Eur J Nutr (2016) 55:1891–1900
DOI 10.1007/s00394-015-1005-z

ORIGINAL CONTRIBUTION

Association between inflammatory potential of diet and mortality among women in the Swedish Mammography Cohort

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Received: 30 March 2015 / Accepted: 21 July 2015 / Published online: 31 July 2015
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Abstract
Purpose Diet and dietary components have been studied previously in relation to mortality; however, little is known about the relationship between the inflammatory potential of overall diet and mortality.
Materials and methods We examined the association between the Dietary Inflammatory Index (DII) and mortality among 33,747 participants in the population-based Swedish Mammography Cohort. The DII score was calculated based on dietary information obtained from a self-administered food frequency questionnaire. Mortality was determined through linkage to the Swedish Cause of Death Registry through 2013. Cox proportional hazard regression was used to estimate hazard ratios (HR). During 15 years of follow-up, 7095 deaths were identified, including 1996 due to cancer, 602 of which were due to digestive-tract cancer, and 2399 due to cardiovascular disease.
Results After adjusting for age, energy intake, education, alcohol intake, physical activity, BMI, and smoking status, analyses revealed a positive association between higher DII score and all-cause mortality. When used as a continuous variable (range −4.19 to 5.10), DII score was associated with all-cause mortality ($HR_{Continuous} = 1.05$; 95 % CI 1.01–1.09) and digestive-tract cancer mortality ($HR_{Continuous} = 1.15$; 95 % CI 1.02–1.29). Comparing subjects in the highest quintile of DII (≥1.91) versus the lowest quintile (DII ≤ −0.67), a significant association was observed for all-cause mortality (HR = 1.25; 95 % CI 1.07–1.47, $P_{trend} = 0.003$).
Conclusion These results indicate that a pro-inflammatory diet, as indicated by higher DII score, was associated with all-cause and digestive-tract cancer mortality.

Keywords Dietary Inflammatory Index · Mortality · Swedish women

Introduction

Inflammation is a result of the body's response to tissue insult or injury, or the presence of inflammatory stimulants such as lipopolysaccharide, interleukins, and other cytokines [1, 2]. The acute inflammatory response represents an important step in the process of wound healing, immune responsiveness, and tissue regeneration that, under normal circumstances, will resolve within a few days [3, 4]. By contrast, chronic inflammation is characterized by positive feedback in which the normal modulating of signaling by cytokines does not "turn off" [5]. It is well known that dietary factors are necessary to mount a competent acute inflammatory response [2, 6], as well as contributing to an individual's underlying state of chronic inflammation [7, 8].

Chronic inflammation is known to be associated with a variety of chronic health conditions including arthritis, diverticulitis, cardiovascular disease (CVD), diabetes

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[9–11], and common epithelial cancers, with colorectal [12–14] being the most extensively studied. Worldwide, CVD is the leading cause of mortality, accounting for about half of deaths among adults [15]. There is growing evidence that specific dietary components influence inflammation [16–18] and this may influence all-cause, cancer, and CVD mortality [19–22].

Research into the role of diet in inflammation and mortality suggests that diet represents a complicated set of exposures which often interact, and whose cumulative effect modifies both inflammatory responses and health outcomes. Several dietary indices exist to assess diet quality, but only one focuses on diet's effects on inflammation. The Dietary Inflammatory Index (DII) was developed to characterize an individual's diet on a continuum from maximally anti- to pro-inflammatory. The DII is grounded in peer-reviewed literature focusing on a specific health-related outcome (i.e., inflammation) and is standardized to dietary intake from numerous populations around the world; combining these developments helps to overcome shortcomings of previous dietary indices [23]. Thus far, the DII has been found to be associated with C-reactive protein [24, 25], interleukin-6 [26–28], and homocysteine [26]. Additionally, DII has been shown to be associated with glucose intolerance and dyslipidemia components of the metabolic syndrome [25, 29], anthropometric measurements in Spain [30], asthma in Australia [28], respiratory conditions in Italy [31], bone mineral density among postmenopausal women in Iran [28], colorectal cancer in two case–control studies in Spain and Italy [32, 33], and three cohort studies in the USA [34–36], pancreatic [37], prostate [38, 39] and esophageal cancers [40, 41] and mortality in a cohort among US women [42].

