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The association of dietary fat and fatty acid intake with ovarian cancer survival: findings from the OOPS, a prospective cohort study

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Abstract

Background Dietary fat and fatty acid intakes impact the occurrence and development of several cancers. However, the evidence regarding fat and fatty acid intake and ovarian cancer (OC) survival is limited.

Methods The Ovarian Cancer Follow-Up Study (OOPS), a prospective cohort study, analyzed data collected from 703 OC patients. Deaths were ascertained via medical records and active follow-up. Dietary intake was derived from a validated food frequency questionnaire. Cox proportional hazard models were used to calculate hazard ratio (HR) and 95% confidence interval (CI) for association evaluation. Furthermore, several subgroup and sensitivity analyses were also performed.

Results A total of 130 patients died during a median follow-up of 37.17 (interquartile: 24.73–50.17) months. Relative to the lowest tertile of intake, patients with the highest tertile of total fat (HR = 1.87, 95% CI = 1.01–3.49), total fatty acid (HR = 2.20, 95% CI = 1.27–3.80), total saturated fatty acid (SFA) (HR = 2.02, 95% CI = 1.22–3.34), shorter-chain SFA (HR = 1.59, 95% CI = 1.03–2.47), long-chain SFA (HR = 1.69, 95% CI = 1.03–2.77), total monounsaturated fatty acid (MUFA) (HR = 1.77, 95% CI = 1.02–3.05), and animal-based MUFA (HR = 2.05, 95% CI = 1.17–3.58) intake had higher all-cause mortality risk. In contrast, individuals in the highest tertile of egg fat (HR = 0.57, 95% CI = 0.35–0.92) and fruit and vegetable fat (HR = 0.48, 95% CI = 0.31–0.75) intake exhibited a reduced risk of all-cause mortality. Additionally, significant positive associations with all-cause mortality were identified for the consumption of several common fatty acids, including capric acid (HR = 1.92, 95% CI = 1.23–3.00), myristic acid (HR = 1.86, 95% CI = 1.15–3.02), palmitic acid (HR = 1.72, 95% CI = 1.07–2.76), stearic acid (HR = 1.93, 95% CI = 1.12–3.31), and oleic acid (HR = 1.96, 95% CI = 1.13–3.40), when comparing the highest to the lowest tertile of intake.

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Conclusions We identified a linkage of higher intake of total fats, total fatty acids, SFAs, shorter-chain SFAs, long-chain SFAs, total MUFAs, and animal-based MUFAs with increased all-cause mortality of OC patients. Conversely, consumption of egg fats and fruit and vegetable fats demonstrated inverse associations with all-cause mortality.

Keywords Cohort study, Diet, Fat, Fatty acid, Ovarian cancer, Survival

Background

Ovarian cancer (OC) is the most fatal gynecologic malignancy due to the absence of early or specific symptoms, and as a result, the disease is often diagnosed at a very late stage [1, 2]. Worldwide, about 324,398 new cases of OC and 206,839 new cases of death occurred in 2022 [3]. Despite advancements in treatment, the age-standardized five-year net survival rate still ranges from 30% to 50% in most countries, with a median survival duration of 3.7 years [4, 5]. Apart from tumor characteristics, limited prognostic factors have been established for OC. Thus, efforts to identify modifiable lifestyle factors that improve OC survival have intensified. Within the last five years, accumulating evidence has indicated that dietary factors might play an important role in OC patient survival [6–9].

Fat is a major component of daily diets and is made up of glycerol and fatty acids. Both fats and fatty acids have an impact on the occurrence and development of several cancers [10–15] through plausible pathways such as systemic inflammation [16], cell metabolic regulation, membrane structure, intracellular signaling, and transcription factor activity [17]. Previously, two Australian prospective studies investigated associations of pre-diagnosis total fat and different types of fat (saturated, monounsaturated, and polyunsaturated) intake with OC patient survival [6, 18]. Only a higher polyunsaturated to monounsaturated fat ratio was found to be associated with better OC patient survival [6]. However, those investigations did not explore the association between dietary fatty acid intake and OC patient survival [6, 18]. Further, other factors such as the source of fats and fatty acids as well as the length of carbon chains were not considered and required further consideration [19, 20].

To fill in the gaps and provide prospective epidemiological evidence, we investigated the associations of dietary fat and fatty acid intake with OC patient survival, using the Ovarian Cancer Follow-Up Study (OOPS).

Methods

Study design and population

The OOPS is a prospective cohort study involving newly diagnosed OC patients, with detailed methodology previously described in the literature [21, 22]. Briefly, among 853 OC patients, 796 patients (93%) were willing to participate and 744 (87%) returned the study questionnaire. We excluded participants from analysis if 11 (10%) or more food items were left blank ($n = 24$) or they reported implausible caloric intakes (< 500 or > 3500 kcal per day) ($n = 17$) [23]. The final analysis included 703 OC patients (Fig. 1). The protocol for OOPS was approved by the Medical Ethics Committee of Shengjing Hospital of China Medical University (2015PS38K). All patients provided written informed consent before participation.

Dietary assessment

Dietary data were obtained using a validated semi-quantitative food frequency questionnaire (FFQ) with reasonable reliability and validity [24]. Detailed information regarding the reliability and validity of the FFQ, the procedures for dietary data collection, and the methodologies for calculating food group consumption and nutrient intake has been comprehensively documented in prior publications [8, 22].

Based on food sources, we separated dietary fats into animal-based fats and plant-based fats. Total fatty acid intake included saturated fatty acids (SFAs), monounsaturated fatty acids (MUFAs), and polyunsaturated fatty acids (PUFAs). According to the length of the carbon chain, SFAs were grouped into shorter-chain SFAs, including saturated butyric (C4), caproic (C6), caprylic (C8), capric (C10), undecanoic (C11), lauric (C12), and tridecanoic (C13) acids and long-chain SFAs, including saturated myristic (C14), pentadecanoic (C15), palmitic (C16), heptadecanoic (C17), stearic (C18), nonadecanoic (C19), arachidic (C20), behenic (C22), and lignoceric (C24) acids. Additionally, MUFAs were further separated into animal-based MUFAs and plant-based MUFAs according to food sources. Based on the position of the double bond, PUFAs were separated into omega-3 PUFAs that included marine omega-3 PUFAs [eicosapentaenoic acid (EPA), docosapentaenoic acid (DPA), docosahexaenoic acid (DHA)], alpha-linolenic acid, parinaric acid, and docosatrienoic acid and into omega-6 PUFAs that included linoleic acid, eicosadienoic acid, arachidonic acid, and docosatetraenoic acid. The reproducibility coefficients were greater than 0.45 for most fats and fatty acids. The crude and energy-adjusted Spearman correlation coefficients between the FFQ and weighed diet records ranged from 0.3 to 0.6 for most fats and fatty acids [24].

Follow-up and outcomes

The vital status (all-cause death) was ascertained through periodic follow-up procedures conducted every six months. These procedures combined active follow-up

