSTRACT During pregnancy, (n-3) PUFA are incorporated into fetal brain and retinal lipids. Docosahexaenoic acid [DHA, 22:6(n-3)], in particular, is required physiologically for optimal development and function of the central nervous system. Maternal intake of (n-3) PUFA must be sufficient to maintain maternal tissues stores and meet fetal accrual. Recommendations for pregnant women include an Acceptable Macronutrient Distribution Range (AMDR) of 0.6–1.2% of energy for (n-3) PUFA intake in the current Dietary Reference Intakes, and ≥300 mg/d of DHA suggested by the International Society for the Study of Fatty Acids and Lipids working group. The present study directly quantitated the (n-3) PUFA intake, including DHA, of pregnant, Canadian women (n = 20) in their 2nd and 3rd trimester. Fatty acid intakes were quantitated in triplicate by lipid extraction and GLC of 3-d duplicate food collections calibrated with an internal standard before homogenization. Total fat intakes were also estimated using dietary analysis software from simultaneous 3-d food records to corroborate biochemical analyses. The mean (n-3) PUFA intake was 0.57 ± 0.06% of energy, with 65% of the women below the AMDR. The mean DHA intake was 82 ± 33 mg/d, with 90% of the women consuming ≤300 mg/d. Nutritional education of pregnant women to ensure adequate intakes of (n-3) PUFA for optimal health of mother and child and the inclusion of DHA in prenatal vitamins may be pertinent. J. Nutr. 135: 206–211, 2005.

KEY WORDS: • (n-3) fatty acids • dietary intakes • pregnant women • direct quantitation • docosahexaenoic acid

The (n-3) PUFA are essential for normal neural development and functioning. Mammalian studies including humans indicate that (n-3) PUFA, specifically docosahexaenoic acid [DHA, 22:6(n-3)], become incorporated into the membrane phospholipids of retina and brain (1–6) mainly during the 3rd trimester of pregnancy and continue to accrue until 18 mo postpartum (7). Salem et al. (8) recently reviewed animal and human studies investigating the role and mechanism of DHA on brain and retinal functions. Low levels of plasma and erythrocyte DHA were associated with poor retinal development, low visual acuity, and poor cognitive developmental performances as measured by behavioral (9–12), electrophysiological (13–17), and olfactory testing (18,19). In addition, (n-3) PUFA have a significant role in gestation and parturition (20–22).

Fetal accretion of (n-3) PUFA in humans was estimated at 67 mg/d during the 3rd trimester (23). It was demonstrated in fetal baboons that maternal α-linolenic acid [α-LNA, 18:3(n-3)] and DHA are sources of fetal brain DHA (24), but that preformed DHA is more readily accreted (25). The conversion of dietary α-LNA to DHA may be elevated in women consuming low amounts of (n-3) highly unsaturated fatty acids (HUFA, ≥20 carbons, ≥3 double bonds) in their diet (26,27), but it was estimated to be <4% by stable isotope techniques (28,29).

In 1990, Health and Welfare Canada recommended in the Recommended Nutrient Intakes (RNI) that adults should consume at least 0.5% of energy as α-LNA (20) in their 2nd trimester of pregnancy of 1.4 g/d and an Acceptable Macronutrient Distribution Range (AMDR) of 0.6–1.2% of energy for (n-3) PUFA with up to 10% of the AMDR consumed as eicosapentaenoic acid [EPA, 20:5(n-3)] and/or DHA (31). Before the DRI recommendations, a working group from the International Society for the Study of Fatty Acids and Lipids (ISSFAL) recommended intakes of 1.0% of energy for α-LNA and 0.3% for EPA + DHA [1.3% for total (n-3) PUFA] for adults and further recommended a minimum intake of 300 mg/d of DHA for pregnant and lactating women (32).
A number of studies have estimated the intake of essential fatty acids [including (n-3) PUFA] in nonpregnant (33–42) and pregnant (43–48) Western populations based mainly on food recall, FFQ, and food disappearance data, which rely on indirect quantitation for (n-3) PUFA content of foods by food composition tables. This approach has several limitations (39,49); in addition, the extensive hydrogenation of some (n-3) enriched vegetable oils (e.g., soybean and canola) in most consumer products often eliminates them as substantial sources of α-LNA. The direct quantitation of fatty acid consumption, including (n-3) fatty acids, can be assessed by biochemical analysis as done in human experimental diets (29) and in 3-d duplicate food collections of free-living, nonpregnant subjects (39). The present investigation is the first to directly quantify the dietary intake of (n-3) PUFA in a sample of pregnant, Canadian women. Individual fatty acid intakes were determined by lipid extraction and GLC (triplicate analyses per food collection) of 3-d duplicate food collections from each participant. The results are compared with current recommendations for North Americans and with previous indirect measurements.

