Eating Fish and Risk of Type 2 Diabetes

A population-based, prospective follow-up study

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OBJECTIVE — To investigate the relation between total fish, type of fish (lean and fatty), and eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) intake and risk of type 2 diabetes in a population-based cohort.

RESEARCH DESIGN AND METHODS — The analysis included 4,472 Dutch participants aged ≥55 years without diabetes at baseline. Dietary intake was assessed with a semiquantitative food frequency questionnaire. Hazard ratios (relative risk [RR]) with 95% CIs were used to examine risk associations adjusted for age, sex, lifestyle, and nutritional factors.

RESULTS — After 15 years of follow-up, 463 participants developed type 2 diabetes. Median fish intake, mainly lean fish (81%), was 10 g/day. Total fish intake was associated positively with risk of type 2 diabetes; the RR was 1.32 (95% CI 1.02–1.70) in the highest total fish group (≥28 g/day) compared with that for non–fish eaters ($P_{trend} = 0.04$). Correspondingly, lean fish intake tended to be associated positively with type 2 diabetes (RR highest group]≥23 g/day] 1.30 [95% CI 1.01–1.68]; $P_{trend} = 0.06$), but fatty fish was not. No association was observed between EPA and DHA intake and type 2 diabetes (RR highest group [≥149.4 mg/day] 1.22 [0.97–1.53]). With additional adjustment for intake of selenium, cholesterol, and vitamin D, this RR decreased to 1.05 (0.80–1.38; $P_{trend} = 0.77$).

CONCLUSIONS — The findings do not support a beneficial effect of total fish, type of fish, or EPA and DHA intake on the risk of type 2 diabetes. Alternatively, other dietary components, such as selenium, and unmeasured contaminants present in fish might explain our results.

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Potential benefits of the intake of fish on the development of type 2 diabetes could be attributed to its high content of dietary n-3 polyunsaturated fatty acids, specifically eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Higher EPA and DHA quantities in the phospholipid cell membranes could increase insulin sensitivity (1). EPA and DHA supplementation increased insulin sensitivity in animal models and in some human studies (2). Results of prospective studies on intake of long-chain n-3 fatty acids and type 2 diabetes risk, however, did not show a relation (3,4). Apart from EPA and DHA, other components within fish, such as selenium and vitamin D, could also be related to type 2 diabetes. Vitamin D could be negatively and selenium could be positively associated with type 2 diabetes (5,6).

Results of studies that investigated the association between fish intake and type 2 diabetes risk are inconclusive. An ecological study reported that high fish intake may reduce the risk of type 2 diabetes in populations with a high prevalence of obesity (7). Cross-sectional studies reported inverse (8,9), no (10,11), or positive (12) associations between habitual fish intake and glycemic status. Prospective evidence suggested that fish intake is either inversely (13,14) or not associated (15) with the risk of type 2 diabetes.

Taken together, the effects of fish intake and EPA and DHA intake on the development of type 2 diabetes are ambiguous. Furthermore, studies conducted in this field did not report associations between different types of fish and type 2 diabetes risk. EPA and DHA are present mainly in fatty fish, which might indicate that it is also important to pay attention to the type of fish that is eaten instead of total fish intake alone.

Therefore, we investigated the relation between the intake of total fish, type of fish (lean or fatty), and EPA and DHA and type 2 diabetes risk in a population of men and women aged ≥55 years. We hypothesized that fish intake and especially fatty fish intake is related inversely to the risk of type 2 diabetes.

RESEARCH DESIGN AND METHODS

The current study was conducted within the Rotterdam Study, an ongoing prospective population-based study, which has been described in detail elsewhere (16). In short, 7,983 inhabitants who resided in the district Ommoord of Rotterdam, the Netherlands, and were aged ≥55 years agreed to participate (response rate 78%). Our study population consisted of 4,472 participants because participants without ($n = 2,339$) or with unreliable ($n = 209$) dietary data, those with known or newly diagnosed diabetes at baseline ($n = 516$), and those who had not sufficient clinical or anthropometric data ($n = 447$) were excluded. The Medical Ethics Committee of Erasmus Medical Center (Rotterdam, the Netherlands) approved the study. All participants gave informed consent.