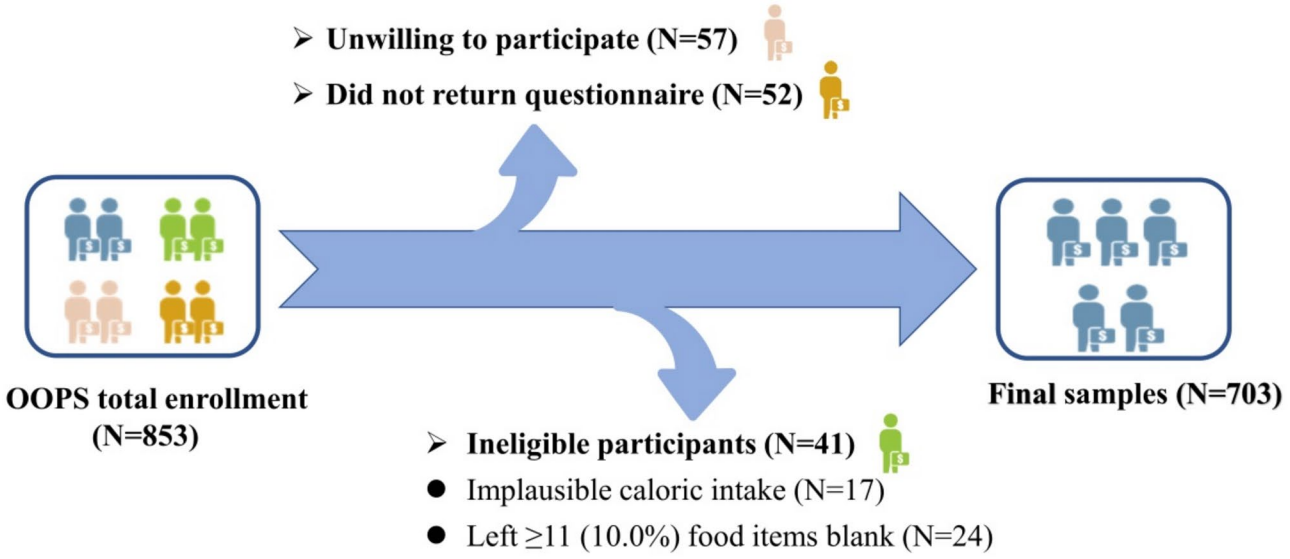

Fig. 1 Flow of participants in the Ovarian Cancer Follow-Up Study (OOPS)

via telephone interviews and passive follow-up through medical records and linkage to the Liaoning Provincial Center for Disease Control and Prevention database. Follow-up duration was calculated as beginning from the date of histologic diagnosis to the time of death or the end of the follow-up (March 31, 2021), whichever came first.

Covariates

The baseline questionnaire included self-reported information on socio-demographic and lifestyle factors, such as physical activity, dietary change, and other factors [22]. Of these, physical activity was calculated by summing the products of time spent on a variety of activities related to work, commuting, household chores, and leisure-time exercise with the average metabolic equivalent task (MET) for that activity during the past year [25, 26]. Dietary change was defined as participants who had deliberately changed their eating habits before diagnosis, with four response options: this year; 1–2 years ago; 3 years ago; and no. Comprehensive documentation of the clinical characteristics of OC patients, as well as the procedures for anthropometric measurements, has been extensively reported in previous studies [9, 22]. Subsequently, we calculated body mass index (BMI) as weight (kg)/height (m^2).

Statistical analysis

Baseline characteristics were summarized with means and standard deviation (SD) or numbers of participants and proportions. Analysis of variance and Chi-square tests or Fisher's exact test were used to assess overall differences across tertiles of energy-adjusted fat and fatty acid intake. Estimates of crude survival probabilities and the plotting of crude survival curves were based on the Kaplan–Meier technique. Intake of all dietary fats and fatty acids was categorized by the tertile distribution. Cox proportional hazards regression analysis was used to calculate the hazard ratios (HRs) and 95% confidence intervals (CIs) for all-cause mortality according to tertiles of pre-diagnosis dietary fat and fatty acid intake, using the lowest tertile as the reference group [27]. The proportional hazards assumptions were tested through modeling an interaction term between each variable within the Cox model and the logarithm of survival time, and no violations were identified (all $P>0.05$). P values for the analysis of linear trends were calculated by assigning the median value of each tertile and entering it as a continuous term in the regression model. Furthermore, several common fatty acid intakes were analyzed separately. In addition, we used pre-diagnosis fat and fatty acid intake as a continuous variable, so that HRs and 95% CIs were calculated per SD increments. We also plotted three knots cubic splines (10th, 50th, and 90th percentiles) to explore the non-linearity association between dietary fat and fatty acid intake and all-cause mortality of OC patients [28].

The selection of covariates in this analysis was based on a combination of prior literature, directed acyclic graphs, clinical significance, and data availability. Ultimately, three types of regression models were fitted to assess the associations. In model 1, we only adjusted for age at diagnosis (continuous, years). To account for lifestyle factors and clinical characteristics, the following variables were included in model 2: smoking status (yes or no), alcohol use (yes or no), education (junior secondary or below, senior high school/technical secondary school, and junior college/university or above), BMI (continuous,

kg/m^2), physical activity (continuous, MET/hours/week), menopausal status (yes or no), parity (≤ 1 or ≥ 2), histological type (serous or non-serous), International Federation of Gynecology and Obstetrics (FIGO) stage (I–II, III–IV, and unknown), histopathologic grade (well, moderately, and poorly differentiated), and residual lesions (none, ≤ 1, and > 1 cm). In model 3, we further adjusted for dietary change (yes or no), total energy (continuous, kcal/day), protein intake (continuous, g/day), and carbohydrate intake (continuous, g/day). In this analysis, all nutrients (including fats, fatty acids, proteins, and carbohydrates) were adjusted for total energy intake using the residual method, which involves regressing the nutrient exposure on total energy intake and entering the residuals from this regression into a secondary model that does not adjust for energy intake [29, 30]. Although the residual method has adjusted nutrient intakes for energy, we still included total energy intake as a covariate to control for potential confounding by total energy itself.

To verify the reliability of the primary results, we conducted numerous subgroup and sensitivity analyses. Subgroup analyses were stratified by age at diagnosis (≤ 50 or > 50 years), menopausal status (no or yes), BMI (< 25 or ≥ 25 kg/m^2), histological type (serous or non-serous), and FIGO stage (I-II or III-IV). Multiplicative interactions were assessed by adding the cross-product of the exposure variable and the stratification variable. In sensitivity analyses, first, we employed the nutrient density method for total energy adjustment to verify the robustness of our findings [30]. Second, we excluded deaths of OC patients occurring within the first year of follow-up to mitigate potential reverse causation. Third, we made further adjustments for age at menarche (continuous, years), oral contraceptive use (yes or no), and family history of OC (yes or no). Last, we mutually adjusted for fats and fatty acids as well as three categories of fatty acids to determine whether the associations were independent of each other. All analyses were performed through SAS version 9.4 (SAS Institute, Cary, NC, USA). Reported *P* values were two-tailed, and differences of $P < 0.05$ were considered statistically significant.

Results

During a median follow-up period of 37.17 (interquartile: 24.73–50.17) months, 130 OC patients died from all causes. Baseline characteristics of the study population depending on total fat and fatty acid intake are summarized in Table 1. Women with higher total fat intake were more likely to consume more dietary proteins, carbohydrates, fiber, and all types of fatty acids (all $P < 0.05$). Further, women who consumed higher dietary fatty acids tended to be poorly educated and consume more dietary proteins, carbohydrates, and various fats, excluding fats derived from fish and eggs (all $P < 0.05$). Additionally, patients with larger residual lesions, non-serous histological subtypes, or advanced FIGO stage had significantly higher all-cause mortality (Supplementary Table 1).

Table 2 shows the HRs and 95% CIs for all-cause mortality according to pre-diagnosis dietary fat and fatty acid intake. Patients in the highest tertile of total fat (HR = 1.87, 95% CI = 1.01–3.49), total fatty acid (HR = 2.20, 95% CI = 1.27–3.80), total SFA (HR = 2.02, 95% CI = 1.22–3.34), shorter-chain SFA (HR = 1.59, 95% CI = 1.03–2.47), long-chain SFA (HR = 1.69, 95% CI = 1.03–2.77), total MUFA (HR = 1.77, 95% CI = 1.02–3.05), and animal-based MUFA (HR = 2.05, 95% CI = 1.17–3.58) intake had higher all-cause mortality, compared to those in the lowest tertile of intake (Supplementary Fig. 1). On the contrary, patients with the highest tertile consumption of egg fats (HR = 0.57, 95% CI = 0.35–0.92) and fruit and vegetable fats (HR = 0.48, 95% CI = 0.31–0.75) exhibited a reduced risk of all-cause mortality compared to those with the lowest tertile of consumption. With the exception of total fats and egg fats, these associations remained significant in continuous variable analysis (per SD increment) and trend analysis (all *P* for trend < 0.05) (Table 2 and Supplementary Fig. 2). Notably, dairy fat intake exhibited a significant positive association with all-cause mortality in the continuous variable analysis (HR = 1.30, 95% CI = 1.10–1.53) and trend analysis (*P* for trend < 0.05), whereas its significance was lost in the third tertile. When mutually adjusted for fats and fatty acids as well as three categories of fatty acids, results were not materially changed (data are not shown). Furthermore, analyses of several common fatty acids indicated that consumption of capric acid (HR = 1.92, 95% CI = 1.23–3.00), myristic acid (HR = 1.86, 95% CI = 1.15–3.02), palmitic acid (HR = 1.72, 95% CI = 1.07–2.76), stearic acid (HR = 1.93, 95% CI = 1.12–3.31), and oleic acid (HR = 1.96, 95% CI = 1.13–3.40) was associated with an increased risk of all-cause mortality when comparing the highest to lowest tertile of intake (Table 3).

