Dietary intake and status of n−3 polyunsaturated fatty acids in a population of fish-eating and non-fish-eating meat-eaters, vegetarians, and vegans and the precursor-product ratio of α-linolenic acid to long-chain n−3 polyunsaturated fatty acids: results from the EPIC-Norfolk cohort[1–3]

Ailsa A Welch, Subodha Shakya-Shrestha, Marleen AH Lentjes, Nicholas J Wareham, and Kay-Tee Khaw

ABSTRACT

Background: Intakes of n−3 (omega-3) polyunsaturated fatty acids (PUFAs) are important for health. Because fish is the major source of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), non-fish-eaters may have suboptimal n−3 PUFA status, although the importance of the conversion of plant-derived α-linolenic acid (ALA) to EPA and DHA is debated.

Objective: The objective was to determine intakes, food sources, and status of n−3 PUFAs according to dietary habit (fish-eaters and non-fish-eating meat-eaters, vegetarians, or vegans) and estimated conversion between dietary ALA and circulating long-chain n−3 PUFAs.

Design: This study included 14,422 men and women aged 39–78 y from the EPIC (European Prospective Investigation into Cancer and Nutrition)-Norfolk cohort with 7-d diary data and a substudy in 4902 individuals with plasma phospholipid fatty acid measures. Intakes and status of n−3 PUFAs were measured, and the precursor-product ratio of ALA to circulating n−3 PUFAs was calculated.

Results: Most of the dietary intake of EPA and DHA was supplied by fish; however, meat was the major source in meat-eaters, and spreading fats, soups, and sauces were the major sources in vegetarians. Total n−3 PUFA intakes were 57–80% lower in non-fish-eaters than in fish-eaters, but status differences were considerably smaller. The estimated precursor-product ratio was greater in women than in men and greater in non-fish-eaters than in fish-eaters.

Conclusions: Substantial differences in intakes and in sources of n−3 PUFAs existed between the dietary-habit groups, but the differences in status were smaller than expected, possibly because the precursor-product ratio was greater in non-fish-eaters than in fish-eaters, potentially indicating increased estimated conversion of ALA. If intervention studies were to confirm these findings, it could have implications for fish requirements. *Am J Clin Nutr* 2010;92:1040–51.

INTRODUCTION

An adequate n−3 (omega-3) polyunsaturated fatty acid (n−3 PUFA) status is important for the maintenance of health and could reduce the risk of chronic and inflammatory diseases, such as coronary artery disease and, potentially, dementia, diabetes, and asthma, although the evidence is weaker (1–6). Dietary n−3 PUFAs are either plant-derived [eg, short-chain α-linolenic acid (ALA)] or marine-derived [eg, longer-chain eicosapentaenoic acid (EPA) and docosahexaenoic acids (DHA)]. ALA is the major plant-based n−3 PUFA and is found in walnuts, flaxseeds, hemp seeds and their oils; in rapeseed (canola) oil; and in smaller amounts in soya oil and green-leafy vegetables (7, 8).

Although conversion from ALA to EPA and DHA occurs, this is limited (9–11). Because fish and fish oils are the most concentrated sources of EPA and DHA, individuals who do not eat fish or fish oils (eg, vegans and non-fish-eating vegetarians and meat-eaters) could be at risk of low or inadequate n−3 PUFA status (12, 13). In addition, because the supply of wild fish is under threat and supplies are compromised, if the maintenance of adequate n−3 PUFA status via conversion of plant-derived ALA was possible this could reduce the requirements for fish and help preserve the fish supply (14, 15).

Fish consumption in the United Kingdom is moderate compared with that in other European countries, because a large proportion of the population does not eat fish and only 15–44% eat oily fish (16–18). Fish intake varies regionally throughout Europe, and a 10-country study found a 6-fold difference between the lowest and the highest intakes (16). Prior studies have found that, although non-fish-eating meat-eaters and vegetarians have much lower intakes of EPA and DHA than do fish-eaters, their n−3 PUFA status is higher than would be expected (13, 19–23).

[1] From the School of Medicine Health Policy and Practice, University of East Anglia, Norwich, United Kingdom (AAW); EPIC-Norfolk UK, Department of Public Health and Primary Care, University of Cambridge, Cambridge, United Kingdom (SSS and K-TK); the MRC Centre for Nutritional Epidemiology in Cancer Prevention and Survival, Department of Public Health and Primary Care, University of Cambridge, Cambridge, United Kingdom (MAHL); and the Medical Research Council Epidemiology Unit, Institute of Metabolic Science, Addenbrooke's Hospital, Cambridge, United Kingdom (NJW).

[2] EPIC-Norfolk is supported by program grants from the Medical Research Council UK and Cancer Research UK.

[3] Address correspondence to AA Welch, School of Medicine Health Policy and Practice, University of East Anglia, Norwich, Norfolk NR4 7TJ, United Kingdom. E-mail: a.welch@uea.ac.uk.

Received March 2, 2010. Accepted for publication August 14, 2010.

First published online September 22, 2010; doi: 10.3945/ajcn.2010.29457.

The conversion of ALA to EPA and DHA takes place via a series of enzymatically controlled steps involving elongase and Δ^5- and Δ^6-desaturase enzymes (**Figure 1**) and is estimated to be ≈5% for EPA and <0.5% for DHA, although more recent research indicates that this could be more variable and tissue specific and it is greater in women of child-bearing age than in men (9, 10, 24–27). The conversion of ALA to EPA can also be negatively affected by dietary linoleic acid [18:2 n−6 (omega-6) PUFAs] because of competitive inhibition of the Δ^5- and Δ^6-desaturase enzymes (25, 28). Smoking habit also positively influences conversion (29). Therefore, because the conversion of ALA to EPA and DHA is variable and the status of non-fish-eaters is higher than expected, it is possible that greater conversion could occur in those consuming less EPA and DHA.

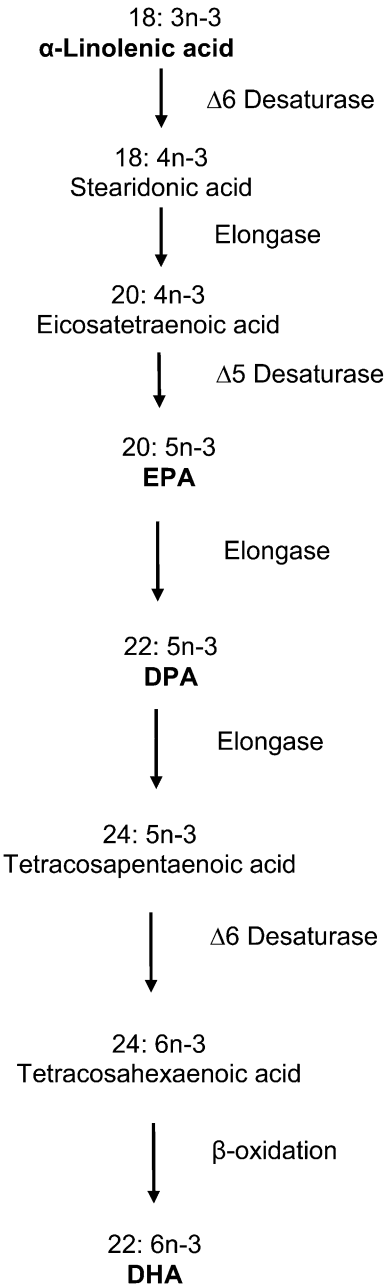

FIGURE 1. Biosynthesis of longer-chain n−3 polyunsaturated fatty acids from α-linolenic acid. EPA, eicosapentaenoic acid; DPA, docosapentaenoic acid; DHA, docosahexaenoic acid.

Because n−3 PUFA status may differ depending on dietary habit and there are few detailed data on intakes and sources of individual n−3 PUFA for the UK population, we investigated intakes and status of n−3 PUFAs in a free-living population in the United Kingdom. The purpose of this study was first to assess detailed n−3 PUFA intakes and food sources (plant, marine, and others) in dietary-habit groups representative of eating habits within the population (fish-eaters and non-fish-eating groups of meat-eaters, vegetarians, or vegans); second, to describe the n−3 PUFA status in these groups; and third, to calculate the precursor-product ratio of dietary ALA to plasma long-chain n−3 PUFAs (PLLC n−3 PUFAs) to statistically estimate whether the increased estimated conversion might occur in non-fish-eaters.