Until now, the DII has not yet been applied to mortality outcomes in a population outside USA. The purpose of this study was to examine the association between the DII and all-cause, overall cancer, digestive-tract cancer, and CVD mortality in a large population-based prospective cohort of Swedish women, the Swedish Mammography Cohort. We are interested in digestive-tract cancers, in particular those of lower gastrointestinal tract as they are strongly related to chronic inflammation [43, 44]. Our working hypothesis was that a higher DII score (indicating a pro-inflammatory diet) is associated with risk of death from any cause, as well as death from specific causes.

Methods

Study population

Recruitment procedures and characteristics of the Swedish Mammography Cohort (SMC) have been described previously [45]. In brief, the SMC is a population-based cohort of 66,651 women born between 1914 and 1948 who were recruited between 1987 and 1990 in Västmanland and Uppsala counties in central Sweden. In 1987, participants completed a questionnaire regarding diet, reproductive, and other factors. In 1997, a second questionnaire was extended to include dietary supplements, physical activity, and smoking status and was sent to participants who were still alive and residing in the study area; 39,227 (70 %) women returned this questionnaire. Completion and return of the self-administered questionnaire were treated as informed consent of study participants. The study was approved by the ethics committee at the Karolinska Institutet.

Because information on several potential confounders (i.e., cigarette smoking, physical activity) was first obtained in 1997, only women who completed the 1997 questionnaire were included in the present study ($N = 38{,}984$ after exclusion of those with an incorrect or missing national registration number). We excluded women with implausible total energy intake [3 standard deviations (SD) from the mean value for $\log_e$-transformed energy intake], a history of stroke, coronary heart disease, diabetes, or cancer (except nonmelanoma skin cancer) before the start of follow-up. This left 33,747 women for the present analysis.

Dietary assessment

Diet was assessed in 1997 using a 96-item food frequency questionnaire (FFQ) [46]. Participants were asked how often, on average, they had consumed each item during the previous year, with eight predefined frequency categories ranging from never to $\geq$3 times per day. Nutrient intakes were calculated by multiplying the frequency of consumption by the nutrient content of age-specific portion sizes by using composition values from the Swedish Food Administration Database [47]. Nutrient intakes were adjusted for energy using the residual method [48]. The questionnaire also asked about use of dietary supplements, including multivitamins with minerals, and some specific vitamin and mineral supplements. The FFQ had been validated previously [49].

Dietary Inflammatory Index (DII)

The DII is based on the literature published through 2010 linking diet to inflammation. Developing the DII involved review and scoring nearly 2000 scientific articles on diet and six inflammatory markers [i.e., CRP, interleukin (IL)-1β, IL-4, IL-6, IL-10, and tumor necrosis factor (TNF)-α] based on cell culture and laboratory animal experiments, and cross-sectional, longitudinal and intervention trials in humans. Individual intakes of food parameters on which the DII is based are then compared to a world standard

database of dietary intake based on datasets from 11 different regions worldwide. A complete description of the DII is available elsewhere. Briefly, to calculate DII for the participants of this study, the dietary data were first linked to the world database that provided a robust estimate of a mean and standard deviation for each parameter [23]. These then become the multipliers to express an individual's exposure relative to the "standard global mean" as a *z*-score. This is achieved by subtracting the "standard global mean" from the amount reported and dividing this value by the standard deviation. To minimize the effect of "right skewing," this value is then converted to a centered percentile score. The centered percentile score for each food parameter for each individual was then multiplied by the respective food parameter effect score, which is derived from the literature review, in order to obtain a food parameter-specific DII score for an individual. All of the food parameter-specific DII scores are then summed to create the overall DII score for each participant in the study [23]. A description of validation of the DII, including comparing hs-CRP values to DII derived from both dietary recalls and a structured questionnaire similar to an FFQ, is available elsewhere [24]. Twenty-seven of the 45 food parameters that could be used for DII calculation were available from this study [23]. The 27 food parameters were energy, carbohydrate, protein, fat, alcohol, fiber, cholesterol, saturated fatty acid, monounsaturated fatty acid, polyunsaturated fatty acid, niacin, thiamin, riboflavin, vitamin B12, vitamin B6, iron, magnesium, zinc, selenium, vitamin A, vitamin C, vitamin D, vitamin E, folic acid, beta carotene, omega 6, and omega 3. Of the remaining 18 food parameters missing, there are some that are consumed in smaller amounts in Sweden, such as turmeric, rosemary, pepper, garlic, and ginger.