Baseline information

Baseline information on current health status was obtained by a questionnaire and clinical examinations between 1990 and 1993. Anthropometric information was obtained during a visit to the research center. BMI was calculated from height and weight (weight in kilograms divided by the square of height in meters). Waist

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circumference (centimeters) was measured at the level midway between the lower rib margin and the iliac crest with participants in the standing position. Blood pressure was measured at the right brachial artery with a random-zero sphygmomanometer with the participants in a sitting position. The mean of two consecutive measurements was used. Hypertension was defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg and/or use of blood pressure–lowering medication.

Blood samples were used to determine serum total cholesterol by an automated enzymatic procedure using CHOD-PAP reagent (Roche Diagnostics). HDL cholesterol was measured with an HDL cholesterol assay (Roche Diagnostics) using polyethylene glycol–modified enzymes and dextran sulfate. A history of coronary heart disease (CHD) was defined as a self-reported myocardial infarction or angina pectoris with hospital admission. A family history of type 2 diabetes was defined as having a parent, sibling, or both with type 2 diabetes.

For a subsample of the population (n = 2,424), the physical activity level was measured with a physical activity questionnaire (LASA Physical Activity Questionnaire) between 1997 and 2000 (17). Body weight, hours of different activities, and the corresponding MET score were used to calculate energy expenditure (kilocalories per day).

Dietary intake

Dietary assessment comprised a self-administered questionnaire followed by a structured interview with a trained dietitian at the research center. Participants had to mark the foods and drinks they had consumed at least twice a month in the preceding year. Subsequently, the dietitian obtained accurate information on the amount of food eaten using a validated 170–food item semiquantitative food frequency questionnaire (18). Food intake data were converted to energy and nutrient intake using a Dutch Food Composition table (1993). For intake of EPA, DHA, and *trans* fatty acids, a later version was used (2006). The amounts of energy from total fat and saturated fat, carbohydrates, and protein were calculated as a percentage of total energy intake (energy percent).

Total fish intake (grams per day) was divided into four categories: no fish intake and approximate tertiles of fish consumers. The variables lean fish (i.e., plaice, stockfish, cod, fish fingers, perch, pike, octopus, pollack, tuna, and sole) and fatty fish (i.e., mackerel, herring, eel, and salmon) were categorized in the same way. Shellfish intake (i.e., mussels and shrimp) was dichotomized. Participants were categorized as fried fish eaters when they ate pollack or cod. The intake of EPA and DHA (milligrams per day) was divided into tertiles.

Follow-up information

Participants were continuously monitored for major events using the information from general practitioners and pharmacy databases. Information on vital status was obtained regularly from the municipal health authorities in Rotterdam. With this information follow-up data could be censored at time of death for 1,337 (30%) participants.

Incident diabetes cases were defined according to the American Diabetes Association 1997 criteria and the World Health Organization 1999 criteria (fasting plasma glucose level ≥7.0 mmol/l and/or random plasma glucose level ≥11.1 mmol/l and/or use of antidiabetes medication and/or treatment by diet) and registered by a general practitioner as having diabetes. Follow-up data were available until July 2005.

Statistical analysis

To compare baseline characteristics across fish intake categories we used ANOVA for continuous variables and χ^2 tests for categorical variables. Hazard ratios (relative risk [RR]) and 95% CIs were calculated to investigate the association between incident type 2 diabetes and *1*) total fish intake, *2*) lean fish intake, *3*) fatty fish intake, and *4*) EPA and DHA intake. Non–fish consumers, nonconsumers of lean or fatty fish (irrespective of their other fish consumption), and low intake of EPA and DHA were considered as the reference group, respectively.

To evaluate whether the risk of type 2 diabetes differed among the intake categories we performed Cox proportional hazard analyses. In the crude model no adjustments were made. In the first model adjustments were made for age (years), sex, smoking (never, former, or current), and level of education (low [primary education], intermediate [lower vocational, secondary general, or vocational education], or high [higher vocational education or university]). The second model was additionally adjusted for dietary factors, i.e., intake of energy (kilocalories per day), *trans* fatty acids (grams per day), fiber (grams per day), and alcohol (no, low [0–3 g/day], medium [3–14 g/day], or high [≥14 g/day]). For lean fish intake as exposure, fatty fish was included as a confounder in the model and vice versa. To investigate whether other components present in fish confounded the association between EPA and DHA intake and type 2 diabetes, for this association only model 2 was additionally adjusted for intake of selenium (micrograms per day), vitamin D (micrograms per day), and cholesterol (milligrams per day). Other potential confounders including family history of diabetes, medically prescribed diet, and intake of saturated fat, monounsaturated fat, linoleic acid, α-linolenic acid, fruit, vegetables, coffee, and meat were examined but did not affect the results. Potential effect modification by sex was tested.

