

# Intakes of essential n-6 and n-3 polyunsaturated fatty acids among pregnant Canadian women<sup>1-3</sup>

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## ABSTRACT

**Background:** Fetal growth requires n-3 docosahexaenoic acid (DHA), which is derived from the essential n-3 fatty acids in the maternal diet. DHA is accumulated in the developing brain and is critical for normal neural and visual function. Available estimates suggest that 67 mg DHA/d is accumulated by the fetus during the third trimester of gestation. Little is known about n-3 fatty acid intakes in pregnant women, although human milk concentrations of DHA have decreased in recent years.

**Objective:** We prospectively determined the n-3 and n-6 fatty acid intakes of 55 pregnant Canadian women.

**Design:** A food-frequency questionnaire was completed at 28 and 35 wk, and plasma n-3 and n-6 fatty acids were measured at 35 wk gestation. The fatty acid composition of  $\approx$ 500 foods was analyzed to allow analysis of dietary intakes from specific foods.

**Results:** Intakes, as a percentage of energy, were ( $\bar{x} \pm$  SEM) total fat,  $28.0 \pm 3.6\%$ ; saturated fat,  $9.8 \pm 0.3\%$ ; monounsaturated fat,  $11.2 \pm 0.4\%$ ; polyunsaturated fat,  $4.7 \pm 0.2\%$ ; linoleic acid,  $3.9 \pm 0.2\%$ ; and  $\alpha$ -linolenic acid,  $0.54 \pm 0.05\%$ . The daily intakes (range) were  $160 \pm 20$  (24–524) mg DHA/d,  $121 \pm 8$  (15–301) mg arachidonic acid/d, and  $78 \pm 2$  (4–125) mg eicosapentaenoic acid/d. The plasma phospholipids had (mg/100 g fatty acid)  $5.0 \pm 0.18$  DHA,  $8.7 \pm 0.18$  arachidonic acid, and  $0.52 \pm 0.32$  eicosapentaenoic acid.

**Conclusion:** The low intake of DHA among some pregnant women highlights the need for studies to address the functional significance of maternal fat intakes during pregnancy on fetal development. *Am J Clin Nutr* 2003;77:473–8.

**KEY WORDS** Essential fatty acids, docosahexaenoic acid, arachidonic acid, fish intakes, pregnancy, fetal growth, brain development, women

## INTRODUCTION

Docosahexaenoic acid (DHA; 22:6n-3) is accumulated in fetal tissues, particularly the central nervous system (1, 2). Available estimates suggest that  $\approx$ 67 mg n-3 fatty acids/d is accumulated in fetal tissue during the third trimester of gestation (2). Estimates for the amounts of n-3 fatty acids accumulated in placental and maternal tissue are not available. Because n-3 fatty acids cannot be formed by animal cells, all of the n-3 fatty acids accumulated in fetal tissues must originate from the maternal diet. Reduced brain and retinal DHA results in decreased visual function and altered learning, behavior, and neurotransmitter metabolism (3–6). Clinical studies showed that a dietary source of DHA

increases the early development of visual acuity and other indexes of neurodevelopment in premature infants (7, 8), showing sensitivity of the third trimester human brain to the supply of DHA. DHA is higher and linoleic acid (LA; 18:2n-6) is lower in fetal than in maternal plasma (9, 10), which could suggest selective transfer of DHA from mother to fetus. Despite this, the maternal dietary intake and plasma concentrations of DHA directly influence the DHA status of the developing fetus (9, 11–13). Further, recent studies have reported more mature electroencephalography patterns in newborn infants with higher plasma phospholipid DHA (12).

DHA can be formed in the liver from the dietary essential fatty acid  $\alpha$ -linolenic acid (ALA; 18:3n-3) (14). Stable isotope tracer studies suggest that only <1–4% of dietary ALA is converted to DHA (15, 16), raising the question of the possible importance of dietary DHA in humans. In this regard, several studies showed that dietary DHA results in higher levels of DHA in tissue phospholipids and higher fetal DHA accretion than does its ALA precursor (4, 17–19). Higher intakes of ALA also fail to increase plasma DHA in infants and adults (20, 21). Arachidonic acid (AA; 20:4n-6), which is formed by desaturation and elongation of LA, is also accumulated in fetal tissues (1, 2) and appears to fulfill the role of n-6 fatty acids in growth (22). LA and ALA are present in vegetable fats and oils, whereas DHA and AA are present only in animal tissue lipids, with concentrations of DHA and its precursor eicosapentaenoic acid (EPA, 20:5n-3) being particularly high in fatty fish (23).

