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Association between the dietary inflammatory index and all-cause mortality in the U.S. cancer survivors: A prospective cohort study using the National Health and Nutrition Examination Survey database

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ABSTRACT

Background: Cancer remains one of the leading causes of mortality worldwide. Diet can impact inflammation and consequently affect cancer outcomes. The Dietary Inflammatory Index (DII) can serve as a tool to assess the inflammatory potential of cancer survivors' diets and further predict their survival.
Objectives: To investigate the relationship between the DII and the survival of cancer survivors in National Health and Nutrition Examination Survey (NHANES).
Methods: An overall sample of 2359 U.S. cancer survivors from the 2005–2014 cohorts of the NHANES were studied. The DII scores were calculated using 28 dietary components and the mortality status was ascertained until December 31, 2015. Based on the multiple analyses, the relationship between DII and all-cause mortality was examined.
Results: The weighted mean age at baseline was 65.17 ± 14.46 years, 53.16 % were female and 71.30 % were non-Hispanic white. The average DII was 1.51 ± 1.97. After accounting for multiple covariates, positive associations were observed ($P < 0.01$). Based on Kaplan-Meier survival curves, their significant relationship remains same and the survival probability was decreased among the groups of anti-inflammatory diets ($DII < 0$) versus pro-inflammatory diets ($DII \geq 0$) significantly (Log rank test; $P = 0.03$). Further analyses were conducted on subgroups and the results are still robust.
Conclusions: An elevated DII was associated with a rising mortality rate among cancer survivors. DII might serve as a potential inflammatory predictor of cancer mortality prognosis, as well as guide nutritional care and even clinical treatment of cancer survivors.

1. Introduction

Cancer remains one of the leading causes of death worldwide. By 2021, it was projected that there will be 608,570 deaths caused by cancer (Siegel et al., 2021). Inflammation promotes all stages of tumorigenesis, conqueringly limiting overall survival (Greten and Grivennikov, 2019). There is growing evidence that markers of inflammation, such as tumor necrosis factor-alpha (TNF-α), interleukins (IL-1, IL-6, IL-8), and C-reactive protein (CRP), are associated with the increased cancer risk and mortality (Zhao et al., 2021; Nøst et al., 2021).

It is noteworthy that diet and nutrition have the potential to influence inflammation and subsequently alter cancer outcomes (Bose et al., 2020; Zhang et al., 2022). Dietary patterns like Dietary Inflammatory Index (DII) (Davis et al., 2021), Healthy Eating Index-2015 (HEI-2015) (Shan et al., 2020), and Alternative Healthy Eating Index (AHEI-2010) (Salazar et al., 2018) more and more becoming the focus of diet-induced chronic inflammation research. The dietary inflammatory index (DII) is such a score developed to assess the overall inflammatory potential of an individual's diet (Shivappa et al., 2014). The DII score is calculated based on its effects on inflammatory markers, including IL1, IL6, TNF,

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and extra IL-4, IL-10, and CRP (Shivappa et al., 2016). Higher DII scores indicate more pro-inflammatory diets, characterized by lower intakes of foods like fruits, vegetables, and whole grains, and higher intakes of foods like processed meats, refined carbohydrates, and saturated fats (Sterling and Bowen, 2019). Several studies have uncovered significant associations between more pro-inflammatory diets, denoted by higher DII scores, and increased risk of cancer (Fowler and Akinyemiju, 2017; Xu et al., 2020). However, research examining the implications of the DII for health outcomes specifically among cancer survivors has been extremely limited to date. Long follow-up periods, large participants samples, comprehensive food questionnaires, as well as enough covariates for diet-mortality analyses have been relatively lacked. Many studies used categorical cut-points for DII analysis (such as tertiles and quartiles), but few studies have presented estimates based on continuous DII. Additionally, the relationship may depend on the type, stage of cancer, and other confounding factors about cancer involved (Fowler and Akinyemiju, 2017). More high-quality research is still needed to determine if the dietary inflammatory index is an independent predictor of mortality in cancer survivors and to understand the mechanisms behind any potential associations.

Basing on this assumption, our study aimed to elucidate the relationship between diet, inflammation, and cancer mortality in cancer survivors. Using National Health and Nutrition Examination Survey (NHANES) data from 2005 to 2014, we tested the hypothesis that a lower DII score, indicating a more anti-inflammatory diet, would be associated with reduced cancer mortality. Gaining a deeper understanding of diet-related inflammation and its links to mortality could have important implications for cancer survivors. Elucidating which dietary components most strongly influence inflammation may reveal actionable targets for dietary interventions aimed at improving outcomes. Analyses stratified by patient factors could help identify subgroups at highest risk from inflammatory diets, who may derive particular benefit from anti-inflammatory dietary changes. Additionally, comparing the dietary inflammatory index to other diet quality scores and biomarkers of inflammation and immune function would provide useful context about its role within the broader nutritional and biological landscape tied to mortality.

2. Materials and methods

2.1. Study design and population

The research data came from the NHANES available on https://www.cdc.gov/nchs/nhanes/. The NHANES was conducted by the Centers for Disease Control and Prevention (CDC) and approved by the institutional review board of the National Center of Health Statistics (NCHS) to investigate the health and nutritional status of Americans. All participants signed the consent form and completed household interviews, physical examinations, laboratory tests, and nutritional status assessments. In our study, we selected data from five cycles of the NHANES survey (2005–2006, 2007–2008, 2009–2010, 2011–2012, 2013–2014). These cycles of NHANES were chosen because only these NHANES cycles had full nutrient and dietary data available. A total of 50,965 individuals took part these times. Firstly, we excluded 20,727 people without mortality follow-up data. Secondly, 3041 participants who had unreliable dietary recall were also excluded from our analysis, including missing data and outliers that have values three times the standard deviation of each nutrient (Ahmed et al., 2021). Thirdly, 24,820 participants without cancer history were eliminated. Last, we excluded 17 participants with incorrect or incomplete covariates. These 17 participants were judged as having outliers which could disturb the results in the following. The depression scores were wrong above the detection limit, or the Body Mass Index (BMI) scores were beyond three standard deviations and last the demography questionnaires were lost. Ultimately, a total of 2359 participants were recruited (Fig. 1).