In additional analysis, the potential intermediates, i.e., BMI (kilograms divided by meters squared), waist circumference (centimeters), total and HDL cholesterol (millimoles per liter), and hypertension (no or yes), were investigated. A linear test for trend across categories was performed based on the median values of each category. All statistical analyses were performed using the statistical program SAS 9.1 for Windows. For all analyses a two-sided P value < 0.05 was considered statistically significant.

RESULTS — The study population consisted of 4,472 participants with an average age of 67.2 ± 7.7 years at baseline. Median follow-up time was 12.4 years during which 463 (10%) incident cases of type 2 diabetes were diagnosed. Mean age of diabetes onset was 73.9 ± 6.9 years. Fifteen participants (0.3%) used fish oil capsules, and 475 (11%) were consuming a prescribed diet.

Zero fish intake was reported by 29% of the population (Table 1). Median fish intake in the total population was 10 g/day. The fish consumed consisted on average of 81% lean fish, 18% fatty fish, and 0.9% shellfish. Of the nonconsumers of lean fish, 88% were also nonconsumers of fatty fish and 43% of the nonconsumers of fatty fish were also nonconsumers of lean fish. Lean fish consumers ate 19 ± 15 g/day, fatty fish consumers ate 9.1 ± 12 g/day, and shellfish consumers ate 4.8 ± 5.0 g/day on average of lean fish, fatty fish, and shellfish, respectively. Median intake

Table 1—*Baseline characteristics of 4,472 Dutch adults aged ≥55 years across categories of total fish intake*

Characteristics	Fish intake				*P*
	No	Low (0–12 g/day)	Moderate (12–28 g/day)	High (≥28 g/day)	
n	1,314	1,061	1,007	1,090	
Age (years)	67.8 ± 8.1	67.6 ± 7.5	66.8 ± 7.5	66.6 ± 7.4	<0.001
Follow-up (years)	10.9 ± 3.7	10.8 ± 3.6	11.0 ± 3.4	10.8 ± 3.6	0.70
Men (%)	38.6	41.9	41.1	42.9	0.16
BMI (kg/m^2)	26.1 ± 3.5	26.3 ± 3.7	26.4 ± 3.8	26.2 ± 3.4	0.31
Waist circumference (cm)	89.1 ± 11.0	90.1 ± 11.0	90.0 ± 10.9	89.8 ± 11.0	0.10
Smokers (%)					0.004
Current	21.8	24.6	22.2	24.0	
Former	40.6	43.4	43.1	46.2	
Never	37.6	32.1	34.7	29.8	
Family history of diabetes (%)	28.2	27.6	26.9	27.9	0.92
History of CHD (%)	11.7	12.7	13.5	10.9	0.28
Hypertension (%)	29.8	32.0	32.7	31.0	0.46
Cholesterol (mmol/l)					
Total cholesterol	6.6 ± 1.3	6.7 ± 1.1	6.7 ± 1.2	6.8 ± 1.2	0.02
HDL cholesterol	1.4 ± 0.36	1.4 ± 0.35	1.4 ± 0.36	1.4 ± 0.40	0.30
Educational level (%)					<0.001
Low	35.5	37.5	31.6	30.9	
Intermediate	54.9	51.7	54.9	53.2	
High	9.6	10.8	13.5	15.9	
Diet prescription (%)	9.6	11.1	11.1	10.9	0.55
Dietary intake					
Energy (kcal/day)	1,962 ± 485	1,953 ± 503	1,989 ± 524	2,025 ± 521	0.004
Carbohydrates (energy %)	45.1 ± 7.1	44.1 ± 7.0	43.9 ± 6.8	43.3 ± 6.8	<0.001
Protein (energy %)	16.5 ± 3.0	16.8 ± 2.9	17.2 ± 3.1	17.8 ± 3.1	<0.001
Total fat (energy %)	36.7 ± 6.4	36.9 ± 6.1	36.5 ± 6.1	35.9 ± 6.1	0.001
Saturated fatty acids (energy %)	14.8 ± 3.2	14.6 ± 3.1	14.2 ± 3.1	14.0 ± 3.3	<0.001
Trans fatty acids (energy %)	1.3 ± 0.48	1.3 ± 0.48	1.2 ± 0.43	1.2 ± 0.45	<0.001
EPA and DHA (mg/day)*	25.2 (13.0–40.7)	63.5 (39.5–95.4)	132 (100–188)	245 (182–374)	<0.001
Fiber (g/day)	16.6 ± 5.0	16.8 ± 5.3	17.1 ± 5.3	17.2 ± 5.0	0.02
Cholesterol (mg/day)	225 ± 80.8	226 ± 79.5	233 ± 80.3	251 ± 85.7	<0.001
Selenium (μg/day)	27.3 ± 7.4	29.5 ± 7.6	33.4 ± 8.1	41.7 ± 11.3	<0.001
Vitamin D (μg/day)	1.9 ± 1.2	2.0 ± 1.2	2.2 ± 1.4	2.6 ± 1.8	<0.001
Alcohol (%)					<0.001
No	23.8	20.4	15.4	16.8	
Low	29.5	30.5	29.7	24.3	
Moderate	26.1	24.4	26.8	26.0	
High	20.6	24.7	28.1	32.9	
Fish (g/day)					
Lean	0	5.1 ± 3.3	14.8 ± 5.5	33.6 ± 15.9	<0.001
Fatty	0	1.3 ± 2.3	2.9 ± 4.7	7.7 ± 14.0	<0.001
Shellfish	0	0.08 ± 0.51	0.21 ± 1.3	0.32 ± 2.0	<0.001

