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Socioeconomic deprivation impact on meat intake and mortality: NIH-AARP Diet and Health Study

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Abstract

Objective—Previous studies have not examined potential interactions between meat intake and characteristics of the local environment on the risk of mortality. This study examined the impact of area socioeconomic deprivation on the association between meat intake and all-cause and cause-specific mortality after accounting for individual-level risk factors.

Methods—In the prospective NIH-AARP Diet and Health Study, we analyzed data from adults, ages 50–71 years at baseline (1995–1996). Individual-level dietary intake and health risk information was linked to the demographic and socioeconomic context of participants' local environment based on census tract data. Deaths (n=33,831) were identified through December 2005. Multilevel Cox models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for quintiles of area deprivation scores.

Results—Associations of red and processed meats with mortality were consistent across deprivation quintiles. Men residing in least-deprived neighborhoods had a stronger protective effect for white meat consumption. No differences by deprivation index were observed for women.

Conclusion—Red and processed meat intake increases mortality risk regardless of level of deprivation within a given neighborhood suggesting biological mechanisms rather than neighborhood contextual factors may underlie these meat-mortality associations. The effect of white meat intake on cancer mortality was modified by area deprivation among men.

Keywords

meat consumption; mortality; census; socioeconomic; clustered survival data

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INTRODUCTION

Poor dietary patterns have been linked to residing in disadvantaged neighborhoods (1, 2), including low fruit and vegetable intake (3), high dietary fat intake (4), and increased alcohol consumption (4, 5). Although the relationship between meat consumption and disease risk is modest and inconsistent, previous studies from the National Institutes of Health (NIH)-AARP Diet and Health Study found a modest positive association between red and processed meat intake and total, cancer and cardiovascular disease (CVD) mortality (6). The socioeconomic characteristics of a person's residential neighborhood may interact with or modify the relationship between the type and amount of meat consumed and subsequent premature mortality, however, no studies to date have attempted to disentangle the potential effects of place of residence on the observed association.

Mortality as well as dietary habits are influenced not only by individual health-risk determinants such as race and education, but also by socioeconomic characteristics of the environment in which one resides (7–10). Several ecological and population-based studies have reported socioeconomic and racial disparities in total and cause-specific mortality (11–19). It is possible that diet-environment interactions exist between meat intake and neighborhood conditions (e.g., socioeconomic and racial composition). Examining whether the association between meat intake and mortality varies according to level of deprivation in the area in which a person resides could benefit public health efforts to change dietary habits if neighborhood-level determinants of diet and mortality exist. The information needed to examine neighborhood differences in dietary patterns is not readily available in standard questionnaires from cohort studies. Information provided by the U.S. Census Bureau may serve as a proxy for disparities in food access (20, 21) (e.g., access to affordable fresh and healthy food or quality and cuts of meat) that we have not collected directly, and may further define meat selection and consumption behaviors.

The aim of the present study is to examine interactions between meat intake and the local environment and their potential impact on mortality, which are not adequately addressed in the current literature. We hypothesize that type and quantity of meat consumption has a more deleterious effect on premature mortality in poorer neighborhoods than more affluent areas. By linking census data to the NIH-AARP Study, and using an innovative analytic method, we will prospectively examine the influence of neighborhood on mortality in a large cohort of over half a million older adults while accounting for known individual-level health risk factors, including age, education, race, smoking, physical activity, alcohol consumption, and fruit and vegetable intake.

MATERIALS AND METHODS

Study population

From 1995 through 1996, men and women between the ages of 50–71 years residing in one of six U.S. states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania) or two metropolitan areas (Atlanta, Georgia, and Detroit, Michigan) were recruited to participate in the NIH-AARP Diet and Health Study, a large prospective cohort study examining the relation between diet and health (22). The NIH-AARP Diet and Health Study was approved by the Special Studies Institutional Review Board of the U.S. National Cancer Institute, and written informed consent was obtained from all participants by completing and returning the baseline questionnaire.