2.2. The dietary inflammatory index (DII)

The DII score used to assess a total of 45 different dietary components according to Shivappa et al (Shivappa et al., 2014; Shivappa et al., 2014). Recent studies confirmed that the value of DII still holds when only 28 food parameters were involved (Harmon et al., 2017; Abulimiti et al., 2020; Ruiz-Canela et al., 2015; Vahid et al., 2017; Denova-Gutiérrez et al., 2018). So our study used 28 food components to calculate the DII scores: energy; protein; carbohydrate; dietary fiber;

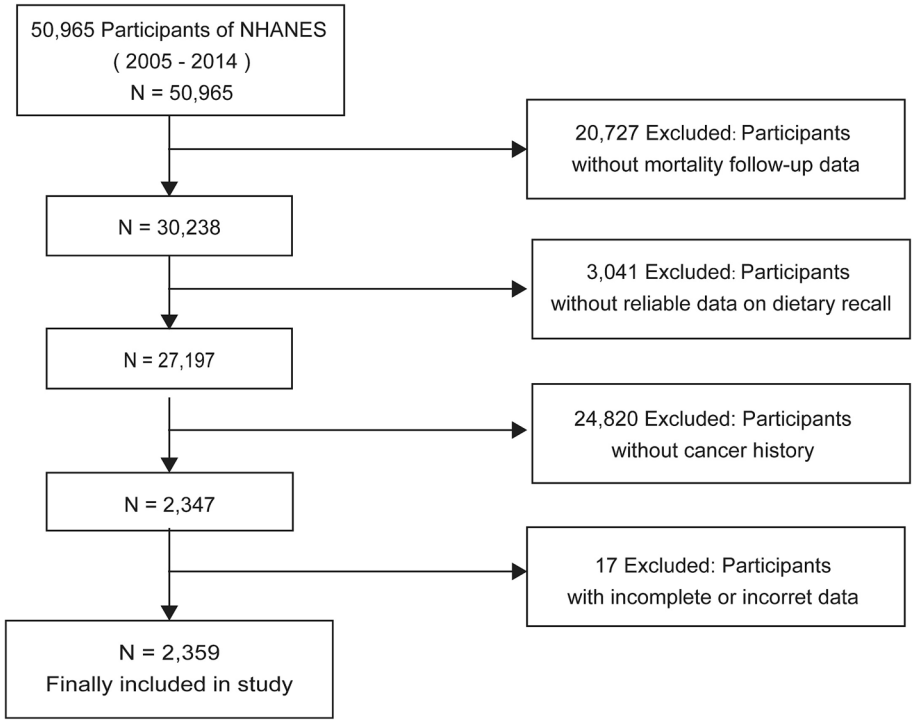

Fig. 1. Flow diagram for exclusion or inclusion in the studied sample.

total fat; saturated; monounsaturated and polyunsaturated fatty acids; ω-3 and ω-6 polyunsaturated fatty acids; cholesterol; beta-carotene; vitamins A, B1 (Thiamin), B2 (Riboflavin), B3 (Niacin), B6, B12, C, D and E; folic acid; magnesium; iron; zinc; selenium; caffeine and alcohol. Intakes of these components are linked to regionally representative world databases that provide robust estimates of mean and standard deviation (Shivappa et al., 2014). The specific calculation steps are as follows: (1) Z-scores = (Individual reported intake - Global daily mean intake) / Standard deviation of the global daily mean intake. (2) Centered percentile score = percentile count of (2* percentile of Z-score −1). (3) Specific DII score = Centered percentile score * Overall inflammatory score. (4) DII score = Summation of all specific DII scores (Shivappa et al., 2014).

2.3. Mortality data

Mortality data was obtained from the NHANES public use linked mortality file and linked to NHANES normal data using each unique respondent sequence number. In the file, the mortality status and follow-up time were included. Survival status was divided into two stages: survival or death. As for follow-up time, the duration was defined from the interview date to the last follow-up or death date through December 31, 2015. During up to 131 months of follow-up (median, 62.94 months) with a total of 2,359 participants (1,105 male and 1,254 female), the survival status was determined. Totally, 474 patients (20.09 %; 283 male and 191 female) were determined to have died, 155 of whom died from cancer (88 male and 67 female).

2.4. Covariates

Two sets of covariates at the time of survey were considered for multiple levels of analysis. For the first set, age, gender, race, education level, and marital status of participants were collected with questionnaires during survey interviews. The mean age was 65.17 ± 14.46 years ranging from 20 to 85 years. Race was classified as Mexican American, other Hispanic, non-Hispanic white, non-Hispanic black, or other races. Education level was categorized as less than high school, high school, or above. Marital status was divided into together (married/living together), separated (widowed/divorced/separated), or never married.