SUBJECTS AND METHODS

Participants and recruitment

Participants recruited for this study were taken from the European Prospective Investigations into Cancer and Nutrition (EPIC)–Norfolk Study of 25,000 men and women from the Norfolk region of the United Kingdom (20, 30). The baseline study was performed between 1993 and 1997. Ethical approval for the study was given by the Norfolk Health District Ethics Committee. This study was undertaken in 14,422 individuals aged 39–78 y who had available entered and cleaned dietary data from 7-d food diaries (referred to as the "whole population") and a substudy of 4902 men and women in whom plasma phospholipid fatty acids were measured.

Data collection

All participants were asked to complete a self-administered detailed health and lifestyle questionnaire. Participants then underwent a health examination, during which measurements and blood samples were obtained by trained nursing staff (30).

Anthropometric measures

Height was measured to the nearest 0.1 cm and weight to the nearest 0.2 kg while participants were wearing light clothing and no shoes (21). Body mass index (BMI) was calculated as weight (in kg)/height^2 (in m).

Dietary data

The 7-d food diary with estimated weights of food consumed consisted of an A5-sized booklet containing 17 sets of color photographs representing portion sizes and instructions to guide the information to be reported (31). Amounts of foods consumed were also described by using household measures and standard units. Nurses, trained to standardized protocols, provided instructions on how to complete the 7-d diary and performed an interviewed 24-h recall at the health check that formed the first day of the record. Participants were asked to complete the remaining 6 d of the diary and to then send it back to the study center. Diary data were entered by using the Data into Nutrients for Epidemiologic Research (DINER) data entry system, and the

entry staff received training with their work checked until it was considered satisfactory (32). A series of data entry checks were also performed before data analysis.

We analyzed 14,422 diaries for intakes of total dietary n−3 PUFAs, ALA, EPA, and DHA. Four categories of eating habit were defined by using the 7-d diary data categorized into 3 groups of non-fish-eaters and one group of fish-eaters, chosen to represent the type of eating habit prevalent within populations: *1*) vegans who reported eating no meat, fish, dairy, or eggs during the period of the dietary record; *2*) vegetarians who reported no fish or meat intake during the period of the diary record; *3*) meat-eaters who did not eat fish but ate meat; and *4*) fish-eaters who ate fish and also, mainly, ate meat (97% ate meat). This categorization is referred to as the "dietary-habit" group throughout this manuscript. These same dietary-habit categories were used in the whole-population and in the substudy analyses.

The EPIC-Norfolk fatty acid nutrient database was compiled for 2480 foods in the DINER database (AA Welch, S Shakya Shrestha, KT Khaw, personal communication, 2010). Analytic fatty acid data from published and unpublished UK sources were included when available. A small proportion of published fatty acid data for single foods from other European countries was also incorporated. When analytic and published data were not available, calculations were carried out to obtain the data from a similar food or a different form of the same food. Data for cooked foods and dishes were calculated by using a newly developed recipe calculation system and with conversion factors derived from the literature (AA Welch, S Shakya Shrestha, KT Khaw, personal communication, 2010).

Intakes of ALA, EPA, and DHA were calculated for each individual for food groups available from the food diary data. Total dietary n−3 PUFA intake was calculated from the sum of ALA, EPA, and DHA. Intake by food group was calculated as the mean intake of each fatty acid according to each food group, for each individual participant. n−3 Docosapentaenoic (DPA) acid could also influence the precursor-product ratio of ALA to LC n−3 PUFAs, but there were insufficient data to include DPA in the nutrient database. However, because meat contains a small amount of DPA (up to 0.06 g/100 g), total meat intake was used to account for this source of DPA in the statistical analyses.

In this article, *LC n−3 PUFAs* refers to the total dietary intake of long-chain EPA and DHA, *total dietary n−3 PUFAs* refers to the intake of ALA in addition to LC n−3 PUFAs, and *PLLC n−3 PUFAs* refers to the sum of EPA, DPA, and DHA circulating in plasma.

Supplement use was defined by questions from the 7-d diary. In version 1, the question was "Please name any vitamins, minerals or other food supplements taken on each day of last week," and information on the brand and name of the supplement and the amount taken was requested. In version 2, the question was "Please name any vitamins, minerals or other food supplements taken on each day of last week. Please write down all the details from each packet/container, and enclose labels(s) giving ingredients and individual amounts where possible," and information on the brand, name, and amount for each day a supplement was consumed was also requested. Data for supplement consumption was identified from the EPIC-Norfolk vitamin and mineral supplement database (33). Supplement takers were identified as those who reported consuming fish oils or cod or halibut liver oils or those identified in the vitamin and mineral database as consuming the nutrients EPA or DHA.

Plasma n−3 PUFAs

Blood samples were collected by venipuncture into tubes containing citrate buffer during the health examination. After overnight storage in the dark at 4–7°C, the samples were centrifuged at 2100 × *g* for 15 min at 4°C. Plasma aliquots (450 μL) were transferred to plastic straws and stored in liquid nitrogen. Lipids were extracted with chloroform-methanol after the addition of 100 μg butylated hydroxytoluene and 20 μg 1,2-dipalmitoyl-D62-*sn*-glycero-3-phosphocholine (Avanti Polar Lipids, Alabaster, AL) internal standard to 200 μL thawed plasma. Plasma phospholipids were isolating via solid-phase extraction chromatography (LC-Si; Supelco, St Louis, MO) and measured by using an HP 5980 gas chromatograph (Agilent, Palo Alto, CA) equipped with a flame ionization detector, as described in detail elsewhere (20). The concentration of each phospholipid fatty acid was expressed as a concentration (μmol/L plasma).

The substudy consisted of 4902 individuals with dietary and plasma n−3 PUFA data who were also nonsupplement users (2256 women and 2646 men). Mean (±SD) PLLC n−3 PUFAs were higher in supplement users than in nonusers: 435 ± 200 μmol/L compared with 360 ± 163 μmol/L in men ($P < 0.001$) and 474 ± 203 compared with 402 ± 170 μmol/L in women ($P < 0.001$). Because there could be interference in the conversion of ALA to EPA by EPA and DHA, those who took supplements were excluded from the substudy analyses (562 men and 593 women).

Individual circulating n−3 PUFAs were analyzed as detailed above. In the analyses, the PLLC n−3 PUFAs excluded ALA but included DPA. The precursor-product ratio of ALA to LC n−3 PUFAs was calculated by relating circulating plasma phospholipid n−3 PUFAs to dietary ALA (DALA) by using the ratio PLLC n−3 PUFAs:DALA (ALA intake in g/d was converted to μmol/d) by summing plasma EPA, DPA, and DHA (μmol/L) and dividing by ALA (μmol/d). This precursor-product ratio provides a statistical method of comparing the potential conversion of dietary ALA to circulating LC n−3 PUFAs between the different dietary-habit groups. We hypothesized that an increased estimated conversion would be observed as a higher precursor-product ratio of ALA to circulating n−3 PUFAs in non-fish-eaters than in fish-eaters. Because the conversion of ALA to EPA and DHA can be affected by age, BMI, and smoking habit, the ratio was adjusted for these covariates. Because this conversion pathway may also involve competition for the Δ^5- and Δ^6-desaturase enzymes by linoleic acid, models 2 and 3 were adjusted for circulating linoleic acid (28). Because the intake of EPA, DHA, and DPA (in meat) may affect the conversion of ALA to EPA and DHA, the fully adjusted model (models 2 and 3) also included these covariates (28).

Statistical analyses

Statistical analyses were performed with STATA statistical software version 10.0 (Stata Corp, College Station, TX). The analyses were stratified by sex, because there were significant interactions between sex and n−3 PUFA intake and plasma phospholipid n−3 PUFAs. Means and SDs of dietary and plasma data and the covariates were calculated by dietary-habit group. Adjusted means of the ratio PLLC n−3-PUFAs:DALA were

calculated by dietary-habit group in the substudy. Two models for adjustment of PLLC n−3 PUFAs:DALA for the covariates were used: *1*) by age, BMI, and smoking habit, and *2*) by age, BMI, smoking habit, circulating plasma linoleic acid, and dietary EPA, DHA, and meat. Further regression analyses were performed with PLLC n−3 PUFAs:DALA to compare the different variables in 3 further models in both men and women in the substudy, using the SD of plasma linoleic acid; dietary EPA, DHA, and meat; and categories of 10 y for age and 4 units for BMI.

RESULTS

Descriptive characteristics of the study population are shown in **Table 1**. Intakes of total dietary n−3 PUFAs, ALA, EPA, DHA, and linoleic acid were significantly higher in men than in women. Intakes of total dietary n−3 PUFAs were highest in fish-eaters and lowest in vegans (in men) and in meat-eaters (in women).

