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Meat intake and mortality: a prospective study of over half a million people

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Abstract

Context—High intakes of red or processed meat may increase risk of mortality.

Objective—Determine the relations of red, white and processed meat intakes to risk for total, and cause-specific mortality.

Design, Setting, and Participants—The NIH-AARP Diet and Health Study cohort of half a million people aged 50-71 years at baseline. Meat intake was estimated from a food frequency questionnaire administered at baseline. Cox proportional hazards regression estimated hazard ratios (HRs) and 95% confidence intervals (CIs) within quintiles of meat intake. The covariates included in the models were: age; education; marital status; family history of cancer (yes/no) (cancer mortality only); race; body mass index; 31-level smoking history; physical activity; energy intake; alcohol intake; vitamin supplement use; fruit consumption; vegetable consumption; and menopausal hormone therapy among women.

Main Outcome Measure—Total mortality, deaths due to cancer, CVD, accidents, and other causes.

Results—There were 47,976 male deaths and 23,276 female deaths during 10 years of follow-up. Men and women in the highest versus lowest quintile of red (HR 1.31, 95% CI 1.27-1.35; HR 1.36, 95% CI 1.30-1.43, respectively) and processed meat intake (HR 1.16, 95% CI 1.12-1.20; HR 1.25, 95% CI 1.20-1.31, respectively) had elevated risks for overall mortality. Regarding cause-specific mortality, men and women had elevated risks for cancer mortality for red (HR 1.22, 95% CI 1.16-1.29; HR 1.20, 95% CI 1.12-1.30, respectively) and processed meats (HR 1.12, 95% CI 1.06-1.19; HR 1.11, 95% CI 1.04-1.19, respectively). Furthermore, CVD risk was elevated for men

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and women in the highest quintile of red (HR 1.27, 95% CI 1.20-1.35; HR 1.50, 95% CI 1.37-1.65, respectively) and processed meat (HR 1.09, 95% CI 1.03-1.15; HR 1.38, 95% CI 1.26-1.51, respectively). When comparing the highest to the lowest quintile of white meat intake, there was an inverse association for total mortality, and cancer mortality, as well as all other deaths for both men and women.

Conclusion—Red and processed meat intakes were associated with modest increases in total mortality, cancer mortality and CVD mortality.

Keywords

Red meat; white meat; processed meat; mortality; CVD; cancer; diet

Introduction

Meat intake varies substantially around the world, but the impact of consuming higher levels of meat in relation to chronic disease mortality is ambiguous.¹⁻⁶ To increase sample size, pooled analyses of meat intake have been carried out in Seventh-day Adventists in the United States^{7,8} and other vegetarian populations in Europe.⁹⁻¹² Vegetarian diets differ from non-vegetarian diets in several respects. The main sources of protein in a vegetarian diet are legumes, grains, and nuts. Vegetarian diets also include higher intakes of vegetables, unsaturated fats, dietary fiber, and antioxidants (carotenoids, vitamins C and E), whereas they contain lower amounts of iron, zinc and vitamin B12. Furthermore, other lifestyle factors, such as smoking, physical activity, and alcohol consumption among vegetarians and members of select religious groups can differ substantially from the general population.

We prospectively investigated red, white and processed meat intakes as risk factors for total mortality, as well as cause-specific mortality, including cancer, and cardiovascular disease (CVD) mortality in a cohort of approximately half a million men and women enrolled in the National Institutes of Health (NIH)-AARP (formerly known as the American Association of Retired Persons) Diet and Health Study. This large prospective study facilitated the investigation of a wide range of meat intakes with chronic disease mortality.

Materials and Methods

Study population

Individuals aged 50 to 71 years were recruited from six U.S. states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania) and two metropolitan areas (Atlanta, Georgia, and Detroit, Michigan) to form a large prospective cohort, the NIH-AARP Diet and Health Study. Questionnaires on demographic and lifestyle characteristics, including dietary habits, were mailed to 3.5 million members of AARP in 1995, described in detail elsewhere.¹³ The NIH-AARP Diet and Health Study was approved by the Special Studies Institutional Review Board of the U.S. National Cancer Institute. Completion of the baseline questionnaire was considered to imply informed consent.

Dietary assessment

A 124-item food frequency questionnaire (FFQ) (<http://riskfactor.cancer.gov/DHQ/forms/files/shared/dhq1.2002.sample.pdf>) was completed at baseline. The FFQ collected information on the usual consumption of foods and drinks and portion sizes over the last twelve months. The validity of the FFQ was estimated using two 24-hour recalls,¹⁴ and the estimated energy adjusted correlations ranged from 0.36 to 0.76 for various nutrients, and attenuation factors ranged from 0.24 to 0.68. Red meat intake was calculated using the frequency of consumption and portion size information of all types of beef

and pork and included bacon, beef, cold cuts, ham, hamburger, hot dogs, liver, pork, sausage, steak and meats in foods such as pizza, chili, lasagna, and stew. White meat included chicken, turkey, and fish and included poultry coldcuts, chicken mixtures, canned tuna, as well as low-fat sausages and low-fat hot dogs made from poultry. Processed meat included bacon, red meat sausage, poultry sausage, luncheon meats (red and white meat), cold cuts (red and white meat), ham, regular hotdogs and low-fat hotdogs made from poultry. The components constituting red or white and processed meats can overlap as both can include meats such as bacon, sausage, and ham, while processed meat can also include smoked turkey and chicken. However, these meat groups are not used in the same models thus they are not duplicated in any one analysis.

In order to investigate whether the overall composition of meat intake was associated with mortality, we created three diet types: high; medium; and low risk meat diet. To form these diet variables, red and white meat consumption was energy adjusted and split into two groups using the median values as a cutpoints. Individuals with red meat consumption in the upper half and white meat consumption in the lower half got a score of 1 (high risk meat diet), those with both red and white meat consumption in the same half got a score of 2 (medium risk meat diet), those with red meat consumption in the lower half and white meat consumption in the upper half got a score of 3 (low risk meat diet).

