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Polyunsaturated fatty acids in relation to incident mobility disability and decline in gait speed; the Age, Gene/Environment Susceptibility-Reykjavik Study

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Abstract

BACKGROUND/OBJECTIVES—Low intake of long chain polyunsaturated fatty acids (PUFAs) are associated with physical disability; however, prospective studies of circulating PUFAs are scarce. We examined associations between plasma phospholipid *n* – 3 and *n* – 6 PUFAs with risk of incident mobility disability and gait speed decline.

SUBJECTS/METHODS—Data are from a subgroup of the Age, Gene/Environment Susceptibility–Reykjavik Study, a population-based study of risk factors for disease and disability in old age. In this subgroup (*n* = 556, mean age 75.1 ± 5.0 years, 47.5% men), plasma phospholipid PUFAs were assessed at baseline using gas chromatography. Mobility disability and usual gait speed were assessed at baseline and after 5.2 ± 0.2 years. Mobility disability was defined as the following: having much difficulty, or being unable to walk 500 m or climb up 10 steps; decline in gait speed was defined as change ≥ 0.10 m/s. Logistic regression analyses were performed to determine associations between sex-specific s.d. increments in PUFAs with risk of

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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incident mobility disability and gait speed decline. Odds ratios (95% confidence intervals) adjusted for demographics, follow-up time, risk factors and serum vitamin D were reported.

RESULTS—In women, but not men, every s.d. increment increase of total *n* – 3 PUFAs and docosahexaenoic acid (DHA) was associated with lower mobility disability risk, odds ratio 0.48 (0.25; 0.93) and odds ratio 0.45 (0.24; 0.83), respectively. There was no association between *n* – 6 PUFAs and the risk of incident mobility disability or gait speed decline.

CONCLUSIONS—Higher concentrations of *n* – 3 PUFAs and, particularly, DHA may protect women from impaired mobility but does not appear to have such an effect in men.

INTRODUCTION

Aging is associated with loss of physical function.[1] With the aging of the general population and the considerable prevalence of older persons with mobility disability, identifying modifiable factors that might delay or prevent loss of physical function is important to promote independence and quality of life for older persons.

Nutrient intake is a modifiable factor that may be important for maintaining the health of aging individuals. Long chain polyunsaturated fatty acids (PUFAs), especially eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), have been associated with improved muscle composition[2] or muscle strength.[2–4] In addition, lower intakes of long chain *n* – 3 PUFAs are cross-sectionally associated with worse physical function.[4,5] Data from the InCHIANTI study showed that higher plasma *n* – 3 PUFA levels were associated with lower risk of poor performance after 3 years of follow-up.[6] Data on long chain *n* – 3 PUFA supplementation (fish oil), although limited, suggest a benefit of 1.2 g of EPA and DHA on gait speed among post-menopausal women.[7] Plasma *n* – 6 PUFAs in relation to physical performance decline have only been investigated in one study. No associations were reported,[6] but further studies are needed to confirm these results.

As most previous studies were limited to cross-sectional measures of function and the majority of the studies estimated *n* – 3 PUFAs using questionnaires rather than measurements of circulating PUFA, further studies are needed. In addition, prior studies focused on long chain *n* – 3 PUFAs, and the role of long chain *n* – 6 PUFAs on physical function is less well known.

The aim of the present study is to determine associations between plasma phospholipid *n* – 3 and *n* – 6 PUFAs with incident mobility disability and gait speed decline assessed over 5 years of follow-up in older adults. We hypothesized that participants with higher plasma phospholipid PUFAs would have lower risk of mobility disability and decline in gait speed.

SUBJECTS AND METHODS

Study population

Data are from the Age, Gene/Environment Susceptibility–Reykjavik (AGES-Reykjavik) Study, a single-center, prospective, ongoing population study of survivors from the Reykjavik Study.[8,9] Details of the study design were previously published.[10] Briefly, baseline data collection among 5764 men and women took place from 2002 to 2006. During

a mean follow-up of 5.2 ± 0.2 years, 1039 participants died, 1198 were not willing to participate and 211 were lost to follow-up. Follow-up measurements took place between 2007 and 2011 in 3316 participants.