ALA intake

ALA intake was highest in fish-eaters and lowest in vegans, and, in women, also in meat-eaters ($P < 0.001$; Table 1). ALA contributed 82% of total dietary n−3 PUFAs in the whole population, but was 80% in fish-eaters, contrasting with 98% of the total in vegetarian men (99% in vegetarian women) and 97% in meat-eaters.

The major sources of ALA in the whole population were the cereals and vegetables food groups (42% of ALA), with total fish contributing 12% to intake and total meat contributing 13% to intake in men and to 12% of intake in women (**Table 2**). Cereals and vegetables supplied 63% of intake in vegetarians, 63% (men) and 73% (women) in vegans, and 44% (men) and 47% (women) in meat-eaters. In fish-eaters, cereals and vegetables supplied 41% (men) and 45% (women) of ALA intake. In meat-eaters, meat supplied ≈17% of ALA and in fish-eaters ≈12% of intake. However, in fish-eaters, total fish contributed to ≈14% of intake.

EPA intake

EPA intakes in meat-eaters were only 15% (men) and 18% (women) of those in fish-eaters and in vegetarians were ≈9% (women) and 15% (men) of those in fish-eaters (Table 1). In fish-eaters, the contribution to EPA intake from total fish was ≈82% and was mainly from fatty fish (61% of total intake; **Table 3**). In comparison, other sources were minor; meats and spreading fats accounted for ≈6% each. In meat-eaters, meat supplied 43% of EPA, spreading fats ≈38% of EPA, and dairy foods and products ≈11% of EPA. In vegetarians, the major sources of EPA were spreading fats (70% in men and 59% in women) and dairy foods and products (18.4% in men and 26.3% in women).

TABLE 1
Characteristics and dietary intakes of n−3 polyunsaturated fatty acids (PUFAs) and linoleic acid in the whole population (n = 14,422)[1]

			Non-fish-eaters			
	All	Fish-eaters	Meat-eaters	Vegetarians	Vegans	P[2]
Men						
n	7056	5952	996	96	12	
Age (y)	62.3 ± 8.7[3,4]	62.7 ± 8.5	60.7 ± 9.0	57.5 ± 9.7	53.2 ± 8.1	<0.001
Weight (kg)	80.3 ± 11.7[3]	80.2 ± 11.5	81.0 ± 12.3	79.8 ± 13.1	84.6 ± 14.4	0.086
Height (cm)	173.4 ± 6.7[3]	173.5 ± 6.7	173.4 ± 6.8	173.1 ± 7.7	173.8 ± 4.2	0.648
BMI (kg/m^2)	26.7 ± 3.4	26.6 ± 3.3	26.9 ± 3.5	26.6 ± 3.8	28.0 ± 4.8	0.033
Current smokers [% (*n*)]	11.2 (787)[5]	10.2 (604)	17.1 (170)	10.4 (10)	25 (3)	<0.001
Total dietary n−3 PUFAs (g/d)	1.50 ± 0.59[3]	1.57 ± 0.58	1.15 ± 0.55	1.27 ± 0.56	1.04 ± 0.71	<0.001
α-Linolenic acid (g/d)	1.23 ± 0.43[3]	1.25 ± 0.41	1.11 ± 0.54	1.25 ± 0.57	1.02 ± 0.71	<0.001
Eicosapentaenoic acid (g/d)	0.11 ± 0.15[3]	0.13 ± 0.16	0.02 ± 0.02	0.02 ± 0.02	0.01 ± 0.001	<0.001
Docosahexaenoic acid (g/d)	0.16 ± 0.22[3]	0.19 ± 0.22	0.02 ± 0.02	0.0007 ± 0.004	0 ± 0	<0.001
Linoleic acid (g/d)	12.35 ± 5.04[3]	12.41 ± 4.8	11.80 ± 5.95	14.78 ± 6.9	12.79 ± 10.80	0.702
Women						
n	7366	6258	938	154	16	
Age (y)	61.3 ± 9.1	61.6 ± 9.0	60.6 ± 9.3	56.9 ± 9.6	54.1 ± 7.7	<0.001
Weight (kg)	68.4 ± 12.1	68.3 ± 11.8	69.7 ± 13.1	65.8 ± 13.6	74.1 ± 16.8	0.201
Height (cm)	160.5 ± 6.3	160.5 ± 6.3	160.3 ± 6.4	161.4 ± 6.6	164.1 ± 6.4	0.384
BMI (kg/m^2)	26.6 ± 4.5	26.5 ± 4.4	27.1 ± 4.9	25.3 ± 4.9	27.5 ± 5.4	0.294
Current smokers [% (*n*)]	10.1 (744)	9.5 (593)	13.9 (130)	13.0 (20)	6.3 (1)	<0.001
Total dietary n−3 PUFAs (g/d)	1.22 ± 0.49	1.27 ± 0.49	0.89 ± 0.33	0.98 ± 0.45	0.91 ± 0.67	<0.001
α-Linolenic acid (g/d)	0.99 ± 0.36	1.01 ± 0.35	0.86 ± 0.33	0.97 ± 0.45	0.86 ± 0.69	<0.001
Eicosapentaenoic acid (g/d)	0.09 ± 0.12	0.11 ± 0.13	0.02 ± 0.01	0.01 ± 0.01	0.02 ± 0.08	<0.001
Docosahexaenoic acid (g/d)	0.13 ± 0.18	0.15 ± 0.19	0.01 ± 0.01	0.0004 ± 0.005	0 ± 0	<0.001
Linoleic acid (g/d)	9.42 ± 3.90	9.52 ± 3.75	8.59 ± 4.15	10.06 ± 6.02	11.91 ± 9.92	0.002

[1] Total dietary n−3 PUFAs represent the sum of α-linolenic acid, eicosapentaenoic acid, and docosahexaenoic acid.
[2] P for difference between the 4 dietary groups (fish-eaters or non-fish-eating meat-eaters, vegetarians, or vegans) calculated by using ANOVA (excludes the group labeled "All").
[3] Significant difference between men and women, $P < 0.001$.
[4] Mean ± SD (all such values).
[5] $P = 0.04$ (2-sample t test).

TABLE 2
Sources of dietary α-linolenic acid in the whole population (All) and in the different dietary groups: fish-eaters and non-fish-eating meat-eaters, vegetarians, or vegans[1]

	Men					Women				
			Non-fish-eaters					Non-fish-eaters		
	All	Fish-eaters	Meat-eaters	Vegetarians	Vegans	All	Fish-eaters	Meat-eaters	Vegetarians	Vegans
n	7056	5952	996	96	12	7366	6258	938	154	16
α-Linolenic acid (g/d)[2]	1.23 ± 0.43	1.25 ± 0.41	1.11 ± 0.54	1.25 ± 0.57	1.02 ± 0.71	0.99 ± 0.36	1.01 ± 0.35	0.86 ± 0.33	0.97 ± 0.45	0.86 ± 0.69
Total cereals (% of intake)	26.7	26.4	28.1	34.6	43.3	25.7	25.3	27.9	33.8	30.2
Rice, pastas, and dishes	1.6	1.6	1.6	3.1	4.5	2.0	1.9	2.1	3.7	6.1
Breads and rolls	6.7	6.6	7.2	9.7	10.7	6.2	6.1	6.7	6.9	5.4
Breakfast cereals	2.5	2.6	2.2	2.6	6.6	2.5	2.4	2.4	3.4	1.2
Biscuits, pastries, buns, and pizzas	15.9	15.6	17.1	19.2	26.8	15.1	14.8	16.7	19.8	18.9
Dairy (% of intake)	6.5	6.3	7.2	9.8	0	6.5	6.3	7.5	9.4	0
Eggs (% of intake)	1.8	1.7	2.3	1.7	0	1.6	1.6	1.9	2.6	0
Potatoes (% of intake)	6.6	6.5	7.7	5.7	4.0	5.8	5.8	6.3	4.8	3.4
Vegetables (% of intake)	14.7	14.3	16.2	28.5	19.5	17.4	16.9	19.2	29.7	42.7
Fruit (% of intake)	1.1	1.2	1.1	1.5	0	1.8	1.8	1.9	3.0	1.7
Nuts (% of intake)	1.1	1.1	1.5	1.5	0	1.3	1.4	0.7	1.5	2.3
Herbs and spices (% of intake)	0.1	0.1	0.2	0.1	0	0.1	0.1	0.1	0	0.5
Total fish (% of intake)	12.1	14.1	0	0	0	12.1	13.9	0	0	0
White fish	0.1	0.1	0	0	0	0.1	0.1	0	0	0
Fatty fish	1.8	2.1	0	0	0	1.9	2.2	0	0	0
Crustaceans	0	0	0	0	0	0	0	0	0	0
Mollusks	0	0	0	0	0	0	0	0	0	0
Fish products and dishes	10.2	11.9	0	0	0	10.1	11.6	0	0	0
Total meat (% of intake)	13.0	12.5	17.5	0	0	12.0	11.7	16.6	0	0
Meats (beef, pork, lamb)	2.3	2.2	3.4	0	0	1.9	1.8	2.6	0	0
Poultry	1.9	1.8	2.6	0	0	1.9	1.9	2.7	0	0
Game	0.0	0.0	0.0	0	0	0.0	0.0	0.0	0	0
Offal	0.1	0.1	0.1	0	0	0.1	0.1	0.1	0	0
Meat products	2.6	2.5	3.6	0	0	2.0	2.0	2.9	0	0
Meat dishes	6.0	5.9	7.9	0	0	6.0	5.9	8.2	0	0
Spreading fats (% of intake)	9.7	9.4	11.5	8.7	10.7	8.1	7.9	9.9	8.0	5.6
Snacks (% of intake)	1.6	1.5	2.1	1.7	0.4	1.8	1.7	2.2	2.1	1.3
Soups and sauces (% of intake)	4.8	4.8	4.7	6.3	14.2	5.7	5.7	5.8	5.1	10.9