Data are means ± SD or % unless otherwise indicated. *Value expressed as median (interquartile range) because of skewed distribution.

of EPA and DHA was 89 (range 35–187) mg/day. Spearman correlation between intake of total fish and EPA and DHA was 0.87 ($P < 0.01$). Participants with higher total fish intake were younger, had higher total cholesterol levels, and were more likely to drink alcohol (Table 1). Intake of *trans* fatty acids was lower in these participants, whereas the intake of fiber, cholesterol, selenium, and vitamin D was higher ($P < 0.05$ for all variables). Fish intake contributed 13, 12, and 5% to the total intake of selenium, vitamin D, and cholesterol, respectively.

In contrast to our hypothesis, we observed a positive association for total fish intake and diabetes risk (Table 2). The RR for those in the highest group of total fish intake compared with that for the non–fish eaters was 1.32 (95% CI 1.02–1.70; $P_{trend} = 0.04$) when adjusted for lifestyle and dietary factors (model 2). When further adjusted for the intake of fried fish (no or yes), total fish intake was borderline significant (RR in the highest group 1.26 [95% CI 0.97–1.64]; $P_{trend} = 0.06$).

When analyses were stratified for types of fish, lean fish intake tended to be associated with a higher risk (RR 1.30 [95% CI 1.01–1.68; $P_{trend} = 0.06$]), whereas fatty fish did not (0.99 [0.71–1.38]). No significant associations were found for shellfish consumers in any

Table 2—*RRs (95% CIs) for incident type 2 diabetes by fish intake categories in 4,472 Dutch adults aged ≥55 years*