Our baseline cohort included 566,401 persons. We excluded individuals whose questionnaire was completed by someone else on their behalf (n=15,760) and subjects who reported having end-stage renal disease, previous cancer, heart disease, stroke or

emphysema (n=136,785). We excluded 3583 (<1%) subjects reporting extreme daily total energy intake defined as more than two interquartile ranges above the 75th percentile or below the 25th percentile according to Box-Cox log-transformed intake values; the energy intake of the participants after exclusions was 322–6144 kcals/day. The exclusion of extreme outliers of energy intake was necessary for appropriate energy-adjustment (23). In addition, 495 individuals were excluded due to missing census information. Individuals were also excluded if their date of death or dropout preceded the date of study entry (n=3 and n=1, respectively). After exclusions, our analytic cohort consisted of 233,205 men and 176,569 women.

Study measures

Vital status and cause-specific mortality—Vital status was ascertained through linkage to the Social Security Administration Death Master File and National Death Index from recruitment (1995–96) to December 31, 2005. The underlying cause of death was ascertained from the National Death Index. In addition to all-cause mortality (n=33,831), we investigated the two leading causes of death: CVD (n=8,952) and cancer (n=14,091).

Neighborhood socioeconomic deprivation index—In the present study, census tracts (n=18,603) were used as proxies for neighborhoods. Study participants were linked to their affiliated census tract by geocoding the residential addresses used for the baseline questionnaire. As previously described (11), a neighborhood deprivation index was constructed for the NIH-AARP Diet and Health Study using principal component analysis based on a priori social and economic indicators available in the 2000 U.S. Census (9). An extensive outline of the process for developing the standardized neighborhood deprivation index is described in detail elsewhere (24). In brief, principal component analysis (PCA) was utilized to reduce the number of census variables (n=21) for established a priori contextual factors related to health outcomes obtained from the literature (9, 24, 25), which include measures of racial and ethnic composition, income and poverty, education, occupation, employment, housing and residential stability. When two or more variables within the same domain were highly correlated with each other (correlation coefficient > 0.90) only one was retained. PCA was re-implemented on the reduced number of variables (n=10) and the resulting first component was used for developing the empirical index based on the unique linear combination that accounted for the largest possible proportion of the total variability (i.e., the first component) in the component measures. The following ten variables were used to calculate a deprivation index score for each census tract: % total with less than high school education, % non-Hispanic blacks, % total unemployed, % females in management, % males in management, % households with income (1999) below poverty, % female head of household, % households with public assistance income, % households with income < 30k, % households with no vehicle. The internal consistency between the 10 variables in our study was high (Chronbach's reliability coefficient = 0.93), indicating strong construct validity. The higher the index score, the higher the neighborhood socioeconomic deprivation.

Demographics and behavioral risk factors—Information on individual-level demographic and health-related behaviors was ascertained through a mailed survey sent to study participants at baseline (1995–96). This survey included a validated (26) 124-item food frequency questionnaire (FFQ) that collected information on a participant's usual dietary intake within the past 12-month and including portion sizes, meat intake, and alcohol intake (www.risk.factor.cancer.gov/DHQ) as well as information on lifestyle (e.g., smoking, physical activity, vitamin use, menopausal hormone use) and medical history (e.g., family history of cancer, self-report of previous diagnosis for having end-stage renal disease, previous cancer, heart disease, stroke or emphysema, and general health status). Daily intake

of red and white meats and fruits and vegetables were energy-adjusted using the nutrient density method (23). Total red meat intake included consumption of bacon, beef, cold cuts, ham, hamburger, hot dogs, liver, pork, sausage, and steak; total white meat included all chicken and turkey meat products and fish. High-risk meat diet was defined a priori as high red, low white meat intake based on median values as cutpoints (6) to allow us to examine the overall composition of meat intake. Individuals who consumed a diet that was high in red meat but low in white meat diet were considered high-risk. Smoking history incorporated information from the baseline questionnaire on smoking status, time since quitting, and smoking dose. Physical activity was assessed on the baseline questionnaire by asking subjects how often they participated in physical activities at work or home, including exercise, sports, and activities such as carrying heavy loads during a typical month in the prior 12 months: vigorous physical activity was defined as activity ≥ 20 minutes (that increased breathing or heart rate, or worked up a sweat) for 5 or more times per week. Self-reported body weight and height were used to derive body mass index (kg/m^2).