[1] Beverages, alcoholic drinks, and miscellaneous foods were not included because of 0% or 0.1% values in intakes of α-linolenic acid, eicosapentaenoic acid, and docosahexaenoic acid.
[2] Values are means ± SDs.

TABLE 3
Sources of dietary eicosapentaenoic acid (EPA) in the whole population (All) and in the different dietary groups: fish-eaters, non-fish-eating meat-eaters, vegetarians, or vegans[1]

	Men					Women				
			Non-fish-eaters					Non-fish-eaters		
	All	Fish-eaters	Meat-eaters	Vegetarians	Vegans	All	Fish-eaters	Meat-eaters	Vegetarians	Vegans
n	7056	5952	996	96	12	7366	6258	938	154	16
EPA (g/d)[2]	0.11 ± 0.15	0.13 ± 0.16	0.02 ± 0.02	0.02 ± 0.02	0.01 ± 0.001	0.09 ± 0.12	0.11 ± 0.13	0.02 ± 0.01	0.01 ± 0.01	0.02 ± 0.08
Total cereals (% of intake)	2.3	2.3	2.8	7.3	20.3	2.7	2.7	4.3	5.5	0
Rice, pastas, and dishes	0.3	0.3	0.0	0	6.8	0.2	0.2	0	0	0
Breads and rolls	0	0	0.2	1.8	0	0.1	0	0.3	0	0
Breakfast cereals	0	0	0	0	0	0	0	0	0	0
Biscuits, pastries, buns, and pizzas	2.0	2.0	2.6	5.5	13.5	2.4	2.4	4.0	5.5	0
Dairy (% of intake)	2.1	1.8	11.3	18.4	0	2.0	1.7	10.7	26.3	0
Eggs (% of intake)	0.3	0.2	1.5	2.3	0	0.3	0.3	1.8	6.2	0
Potatoes (% of intake)	0.2	0.2	1.1	0	0	0.2	0.2	1.0	1.8	0.8
Vegetables (% of intake)	0	0	0.1	0	0	0	0	0.2	1.2	0.4
Fruit (% of intake)	0	0	0	0	0	0	0	0	0	0
Nuts (% of intake)	0	0	0	0	0	0	0	0	0	0
Herbs and spices (% of intake)	0	0	0	0	0	0	0	0	0	0
Total fish (% of intake)	80.6	83.0	0	0	0	81.4	83.5	0	0	0
White fish	3.0	3.1	0	0	0	3.6	3.7	0	0	0
Fatty fish	59.6	61.4	0	0	0	59.6	61.1	0	0	0
Crustaceans	2.8	2.9	0	0	0	3.1	3.2	0	0	0
Mollusks	0.4	0.4	0	0	0	0.2	0.2	0	0	0
Fish products and dishes	14.8	15.2	0	0	0	14.8	15.2	0	0	0
Total meat (% of intake)	6.5	5.5	43.0	0	0	6.1	5.3	42.7	0	0
Meats (beef, pork, lamb)	3.0	2.5	20.2	0	0	2.6	2.2	17.9	0	0
Poultry	0.6	0.5	4.2	0	0	0.7	0.6	5.5	0	0
Game	0	0	0	0	0	0	0	0	0	0
Offal	0.4	0.3	2.1	0	0	0.4	0.4	1.3	0	0
Meat products	0.7	0.6	4.0	0	0	0.7	0.6	4.8	0	0
Meat dishes	1.8	1.5	12.7	0	0	1.8	1.5	13.1	0	0
Spreading fats (% of intake)	7.3	6.3	38.3	70.2	72.0	6.4	5.6	36.8	58.9	22.8
Snacks (% of intake)	0	0	0	0	0	0	0	0.1	0	0
Soups and sauces (% of intake)	0.6	0.6	1.7	1.9	7.7	0.8	0.7	2.4	0	76.0

[1] Beverages, alcoholic drinks, and miscellaneous foods were not included because of 0% or 0.1% values in intakes of α-linolenic acid, EPA, and docosahexaenoic acid.
[2] Values are means ± SDs.

TABLE 4
Sources of dietary docosahexaenoic acid (DHA) in the whole population (All) and in the different dietary groups: fish-eaters and non-fish-eating meat-eaters, vegetarians, or vegans[1]

	Men					Women				
			Non-fish-eaters					Non-fish-eaters		
	All	Fish-eaters	Meat-eaters	Vegetarians	Vegans	All	Fish-eaters	Meat-eaters	Vegetarians	Vegans
n	7056	5952	996	96	12	7366	6258	938	154	16
DHA (g/d)[2]	0.16 ± 0.22	0.19 ± 0.22	0.02 ± 0.02	0.0007 ± 0.004	0	0.13 ± 0.18	0.15 ± 0.19	0.01 ± 0.01	0.0004 ± 0.005	0
Total cereals (% of intake)	2.5	2.5	0.0	0.0	0	2.7	2.8	0.0	0.0	0
Rice, pastas, and dishes	0.4	0.4	0.0	0.0	0	0.4	0.4	0.0	0.0	0
Breads and rolls	0.0	0.0	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Breakfast cereals	0.0	0.0	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Biscuits, pastries, buns, and pizzas	2.0	2.1	0.0	0.0	0	2.4	2.4	0.0	0.0	0
Dairy (% of intake)	0.0	0.0	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Eggs (% of intake)	0.1	0.1	1.6	16.0	0	0.1	0.1	1.0	88.2	0
Potatoes (% of intake)	0.0	0.0	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Vegetables (% of intake)	0.0	0.0	0.1	0.0	0	0.0	0.0	0.0	0.0	0
Fruit (% of intake)	0.0	0.0	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Nuts (% of intake)	0.0	0.0	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Herbs and spices (% of intake)	0.0	0.0	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Total fish (% of intake)	88.7	89.9	0.0	0.0	0	88.8	89.8	0.0	0.0	0
White fish	5.2	5.3	0.0	0.0	0	6.2	6.2	0.0	0.0	0
Fatty fish	63.3	64.2	0.0	0.0	0	63.2	64.0	0.0	0.0	0
Crustaceans	1.8	1.8	0.0	0.0	0	1.9	1.9	0.0	0.0	0
Mollusks	0.1	0.1	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Fish products and dishes	18.3	18.5	0.0	0.0	0	17.5	17.7	0.0	0.0	0
Total meat (% of intake)	7.8	6.6	92.6	0.0	0	7.3	6.3	92.6	0.0	0
Meats (beef, pork, lamb)	2.0	1.6	25.6	0.0	0	1.5	1.3	20.5	0.0	0
Poultry	2.4	2.0	29.3	0.0	0	2.6	2.2	34.5	0.0	0
Game	0.0	0.0	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Offal	0.4	0.4	4.7	0.0	0	0.4	0.3	2.6	0.0	0
Meat products	0.9	0.8	9.6	0.0	0	0.8	0.7	9.9	0.0	0
Meat dishes	2.1	1.8	23.5	0.0	0	2.0	1.7	25.1	0.0	0
Spreading fats (% of intake)	0.0	0.0	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Snacks (% of intake)	0.0	0.0	0.0	0.0	0	0.0	0.0	0.0	0.0	0
Soups and sauces (% of intake)	0.9	0.8	5.7	84.0	0	1.0	0.9	6.3	11.8	0

[1] Beverages, alcoholic drinks, and miscellaneous foods were not included because of 0% or 0.1% values in intakes of α-linolenic acid, eicosapentaenoic acid, and DHA.
[2] Values are means ± SDs.