Outcome assessment

Date and cause(s) of death were identified through linkage to the Swedish Cause of Death Registry at National Board of Health and Welfare. It is estimated that 93 % of all deaths in Sweden are reported within 10 days and 100 % are reported within 30 days [50]. Cause of death was determined by International Classification of Diseases (ICD) codes.

Statistical analysis

Cox proportional hazard models were used to calculate hazard ratios (HRs) and 95 % confidence intervals (95 % CIs) for death. Participants contributed person-time from September 15, 1997, until death from any cause, or end of follow-up on December 31, 2013. We also examined death from any cancer, digestive-tract cancer, and cardiovascular disease as the endpoints with end of follow-up on December 31, 2012, as information on the specific cause of death was not available after this time. The DII was categorized into quintiles, with the lowest quintile serving as the reference group.

We adjusted our multivariable models for age in 1997 (continuous), total energy intake (continuous), BMI (<25, 25–29.9, ≥30 kg/m^2), education (primary school, high school, university), smoking status (never, former <20 pack-years, former ≥20 pack-years, current <20 pack-years, current ≥20 pack-years), physical activity (quartiles of metabolic equivalent [MET] hours/day; <38.9, 38.9–42.2, 42.3–45.9, >45.9), and alcohol intake (nondrinker or <3.4, 3.4–9.9, ≥10 g/day). Categories were created for missing data.

The covariates were chosen a priori as they previously had been shown to be strong risk factors for mortality in this cohort. Other covariates commonly reported to alter inflammation (NSAIDS, aspirin) or mortality (sedentary time) were explored and not shown to be significant and thus not included in the models. Tests for linear trend were performed by assigning the median value of each category to each participant in that group. All tests of statistical significance were two-sided, and analyses were performed using SAS version 9.2 (SAS Institute Inc., Cary, NC, USA).

Results

DII had a mean ± SD value of 0.64 ± 1.45, with a range from −4.19 (most anti-inflammatory score) to +5.10 (most pro-inflammatory score). Women in higher quintiles had lower age, BMI, physical activity (MET hours/day), percentage of women with postsecondary education, nonalcohol drinkers, and higher total energy intake and percentage of current smokers. Similarly, total, cancer, digestive-tract cancer, and CVD deaths were associated with higher DII values (Table 1). Table 2 describes the distribution of servings of 30 food groups across DII quintiles, with percentage difference between quintiles 5 and 1. Compared to first quintile, women in fifth quintile reported lower consumption of green leafy vegetables or potatoes (65 %), green leafy vegetables (64 %), and fruits (60 %). Similarly, women in fifth quintile reported higher consumption of beer (24 %), French fries (25 %), sweets (not including chocolate) (32 %), liquor (50 %), whole grains (58 %), refined grains (69 %), chocolate (100 %), high-fat diary (142 %), and soft drinks/soda (150 %).

In total, 7095 total deaths were identified during the 15 years of follow-up, including 1996 all cancer deaths, 602 digestive-tract cancer deaths, and 2399 CVD deaths. In age- and energy-adjusted analyses using continuous DII, a 1-unit increment in DII (corresponding to 11 % range in this cohort study) showed significant positive associations

Table 1 Characteristics at baseline (1997) of 33,747 women in the Swedish Mammography Cohort by quintile of Dietary Inflammatory Index