Participants were drawn from the random cohorts of two sub studies in the AGES-Reykjavik Study (n = 1028) that had data on PUFAs. Participants who met the criteria for magnetic resonance imaging were identified and randomly selected to take part in the Iceland-MI study (n = 702).[11] Participants who were identified as candidates for Iceland-MI but who did not participate were also randomly selected (n = 326). Participants without baseline and follow-up data on mobility disability or gait speed (n = 440) were excluded in the present analytic sample. As we were interested in incident mobility disability, we excluded participants who reported difficulty walking 500 m or climb 10 steps at baseline (n = 32), resulting in 556 participants with complete data to assess incidence. Compared with the included sample, those who were excluded were older at baseline, were less moderate to vigorous physically active and had lower serum 25-hydroxyvitamin D (25OHD) concentration (Supplementary Table 1).

All participants provided written informed consent, and the study was approved (VSN 00-063) by the National Bioethics Committee in Iceland, as well as the Institutional Review Board of the Intramural Research Program of the National Institute on Aging.

Determination of PUFAs

Baseline blood samples were collected following an overnight fast and stored at − 80 °C. PUFAs were measured in plasma phospholipids, which reflect short-term dietary intake and fatty acids available to the periphery. Detailed description of determination has been previously described.[2] In brief, phospholipids were separated from other lipids by one dimensional thin layer chromatography.[12] Fatty acid methyl esters were prepared by direct transesterification[13] and separated using gas chromatography. PUFAs are expressed as a relative percent of the total phospholipid fatty acids analyzed. For this study, we focused on total and individual long chain n – 3 PUFAs (EPA+DPA+DHA), alpha-linolenic acid, total and individual long chain n – 6 PUFAs (linoleic acid+arachidonic acid). All coefficients of variation from pooled quality control samples for EPA, docosapentaenoic acid, DHA, alpha-linolenic acid, linoleic acid and arachidonic acid were all <2.5%.

Determination of self-reported mobility disability

Self-reported mobility disability was assessed at baseline and follow-up with the following questions: ‘Because of health or physical problems do you have any difficulty walking 500 m by yourself or without the use of aids?’, and ‘Do you have any difficulty climbing 10 steps without resting when you are by yourself and without the use of aids?’ Mobility disability was defined as having much difficulty or being unable to walk 500 m and/or climb 10 steps at follow-up.

Determination of gait speed

Gait speed was used as an objective measure of physical performance. Usual 6 m walking speed was assessed using the same standardized protocol at both baseline and follow-up.

Gait speed was calculated by dividing the distance with the walking time expressed in meters per second (m/s). Change in gait speed over time was calculated. A decline of ≥ 0.10 m/s in gait speed, a clinically meaningful change,[14] was used to categorize participants according to whether or not they had a decline in gait speed.

Covariates

All covariates were assessed at the baseline examination. Body mass index (kg/m^2) was calculated from measured weight and height, and waist circumference (cm) was measured using standardized protocols.[10] Education (primary, secondary, college and university education), smoking status (never, former and current) and physical activity (hours per week of moderate to vigorous activity in prior year) were assessed by questionnaire. Circulating vitamin D, 25OHD, was determined using the Liaison chemiluminescence immunoassay (Stillwater, MN, USA) as measured in nmol/l. Blood pressure was assessed from the mean value of two measurements with a large-cuff mercury sphygmomanometer. Medical conditions (hypertension, diabetes, coronary heart disease) were determined from self-report, medications and clinical assessments.

Dietary consumption was assessed by food frequency questionnaire in early life (ages 14–19), midlife (ages 40–50) and later life (AGES-Reykjavik baseline).[15] The food frequency questionnaire assessed frequency of intake of 10 common foods and food groups, including fish and fish oil, using the same questions for all three time periods. The most commonly consumed fish in Iceland are cod and haddock,[16] and both contain low levels of *n* – 3 PUFAs. Therefore, we focused on fish liver oil consumption (referred to as fish oil hereafter), which is rich in *n* – 3 PUFAs, as well as vitamin D. Fish oil consumption was categorized as never, < daily (< once a month, 1–3 times a month, 1–2 times a month or 5–6 times a week) or daily.

Statistical analysis

Because of known differences in physical function in older men and women,[17] we stratified analyses by sex. Differences in baseline characteristics were examined using analysis of variance for continuous variables and χ^2-tests for categorical variables.