Cohort follow-up and case ascertainment

Cohort members were followed-up from the date the baseline questionnaire was returned (beginning 1995) through December 31, 2005 by annual linkage of the cohort to the National Change of Address database maintained by the U.S. Postal Service and through processing of undeliverable mail, other address change update services, and directly from cohort members' notifications. For matching purposes, we have virtually complete data on first and last name, address history, gender, and date of birth. Follow-up for vital status is performed by annual linkage of the cohort to the Social Security Administration Death Master File in the U.S. Verification of vital status and cause of death information is provided by follow-up searches of the National Death Index (NDI) Plus with the current follow-up for mortality covered until 2005.

Cause-Specific Case Ascertainment

Cancer (ICD9: 140-239; ICD10: C00-C44, C45.0, C45.1, C45.7, C45.9, C48-C97, D12-D48) - mortality included deaths due to cancers of the oral cavity and pharynx, digestive tract, respiratory tract, soft tissue (including heart), skin (excluding basal and squamous cell carcinoma), female genital system and breast, male genital system, urinary tract, endocrine system, lymphoma, leukemia, and other miscellaneous cancers.

Cardiovascular disease (CVD) (ICD9: 390-398, 401-404, 410-438, 440-448; ICD10: I00-I09, I10-I13, I20-I51, I60-I78) - mortality was from a combination of diseases of the heart, hypertension without heart disease, cerebrovascular diseases, atherosclerosis, aortic aneurysm and dissection, and other diseases of the arteries, arterioles, and capillaries.

Mortality from injuries and sudden deaths (ICD9: 800-978; ICD10: U01-U03, V01-Y09, Y35, Y85-Y86, Y87.0, Y87.1 Y89.0) - included accidents, adverse effects, suicide, self-inflicted injury, homicide, and legal intervention.

All others deaths included mortality from tuberculosis, human immunodeficiency virus, other infectious and parasitic diseases, septicemia, diabetes mellitus, Alzheimer's, stomach and duodenal ulcers, pneumonia and influenza, chronic obstructive pulmonary disease and allied conditions, chronic liver disease and cirrhosis, nephritis, nephrotic syndrome and nephrosis;

congenital anomalies; certain conditions originating in the perinatal period, ill-defined conditions, and unknown causes of death.

Total mortality is a combination of all of the above mentioned causes of deaths.

Statistical analysis

A total of 617,119 persons returned the baseline questionnaire; of these, we excluded individuals who moved out of the eight study areas before returning the baseline questionnaire (n = 321), requested to be withdrawn from the study (n = 829), died before study entry (n = 261), had duplicate records (n = 179), indicated that they were not the intended respondent and did not complete the questionnaire (n = 13,442), provided no information on gender (n = 6), did not answer substantial portions of the questionnaire or had greater than 10 recording errors (n = 35,679). After these exclusions, we further removed individuals whose questionnaire was filled in by someone else on their behalf (n = 15,760). We excluded 4,849 subjects reporting extreme daily total energy intake defined as more than two inter-quartile ranges above the 75th percentile or below the 25th percentile and 140 people who had zero person years of follow up. After all exclusions, our analytic cohort consisted of 322,263 men and 223,390 women.

We estimated hazard ratios (HRs) and 95% confidence intervals (CIs) with time since entry into the study as the underlying time metric using Cox proportional hazards regression. Quintile cut-points were based on the entire cohort and multivariate adjusted HRs are reported using the lowest quintile as the referent category. The violation of the proportional hazard assumption was investigated by testing an interaction between a time dependent binary covariate, which indicated if follow-up was in the first 5 years or in the second 5 years, and the quintile terms for meat consumption. Dietary variables were energy adjusted using the nutrient density method and meat variables in each model added up to total meat (addition model). For example, one model contained both red and white meat while the processed meat model also contained a non-processed meat variable.

In order to address confounding we used forward stepwise variable selection to include covariates to develop the fully adjusted model. Smoking was the largest confounder of the association between meat intake and mortality. Physical activity and education were also important covariates, but not to the same degree as smoking. The final model included: age (continuous); education (less than 8 years or unknown, 8 to 11 years, 12 years (high school), some college, college graduate); marital status (married: yes/no); family history of cancer (yes/no) (cancer mortality only); race (non-Hispanic white, non-Hispanic black, Hispanic/Asian/Pacific Islander/American Indian/Alaskan native or unknown); body mass index (18.5-<25, 25-<30, 30-<35, ≥ 35 kg/m²); 31-level smoking history using smoking status (never, former, current), time since quitting for former smokers, and smoking dose; frequency of vigorous physical activity (never/rarely, 1-3 times/month, 1-2 times/week, 3-4 times/week, 5 or more times/week); total energy intake (continuous); alcohol intake (none, 0-<5, 5-<15, 15-<30, ≥ 30 g/day); vitamin supplement user (one or more supplement per month); fruit consumption (0 - <0.7, 0.7-< 1.2, 1.2-< 1.7, 1.7-< 2.5, ≥ 2.5 servings/1000 kcal); vegetable consumption (0-<1.3, 1.3-< 1.8, 1.8-< 2.2, 2.2-<3.0, ≥ 3.0 servings/1000 kcal); and menopausal hormone therapy among women in the multivariate models.

In sub-analyses, we investigated the relation between meat intake and mortality by smoking status. We used median values of each quintile to test for linear trend with two-sided P-values. We also calculated population attributable risks (PAR) as an estimate of the percent of mortality that could be prevented if individuals adopted intake levels of participants within the first quintile. This was computed as one minus the ratio consisting of the sum of the estimated hazards (derived from the Cox proportional hazard models) of each member of the cohort divided by the sum of the estimated hazards where meat exposure was assigned to the lowest

or highest quintile, depending on which quintile was the ideal level of meat consumption. The PAR was multiplied by 100 to convert them to a percentage. All statistical analyses were carried out using Statistical Analytic Systems (SAS) software (SAS Institute Inc, Cary, NC).

Results

During 10 years of follow-up, there were 47,976 male deaths and 23,276 female deaths. In general, those in the highest quintile of red meat intake tended to consume a slightly lower amount of white meat, but a higher amount of processed meat than those in the lowest quintile. Subjects who consumed more red meat tended to be married, more likely to be of non-Hispanic white ethnicity, more likely to be a current smoker, have a higher body mass index, and a higher daily intake of energy, total fat and saturated fat; whereas they tended to have a lower education level, were less physically active and consumed less fruits, vegetables, fiber and vitamin supplements (Table 1).