DHA intakes

DHA intakes in vegetarians were ≈0.3% of those in fish-eaters, and DHA intakes in meat-eaters were 10.5% (men) and 6.7% (women) of those in fish-eaters (Table 1). In fish-eaters, 90% of DHA came from fish (64% from fatty fish), with meat contributing ≈6% to intake (**Table 4**). In meat-eaters, 93% of DHA was supplied by meats and meat products, with the greatest contribution from poultry; 29% in men and 35% in women. In vegetarians, the major contributors to DHA intake were from foods in the soups and sauces groups (84% and 11.8% in men and women, respectively), and 16% of the DHA intake in men and 88.2% of the intake in women were supplied by eggs.

Relation between diet and plasma n−3 PUFAs in the substudy

In the substudy, as in the whole population, there were significant differences between men and women for age, weight, height, dietary n−3 PUFAs, and linoleic acid and for circulating plasma n−3 PUFA concentrations (**Table 5**). Differences between the dietary-habit groups for weight, height, BMI, and

TABLE 5
Characteristics, dietary intakes, and circulating plasma phospholipid n−3 polyunsaturated fatty acids (PUFAs) and plasma linoleic acid in the substudy in 4902 men and women aged 39–78 y with plasma measures of n−3 PUFAs who were not fish-oil-supplement consumers[1]

			Non-fish-eaters			
	All	Fish-eaters	Meat-eaters	Vegetarians	Vegans	P^2
Men						
n	2646	2257	359	25	5	
Age (y)	64.4 ± 7.7[3,4]	64.7 ± 7.6	63.3 ± 8.3	61.4 ± 9.9	54.4 ± 11.8	<0.001
Weight (kg)	80.6 ± 11.8[3]	80.5 ± 11.7	81.3 ± 12.5	79.6 ± 9.6	80.9 ± 10.5	0.43
Height (cm)	173.0 ± 6.6[3]	173.0 ± 6.6	173.2 ± 6.5	172.1 ± 6.4	172.8 ± 4.7	0.83
BMI (kg/m^2)	26.9 ± 3.4	26.9 ± 3.4	27.0 ± 3.6	26.8 ± 2.8	27.1 ± 3.5	0.44
Current smokers [% (*n*)]	11.9 ± 315	11.0 ± 248	17.6 ± 63	8.0 ± 2	40.0 ± 2	<0.001
Diet (g/d)						
Total n−3 PUFAs	1.46 ± 0.57[3]	1.52 ± 0.57	1.13 ± 0.45	1.16 ± 0.55	0.87 ± 0.61	<0.001
α-Linolenic acid	1.19 ± 0.41[3]	1.21 ± 0.4	1.09 ± 0.45	1.15 ± 0.55	0.84 ± 0.61	<0.001
Eicosapentaenoic acid	0.12 ± 0.15[3]	0.13 ± 0.16	0.02 ± 0.02	0.01 ± 0.01	0.009 ± 0.008	<0.001
Docosahexaenoic acid	0.16 ± 0.21[3]	0.18 ± 0.22	0.02 ± 0.02	0.002 ± 0.007	0	<0.001
Linoleic acid	11.92 ± 4.87[3]	11.99 ± 4.63	11.43 ± 6	13.46 ± 6.5	8.53 ± 9.3	0.135
Plasma (μmol/L)						
α-Linolenic acid	11.1 ± 6.0[3]	10.9 ± 5.7	11.8 ± 7.0	13.6 ± 10.1	15.8 ± 9.7	<0.001
Eicosapentaenoic acid	56.1 ± 41.8[3]	57.5 ± 43.2	47.4 ± 30.3	55.9 ± 45.3	65.1 ± 45.5	0.001
Docosapentaenoic acid	67.7 ± 30.1[3]	67.3 ± 29.4	70.0 ± 33.4	77.5 ± 38.8	67.2 ± 26.8	0.038
Docosahexaenoic acid	236.2 ± 105.5[3]	239.7 ± 106.2	215.6 ± 96.4	222.2 ± 138.4	195.0 ± 58.8	<0.001
Total long-chain n−3 PUFAs	360.0 ± 163.3[3]	364.5 ± 164.8	333.0 ± 147.7	355.5 ± 211.1	327.4 ± 123.6	0.002
Linoleic acid	1171.0 ± 331.4	1164.1 ± 329.5	1207.9 ± 333.3	1238.2 ± 421.6	1337.7 ± 414.1	<0.001
Women						
n	2256	1891	309	51	5	
Age (y)	62.3 ± 8.8	62.4 ± 8.7	61.8 ± 9.3	60.1 ± 9.2	48.4 ± 5.0	0.002
Weight (kg)	68.8 ± 11.9	68.8 ± 11.8	69.1 ± 12.7	66.1 ± 11.5	69.4 ± 9.5	0.53
Height (cm)	160.4 ± 6.2	160.4 ± 6.2	160.1 ± 6.1	160.7 ± 6.6	164.3 ± 6.3	0.91
BMI (kg/m^2)	26.8 ± 4.4	26.7 ± 4.4	27.0 ± 4.7	256 ± 4.1	25.9 ± 4.7	0.70
Current smokers [% (*n*)]	11.7 ± 263	11.5 ± 217	12.3 ± 38	15.7 ± 8	0 ± 0	0.65
Diet (g/d)						
Total n−3 PUFAs	1.18 ± 0.46	1.24 ± 0.46	0.89 ± 0.34	0.87 ± 0.39	0.72 ± 0.33	<0.001
α-Linolenic acid	0.97 ± 0.33	0.99 ± 0.32	0.86 ± 0.33	0.86 ± 0.39	0.71 ± 0.33	<0.001
Eicosapentaenoic acid	0.09 ± 0.12	0.1 ± 0.13	0.02 ± 0.01	0.01 ± 0.01	0.002 ± 0.004	<0.001
Docosahexaenoic acid	0.13 ± 0.17	0.15 ± 0.18	0.01 ± 0.01	0.002 ± 0.007	0 ± 0	<0.001
Linoleic acid	9.18 ± 3.86	9.33 ± 3.73	8.25 ± 3.94	9.02 ± 5.81	10.89 ± 10.86	<0.001
Plasma (μmol/L)						
α-Linolenic acid	12.5 ± 6.3	12.4 ± 6.1	13.1 ± 7.3	12.3 ± 4.8	13.71 ± 8.10	0.22
Eicosapentaenoic acid	63.4 ± 43.0	64.7 ± 43.4	57.1 ± 38.4	55.1 ± 52.5	50.0 ± 29.4	0.001
Docosapentaenoic acid	72.3 ± 30.4	71.8 ± 29.6	74.7 ± 34.2	75.0 ± 32.2	90.6 ± 54.0	0.056
Docosahexaenoic acid	266.0 ± 113.8	271.2 ± 113.1	241.3 ± 109.6	223.5 ± 137.8	286.4 ± 211.7	<0.001
Total long-chain n−3 PUFAs	401.7 ± 170.2	407.7 ± 169.3	373.1 ± 166.2	353.5 ± 191.5	426.8 ± 284.0	<0.001
Linoleic acid	1244.0 ± 334.3	1236.9 ± 328.4	1271.2 ± 373.9	1325.9 ± 278.6	1406 ± 162.1	<0.001

[1] Total dietary n−3 PUFAs represent the sum of α-linolenic acid, eicosapentaenoic acid, and docosahexaenoic acid. Total long-chain n−3 PUFAs represent the sum of eicosapentaenoic acid, docosapentaenoic acid, and docosahexaenoic acid.

[2] *P* for the difference between the 4 dietary groups (fish-eaters and non-fish-eating meat-eaters, vegetarians, or vegans) calculated by using ANOVA (excludes the group labeled "All").

[3] Significant difference between men and women, $P < 0.001$ (2-sample *t* test).

[4] Mean ± SD (all such values)

current smoking in women were not significant; for age were significant in both men and women; and for current smoking habit were significant in men.

Although intakes of total dietary n−3 PUFA in the substudy population and in each of the 4 dietary-habit groups were higher in men than in women, circulating concentrations were lower in men (Table 5).

Circulating concentrations of plasma phospholipid fatty acids (ALA, EPA, DPA, DHA, and total LC n−3 PUFAs) were significantly different between dietary-habit groups in men and women, with the exception of ALA and DPA in women; however, there was a trend toward significance for DPA in women (P = 0.056). Overall, the percentage differences between dietary-habit groups were smaller for total plasma PLLC n−3 PUFAs than for the estimated intakes from diet.