Multivariate logistic regression analyses were used to examine baseline PUFAs in relation to odds of developing incident mobility disability and decline in gait speed. Effect estimates were expressed as odds ratios (OR) and corresponding 95% confidence intervals (CI) per sex-specific s.d. increments of PUFAs. Three models were fit; Model 1 was adjusted for age, waist circumference, education and the time between baseline and follow-up examination. Model 2 was adjusted for all variables of Model 1 plus smoking status, physical activity, hypertension, diabetes and coronary heart disease. The prevalence of daily fish oil consumption in the study population is high (62%). Besides being the main source of *n* – 3 PUFAs, it is also rich in vitamin D. Higher concentrations of *n* – 3 PUFAs[18–20] and vitamin D[21] are associated with lower cardiovascular disease events and cardiovascular disease risk factors. In turn, cardiovascular diseases are associated with mobility disability[22] or limited activity.[23,24] Therefore, we additionally adjusted for serum 25OHD in Model 3 to investigate associations between *n* – 3 PUFAs and odds of developing mobility disability

and decline in gait speed, independent of vitamin D status. All models of decline in gait speed were additionally adjusted for gait speed at baseline. All *P*-values are two-tailed (α = 0.05). All analyses were performed using STATA version 12.1 (StataCorp, College Station, TX, USA).

RESULTS

The mean age of the analytic sample was 75.1 ± 5.0 years, with a body mass index of 27.3 ± 3.9 kg/m^2. Differences between men and women are shown in Table 1. Compared with women, men had a larger waist circumference, were more educated, less likely to report never smoking, more physically active, more likely to have coronary heart disease and had higher concentrations of vitamin D, total and individual long chain *n* – 3 PUFAs ($P < 0.05$ for all).

PUFAs in relation to incident mobility disability and decline in gait speed

At follow-up, 17 (6.4%) men and 25 (8.6%) women reported mobility disability. Associations between s.d. increments in PUFAs with risk of mobility disability are presented in Table 2. In women, total long chain *n* – 3 PUFAs were associated with lower risk of mobility disability with minimal (Model 1) and further adjustments for life style factors and diseases (Model 2): OR 0.49 (95% CI 0.26; 0.93). Associations remained after further adjustment for serum 25OHD (Model 3): OR 0.48 (95% CI 0.25; 0.93). The protective effect of total long chain *n* – 3 PUFAs in relation to mobility disability mainly reflected associations between DHA and mobility disability risk. DHA was inversely associated with mobility disability risk in all models, with OR 0.45 (95% CI 0.24; 0.83) in Model 3. No other associations were observed for PUFAs in relation to mobility disability risk for men or for women.

During follow-up, 101 (38.3%) men and 104 (35.6%) women had a clinically significant decline in gait speed. Table 3 depicts associations between s.d. increments in PUFAs with risk of gait speed decline. PUFAs were not associated with risk of gait speed decline in men or in women.

DISCUSSION

In this study, plasma phospholipid long-chain *n* – 3 PUFAs, and in particular DHA, were associated with lower risk of mobility disability in women but not in men. We observed no associations for plasma phospholipid long-chain *n* – 3 PUFAs with decline in gait speed. Plasma phospholipid long chain *n* – 6 PUFAs were not associated with mobility disability or decline in gait speed.

Few studies have investigated relations of plasma phospholipid PUFAs with longitudinal measures of physical function in older populations. Consistent with our finding, results from the InCHIANTI study showed that baseline plasma *n* – 3 PUFAs were inversely associated with the risk of developing impaired physical performance, but no associations for long chain *n* – 6 PUFAs were observed.[6] In a Japanese study, higher intake of long chain *n* – 3 PUFAs was associated with shorter timed up and go tests in men but not in women.[5] In

contrast, another study showed no associations between self-reported *n* – 3 PUFAs and physical performance measures such as chair rise, grip strength and walking speed,[3] casting doubt over the role of *n* – 3 PUFAs in physical function. In addition to our analyses between plasma phospholipid PUFAs with physical function, we determined associations for fish consumption in relation to physical function. Spearman correlations showed that current fish oil consumption was moderately correlated with EPA, $r = 0.42$, and DHA levels, $r = 0.40$ (both $P < 0.001$). Our results do not appear to support a major role for fish oil consumption in relation to mobility disability or decline in gait speed (Supplementary Table 2). This may be explained by the limited number of events per fish oil intake group, which might have resulted in low statistical power to detect significant differences. Our study population is also typified by a high intake of fish and fish oil, which may mean that the reference group has a more favorable fatty acid profile compared with other studies.

