Null Results in Brief

Cancer Epidemiology, Biomarkers & Prevention

Inflammatory Potential of Diet, Inflammation-Related Lifestyle Factors, and Risk of Pancreatic Cancer: Results from the NIH-AARP Diet and Health Study

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Abstract

Background: Chronic inflammation is implicated in pancreatic cancer, and can be modulated by diet and other lifestyle factors. We examined the association between Dietary Inflammatory Index (DII) scores and pancreatic cancer risk in the NIH-AARP Diet and Health Study, and examined effect modification by inflammation-related lifestyle factors, including body mass index, cigarette smoking, diabetes, alcohol drinking, and use of non-steroidal anti-inflammatory drugs.

Methods: Energy-adjusted DII scores (E-DII) were computed on the basis of food frequency questionnaire responses for foods and dietary supplements. Cox proportional hazards models were fitted and effect modification was examined by adding a cross-product of each effect modifier with E-DII quintile in the multivariable-adjusted model.

Results: There were 2,824 primary incident pancreatic cancers diagnosed during a median of 13.4 years follow-up, and there was no association between E-DII scores and pancreatic cancer risk among either men [HR_{Q5vsQ1}, 1.00; 95% confidence interval (CI), 0.86–1.16] or women (HR_{Q5vsQ1}, 1.00; 95% CI, 0.82–1.21) in the multivariable-adjusted model, and no association was detected by any cancer stage. The E-DII and pancreatic cancer association was not modified by any of the inflammation-related lifestyle factors examined.

Conclusions: Results from this large prospective study did not support an association between inflammatory potential of diet and pancreatic cancer, or effect modification by other inflammation-related lifestyle factors.

Impact: Inflammatory potential of diet may not be related to pancreatic cancer risk. Future cohort studies with more frequent dietary measures could be useful in determining the appropriate timing of dietary intake in relation to pancreatic cancer etiology.

Introduction

Chronic inflammation is implicated in pancreatic carcinogenesis and can be modulated by diet and other lifestyle factors (1, 2). The Dietary Inflammatory Index (DII) assesses dietary inflammatory potential (3) and has been examined in relation to pancreatic cancer in both case–control and prospective studies (4–7), with inconsistent results. In the NIH—American Association of Retired Persons (NIH–AARP) Diet and Health Study, the largest diet and health prospective cohort study in the United States, we aimed to examine and verify the findings of the relationship between the E-DII and pancreatic cancer risk and effect modifications by important inflammation-related lifestyle factors including body mass index (BMI), cigarette smoking, alcohol consumption, non-steroidal anti-inflammatory drugs use, and diabetes history. We also aimed to explore the E-DII and pancreatic cancer association by cancer stage.

Materials and Methods

The NIH-AARP study (8) was a prospective cohort of 617,119 American adults ages 50 to 71 years old who responded to the baseline questionnaire (BQ) between 1995 and 1996. In the current analysis, participants were excluded if they had unreliable responses on the BQ ($n = 50{,}721$), had BQs filled out by proxy ($n = 15{,}760$), had a personal history of cancer ($n = 8{,}828$), reported an extreme energy intake or BMI (2 interquartile ranges below the sex-specific 25th percentile or above the 75th percentile of log-transformed energy intake or BMI; $n = 4{,}261$ and 4,214, respectively), or moved out of the study area or died before baseline ($n = 79$), bringing the analytic sample to 533,256.

The E-DII score (3) was calculated by summing across the product of energy-adjusted and global dietary database-standardized intake of each of the 33 available DII components, derived from self-reports of previous-year intake using the 124-item NCI-developed food frequency questionnaire (FFQ), with the DII component-specific inflammatory effect scores (3, 9). Incident

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pancreatic cancer cases were mainly identified through linkage with state cancer registries (9). The HRs (95% confidence intervals, 95% CI) of cancer risk associated with each one-unit increase of a standard deviation of the continuous E-DII score or with the E-DII quintiles (the first E-DII quintile as the referent group representing the most anti-inflammatory diets) were estimated using Cox proportional hazards models. We evaluated associations for men, women, and both combined. Effect modification was assessed by adding a cross-product of each categorical effect modifier with E-DII quintile in the multivariable-adjusted model. In addition, each pancreatic cancer stage was analyzed as a separate outcome. Multivariable models controlled for potential confounders listed in Table 1 and included as a footnote of Table 2. Sensitivity analyses included: (i) excluding subjects