Characteristics[a,b]	Quintile of DII				
	1	2	3	4	5
Age at baseline (1997)	61.1	61.6	61.5	61.0	60.9
Postsecondary education (%)	22.5	20.9	18.7	18.8	15.9
Body mass index (kg/m^2)	25.2	25.0	25.0	24.8	24.7
Alcohol intake among drinkers (g/day)	5.1	5.2	5.2	5.2	4.9
Nondrinkers (%)	17.8	14.0	15.3	15.0	15.3
Total energy intake (kcal/day)	1516	1587	1616	1626	1651
Physical activity (MET hours/day)	43.0	42.7	42.6	42.5	42.0
Smoking status (%)					
Never (%)	53.1	54.3	55.1	52.7	47.0
Past (%)	25.8	22.7	22.1	20.8	20.9
Current (%)	19.4	19.9	21.1	24.9	30.0
Total deaths	1244	1388	1413	1447	1603
Cancer deaths	340	398	417	417	424
Digestive cancer deaths[c]	100	121	123	128	130
CVD deaths	445	457	472	465	560

[a] Data represent mean unless otherwise indicated

[b] Percents may not equal 100 due to missing values

[c] Includes cancers from the beginning of the oral cavity to rectum and cancers of pancreas and hepato-biliary system

with risk of all-cause mortality (HR = 1.08; 95 % CI 1.04–1.11), cancer mortality (HR = 1.06; 95 % CI 1.00–1.12), digestive-tract cancer mortality (HR = 1.14; 95 % CI 1.02–1.28), and CVD mortality (HR = 1.06; 95 % CI 1.00–1.13). The results were almost identical after additional adjustment for education, alcohol intake, physical activity, BMI, and smoking status, for all-cause mortality (HR = 1.05; 95 % CI 1.01–1.09) and digestive-tract cancer mortality (HR = 1.15; 95 % CI 1.02–1.29). The results were in the hypothesized direction for cancer mortality and CVD mortality. The direction of associations was along expected lines for all-cause and digestive-tract cancer mortality; however, results did not achieve statistical significance.

Age- and energy-adjusted analyses with DII categorized as quintiles revealed significantly higher risk for subjects in the fifth quintile compared to those in the first quintile for all-cause mortality (HR = 1.41; 95 % CI 1.21–1.64, $P_{trend} < 0.0001$), cancer mortality (HR = 1.32; 95 % CI 1.02–1.72, $P_{trend} = 0.03$), digestive-tract cancer mortality (HR = 1.38; 95 % CI 0.81–2.33, $P_{trend} = 0.04$), and CVD mortality (HR = 1.35; 95 % CI 1.01–1.81, $P_{trend} = 0.02$). Multivariate analyses revealed significantly higher risk among subjects in the fifth quintile compared to those in the first quintile for all-cause mortality (HR = 1.25; 95 % CI 1.07–1.47, $P_{trend} = 0.003$) (Table 3).

Discussion

In this large, nationally representative, prospective cohort study of women, consumption of a more pro-inflammatory diet, as reflected by higher DII scores, was associated with increased risk of death from any cause and deaths due to digestive-tract cancers. Compared to women in the first quintile, women in the fifth quintile were 25 % more likely to die from any cause. Similarly, for every one-unit increase in DII, the risk of dying from any cause and from digestive-tract cancer increased by 5 and 15 %, respectively. Among digestive-tract cancers, colorectal cancer is known to be strongly related to inflammation [51–54] and represents the majority of digestive-tract cancers [55, 56]. We also observed that higher intake of healthy food items such as fruits and vegetables was observed among women with lower quintiles of DII, whereas higher intake of less healthy food items such as French fries and refined grains was seen among women in lower quintiles.

The DII is different from other dietary indices, virtually all of which fall into three main categories: (1) those derived from specific dietary recommendations based on some external standard (e.g., Healthy Eating Index (HEI) which was derived from the adherence to the US Dietary guidelines [57]); (2) those derived empirically from findings within particular study populations (e.g., computing a pattern using principal component analysis (PCA) [58]); or (3) those that link to particular cultural patterns of dietary intake (e.g., the Mediterranean diet score [59]). Studies have been conducted examining various dietary patterns and indices in relation to mortality [60–62]. In the NHANES III cohort study, the HEI was found to be inversely associated with overall and CVD mortality [61]. In a study conducted in the National Institutes of Health-AARP Diet and Health Study, the Mediterranean diet score was associated with reduced all-cause and cause-specific mortality [60], while another report from the National Institutes of Health-AARP Diet and Health Study showed various indices [HEI-2010, the Alternative Healthy Eating Index-2010 (AHEI-2010), the alternate Mediterranean Diet (aMED), and Dietary Approaches to Stop Hypertension (DASH)] to be protective against all-cause mortality, CVD, and cancer mortality [63]. In contrast, in the Whitehall cohort study, which was conducted in UK with a predominantly European population, the AHEI was not associated with cancer mortality or noncancer/non-CVD mortality [62].