A potential mechanism for the observed associated between *n* – 3 PUFAs in relation to lower mobility disability risk is the incorporation of PUFAs into membranes of the skeletal muscle. Several trials in animals[25] and humans[26–28] have shown that supplementation of EPA and DHA increased PUFA content in muscle phospholipids. This leads to improved muscle composition due to the anti-inflammatory properties of long chain *n* – 3 PUFAs.[29–31] Previously, in this study population, we showed that higher concentrations of total PUFAs cross-sectionally were associated with larger muscle size and with greater knee extension strength, and alpha-linolenic acid was associated with increased knee extension strength over time.[2] As muscle size, composition and strength are related to physical function[32,33] this may help explain our finding of an inverse relationship between *n* – 3 PUFA and risk of mobility disability among women. However, the reason for the null relationship in men is unclear. One other study reported gender differences whereby there were relationships between PUFAs and faster time up and go test in men but not women.[5] In our study concentrations of *n* – 3 PUFAs were lower in women than men. It is possible that the odds of detecting associations are greater in women because the reference group includes individuals with a less favorable fatty acid profile compared with the reference group in men. However, further research is needed to fully understand the potential biological differences in these associations. There are also more indirect mechanisms whereby long chain *n* – 3 PUFAs decrease risk of mobility disability such as through their cardioprotective effects, for example, improved lipid profile, lower blood pressure, reduced heart rate and less arrhythmia.[18,20] Those improvements, in turn, are associated with healthy aging[34] and lower risk of physical decline.[35] In addition, *n* – 3 PUFAs are also associated with decreased depression risk, which can indirectly lead to a lower risk of physical inactivity. A recent meta-analysis showed that supplementation of *n* – 3 PUFAs is beneficial in the treatment of patients with diagnosis of major depressive disorder.[36] As depression itself is associated with worse physical function,[37–39] higher PUFAs levels may in turn contribute to more active life, engagement in more physical activity and finally to better health status.

Interestingly, our results suggest that divergent relationships between *n* – 3 PUFAs and mobility disability and gait speed decline. In contrast to what we had hypothesized, we did not observe associations between plasma phospholipid PUFAs and decline in gait speed. Although, both mobility disability and gait speed provide indications of lower extremity

function, mobility disability may capture more advanced decline in physical function as participants had to self-report much difficulty or being unable to walk a quarter mile or climb 10 steps. In comparison, a decline in gait speed of 0.10 m/s while walking on a straight, level surface, while clinically meaningful, may represent changes in function early in the pathway to disability. In our study, there was a higher percentage of participants with a decline in gait speed compared with incident mobility disability, 36.9% versus 7.6%, respectively. It is possible that *n* – 3 PUFAs are involved in processes that manifest later as a more severe mobility impairment. However, a recent randomized controlled trial performed in women showed that gait speed increased after fish oil supplementation of 1.2 g of EPA and DHA per day.[7] It is possible that discrepancies are due to dose effects of fish oil.

Strengths and limitations

A strength of our study is the objective determination of plasma phospholipids fatty acids. This approach offers an advantage in that it reflects the absorption and metabolism of fatty acids and provides a more precise measure of fatty acid status than dietary estimates. It also has the advantage of quantifying individual PUFAs and therefore facilitates identification of fatty acids that may be particularly important for functional outcomes. Further strengths were that physical function was assessed at two time points using both self-reported and objectively measured mobility, and our analytic sample was restricted to participants who reported no or some mobility disability at baseline, which minimizes the possibility of reverse causation. However, our study was not without limitations. Plasma phospholipid PUFAs were determined at baseline only, and therefore we were not able to determine changes in PUFAs levels over time. Having multiple measurements of PUFAs might be of additional value as it would allow us to examine the influence of dynamic circulating PUFAs in relation to risk of incident mobility disability or gait speed decline. In addition, our study is limited by a small sample size. Future large longitudinal studies with multiple measurements of PUFAs could investigate changes in exposure status over time in relation to the physical function measures. Another limitation is our limited external validity as fish oil intake in Iceland is high and, for several decades, has been extremely common; 62% of study participants consumed fish oil daily. Finally, as we excluded individuals with mobility disability at baseline, our sample was likely healthier compared with other populations of comparable age and only consisted of Caucasians; therefore, our results may not be applicable to a general population of older adults.