Table 1. Baseline characteristics of 533,256 subjects in the NIH-AARP Diet and Health Study by quintile of E-DII score

	Most anti-inflammatory diet E-DII Quintile 1 (−7.91 to −5.61)	**E-DII Quintile 2 (−5.60 to −4.51)**	**E-DII Quintile 3 (−4.50 to −3.29)**	**E-DII Quintile 4 (−3.28 to −1.51)**	**Most pro-inflammatory diet E-DII Quintile 5 (−1.50 to 6.66)**
N	106,650	106,652	106,652	106,652	106,650
	Mean (SE)	Mean (SE)	Mean (SE)	Mean (SE)	Mean (SE)
Age at baseline (y)	62.0 (0.02)	61.9 (0.02)	61.8 (0.02)	61.6 (0.02)	61.1 (0.02)
Total energy intake (*k*cal/d)	1,564.8 (1.8)	1,722.9 (2.2)	1,817.2 (2.4)	1,937.7 (2.6)	2,180.1 (3.1)
BMI at baseline (kg/m^2)	26.3 (0.01)	26.8 (0.01)	27.1 (0.01)	27.4 (0.01)	27.6 (0.01)
	n (%)[a]	*n* (%)[a]	*n* (%)[a]	*n* (%)[a]	*n* (%)[a]
Sex					
Male	48,767 (45.7)	56,690 (53.2)	62,311 (58.4)	69,003 (64.7)	77,368 (72.5)
Female	57,883 (54.3)	49,962 (46.8)	44,341 (41.6)	37,649 (35.3)	29,282 (27.5)
Race/ethnicity					
White non-Hispanic	98,306 (92.2)	97,676 (91.6)	97,089 (91.0)	97,167 (91.1)	97,241 (91.2)
Black non-Hispanic	3,248 (3.1)	3,915 (3.7)	4,359 (4.1)	4,319 (4.1)	4,532 (4.3)
Hispanic	1,675 (1.6)	1,901 (1.8)	2,114 (2.0)	2,195 (2.1)	2,015 (1.9)
Other races	2,169 (2.0)	1,873 (1.8)	1,709 (1.6)	1,457 (1.4)	1,353 (1.3)
Unknown	1,252 (1.2)	1,287 (1.2)	1,381 (1.3)	1,514 (1.4)	1,509 (1.4)
Education level					
Less than or equal to 11 years	3,689 (3.5)	5,050 (4.7)	6,106 (5.7)	7,429 (7.0)	9,593 (9.0)
High school completion	16,654 (15.6)	19,180 (18.0)	20,309 (19.0)	22,155 (20.8)	25,815 (24.2)
Post high school training other than college	9,548 (9.0)	10,031 (9.4)	10,336 (9.7)	10,810 (10.1)	11,630 (10.9)
Some college	25,270 (23.7)	25,050 (23.5)	24,512 (23.0)	24,575 (23.0)	24,327 (22.8)
College and postgraduate	48,559 (45.5)	44,209 (41.5)	42,190 (39.6)	38,554 (36.2)	32,089 (30.1)
Unknown	2,930 (2.8)	3,132 (2.9)	3,199 (3.0)	3,129 (2.9)	3,196 (3.0)
Detailed smoking variable					
Never smoked	41,413 (38.8)	40,370 (37.9)	38,425 (36.0)	35,937 (33.7)	29,962 (28.1)