Further investigations are required to exclude other confounding factors. Additionally, smoking, alcohol, coronary heart disease (CHD), hypertension, diabetes, kidney injury, liver disease, stroke, BMI, depression, and tumor category were included to adjust for analysis. Smoking status was classified as never, former, and current. Never smokers reported smoking less than 100 cigarettes during their lifetime, while current or former smokers reported smoking over 100 cigarettes, and then divided according to whether they were currently smoking. Alcohol status was grouped into 'yes or no', alcohol use was assessed by asking the question "Had at least 12 alcohol drinks/1 year?" Hypertension was defined as having an SBP ≥ 140 or / and DBP ≥ 90 mmHg, and the ones who have been told by a doctor or other health professional that they have high blood pressure (Seo et al., 2020). Diabetes status was confirmed when the participants had a positive response to the question, "Have you ever been told by a doctor that you have diabetes?". CHD, kidney injury, liver disease, and stroke were all the same. The Body Mass Index (BMI) was calculated by dividing the measured weight (kg) by the square of the height (m^2), and the mean is (28.02 ± 6.03) kg/m^2. Diagnosis of depressive disorders used the Patient Health Questionnaire-9 (PHQ-9) instrument and a score of ≥ 10 has been valid for depression diagnosis. Tumor categories including skin, prostate, cervix, colon, bladder, lung, ovary, thyroid, endometrium, and other cancers, have been considered in terms of patient's recollection of cancer history.

2.5. Statistical analysis

In the statistical analysis, the complex survey design factors including weights, clustering, and stratification were all considered. The statistical differences between continuous or quartiles of DII with all the variables were tested by linear regression model and weighted chi-square test. In the next step, we estimated hazard ratios (HRs) and 95 % confidence intervals (CIs) using univariate and multivariate Cox proportional hazards models. Together with the above results, Kaplan-Meier analysis was applied to determine the association between DII levels and all-cause mortality. Kaplan-Meier cumulative hazard plots were calculated for the situation where the DIIs were divided into four quartiles. Kaplan-Meier survival curves were for two groups as anti-inflammatory diets and proinflammatory diets. The multivariate models were adjusted for potential confounding factors such as demographic variables, lifestyle behaviors, medical history, and so on. As for model 2, age, gender, race, education level, and marital status were adjusted. As for model 3, we further account for smoking, alcohol, CHD, hypertension, diabetes, kidney injury, liver disease, stroke, BMI, depression, and tumor category. In addition, we conducted subgroup to examine the association between all-cause mortality and covariates. To determine the statistical significance of interactions, the likelihood ratio test was used by creating interaction terms between continuous DII and the demographic and disease variables in subgroups. For missing values in covariates, median interpolation was used for continuous variables, and third categories were added for classified variables to assess the effect of missingness on the outcome. In this study, R software, and Empower Stats were used for data analysis.

3. Results

3.1. Baseline participants characteristics

Sociodemographic and clinical characteristics at baseline of 2359 participants in quartiles of DII are presented in Table 1 (DII < −0.266, −0.266 ≤ DII < 1.317, 1.317 ≤ DII < 2.692, DII ≥ 2.692). Data were presented as mean ± standard deviation (SD) for continuous variables and as numbers (%) for categorical variables. The weighted mean age at baseline was 65.17 ± 14.46 years, 1,254 (53.16 %) were female and 1,682 (71.30 %) were non-Hispanic white. The average DII was 1.51 ± 1.97. As for sociodemographic characteristics, lower levels of DII were more associated with individuals who are male, together with partner, having attended higher education, alcohol, and former smokers. To mention clinical characteristics, the prevalence of kidney injury increases across levels. Also, a new finding is that individuals with quarter 2 DII tends to have less stroke, liver injury, depression, however higher BMI. As for age, race/ethnicity, Hypertension, and CHD, quantities were similar among quartiles. When it comes to cancer categories, skin and prostate cancer tend to own lower DII, while cervical, colon, and lung have higher DII. Regarding their dietary habits, consumption of food elements reduced across the DII quartiles except cholesterol (**Table S1**).

3.2. Survival analysis of DII for mortality risk

During a follow-up of 149,043 person months, 474 individuals with cancer died from all-causes. DIIs were significantly higher in participants who died than in those who did not (1.37 ± 1.94 vs. 1.05 ± 1.94, p < 0.001). After multiple cox regression analyses, the estimated HR, and CIs of all-causes mortality across DII are shown in Table 2. In continuous models, we found that every 1 increase in DII was linked with an 8 % rise in the risk of all-cause mortality (HR 1.08, 95 % CI 1.02–1.14, P = 0.0053) in the multivariable model III. In categorical models, compared with the lowest quartile of DII (Q1), the HRs and 95 % CIs for all-cause mortality from Q2 to Q4 in the multivariable model III were respective 1.11 (0.84–1.48), 1.39 (1.06–1.82), and 1.39 (1.05–1.84). There was a graded increase in mortality risk for the higher two models across the quartiles of DII (P for trend = 0.0084). Individuals in the fourth quartile were 39 % more likely to die compared to the first quartile. The differences can be seen even more clearly in the

Table 1
Baseline characteristics of the research population according to DII quartiles in NHANES 2005–2014.