Ratio of ALA intake to the sum of EPA and DHA intakes

The mean (±SD) ratio of PLLC n−3 PUFAs:DALA was higher in women than in men: 0.135 ± 0.982 compared with 0.097 ± 0.062 (28% difference, $P < 0.001$). The maximally adjusted ratio was also higher in women than in men within each of the dietary-habit groups: a difference of 28% in fish-eaters, 29% in meat-eaters, 23% in vegetarians, and 18% in vegans (**Table 6**).

Comparison of the PLLC n−3 PUFAs:DALA ratio between dietary-habit groups showed that it was 209% higher in vegan men and 184% higher in vegan women than in fish-eaters, was 14% higher in vegetarian men and 6% higher in vegetarian women than in fish-eaters, and was 17% and 18% higher in male and female meat-eaters, respectively, than in fish-eaters (Table 6). This suggests that that statistically estimated conversion may be higher in non-fish-eaters than in fish-eaters.

The PLLC n−3 PUFAs:DALA ratio was 0.0327 higher in women than in men ($P < 0.001$) and was 0.0077 higher for those with a current smoking habit (model 3; **Table 7**). The ratio was also 0.0189 higher per SD of circulating linoleic acid ($P < 0.001$), 0.0145 higher per SD of DHA intake, and 0.1054 higher in vegans ($P < 0.001$) than in fish-eaters and 0.0195 higher in meat-eaters than in fish-eaters ($P < 0.001$) and was 0.0103 higher in vegetarians than in fish-eaters but not significantly so (model 3; Table 7).

DISCUSSION

In this study we found clear differences in the total intake and dietary sources of n−3 PUFAs, ALA, EPA, and DHA between fish-eaters and non-fish-eaters. Although the estimated dietary intake of n−3 PUFAs in non-fish-eaters was only between 57% and 80% of that of fish-eaters, the differences between these groups were smaller for plasma n−3 PUFA status. One explanation for this observation may be due to increased conversion, and our data suggest that the precursor-product ratio from plant-derived ALA to circulating LC n−3 PUFAs was significantly greater in non-fish-eaters than in those who ate fish. Although there have been many small, careful metabolic studies determining the extent of the conversion, we believe this to be the first large population study to investigate intakes, status, and the precursor-product ratio by using statistical models as, surrogate, estimates of conversion of ALA to LC n−3 PUFAs in different dietary-habit groups.

TABLE 6
The precursor-product ratio of circulating eicosapentaenoic acid, docosapentaenoic acid, and docosahexaenoic acid to dietary α-linolenic acid (converted to μmol/d) in different dietary-habit groups of 4902 men and women aged 39–78 y[1]

	Fish-eaters			Non-fish-eaters									
				Meat-eaters			Vegetarians			Vegans			
	n	Mean ± SE	95% CI	n	Mean ± SE	95% CI	n	Mean ± SE	95% CI	n	Mean ± SE	95% CI	P^2
Men	2257	—	—	359	—	—	25	—	—	5	—	—	—
Unadjusted	—	0.093 ± 0.001	0.091, 0.096	—	0.101 ± 0.004	0.093, 0.108	—	0.108 ± 0.012	0.085, 0.132	—	0.199 ± 0.027	0.146, 0.252	<0.001
Adjusted[3]	—	0.093 ± 0.001	0.091, 0.096	—	0.101 ± 0.003	0.095, 0.107	—	0.111 ± 0.012	0.088, 0.135	—	0.206 ± 0.027	0.153, 0.258	<0.001
Adjusted[4]	—	0.092 ± 0.001	0.090, 0.095	—	0.108 ± 0.003	0.102, 0.114	—	0.105 ± 0.011	0.083, 0.128	—	0.193 ± 0.025	0.144, 0.248	<0.001
Women	1891	—	—	309	—	—	51	—	—	5	—	—	—
Unadjusted	—	0.129 ± 0.002	0.125, 0.133	—	0.142 ± 0.005	0.132, 0.153	—	0.141 ± 0.011	0.117, 0.164	—	0.230 ± 0.037	0.158, 0.303	0.002
Adjusted[3]	—	0.127 ± 0.002	0.123, 0.131	—	0.141 ± 0.005	0.132, 0.151	—	0.152 ± 0.011	0.130, 0.173	—	0.249 ± 0.037	0.177, 0.320	0.002
Adjusted[4]	—	0.128 ± 0.002	0.124, 0.131	—	0.152 ± 0.005	0.142, 0.161	—	0.136 ± 0.011	0.114, 0.159	—	0.235 ± 0.035	0.165, 0.304	<0.001

[1] The analysis was performed by using linear regression.
[2] P for difference between different dietary-habit groups by ANOVA.
[3] Model 1 adjusted for age, BMI, and smoking habit.
[4] Model 2 adjusted for age, BMI, smoking habit, circulating plasma linoleic acid, dietary eicosapentaenoic acid and docosahexaenoic acid, and meat.

TABLE 7
Linear regression coefficients of the ratio of circulating eicosapentaenoic acid (EPA), docosapentaenoic acid, and docosahexaenoic acid (DHA) to dietary α-linolenic acid (converted to μmol/d) in different dietary-habit groups of 4902 men and women aged 39–78 y[1]

	β	95% CI	P	R^2
Model 1				
Sex (women vs men)	0.0390	0.035, 0.043	<0.001	0.075
Age (per 10 y)	0.0103	0.008, 0.013	<0.001	
BMI, per 4 units (kg/m^2)	0.0014	−0.001, 0.003	0.165	
Current smoking (yes vs no)	0.0053	−0.001,0.011	0.095	
Model 2				
Sex (women vs men)	0.0389	0.035, 0.043	<0.001	0.083
Age (per 10 y)	0.0109	0.009, 0.013	<0.001	
BMI, per 4 units (kg/m^2)	0.0014	−0.001, 0.003	0.174	
Current smoking (yes vs no)	0.0047	−0.001, 0.011	0.135	
Vegans vs fish-eaters	0.117	0.073, 0.160	<0.001	
Vegetarians vs fish-eaters	0.162	0.001, 0.032	0.048	
Meat-eaters vs fish-eaters	0.0111	0.005, 0.017	<0.001	
Model 3				
Sex (women vs men)	0.0327	0.029, 0.037	<0.001	0.168
Age (per 10 y)	0.0105	0.008, 0.013	<0.001	
BMI, per 4 units (kg/m^2)	0.0020	0.000, 0.004	0.038	
Current smoking (yes vs no)	0.0077	0.002, 0.014	0.011	
Plasma linoleic acid, per SD (μmol/L)	0.0189	0.017, 0.021	<0.001	
Dietary EPA, per SD (g/d)	−0.0045	−0.011, 0.001	0.154	
Dietary DHA, per SD (g/d)	0.0145	0.008, 0.021	<0.001	
Meat, per SD (g/d)	−0.0064	−0.008, −0.004	<0.001	
Vegans vs fish-eaters	0.1054	0.063, 0.147	<0.001	
Vegetarians vs fish-eaters	0.0103	−0.005, 0.026	0.201	
Meat-eaters vs fish-eaters	0.0195	0.014, 0.025	<0.001	

[1] The β coefficients represent the difference in the precursor-product ratio according to the variable specified: BMI per 4 units, age per 10 y, smoking (yes vs no), plasma linoleic acid divided by its SD (340 μmol), dietary EPA divided by its SD (0.14 g), dietary DHA divided by its SD (0.20 g), and meat divided by its SD (42.5 g).

Our estimates of total dietary n−3 PUFA intake were lower than in previous studies in UK populations and, although few data for specific long- and short-chain n−3 PUFA intakes exist to compare with our study, our results were similar to one small UK study of younger people (34). However, compared with other European populations, intakes in our study were higher than in France and Belgium for ALA, were of a similar scale for LC n−3 PUFAs in German and Belgian populations, and were lower than in France (35–37). LC n−3 PUFA intakes in fish-eaters were higher than the current UK recommendation of 0.2 g/d; however, neither the whole population nor the dietary-habit groups met the current US recommendations for intakes of ALA in this age group (17, 18, 38–40).

The main food sources of ALA in the whole population and in the dietary-habit groups were the cereals and vegetables food groups. There are few European data for food sources of ALA; however, in contrast with our study, fats and oils supplied most of the ALA in Belgium and France (35, 36). In common with other populations in Belgium, Germany, France, and Norway, fish contributed most to the intake of EPA and DHA in our whole population and in fish-eaters (37, 41–43). However, in the vegetarians in our study, EPA was mainly supplied by fat spreads and dairy foods, and, in meat-eaters, most of the LC n−3 PUFAs was supplied by meats.