Saturated fat intakes have declined from 18–20% to  $\approx$ 11% of total energy over the past 3–4 decades, and meat consumption has decreased in North America (24, 25). The effect of these dietary trends on the intakes of n-3 fatty acids, particularly DHA by pregnant women, is not known. Human milk concentrations of DHA, however, appear to have decreased by  $\approx$ 50% in Canada and Australia during the past 15 y (26–28). Whether this reflects a decline in n-3 fatty acid intakes is not known. In the present

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study, we determined the intakes of LA, AA, ALA, DHA, and EPA with a validated food-frequency questionnaire (29), with corroboration of the differences in intake through biochemical analysis of plasma phospholipid fatty acids in 55 pregnant women. Because of the limited data on the AA, EPA, and DHA in foods, we also analyzed the fatty acid composition of  $\approx 500$  foods and used this to calculate dietary fatty acid intakes.

## SUBJECTS AND METHODS

### Subjects

This was a prospective study of women identified, without knowledge of dietary practices or family background, from registrations for low-risk delivery at the British Columbia Women's Hospital. Eligible women were 20–40 y of age, 12–16 wk gestation, expected to deliver a full-term (37–42 wk) single-birth infant between June 1 and July 19, 1998, with no known history of disease such as diabetes, cardiac disease, renal disease, tuberculosis, HIV-AIDS, hepatitis, previous pregnancy complications, or substance abuse. Of 174 women contacted, 23 did not meet the eligibility criteria, and 70 agreed to participate. These 70 women were part of a larger study on dietary fat intakes, including *trans* fatty acids (9, 29). The 55 women reported in the present study are all those who completed dietary assessments at both 28 and 35 wk gestation and provided a blood sample at 35 wk gestation. The study protocol was approved by the University of British Columbia's Clinical Screening Committee for Research and Other Studies Involving Human Subjects and the British Columbia Women's Hospital Research Coordinating Committee. All the subjects provided written informed consent.

### Dietary assessment

We used a validated food questionnaire that contained 105 food categories to determine the fat and fatty acid intakes of pregnant women over 4 wk (29). To address reproducibility and the possibility of changes in dietary intakes the food questionnaire was given at both 28 wk and 35 wk gestation. The food questionnaire was completed in an interview format with a trained dietitian with the aid of food models, scales, cups, and spoons. The same dietitian completed all of the dietary interviews. Information was collected on the specific food (eg, cow, goat, soy, or other milk and the milk fat content); the frequency with which each food was eaten; the portion size, brand name, or place of purchase; the method of preparation (eg, roasting, grilling, frying); and the types of margarine, shortenings, and other fats and oils. We analyzed the fatty acid composition of  $\approx 500$  local foods, including different meats; poultry; wild, farmed, and canned fish; shellfish and crustaceans; eggs; dairy products; bakery products; snacks; prepared meals; margarine; shortenings; and fats and oils. Total lipids were extracted and the fatty acid composition of the food analyzed by using gas liquid chromatography with 100-m capillary columns (9, 26, 29). The fatty acid composition of each food, identified by food and brand name, was entered into a nutrient database (FOOD PROCESSOR 11, version 7; ESHA Research, Salem, OR). Total energy intakes and the intakes of total fat and individual fatty acids were calculated from the portion size and frequency of food consumption. The contribution of fish, meats, poultry, and eggs to the intakes of AA, EPA, and DHA was also calculated.

### Blood collection and fatty acid analysis

Venous blood was collected from all the subjects after they had fasted overnight into tubes containing 0.1% EDTA. The plasma

**TABLE 1**

Use of fat-reduced milks, dietary fats, oils, meat, fish, and eggs by study subjects<sup>1</sup>

Food	No. of women <i>n</i> (%)
Milk	
Whole	1 (1.8)
2% fat	12 (21.8)
1% fat	16 (52.7)
Skim	23 (41.8)
Soy or rice	4 (7.2)
Table spread	
Butter	25 (45.4)
Margarine, nonhydrogenated	17 (30.9)
Margarine, hydrogenated	13 (23.6)
None	8 (14.5)
Cooking or salad oil	
Canola	33 (60.0)
Olive	31 (56.4)
Corn	1 (1.8)
Safflower or sunflower	6 (10.9)
Other <sup>2</sup>	3 (5.4)
Meat	50 (90.9)
Fish and seafood	50 (90.9)
Poultry	52 (94.5)
Eggs <sup>3</sup>	49 (89.1)

<sup>1</sup>*n* = 55. More than one milk, table spread, or oil was used by some subjects.