DII quartiles	All	Q1 (<−0.266)	Q2 (<1.317)	Q3 (<2.692)	Q4 (≥2.692)	P value
Number	2359	590 (25.01)	589 (24.97)	590 (25.01)	590 (25.01)	
Age (years)	65.17 ± 14.46	65.92 ± 14.28	65.26 ± 13.68	65.08 ± 14.72	64.40 ± 15.12	0.350
BMI (kg/m2)	28.02 ± 6.03	27.32 ± 5.79	28.30 ± 5.93	28.21 ± 6.06	28.23 ± 6.29	0.015
Cancer categories (n,%)						<0.001
Skin	325 (13.78)	190(32.20 %)	142(24.11 %)	133(22.54 %)	100(16.95 %)	
Prostate	309 (13.10)	95 (16.10)	85 (14.43)	75 (12.71)	54 (9.15)	
Cervical	170 (7.21)	20 (3.39)	42 (7.13)	44 (7.46)	64 (10.85)	
Colon	119 (5.04)	21 (3.56)	28 (4.75)	31 (5.25)	39 (6.61)	
Bladder	38 (1.61)	10 (1.69)	8 (1.36)	12 (2.03)	8 (1.36)	
Lung	42 (1.78)	7 (1.19)	10 (1.70)	10 (1.69)	15 (2.54)	
Ovarian	44 (1.87)	10 (1.69)	10 (1.70)	10 (1.69)	14 (2.37)	
Thyroid	44 (1.87)	11 (1.86)	13 (2.21)	7 (1.19)	13 (2.20)	
Endometrial	82 (3.48)	18 (3.05)	16 (2.72)	25 (4.24)	23 (3.90)	
Gender (n,%)						<0.001
Male	1105 (46.84)	350 (59.32)	313 (53.14)	263 (44.58)	179 (30.34)	
Female	1254 (53.16)	240 (40.68)	276 (46.86)	327 (55.42)	411 (69.66)	
Race/ethnicity (n, %)						0.056
Mexican American	136 (5.77)	24 (4.07)	41 (6.96)	36 (6.10)	35 (5.93)	
Other Hispanic	116 (4.92)	21 (3.56)	29 (4.92)	39 (6.61)	27 (4.58)	
Non-Hispanic White	1682 (71.30)	460 (77.97)	425 (72.16)	403 (68.31)	394 (66.78)	
Non-Hispanic Black	338 (14.33)	61 (10.34)	72 (12.22)	88 (14.92)	117 (19.83)	
Others	87 (3.69)	24 (4.07)	22 (3.74)	24 (4.07)	17 (2.88)	
Education (n, %)						<0.001
Less	227 (9.62)	37 (6.27)	53 (9.00)	53 (8.98)	84 (14.24)	
High school	317 (13.44)	51 (8.64)	65 (11.04)	91 (15.42)	110 (18.64)	
Above	1815 (76.94)	502 (85.08)	471 (79.97)	446 (75.59)	396 (67.12)	
Marital status (n, %)						<0.001
Together	1449 (61.42)	376 (63.73)	391 (66.38)	372 (63.05)	310 (52.54)	
Separated	769 (32.60)	193 (32.71)	154 (26.15)	188 (31.86)	234 (39.66)	
Never	139 (5.89)	21 (3.56)	43 (7.30)	30 (5.08)	45 (7.63)	
Smoke (n, %)						<0.001
Never	1057 (44.81)	274 (46.44)	261 (44.31)	271 (45.93)	251 (42.54)	
Current	368 (15.60)	64 (10.85)	66 (11.21)	83 (14.07)	155 (26.27)	
Former	934 (39.59)	252 (42.71)	262 (44.48)	236 (40.00)	184 (31.19)	
Alcohol drinking (n, %)						<0.001
Yes	1567 (66.43)	453 (76.78)	405 (68.76)	382 (64.75)	327 (55.42)	
No	680 (28.83)	118 (20.00)	156 (26.49)	175 (29.66)	231 (39.15)	
Depression (n, %)						<0.001
Yes	239 (10.13)	50 (8.47)	38 (6.45)	61 (10.34)	90 (15.25)	
No	2120 (89.87)	540 (91.53)	551 (93.55)	529 (89.66)	500 (84.75)	
Hypertension (n, %)						0.513
Yes	1357 (57.52)	320 (54.24)	321 (54.50)	351 (59.49)	365 (61.86)	
No	996 (42.22)	269 (45.59)	265 (44.99)	238 (40.34)	224 (37.97)	
Diabetes (n, %)						0.064
Yes	444 (18.82)	85 (14.41)	109 (18.51)	113 (19.15)	137 (23.22)	
No	1833 (77.70)	480 (81.36)	458 (77.76)	459 (77.80)	436 (73.90)	
CHD (n, %)						0.582
Yes	208 (8.82)	58 (9.83)	46 (7.81)	48 (8.14)	56 (9.49)	
No	2135 (90.50)	529 (89.66)	538 (91.34)	540 (91.53)	528 (89.49)	
Stroke (n, %)						<0.001
Yes	210 (8.90)	43 (7.29)	36 (6.11)	54 (9.15)	77 (13.05)	
No	2143 (90.84)	544 (92.20)	553 (93.89)	535 (90.68)	511 (86.61)	
Liver injury (n, %)						0.034
Yes	128 (5.43)	30 (5.08)	23 (3.90)	39 (6.61)	36 (6.10)	
No	2226 (94.36)	560 (94.92)	566 (96.10)	550 (93.22)	550 (93.22)	
Kidney injury (n, %)						<0.001
Yes	146 (6.19)	23 (3.90)	33 (5.60)	30 (5.08)	60 (10.17)	
No	2211 (93.73)	567 (96.10)	555 (94.23)	560 (94.92)	529 0(89.66)	

Notes: [†] The complex survey design was accounted for when computing means, standard deviation and proportions. [‡] Values are standardized to four quartiles of DII distribution of the study population except DII itself. [§] For continuous variables, data were mean if the carriable distribution is normal and P value was calculated by weighted linear regression model. For categorical variables, data were presented as n (%) and P value was calculated by weighted chi-square test.
Abbreviations: DII, Dietary Inflammatory Index; BMI, Body Mass Index; CHD, Coronary Heart Disease; Q, Quartile.