	Categories of fish intake				
Total fish	No	Low (0–12 g/day)	Moderate (12–28 g/day)	High (≥28 g/day)	P_{trend}
Median intake	0	6.6	17.5	35.6	
No. participants/cases	1,314/121	1,061/112	1,007/107	1,090/123	
Person-years	14,267	11,492	11,073	11,819	
Crude model	1	1.15 (0.89–1.49)	1.13 (0.87–1.47)	1.22 (0.95–1.56)	0.18
Model 1	1	1.14 (0.88–1.47)	1.14 (0.88–1.48)	1.23 (0.96–1.58)	0.14
Model 2	1	1.15 (0.89–1.48)	1.19 (0.92–1.54)	1.32 (1.02–1.70)	0.04
Lean fish*	No	Low (0–10 g/day)	Moderate (10–23 g/day)	High (23 g/day)	P_{trend}
Median intake	0	6.5	14.3	30.6	
No. participants/cases	1,488/139	992/110	992/99	1,000/115	
Person-years	16,119	10,769	10,974	10,788	
Crude model	1	1.18 (0.92–1.52)	1.03 (0.80–1.34)	1.22 (0.96–1.57)	0.19
Model 1	1	1.17 (0.90–1.51)	1.05 (0.81–1.37)	1.24 (0.96–1.60)	0.16
Model 2	1	1.15 (0.89–1.49)	1.07 (0.82–1.40)	1.30 (1.01–1.68)	0.06
Fatty fish*	No	Low (0–3 g/day)	Moderate (3–7 g/day)	High (≥7 g/day)	P_{trend}
Median intake	0	1.6	5.3	15.7	
No. participants/cases	3,087/313	461/51	499/57	425/42	
Person-years	33,586	5,124	5,329	4,612	
Crude model	1	1.05 (0.78–1.42)	1.14 (0.86–1.51)	0.97 (0.70–1.34)	0.98
Model 1	1	1.01 (0.74–1.36)	1.07 (0.80–1.43)	0.92 (0.66–1.28)	0.70
Model 2	1	1.04 (0.77–1.42)	1.11 (0.83–1.49)	0.99 (0.71–1.38)	0.93

Data are RRs (95% CIs). Model 1: adjusted for age, sex, smoking, and education level. Model 2: as model 1 with additional adjustments for intake of energy, alcohol, *trans* fatty acids, and fiber. *Models 1 and 2: lean fish is adjusted for fatty fish; fatty fish is adjusted for lean fish.

model compared with nonconsumers of shellfish (RR model 2 1.04 [95% CI 0.61–1.77]) (data not shown).

Furthermore, no associations were observed for EPA and DHA intake (Table 3). The RR was 1.22 (95% CI 0.97–1.53) for the highest level of EPA and DHA intake compared with the lowest. Additional adjustments for intake of selenium, vitamin D, and cholesterol decreased the RR to 1.05 (0.80–1.38).

In a subsample (n = 2,424), energy expenditure was added to model 2, but the RRs did not change appreciably (data not shown). Furthermore, when BMI and waist circumference were taken into account in addition to model 2, the RRs did not alter substantially (RR highest total fish intake 1.29 [95% CI 1.00–1.67]). The other potential intermediates, i.e., total cholesterol, HDL cholesterol, and hypertension, did not change any of the RRs either. Sex did not modify the observed relations (RR highest total fish intake for men 1.38 [0.94–2.02] and for women 1.26 [0.90–1.78]). In all analyses, after exclusion of participants with previous CHD at baseline (n = 544), those who consumed fish oil capsules (n = 15), or those who did not eat fish or meat (n = 18) separately, the results did not change.

Table 3—*RRs (95% CIs) for incident type 2 diabetes by tertiles of EPA and DHA intake in 4,472 Dutch adults aged ≥55 years*

	Tertiles of EPA and DHA intake (mg/day)			
	Low (<49.1 mg/day)	Moderate (49.1–149.4 mg/day)	High (≥149.4 mg/day)	P_{trend}
Median intake	23.8	89.4	236.8	
No. participants/cases	1,490/142	1,491/158	1,491/163	
Person-years	16,085	16,303	16,263	
Crude model	1	1.09 (0.87–1.37)	1.12 (0.89–1.40)	0.38
Model 1	1	1.10 (0.88–1.38)	1.13 (0.90–1.42)	0.33
Model 2	1	1.13 (0.90–1.42)	1.22 (0.97–1.53)	0.11
Model 3	1	1.06 (0.84–1.34)	1.05 (0.80–1.38)	0.77

Data are RRs (95% CIs). Model 1: adjusted for age, sex, smoking, and education level. Model 2: as model 1 with additional adjustments for intake of energy, alcohol, *trans* fatty acids, and fiber. Model 3: as model 2 with additional adjustments for intake of selenium, vitamin D, and cholesterol.

CONCLUSIONS — The results of this prospective study in older Dutch men and women with a low habitual level of fish intake do not support the hypothesis that fish intake could protect against risk of type 2 diabetes. On the contrary, we observed a positive association between total fish intake and type 2 diabetes. This result was mainly due to lean fish intake, which accounted for 81% of total fish intake. Intake of fatty fish and EPA and DHA was not related to type 2 diabetes risk.