Quit ≥ 10 years, ≤20 cigs/d	26,668 (25.0)	24,272 (22.8)	23,208 (21.8)	21,761 (20.4)	18,272 (17.1)
Quit ≥ 10 years, >20 cigs/d	15,482 (14.5)	15,746 (14.8)	16,107 (15.1)	16,505 (15.5)	16,465 (15.4)
Quit 5–9 years, ≤20 cigs/d	3,808 (3.6)	3,546 (3.3)	3,369 (3.2)	3,393 (3.2)	3,058 (2.9)
Quit 5–9 years, >20 cigs/d	3,431 (3.2)	3,738 (3.5)	3,908 (3.7)	4,258 (4.0)	4,986 (4.7)
Quit 1–4 years, ≤20 cigs/d	2,279 (2.1)	2,144 (2.0)	2,139 (2.0)	2,140 (2.0)	2,137 (2.0)
Quit 1–4 years, >20 cigs/d	1,545 (1.4)	1,923 (1.8)	2,091 (2.0)	2,419 (2.3)	3,202 (3.0)
Quit < 1 year or current smoker, ≤20 cigs/d	6,376 (6.0)	7,972 (7.5)	9,109 (8.5)	10,318 (9.7)	13,563 (12.7)
Quit < 1 year or current smoker, >20 cigs/d	1,835 (1.7)	3,112 (2.9)	4,251 (4.0)	5,945 (5.6)	10,696 (10.0)
Unknown	3,813 (3.6)	3,829 (3.6)	4,045 (3.8)	3,976 (3.7)	4,309 (4.0)
Alcohol intake (drinks/day)					
0	21,480 (20.1)	25,517 (23.9)	27,369 (25.6)	27,915 (26.2)	28,428 (26.7)
0–3	81,323 (76.3)	75,906 (71.2)	73,027 (68.5)	70,578 (66.2)	63,174 (59.2)
>3	3,847 (3.6)	5,229 (4.9)	6,256 (5.9)	8,159 (7.6)	15,048 (14.1)
History of diabetes					
No	98,632 (92.5)	96,901 (90.9)	96,098 (90.1)	95,785 (89.8)	97,558 (91.5)
Yes	8,018 (7.5)	9,751 (9.1)	10,554 (9.9)	10,867 (10.2)	9,092 (8.5)
Aspirin products use frequency					
No use	17,068 (16.0)	16,650 (15.6)	16,996 (15.9)	16,913 (15.9)	16,799 (15.8)
1–3 times/mo	19,526 (18.3)	19,138 (17.9)	19,265 (18.1)	19,542 (18.3)	20,163 (18.9)
1–6 times/wk	11,384 (10.7)	11,271 (10.6)	10,465 (9.8)	10,226 (9.6)	9,258 (8.7)
≥1 time/d	17,981 (16.9)	17,056 (16.0)	16,267 (15.3)	14,955 (14.0)	12,750 (12.0)
Unknown	40,691 (38.1)	42,537 (39.9)	43,659 (40.9)	45,016 (42.2)	47,680 (44.7)
Ibuprofen products use frequency					
No use	26,405 (24.8)	26,657 (25.0)	27,365 (25.7)	27,689 (26.0)	27,909 (26.2)
1–3 times/mo	22,738 (21.3)	21,332 (20.0)	20,562 (19.3)	19,645 (18.4)	18,402 (17.3)
1–6 times/wk	9,463 (8.9)	9,033 (8.5)	8,338 (7.8)	8,057 (7.6)	6,955 (6.5)
≥1 time/d	7,133 (6.7)	6,899 (6.5)	6,519 (6.1)	5,994 (5.6)	5,459 (5.1)
Unknown	40,911 (38.4)	42,731 (40.1)	43,868 (41.1)	45,267 (42.4)	47,925 (44.9)