Cumulative Hazard curves (Fig. 2). Besides this classification, participants can be further categorized according to their dietary polarization into pro-inflammatory groups (DII $\geq$ 0) or anti-inflammatory groups (DII < 0). The significance still held that $p < 0.05$ and pro-inflammatory ones were 33 % higher than anti-inflammatory ones (**Table S2**). As shown in Kaplan-Meier survival curves (Fig. 3), the survival probability was significantly decreased among pro-inflammatory diets (DII < 0) groups versus anti-inflammatory diets (DII $\geq$ 0) groups (Log rank test; P = 0.03). In addition, we also assessed the correlations between DII score of each dietary component and risk of all-cause mortality in cancer survivors (Qi et al., 2014; Petimar et al., 2017; Zwakenberg et al., 2017; Zhang et al., 2022). Thus, we observed a positive association between each Fiber, PUFA, Alcohol, Mg, SE, Niacin, n-3 Fatty acids, and cancer mortality after adjustment (**Table S3**). We aimed to further find a simplified-DII index with less and more relevant elements specifically. A new cancer DII index may be made up specifically applicable to cancer

Table 2
The correlation between risk of all-cause mortality and DII among 2359 Participants between 131 Months.

Exposure	HR (95 % CI), P-value		
	Model I[†]	Model II[‡]	Model III[§]
DII (per 1 increased)	1.08 (1.02, 1.13) 0.0033**	1.11(1.05, 1.17) <0.0001***	1.08 (1.02, 1.14) 0.0053**
DII quartiles			
Q1<-0.266	1.0	1.0	1,0
Q2<1.317	0.96 (0.72, 1.26)	1.08 (0.82, 1.44)	1.11 (0.84, 1.48)
Q3<2.692	1.32 (1.02, 1.70) *	1.44 (1.11, 1.88) **	1.39 (1.06, 1.82) *
Q4 ≥ 2.692	1.36 (1.05, 1.76) *	1.56 (1.19, 2.04) **	1.39 (1.05, 1.84) *
P for trend	0.0034**	0.0003***	0.0084**

Notes: [†]. Not adjusted. [‡]. Further adjusted for age, gender, race, education, and marital status. [§]. The following variables of BMI, smoking status, alcohol status, presence of diabetes, hypertension, CHD, depression, stroke, kidney and liver injury were also included to control for the effects of continuous DII. For categorical covariates, four quartiles of DII variables were used for logistic regression models. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.
Abbreviations: DII, Dietary Inflammatory Index; Q, Quartile; HR, hazard ratio; CI, confidence interval.

mortality or any other fields in the future.

3.3. Subgroup analyses

Further analyses were conducted on subgroups of age, gender, education level, cancer type, alcohol, smoke, BMI, and baseline medical condition to determine whether DII levels were associated with all-cause mortality (Table 3). Across subgrouping variables, the trend between DII levels and all-cause mortality was obvious in all ages, male, higher educational subgroup, BMI ≥ 30, alcohol and no diseases of diabetes, depression, liver injury, stroke, CHD, and hypertension (P for trend < 0.05). Among the cancer types, the skin cancer type is most pronounced (P for trend = 0.0044), higher DII was associated with more than two times higher incidence of skin cancer (RR: 2.68, 95 % CI: 1.48–4.84). Moreover, no significant interactions were detected between DII levels and these stratifying variables (P interactions > 0.05).

4. Discussion

In our prospective study, we have found that the pro-inflammatory potential of the diet, as calculated by the DII, was significantly associated with the risk of all-cause mortality among US cancer survivors. The positive correlation was consistent in all ages, male, higher educational subgroup, BMI ≥ 30, alcohol and individuals without diabetes, depression, liver injury, stroke, CHD, or hypertension as subgroup analyses indicated.

The positive association between DII and cancer mortality has been reported in many studies. Observational studies across geographies (Li et al., 2018), races (Lopez-Pentecost et al., 2022; Veronese et al., 2020; Shivappa et al., 2018) and cancer types (Jayedi et al., 2018) were the most common kind of evidence between them. A multiethnic study found an increased risk of cancer mortality with DII in the top quintile by 53 % (Park et al., 2018). A randomized controlled trial with low-dose antioxidants reported an increased risk of cancer mortality with higher DII scores in the placebo group, but not in the antioxidant-supplemented group (Graffouillère et al., 2016). A *meta*-analysis of cohort studies have also supported that the highest DII category was associated with a 67 % increased risk of cancer mortality (Fowler and Akinyemiju, 2017).

In terms of specific research data, our analysis found that participants in USA shared higher DII in general. The mean DII in our analysis was 1.112 ± 1.943, which was slightly higher than cohort studies conducted in Mediterranean area (Veronese et al., 2020) or in Augsburg, German (Shivappa et al., 2018). Probably patients in the USA showed less adherent to a Mediterranean diet, which was inversely related to the DII score, than those living in the Mediterranean or Germany (Fowler and Akinyemiju, 2017; Tosti et al., 2018; Wirth et al., 2016). In terms of our main outcomes, the higher DII probably related to pro-inflammatory dietary patterns have been associated with increased cancer mortality. Consistent result was found in lung (Sadeghi et al., 2022), colorectal (Zheng et al., 2020), prostate (Zucchetto et al., 2016), breast (Jang et al., 2018), and primary liver cancer (Zhong et al., 2020). A *meta*-analysis found a 16 % increase between the diet-mortality relationship (95 % CI:

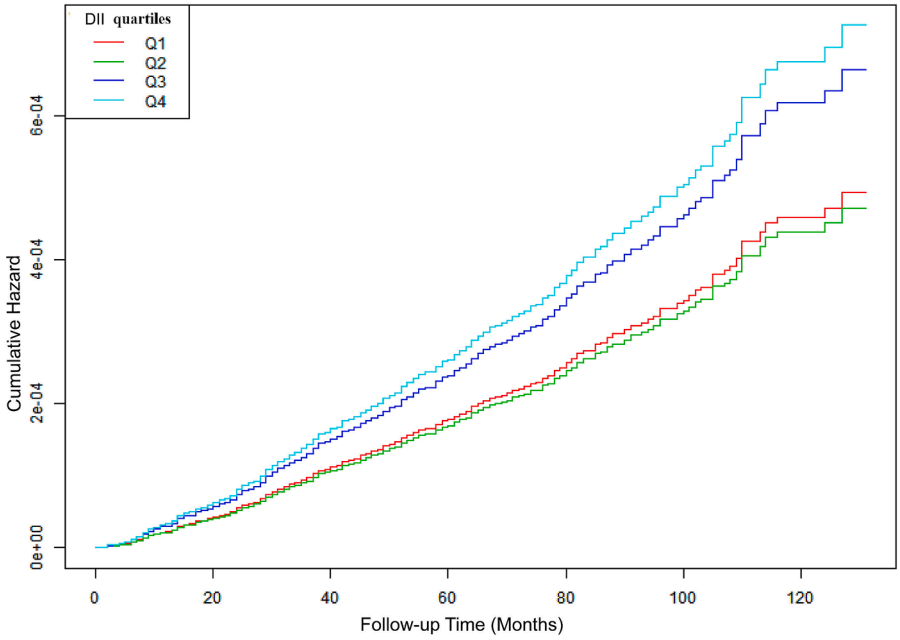

Fig. 2. Kaplan–Meier estimated cumulative survival curves based on DII levels, 2005–2014. Notes: Each red, green, dark blue and light blue line represents each DII in four quartiles (DII <−0.266, −0.266 ≤ DII <1.317, 1.317 ≤ DII <2.692, DII ≥ 2.692). Abbreviations: DII, Dietary Inflammatory Index. Q, quarter. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

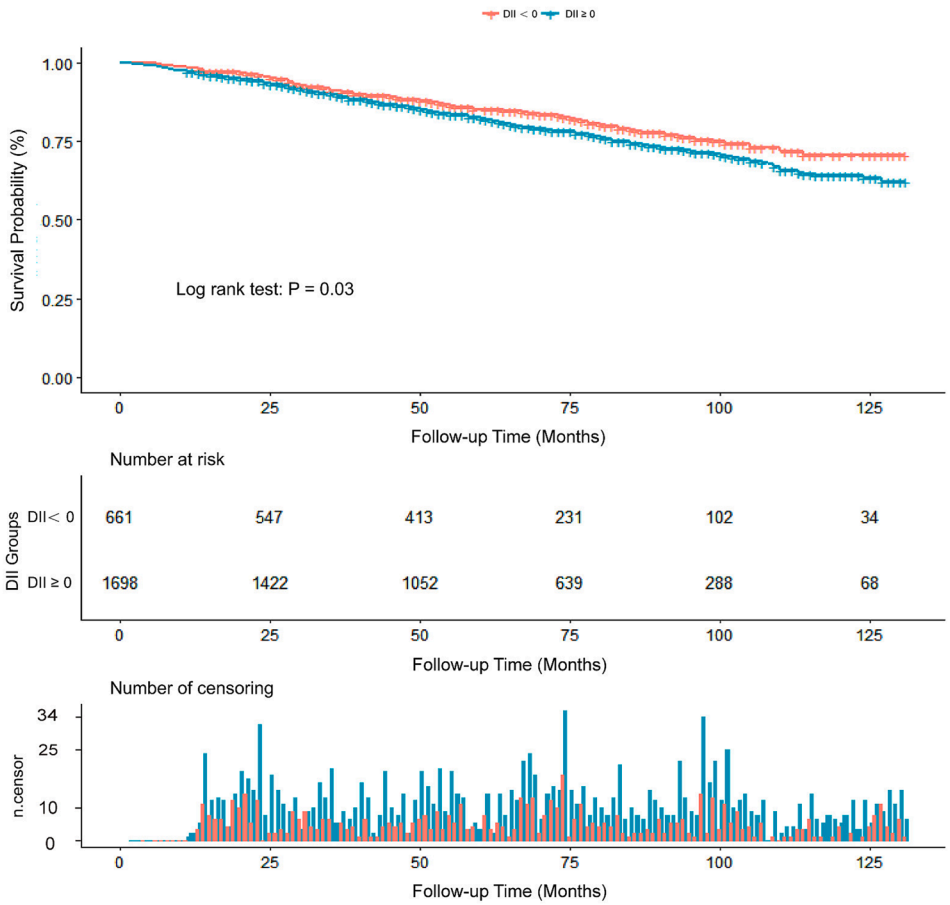

Fig. 3. Kaplan–Meier estimated cumulative hazard curves based on DII levels, 2005–2014. Notes: Each red line represents DII < 0 and each blue line represents DII ≥ 0. Abbreviations: DII, Dietary Inflammatory Index. Q, quarter. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