SUBJECTS AND METHODS

Subjects. All procedures received prior review and approval by the University of Guelph Human Subjects Committee. Pregnant women (n = 20) were recruited through an electronic mail message to University of Guelph faculty, staff, and graduate students, electronic mail bulletin to faculty and students of the neighboring University of Waterloo, and personal communication to prenatal exercise and childbirth classes in the surrounding area. Candidates expressing an interest in participating in the study were met individually for an informational session.

Food collection and dietary records. Participants were not informed of the particular nutrient under investigation to prevent adjustments of their typical food consumption. On 3 separate days, participants collected a duplicate of all food and beverages consumed. Daily food collections were stored in either the refrigerator or freezer until collection the next day for storage at the University of Guelph. Participants also completed a 3-d dietary record using household measures on the duplicate collection days. All vitamin and nutritional supplements were collected into the duplicate sample and recorded in the food record during the experimental period to ensure that an accurate account of dietary (n-3) PUFA was obtained. Food records were checked by a nutritionist, and analyzed using CanWest Diet Analysis Plus software (West Publishing). To corroborate energy intake values calculated by the CanWest Diet Analysis Plus software program, gross energy of the duplicate food collections was analyzed by bomb calorimeter (Parr Adiabatic Calorimeter).

Biochemical analysis of (n-3) PUFA intake. Food duplicate collections were analyzed biochemically for (n-3) PUFA content by lipid extraction and GLC. The 3-d duplicate collections of total food and beverage of each subject were combined and blended to homogeneity using a high volume Waring blender (VWR Scientific). The total weight of oil was recorded and divided by 3 to quantify daily amounts. A representative sample of the mixture (~0.25 g) was measured into a test tube and the exact mass recorded. The procedure was performed in triplicate to validate the results. To each test tube, 100 µL of concentrated internal standard containing 20 g triheptadecanoin/L (Nuchek Prep) was added. Lipid extraction using the method of Folch et al. (50) was performed. Briefly, 5 mL of chloroform/methanol (2:1, v:v) was added and each test tube was mixed on a vortex for 2 min to guarantee full degradation and reaction of the sample and solvent. To remove nonlipid contaminants, samples were mixed on a vortex with 1 mL of 0.188 mol/L potassium chloride for 30 s, left to sit for 30 min, and then centrifuged at 1000 × g for 25 min at 4°C. The lower lipid and chloroform phase was removed and a second extraction of the sample was performed with 3.33 mL of chloroform. The lipid extractions were combined and percolated through a pipette stuffed with a small amount of cotton and ~2.5 cm of sodium sulfate anhydrous (Na2SO4) to remove moisture and contaminants. A 200-µL aliquot of lipid extract was dried under nitrogen on a 50°C sand bath and transmethylated with 3 mL of 6% (by volume) H2SO4 in methanol at 80°C for 3 h. After cooling, the FAME were collected after the addition of 2 mL of petroleum ether and 1 mL of deionized H2O, and centrifugation at 1000 × g for 15 min at 4°C. Samples were percolated through a pipette of cotton and Na2SO4 a second time and then stored at −20°C until analysis. FAME were analyzed by GLC (HP 5890 Series II; Hewlett Packard) using an approach similar to that described previously (51).

Statistical analyses. All data are expressed as means ± SEM and analyses were completed with SPSS for Windows statistical software (release 11.5.1; SPSS). Paired t tests and Pearson’s correlations were used to compare and examine the relation between the 3-d food record estimates and the biochemical quantitations of fat and energy intakes. Spearman’s nonparametric correlations were used to examine relations between the directly quantitated, dietary fatty acids. Differences were considered significant when P < 0.05.