Our finding of lower circulating LC n−3 PUFAs in non-fish-eaters than in fish-eaters is similar to that of a recent Austrian study of vegetarians, although other studies found greater differences between meat-eaters and vegetarians of 40–76% and between meat-eaters and vegans of 40–65% (13, 19, 21–23, 44). However, a comparison between different countries is difficult because of differences in the composition of meat-eating and vegetarian diets and in analytic methods for measuring n−3 PUFA status. Our findings of higher circulating linoleic acid concentrations in vegetarians than in meat-eaters confirmed previous studies, and our observation of a higher precursor-product ratio in smokers than in nonsmokers was compatible with results from a metabolic study (13, 23, 29).

In our study, the calculated LC n−3 PUFA:DALA ratio was higher in women than in men, which indicated a greater estimated conversion from dietary ALA and reflected results of previous metabolic and animal studies, possibly because of the effects of estrogen on mRNA expression of the Δ^5- and Δ^6-desaturase genes (9, 11, 24, 44–46). The greater LC n−3 PUFAs:DALA ratio in non-fish-eaters than in fish-eaters also indicated a greater estimated conversion, and, given that intakes of total dietary n−3 PUFAs are lower, it might explain the smaller than expected differences between circulating n−3 PUFA concentrations that we and other studies have found between fish-eaters and non-fish-eaters.

The advantages of this study are that the dietary data were derived from 7-d diaries, which provide good precision for fish intakes, and the sample size was large (20). The limitations of this study are that we were only able to estimate a precursor-product ratio as a statistical estimate of potential conversion, actual conversion was not measured in a metabolic study, and this ratio does not inform the mechanisms of conversion or the metabolic

fate of ALA. However, metabolic studies are, of necessity, small and are unable to investigate relations between intake and circulating concentrations in the general population. We would have liked to include dietary intakes of DPA in our analyses, but were unable to do so because our database, like others, does not include DPA, although we included meat in our fully adjusted models. A further limitation was the potential measurement error caused by misclassification of individuals, but our findings were as expected from metabolic studies for sex differences, and in smokers, which indicated that this is an unlikely explanation.

Current dietary recommendations for maintenance of n−3 PUFA status are to consume one or more portions of oily fish per week; however, the supply of wild fish is dwindling and efforts to conserve the fish supply are needed (14, 38). So, further research to investigate the potential conversion of ALA to long-chain n−3 PUFAs for maintenance of adequate status in non-fish and fish-oil consumers is required.

In conclusion, this study found substantial differences in status and detailed intakes of n−3 PUFAs and their sources in different dietary-habit groups in a general population of middle- and older-aged men and women. The precursor-product ratio of ALA to circulating n−3 PUFAs was significantly greater in women than in men and in non-fish-eaters than in fish-eaters, which indicated a potentially greater estimated conversion. There were smaller differences than expected in status between fish-eaters and non-fish-eaters, which may also be explained by the greater estimated conversion of ALA to LC n−3 PUFAs in the non-fish-eaters. The implications of this study are that, if conversion of plant-based sources of n−3 PUFAs were found to occur in intervention studies, and were sufficient to maintain health, it could have significant consequences for public health recommendations and for preservation of the wild fish supply.

We thank all of the participants in this study and the EPIC-Norfolk study staff at the University of Cambridge, Department of Public Health and Primary Care. Sheila A Rodwell (Bingham), a principal investigator in the EPIC-Norfolk study, read an initial draft of this manuscript; it is with deep regret that we note her death in June 2009.

The authors' responsibilities were as follows—AAW: initiated the study and provided the dietary data, performed the statistical analyses, and wrote the manuscript; and K-TK and NJW: are principal investigators of the EPIC-Norfolk Study. All authors were involved in interpreting the data, contributed to the writing of the manuscript, and read and approved the final manuscript. All authors declared that they had no financial or personal interests in any company or organization sponsoring the research currently or at the time the research was done.

REFERENCES

1. Lee JH, O'Keefe JH, Lavie CJ, Marchioli R, Harris WS. Omega-3 fatty acids for cardioprotection. Mayo Clin Proc 2008;83:324–32.
2. Hooper L, Thompson RL, Harrison RA, et al. Risks and benefits of omega 3 fats for mortality, cardiovascular disease, and cancer: systematic review. BMJ 2006;332:752–60.
3. von Schacky C, Harris WS. Cardiovascular risk and the omega-3 index. J Cardiovasc Med (Hagerstown) 2007;8(suppl 1):S46–9.
4. von Schacky C, Harris WS. Cardiovascular benefits of omega-3 fatty acids. Cardiovasc Res 2007;73:310–5.
5. Nair GM, Connolly SJ. Should patients with cardiovascular disease take fish oil? CMAJ 2008;178:181–2.
6. Balk EM, Horsley TA, Newberry SJ, et al. A collaborative effort to apply the evidence-based review process to the field of nutrition: challenges, benefits, and lessons learned. Am J Clin Nutr 2007;85:1448–56.
7. Davis BC, Kris-Etherton PM. Achieving optimal essential fatty acid status in vegetarians: current knowledge and practical implications. Am J Clin Nutr 2003;78(suppl):640S–6S.
8. Harris WS. Alpha-linolenic acid: a gift from the land? Circulation 2005; 111:2872–4.
9. Brenna JT, Salem N Jr, Sinclair AJ, Cunnane SC. alpha-Linolenic acid supplementation and conversion to n−3 long-chain polyunsaturated fatty acids in humans. Prostaglandins Leukot Essent Fatty Acids 2009; 80:85–91.
10. Plourde M, Cunnane SC. Extremely limited synthesis of long chain polyunsaturates in adults: implications for their dietary essentiality and use as supplements. Appl Physiol Nutr Metab 2007;32:619–34.
11. Burdge GC. Metabolism of alpha-linolenic acid in humans. Prostaglandins Leukot Essent Fatty Acids 2006;75:161–8.
12. Sanders TA, Ellis FR, Dickerson JW. Studies of vegans: the fatty acid composition of plasma choline phosphoglycerides, erythrocytes, adipose tissue, and breast milk, and some indicators of susceptibility to ischemic heart disease in vegans and omnivore controls. Am J Clin Nutr 1978;31: 805–13.
13. Rosell MS, Lloyd-Wright Z, Appleby PN, Sanders TA, Allen NE, Key TJ. Long-chain n−3 polyunsaturated fatty acids in plasma in British meat-eating, vegetarian, and vegan men. Am J Clin Nutr 2005;82: 327–34.
14. Brunner E. Oily fish and omega 3 fat supplements. BMJ 2006;332: 739–40.
15. Brunner EJ, Jones PJ, Friel S, Bartley M. Fish, human health and marine ecosystem health: policies in collision. Int J Epidemiol 2009;38: 93–100.
16. Welch AA, Lund E, Amiano P, et al. Variability of fish consumption within the 10 European countries participating in the European Investigation into Cancer and Nutrition (EPIC) study. Public Health Nutr 2002;5:1273–85.
17. Nelson M. Great Britain. Food standards. Low income diet and nutrition survey. Norwich, United Kingdom: The Stationery Office, 2007.
18. Henderson L, Gregory J, Irving K, et al. The National Diet & Nutrition Survey: adults aged 19 to 64 years; energy, protein, carbohydrate, fat and alcohol intake. London, United Kingdom: Stationery Office, 2003.
19. Lee HY, Woo J, Chen ZY, Leung SF, Peng XH. Serum fatty acid, lipid profile and dietary intake of Hong Kong Chinese omnivores and vegetarians. Eur J Clin Nutr 2000;54:768–73.
20. Welch AA, Bingham SA, Ive J, et al. Dietary fish intake and plasma phospholipid n−3 polyunsaturated fatty acid concentrations in men and women in the European Prospective Investigation into Cancer-Norfolk United Kingdom cohort. Am J Clin Nutr 2006;84:1330–9.
21. Sanders TA, Roshanai F. Platelet phospholipid fatty acid composition and function in vegans compared with age- and sex-matched omnivore controls. Eur J Clin Nutr 1992;46:823–31.
22. Mann N, Pirotta Y, O'Connell S, Li D, Kelly F, Sinclair A. Fatty acid composition of habitual omnivore and vegetarian diets. Lipids 2006;41: 637–46.
23. Kornsteiner M, Singer I, Elmadfa I. Very low n−3 long-chain polyunsaturated fatty acid status in Austrian vegetarians and vegans. Ann Nutr Metab 2008;52:37–47.
24. Burdge GC, Calder PC. Conversion of alpha-linolenic acid to longer-chain polyunsaturated fatty acids in human adults. Reprod Nutr Dev 2005;45:581–97.
25. Harnack K, Andersen G, Somoza V. Quantitation of alpha-linolenic acid elongation to eicosapentaenoic and docosahexaenoic acid as affected by the ratio of n6/n3 fatty acids. Nutr Metab (Lond) 2009;6:8.
26. Barcelo-Coblijn G, Murphy EJ. Alpha-linolenic acid and its conversion to longer chain n−3 fatty acids: benefits for human health and a role in maintaining tissue n−3 fatty acid levels. Prog Lipid Res 2009;48: 355–74.
27. Barcelo-Coblijn G, Murphy EJ, Othman R, Moghadasian MH, Kashour T, Friel JK. Flaxseed oil and fish-oil capsule consumption alters human red blood cell n−3 fatty acid composition: a multiple-dosing trial comparing 2 sources of n−3 fatty acid. Am J Clin Nutr 2008;88:801–9.
28. Goyens PL, Spilker ME, Zock PL, Katan MB, Mensink RP. Conversion of alpha-linolenic acid in humans is influenced by the absolute amounts of alpha-linolenic acid and linoleic acid in the diet and not by their ratio. Am J Clin Nutr 2006;84:44–53.
29. Pawlosky RJ, Hibbeln JR, Salem N Jr. Compartmental analyses of plasma n−3 essential fatty acids among male and female smokers and nonsmokers. J Lipid Res 2007;48:935–43.
30. Day N, Oakes S, Luben R, et al. EPIC-Norfolk: study design and characteristics of the cohort. European Prospective Investigation of Cancer. Br J Cancer 1999;80(Suppl 1):95–103.