Previous studies also have examined the effect of specific food items on mortality, including high-fat dairy [66, 67], and nutrients such as magnesium [68], and vitamin E [69], and the results have been inconsistent. High-fat dairy is rich in saturated fat and total fat which form the pro-inflammatory

Table 2 Distribution of food groups across quintiles of Dietary Inflammatory Index (DII)

Food groups (servings/week)	Direction increase with DII	% diff (Q5–Q1)/Q1	Quintile of DII				
			1	2	3	4	5
Other vegetables	−	−65	32.1	22.2	17.6	14.6	11.1
Green leafy vegetables	−	−64	6.4	4.5	3.6	3.0	2.3
Fruit	−	−60	19.1	15.5	12.8	10.3	7.7
Legumes	−	−59	2.7	2.0	1.6	1.4	1.1
Fruit juice	−	−54	2.8	2.5	2.1	1.7	1.3
Poultry	−	−38	0.91	0.78	0.66	0.62	0.56
Low-fat dairy	−	−38	16.9	16.1	15.8	12.4	10.4
Fish/seafood	−	−37	4.6	4.2	3.7	3.4	2.9
Hot cereal	−	−35	2.3	2.2	2.1	1.9	1.5
Organ meats	−	−33	1.8	1.9	1.8	1.6	1.2
Cold cereal/muesli	−	−31	3.6	3.5	3.3	3.0	2.5
Condiments	−	−27	2.6	8.1	2.2	2.1	1.9
Nuts	−	−27	0.41	0.36	0.34	0.33	0.30
Potatoes	−	−18	5.6	5.3	5.1	5.0	4.6
Eggs	−	−13	1.5	1.5	1.4	1.3	1.3
Wine	−	−8	0.89	0.93	0.91	0.89	0.82
Red meat	−	−6	3.3	3.3	3.2	3.2	3.1
Tea	−	−4	7.9	7.8	7.7	7.7	7.6
Coffee	+	14	20.5	20.9	21.7	22.2	23.4
Salty snacks	+	15	0.26	0.28	0.28	0.30	0.30
Beer	+	24	3.3	3.7	3.9	4.2	4.1
French fries	+	25	0.32	0.37	0.32	0.30	0.40
Sweets without chocolate	+	32	8.5	10.0	10.4	10.7	11.2
Liquor	+	50	0.20	0.20	0.30	0.30	0.30
Whole grains	+	58	16.0	18.9	20.4	21.3	25.2
Refined grains	+	69	8.8	10.6	11.6	12.8	14.9
Chocolate	+	100	0.70	0.90	0.90	1.1	1.4
High-fat dairy	+	142	15.4	21.9	24.8	28.9	37.2
Soft drinks/soda	+	150	3.0	4.0	4.8	5.6	7.5

components in DII calculation. Vitamin E and magnesium have an anti-inflammatory effect score in the DII calculation [23]. No association was observed between magnesium and calcium and cancer-related mortality in the EPIC-Heidelberg study [68]. In a prospective study conducted by Pocobelli et al. [69], vitamin E was found to significantly reduce CVD mortality; however, no association was observed with cancer mortality. A limitation of examining individual food items or nutrients is that whole foods or nutrients are usually consumed with other food items and nutrients; thus, dietary inter-correlations may attenuate or accentuate the actual effects of the individual food or nutrient under study. A very high correlation between nutrients and among foods can result in instability in risk estimation and possible loss of statistical power. In formulating the DII [23], an entirely different approach was taken by focusing on the functional effects of foods and nutrients. As such, it relies on a careful review and scoring of the medical literature in specific relation to inflammation. Also, it standardizes individuals' dietary intakes of pro- and anti-inflammatory food constituents to world referent values.