In conclusion, in this study of older men and women without mobility disability at baseline, we showed that higher concentrations of long chain *n* – 3 PUFAs, especially DHA, are associated with lower mobility disability risk in women after 5 years of follow-up. Other longitudinal studies are needed to confirm these associations and to examine mechanisms.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Baseline characteristics from a subgroup of the AGES-Reykjavik Study

	Men* n = *264	***Women* n = *292***	**P-*value***
Age (years)	75.1 ± 4.69	75.1 ± 5.26	0.993
Body mass index (kg/m^2)	27.0 ± 3.56	27.4 ± 4.12	0.232
Waist circumference (cm)	102.4 ± 9.74	98.0 ± 11.9	< 0.001
Education, n (%)			0.004
< High School	178 (67)	219 (75)	
High School	42 (16)	51 (17)	
Postsecondary	44 (17)	22 (8)	
Smoking status, n (%)			< 0.001
Never	81 (31)	155 (53)	
Former	157 (59)	105 (36)	
Current	26 (10)	32 (11)	
Moderate to vigorous activity (hours/week)	2.07 ± 2.73	1.43 ± 2.36	0.004
Vitamin D (nmol/l)	59.3 ± 26.4	54.4 ± 26.5	0.029
Systolic blood pressure (mm Hg)	143 ± 20	141 ± 18	0.102
Hypertension, *n* (%)	214 (81)	223 (76)	0.178
Type 2 diabetes mellitus, *n* (%)	28 (11)	18 (6)	0.058
Coronary heart disease, *n* (%)	77 (29)	39 (13)	< 0.001
Current fish oil intake, n (%)			0.782
Never	64 (24)	72 (25)	
< Daily	38 (14)	36 (12)	
Daily	162 (61)	183 (63)	
Plasma polyunsaturated fatty acids (relative % of total fatty acids)			
Long chain *n* - 3 PUFAs	10.8 ± 3.11	9.93 ± 2.93	< 0.001
Eicosapentaenoic acid	3.13 ± 1.68	2.74 ± 1.60	0.006
Docosapentaenoic acid	1.20 ± 0.21	1.11 ± 0.18	< 0.001
Docosahexaenoic acid	6.51 ± 1.51	6.08 ± 1.44	< 0.001
Alpha-linoleic acid	0.22 ± 0.07	0.22 ± 0.07	0.844
Long chain *n* - 6 PUFAs	24.6±3.29	25.0 ± 2.75	0.139
Linoleic acid	17.6 ± 3.10	17.9 ± 2.56	0.185
Arachidonic acid	7.02 ± 1.65	7.08 ± 1.70	0.672

Abbreviations: AGES, Age, Gene/Environment Susceptibility; PUFAs, poly-unsaturated fatty acids. Values are presented as mean ± s.d. for continuous variables and number (%) for categorical variables.

Table 2

Associations between plasma phospholipid PUFAs in relation to incident mobility disability risk

	Men* n = *264			***Women* n = *292***		
	Model 1 (OR (95% CI))	***Model 2 (OR (95% CI))***	***Model 3 (OR (95% CI))***	***Model 1 (OR (95% CI))***	***Model 2 (OR (95% CI))***	***Model 3 (OR (95% CI))***
Long chain *n* - 3 PUFAs	0.94 (0.49; 1.78)	1.07 (0.54; 2.14)	1.24 (0.57; 2.70)	0.47 (0.25; 0.89)	0.49 (0.26; 0.93)	0.48 (0.25; 0.93)
Eicosapentaenoic acid	0.83 (0.42; 1.64)	0.96 (0.45; 2.01)	1.05 (0.47; 2.32)	0.53 (0.28; 1.03)	0.55 (0.28; 1.07)	0.55 (0.28; 1.10)
Docosapentaenoic acid	0.79 (0.43; 1.45)	0.82 (0.41; 1.60)	0.85 (0.43; 1.70)	1.02 (0.67; 1.56)	1.07 (0.70; 1.66)	1.12 (0.71; 1.74)
Docosahexaenoic acid	1.11 (0.60; 2.08)	1.22 (0.65; 2.29)	1.46 (0.70; 3.04)	0.45 (0.25; 0.81)	0.46 (0.26; 0.84)	0.45 (0.24; 0.83)
Alpha-linoleic acid	1.16 (0.69; 1.93)	1.12 (0.65; 1.93)	1.11 (0.64; 1.94)	1.36 (0.94; 1.97)	1.42 (0.96; 2.09)	1.40 (0.95; 2.07)
Long chain *n* - 6 PUFAs	0.92 (0.52; 1.65)	0.79 (0.42; 1.48)	0.72 (0.37; 1.41)	1.35 (0.83; 2.19)	1.32 (0.81; 2.16)	1.29 (0.78; 2.15)
Linoleic acid	1.11 (0.60; 2.06)	0.93 (0.46; 1.86)	0.90 (0.44; 1.83)	1.32 (0.83; 2.12)	1.29 (0.80; 2.07)	1.27 (0.78; 2.05)
Arachidonic acid	0.69 (0.34; 1.39)	0.69 (0.34; 1.39)	0.60 (0.28; 1.31)	1.05 (0.67; 1.64)	1.05 (0.65; 1.70)	1.03 (0.63; 1.67)