RESULTS

Demographic information. All pregnant women (n = 20) recruited for the study completed the 3-d duplicate food collection and 3-d food record; selected participant characteristics were recorded (Table 1). Women had completed 27.7 ± 1.5 wk of gestation, with all women in their 2nd or 3rd trimester of pregnancy. The women were 28.6 ± 0.8 y old and their height and weight were 1.65 ± 0.02 m and 74.3 ± 3.5 kg, respectively. Recruitment of the participants was 85% through prenatal class visits and 15% through broadcast email message; therefore, the majority of the subjects were likely primigravidas. Further optional demographic information was supplied by 85% of the participants. Of the responding participants; 11% had completed high school, 65% university or college, and 24% postgraduate education; 29% smoked before their pregnancy; and 53% noted that their typical diet changed significantly upon pregnancy.

Food record collection and analysis. Estimated dietary intakes from 3-d food records were calculated and biochemical quantitations for fat and energy intakes were determined (Table 2). The estimated percentage of energy from carbohydrate, protein, and fat were within the AMDR. The estimated intake of dietary fiber of 26.0 ± 2.6 g/d was slightly below the AI value of 28 g/d. The directly analyzed values for total fat intake were significantly lower than the estimated fat values. The total fat intake by direct quantitation was 45.1 ± 3.1 g/d, whereas 70.6 ± 5.8 g/d was estimated from food records. The discrepancy was due to significant overestimations largely of saturated fat content (26.4 ± 2.2 vs. 15.3 ± 0.9 g/d, P < 0.001) and to a lesser extent of monounsaturated fat content (24.6 ± 2.2 vs. 20.0 ± 1.6 g/d, P < 0.01) and polyunsaturated fat content (13.3 ± 1.6 vs. 9.8 ± 0.9 g/d, P < 0.01) of food products by the CanWest Diet Analysis Plus software program compared with direct quantitation. Neither the weight of the food consumed per day nor the total energy

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>1 Values are means ± SEM, n = 20.</th>
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<tbody>
<tr>
<td>Age, y</td>
<td>28.6 ± 0.8</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.65 ± 0.02</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>74.3 ± 3.5</td>
</tr>
<tr>
<td>Pregnancy BMI, kg/m²</td>
<td>27.2 ± 1.0</td>
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<tr>
<td>Pregnancy duration, wk</td>
<td>27.7 ± 1.5</td>
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</table>
intake differed between the food record estimate and the direct measurements. Both the estimated total energy intake (9.55 ± 0.51 MJ/d) and the bomb calorimetry quantitated energy intake (9.05 ± 0.37 MJ/d) were lower than the Estimated Energy Requirement of 10.74 MJ/d for these women as determined by calculations in the DRI for energy (31), suggesting some energy restriction during the experiment. All of the food intake estimates correlated significantly (P < 0.01) with the biochemical quantitations.

The participants indicated that they consumed 0.7 ± 0.2 fish meals/wk when surveyed regarding fish consumption patterns. Potential DHA sources reported in the food records included 9 subjects consuming some form of fish and/or seafood, whereas 10 subjects reported eating poultry and 10 subjects reported eating eggs. Two of the subjects did not consume seafood, poultry, or eggs, and several subjects consumed low amounts of these potential DHA-containing foods. The types of fish and number of subjects consuming each type were as follows: tuna, 6 subjects; salmon, 3 subjects; haddock, 2 subjects; sardines, 1 subject; and shrimp, 1 subject. The subject with the highest biochemically quantitated DHA intake reported consuming ~85 g of salmon, 143 g of poultry, and 275 g of eggs. Identifying specific sources of α-LNA is this study is difficult. One subject reported consuming 2.5 slices of flax bread and using canola oil, whereas 75% of the subjects reported consuming leafy green vegetables (mainly romaine lettuce). A significant proportion of the α-LNA intake was likely soybean oil and canola oil as a component of margarine and prepared foods.

Direct quantitation of dietary fatty acids. The biochemical analyses of individual dietary fatty acids were determined as a percentage of total fatty acids, mg/d of fatty acid, and a percentage of total energy (Table 3). Unlike the food record estimates, when fat intakes were quantitated directly, monounsaturated fatty acids (MUFAs) were the largest source of dietary fat (43.6 ± 1.0%), rather than SFA (35.2 ± 1.7%). PUFA intake was 21.2 ± 1.1% with considerably more (n-6) PUFA (18.1 ± 0.9%) than (n-3) PUFA (3.1 ± 0.3%). Approximately 90% of dietary fat intake was accounted for by individual fatty acids, i.e., palmitic acid (16:0, ~22%); stearic acid (18:0, ~10%); oleic acid [18:1 (n-9), ~41%]; and linoleic acid [18:2 (n-6), ~17%].