<sup>2</sup>Includes sesame and other nut oils. None of the subjects reported that they used soybean oil in cooking or salad dressings.

<sup>3</sup>Used in home food preparation but not in purchased baked or other commercially prepared foods.

was separated from blood cells by centrifugation at  $1216 \times g$  for 15 min at 4 °C, stored at  $-80$  °C, and the fatty acid composition of phospholipids determined (9, 18, 19, 21).

### Statistical analysis

Results are presented as means  $\pm$  SEMs, unless otherwise stated; *n* = 55 for all measures. Two-sample paired *t* tests and one-way analysis of variance were used to test for differences in the mean intakes of fat and individual fatty acids calculated from the food-frequency questionnaires given at 28 and 35 wk gestation. Regression analysis was used to determine the relation between the dietary intakes of LA, AA, EPA, and DHA and the concentrations of AA, EPA, and DHA in the plasma phospholipids and included adjustment for total energy intake. Pearson correlation coefficients were calculated to determine the relation between fish intake and plasma lipid fatty acids. A standard serving size was 85 g edible (cooked) food and for eggs, 1 egg. All analysis was done with SPSS for WINDOWS (version 10; SPSS Inc, Chicago). The level of significance used was 0.05.

## RESULTS

The mean age of the women was 32.5 y (range: 20–40 y). The mean ( $\pm$  SE) body weight before pregnancy was  $62.8 \pm 2.8$  kg and  $70.9 \pm 2.7$  and  $74.8 \pm 2.6$  kg at 28 and 35 wk gestation, respectively. Of the 55 women in our study, 1 ate fish but no meat or poultry, 2 ate fish and poultry but no meat, and 2 ate no fish, meat, or poultry (**Table 1**). Six women did not eat eggs, and 5 ate no fish

**TABLE 2**  
Daily intakes of total fat and individual n-6 and n-3 fatty acids<sup>1</sup>

	Intake
Energy (kJ)	10931 ± 345 (5230–17712)
Fat (g)	
Total	79.8 ± 3.6 (29.7–142.4)
Saturated	28.0 ± 1.3 (7.6–59.6)
Monounsaturated	29.2 ± 1.5 (7.6–65.6)
Polyunsaturated	13.4 ± 0.6 (4.3–28.3)
Fatty acids	
Linoleic (18:2n-6) (g)	11.2 ± 0.4 (2.9–24.2)
α-Linoleic (18:3n-3) (g)	1.6 ± 0.10 (0.16–3.6)
Arachidonic (20:4n-6) (mg)	121 ± 8 (15–301)
Eicosapentaenoic (20:5n-3) (mg)	78 ± 2 (4–125)
Docosahexaenoic (22:6n-3) (mg)	160 ± 20 (24–524)

<sup>1</sup> $\bar{x} \pm \text{SEM}$ , calculated as the average intake of each subject determined from 2 dietary assessments; range in parentheses.  $n = 55$ .

or seafood. Most of the women used canola oil (49%), olive oil (56%), or both; soybean oil was not used in home food preparation. The food-specific fatty acid composition data were entered into the nutrient analysis program for each woman and used for analysis of each food frequency.

The energy intakes from fat and fatty acids estimated from the first and second food-frequency questionnaires were not different ( $P > 0.05$ , data not shown). The correlation coefficients between the energy intakes from fat, saturated fat, monounsaturated fat, and polyunsaturated fat calculated from the 2 questionnaires were  $r = 0.69, 0.75, 0.64$ , and  $0.61$ , respectively. Thus, mean intakes for the group were calculated from the average of the 2 dietary assessments for each woman. The mean intakes were  $10931 \pm 345$  kJ/d, with  $28.0 \pm 3.6\%$  of total energy from fat, and  $9.8 \pm 0.3, 11.2 \pm 0.4, 4.7 \pm 0.2$ , and  $1.4 \pm 0.06\%$  of energy from saturated fat, monounsaturated fat, polyunsaturated fat, and *trans* fatty acids, respectively (Table 2). The mean intake of LA was  $11.2 \pm 0.4$  g/d ( $3.9 \pm 0.2\%$  total energy) and of ALA was  $1.62 \pm 0.10$  g/d ( $0.54 \pm 0.05$  total energy). The mean intakes of AA, EPA, and DHA were  $121 \pm 8, 78 \pm 2$ , and  $160 \pm 20$  mg/d, respectively.