When the cohort was stratified by several demographic and clinical characteristics, the results were consistent with the main findings (Supplementary Table 2). For example, the significant positive associations of dietary fat and fatty acid intake with all-cause mortality of OC patients were more frequently observed in those with an age at diagnosis older than 50 years. One exception, however, was total SFA intake, which was significantly associated with all-cause mortality in both age groups (≤ 50 years: HR = 2.82, 95% CI = 1.02–7.83; >50 years: HR = 2.19, 95% CI = 1.19–4.03). The wider CI in younger group suggested reduced statistical precision likely attributable to smaller subgroup sample sizes ($n = 258$ versus 445 in older group). Of note, there were several significant interactions between the stratification variable and dietary fat and fatty acid intake. For instance,

Table 1 General characteristics of ovarian cancer patients by tertiles of total dietary fat and fatty acid intake in the OOPS ($N=703$)

Characteristics	Tertiles of energy-adjusted intake*					
	Total fat			Total fatty acid		
	T1	T2	T3	T1	T2	T3
Range (g/day)	< 31.61	31.61-< 37.66	≥ 37.66	< 20.84	20.84-< 26.17	≥ 26.17
No. of deaths/patients	46/234	35/234	49/235	38/234	39/234	53/235
Mean (SD) age at diagnosis (years)	54.26 (9.65)	52.54 (9.59)	54.09 (9.04)	54.09 (9.45)	52.52 (9.32)	54.27 (9.51)
Mean (SD) follow-up time (months)	33.37 (16.56)	31.12 (16.29)	33.79 (16.66)	32.78 (16.66)	33.65 (16.35)	31.86 (16.58)
Mean (SD) body mass index (kg/m^2)	23.41 (3.89)	23.01 (3.12)	23.32 (3.69)	23.34 (3.98)	23.00 (3.21)	23.40 (3.52)
Mean (SD) physical activity (MET h/d)	15.51 (12.03)	16.11 (10.52)	15.64 (11.18)	15.60 (11.94)	15.83 (11.59)	15.82 (10.17)
Mean (SD) age at menarche (years)	14.83 (1.81)	14.84 (2.03)	14.58 (1.73)	14.84 (1.93)	14.76 (1.92)	14.65 (1.74)
Ever cigarette smoking	24 (10.26)	22 (9.40)	22 (9.36)	19 (8.12)	25 (10.68)	24 (10.21)
Ever alcohol drinking	58 (24.79)	47 (20.09)	44 (18.72)	56 (23.93)	49 (20.94)	44 (18.72)
Ever dietary change	52 (22.22)	51 (21.79)	65 (27.66)	54 (23.08)	52 (22.22)	62 (26.38)
Ever menopause	177 (75.64)	159 (67.95)	172 (73.19)	176 (75.21)	169 (72.22)	163 (69.36)
Ever oral contraceptive use	25 (10.68)	23 (9.83)	22 (9.36)	17 (7.26)	31 (13.25)	22 (9.36)
Ever a family history of ovarian cancer [a]	4 (1.71)	1 (0.43)	0 (0.00)	3 (1.28)	2 (0.85)	0 (0.00)
Parity						
≤ 1	167 (71.37)	169 (72.22)	169 (71.91)	169 (72.22)	166 (70.94)	170 (72.34)
≥ 2	67 (28.63)	65 (27.78)	66 (28.09)	65 (27.78)	68 (29.06)	65 (27.66)
Histological type						
Serous	163 (69.66)	160 (68.38)	156 (66.38)	166 (70.94)	156 (66.67)	157 (66.81)
Non-serous	71 (30.34)	74 (31.62)	79 (33.62)	68 (29.06)	78 (33.33)	78 (33.19)
FIGO stage						
I-II	120 (51.28)	106 (45.30)	116 (49.36)	116 (49.57)	118 (50.42)	108 (45.96)
III-IV	107 (45.73)	116 (49.57)	115 (48.94)	109 (46.58)	107 (45.73)	122 (51.91)
Unknown	7 (2.99)	12 (5.13)	4 (1.70)	9 (3.85)	9 (3.85)	5 (2.13)
Histopathologic grade						
Well differentiated	19 (8.12)	22 (9.40)	15 (6.38)	17 (7.26)	21 (8.97)	18 (7.66)
Moderately differentiated	14 (5.98)	17 (7.26)	17 (7.23)	14 (5.98)	21 (8.97)	13 (5.53)
Poorly differentiated	201 (85.90)	195 (83.34)	203 (86.39)	203 (86.76)	192 (82.06)	204 (86.81)
Residual lesions						
No	180 (76.92)	187 (79.92)	186 (79.15)	183 (78.21)	191 (81.62)	179 (76.17)
≤ 1 cm	35 (14.96)	36 (15.38)	35 (14.89)	34 (14.53)	31 (13.25)	41 (17.45)
> 1 cm	19 (8.12)	11 (4.70)	14 (5.96)	17 (7.26)	12 (5.13)	15 (6.38)
Educational level						
Junior secondary or below	135 (57.69)	130 (55.56)	110 (46.81)	**143 (61.11)**	**121 (51.71)**	**111 (47.22)**
Senior high/technical secondary school	46 (19.66)	46 (19.66)	55 (23.40)	**36 (15.38)**	**49 (20.94)**	**62 (26.38)**
Junior college/university or above	53 (22.65)	58 (24.78)	70 (29.79)	**55 (33.51)**	**64 (27.35)**	**62 (26.38)**
Income per month (Yuan)						
< 5000	141 (60.26)	144 (61.54)	136 (57.87)	143 (61.11)	143 (61.11)	135 (57.45)
5000–10,000	72 (30.77)	60 (25.64)	62 (26.38)	66 (28.21)	57 (24.36)	71 (30.21)
≥ 10,000	21 (8.97)	30 (12.82)	37 (15.75)	25 (10.68)	34 (14.53)	29 (12.34)
Mean (SD) total energy intake (kcal/day)	1503.51 (572.25)	1373.18 (465.08)	1490.43 (603.73)	1527.67 (535.88)	1363.96 (502.05)	1475.55 (604.43)
Mean (SD) total protein intake (g/day)	**52.39 (8.98)**	**57.72 (6.57)**	**65.22 (9.58)**	**53.60 (8.52)**	**57.55 (7.90)**	**64.18 (10.33)**
Mean (SD) total carbohydrate intake (g/day)	**245.66 (24.42)**	**230.49 (13.85)**	**204.64 (17.96)**	**244.59 (23.07)**	**228.57 (18.28)**	**207.62 (20.44)**
Mean (SD) total fiber intake (g/day)	**16.89 (5.77)**	**17.32 (4.45)**	**18.32 (4.99)**	17.58 (5.51)	16.97 (4.74)	17.98 (5.08)
Mean (SD) total fat intake (g/day)	**27.00 (4.74)**	**34.49 (1.81)**	**43.58 (5.25)**	**28.49 (6.26)**	**33.99 (3.89)**	**42.61 (5.97)**
Mean (SD) animal-based fat intake (g/day)	**11.58 (5.54)**	**16.53 (4.68)**	**20.54 (7.20)**	**12.89 (5.74)**	**15.86 (5.59)**	**19.90 (7.48)**
Mean (SD) plant-based fat intake (g/day)	**15.43 (5.50)**	**17.97 (4.57)**	**23.04 (7.25)**	**15.60 (5.75)**	**18.12 (4.63)**	**22.71 (7.31)**
Mean (SD) dairy fat intake (g/day)	**2.16 (2.80)**	**2.80 (2.73)**	**3.19 (3.04)**	**2.07 (2.76)**	**2.85 (2.82)**	**3.23 (2.96)**
Mean (SD) meat fat intake (g/day)	**4.17 (3.46)**	**6.63 (3.95)**	**9.08 (5.72)**	**4.08 (3.35)**	**6.34 (3.78)**	**9.46 (5.68)**
Mean (SD) fish fat intake (g/day)	**1.35 (1.85)**	**1.44 (1.17)**	**1.80 (1.57)**	1.29 (1.74)	1.57 (1.36)	1.73 (1.54)