Red Meat

There was an overall increased risk of total, cancer, and CVD mortality, as well as all other deaths in both men (Table 2) and women (Table 3) in the highest compared to the lowest quintile of red meat intake in the fully adjusted model. There was an increased risk associated with accidental deaths with higher consumption of red meat in men but not in women.

White meat

When comparing the highest to the lowest quintile of white meat intake, there was an inverse association for total mortality, and cancer mortality, as well as all other deaths for both men (Table 2) and women (Table 3). In contrast, there was a small increase in risk for CVD mortality in men with higher intake of white meat. There was no association between white meat consumption and accidental death in men or women.

Processed Meat

There was an overall increased risk of total, cancer, and CVD mortality, as well as all other deaths in both men (Table 2) and women (Table 3) in the highest compared to the lowest quintile of processed meat intake. In contrast, there was no association for processed meat intake and accidental deaths in either gender.

A lag analysis, excluding deaths occurring in the first two years of follow up, produced results consistent with the main findings in Tables 2 and 3. For example, the HRs for total mortality in men for red meat was: 2nd quintile HR 1.05, 95% CI 1.01-1.09; 3rd quintile HR 1.13, 95% CI 1.09-1.17; 4th quintile HR 1.20, 95% CI 1.16-1.24; 5th quintile HR 1.30, 95% CI 1.26-1.35. For women, the HRs were: 2nd quintile HR 1.07, 95% CI 1.02-1.12; 3rd quintile HR 1.15, 95% CI 1.11-1.21; 4th quintile HR 1.27, 95% CI 1.21- 1.33; 5th quintile HR 1.35, 95% CI 1.28-1.42. Furthermore, we investigated our models for a violation of the proportional hazard assumption. Proportional hazard assumption was not rejected for all analyses except one, the model with red and white meat among the women for total mortality ($p=0.008$). Upon further examination in that model of the relative hazard between the first 5 years of follow up and the second 5 years of follow up, the red meat results were consistent between the two follow-up time periods. However, for white meat the second 5 year period showed little inverse trend as compared to the first 5 year period (data not shown).

We investigated whether people who consumed a high risk meat diet had mortality risk profiles that were different than people who consumed a low risk meat diet. Both men and women who consumed a low risk meat diet had statistically significant lower HRs compared to people who consumed a high risk meat diet for all cause, cancer, CVD mortality, as well as all other deaths;

for example, for all cause mortality, the HR for a low risk meat diet was 0.92, 95% CI 0.80-0.94 for men and 0.80, 95% CI 0.78-0.84 for women.

To further explore possible confounding by smoking, we analyzed meat intake and mortality in two subgroups - never-smokers (15,413 deaths among 190,135 never-smokers) and past/current smokers (n= 52,754 deaths among 335,036 past/current smokers). For men, the risks in the 5th quintile of red meat intake for never and past/current smokers, respectively, were: HR 1.28, 95% CI 1.19-1.38 and HR 1.25, 95% CI 1.20-1.30 for total mortality; HR 1.16, 95% CI 1.02-1.33 and 95% HR 1.17, 95% CI 1.09-1.24 for cancer mortality; 1.43, 95% CI 1.25-1.63 and HR 1.17; 95% CI 1.10-1.26 for CVD mortality. In women, the risks in the 5th quintile of red meat intake for never and past/current smokers, respectively, were: HR 1.36, 95% CI 1.25-1.48 and HR 1.28; 95% CI 1.21-1.35 for total mortality; HR 1.10, 95% CI 0.95-1.27 and HR 1.16, 95% CI 1.06-1.27 for cancer mortality; HR 1.63, 95% CI 1.38-1.93 and HR 1.34, CI 1.18-1.51 for CVD mortality. Risks were similar for the two smoking categories in most instances for processed meat except for cancer mortality where we found a null relation for both genders in never-smokers (men: HR 1.01, 95% CI 0.88-1.15; women: HR 1.02; 95% CI 0.89-1.17), but in ever/current smokers we found higher risks (men: HR 1.12, 95% CI 1.05-1.19; women: HR 1.11, 95% CI 1.02-1.21). Intriguingly, there was increased risk with higher intake of white meat for CVD mortality in never-smokers (men: HR 1.24, 95% CI 1.10-1.40; women: HR 1.20, CI 1.03-1.41).

We calculated the population attributable risks, representing the percentage of deaths that could be prevented if individuals adopted red or processed meat intake levels of participants within the first quintile. For overall mortality, an 11% of deaths in men and 16% of deaths in women could be prevented if people decreased their red meat consumption to the level of intake in the first quintile. The impact on CVD mortality was an 11% decrease in men and a 21% decrease in women if the red meat consumption was decreased to the amount consumed by individuals in the first quintile. The median red meat consumption based on men and women in the 1st quintile was 9.8 g/1000kcal per day as compared to 62.5 g/1000kcal per day in the 5th quintile. For women eating processed meat at the first quintile level the decrease in CVD mortality was approximately 20%. The median processed meat consumption based on men and women in the 1st quintile was 1.6g/1000kcal per day as compared to 22.6 g/1000kcal per day in the 5th quintile.

Discussion

We examined total and cause-specific mortality in relation to meat consumption in a large prospective study. We found modest increases in risk for total mortality, as well as cancer and CVD mortality with higher intakes of red and processed meat in both men and women. In contrast, higher white meat consumption was associated with a small decrease in total and cancer mortality in men and women.

The principal strength of this study is the large size of the cohort, which provided us the ability to investigate the relationship of many deaths (47,976 male deaths and 23,276 female deaths) within the context of a single study with a standardized protocol and a wide range of meat consumption. In contrast, other reports investigating meat intake in relation to mortality have pooled data from different studies conducted in California, the United Kingdom, and Germany because the numbers of events were limited in each study.¹⁵⁻²⁶ The protocols and questionnaires in these studies were different as were the populations: Seventh-day Adventists in California and vegetarians and non-vegetarians in Europe. Pooled analyses of specialized populations with distinct healthy lifestyles, are subject to unmeasured confounding. Furthermore, recall bias and reverse causality were minimized in our study as diet was assessed prior to the diagnosis of the conditions that led to death.