Statistical analysis

Descriptive statistics were calculated for baseline characteristics of participants by gender. General linear models were used to examine age-adjusted characteristics across quintiles of deprivation. Quintiles for person-level variables (e.g., meat intake) were based on the full analytic cohort; quintiles for area deprivation were based on the distribution of the study's census tracts. Multivariable Cox proportional hazard regression models with a robust variance estimator that accounts for the correlation of individuals residing within the same census tract were used to assess associations between neighborhood deprivation quintiles, meat intake, and all-cause as well as CVD and cancer mortality (27, 28). Analyses were performed separately in men and in women. We examined the within and between components of variability using Cox regression models with gamma frailties (29, 30), which gave almost identical estimates of relative hazards due to the between-neighborhood variability being small, therefore findings from the former models are reported in the present study. Time since entry into the study was used as the underlying time metric. The proportional hazards assumption was assessed by modeling interaction terms of meat intake and time. Participants were followed from the date the baseline questionnaire was returned to the date of death or the end of study follow-up (December 31, 2005), whichever came first. Potential interactions were evaluated using cross-product terms along with the main effects in the model. The hazard ratio (HR) and 95% confidence interval (CI) were calculated for each variable in the Cox models. Fully adjusted models included age (continuous), education, race/ethnicity, marital status, family history of cancer (for cancer mortality only), body mass index (<18.5 , 18.5 to <25 , 25 to <30 , ≥ 30 kg/m^2), smoking (never, former ≤ 20 cigarettes/d, former >20 cigarettes/d, current ≤ 20 cigarettes/d, current >20 cigarettes/d, missing), vigorous physical activity (≥ 5 times/week), self-reported health status (excellent/great, good, fair/poor), vitamin or mineral use ($\geq$ once a month), total energy intake (continuous), alcohol intake (none, 0 to <5 , 5 to <15 , 15 to <30 , ≥ 30 g/day), and intakes of red meat, white meat, fruits, and vegetables (g/1000 kcal categorized into quintiles to examine patterns in diet). Analyses on women also considered menopausal hormone use (never, former, current). Covariates were progressively introduced in the model. Final models included covariates that altered the risk estimates by at least 10%. Multicollinearity was assessed by examining tolerance values; none was detected. Statistical significance was based on two-sided P values of <0.05 . Data were analyzed using SAS® (version 9.2, SAS Institute Inc., Cary, NC), and Survival package in R software (version 2.10) was used for the Cox regression with gamma frailties modeling.

RESULTS

Subject characteristics were examined across quintiles of deprivation for men and women separately (Table 1). In our study population, 6% men and 9% women resided in the most deprived areas (deprivation quintile 5). In general, those residing in the most deprived neighborhoods were more likely to be of African-American descent or of another non-white race, less educated, unmarried, current smokers, have higher daily intakes of total energy, consume less alcohol per day, and less likely to report being in good health or use vitamin/mineral supplements. Age-adjusted mean intakes of meat across neighborhood deprivation quintiles were significant ($P<0.001$).

Over a maximum follow-up of ten years, 33,831 decedents were reported. The leading causes of death included CVD and cancer (combined, approximately 70% of total deaths). The multivariable-adjusted HRs and corresponding 95% CIs for all-cause mortality according to quintiles of the neighborhood deprivation index are presented in Table 2. Estimates on cancer and CVD mortality are reported in Tables 3 and 4, respectively. Tests for interactions were not significant between red meats and quintiles of neighborhood deprivation, when examining all-cause or cause-specific mortality. Similarly, tests for interactions were not significant between processed meats and quintiles of neighborhood deprivation. For men, an effect modification by neighborhood was suggested when examining total and cancer mortality ($P=0.06$ and $P=0.03$, respectively) and white meat intake (Figure 1). Men residing in the least deprived neighborhoods had a stronger protective effect of white meat consumption compared to those in the most deprived areas, with the greatest reductions being observed for cancer mortality (total mortality, HR=0.79, 95% CI: 0.72–0.86; cancer mortality, HR=0.74, 95% CI: 0.64–0.84). These decreased risks were observed after adjusting for known risk factors, including age, education, smoking, physical activity and health status. A similar pattern was observed in women for cancer mortality, however, the modifying effect of neighborhood deprivation was not as strong and did not reach statistical significance ($P=0.09$). Risk of dying among women, but not men, with high-risk meat (high red, low white meat intake) diets was elevated for those residing in most-deprived neighborhoods (HR=1.45, 95% CI: 1.23–1.72). However, the interactions were not significant (P -values=0.06, 0.32, 0.24 for total, cancer, and CVD-related mortality, respectively). Tests for linear trend were significant for each meat type for quintiles of deprivation (P 0.01; data not shown), except for white meat consumption among women when modeling CVD-related mortality.