Table 1 (continued)

Characteristics	Tertiles of energy-adjusted intake*					
	Total fat			Total fatty acid		
	T1	T2	T3	T1	T2	T3
Mean (SD) egg fat intake (g/day)	**3.90 (2.67)**	**5.65 (3.14)**	**6.47 (3.16)**	5.45 (3.44)	5.09 (3.15)	5.48 (2.93)
Mean (SD) grain fat intake (g/day)	**8.09 (5.23)**	**8.52 (3.91)**	**10.33 (6.42)**	**8.31 (4.95)**	**8.54 (4.09)**	**10.09 (6.61)**
Mean (SD) nut fat intake (g/day)	**2.25 (2.74)**	**3.32 (2.64)**	**4.60 (4.56)**	**2.09 (2.32)**	**3.43 (2.93)**	**4.66 (4.56)**
Mean (SD) legume fat intake (g/day)	**3.69 (3.40)**	**4.47 (3.14)**	**6.40 (4.94)**	**3.59 (3.16)**	**4.51 (3.42)**	**6.46 (4.87)**
Mean (SD) fruit and vegetable fat intake (g/day)	1.03 (0.54)	1.07 (0.42)	1.11 (0.48)	**1.10 (0.55)**	**1.05 (0.42)**	**1.06 (0.47)**
Mean (SD) total fatty acid intake (g/day)	**17.75 (5.31)**	**23.28 (3.84)**	**30.36 (6.68)**	**16.37 (4.56)**	**23.46 (1.59)**	**31.56 (5.34)**
Mean (SD) total SFA intake (g/day)	**6.78 (2.59)**	**8.88 (2.25)**	**11.37 (3.32)**	**6.07 (2.01)**	**8.94 (1.68)**	**12.01 (2.96)**
Mean (SD) total MUFA intake (g/day)	**6.65 (2.34)**	**9.11 (1.67)**	**12.07 (3.00)**	**6.16 (2.08)**	**9.14 (1.01)**	**12.53 (2.52)**
Mean (SD) total PUFA intake (g/day)	**4.17 (1.69)**	**5.06 (1.52)**	**6.65 (2.35)**	**3.99 (1.54)**	**5.16 (1.55)**	**6.73 (2.30)**
Mean (SD) shorter-chain SFA intake (g/day) [b]	**0.35 (0.29)**	**0.45 (0.35)**	**0.54 (0.33)**	**0.31 (0.26)**	**0.46 (0.31)**	**0.57 (0.37)**
Mean (SD) long-chain SFA intake (g/day) [c]	**6.68 (2.40)**	**8.78 (2.10)**	**11.24 (3.18)**	**6.01 (1.87)**	**8.82 (1.47)**	**11.87 (2.80)**
Mean (SD) animal-based MUFA intake (g/day)	**3.13 (1.94)**	**4.64 (1.92)**	**6.07 (2.93)**	**3.09 (1.85)**	**4.52 (1.89)**	**6.22 (2.90)**
Mean (SD) plant-based MUFA intake (g/day)	**3.52 (2.08)**	**4.47 (1.80)**	**6.01 (3.04)**	**3.07 (1.84)**	**4.62 (1.63)**	**6.31 (2.93)**
Mean (SD) total omega-3 PUFA intake (g/day) [d]	**0.49 (0.26)**	**0.60 (0.21)**	**0.79 (0.33)**	**0.47 (0.23)**	**0.61 (0.23)**	**0.80 (0.33)**
Mean (SD) marine omega-3 PUFA intake (g/day) [e]	**0.07 (0.11)**	**0.07 (0.08)**	**0.09 (0.09)**	0.06 (0.11)	0.08 (0.08)	0.09 (0.09)
Mean (SD) total omega-6 PUFA intake (g/day) [f]	**3.64 (1.50)**	**4.45 (1.37)**	**5.81 (2.11)**	**3.47 (1.39)**	**4.53 (1.35)**	**5.90 (2.07)**
Mean (SD) omega-6/omega-3 ratio intake	**8.39 (3.02)**	**7.82 (2.22)**	**7.73 (2.54)**	8.23 (2.82)	7.97 (2.55)	7.74 (2.49)

MET, metabolic equivalents of task; MUFA, monounsaturated fatty acid; OOPS, the ovarian cancer follow-up study; PUFA, polyunsaturated fatty acid; SD, standard deviation; SFA, saturated fatty acid; T, tertile

Values are numbers (percentages) unless stated otherwise

Values in bold indicate statistically significant results

*Energy adjustment by the residual method

[a] *P* values were calculated with the use of Fisher's exact test

[b] Short-chain SFA included saturated butyric (C4), caproic (C6), caprylic (C8), capric (C10), undecanoic (C11), lauric (C12), tridecanoic (C13) acids

[c] Long-chain SFA included saturated myristic (C14), pentadecanoic (C15), palmitic (C16), heptadecanoic (C17), stearic (C18), nonadecanoic (C19), arachidic (C20), behenic (C22), lignoceric (C24) acids

[d] Total omega-3 PUFA included alpha-linolenic acid, parinaric acid, docosatrienoic acid, eicosapentaenoic acid (EPA), docosapentaenoic acid (DPA), and docosahexaenoic acid (DHA)

[e] Marine omega-3 PUFA included eicosapentaenoic acid (EPA), docosapentaenoic acid (DPA), and docosahexaenoic acid (DHA)

[f] Total omega-6 PUFA included linoleic acid, eicosadienoic acid, arachidonic acid, and docosatetraenoic acid

significant interactions were emerged between age at diagnosis and dietary total SFA, shorter-chain SFA, long-chain SFA, total PUFA, total omega-3 PUFA, and total omega-6 PUFA intake (all *P* for interaction < 0.05), underscoring the importance of considering age at diagnosis in the dietary analysis of OC mortality.

Sensitivity analyses, which adjusted for energy with nutrient density, yielded similar results (Supplementary Table 3). Further, after excluding deaths occurring within one year of follow-up, we found that the highest tertile of total fatty acid and animal-based MUFA intakes were associated with higher all-cause mortality compared to the lowest tertile of intake (Supplementary Table 3). Moreover, when additional adjustments were made for reproductive factors, the results remained substantially unchanged (Supplementary Table 4).

Discussion

Findings from this prospective cohort study revealed that higher intakes of total fats, total fatty acids, total SFAs, shorter-chain SFAs, long-chain SFAs, total MUFAs, and animal-based MUFAs were significantly associated with higher all-cause mortality in OC patients. In contrast, consumption of egg fats and fruit and vegetable fats was inversely linked to all-cause mortality. Further, several common fatty acids, including capric acid, myristic acid, palmitic acid, stearic acid, and oleic acid, were found to be positively correlated with all-cause mortality among OC patients.