The mean intake, in servings/wk, of fatty fish (predominately Pacific salmon) was  $0.4 \pm 0.07$ ; lean fish (predominately sole, cod, halibut, and tuna canned in water),  $0.8 \pm 0.09$ ; shellfish and

crustaceans (predominately shrimp and prawns),  $0.3 \pm 0.06$ ; meat (beef, pork, and lamb, including cured meats),  $2.5 \pm 0.2$ ; and poultry,  $2.1 \pm 0.18$ . The average use of eggs was  $2.3 \pm 0.25$ /wk per person. Fish and other seafoods contributed  $\approx 80\%$  of dietary DHA and  $65\%$  of EPA (Table 3). Meat and poultry contributed  $\approx 80\%$  of dietary AA,  $32\%$  and  $9\%$  of dietary EPA and DHA, respectively, and eggs contributed  $27\%$  of AA,  $1\%$  of EPA, and  $10\%$  of DHA.

The plasma phospholipids of the women at 35 wk gestation had  $20.5 \pm 0.42$  g LA,  $0.36 \pm 0.02$  g ALA,  $8.7 \pm 0.18$  g AA,  $0.52 \pm 0.32$  g EPA, and  $5.0 \pm 0.18$  g DHA/100 g fatty acids. The mean daily intakes of DHA and EPA were significantly related to the concentrations of DHA and EPA in the plasma phospholipids (Figure 1). The concentrations of DHA and EPA in the plasma phospholipids also increased with increasing intakes of fish (Table 4). This significant relation was explained by the relation between the intake of fatty fish and the concentrations of DHA ( $r = 0.61, P < 0.0001$ ) and EPA ( $r = 0.55, P < 0.0001$ ) in the plasma phospholipids rather than by a relation between the intake of lean fish and the concentrations of DHA ( $r = 0.29, P = 0.03$ ) and EPA ( $r = 0.22, P > 0.05$ ) in the plasma phospholipids. There was no significant relation between fish intake and the concentration of LA, ALA, or AA in the plasma phospholipids (Table 4) or between the intake of meat and poultry and the concentration of AA in the plasma phospholipids (data not shown). The intake of AA was not significantly related to the concentration of AA in the plasma phospholipids (Figure 1). However, the concentration of AA in the plasma phospholipids decreased with increasing LA intake. The concentrations of AA in the plasma phospholipids for women with the lowest to highest tertiles of LA intake were  $9.5 \pm 0.3$  ( $n = 18$ ),  $8.5 \pm 0.3$  ( $n = 26$ ), and  $7.5 \pm 0.2$  ( $n = 11$ ) g/100 g fatty acids, respectively,  $P < 0.01$ .