Abbreviation: SE, standard error.

[a]Sum of percentages may not add up to 100% because of rounding or missing.

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with follow-up <5 years; (ii) removing subjects who self-reported diabetes history at baseline; (iii) imputing cases' diagnosis dates by subtracting survival time (estimated on the basis of SEER data) from the death date if diagnosis date was same as death date.

Results

A total of 2,824 primary pancreatic cancers occurred during a median follow-up of 13.4 years. Population characteristics across E-DII quintiles are shown in Table 1. The E-DII was not associated with any pancreatic cancer outcome among both men and women

Table 2. The association between quintiles of E-DII score and pancreatic cancer risk among 533,256 subjects in the NIH-AARP Diet and Health Study

	E-DII Quintile 1	E-DII Quintile 2	E-DII Quintile 3	E-DII Quintile 4	E-DII Quintile 5	P_{trend}[a]	HR (95% CI)[b]
Total subjects							
Total sample	106,650	106,652	106,652	106,652	106,650		
Number of cases	575	524	572	553	600		
HR (95% CI)[c]	1.00	0.89 (0.79–1.00)	0.96 (0.85–1.08)	0.91 (0.80–1.02)	0.96 (0.85–1.08)	0.87	1.00 (0.96–1.04)
Males							
Total sample	62,827	62,830	62,828	62,826	62,828		
Number of cases	358	340	367	330	370		
HR (95% CI)[c]	1.00	0.94 (0.81–1.09)	1.01 (0.87–1.17)	0.90 (0.77–1.05)	1.00 (0.86–1.16)	0.89	1.00 (0.96–1.06)
Females							
Total sample	43,825	43,820	43,826	43,823	43,823		
Number of cases	218	212	181	217	231		
HR (95% CI)[c]	1.00	0.96 (0.79–1.16)	0.81 (0.66–0.99)	0.97 (0.80–1.17)	1.00 (0.82–1.21)	0.87	1.01 (0.94–1.07)
Localized pancreatic cancer[d]							
Number of cases	25	33	31	22	37		
HR (95% CI)[c]	1.00	1.32 (0.78–2.22)	1.23 (0.72–2.10)	0.88 (0.49–1.58)	1.52 (0.88–2.61)	0.08	1.16 (0.98–1.37)
Regional pancreatic cancer[d]							
Number of cases	122	108	119	102	122		
HR (95% CI)[c]	1.00	0.87 (0.67–1.13)	0.95 (0.73–1.22)	0.80 (0.61–1.05)	0.95 (0.73–1.25)	0.87	0.99 (0.91–1.09)
Distant metastasized pancreatic cancer[d]							
Number of cases	190	150	169	188	199		
HR (95% CI)[c]	1.00	0.75 (0.61–0.93)	0.82 (0.67–1.01)	0.88 (0.71–1.08)	0.87 (0.70–1.07)	0.77	1.01 (0.94–1.08)
Stratified analyses by inflammation-related lifestyle factors[e]							
	HR	**HR (95% CI)**	**HR (95% CI)**	**HR (95% CI)**	**HR (95% CI)**	P_{trend}[a]	$P_{interaction}$[f]
BMI							**0.29**
Underweight	1.00	1.40 (0.39–5.01)	1.96 (0.59–6.50)	0.20 (0.02–1.87)	0.80 (0.20–3.25)	0.51	
Normal	1.00	0.90 (0.74–1.10)	1.07 (0.88–1.30)	0.82 (0.66–1.02)	0.92 (0.74–1.15)	0.64	
Overweight	1.00	0.87 (0.73–1.05)	0.98 (0.82–1.17)	0.91 (0.76–1.10)	1.00 (0.84–1.21)	0.43	
Obese	1.00	0.83 (0.64–1.07)	0.72 (0.55–0.94)	0.86 (0.67–1.11)	0.80 (0.62–1.04)	0.32	
Smoking status							**0.72**
Never smokers	1.00	0.84 (0.69–1.02)	0.82 (0.67–1.01)	0.85 (0.69–1.04)	0.89 (0.72–1.11)	0.65	
Former smokers	1.00	0.90 (0.76–1.06)	0.97 (0.82–1.15)	0.97 (0.81–1.14)	1.01 (0.84–1.20)	0.46	
Current smokers	1.00	0.94 (0.64–1.37)	1.21 (0.85–1.72)	0.93 (0.65–1.32)	1.06 (0.76–1.49)	0.63	
Alcohol intake (drinks/d)							**0.52**
0	1.00	0.76 (0.59–0.98)	0.97 (0.77–1.23)	0.79 (0.61–1.00)	0.94 (0.73–1.19)	0.68	
0–3	1.00	0.93 (0.81–1.07)	0.92 (0.80–1.06)	0.94 (0.82–1.09)	0.95 (0.81–1.10)	0.86	
>3	1.00	1.02 (0.59–1.78)	1.32 (0.79–2.21)	1.07 (0.64–1.80)	1.14 (0.70–1.84)	0.80	
Frequency of aspirin products use							**0.38**
Never use	1.00	0.82 (0.60–1.11)	0.99 (0.74–1.32)	0.93 (0.69–1.26)	1.07 (0.79–1.45)	0.14	
Monthly use	1.00	0.81 (0.60–1.08)	1.12 (0.85–1.47)	0.96 (0.72–1.27)	0.86 (0.64–1.15)	0.44	
Weekly or daily use	1.00	1.00 (0.80–1.24)	0.95 (0.76–1.20)	0.89 (0.70–1.13)	1.15 (0.90–1.46)	0.31	
Frequency of ibuprofen products use							**0.60**
Never use	1.00	0.98 (0.77–1.24)	1.19 (0.95–1.50)	0.98 (0.77–1.25)	1.12 (0.88–1.42)	0.54	
Monthly use	1.00	0.82 (0.63–1.07)	1.00 (0.77–1.29)	0.93 (0.71–1.21)	1.02 (0.77–1.34)	0.25	
Weekly or daily use	1.00	0.88 (0.65–1.19)	0.78 (0.56–1.07)	0.86 (0.62–1.19)	0.92 (0.65–1.30)	0.98	
Diabetes history							**0.46**
No	1.00	0.90 (0.79–1.02)	0.99 (0.88–1.12)	0.90 (0.79–1.03)	0.96 (0.84–1.09)	0.96	
Yes	1.00	0.82 (0.58–1.14)	0.74 (0.53–1.04)	0.91 (0.65–1.26)	0.92 (0.65–1.30)	0.59	