In this study, it is unlikely that the association was obscured because of misclassification of diabetes incidence. Onset of diabetes was monitored continuously through general practitioners and follow-up visits. The extensive information

on potential confounders, which minimized the possibility of residual confounding, also strengthened our results. Information about physical activity was available for only a subsample of the population. Adjustment for energy expenditure did not affect the RR, through which it is unlikely that confounding by physical activity explained our results. Another strength of our study is the large reference group, which enabled us to show an association, if one would have existed. Within fish eaters, however, the contrast of fish intake appeared to be small. The total fish intake is rather low (~10 g/day) in this population, which limited the possibility of studying the effects of high fish intake on diabetes onset. Furthermore, the investigation into the effect of fatty fish intake might have been restricted because of the high intake of lean fish relative to fatty fish. We cannot rule out potential misclassification of fish intake due to changes in fish intake during follow-up. However, participants with type 2 diabetes or CHD at baseline who were likely to change their diet as a consequence of disease were excluded or did not change the results, respectively.

In contrast with our findings, two earlier cohort studies showed protective effects of fish intake (13,14). The study of Feskens et al. (13), which showed an odds ratio of 0.47 for fish eaters compared with non–fish eaters, was smaller (59 cases) and had a shorter follow-up period (4 years) than our study. In the Dutch and Finnish cohorts of the Seven Countries Study, which used 2-h blood glucose levels instead of type 2 diabetes risk, a change in fish intake was also associated with a reduced risk ($\beta = -0.18$) (14). In the Nurses Health Study II an association between fish intake and risk of type 2 diabetes was not found (RR ≥2 portions/week vs. <1 portion/week 1.04) (15). Differences in range, type, and preparation of fish might explain in part the observed differences in risk estimates among studies.

Concerning n-3 fatty acids, in line with our study two other prospective studies also showed no association. In the Health Professionals Follow-up Study (3), an RR of 1.01 (upper vs. lower quintile) was observed, and the Iowa Women's Health Study (4) showed an RR of 1.11 between the upper and lower quintiles of long-chain n-3 fatty acid intake and diabetes risk.

The different findings between the intakes of fish and EPA and DHA might be partly explained by the intake of deep-fried fish that is generally lean fish. Deep-fat frying can affect the potential benefits of fish by lowering the EPA and DHA content (19). Indeed, although detailed information on the preparation method was not available, after additional adjustment for fried fish intake, the RR decreased for the highest total fish intake category in our cohort.

Furthermore, the potential beneficial effect of EPA and DHA intake could be counteracted by total cholesterol intake, which was associated significantly with fish intake in our study. Elevated cholesterol levels may impair pancreatic β-cell function and insulin secretion (20). It should be noted, however, that when additional adjustments were made for intake of cholesterol, selenium, and vitamin D, the RR for the highest intake group of EPA and DHA compared with the lowest group was especially reduced after adjustment for selenium intake. Plasma selenium levels increase with increasing fish intake (21) and may increase diabetes risk (22). Selenium supplementation increased diabetes risk in a trial among 1,202 dermatology patients (6) and tended to increase diabetes risk in the Selenium and Vitamin E Cancer Prevention Trial (SELECT) including 35,533 men (23). An adverse effect of selenium on diabetes risk might therefore explain the higher risk in the highest total fish and EPA and DHA intake groups.

Finally, particularly at high exposure levels, it may be that the potential beneficial effect of EPA and DHA was counteracted by ingestion of contaminated fish, especially lean freshwater fish. Mice models showed that elevated blood mercury levels decreased plasma insulin and elevated blood glucose levels (24). Serum concentrations of persistent organic pollutants were strongly related with diabetes prevalence in a cross-sectional study (25). Unfortunately, we do not have information available on intake of contaminants in the current study.

In summary, the findings of this prospective study do not support a protective effect of total fish, type of fish, nor EPA and DHA intake on the development of type 2 diabetes. Total fish intake even appeared to be positively associated with risk of type 2 diabetes in this study. Dietary components and contaminants present in fish should be studied extensively when the potential role of fish in the development of type 2 diabetes is examined further. At this point, given the conflicting results on fish intake and type 2 diabetes risk, we think it is too early to give recommendations regarding fish intake in relation to type 2 diabetes.

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