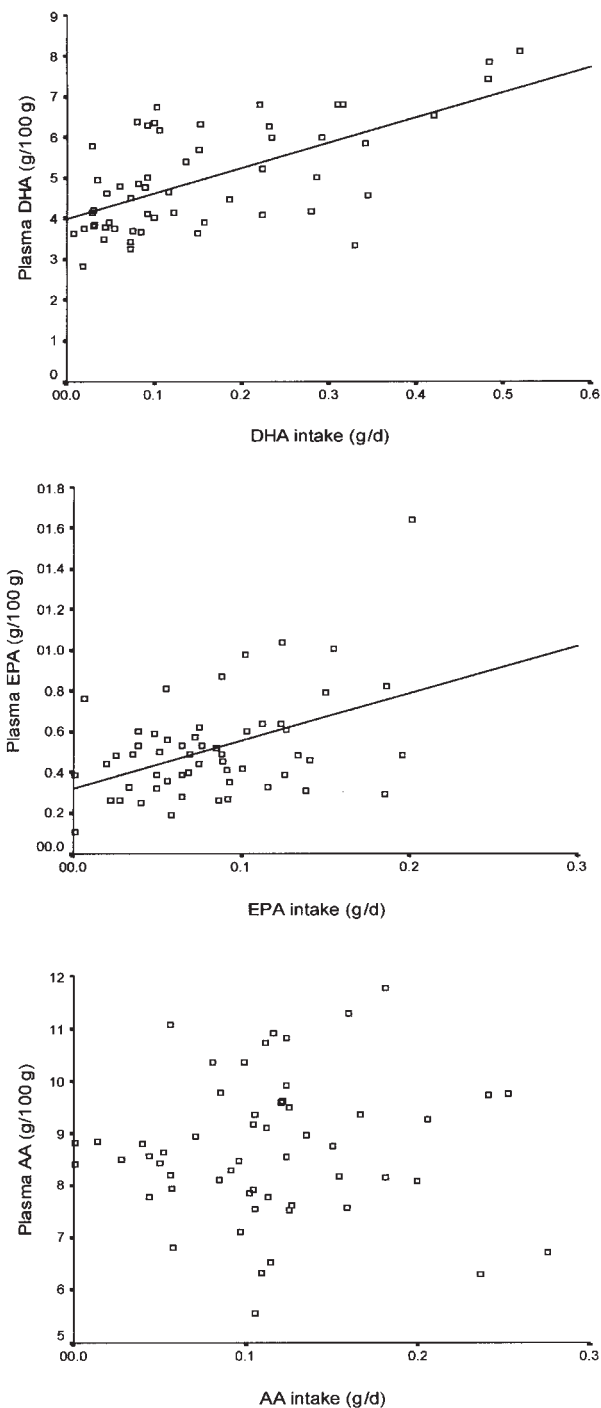
## DISCUSSION

We calculated the intakes of DHA, EPA, and AA of pregnant Canadian women with 2 food-frequency questionnaires and by analysis of  $\approx 500$  local foods in our laboratory. We used a food-frequency questionnaire because fish and seafoods are not typically eaten on a daily basis; thus, estimation of intakes is better represented by a method that covers a longer reporting time than

**TABLE 3**  
Intakes of arachidonic acid (AA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA) from animal foods<sup>1</sup>

	AA	EPA	DHA
	mg/d		
Fatty fish	3.5 ± 0.6 (0.0–16.0)	9.1 ± 3.3 (0.0–88.1)	83.5 ± 14.4 (0.0–384.4)
Lean fish	3.1 ± 0.3 (0.0–10.7)	14.9 ± 1.6 (0.0–51.6)	27.4 ± 3.0 (0.09–5.3)
Shellfish and crustaceans	7.6 ± 1.2 (0.0–38.8)	16.9 ± 2.7 (0.0–86.2)	14.7 ± 2.4 (0.0–75.0)
Total fish and seafood	14.2 ± 1.8 (0.0–50.8)	50.4 ± 6.1 (0.0–58.7)	125.7 ± 17.0 (0.0–426.5)
Chicken	34.6 ± 3.8 (0.0–120.1)	3.8 ± 0.4 (0.0–13.3)	6.7 ± 0.7 (0.0–23.2)
Turkey	24.3 ± 5.7 (0.0–188.9)	1.1 ± 0.2 (0.0–8.2)	3.8 ± 0.9 (0.0–29.1)
Total poultry	58.8 ± 6.5 (0.0–202.9)	4.9 ± 0.5 (0.0–13.3)	10.4 ± 1.1 (0.0–31.8)
Beef and beef products	20.5 ± 0.2 (0.0–63.1)	15.7 ± 1.6 (0.0–4.8)	2.7 ± 0.3 (0.0–8.5)
Pork and pork products	14.4 ± 1.8 (0.0–49.1)	1.4 ± 0.1 (0.0–4.9)	1.4 ± 0.1 (0.0–4.9)
Lamb and lamb products	1.5 ± 0.3 (0.0–8.7)	1.2 ± 0.3 (0.0–6.7)	0.4 ± 0.1 (0.0–2.2)
Total meat	36.4 ± 3.3 (0.0–97.4)	18.4 ± 1.8 (0.0–43.5)	4.6 ± 0.4 (0.0–11.9)
Eggs	32.7 ± 3.6 (0.0–82.7)	0.31 ± 0.04 (0.0–0.9)	14.6 ± 1.6 (0.0–37.0)
Total	112.0 ± 7.8 (0.5–275.8)	73.5 ± 6.3 (0.4–186.8)	153.6 ± 17.9 (17.7–518.6)

<sup>1</sup> $\bar{x} \pm \text{SEM}$ , calculated as the average intake of each subject determined from 2 dietary assessments; range in parentheses.  $n = 55$ .