[a]P_{trend} was calculated using the continuous E-DII score in the model after the restricted cubic spline test indicated the linear assumption was sufficient.
[b]The HR and 95% CI were estimated with the Cox proportional hazards model with continuous E-DII score, and it represents HR for one standard deviation increase of E-DII score.
[c]The multivariable-adjusted model was adjusted for age group (<62 and ≥62-years-old), sex (male or female), body mass index category (underweight, normal, overweight, obese, missing), smoking, quit and dose combined (nonsmoker, quit ≥ 10 years,≤20 cigs/d, quit ≥ 10 years,>20 cigs/d, quit 5–9 years,≤20 cigs/d, quit 5–9 years,>20 cigs/d, quit 1–4 years,≤20 cigs/d, quit 1–4 years,>20 cigs/d, quit<1 year or current,≤20 cigs/d, quit<1 year or current,>20 cigs/d, unknown), total energy (kcal/d), alcohol drinks per day (0, 0–3,>3), diabetes history (yes/no), education level (≤11 years, 12 years or completed high school, post-high school, some college, college and post graduate, unknown).
[d]The association between E-DII and each pancreatic cancer stage was conducted among total population after controlling for all the confounders listed in the footnote [c], because the interaction by sex was not statistically significant.
[e]The hazard ratios and 95% CIs comparing E-DII quintile 5,4,3, 2 with the first quintile were computed in the multivariable-adjusted model that included all variables in footnote [c], while adding the cross-product term of the quintile E-DII and each categorical effect modifier in the model among men and women combined. We did not present sex-specific interaction because of no difference in the significance of interactions by sex.
[f]$P_{interaction}$ was calculated with the cross-product of each effect modifier and E-DII quintile in the separate multivariable-adjusted COX proportional hazards model.

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(Table 2). The E-DII and pancreatic cancer association was not modified by any of the inflammation-related lifestyle factors examined (Table 2). All sensitivity analyses produced null results.

Discussion

The null association between the E-DII and pancreatic cancer risk found in this large prospective cohort study did not support previous case–control studies, which observed an approximate 2.5-fold increased risk among subjects consuming the most pro-inflammatory diets (4–6). Likewise, in contrast to our null findings, two case–control studies found evidence of effect modification by smoking status, BMI, or diabetes (4, 5). One study identified significant positive associations of DII with pancreatic cancer in all cancer stages (4). A pooled analyses of five nested case–control studies within the Pancreatic Cancer Cohort Consortium (PanScan) reported a modest positive association with E-DII ($OR_{Q5\ vs.\ Q1}$, 1.23; 95% CI, 0.92–1.66; $P_{trend} = 0.008$; ref. 6). Our findings agreed with previously conducted analyses using the prospective PLCO study, which reported an overall null association among 101,449 participants with baseline demographics similar to this study (7). Possible selection bias, frequent use of proxy responses, and other sources of information bias in pancreatic cancer case–control studies that may contribute to differential misclassification could help to explain differences in results from cohort studies.

The PLCO cohort suggested reverse causality for E-DII and pancreatic cancer association, where an inverse association between pro-inflammatory diets and pancreatic cancer risk was observed in follow up of <4 years (7). The current study did not support this phenomenon, even in the lag time analysis. The differences in study follow-up time (PLCO median follow-up is 8.5 years) and use of a FFQ with different components for computing the DII score may partially explain differing results. The effect of preclinical disease on dietary intake should be further explored in future studies. In addition to the possible true null association, it is possible that a one-time dietary assessment many years prior to diagnosis may not be an etiologically relevant timeframe for assessing exposure. Therefore, a large prospective study with more frequent dietary measures could be useful in determining the appropriate timing of dietary intake in relation to pancreatic cancer etiology.

Disclosure of Potential Conflicts of Interest

M.D. Wirth is a senior research scientist at Connecting Health Innovations, LLC. J.R. Hebert is a president and has ownership interest (including stock, patents, etc.) in Connecting Health Innovations LLC. No potential conflicts of interest were disclosed by the other authors.

Disclaimer

The views expressed herein are solely those of the authors and do not necessarily reflect those of the FCDC or FDOH.

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