DISCUSSION

This study was undertaken to test whether the effect of dietary meat on mortality differed according to levels of neighborhood socioeconomic status, using a large prospective study of older U.S. adults. The primary question of interest was whether the type and quantity of meat consumption had a more deleterious effect in poorer neighborhoods than more affluent areas based on a census-tract socioeconomic deprivation index. We found that the risk of mortality from consumption of red and processed meats did not differ across neighborhoods of varying levels of socioeconomic deprivation. With regards to white meat intake, results suggest potential socioeconomic inequalities in total cancer mortality in men (largest effects being observed in the least deprived census tracts), whereas in women these inequalities were not as apparent.

Our findings of sex differences between deprivation and mortality are in accord with previous studies that found the socioeconomic-mortality association is weaker in women than men for person-level indicators of SES (31–33). The risks of mortality for men and

women with high meat intakes are consistent in both magnitude and significance (e.g., linear trends) with those previously reported by Sinha and colleagues (6).

The mechanism by which neighborhood deprivation influences the protective effect of white meat consumption, but not that of red or processed meats, is not clear. The observed benefits may partly be explained by differences in the physical environment or social norms. In particular, men residing in areas with higher deprivation may have issues such as limited availability of or access to healthy foods and health care, and lack of social networks that might impact mortality risk independently of the characteristics of the people living in those areas (20, 21). There might also be differences in the preparation or type of white meat consumed (e.g., more fried chicken, less fish) for those living in more socioeconomically disadvantaged neighborhoods. Eating white meat may be correlated with other health-promoting behaviors that were not measured or inadequately adjusted particularly among men.

Among the inherent strengths of the present study is the prospective design in which neighborhood deprivation and covariates were measured prior to mortality, which unlike ecologic study designs, permits examination of causation and provides data at both the individual- and aggregate-level. In addition, the NIH-AARP Study consisted of participants from 6 states and 2 metropolitan areas and was not limited to a specific geographical location as with other U.S. prospective studies examining area-based effects on mortality. Extensive data collection of information on lifestyle and medical history allowed us to control for possible confounding on a broad array of characteristics and lifestyle factors. In addition, we employed an innovative analytic method, the extended Cox model with a robust variance estimator, to simultaneously examine associations of person-level and area-level risk factors with mortality, which is unique in research examining the influence of neighborhood on mortality. Further, the large size of the NIH-AARP Diet and Health Study allowed us to stratify the study population by gender and examine interactions while maintaining study power.

A limitation of our study is that the cohort was predominantly upper-to-middle class Caucasians; therefore, results may not apply to more diverse populations. However, our study was population-based and more broadly generalizable to the general U.S. population than prospective studies examining meat intake and mortality in specific geographic locations or specific study populations (34, 35). Even within the NIH-AARP Diet and Health Study population that may have a limited range of neighborhood deprivation scores, we were still able to observe an effect modification by deprivation on the association between white meat intake and mortality. It is possible that this effect might actually be stronger for a population that has more diversity in socioeconomic status. Alternatively, it is possible that differences across categories of neighborhood deprivation reflect incomplete adjustment for person-level factors, including household income and occupation. In addition, data on the social norms, physical environment, quality of foods and the availability or access to healthy foods and health care was not ascertained; therefore, it was not possible to directly assess inequalities on these measures. To our knowledge, other prospective studies have not yet examined effect modification of these four mentioned factors on the association between meat intake and mortality and that the present study is the first to examine the impact of neighborhood deprivation on the associations. Although this information was not ascertained in the present study, the analyses were adjusted for a wide range of characteristics and lifestyle factors indicative of cancer and cardiovascular disease, including an extensive in-depth measure of smoking history and dietary intake. As it was not feasible to obtain clinical measures from a cohort of this magnitude, BMI was based on self-reported body weight and height. Self-reported and actual weight has been reported to be strongly correlated among U.S. adults (36, 37). While residual confounding of BMI may

exist, the consistency of our results for BMI with those of other studies would suggest that the influence of this type of bias was likely weak. Because we investigated multiple endpoints, it is possible that significant results may be due to chance. Future studies are needed to replicate these findings and to examine direct measures of neighborhood deprivation.