1.01–1.32) (Zahedi et al., 2020). In comparison, our findings were an 8 % rise (HR 1.08, 95 % CI 1.02–1.14, P = 0.0053). The above research indicated that the linearity of DII in quartile variables show a greater degree of variation with mortality than continuous variables. However, no significant association has been found in some research (Okada et al., 2019), such as a collaborative cohort study that a total of 58,782 Japanese with a 19.3-year median follow-up period, 11,693 participants died, and no significant relation were observed to either total cancer or digestive cancer (Okada et al., 2019). Not only the cancer type, but also the regional culture that different dietary habits in Asia or Western populations may account for this dissimilarity. In the subgroup analyses, result seemed more significant in males. Interestingly, two case-control studies indicated that the positive association remained only in males (Sadeghi et al., 2022; Shivappa et al., 2015). Another study revealed the indistinctive finding in postmenopausal Hispanic women (Lopez-Pentecost et al., 2022). Anti-inflammatory diets appear to be safer for women than pro-inflammatory diets, especially among postmenopausal women (Shivappa et al., 2018; Zheng et al., 2020; Shivappa et al., 2015). A small sample size or regional or type specific differences may account for the observed differences. Further studies have been needed to determine if special groups in subgroup analysis were generally exposed to pro-inflammatory dietary components. Last but not the least, we found the strong relation in certain skin cancer survivors, and this was the first prospective study concerning it.

There are several potential mechanisms that could explain the association between a pro-inflammatory diet, as shown by higher DII scores, and cancer mortality risk. In our report, the DII for each Fiber, PUFA, Alcohol, Mg, SE, Niacin, and n-3 Fatty acids stood out to be the most significant ones in the relationship between cancer mortality and dietary inflammation. Recent studies have revealed a pro-inflammatory diet reported with lower fiber (Lee et al., 2016; Katagiri et al., 2020), higher saturated fats (Brown et al., 2019; Hanson et al., 2020; Liyanage et al., 2019), or lower microelement (Anderson et al., 2019; Wang et al., 20182018; Cortés-Jofré et al., 2020) may increase the levels of cytokines, which may lead to inflammatory tumor microenvironment that drives metastasis and poor patient outcomes (Fowler and Akinyemiju, 2017; Liu et al., 2019). It is worth mentioning that the gut microbiome appears to be a hotspot medium between the diet and cancer mortality (Hu et al., 2012). By inhibiting the absorption or modulating the effects of dietary components such as fiber, the gut microbiome may play a potential role in maintaining intestinal immune homeostasis and preventing inflammation (Neuman et al., 2015; Murga-Garrido et al., 2021; Tong et al., 2021). Another possible indirect way is that multiple metabolic risk factors affected by a pro-inflammatory diet, including insulin resistance (Saad et al., 2016), and high cholesterol (Hu et al., 2012), could promote cancers of the lung, colon, stomach, breast (Fowler and Akinyemiju, 2017), et al. In brief, the DII score could provide a useful measurement of the inflammatory potential to estimate the burden of cancer linked to diet.

Our study has some strengths. To begin with, as a composite of up to 28 food parameters, the DII obviates problems with inter-correlations between these dietary factors to a large extent. Second, we utilized the generalizability of NHANES data, which contained representative non-institutionalized Americans, which allowed our findings to be presented with generalizability. we adjusted for covariates associated with risk of mortality to minimize potential sources of bias. Thirdly, all covariates were measured without participants knowing their ultimate results. Then, with the prospective nature of mortality follow-up and

Table 3
Subgroup analysis for DII quartiles with all-cause mortality in 2359 cancer survivors.