The effect of red meat is in the opposite direction of expectation. However, it should be noted that red meat is one among several other food items that influence inflammation. There are other food items such as vegetables, fruits, and fish that exert strong anti-inflammatory effects per unit exposure, and their distributions are along expected lines. Because the DII takes into account diet as a whole, and red meat eaters also consumed these other vegetable components, spices, etc., the totality of their diet was anti-inflammatory. Red meat was positively correlated with both energy and sum of fruit and vegetable intake ($r = 0.40$ and 0.14, respectively). In addition to this, the percentage decrease across quintiles is 6 % which is very low

Table 3 Hazard ratio for mortality outcomes by Dietary Inflammatory Index (DII) among 33,747 women in the Swedish Mammography Cohort, 1997–2013

	Overall mortality ($N = 7095$)		Cancer mortality ($N = 1996$)		Digestive cancer mortality[c] ($N = 602$)		Cardiovascular disease mortality ($N = 2399$)	
	HR[a]	HR[b]	HR[a]	HR[b]	HR[a]	HR[b]	HR[a]	HR[b]
DII (continuous)	1.08 (1.04–1.11)	1.05 (1.01–1.09)	1.06 (1.00–1.12)	1.04 (0.99–1.11)	1.14 (1.02–1.28)	1.15 (1.02–1.29)	1.06 (1.00–1.13)	1.04 (0.98–1.12)
Quintile 1	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
Quintile 2	1.10 (0.95–1.29)	1.09 (0.93–1.27)	1.10 (0.85–1.43)	1.15 (0.88–1.49)	0.87 (0.52–1.44)	0.95 (0.56–1.62)	1.09 (0.82–1.47)	1.08 (0.79–1.47)
Quintile 3	1.09 (0.94–1.27)	1.05 (0.90–1.23)	1.15 (0.89–1.49)	1.16 (0.89–1.52)	0.99 (0.59–1.66)	1.01 (0.59–1.72)	1.03 (0.77–1.37)	1.06 (0.78–1.43)
Quintile 4	1.21 (1.04–1.41)	1.17 (1.00–1.36)	1.16 (0.90–1.51)	1.15 (0.88–1.50)	1.40 (0.85–2.31)	1.52 (0.90–2.56)	1.30 (0.97–1.74)	1.30 (0.95–1.77)
Quintile 5	1.41 (1.21–1.64)	1.25 (1.07–1.47)	1.32 (1.02–1.72)	1.25 (0.96–1.64)	1.38 (0.81–2.33)	1.42 (0.82–2.49)	1.35 (1.01–1.81)	1.26 (0.93–1.70)
P_{trend}	<0.0001	0.003	0.03	0.14	0.04	0.045	0.02	0.08

[a] Adjusted for age and energy intake

[b] Adjusted for age (continuous), energy intake (continuous), BMI (<25, 25–29.9, ≥30 kg/m^2), education (primary school, high school, university), smoking status (never, former <20 pack-years, former ≥20 pack-years, current <20 pack-years, current ≥20 pack-years), physical activity (quartiles of metabolic equivalent hours/day), and alcohol intake (nondrinker or <3.4, 3.4–9.9, ≥10 g/day)

[c] Includes cancers from the beginning of the oral cavity to rectum and cancers of pancreas and hepato-biliary system

compared to decrease in other food items like fruits (60 %) and green leafy vegetables (64 %).

However, our previous experience with using the DII indicates that those conditions that we know from other lines of research (e.g., laboratory animal experiments) to be most strongly related to inflammation tend to produce the most strongly positive results. Most notably, this includes colorectal cancer (which, because of its relatively high incidence, drives digestive-tract cancer rates) [32, 34–36], pancreatic cancer [37], and asthma, lung and other respiratory conditions [28, 31]. In contrast, results have been equivocal for factors related to metabolic syndrome and thereby for cardiovascular outcomes as well [25, 26, 29]. This makes sense given the fact that cardiovascular outcomes are strongly determined by other, nondietary factors, most notably physical activity [60, 61].