There has been scarce evidence for associations between fat and fatty acid intake and OC survival. To our best knowledge, only two relevant prospective cohort studies from Australia have investigated this topic. These studies only focused on pre-diagnosis total fat and different types of fat intake, and both revealed that total fat, saturated fat, monounsaturated fat, and polyunsaturated fat intakes were unrelated to OC survival [6, 18]. However, our findings indicated that total fat intake, when analyzed categorically, was associated with increased mortality risk. This discrepancy might be attributed to differences in population characteristics and total fat intake levels. Specifically, the study population of Nagle

Table 2 Adjusted hazard ratio (HR) and 95% confidence interval (CI) for the association of dietary fat and fatty acid intake with all-cause mortality of ovarian cancer patients [*]

Characteristics		All-cause mortality			
		Deaths (% of total deaths)	Model 1	Model 2	Model 3
Total fat (g/day)[†]	T1 (< 31.61)	46 (35.38)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (31.61-< 37.66)	35 (26.94)	0.85 (0.55–1.32)	0.89 (0.57–1.40)	1.05 (0.65–1.70)
	T3 (≥ 37.66)	49 (37.68)	1.06 (0.71–1.58)	1.18 (0.78–1.79)	**1.87 (1.01–3.49)**
	Continuous (per SD increment)		0.97 (0.82–1.15)	1.00 (0.85–1.17)	1.14 (0.89–1.45)
	P for trend [**]		0.73	0.39	0.05
Animal-based fat (g/day)[†]	T1 (< 13.11)	37 (28.48)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (13.11-< 18.58)	50 (38.44)	1.36 (0.89–2.09)	1.50 (0.97–2.32)	**1.74 (1.08–2.82)**
	T3 (≥ 18.58)	43 (33.08)	1.14 (0.74–1.78)	1.13 (0.72–1.77)	1.43 (0.83–2.47)
	Continuous (per SD increment)		1.00 (0.84–1.18)	0.99 (0.84–1.18)	1.12 (0.88–1.43)
	P for trend [**]		0.62	0.70	0.28
Dairy fat (g/day)[†]	T1 (< 1.39)	36 (27.69)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (1.39-< 2.89)	41 (31.54)	1.02 (0.65–1.61)	0.98 (0.62–1.56)	1.01 (0.62–1.63)
	T3 (≥ 2.89)	53 (40.77)	**1.54 (1.01–2.35)**	1.50 (0.97–2.31)	1.55 (0.99–2.43)
	Continuous (per SD increment)		**1.21 (1.04–1.41)**	**1.27 (1.08–1.49)**	**1.30 (1.10–1.53)**
	P for trend [**]		**< 0.05**	**< 0.05**	**< 0.05**
Meat fat (g/day)[†]	T1 (< 4.38)	38 (29.24)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (4.38-< 7.64)	46 (35.38)	1.24 (0.81–1.91)	1.17 (0.76–1.80)	1.28 (0.80–2.04)
	T3 (≥ 7.64)	46 (35.38)	1.19 (0.77–1.83)	1.14 (0.74–1.77)	1.37 (0.82–2.28)
	Continuous (per SD increment)		1.04 (0.88–1.24)	1.02 (0.86–1.22)	1.10 (0.89–1.37)
	P for trend [**]		0.49	0.59	0.25
Fish fat (g/day)[†]	T1 (< 0.82)	44 (33.85)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (0.82-< 1.72)	51 (39.23)	1.10 (0.73–1.65)	1.13 (0.74–1.71)	1.19 (0.76–1.85)
	T3 (≥ 1.72)	35 (26.92)	0.65 (0.42–1.01)	0.65 (0.42–1.03)	0.69 (0.42–1.14)
	Continuous (per SD increment)		0.82 (0.67–1.01)	0.82 (0.67–1.01)	0.83 (0.66–1.05)
	P for trend [**]		**< 0.05**	**< 0.05**	0.09
Egg fat (g/day)[†]	T1 (< 3.54)	52 (40.00)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (3.54-< 6.45)	47 (36.15)	0.94 (0.63–1.40)	0.85 (0.57–1.28)	0.87 (0.57–1.32)
	T3 (≥ 6.45)	31 (23.85)	**0.58 (0.37–0.91)**	**0.56 (0.36–0.88)**	**0.57 (0.35–0.92)**
	Continuous (per SD increment)		**0.83 (0.69–0.99)**	**0.81 (0.67–0.97)**	0.81 (0.66-1.00)
	P for trend [**]		**< 0.05**	**< 0.05**	**< 0.05**
Plant-based fat (g/day)[†]	T1 (< 16.48)	43 (33.08)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (16.48-< 20.40)	41 (31.54)	1.05 (0.69–1.62)	0.99 (0.64–1.54)	1.02 (0.65–1.60)
	T3 (≥ 20.40)	46 (35.38)	1.15 (0.76–1.75)	1.25 (0.82–1.92)	1.33 (0.85–2.08)
	Continuous (per SD increment)		0.97 (0.81–1.15)	1.00 (0.85–1.19)	1.02 (0.86–1.21)
	P for trend [**]		0.52	0.28	0.21
Grain fat (g/day)[†]	T1 (< 7.13)	40 (30.77)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (7.13-< 9.65)	36 (27.69)	0.86 (0.55–1.35)	0.86 (0.55–1.36)	0.83 (0.51–1.36)
	T3 (≥ 9.65)	54 (41.54)	1.30 (0.86–1.95)	1.13 (0.74–1.71)	1.07 (0.70–1.66)
	Continuous (per SD increment)		1.07 (0.91–1.25)	1.06 (0.90–1.24)	1.03 (0.88–1.22)
	P for trend [**]		0.18	0.52	0.64
Nut fat (g/day)[†]	T1 (< 2.05)	48 (36.92)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (2.05-< 3.56)	44 (33.85)	0.94 (0.62–1.41)	0.89 (0.59–1.35)	0.88 (0.56–1.38)
	T3 (≥ 3.56)	38 (29.23)	0.79 (0.51–1.21)	0.87 (0.56–1.35)	0.87 (0.55–1.38)
	Continuous (per SD increment)		0.96 (0.80–1.16)	1.01 (0.85–1.21)	1.02 (0.86–1.22)
	P for trend [**]		0.26	0.54	0.59
Legume fat (g/day)[†]	T1 (< 2.97)	44 (33.85)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (2.97-< 5.23)	46 (35.38)	0.97 (0.64–1.47)	0.98 (0.64–1.50)	1.02 (0.65–1.60)
	T3 (≥ 5.23)	40 (30.77)	0.85 (0.56–1.31)	0.87 (0.56–1.35)	0.97 (0.59–1.59)
	Continuous (per SD increment)		0.93 (0.77–1.11)	0.97 (0.80–1.17)	1.04 (0.83–1.31)
	P for trend [**]		0.45	0.51	0.89

Table 2 (continued)