In conclusion, red and processed meat intake increases mortality risk regardless of the level of deprivation within a given neighborhood, even after accounting for a large number of known risk factors for mortality. Findings suggest biological mechanisms rather than neighborhood contextual factors may underlie these particular meat-mortality associations. Possible socioeconomic inequalities may exist among men with regards to white meat intake and risk of cancer mortality. Our findings require confirmation in other U.S. populations, including younger age groups and populations with a wider range of SES. If confirmed, the information gained could potentially be useful in identifying inequalities in lifestyle as well as the geographical areas most likely to benefit from strategies aimed at promoting a healthy environment.

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Figure 1.

Potential effect modification by neighborhood among men when examining all-cause and cancer mortality ($P=0.06$ and $P=0.03$, respectively) and white meat intake. Models adjusted for age, education, race, marital status, family history of cancer (cancer mortality), physical activity, smoking, health status, BMI, energy, cholesterol (CVD mortality), alcohol use, fruit/vegetables/meat intakes, and vitamin use.

Table 1

Age-adjusted characteristics across quintiles of the deprivation index

Parameter	Men (n=233,205)					Women (n=176,570)				
	Deprivation Index Quintiles					Deprivation Index Quintiles				
	Q1	Q3	Q5	Q1	Q5	Q1	Q3	Q5	Q1	Q5
Participants, n	77132	49366	13686	47894	39549	15749				
Total deaths, %	7.4	10.4	12.3	5.5	7.1	8.7				
CVD deaths, %	1.9	2.9	3.8	1.2	1.7	2.2				
Cancer deaths, %	3.2	4.2	4.5	2.7	3.0	3.5				
Age, years	61.4	62.0	61.6	61.2	61.9	61.7				
Race, %										
Non-Hispanic white	94.7	93.2	70.6	94.2	92.4	58.5				
Non-Hispanic black	0.9	2.1	20.8	1.4	3.0	33.0				
Other	4.4	4.7	8.6	4.4	4.6	8.4				
Currently married, %	88.5	84.6	72.3	49.0	45.4	34.6				
Family history of cancer, %	48.3	46.8	43.4	51.8	51.6	47.6				
BMI, kg/m ²	26.8	27.5	27.7	25.7	27.0	28.4				
Smoking history, %										
Never	37.0	29.7	30.3	46.0	47.4	48.6				
Former	53.9	56.4	51.7	40.8	35.7	31.9				
Current or quit <1yr	9.0	14.0	18.0	13.2	17.0	19.6				
Education, college grad+, %	64.1	35.1	29.6	44.1	24.2	22.8				
Activity 5+ times/wk, %	21.5	20.9	19.1	18.7	15.9	13.8				
Health status, %										
Excellent/great	68.6	57.7	49.4	65.7	54.3	43.8				
Good	27.2	34.9	38.6	28.4	36.0	40.2				
Fair/poor	4.2	7.4	12.0	5.9	9.7	16.0				
Current HRT use, %				67.0	61.1	52.5				
Dietary intake										
Energy, kcal/d	1961	2069	2131	1514	1572	1690				
Fruit, srv/1000 kcal	1.6	1.5	1.6	2.0	1.9	2.1				