**FIGURE 1.** Relations between dietary intakes and plasma phospholipid concentrations of docosahexaenoic acid (DHA; 22:6n-3),  $r = 0.63$ ,  $P < 0.0001$ ; eicosapentaenoic acid (EPA; 20:5n-3),  $r = 0.44$ ,  $P < 0.001$ ; and arachidonic acid (AA; 20:4n-6),  $P > 0.05$ , among pregnant Canadian women.  $n = 55$ .

is practical with weighed dietary records. We used biochemical analysis of the plasma phospholipid fatty acids to further validate the dietary assessments and found correlation coefficients between the intakes of DHA and EPA and the concentrations of DHA and EPA in the plasma phospholipids of  $r = 0.63$  and  $0.44$ , respectively. Estimates of energy expenditure with the use of doubly

labeled water have highlighted the discrepancy between actual energy intake and estimates of energy intake derived from dietary assessments (30). The relative error between the estimated and actual intake, however, differs among nutrients estimated by the same dietary instrument (31). The extent to which the food frequency used here under- or overestimated the actual intake of n-6 and n-3 fatty acids, which are distributed among specific foods, is not known.

The importance of DHA for brain and retinal development is recognized (3, 7, 8), but little information is available on which to base dietary reference intakes for pregnant and lactating women. In the present study, polyunsaturated fat represented  $4.7 \pm 0.2\%$  of total energy intake, with mean intakes of 121, 78, and 160 mg/d of AA, EPA, and DHA, respectively. The Food and Agriculture Organization of the World Health Organization noted that 2.2 g n-6 plus n-3 fatty acids/d are deposited in maternal and fetal tissue during pregnancy (32). This value may underestimate the dietary requirement in that fatty acid turnover in eicosanoid synthesis,  $\beta$ -oxidation, and membrane turnover is not considered. The International Society for the Study of Fatty Acids and Lipids, a scientific society, recommends adequate intakes of 4.44 g LA and 2.22 g ALA, with  $\geq 0.22$  g DHA and 0.22 g EPA for adults, and  $\geq 0.3$  g DHA/d for pregnant women (33). Extrapolations from autopsy analysis indicate that the fetus accumulates  $\approx 0.067$  g DHA/d during the last trimester of pregnancy (2). Assuming the fetus represents  $\approx 25\%$  of total weight gain at term pregnancy, then the maternal intake of n-3 fatty acids should exceed the amount accumulated in fetal tissues. In the present study, 1 in 6 women consumed  $< 67$  mg DHA/d during the latter part of gestation, 60% had an intake of  $< 150$  mg DHA/d, and 16% consumed  $> 300$  mg DHA/d.

The extent to which the women in our study are comparable to other women is important. However, only limited information is available on the n-3 and n-6 fatty acid intakes of pregnant women in North America. The intakes of total energy from total fat, saturated fat, monounsaturated fat, and polyunsaturated fat that we found are comparable to recent data from 24-h dietary recalls by 1544 Canadians that found intakes of 28.8%, 9.5%, 10.6%, and 5.0% energy from total fat, saturated fat, monounsaturated fat, and polyunsaturated fat, respectively, for women 18-34 y (34). The mean intake of 1.6 g ALA/d in our study, however, is higher than the estimated intake of 1.0 g ALA/d derived from the 1987-1988 US Department of Agriculture Nationwide Food Consumption Survey (35, 36). Studies in first-trimester pregnant, lactating, and nonlactating Dutch women found intakes of 1.0, 1.2, and 0.85 g ALA/d, respectively (37, 38), and studies in Belgium found intakes of 1.4 g ALA/d among pregnant women (39). Data on the per capita availability of fatty acids estimated intakes of 0.046 g EPA and 0.078 g DHA/d per person in the United States in 1985, with 90% of the EPA and 75% of the DHA available from fish. Estimations based on the 1987-1988 US Department of Agriculture Nationwide Food Consumption Survey similarly suggested intakes of 0.1 g EPA + DHA/d (35). Other recent studies that used food-frequency questionnaires reported intakes of 0.05 g EPA/d and 0.085 g DHA/d among lactating women and 0.08 g EPA/d and 0.14 g DHA/d among pregnant women in Holland (37, 38), similar to our calculated intakes of 0.08 g EPA/d and 0.16 g DHA/d for pregnant Canadian women.