In gram amounts, total (n-3) PUFA intake was 1.45 ± 0.18 g/d and (n-6) PUFA intake was 8.35 ± 0.77 g/d in the study participants. The ratio of (n-6) to (n-3) PUFA was 6.3 ± 0.4. α-LNA was the major (n-3) PUFA, with an intake of 1.30 ± 0.16 g/d, representing 89% of the total (n-3) PUFA intake. The intakes of EPA and DHA were 35 ± 19 and 82 ± 33 mg/d, respectively. The dominant dietary (n-6) PUFAs were linoleic acid (8.00 ± 0.75 g/d) followed by minor contributions from arachidonic acid [AA, 20:4 (n-6)] (99 ± 19 mg/d) and docosapentaenoic acid (n-6) [DPAn-6, 22:5 (n-6)] (114 ± 10 mg/d). Linoleic acid accounted for 96% of the total (n-6) PUFA intake. Total (n-3) PUFA was 0.57 ± 0.06% and (n-6) PUFA was 3.4 ± 0.3% of total energy intakes.

Individual (n-3) fatty acid intakes and current recommendations. Recommendations for (n-3) PUFA as a percentage of energy include the RNI of 0.5%, the AMDR of 0.6–1.2% of energy, and the ISSFAL working group recommendation of 1.3%. Only 50% of the participants met the RNI value, which was designed to cover 97.5% of the Canadian population, and only 35% of the participants met the lower limit of the more recent AMDR (Fig. 1). None of the participants in this study met the ISSFAL working group recommendation for (n-3) PUFA intake. The ISSFAL working group recommendation of 300 mg/d of DHA was met by 10% of the participants in this study, and 20% of the participants had only trace amounts (<2 mg/d) of DHA intake after triplicate analysis (Fig. 2).

### Table 2

<table>
<thead>
<tr>
<th>Dietary intake</th>
<th>3-d food record estimates</th>
<th>Biochemical quantitations</th>
<th>Paired differences</th>
<th>Correlation</th>
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<tbody>
<tr>
<td>Carbohydrate</td>
<td>57.8 ± 1.7</td>
<td>ND</td>
<td>ND</td>
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<tr>
<td>% of energy</td>
<td>15.1 ± 0.5</td>
<td>ND</td>
<td>ND</td>
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<tr>
<td>Protein</td>
<td>87.1 ± 5.1</td>
<td>ND</td>
<td>ND</td>
<td></td>
</tr>
<tr>
<td>% of energy</td>
<td>27.3 ± 1.7</td>
<td>18.1 ± 1.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10.0 ± 1.4</td>
<td>0.59&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fat</td>
<td>70.6 ± 5.8</td>
<td>45.1 ± 3.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>25.4 ± 4.2</td>
<td>0.69&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>% of energy</td>
<td>10.6 ± 0.8</td>
<td>6.2 ± 0.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.4 ± 0.5</td>
<td>0.80&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Saturated fat</td>
<td>26.4 ± 2.2</td>
<td>15.3 ± 0.9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>11.1 ± 1.6</td>
<td>0.79&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>% of energy</td>
<td>9.7 ± 0.6</td>
<td>8.0 ± 0.6&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.8 ± 0.4</td>
<td>0.84&lt;sup&gt;d&lt;/sup&gt;</td>
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<tr>
<td>Monounsaturated fat</td>
<td>13.3 ± 1.6</td>
<td>9.8 ± 0.9&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.5 ± 1.1</td>
<td>0.75&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>% of energy</td>
<td>5.2 ± 0.5</td>
<td>3.9 ± 0.4&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.3 ± 0.4</td>
<td>0.73&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Polysaturated fat</td>
<td>26.0 ± 2.6</td>
<td>2.19 ± 0.10</td>
<td>0.10 ± 0.08</td>
<td>0.77&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Dietary fiber, g</td>
<td>2.29 ± 0.12</td>
<td>9.05 ± 0.37</td>
<td>0.50 ± 0.33</td>
<td>0.77&lt;sup&gt;d&lt;/sup&gt;</td>
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</table>

<sup>1</sup> Values are means ± SEM, n = 20. <sup>a,b</sup> Indicate biochemical quantitation was significantly different than food record estimate by paired t test, *P* < 0.001; <sup>c,d</sup> Indicate significant Pearson’s correlation between 3-d food estimates and biochemical quantitation, *c P* < 0.01; *d P* < 0.001. ND, not determined.