Parameter	Men (n=233,205)				Women (n=176,570)			
	Deprivation Index Quintiles				Deprivation Index Quintiles			
	Q1	Q3	Q5		Q1	Q3	Q5	
Vegetables, srv/1000 kcal	2.1	2.0	1.9		2.6	2.5	2.3	
Alcohol, g/d	17.6	16.8	15.7		7.3	5.4	4.0	
Saturated fat, g/1000 kcal	10.4	10.9	10.9		10.0	10.5	10.4	
Cholesterol, mg	216	237	250		157	169	184	
Vitamin, mineral use l/mo	61.0	56.6	52.1		71.4	66.6	58.0	
Meat intake								
Red meat, g/1000 kcal	36.5	40.1	38.9		26.8	30.7	29.9	
White meat, g/1000 kcal	34.3	29.5	29.9		38.7	34.4	35.7	
Processed meat, g/1000 kcal	11.2	12.4	12.4		7.3	8.5	9.1	

Data are given as mean value or percentage of participants. Generalized linear models were used to estimate age-adjusted mean values for continuous variables and logistic regression for proportions within each deprivation quintile. There were approximately 3720 census tracts within each deprivation quintile.

Table 2
Associations of meat intake and all-cause mortality by quintiles of neighborhood deprivation

Meat intake	Men (n=233,205)					Women (n=176,570)				
	Deprivation Index Quintiles					Deprivation Index Quintiles				
	Least - Q1	Q3	Most - Q5	P		Least - Q1	Q3	Most - Q5	P	
Red meat				0.41					0.94	
Quintile 1 (ref)	1.0	1.0	1.0			1.0	1.0	1.0		
Quintile 2	1.03 (0.93–1.13)	0.96 (0.86–1.07)	1.01 (0.85–1.20)			1.10 (0.98–1.22)	1.02 (0.90–1.14)	0.96 (0.81–1.14)		
Quintile 3	1.15 (1.05–1.26)	1.08 (0.97–1.20)	1.00 (0.84–1.19)			1.16 (1.03–1.31)	1.20 (1.06–1.35)	1.09 (0.92–1.30)		
Quintile 4	1.21 (1.11–1.33)	1.11 (1.10–1.23)	1.07 (0.90–1.27)			1.25 (1.10–1.42)	1.31 (1.16–1.48)	1.31 (1.10–1.55)		
Quintile 5	1.28 (1.16–1.40)	1.25 (1.13–1.38)	1.21 (1.03–1.42)			1.36 (1.18–1.56)	1.30 (1.13–1.48)	1.29 (1.07–1.56)		
White meat				0.06					0.59	
Quintile 1 (ref)	1.0	1.0	1.0			1.0	1.0	1.0		
Quintile 2	0.82 (0.75–0.88)	0.94 (0.87–1.01)	0.91 (0.79–1.04)			0.85 (0.75–0.97)	0.95 (0.84–1.07)	0.98 (0.83–1.16)		
Quintile 3	0.86 (0.80–0.93)	0.91 (0.84–0.98)	0.79 (0.69–0.92)			0.86 (0.76–0.98)	0.95 (0.84–1.07)	0.80 (0.67–0.95)		
Quintile 4	0.78 (0.72–0.85)	0.91 (0.83–0.99)	0.80 (0.69–0.94)			0.82 (0.72–0.93)	0.97 (0.86–1.09)	0.88 (0.74–1.05)		
Quintile 5	0.79 (0.72–0.86)	0.92 (0.83–1.01)	0.90 (0.76–1.05)			0.82 (0.73–0.93)	0.91 (0.81–1.03)	0.85 (0.71–1.01)		
Processed meat				0.40					0.50	
Quintile 1 (ref)	1.0	1.0	1.0			1.0	1.0	1.0		
Quintile 2	0.97 (0.89–1.07)	0.94 (0.84–1.04)	1.06 (0.88–1.28)			0.98 (0.88–1.09)	0.93 (0.83–1.04)	1.12 (0.95–1.33)		
Quintile 3	1.04 (0.95–1.14)	1.02 (0.92–1.13)	1.08 (0.90–1.30)			1.02 (0.91–1.15)	0.97 (0.87–1.10)	1.14 (0.96–1.36)		
Quintile 4	1.10 (1.00–1.20)	1.03 (0.93–1.14)	1.17 (0.98–1.40)			1.08 (0.95–1.22)	1.14 (1.01–1.29)	1.24 (1.04–1.48)		
Quintile 5	1.21 (1.11–1.32)	1.11 (1.00–1.23)	1.33 (1.13–1.58)			1.27 (1.11–1.44)	1.15 (1.01–1.30)	1.36 (1.14–1.63)		

HRs (95% CIs) reported. *P*-value for test of interaction.