There is a possibility that some residual confounding by smoking may remain; however, we used a detailed 31-level smoking history variable and repeated the analyses within smoking status strata. Within smoking sub-groups we found consistent results for red, white and processed meat intakes; however, there were some intriguing differences that could be further investigated; we found a positive association for processed meat intake and cancer mortality among past/current smokers, but not among never-smokers. This may be because we were still not able to fully statistically adjust for residual confounding of smoking as people who eat processed meat may also smoke. An additional reason could be that smokers are inhaling carcinogenic chemicals as well as being exposed to *N*-nitroso-compounds from processed meats. The possible reason why there was increased risk with white meat consumption among never-smokers is not readily apparent

Because our cohort was predominantly non-Hispanic white, more educated, consumed less fat and red meat and more fiber and fruits and vegetables, and had somewhat fewer current smokers than comparably aged adults in the US population, caution should be applied when attempting to generalize our findings to other populations,²⁷ although this caution is somewhat tempered as it is unlikely that the mechanisms relating meat to mortality differs quantitatively between our study population and other Caucasian populations over 50 years old. Furthermore, the population attributable risks in our cohort may be conservative estimates because red and processed meat consumption may be higher in the general population than in our cohort.

The inherent limitations of measurement error in this study are similar to those of any nutritional epidemiology study that is based on recall of usual intake over a given period. We attempted to reduce measurement error by adjusting our models for reported energy intake.²⁸ Correlations for red meat assessed from the FFQ compared with two 24-hour recall diaries were 0.62 for men and 0.70 for women as reported previously by Schatzkin et al.²⁷ The problem of residual confounding may still exist and could explain the relatively small associations found throughout this paper despite the care taken to adjust for known confounders.

Overall, we did not find statistically significant association between meat consumption and accidental deaths in most instances. The relative hazards of meat consumption with the other causes of death (total, cancer and CVD mortality) were similar in magnitude in some cases to those of accidental deaths; however, the number of accidental deaths was fewer than the other causes of deaths and thus the HRs were generally not statistically significant. We did observe a higher risk with the category that included “all other deaths”; this is a broad category with many heterogeneous conditions (such as diabetes mellitus, Alzheimer's, stomach and duodenal ulcers, chronic liver disease, cirrhosis, nephritis, nephrotic syndrome and nephrosis, etc.), some of which may be positively related to meat intake.

There are various mechanisms by which meat may be related to mortality. In relation to cancer, meat is a source of several multi-site carcinogens, including heterocyclic amines and polycyclic aromatic hydrocarbons,²⁹⁻³⁴ which are both formed during high-temperature cooking of meat, as well as *N*-nitroso compounds.^{35,36} Iron in red meat may increase oxidative damage and increase the formation of *N*-nitroso-compounds.³⁷⁻⁴⁰ Furthermore, meat is a major source of saturated fat, which has been positively associated with breast⁴¹⁻⁴³ and colorectal cancer.⁴⁴

In relation to CVD, elevated blood pressure has been shown to be positively associated with higher intakes of red and processed meat, even though the mechanism is unclear except possibly meat may substituted for other beneficial foods such as grains, fruits or vegetables.⁴⁵ Mean plasma total cholesterol, low density-lipoprotein cholesterol, very-low-density-lipoprotein cholesterol, and triglycerides were found to decrease in subjects who substituted red meat with fish.^{46,47} Vegetarians have a lower proportion of arachadonic acid, eicosapentaenoate and docosahexaenoate in platelet phospholipids and higher platelet phospholipids linoleate and

antioxidants; such a biochemical profile may be related to decreased atherogenesis and thrombogenesis.⁴⁸⁻⁵⁰

Red and processed meat intakes, as well as a high risk meat diet, were associated with a modest increase in risk of total mortality, cancer, and CVD mortality in both men and women. In contrast, high white meat intake and a low risk meat diet was associated with a small decrease in total and cancer mortality. These results complement the recommendations by the American Institute for Cancer Research and the World Cancer Research Fund to reduce red and processed meat intake to decrease cancer incidence.⁴⁴ Future research should investigate the relation between sub-types of meat and specific causes of mortality.

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Reference List

1. Fraser GE. Associations between diet and cancer, ischemic heart disease, and all-cause mortality in non-Hispanic white California Seventh-day Adventists. *Am J Clin Nutr* 1999;70:532S–538S. [PubMed: 10479227]
2. Kahn HA, Phillips RL, Snowdon DA, Choi W. Association between reported diet and all-cause mortality. Twenty-one-year follow-up on 27,530 adult Seventh-Day Adventists. *Am J Epidemiol* 1984;119:775–787. [PubMed: 6720674]
3. Appleby PN, Key TJ, Thorogood M, Burr ML, Mann J. Mortality in British vegetarians. *Public Health Nutr* 2002;5:29–36. [PubMed: 12001975]
4. Key TJ, Fraser GE, Thorogood M, et al. Mortality in vegetarians and non-vegetarians: a collaborative analysis of 8300 deaths among 76,000 men and women in five prospective studies. *Public Health Nutr* 1998;1:33–41. [PubMed: 10555529]
5. Key TJ, Fraser GE, Thorogood M, et al. Mortality in vegetarians and nonvegetarians: detailed findings from a collaborative analysis of 5 prospective studies. *Am J Clin Nutr* 1999;70:516S–524S. [PubMed: 10479225]
6. Thorogood M, Mann J, Appleby P, McPherson K. Risk of death from cancer and ischaemic heart disease in meat and non-meat eaters. *BMJ* 1994;308:1667–1670. [PubMed: 8025458]
7. Fraser GE. Associations between diet and cancer, ischemic heart disease, and all-cause mortality in non-Hispanic white California Seventh-day Adventists. *Am J Clin Nutr* 1999;70:532S–538S. [PubMed: 10479227]
8. Kahn HA, Phillips RL, Snowdon DA, Choi W. Association between reported diet and all-cause mortality. Twenty-one-year follow-up on 27,530 adult Seventh-Day Adventists. *Am J Epidemiol* 1984;119:775–787. [PubMed: 6720674]