Characteristics		All-cause mortality			
		Deaths (% of total deaths)	Model 1	Model 2	Model 3
Fruit and vegetable fat (g/day)[†]	T1 (< 0.85)	54 (41.54)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (0.85-< 1.20)	39 (30.00)	**0.63 (0.42–0.95)**	**0.58 (0.38–0.88)**	**0.58 (0.37–0.91)**
	T3 (≥ 1.20)	37 (28.46)	**0.56 (0.37–0.85)**	**0.47 (0.31–0.73)**	**0.48 (0.31–0.75)**
	Continuous (per SD increment)		**0.82 (0.68–0.98)**	**0.75 (0.62–0.91)**	**0.76 (0.62–0.93)**
	P for trend [**]		**< 0.05**	**< 0.05**	**< 0.05**
Total FA (g/day)[†]	T1 (< 20.84)	38 (29.23)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (20.84-< 26.17)	39 (30.02)	1.01 (0.64–1.58)	1.14 (0.72–1.80)	1.33 (0.83–2.15)
	T3 (≥ 26.17)	53 (40.75)	1.39 (0.91–2.11)	1.46 (0.95–2.24)	**2.20 (1.27–3.80)**
	Continuous (per SD increment)		1.13 (0.96–1.33)	1.17 (1.00-1.38)	**1.38 (1.12–1.71)**
	P for trend [**]		0.11	0.08	**< 0.05**
Total SFA (g/day)[†]	T1 (< 7.58)	32 (24.63)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (7.58-< 10.13)	45 (34.62)	1.43 (0.91–2.25)	1.37 (0.87–2.16)	1.56 (0.97–2.52)
	T3 (≥ 10.13)	53 (40.75)	**1.68 (1.08–2.61)**	**1.61 (1.03–2.51)**	**2.02 (1.22–3.34)**
	Continuous (per SD increment)		**1.20 (1.03–1.41)**	**1.25 (1.06–1.47)**	**1.31 (1.11–1.56)**
	P for trend [**]		**< 0.05**	**< 0.05**	**< 0.05**
Shorter-chain SFA[a](g/day)[†]	T1 (< 0.31)	36 (27.69)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (0.31-< 0.50)	38 (29.23)	1.05 (0.67–1.66)	1.01 (0.63–1.61)	1.05 (0.65–1.69)
	T3 (≥ 0.50)	56 (43.08)	**1.68 (1.10–2.55)**	**1.55 (1.01–2.38)**	**1.59 (1.03–2.47)**
	Continuous (per SD increment)		**1.27 (1.11–1.46)**	**1.30 (1.12–1.51)**	**1.30 (1.12–1.52)**
	P for trend [**]		**< 0.05**	**< 0.05**	**< 0.05**
Long-chain SFA[b](g/day)[†]	T1 (< 7.46)	37 (28.48)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (7.46-< 9.92)	41 (31.54)	1.12 (0.72–1.75)	1.12 (0.71–1.75)	1.23 (0.78–1.97)
	T3 (≥ 9.92)	52 (39.98)	1.41 (0.93–2.16)	1.38 (0.90–2.11)	**1.69 (1.03–2.77)**
	Continuous (per SD increment)		**1.18 (1.01–1.38)**	**1.22 (1.03–1.43)**	**1.30 (1.09–1.54)**
	P for trend [**]		0.10	0.14	**< 0.05**
Total MUFA (g/day)[†]	T1 (< 7.98)	39 (30.02)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (7.98-< 10.35)	41 (31.54)	1.01 (0.65–1.57)	1.12 (0.71–1.76)	1.29 (0.80–2.09)
	T3 (≥ 10.35)	50 (38.44)	1.25 (0.82–1.89)	1.30 (0.84-2.00)	**1.77 (1.02–3.05)**
	Continuous (per SD increment)		1.10 (0.93–1.30)	1.12 (0.95–1.31)	**1.26 (1.04–1.54)**
	P for trend [**]		0.29	0.23	**< 0.05**
Animal-based MUFA (g/day)[†]	T1 (< 3.45)	31 (23.85)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (3.45-< 5.21)	51 (39.23)	**1.73 (1.11–2.70)**	**1.82 (1.16–2.86)**	**2.24 (1.36–3.67)**
	T3 (≥ 5.21)	48 (36.92)	1.50 (0.96–2.37)	1.45 (0.92–2.29)	**2.05 (1.17–3.58)**
	Continuous (per SD increment)		1.11 (0.94–1.31)	1.09 (0.92–1.29)	**1.29 (1.03–1.63)**
	P for trend [**]		0.12	0.18	**< 0.05**
Plant-based MUFA (g/day)[†]	T1 (< 3.82)	44 (33.84)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (3.82-< 5.12)	37 (28.48)	0.90 (0.58–1.39)	0.94 (0.60–1.46)	0.95 (0.60–1.50)
	T3 (≥ 5.12)	49 (37.68)	1.15 (0.76–1.73)	1.43 (0.94–2.19)	1.53 (0.99–2.39)
	Continuous (per SD increment)		1.02 (0.86–1.21)	1.07 (0.91–1.26)	1.07 (0.91–1.26)
	P for trend [**]		0.47	0.09	0.06
Total PUFA (g/day)[†]	T1 (< 4.32)	44 (33.84)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (4.32-< 5.75)	46 (35.38)	1.02 (0.67–1.55)	1.09 (0.71–1.67)	1.19 (0.75–1.90)
	T3 (≥ 5.75)	40 (30.78)	0.89 (0.58–1.36)	1.07 (0.69–1.65)	1.33 (0.78–2.29)
	Continuous (per SD increment)		0.98 (0.82–1.16)	1.05 (0.88–1.26)	1.23 (0.97–1.57)
	P for trend [**]		0.55	0.80	0.31
Total omega-3 PUFA[c](g/day)[†]	T1 (< 0.48)	49 (37.68)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (0.48-< 0.69)	41 (31.54)	0.79 (0.52–1.20)	0.77 (0.51–1.18)	0.80 (0.50–1.27)
	T3 (≥ 0.69)	40 (30.78)	0.74 (0.49–1.13)	0.77 (0.50–1.18)	0.82 (0.47–1.46)
	Continuous (per SD increment)		0.95 (0.80–1.13)	0.99 (0.82–1.19)	1.16 (0.89–1.52)
	P for trend [**]		0.18	0.27	0.55

Table 2 (continued)

Characteristics		All-cause mortality			
		Deaths (% of total deaths)	Model 1	Model 2	Model 3
Marine omega-3 PUFA[d](g/day)[†]	T1 (< 0.04)	42 (32.31)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (0.04-< 0.08)	46 (35.38)	1.07 (0.70–1.63)	1.01 (0.66–1.54)	1.09 (0.69–1.74)
	T3 (≥ 0.08)	42 (32.31)	0.91 (0.59–1.39)	0.85 (0.55–1.32)	0.93 (0.57–1.50)
	Continuous (per SD increment)		0.93 (0.77–1.14)	0.93 (0.76–1.14)	0.96 (0.77–1.20)
	P for trend [**]		0.57	0.42	0.66
Total omega-6 PUFA[e](g/day)[†]	T1 (< 3.79)	46 (35.38)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (3.79-< 5.00)	44 (33.84)	0.92 (0.61–1.39)	0.88 (0.57–1.35)	0.92 (0.58–1.47)
	T3 (≥ 5.00)	40 (30.78)	0.84 (0.55–1.28)	0.97 (0.63–1.49)	1.13 (0.67–1.90)
	Continuous (per SD increment)		0.99 (0.83–1.17)	1.06 (0.89–1.27)	1.21 (0.97–1.50)
	P for trend [**]		0.43	0.94	0.60
Omega-6/omega-3 ratio[†]	T1 (< 6.75)	44 (33.84)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
	T2 (6.75-< 8.48)	43 (33.08)	1.07 (0.70–1.64)	1.07 (0.69–1.64)	1.05 (0.68–1.63)
	T3 (≥ 8.48)	43 (33.08)	1.04 (0.69–1.59)	1.15 (0.75–1.76)	1.07 (0.68–1.69)
	Continuous (per SD increment)		1.04 (0.87–1.24)	1.08 (0.90–1.30)	1.04 (0.85–1.27)
	P for trend [**]		0.86	0.53	0.78

CI, confidence interval; FA, fatty acid; HR, hazard ratio; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid; Ref, reference; SD, standard deviation; SFA, saturated fatty acid; T, tertile

Values in bold indicate statistically significant results

[*] HR and 95% CI were calculated through the Cox proportional hazards regression model

[†] Adjusted for energy by the residual method

[**] Test for trend based on variables containing the median value for each tertile

[a] Shorter-chain SFA included saturated butyric (C4), caproic (C6), caprylic (C8), capric (C10), undecanoic (C11), lauric (C12), tridecanoic (C13) acids

[b] Long-chain SFA included saturated myristic (C14), pentadecanoic (C15), palmitic (C16), heptadecanoic (C17), stearic (C18), nonadecanoic (C19), arachidic (C20), behenic (C22), and lignoceric (C24) acids

[c] Total omega-3 PUFA included alpha-linolenic acid, parinaric acid, docosatrienoic acid, eicosapentaenoic acid (EPA), docosapentaenoic acid (DPA), and docosahexaenoic acid (DHA)

[d] Marine omega-3 PUFA included eicosapentaenoic acid (EPA), docosapentaenoic acid (DPA), and docosahexaenoic acid (DHA)

[e] Total omega-6 PUFA included linoleic acid, eicosadienoic acid, arachidonic acid, and docosatetraenoic acid

Model 1 was adjusted for age at diagnosis

Model 2 same as Model 1 and further adjusted for education, smoking status, alcohol use, body mass index, physical activity, FIGO stage, histological type, histopathologic grade, menopausal status, parity, residual lesions

Model 3 same as Model 2 and further adjusted for dietary change, total energy, protein intake, and carbohydrate intake

et al. [18] and Playdon et al. [6] were both from Australia with a late FIGO stage (III–IV: Nagle et al., 65%; Playdon et al., 71%) and an older age at diagnosis (≥ 50 years: Nagle et al., 66%; Playdon et al., 82%), while our study population was from China with a relatively earlier FIGO stage (III–IV: 46%) and a younger age at diagnosis (> 50 years: 63%). Furthermore, the median of the highest category for total fat intake in the studies of Nagle et al. [18] and Playdon et al. [6] were both 86 g/day, whereas this value in our study was 42 g/day (energy-adjusted) or 51 g/day (unadjusted). In addition, a randomized controlled trial from the Women's Health Initiative, across 40 clinical centers in the United States, evaluated the association between a low-fat dietary pattern and OC mortality. After a cumulative follow-up of 17.7 years, this study found no link between low-fat dietary interventions and either OC mortality or overall mortality [31]. Therefore, future studies are needed to further explore the association between dietary fat intake and OC mortality across diverse populations.