Deprivation quintiles: Q1=least-deprived, Q5=most-deprived.

Models adjusted for age, education, race, marital status, family history of cancer (cancer mortality), exercise, smoking, health status, BMI, energy, cholesterol (CVD mortality), alcohol, fruit/vegetables intakes, vitamin use. Models for women additionally adjusted for menopausal hormone use.

Table 3

Associations of meat intake and cancer mortality by quintiles of neighborhood deprivation

Meat intake	Men (n=233,205)					Women (n=176,569)				
	Deprivation Index Quintiles					Deprivation Index Quintiles				
	Least - Q1	Q3	Most - Q5	P		Least - Q1	Q3	Most - Q5	P	
Red meat				0.48					0.45	
Quintile 1 (ref)	1.0	1.0	1.0			1.0	1.0	1.0		
Quintile 2	0.97 (0.84–1.12)	0.91 (0.76–1.08)	0.99 (0.74–1.33)			1.09 (0.93–1.28)	0.91 (0.76–1.09)	0.96 (0.75–1.24)		
Quintile 3	1.05 (0.91–1.21)	1.06 (0.90–1.26)	1.01 (0.76–1.35)			1.08 (0.91–1.28)	1.12 (0.94–1.34)	0.94 (0.71–1.24)		
Quintile 4	1.13 (0.98–1.30)	1.11 (0.94–1.31)	1.13 (0.86–1.50)			1.15 (0.96–1.38)	1.19 (0.99–1.44)	1.10 (0.83–1.45)		
Quintile 5	1.14 (0.99–1.31)	1.22 (1.04–1.44)	1.16 (0.88–1.53)			1.27 (1.03–1.56)	1.02 (0.83–1.26)	1.06 (0.79–1.43)		
White meat				0.03					0.09	
Quintile 1 (ref)	1.0	1.0	1.0			1.0	1.0	1.0		
Quintile 2	0.83 (0.73–0.93)	1.03 (0.92–1.17)	1.03 (0.83–1.28)			0.84 (0.69–1.02)	1.02 (0.85–1.23)	1.05 (0.82–1.36)		
Quintile 3	0.84 (0.75–0.95)	1.01 (0.89–1.15)	0.72 (0.56–0.93)			0.87 (0.72–1.05)	0.99 (0.82–1.19)	0.73 (0.55–0.96)		
Quintile 4	0.82 (0.72–0.93)	0.90 (0.78–1.03)	0.81 (0.62–1.04)			0.89 (0.74–1.07)	0.95 (0.78–1.14)	0.81 (0.61–1.07)		
Quintile 5	0.74 (0.64–0.84)	1.00 (0.86–1.16)	0.91 (0.70–1.19)			0.87 (0.73–1.04)	0.94 (0.78–1.14)	0.79 (0.60–1.04)		
Processed meat				0.77					0.50	
Quintile 1 (ref)	1.0	1.0	1.0			1.0	1.0	1.0		
Quintile 2	0.91 (0.79–1.05)	0.97 (0.82–1.16)	1.03 (0.76–1.39)			1.03 (0.88–1.20)	0.82 (0.69–0.97)	1.16 (0.90–1.51)		
Quintile 3	1.00 (0.87–1.15)	1.06 (0.89–1.25)	0.99 (0.73–1.34)			1.02 (0.86–1.22)	0.89 (0.74–1.06)	1.14 (0.87–1.50)		
Quintile 4	0.93 (0.81–1.07)	1.10 (0.93–1.30)	1.14 (0.86–1.52)			1.06 (0.88–1.27)	1.01 (0.84–1.21)	0.99 (0.74–1.32)		
Quintile 5	1.02 (0.88–1.17)	1.17 (0.99–1.38)	1.22 (0.93–1.62)			1.13 (0.93–1.38)	0.97 (0.80–1.18)	1.12 (0.83–1.50)		

HRs (95% CIs) reported. *P*-value for test of interaction.

Deprivation quintiles: Q1=least-deprived, Q5=most-deprived.