9. Appleby PN, Key TJ, Thorogood M, Burr ML, Mann J. Mortality in British vegetarians. *Public Health Nutr* 2002;5:29–36. [PubMed: 12001975]
10. Key TJ, Fraser GE, Thorogood M, et al. Mortality in vegetarians and non-vegetarians: a collaborative analysis of 8300 deaths among 76,000 men and women in five prospective studies. *Public Health Nutr* 1998;1:33–41. [PubMed: 10555529]
11. Key TJ, Fraser GE, Thorogood M, et al. Mortality in vegetarians and nonvegetarians: detailed findings from a collaborative analysis of 5 prospective studies. *Am J Clin Nutr* 1999;70:516S–524S. [PubMed: 10479225]
12. Thorogood M, Mann J, Appleby P, McPherson K. Risk of death from cancer and ischaemic heart disease in meat and non-meat eaters. *BMJ* 1994;308:1667–1670. [PubMed: 8025458]
13. Schatzkin A, Subar AF, Thompson FE, et al. Design and serendipity in establishing a large cohort with wide dietary intake distributions : the National Institutes of Health-American Association of Retired Persons Diet and Health Study. *Am J Epidemiol* 2001;154:1119–1125. [PubMed: 11744517]
14. Thompson FE, Kipnis V, Midthune D, et al. Performance of a food-frequency questionnaire in the US NIH-AARP (National Institutes of Health-American Association of Retired Persons) Diet and Health Study. *Public Health Nutr* 2007:1–13.
15. Fraser GE. Associations between diet and cancer, ischemic heart disease, and all-cause mortality in non-Hispanic white California Seventh-day Adventists. *Am J Clin Nutr* 1999;70:532S–538S. [PubMed: 10479227]
16. Beeson WL, Mills PK, Phillips RL, Andress M, Fraser GE. Chronic disease among Seventh-day Adventists, a low-risk group. Rationale, methodology, and description of the population. *Cancer* 1989;64:570–581. [PubMed: 2743251]
17. Kahn HA, Phillips RL, Snowdon DA, Choi W. Association between reported diet and all-cause mortality. Twenty-one-year follow-up on 27,530 adult Seventh-Day Adventists. *Am J Epidemiol* 1984;119:775–787. [PubMed: 6720674]
18. Sanjoaquin MA, Appleby PN, Thorogood M, Mann JI, Key TJ. Nutrition, lifestyle and colorectal cancer incidence: a prospective investigation of 10998 vegetarians and non-vegetarians in the United Kingdom. *Br J Cancer* 2004;90:118–121. [PubMed: 14710217]
19. Appleby PN, Key TJ, Thorogood M, Burr ML, Mann J. Mortality in British vegetarians. *Public Health Nutr* 2002;5:29–36. [PubMed: 12001975]
20. Key TJ, Fraser GE, Thorogood M, et al. Mortality in vegetarians and non-vegetarians: a collaborative analysis of 8300 deaths among 76,000 men and women in five prospective studies. *Public Health Nutr* 1998;1:33–41. [PubMed: 10555529]
21. Appleby PN, Thorogood M, Mann JI, Key TJ. The Oxford Vegetarian Study: an overview. *Am J Clin Nutr* 1999;70:525S–531S. [PubMed: 10479226]
22. Key TJ, Fraser GE, Thorogood M, et al. Mortality in vegetarians and nonvegetarians: detailed findings from a collaborative analysis of 5 prospective studies. *Am J Clin Nutr* 1999;70:516S–524S. [PubMed: 10479225]
23. Appleby PN, Thorogood M, Mann JI, Key TJ. Low body mass index in non-meat eaters: the possible roles of animal fat, dietary fibre and alcohol. *Int J Obes Relat Metab Disord* 1998;22:454–460. [PubMed: 9622343]
24. Mann JI, Appleby PN, Key TJ, Thorogood M. Dietary determinants of ischaemic heart disease in health conscious individuals. *Heart* 1997;78:450–455. [PubMed: 9415002]
25. Thorogood M, Mann J, Appleby P, McPherson K. Risk of death from cancer and ischaemic heart disease in meat and non-meat eaters. *BMJ* 1994;308:1667–1670. [PubMed: 8025458]
26. Snowdon DA, Phillips RL, Fraser GE. Meat consumption and fatal ischemic heart disease. *Prev Med* 1984;13:490–500. [PubMed: 6527990]
27. Schatzkin A, Subar AF, Thompson FE, et al. Design and serendipity in establishing a large cohort with wide dietary intake distributions : the National Institutes of Health-American Association of Retired Persons Diet and Health Study. *Am J Epidemiol* 2001;154:1119–1125. [PubMed: 11744517]
28. Kipnis V, Subar AF, Midthune D, et al. Structure of dietary measurement error: results of the OPEN biomarker study. *Am J Epidemiol* 2003;158:14–21. [PubMed: 12835281]