Notably, findings from the current study suggested that the association between fat intake and OC mortality might depend on food sources. Specifically, egg fat and fruit and vegetable fat intakes were inversely associated with OC mortality. Although significance in categorical comparisons was diminished for dairy fat intake, it was positively associated with increased mortality in continuous and trend analyses. These might be attributed to the distinct characteristics and biological activities of fats from various sources. For instance, dairy fat has been associated with increased circulating levels of estrogen and insulin-like growth factor 1 [32, 33], both of which have been implicated in promoting OC progression [34, 35]. Conversely, egg fat demonstrates excellent functional properties, including anti-inflammatory and antioxidant activities [36, 37], which may play a protective role in slowing disease progression in patients with OC.

Table 3 Adjusted hazard ratio (HR) and 95% confidence interval (CI) for all-cause mortality by the tertiles of common fatty acid intake among ovarian cancer patients

Characteristics		All-cause mortality (*N* = 703)	
		Deaths (% of total)	HR (95% CI)
Capric acid (g/day)†	T1 (< 0.08)	34 (26.15)	1.00 (Ref)
	T2 (0.08-< 0.13)	37 (28.46)	1.22 (0.74–1.99)
	T3 (≥ 0.13)	59 (45.38)	**1.92 (1.23-3.00)**
	P for trend *		**< 0.05**
Lauric acid (g/day)†	T1 (< 0.19)	37 (28.46)	1.00 (Ref)
	T2 (0.19-< 0.30)	37 (28.46)	1.04 (0.65–1.68)
	T3 (≥ 0.30)	56 (43.08)	1.49 (0.96–2.29)
	P for trend *		**< 0.05**
Myristic acid (g/day)†	T1 (< 0.45)	32 (24.62)	1.00 (Ref)
	T2 (0.45-< 0.70)	47 (36.15)	1.41 (0.87–2.28)
	T3 (≥ 0.70)	51 (39.23)	**1.86 (1.15–3.02)**
	P for trend *		**< 0.05**
Palmitic acid (g/day)†	T1 (< 5.30)	39 (30.00)	1.00 (Ref)
	T2 (5.30-< 6.98)	35 (26.92)	1.03 (0.63–1.69)
	T3 (≥ 6.98)	56 (43.08)	**1.72 (1.07–2.76)**
	P for trend *		**< 0.05**
Stearic acid (g/day)†	T1 (< 1.42)	35 (26.92)	1.00 (Ref)
	T2 (1.42-< 2.00)	45 (34.62)	**1.78 (1.08–2.94)**
	T3 (≥ 2.00)	50 (38.46)	**1.93 (1.12–3.31)**
	P for trend *		**< 0.05**
Arachidic acid (g/day)†	T1 (< 0.03)	44 (33.85)	1.00 (Ref)
	T2 (0.03-< 0.05)	42 (32.31)	1.00 (0.62–1.63)
	T3 (≥ 0.05)	44 (33.85)	1.25 (0.70–2.24)
	P for trend *		0.41
Palmitoleic acid (g/day)†	T1 (< 0.36)	35 (26.92)	1.00 (Ref)
	T2 (0.36-< 0.51)	49 (37.69)	**1.66 (1.02–2.71)**
	T3 (≥ 0.51)	46 (35.38)	1.56 (0.89–2.74)
	P for trend *		0.23
Oleic acid (g/day)†	T1 (< 6.99)	39 (30.00)	1.00 (Ref)
	T2 (6.99-< 9.12)	40 (30.77)	1.27 (0.78–2.06)
	T3 (≥ 9.12)	51 (39.23)	**1.96 (1.13–3.40)**
	P for trend *		**< 0.05**
Erucic acid (g/day)†	T1 (< 0.10)	47 (36.15)	1.00 (Ref)
	T2 (0.10-< 0.21)	28 (21.54)	**0.54 (0.32–0.91)**
	T3 (≥ 0.21)	55 (42.31)	1.15 (0.75–1.77)
	P for trend *		0.10
Linoleic acid (g/day)†	T1 (< 3.77)	45 (34.62)	1.00 (Ref)
	T2 (3.77-< 4.97)	45 (34.62)	0.95 (0.60–1.52)
	T3 (≥ 4.97)	40 (30.77)	1.15 (0.69–1.94)
	P for trend *		0.56
α-linolenic acid (g/day)†	T1 (< 0.41)	45 (34.62)	1.00 (Ref)
	T2 (0.41-< 0.57)	43 (33.08)	0.84 (0.52–1.34)
	T3 (≥ 0.57)	42 (32.31)	1.06 (0.62–1.82)
	P for trend *		0.69
Arachidonic acid (g/day)†	T1 (< 0.01)	41 (31.54)	1.00 (Ref)
	T2 (0.01-< 0.02)	46 (35.38)	1.13 (0.71–1.79)
	T3 (≥ 0.02)	43 (33.08)	0.93 (0.55–1.58)
	P for trend *		0.73

Table 3 (continued)

Characteristics		All-cause mortality (*N* = 703)	
		Deaths (% of total)	HR (95% CI)
Eicosapentaenoic acid (g/day)[†]	T1 (< 0.02)	38 (29.23)	1.00 (Ref)
	T2 (0.02-< 0.04)	51 (39.23)	1.38 (0.86–2.20)
	T3 (≥ 0.04)	41 (31.54)	1.05 (0.64–1.72)
	P for trend [*]		0.87
Docosahexaenoic acid (g/day)[†]	T1 (< 0.01)	43 (33.08)	1.00 (Ref)
	T2 (0.01-< 0.04)	45 (34.62)	1.05 (0.66–1.68)
	T3 (≥ 0.04)	42 (32.31)	0.88 (0.55–1.42)
	P for trend [*]		0.51

CI, confidence interval; HR, hazard ratio; Ref, reference; T, tertile

Values in bold indicate statistically significant results

[*] HR and 95% CI were calculated through the Cox proportional hazards regression model with adjustment for age at diagnosis, education, smoking status, alcohol use, body mass index, physical activity, FIGO stage, histological type, histopathologic grade, menopausal status, parity, residual lesions, dietary change, total energy, total protein intake, and total carbohydrate intake

[†] Adjusted for energy by the residual method

[*] Test for trend based on variables containing the median value for each tertile

Furthermore, while fruits and vegetables are relatively low in fats, they are abundant in antioxidant vitamins and phytochemicals, which may synergize with their fat content to exert beneficial effects on OC survival [38, 39]. These findings highlight the critical need to consider specific dietary fat sources in evaluating their impact on OC mortality; however, further research is required to validate these observations.

Although no study has investigated the impact of fatty acid intake on OC survival, our findings were similar in part to several studies exploring other health outcomes such as all-cause mortality and cancer-specific mortality [10, 11]. For example, a recent meta-analysis synthesizing data from 101 prospective cohort studies revealed a significant positive correlation between SFA intake and cancer-specific mortality [10]. Similarly, a large prospective cohort study encompassing 521,120 participants observed a significant positive association between SFA intake and all-cause mortality, cardiovascular disease mortality, cancer-specific mortality, respiratory disease mortality, diabetes mortality, infectious disease mortality, and chronic liver disease mortality [11]. These suggested that dietary SFA intake might be positively linked to various mortality outcomes, aligning with the findings of the present study. Regarding the chain length of SFAs, a systematic review indicated that long-chain SFAs appeared to be positively related to the risk of cardiovascular diseases, whereas short-chain and medium-chain SFAs might exhibit neutral or beneficial associations with cardiovascular disease risk [40]. Moreover, a cohort study involving 525 Swedish prostate cancer patients suggested that shorter-chain SFA intake might be associated with increased mortality risk in patients with localized prostate cancer [19]. An analysis of data from the China Health and Nutrition Survey found a significant positive association between long-chain SFA intake and all-cause mortality among Chinese women [41]. These findings support the positive association of both shorter-chain and long-chain SFA intake with OC mortality observed in the current study.