Interpretation of the physiologic significance of the intake of DHA during pregnancy is complex because DHA can be formed from its precursor, ALA (14). In stable-isotope-tracer studies,

TABLE 4

Relation of fish consumption to concentrations of n-6 and n-3 fatty acids in plasma phospholipids<sup>1</sup>

Fatty acid	Servings of fish/wk				<i>P</i> <sup>2</sup>
	0	0.1–0.9	1–1.9	>2	
	<i>mg/100 g fatty acids</i>				
DHA	3.89 ± 0.33	4.48 ± 0.25	5.19 ± 0.27	5.75 ± 0.39	<0.01
EPA	0.26 ± 0.05	0.47 ± 0.04	0.49 ± 0.04	0.71 ± 0.11	<0.01
AA	8.33 ± 0.30	8.55 ± 0.26	8.76 ± 0.36	8.29 ± 0.44	NS
LA	23.6 ± 0.78	20.2 ± 0.66	20.7 ± 0.64	21.0 ± 1.0	NS
ALA	0.40 ± 0.10	0.33 ± 0.07	0.35 ± 0.02	0.43 ± 0.03	NS

<sup>1</sup> $\bar{x} \pm \text{SEM}$ ; *n* = 55. DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; AA, arachidonic acid; LA, linoleic acid; ALA,  $\alpha$ -linolenic acid. Concentrations of fatty acids were measured at 35 wk gestation. Total fish (fatty and lean) intake was calculated as the average intake of each subject determined from 2 dietary assessments.


<sup>2</sup>Correlation was used to determine the association between fish intake and the fatty acids. *P* values > 0.05 were considered not significant.

adult humans converted < 1–4% ALA to DHA (15, 16). This suggests that when provided as ALA, the amount of ALA required for fetal-tissue DHA accretion could be  $\geq 25$ -fold, higher than the requirement as preformed DHA. The mean ALA intake in the present study was 1.62 g/d, with a range of 0.16–3.6 g/d; 45% of the women consumed < 0.5% of energy as ALA. Although the physiologic importance of the intakes of ALA found in our study is unknown, it is known that higher intakes of ALA do not increase the concentrations of DHA in the blood lipids of infants or adults (20, 21).

Information on the AA content of Western diets is limited. Data for Australia suggest that AA intakes in that country are 100–200 mg/d (40). The present study found mean intakes of 121 ± 8 mg/d. Autopsy data suggest that the fetus accumulates  $\approx 55.2$  mg n-6 fatty acids/d, which includes  $\approx 250$ –400 mg AA/d, during the third trimester of gestation (1, 2). We found an inverse association between LA intake and plasma AA concentrations, consistent with stable-isotope studies that showed lower conversion of LA to AA at higher LA intakes (15). We also recently showed an inverse association between AA status and birth weight and a significant association between maternal and newborn infant AA concentrations (9). Whether higher maternal intakes of LA are of physiologic relevance to fetal growth and tissue AA accumulation is not yet clear.

It is known that the maternal intake of DHA during pregnancy determines the DHA status of the infant at birth and for several weeks following birth (9, 11–13, 41, 42). Several clinical studies showed that a dietary source of DHA increases the development of visual acuity and scores on some tests of motor skill and language development in preterm infants (7, 8). An association between less premature electroencephalography patterns at birth and higher concentrations of DHA in the plasma phospholipids in the newborn infant (12) may also suggest that the supply of DHA during gestation influences neural development in the human brain. Studies on the importance of dietary DHA for neural development after birth in term infants, however, have yielded inconsistent findings (43–45). We recently found a significant relation between plasma and red blood cell DHA concentrations at 2 mo of age and later visual acuity and language development in breast-fed term infants (46). Possibly, this might have involved habitual differences in maternal DHA intake, which were also present during gestation, suggesting the need to consider the DHA status of the infant at birth as a potentially important variable.

In conclusion, we showed that the intakes of DHA among some Canadian women during the third trimester of gestation appear to be below possible needs for fetal and maternal tissue DHA

accretion. This raises the need for studies combining functional outcome measures of infant neural development, dietary fat intake, and DHA. 

SMI was the principal investigator. SLE collected the dietary information and undertook the blood analyses as part of the requirements for completion of a MSc degree.

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