31. Bingham SA, Welch AA, McTaggart A, et al. Nutritional methods in the European Prospective Investigation of Cancer in Norfolk. Public Health Nutr 2001;4:847–58.
32. Welch AA, McTaggart A, Mulligan AA, et al. DINER (Data Into Nutrients for Epidemiological Research)—a new data-entry program for nutritional analysis in the EPIC-Norfolk cohort and the 7-day diary method. Public Health Nutr 2001;4:1253–65.
33. Lentjes M, Mulligan AA, Khaw KT, Welch AA. Development of vitamin and mineral database for the EPIC-Norfolk Study. Public Health Nutr (in press).
34. Bakewell L, Burdge GC, Calder PC. Polyunsaturated fatty acid concentrations in young men and women consuming their habitual diets. Br J Nutr 2006;96:93–9.
35. Astorg P, Arnault N, Czernichow S, Noisette N, Galan P, Hercberg S. Dietary intakes and food sources of n−6 and n−3 PUFA in French adult men and women. Lipids 2004;39:527–35.
36. Sioen IA, Pynaert I, Matthys C, De Backer G, Van Camp J, De Henauw S. Dietary intakes and food sources of fatty acids for Belgian women, focused on n−6 and n−3 polyunsaturated fatty acids. Lipids 2006;41:415–22.
37. Bauch A, Lindtner O, Mensink GB, Niemann B. Dietary intake and sources of long-chain n−3 PUFAs in German adults. Eur J Clin Nutr 2006;60:810–2.
38. SACN Great Britain. Advice on fish consumption: benefits and risks. London, United Kingdom: The Stationery Office, 2004. Available from: http://cot.food.gov.uk/pdfs/fishreport2004full.pdf (cited 1 September 2010).
39. Committee on Medical Aspects of Food Policy. Nutritional aspects of cardiovascular disease: report of the Cardiovascular Review Group Committee on Medical Aspects of Food Policy (report on health and social subjects) London, United Kingdom: Her Majesty's Stationery Office, 1994.
40. Otten JJ, Hellwig JP, Meyers LD. DRI, dietary reference intakes: the essential guide to nutrient requirements. Washington, DC: National Academies Press, 2006.
41. Sioen I, Matthys C, De Backer G, Van Camp J, Henauw SD. Importance of seafood as nutrient source in the diet of Belgian adolescents. J Hum Nutr Diet 2007;20:580–9.
42. Flood VM, Webb KL, Rochtchina E, Kelly B, Mitchell P. Fatty acid intakes and food sources in a population of older Australians. Asia Pac J Clin Nutr 2007;16:322–30.
43. Meyer BJ, Mann NJ, Lewis JL, Milligan GC, Sinclair AJ, Howe PR. Dietary intakes and food sources of omega-6 and omega-3 polyunsaturated fatty acids. Lipids 2003;38:391–8.
44. Williams CM, Burdge G. Long-chain n−3 PUFA: plant v. marine sources. Proc Nutr Soc 2006;65:42–50.
45. Extier A, Langelier B, Perruchot MH, et al. Gender affects liver desaturase expression in a rat model of n−3 fatty acid repletion. J Nutr Biochem 2010;21(3):180–7.
46. McNamara RK, Able J, Jandacek R, Rider T, Tso P. Gender differences in rat erythrocyte and brain docosahexaenoic acid composition: role of ovarian hormones and dietary omega-3 fatty acid composition. Psychoneuroendocrinology 2009;34:532–9.

Erratum

Welch AA, Shakya-Shrestha S, Lentjes MAH, Wareham NJ, Khaw K-T. Dietary intake and status of n–3 polyunsaturated fatty acids in a population of fish-eating and non-fish-eating meat eaters, vegetarians, and vegans and the precursor-product ratio of α-linolenic acid to long-chain n–3 polyunsaturated fatty acids: results from the EPIC-Norfolk cohort. Am J Clin Nutr 2010;92:1040–51.

The term ''precursor-product ratio'' used throughout the article would be more correctly called ''product-precursor ratio.'' Despite this name change, the ratio and the data and their interpretation remain correct.

In addition, inaccurate wording appears in the second sentence of the Results section of the abstract (page 1040). As published, the sentence reads: ''Total n–3 PUFA intakes were 57–80% lower in non-fish-eaters than in fish-eaters, but status differences were considerably smaller.'' Instead, the sentence should read as follows: ''Total n–3 PUFA intakes in non-fish-eaters were 57–80% of those in fish-eaters, but status differences were considerably smaller.'' These figures are referred to correctly in the second sentence of the Discussion section on page 1048.

doi: 10.3945/ajcn.110.011346.

Erratum

Sluijs I, van der Schouw YT, van der A DL, et al. Carbohydrate quantity and quality and risk of type 2 diabetes in the European Prospective Investigation into Cancer and Nutrition–Netherlands (EPIC-NL) study. Am J Clin Nutr 2010;92:905–11.

In the second sentence of the Results section of the abstract on page 905, the hazard ratio and 95% CI are incorrect. The sentence should read as follows:

''Dietary GL was associated with an increased diabetes risk after adjustment for age, sex, established diabetes risk factors, and dietary factors [hazard ratio (HR) per SD increase: 1.27; 95% CI: 1.11, 1.44; $P < 0.001$].''

doi: 10.3945/ajcn.110.011361.

Erratum

Freedman DS, Fulton JE, Dietz WH, et al. The identification of children with adverse risk factor levels by body mass index cutoffs from 2 classification systems: the Bogalusa Heart Study. Am J Clin Nutr 2010;92:1298–305.

After our article (1) was published online, we became aware that the FitnessGram cutoffs for body composition had been revised. (This revision was announced in an e-mail dated November 2010.) According to the FitnessGram website (2), the previous standards for percentage body fat and body mass index (BMI) ''were based on the best available research at the time they were developed, . . . but some inconsistencies became apparent.''

The new FitnessGram BMI cutoffs (3) categorize children and adolescents into 4 groups: *1*) very lean, *2*) within the Healthy Fitness Zone, *3*) needs improvement—some risk, and *4*) needs improvement—high risk. The previous and revised cutoffs for the upper categories in FitnessGram, along with the CDC (Centers for Disease Control and Prevention) 85th and 95th percentiles of BMI (4), are shown in **Figure 1**. As noted in our article (1), the previous FitnessGram BMI cutoffs resulted in marked differences (ranging from 2% to 20%) in the prevalence of children who had a high FitnessGram BMI across ages.

The revised FitnessGram BMI cutoffs are fairly close to the CDC 85th percentile, with the cutoffs for ''some risk'' varying from the CDC 79th to 83rd percentiles of BMI by sex and age. The ''high risk'' cutoffs range from the CDC 87th to 91st percentiles. On the basis of these revised cutoffs, it is likely *1*) that the prevalence of children with a high FitnessGram

(*Continued on next page*)