<sup>2</sup> Food record analysis determined by CanWest Diet Analysis Plus software (West Publishing Company, St. Paul, MN).

<sup>3</sup> Quantitative analysis determined by total lipid extraction followed by GLC (fat) and by bomb calorimetry (energy).
Individual percentages of EPA + DHA in total (n-3) PUFA were calculated from energy percentage data and the mean was 7.1 ± 2.4%; however, 65% of the participants had values <5% and 20% had values >10%. Two subjects had EPA + DHA intakes that were 32.5 and 39.5% of total (n-3) PUFA, respectively.

**Correlations between directly quantitated fatty acids.** Spearman’s correlations were used to examine the relations of the directly quantitated fatty acids with each other. The intake of α-LNA was highly correlated with total (n-3) PUFA intake \((r = 0.93, P < 0.001)\) but also with total PUFA, (n-6) PUFA, linoleic acid, total MUFA \((r\text{-values} \approx 0.64, P\text{-values} \approx 0.002)\), and total saturates to a lesser degree \((r = 0.51, P = 0.022)\). DHA intake was highly correlated with intakes of 22:2(n-6), 22:5(n-3) and 20:4(n-3) \((r\text{-values} \approx 0.58, P\text{-values} \approx 0.007)\) and also significantly correlated with intakes of EPA, AA, and 24:1 \((r\text{-values} \approx 0.49, P\text{-values} \approx 0.03)\). DHA was not correlated with either total (n-3) PUFA or α-LNA. Also, none of the other dietary (n-3) PUFAs were correlated with α-LNA intake, except 18:4(n-3), which was negatively correlated \((r = -0.50, P = 0.026)\). These results suggest that α-LNA is somewhat ubiquitous, although at a low amount, in the dietary fat of the North American food supply, and is associated with longer chain (n-3) PUFA such as DHA.

**DISCUSSION**

The primary objective of this study was to quantitate (n-3) PUFA intakes of pregnant, Canadian women and compare these to the current recommendations for North American pregnant women. The importance and mechanisms of action of DHA in the central nervous system were reviewed (8), and the requirements of PUFA intakes during pregnancy were discussed recently (43,52). Previous (n-3) PUFA intake estimates in pregnant Western women vary and are based on either food records or FFQ. In these studies, daily intakes of α-LNA were estimated to be between 1.0 and 1.6 g/d and intakes of DHA between 48 and 300 mg/d (43,45–48). In nonpregnant Japanese women, dietary intakes of α-LNA are similar and were estimated to be 1.6 g/d; however DHA intake estimates were much higher at 571 mg/d (53).

In the current study, intakes of 1.3 ± 0.2 g/d of α-LNA and 82 ± 33 mg/d of DHA were determined by direct quantitation of duplicate food collections. These values are similar to esti-
mated intakes of 1.4 g/d of α-LNA and 100–200 mg/d of EPA + DHA (117 ± 51 mg/d EPA + DHA in current study) for the United States as determined by food disappearance data (36). Innis and Elias (43) recently estimated a higher intake of DHA (160 ± 20 mg/d) in pregnant Canadian women by FFA; however, that study was located in the Pacific coastal city of Vancouver and subjects reported consuming 1.5 seafood meals/wk with 5.0% DHA in plasma phospholipid fatty acids. The seafood consumption in the present study (0.7 ± 0.2 meals/wk) agrees with previous seafood intake reports for Guelph area, premenopausal women at 0.7 seafood meals/wk with a corresponding 3.2% DHA in plasma phospholipid fatty acids (54).

Energy intake as determined by bomb calorimetry and corroborated by food records was 84% of the calculated Estimated Energy Requirement for the participants in this study, and suggests that energy restriction during the study may have occurred. In addition, the participants were highly educated and 80% recorded taking a prenatal vitamin supplement during their pregnancy. This indicates that the participants, as a group, were health conscious, a factor that was associated previously with energy underreporting (55). Adjusting intakes to the Estimated Energy Requirement would increase the α-LNA intake to 1.5 g/d and the DHA intake to 97 mg/d. This adjustment had no effect on the number of individual participants failing to meet the DHA intake recommendations by the ISSFAL working group (Fig. 2). Lower intakes of (n-3) PUFAs and DHA in particular were demonstrated in women of lower socioeconomic status (45).