Abbreviations: CI, confidence interval; OR, odds ratio; PUFA, polyunsaturated fatty acids. Mobility disability was defined as having much difficulty or unable to walk 500 m and/or climb 10 steps at follow-up. Seventeen men and twenty-five women reported having mobility disability. Model 1 was adjusted for age, education, waist circumference and follow-up time. Model 2 was adjusted for model 1 plus smoking status, physical activity, hypertension, diabetes mellitus and heart disease. Model 3 was adjusted for model 2 plus serum vitamin D.

Table 3

Associations between plasma phospholipid PUFAs in relation to risk of decline in gait speed

	Men* n = *264			***Women* n = *292***		
	Model 1 (OR (95% CI))	***Model 2 (OR (95% CI))***	***Model 3 (OR (95% CI))***	***Model 1 (OR (95% CI))***	***Model 2 (OR (95% CI))***	***Model 3 (OR (95% CI))***
Long chain *n* - 3 PUFAs	0.89 (0.67; 1.19)	0.89 (0.67; 1.19)	0.79 (0.57; 1.09)	0.86 (0.65; 1.12)	0.84 (0.63; 1.11)	0.90 (0.66; 1.23)
Eicosapentaenoic acid	0.85 (0.63; 1.14)	0.86 (0.64; 1.16)	0.76 (0.54; 1.06)	0.84 (0.64; 1.11)	0.82 (0.61; 1.10)	0.88 (0.65; 1.20)
Docosapentaenoic acid	1.06 (0.81; 1.39)	1.06 (0.81; 1.41)	1.04 (0.78; 1.37)	1.05 (0.81; 1.36)	1.05 (0.80; 1.38)	1.11 (0.84; 1.47)
Docosahexaenoic acid	0.94 (0.71; 1.24)	0.93 (0.70; 1.23)	0.83 (0.61; 1.14)	0.87 (0.67; 1.15)	0.86 (0.65; 1.14)	0.92 (0.68; 1.25)
Alpha-linoleic acid	0.94 (0.71; 1.24)	0.94 (0.70; 1.25)	0.94 (0.70; 1.25)	1.09 (0.85; 1.41)	1.12 (0.87; 1.46)	1.12 (0.86; 1.45)
Long chain *n* - 6 PUFAs	1.14 (0.86; 1.50)	1.14 (0.86; 1.52)	1.22 (0.91; 1.65)	0.98 (0.75; 1.28)	0.99 (0.75; 1.32)	0.93 (0.70; 1.25)
Linoleic acid	1.22 (0.92; 1.62)	1.26 (0.94; 1.69)	1.30 (0.97; 1.75)	0.99 (0.76; 1.30)	0.99 (0.75; 1.30)	0.96 (0.72; 1.26)
Arachidonic acid	0.90 (0.68; 1.19)	0.86 (0.64; 1.15)	0.90 (0.66; 1.22)	0.98 (0.75; 1.28)	1.01 (0.76; 1.34)	0.97 (0.72; 1.29)

Abbreviations: CI, confidence interval; OR, odds ratio; PUFA, polyunsaturated fatty acids. 101 men and 104 women had a decline in gait speed (decline of ≥ 0.10 m/s). Model 1 was adjusted for age, education, waist circumference, baseline gait speed and follow-up time. Model 2 was adjusted for model 1 plus smoking status, physical activity, hypertension, diabetes mellitus and heart disease. Model 3 was adjusted for model 2 plus serum vitamin D.