29. Knize MG, Dolbeare FA, Carroll KL, Moore DH, Felton JS. Effect of cooking time and temperature on the heterocyclic amine content of fried beef patties. *Food Chem Toxicol* 1994;32:595–603. [PubMed: 8045472]
30. Sinha R, Knize MG, Salmon CP, et al. Heterocyclic amine content of pork products cooked by different methods and to varying degrees of doneness. *Food Chem Toxicol* 1998;36:289–297. [PubMed: 9651045]
31. Sinha R, Rothman N, Salmon CP, et al. Heterocyclic amine content in beef cooked by different methods to varying degrees of doneness and gravy made from meat drippings. *Food Chem Toxicol* 1998;36:279–287. [PubMed: 9651044]
32. Skog K, Steineck G, Augustsson K, Jagerstad M. Effect of cooking temperature on the formation of heterocyclic amines in fried meat products and pan residues. *Carcinogenesis* 1995;16:861–867. [PubMed: 7728968]
33. Sugimura T, Wakabayashi K, Ohgaki H, Takayama S, Nagao M, Esumi H. Heterocyclic amines produced in cooked food: unavoidable xenobiotics. *Princess Takamatsu Symp* 1990;21:279–288. [PubMed: 2134681]
34. Kazerouni N, Sinha R, Hsu CH, Greenberg A, Rothman N. Analysis of 200 food items for benzo[a]pyrene and estimation of its intake in an epidemiologic study. *Food Chem Toxicol* 2001;39:423–436. [PubMed: 11313108]
35. Hughes R, Cross AJ, Pollock JR, Bingham S. Dose-dependent effect of dietary meat on endogenous colonic N-nitrosation. *Carcinogenesis* 2001;22:199–202. [PubMed: 11159760]
36. Cross AJ, Sinha R. Meat-related mutagens/carcinogens in the etiology of colorectal cancer. *Environ Mol Mutagen* 2004;44:44–55. [PubMed: 15199546]
37. Kato I, Dnistrian AM, Schwartz M, et al. Iron intake, body iron stores and colorectal cancer risk in women: a nested case-control study. *Int J Cancer* 1999;80:693–698. [PubMed: 10048969]
38. Kabat GC, Miller AB, Jain M, Rohan TE. A cohort study of dietary iron and heme iron intake and risk of colorectal cancer in women. *Br J Cancer* 2007;97:118–122. [PubMed: 17551493]
39. Lee DH, Jacobs DR Jr, Folsom AR. A hypothesis: interaction between supplemental iron intake and fermentation affecting the risk of colon cancer. *The Iowa Women's Health Study. Nutr Cancer* 2004;48:1–5. [PubMed: 15203371]
40. Wurzelmann JI, Silver A, Schreinemachers DM, Sandler RS, Everson RB. Iron intake and the risk of colorectal cancer. *Cancer Epidemiol Biomarkers Prev* 1996;5:503–507. [PubMed: 8827353]
41. Bingham SA, Luben R, Welch A, Wareham N, Khaw KT, Day N. Are imprecise methods obscuring a relation between fat and breast cancer? *Lancet* 2003;362:212–214. [PubMed: 12885485]
42. Thiebaut AC, Kipnis V, Chang SC, et al. Dietary fat and postmenopausal invasive breast cancer in the National Institutes of Health-AARP Diet and Health Study cohort. *J Natl Cancer Inst* 2007;99:451–462. [PubMed: 17374835]
43. Midthune D, Kipnis V, Freedman LS, Carroll RJ. Binary Regression in Truncated Samples, with Application to Comparing Dietary Instruments in a Large Prospective Study. *Biometrics*. 2007
44. The World Cancer Research Fund / American Institute for Cancer Research. *Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective*. Washington, DC: AICR; 2007.
45. Steffen LM, Kroenke CH, Yu X, et al. Associations of plant food, dairy product, and meat intakes with 15-y incidence of elevated blood pressure in young black and white adults: the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Am J Clin Nutr* 2005;82:1169–1177. [PubMed: 16332648]
46. Gascon A, Jacques H, Moorjani S, Deshaies Y, Brun LD, Julien P. Plasma lipoprotein profile and lipolytic activities in response to the substitution of lean white fish for other animal protein sources in premenopausal women. *Am J Clin Nutr* 1996;63:315–321. [PubMed: 8602586]
47. Wolmarans P, Benade AJ, Kotze TJ, Daubitzer AK, Marais MP, Laubscher R. Plasma lipoprotein response to substituting fish for red meat in the diet. *Am J Clin Nutr* 1991;53:1171–1176. [PubMed: 2021128]
48. Jacques H, Gascon A, Bergeron N, et al. Role of dietary fish protein in the regulation of plasma lipids. *Can J Cardiol* 1995;11:63G–71G.

49. Wolmarans P, Benade AJ, Kotze TJ, Daubitzer AK, Marais MP, Laubscher R. Plasma lipoprotein response to substituting fish for red meat in the diet. *Am J Clin Nutr* 1991;53:1171–1176. [PubMed: 2021128]
50. Sanders TA, Oakley FR, Miller GJ, Mitropoulos KA, Crook D, Oliver MF. Influence of n-6 versus n-3 polyunsaturated fatty acids in diets low in saturated fatty acids on plasma lipoproteins and hemostatic factors. *Arterioscler Thromb Vasc Biol* 1997;17:3449–3460. [PubMed: 9437192]

Abbreviations

AARP	formerly known as the American Association of Retired Persons
CI	confidence interval
CVD	cardiovascular disease
FFQ	food frequency questionnaire
HR	hazard ratio
RR	relative risk

Table 1
Selected age-adjusted characteristics of AARP cohort (n=545,653) by red meat quintile category means and proportions*

Characteristics	Quintile red meat, g/1000 kcal				
	Q1	Q2	Q3	Q4	Q5
Men (n=322,263)					
Red meat (g/1000 kcal)	9.3	21.4	31.5	43.1	68.1
White meat (g/1000kcal)	36.6	32.2	30.7	30.4	30.9
Processed meat (g/1000kcal)	5.1	7.8	10.3	13.3	19.4
Age (years)	62.8	62.8	62.5	62.3	61.7
Race					
Non-Hispanic white (%)	88.6	91.8	93.1	94.0	94.1
Non-Hispanic black (%)	4.2	3.2	2.7	2.2	1.9
Hispanic, Asian, Pacific Islander, American Indian, Alaskan native or unknown (%)	7.2	5.0	4.2	3.8	4.0
Positive family history of cancer (%)	47.0	47.7	48.4	48.6	47.8
Currently married (%)	80.8	84.4	86.1	86.7	85.6
Body Mass Index, kg/m ²	25.9	26.7	27.1	27.6	28.3
Smoking history [‡]					
Never smoker (%)	34.4	30.5	28.8	27.6	25.4
Former smoker (%)	56.5	58.1	57.5	57.1	55.8
Current smoker or having quit < 1 year ago (%)	4.9	7.6	9.9	11.4	14.8
Education, college graduate or post graduate (%)	53.0	47.3	45.1	42.3	39.1
Vigorous physical activity ≥ 5 times per week (%)	30.7	23.6	20.5	18.6	16.3
Dietary intake					
Energy (kcal/day)	1899	1955	1998	2038	2116
Fruit (servings/1000 kcal)	2.3	1.8	1.6	1.4	1.1
Vegetables (servings/1000 kcal)	2.4	2.1	2.0	2.0	1.9
Alcohol (g/day)	20.2	20.4	17.6	15.3	12.5
Total fat (g/1000 kcal)	25.8	30.5	33.5	35.9	39.4
Saturated fat (g/1000 kcal)	7.6	9.4	10.5	11.3	12.7
Fiber (g/1000 kcal)	13.2	11.0	10.2	9.6	8.8