Although the actual mechanism of how a healthy diet reduces mortality is not clearly known, one of the possible mechanisms for this inverse association might be through the effect of a pro-inflammatory diet on insulin resistance by increasing systemic inflammation [70, 71]. Consumption of food items such as meat and butter has been shown to increase systemic inflammation by increasing levels of high-sensitivity CRP, E-selectin, and soluble vascular cell adhesion molecule-1 [70], which are then responsible for increasing insulin resistance [71]. Insulin resistance caused by increasing circulating levels of insulin, triglycerides, and nonesterified fatty acids [43, 44] is associated with digestive-tract cancers and various cardiovascular diseases, all of which, if left uncontrolled, result in death. As mentioned previously, there are various dietary factors that have different effects on inflammation; for example, red meat consumption increases inflammation while green leafy vegetables reduce inflammation [43, 44]. In support of our findings, previous work in the SMC examining diet and mortality, have shown significant inverse associations between anti-inflammatory food parameters such as fruits and vegetables [72], which is consistent with results from other studies such as ARIC [73], 2001–2008 Health Surveys for England [74], and a cohort in Taiwan [75]. Previous analyses in the SMC also have shown increasing vitamin C and selenium intake to be inversely associated with breast cancer-related mortality [76, 77], and consumption of red meat was associated with shorter overall survival [78]. Vitamin C and selenium are among anti-inflammatory components of DII [23]. At the same time, there are also other studies that have shown no association between red meat, vitamin C, and selenium; and inflammation [79–81] and mortality [82, 83].

Our study has several strengths. First, it is population-based and employs a prospective design. It also benefits from a large sample size, complete ascertainment of deaths through the National Cause of Death Registry, and detailed information on diet. The small fraction of missing data reported on single items, which were treated with the zero-consumption approach, is unlikely to represent a source of bias for the observed findings [84]. This study also had a long follow-up with a large number of events for the outcomes studied. This is the first time the DII has been used in a cohort study outside USA with mortality as outcome. The main limitation of this study was that information on diet was self-reported, which can lead to a potential misclassification of the exposure. Classification errors in our prospective study, however, were nondifferential with respect to the occurrence of death and most likely led to an attenuation of the results. Dietary assessment was available only at one time point. Participants' dietary habit might have changed during the follow-up period. However, previous studies reported that dietary pattern classification is moderately stable over long periods of time during adulthood [85–90]. Other limitations of the study include nonavailability of screening data, stage of disease at diagnosis for most cancers, and no evidence of DII being associated with inflammatory markers in this study. While it is true we do not have screening data, the universal health care system of Sweden makes this less of an issue than in a country such as the USA where access to healthcare is confounded by many other unmeasured factors.

In conclusion, individuals who reported consumption of a more pro-inflammatory diet were at greater risk of dying from any cause and from digestive-tract cancers compared to individuals who reported a more anti-inflammatory dietary intake. Our results provide further evidence for the benefits of a diet in high anti-inflammatory foods such as vegetables and fruits, nuts, low-fat dairy products, and fish, and low in fried foods, processed meats, and refined grains. Future steps might include investigating how the DII predicts mortality in cohort studies among men and how it behaves longitudinally in an intervention trial among individuals who have had cancer or CVD to examine if improvement in the DII scores over time is associated with reduced risk of inflammation-associated disease and/or death.

Acknowledgments Funding Drs. Shivappa and Hébert were supported by Grant Number R44DK103377 from the US National Institute of Diabetes and Digestive and Kidney Diseases. Dr. Wolk was supported by the Distinguished Professor Award from Karolinska Institute. The Swedish Mammography Cohort is maintained by grants from the Swedish Research Council Committee for infrastructure. Dr Harris was supported by a grant from the Swedish Cancer Foundation.

Disclosure Dr. James R. Hébert owns controlling interest in Connecting Health Innovations LLC (CHI), a company planning to license the right to his invention of the Dietary Inflammatory Index (DII) from the University of South Carolina in order to develop computer and smart phone applications for patient counseling and dietary intervention in clinical settings. Dr. Nitin Shivappa is an employee of CHI. The subject matter of this paper will not have any direct

bearing on that work, nor has that activity exerted any influence on this project.

Compliance with ethical standards

Conflict of interest The authors have declared no conflicts of interest.

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