Variables	DII Lever, Hazard Ratio (95 % CI)					P interaction
	Q1 (<-0.266)	Q2 (<1.317)	Q3 (<2.692)	Q4 (≥2.692)	P trend	
Age						0.7432
20–60	1.0	1.00 (0.28, 3.57)	2.50 (0.83, 7.55)	2.54 (0.84, 7.66)	0.0432	
60–85	1.0	1.13 (0.84, 1.51)	1.46(1.10, 1.93) **	1.43(1.07, 1.93) *	0.0049	
Gender						0.7531
Male	1.0	1.23 (0.88, 1.73)	1.57(1.13,2.20) **	1.39 (0.94, 2.05)	0.0211	
Female	1.0	0.85 (0.49, 1.46)	1.12 (0.70, 1.78)	1.24 (0.79, 1.93)	0.2055	
Education level						0.1265
Less	1.0	1.09 (0.42, 2.85)	1.67 (0.67, 4.14)	1.78 (0.79, 4.02)	0.1216	
High	1.0	1.16 (0.56, 2.40)	1.18 (0.59, 2.36)	0.79 (0.39, 1.59)	0.4079	
Above	1.0	1.04 (0.74, 1.46)	1.39(1.01, 1.90) *	1.54(1.09, 2.17) *	0.0053	
Cancer categories						0.1812
Skin	1.0	1.50 (0.82, 2.77)	1.33 (0.73, 2.43)	2.68(1.48, 4.84) *	0.0044	
Digest	1.0	1.34 (0.39, 4.59)	2.57 (0.91, 7.26)	1.59 (0.54, 4.73)	0.3016	
Urinary	1.0	0.70 (0.09, 5.28)	0.61 (0.09, 4.36)	0.89 (0.08, 10.24)	0.8813	
Lung	1.0	0.00(0.00, 0.14) *	0.00(0.00, 0.28) *	0.64 (0.01, 53.17)	0.1904	
Others	1.0	1.12 (0.61, 2.07)	2.21(1.26, 3.90) **	1.79 (0.91, 3.49)	0.0096	
Alcohol						0.2015
Yes	1.0	1.27 (0.89, 1.80)	1.56(1.11, 2.18) **	1.65(1.15, 2.37) **	0.0024	
No	1.0	0.80 (0.46, 1.40)	0.99 (0.59, 1.64)	0.85 (0.51, 1.42)	0.7028	
Diabetes						0.3083
Yes	1.0	1.14 (0.63, 2.04)	1.11 (0.61, 2.00)	1.02 (0.55, 1.88)	0.9793	
No	1.0	1.09 (0.78, 1.54)	1.44(1.06, 1.97) *	1.10(1.03, 1.17) **	0.0060	
Depression						0.2898
Yes	1.0	2.65 (0.92, 7.61)	1.98 (0.80, 4.91)	2.52 (1.04, 6.09)	0.0898	
No	1.0	1.03 (0.77, 1.38)	1.42(1.08, 1.87) *	1.46(1.10, 1.95) **	0.0295	
Smoke						0.1761
Never	1.0	1.27 (0.77, 2.09)	1.55 (0.96, 2.50)	1.49 (0.91, 2.42)	0.0844	
Current	1.0	1.12 (0.40, 3.14)	0.94 (0.38, 2.32)	1.80 (0.83, 3.89)	0.1293	
Former	1.0	0.98 (0.67, 1.43)	1.38 (0.96, 1.99)	1.09 (0.71, 1.67)	0.2814	
BMI						0.5984
<25	1.0	1.38 (0.87, 2.20)	1.41 (0.91, 2.18)	1.53 (0.97, 2.42)	0.0654	
>=25, <30	1.0	0.66 (0.40, 1.09)	1.24 (0.80, 1.93)	1.06 (0.66, 1.70)	0.3959	
>=30	1.0	1.91 (0.99, 3.68)	2.10(1.13, 3.88) *	1.98(1.04, 3.78) *	0.0386	
CHD						0.9704
Yes	1.0	0.67 (0.31, 1.45)	0.96 (0.47, 1.99)	1.16 (0.55, 2.46)	0.7693	
No	1.0	1.21 (0.88, 1.66)	1.49(1.11, 2.01) **	1.45(1.06, 1.98) *	0.0083	
Stroke						0.6847
Yes	1.0	0.79 (0.32, 1.96)	1.39 (0.68, 2.81)	1.46 (0.71, 2.97)	0.2278	
No	1.0	1.16 (0.85, 1.57)	1.38(1.03, 1.85) *	1.37 (1.00, 1.87)	0.0254	
Liver injury						0.2012
Yes	1.0	1.36 (0.33, 5.69)	6.22(1.44, 26.85) *	2.83 (0.60, 13.31)	0.0624	
No	1.0	1.10 (0.82, 1.48)	1.30 (0.99, 1.72)	1.40 (1.05, 1.88) *	0.0129	
Hypertension						0.9598

(continued on next page)

Table 3 (*continued*)

Variables	DII Lever, Hazard Ratio (95 % CI)					P interaction
	Q1 (<-0.266)	Q2 (<1.317)	Q3 (<2.692)	Q4 (≥2.692)	P trend	
Yes	1.0	1.15 (0.80, 1.66)	1.51(1.08, 2.12) *	1.24 (0.88, 1.76)	0.1692	
No	1.0	1.13 (0.71, 1.82)	1.30 (0.81, 2.09)	1.90 (1.14, 3.16)	0.0188	

Notes: †· When analysis a subgroup variable, age at interview, gender, education level, BMI, smoking status, alcohol status, presence of diabetes, hypertension, CHD, stoke, liver injury, depression and cancer categories were all adjusted except the variable itself. ‡· For categorical covariates, four quartiles of DII variables were used for logistic regression models. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.
Abbreviations: DII, Dietary Inflammatory Index; Q, Quartile; HR, hazard ratio; CI, confidence interval.

spending a longer period (median, 62.94 months), we could eliminate the possibility of recall bias from our analysis. Finally, our initial results remained in extra subgroup analyses, suggesting the robustness of the relation.

We also found limitations in our study. Firstly, our analysis of a single DII score at baseline may lead to bias. Dietary habits could change over time that longitudinal studies can compensate for this limitation (Pierre and Almaroof, 2022). Secondly, due to the nature of the observational design, we could not exclude all the possible effects of covariates that might be related to both diet and mortality. Furthermore, the death data we obtained from the National Death Index might introduce some biases due to the possible incomplete linking, and inaccurate death certificates. Lastly, this database has a relatively small group of cancer survivors and whether these associations remain for certain patients who were hospitalized or highly coordinated requires further investigation.

There are still some issues to be clarified. Based on the variation of DII measurements according to clinical condition, DII's effect size and cut-off variables could differ according to the type and progression of cancer. The optimal condition and cut-off value for DII should be validated for future research needs and clinical applications. Thus, a larger sample size is required. In view of the epidemiology statement, a greater number of patients with lung and colon cancers should be studied, and not just the non-institutionalized United States. Furthermore, research should investigate the dose–response relationship between DII and cancer mortality to determine whether the association is linear, or threshold related. Research on the relationship between food, inflammation, and cancer still requires further investigation, with mediums such as gut microbiome tenting to be an appropriate research avenue.

5. Conclusions

Our findings suggest that a more pro-inflammatory potential of the diet, as measured by the DII, was significantly associated with the higher risk of all-cause mortality among US cancer survivors. In clinical practice, the DII might serve as a potential inflammatory predictor of cancer mortality prognosis, as well as guide nutritional care and even inspire treatment of cancer survivors.

Ethical approval

Not applicable.

Author contributions

Xiaohe Sun: Data curation, Methodology, Writing – original draft, Writing – review & editing. **Shuai Chen:** Data curation, Methodology, Writing – original draft. **Guowei Zhou:** Methodology. **Haibo Cheng:** Funding acquisition, Project administration, Writing – original draft, Writing – review & editing.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.pmedr.2023.102582.

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