Characteristics	Quintile red meat, g/1000 kcal				
	Q1	Q2	Q3	Q4	Q5
Vitamin supplement use 1 or more/month	67.3	62.1	59.1	55.8	52.0
Women (n=223,390)					
Red meat (g/1000 kcal)	9.1	21.2	31.2	42.8	65.9
White meat (g/1000kcal)	37.4	35.6	34.9	35.1	35.3
Processed meat (g/1000kcal)	3.8	6.4	8.7	11.3	16.0
Age (years)	62.2	62.2	62.0	61.7	61.3
Race					
Non-Hispanic white (%)	86.2	89.9	91.0	91.8	91.4
Non-Hispanic black (%)	7.5	5.5	4.8	4.1	3.8
Hispanic, Asian, Pacific Islander, American Indian, Alaskan native or unknown (%)	6.3	4.5	4.3	4.1	4.9
Positive family history of cancer (%)	51.4	53.0	52.9	52.4	51.5
Currently married (%)	37.2	42.4	46.3	48.8	50.7
Body Mass Index, kg/m ²	25.6	26.6	27.1	27.7	28.4
Never on hormone replacement (females only)	46.6	46.3	47.1	48.1	50.5
Smoking history [‡]					
Never smoker (%)	45.5	44.3	43.23	42.2	40.0
Former smoker (%)	41.8	39.5	38.1	37.0	35.4
Current smoker or having quit < 1 year ago (%)	8.8	12.7	15.3	17.7	21.2
Education, college graduate or post graduate (%)	37.1	30.7	27.7	25.6	22.7
Vigorous physical activity ≥ 5 times per week (%)	22.5	16.3	13.9	12.0	11.0
Dietary intake					
Energy (kcal/day)	1526	1539	1584	1613	1646
Fruit (servings/1000 kcal)	2.5	2.0	1.8	1.5	1.3
Vegetables (servings/1000 kcal)	2.8	2.5	2.4	2.3	2.3
Alcohol (g/day)	5.8	6.3	6.2	5.7	5.1
Total fat (g/1000 kcal)	27.7	32.1	34.7	37.0	40.1
Saturated fat (g/1000 kcal)	8.3	9.9	10.8	11.6	12.7
Fiber (g/1000 kcal)	13.8	11.7	10.9	10.3	9.5

Characteristics	Quintile red meat, g/1000 kcal				
	Q1	Q2	Q3	Q4	Q5
Vitamin supplement use 1 or more/month	72.2	68.4	66.1	63.7	58.8

* Generalized linear models used to estimate mean values for the continuous variables and frequencies for dichotomous proportions within each red meat intake quintile

‡ 12,597 (3.9%) men and 7,885 (3.5%) women have missing smoking history data.

Table 2

Multivariate HRs and CIs for red, white, and processed meat intake and total and cause specific mortality in men in the NIH-AARP Diet and Health Study

Men	Red meat quintiles ¹					P for trend
	Q1	Q2	Q3	Q4	Q5	
<u>All Mortality</u>						
Deaths	6437	7835	9366	10988	13350	
Basic model ⁴	1.00	1.07 (1.03-1.10)	1.17 (1.13-1.21)	1.27 (1.23-1.31)	1.48 (1.43-1.52)	P<0001
Adjusted model ⁵	1.00	1.06 (1.03-1.10)	1.14 (1.10-1.18)	1.21 (1.17-1.25)	1.31 (1.27-1.35)	P<0001
<u>Cancer Mortality</u>						
Deaths	2136	2701	3309	3839	4448	
Basic model ⁴	1.00	1.10 (1.04-1.17)	1.23 (1.16-1.29)	1.31 (1.24-1.39)	1.44 (1.37-1.52)	P<0001
Adjusted model ⁵	1.00	1.05 (0.99-1.11)	1.13 (1.07-1.20)	1.18 (1.12-1.25)	1.22 (1.16-1.29)	P<00010
<u>CVD Mortality</u>						
Deaths	1997	2304	2703	3256	3961	
Basic model ⁴	1.00	1.02 (0.96-1.08)	1.10 (1.04-1.17)	1.24 (1.17-1.31)	1.44 (1.37-1.52)	P<0001
Adjusted model ⁵	1.00	0.99 (0.96-1.09)	1.08 (1.02-1.15)	1.18 (1.12-1.26)	1.27 (1.20-1.35)	P<0001
<u>Accidental deaths</u>						
Deaths	184	216	228	280	343	
Basic model ⁴	1.00	1.02 (0.84-1.24)	0.97 (0.80-1.18)	1.09 (0.90-1.31)	1.24 (1.03-1.49)	P=0.01
Adjusted model ⁵	1.00	1.06 (0.86-1.29)	1.01 (0.83-1.24)	1.14 (0.94-1.39)	1.26 (1.04-1.54)	P=0.008
<u>All other deaths</u>						
Deaths	1268	1636	1971	2239	2962	
Basic model ⁴	1.00	1.13 (1.05-1.22)	1.25 (1.17-1.35)	1.33 (1.24-1.42)	1.68 (1.57-1.80)	P<0001
Adjusted model ⁵	1.00	1.17 (1.09-1.26)	1.28 (1.19-1.38)	1.34 (1.25-1.44)	1.58 (1.47-1.70)	P<0001
<u>White meat quintiles²</u>						
Men	Q1	Q2	Q3	Q4	Q5	P for trend
<u>All Mortality</u>						
Deaths	12521	10442	9359	8444	7210	
Basic model ⁴	1.00	0.83 (0.81-0.85)	0.77 (0.75-0.79)	0.74 (0.72-0.76)	0.74 (0.72-0.76)	P<0001