The bioconversion rate for α-LNA to DHA in humans is controversial. Recently, Pawlosky et al. (29,56) estimated the bioconversion of plasma α-LNA to DHA to be <0.2% using a kinetic compartmental model. Emken et al. (28) estimated the rate to be 3.8% for young adult males. It was also suggested that women of childbearing age have a greater capacity to convert α-LNA to DHA (26,27). The capacity of pregnant women to convert α-LNA to DHA has not been examined, but concentrations of DHA in plasma were shown to be elevated during pregnancy (52,57). Differences in (n-3) PUFA conversion capacity between genders and during pregnancy may be mediated by hormonal factors and possibly estrogen in particular, as demonstrated in recent studies examining fatty acids and hormone replacement therapy (54,58). On the basis of measurements in baboons (24), 0.45% energy of α-LNA or 0.03% of energy of DHA is required to meet growing fetal brain requirements. In the current study, 55% of the participants had α-LNA intakes of <0.45% energy (1.1 g/d), and 75% of the subjects had DHA intakes < 0.03% energy (72 mg/d).

Specific recommendations for DHA intakes during pregnancy for North Americans are limited to those made by the ISSFAL working group of a minimum of 300 mg/d (32). The recent DRI recommendations (31) differ little from the RNI recommendations made in 1990 for (n-3) PUFA intakes (30). For specific (n-3) fatty acids, the DRIs limits EPA and/or DHA intakes to 10% of the AMDR for α-LNA. Based on the Estimated Energy Requirement for the participants of this study, the AMDR recommendations would result in a limit of 170–350 mg/d of EPA and/or DHA. In contrast, the American Heart Association recommended EPA and/or DHA intakes of 1 g/d to reduce coronary heart disease risk and 2–4 g/d to lower triglycerides (59).

The discrepancy between food record fat intakes and directly quantitated fat intakes in the present study was not expected and is difficult to interpret. This may be a limitation of the dietary software used, but could indicate that dietary fat estimates in nutrient databases may be inaccurate. This may be due to the dynamic nature of the food supply with the introduction of food products such as low-fat foods and the substitution of monounsaturated fats for saturated fatty acids in processed foods.

A link between maternal DHA intake and infant DHA was established, and the potential benefits of DHA sufficiency in preterm and full-term infants were reviewed (8). DHA supplementation was also demonstrated to increase gestation length (22), and low fish consumption was identified as a risk factor for preterm delivery and low birth weight (21). Health benefits for the mother as a result of increased (n-3) PUFA intake must also be considered because a higher prevalence of postpartum depression has been associated with decreased DHA content in mother’s milk and lower seafood consumption (60). These observations were confirmed because women identified as “possibly depressed” on a depression questionnaire had a lower ratio of DHA/ EPA (n-6) in plasma phospholipid compared with nondepressed women (61).

The (n-3) PUFA intakes of pregnant women reported herein raise concerns and implications for public health. The richest sources of EPA and DHA are found in fatty fish such as herring, mackerel, sardines, and salmon (300–2100 mg of EPA + DHA/100 g) (62). Poultry (30–70 mg/100 g) and eggs (~40 mg/100 g) also provide small, but useful amounts of DHA (62). People who do not eat meat and fish, such as vegans and vegetarians, must rely on much higher intakes of α-LNA by consumption of flax (18 g/100 g of seed, 53 g/100 g of oil) and green leafy vegetables such as lettuce (39–113 mg/100 g), broccoli (21–129 mg/100 g), and spinach (27–165 mg/100 g) (62). English walnuts (9 g/100 g), walnut oil (10 g/100 g), canola oil (9 g/100 g), and soybean oil (7 g/100 g) contain α-LNA, but also contain high amounts of linoleic acid (62).

Correlations between dietary fatty acids in this study indicated that α-LNA is associated with overall fat intake, but DHA is not. There is a need to create greater awareness, educational programs, and counseling regarding DHA intake targeted at women planning to become pregnant. Optimal nutrition during pregnancy must include the regular consumption of foods high in (n-3) PUFA and EPA + DHA. Recommendations specific for DHA during pregnancy and the addition of (n-3) PUFA and DHA in particular to prenatal vitamins may be pertinent for Western societies. 

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LITERATURE CITED