In our study, the positive association of MUFAs with all-cause mortality in OC patients was mainly driven by animal-based MUFAs. This might be consistent with previous research on the association between MUFA intake and other survival outcomes. For instance, a large-scale study revealed that higher animal-derived MUFA intake was associated with increased all-cause mortality, cardiovascular mortality, and cancer mortality, while plant-derived MUFA intake showed no significant association with risk of death from cardiovascular disease and cancer [12]. These findings underscore the importance of differentiating the sources of MUFA in individual dietary choices.

Previous studies exploring the association between common fatty acid intake and various survival outcomes also support the findings of this study. For example, a study revealed that palmitic acid and stearic acid intakes were positively associated with cardiovascular disease mortality, and higher intake of capric acid, lauric acid, and myristic acid was linked to increased cerebrovascular disease mortality [42]. Similarly, a National Health and Nutrition Examination Survey in the United States found that capric acid, myristic acid, palmitic acid, and stearic acid intakes were positively associated with overall mortality among middle-aged and older adults (age ≥ 50 years) [43]. Furthermore, oleic acid, a key component of MUFAs, has been found to be positively associated with OC mortality. And evidence suggested that the association of oleic acid with mortality might be influenced by its food source (animal or plant) [44], aligning with the analysis for MUFA in our study. Given the lack of relevant

literature, further studies are warranted to confirm these associations.

The distinction between fats and fatty acids requires further clarification. Fat is composed of one glycerol molecule and three fatty acid molecules, with its function largely determined by the properties of its constituent fatty acids. This aligns with our findings, where significant associations with OC mortality were more pronounced for various fatty acid intake, such as SFA and MUFA, than for fat intake. Certain fatty acids, including SFAs, MUFAs, and PUFAs, might exert distinct metabolic or inflammatory effects, which could contribute more significantly to health outcomes compared to fat consumption [45, 46]. Additionally, detailed categorization of fatty acid intake might better capture underlying dietary patterns. For example, individuals with higher SFA intake might adhere to poorer overall dietary habits [47]. Moreover, specific biochemical pathways associated with individual fatty acids might have more direct influences on metabolic pathways and bioavailability than those attributable to dietary fat consumption [48].

Although biological mechanisms are not fully elucidated, several causal pathways have been proposed for the association of fat and fatty acid intake with OC patient survival. First, hormone-related mechanisms may play an important role in these associations. A meta-analysis of 13 intervention studies showed a positive association between dietary fat intake and estrogen levels [49]. Further, elevated levels of estrogen may promote the growth and proliferation of OC cells [50]. In this study, animal-based MUFAs were found to be the main contributor to the positive association of total MUFA intake with all-cause mortality in OC patients. It is possible that the consumption of animal-derived food adversely affects the development of hormone-dependent cancers, including OC [51]. Second, high-fat diets have been shown to induce systemic, chronic low-grade inflammation [16], and a high systemic immune-inflammatory index was reported to be related to poor OC patient survival [52]. Third, the uptake and metabolism of exogenous fatty acids by human adipocytes are associated with OC progression and metastasis [53–55]. Fourth, fatty acids not only provide energy for cancer cells, but also regulate the proliferation of cancer cells by affecting membrane structure, intracellular signaling processes, and transcription factors [17]. Fifth, a bio-informatic analysis using samples from public databases strongly pointed to a critical role of fatty acid metabolism in OC prognosis and therapy response [48]. Taken together, these biological mechanisms may contribute to the development of OC.

The major strength of the current study is that, for the first time, we assessed the associations of pre-diagnosis dietary fat and fatty acid intake from different sources and categories with OC patient survival. Additional merits stem from the design of the OOPS, detailed elsewhere [21, 22]. Furthermore, numerous subgroup analyses and sensitivity analyses yielded similar results, which confirmed the robust nature of the findings.

There are several potential limitations to this study. First, the application of FFQs may introduce measurement errors in the assessment of dietary intake, as outlined and explained in detail in prior research [22]. Second, dietary information was collected based on participants' recall of "usual" consumption in the year prior to OC diagnosis, whereas some participants might change their dietary habits due to a variety of reasons such as dieting to lose weight and dietary therapy for metabolic diseases. Nevertheless, our data showed that only a small number of patients (23.9%) changed their dietary habits recently and we have adjusted for dietary change in the Cox models. Furthermore, our investigation was specifically focused on understanding the association between pre-diagnosis dietary fat and fatty acid intake and OC survival, rather than post-diagnosis dietary intakes. Consequently, further research, ideally with prospectively collected post-diagnosis dietary data, is necessary to validate our findings. Nevertheless, using pre-diagnosis data allows us to leverage a large and well-characterized cohort that would otherwise be difficult to assemble for post-diagnosis dietary intake, thereby providing robust evidence despite this limitation. Third, due to the absence of data on trans fatty acids in the Chinese Food Composition Table [56], we were unable to assess the association between trans fatty acid intake and OC survival. Nevertheless, existing evidence indicated that trans fatty acid intake within the Chinese population was relatively low [57]. Fourth, despite rigorous control for potential confounding factors, the possibility of residual confounding and unmeasured confounders, such as tubal ligation and hysterectomy, could not be dismissed due to the observational nature of the study, which might affect the associations observed. Besides, given the high uniformity of surgical and chemotherapy protocols (carboplatin and paclitaxel) across patients, these were unadjusted in the final model, subsequent research could investigate the influence of divergent surgical and chemotherapy strategies on OC survival. Finally, all the participants were Chinese and therefore, generalization to other populations requires careful consideration.

Conclusions

In this prospective cohort of OC patients, higher pre-diagnosis intakes of total fats, total fatty acids, total SFAs, shorter-chain SFAs, long-chain SFAs, total MUFAs, and animal-based MUFAs were significantly associated with an increased all-cause mortality of OC patients. Conversely, consumption of egg fats and fruit and vegetable fats exhibited an inverse correlation with all-cause

mortality. These findings contribute novel evidence to the growing body of research on the associations between dietary intake and OC survival. Clinically, these results highlight the potential importance of dietary modifications, particularly in fat and fatty acid intake, as part of the management strategy for OC patients. Given the association between higher intake of certain fats and fatty acids and poorer survival outcomes, healthcare providers could consider incorporating dietary counseling focused on reducing SFAs and animal-based MUFAs while encouraging beneficial fats such as egg fats, as part of comprehensive patient care. However, these findings also call for further randomized controlled trials and experimental studies to validate the associations and explore the underlying biological mechanisms, which could inform future clinical guidelines and interventions to improve patient outcomes.

Abbreviations

BMI	body mass index
CIs	confidence intervals
DHA	docosahexaenoic acid
DPA	docosapentaenoic acid
EPA	eicosapentaenoic acid
FIGO	Federation of Gynecology and Obstetrics
FFQ	food frequency questionnaire
HRs	hazard ratios
MET	metabolic equivalent task
MUFA	monounsaturated fatty acid
OC	ovarian cancer
OOPS	Ovarian Cancer Follow-Up Study
PUFA	polyunsaturated fatty acid
SFA	saturated fatty acid
SD	standard deviation

Supplementary Information

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Supplementary Material 1

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Author contributions

Y-FW, ML, T-TG, and Q-JW contributed to the study design. XQ, SY, SG, JX, and T-TG collected the data. Y-FW, Y-ZL, S-HH, and F-HL analyzed the data. Y-FW, Y-LX, ML, T-TG, and Q-JW wrote the first draft of the manuscript and edited the manuscript. All authors read and approved the final manuscript. Y-FW and Y-LX contributed equally to this work.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of the Ethics Committee of Shengjing Hospital of China Medical University. And written informed consent was obtained from all the participants. The study was performed in accordance with the declaration of Helsinki.

Consent for publication

Not applicant.

Competing interests

The authors declare no competing interests.

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