Models adjusted for age, education, race, marital status, family history of cancer (cancer mortality), exercise, smoking, health status, BMI, energy, cholesterol (CVD mortality), alcohol, fruit/vegetables intakes, vitamin use. Models for women additionally adjusted for menopausal hormone use.

Table 4

Associations of meat intake and CVD mortality by quintiles of neighborhood deprivation

Meat intake	Men (n=233,205)					Women (n=176,569)				
	Deprivation Index Quintiles					Deprivation Index Quintiles				
	Least - Q1	Q3	Most - Q5	P		Least - Q1	Q3	Most - Q5	P	
Red meat				0.81					0.82	
Quintile 1 (ref)	1.0	1.0	1.0			1.0	1.0	1.0		
Quintile 2	1.03 (0.86–1.25)	0.93 (0.77–1.14)	0.76 (0.56–1.04)			1.03 (0.80–1.32)	0.96 (0.76–1.22)	0.98 (1.70–1.38)		
Quintile 3	1.25 (1.04–1.49)	0.97 (0.80–1.18)	0.84 (0.62–1.14)			1.32 (1.03–1.70)	1.18 (0.94–1.50)	1.33 (0.95–1.86)		
Quintile 4	1.26 (1.05–1.50)	1.06 (0.88–1.29)	1.03 (0.77–1.38)			1.46 (1.12–1.91)	1.20 (0.94–1.54)	1.75 (1.25–2.45)		
Quintile 5	1.36 (1.13–1.63)	1.22 (1.01–1.47)	1.17 (0.88–1.55)			1.45 (1.07–1.98)	1.33 (1.03–1.73)	1.50 (1.03–2.18)		
White meat				0.60					0.55	
Quintile 1 (ref)	1.0	1.0	1.0			1.0	1.0	1.0		
Quintile 2	0.85 (0.73–1.00)	0.88 (0.76–1.02)	0.88 (0.69–1.12)			0.85 (0.64–1.11)	0.84 (0.66–1.07)	1.00 (0.71–1.40)		
Quintile 3	0.92 (0.79–1.07)	0.87 (0.74–1.01)	0.84 (0.64–1.09)			0.93 (0.72–1.22)	1.10 (0.87–1.38)	0.99 (0.71–1.39)		
Quintile 4	0.76 (0.64–0.90)	0.95 (0.81–1.11)	0.81 (0.62–1.07)			0.85 (0.65–1.11)	1.01 (0.79–1.29)	0.92 (0.64–1.31)		
Quintile 5	0.94 (0.80–1.11)	0.95 (0.80–1.13)	1.00 (0.75–1.32)			0.77 (0.59–1.01)	1.06 (0.83–1.34)	0.95 (0.67–1.34)		
Processed meat				0.71					0.52	
Quintile 1 (ref)	1.0	1.0	1.0			1.0	1.0	1.0		
Quintile 2	1.06 (0.88–1.28)	0.93 (0.76–1.14)	0.91 (0.65–1.26)			0.95 (0.74–1.23)	1.07 (0.84–1.37)	1.40 (0.95–2.05)		
Quintile 3	1.13 (0.94–1.36)	1.04 (0.86–1.26)	0.94 (0.68–1.29)			1.01 (0.76–1.33)	1.98 (0.75–1.29)	1.45 (0.98–2.16)		
Quintile 4	1.18 (0.98–1.41)	0.93 (0.76–1.13)	1.06 (0.78–1.44)			1.25 (0.93–1.68)	1.17 (0.90–1.53)	1.90 (1.29–2.78)		
Quintile 5	1.35 (1.13–1.61)	1.16 (0.97–1.39)	1.22 (0.91–1.64)			1.52 (1.13–2.04)	1.35 (1.05–1.75)	1.71 (1.14–2.57)		

HRs (95% CIs) reported. *P*-value for test of interaction.

Deprivation quintiles: Q1=least-deprived, Q5=most-deprived.

Models adjusted for age, education, race, marital status, family history of cancer (cancer mortality), exercise, smoking, health status, BMI, energy, cholesterol (CVD mortality), alcohol, fruit/vegetables intakes, vitamin use. Models for women additionally adjusted for menopausal hormone use.