Men	Red meat quintiles ¹					P for trend
	Q1	Q2	Q3	Q4	Q5	
Adjusted model⁵	1.00	0.92 (0.90-0.95)	0.90 (0.88-0.93)	0.90 (0.88-0.93)	0.92 (0.89-0.94)	P<.0001
<u>Cancer Mortality</u>						
Deaths	4424	3647	3203	2830	2329	
Basic model⁴	1.00	0.82 (0.79-0.86)	0.74 (0.71-0.78)	0.71 (0.67-0.74)	0.68 (0.65-0.72)	P<.0001
Adjusted model⁵	1.00	0.91 (0.87-0.95)	0.87 (0.83-0.91)	0.85 (0.81-0.90)	0.84 (0.80-0.88)	P<.0001
<u>CVD Mortality</u>						
Deaths	3521	3015	2771	2578	2336	
Basic model⁴	1.00	0.85 (0.81-0.89)	0.81 (0.77-0.85)	0.81 (0.77-0.85)	0.86 (0.81-0.90)	P<.0001
Adjusted model⁵	1.00	0.96 (0.91-1.00)	0.96 (0.91-1.01)	0.99 (0.94-1.04)	1.05 (1.00-1.11)	P=0.009
<u>Accidental deaths</u>						
Deaths	333	266	249	219	184	
Basic model⁴	1.00	0.81 (0.69-0.95)	0.78 (0.66-0.93)	0.73 (0.62-0.87)	0.71 (0.59-0.85)	P=0.0003
Adjusted model⁵	1.00	0.89 (0.76-1.05)	0.90 (0.76-1.06)	0.86 (0.72-1.03)	0.85 (0.70-1.02)	P=0.11
<u>All other deaths</u>						
Deaths	2775	2206	1948	1722	1425	
Basic model⁴	1.00	0.79 (0.75-0.83)	0.72 (0.68-0.76)	0.68 (0.64-0.73)	0.67 (0.63-0.72)	P<.0001
Adjusted model⁵	1.00	0.90 (0.85-0.95)	0.88 (0.83-0.93)	0.86 (0.81-0.92)	0.86 (0.80-0.92)	P<.0001
Processed meat quintiles³						
Men	Q1	Q2	Q3	Q4	Q5	P for trend
Deaths	6235	7738	9435	11249	13319	
Basic model⁴	1.00	1.04 (1.01-1.08)	1.13 (1.09-1.16)	1.20 (1.16-1.24)	1.30 (1.26-1.34)	P<.0001
Adjusted model⁵	1.00	1.01 (0.98-1.04)	1.07 (1.04-1.11)	1.12 (1.08-1.16)	1.16 (1.12-1.20)	P<.0001
<u>Cancer Mortality</u>						
Deaths	2032	2784	3334	3906	4377	
Basic model⁴	1.00	1.15 (1.08-1.22)	1.22 (1.15-1.29)	1.28 (1.21-1.35)	1.32 (1.25-1.39)	P<.0001
Adjusted model⁵	1.00	1.07 (1.01-1.14)	1.11 (1.05-1.17)	1.14 (1.07-1.20)	1.12 (1.06-1.19)	P=0.001
<u>CVD Mortality</u>						
Deaths	1977	2225	2752	3255	4012	

Men	Red meat quintiles ¹					P for trend
	Q1	Q2	Q3	Q4	Q5	
Basic model⁴	1.00	0.94 (0.88-1.00)	1.02 (0.96-1.09)	1.08 (1.02-1.14)	1.22 (1.15-1.29)	P<0001
Adjusted model⁵	1.00	0.92 (0.87-0.98)	0.99 (0.93-1.05)	1.02 (0.96-1.08)	1.09 (1.03-1.15)	P=0.0001
<i>Accidental deaths</i>						
Deaths	190	201	257	273	330	
Basic model⁴	1.00	0.87 (0.72-1.07)	0.98 (0.81-1.19)	0.93 (0.77-1.13)	1.04 (0.86-1.25)	P=0.24
Adjusted model⁵	1.00	0.88 (0.72-1.08)	0.99 (0.81-1.20)	0.93 (0.76-1.13)	1.00 (0.83-1.21)	P=0.48
<i>All other deaths</i>						
Deaths	1259	1548	1896	2430	2943	
Basic model⁴	1.00	1.05 (0.97-1.13)	1.15 (1.07-1.23)	1.31 (1.22-1.41)	1.46 (1.36-1.56)	P<0001
Adjusted model⁵	1.00	1.05 (0.97-1.13)	1.14 (1.06-1.23)	1.28 (1.19-1.38)	1.33 (1.24-1.43)	P<0001

Total and cause specific mortality in men (n=322,263 deaths) among cohort of 545,653 in the NIH-AARP Diet and Health Study

¹Median red meat based on men and women (g/1000 kcal): 1st quintile 9.8; 2nd quintile 21.4; 3rd quintile 31.3; 4th quintile 42.8; 5th quintile 62.5.

²Median white meat based on men and women (g/1000 kcal): 1st quintile 18.4; 2nd quintile 18.4; 3rd quintile 27.4; 4th quintile 39.4; 5th quintile 64.6.

³Median processed meat based on men and women (g/1000 kcal): 1st quintile 1.6; 2nd quintile 4.4; 3rd quintile 7.4; 4th quintile 12.2; 5th quintile 22.6.

⁴Basic model: age (continuous); race (non-Hispanic white, non-Hispanic black, Hispanic/Asian/Pacific Islander/American Indian/Alaskan native or unknown); total energy intake (continuous).

⁵Adjusted model: Basic model plus education (less than 8 years or unknown, 8 to 11 years, 12 years (high school), some college, college graduate); marital status (married: yes/no); family history of cancer (yes/no) (cancer mortality only); body mass index (18.5-<25, 25-<30, 30-<35, ≥35 kg/m²); 31-level smoking history using smoking status (never, former, current), time since quitting for former smokers, and smoking dose, frequency of vigorous physical activity (never/rarely, 1-3 times/month, 1-2 times/week, 3-4 times/week, 5 or more times/week); alcohol intake (none, 0-<5, 5-<15, 15-<30, ≥30 servings/1000 kcal), vitamin supplement user (one or more supplement per month); fruit consumption (0-<0.7, 0.7-1.2, 1.2-<1.7, 1.7-<2.5, ≥2.5 servings/1000 kcal); vegetable consumption (0-<1.3, 1.3-<1.8, 1.8-<2.2, 2.2-<3.0, ≥3.0 serving/1000 kcal).

Multivariate HRs and CIs for red, white, and processed meat intake and total and cause specific mortality in women in the NIH-AARP Diet and Health Study

Table 3

	Red meat quintiles ¹					P for trend
	Q1	Q2	Q3	Q4	Q5	
Women						
<i>All Mortality</i>						
Deaths	5314	5081	4734	4395	3752	
Basic model ⁴	1.00	1.11 (1.07-1.16)	1.24 (1.20-1.29)	1.43 (1.38-1.49)	1.63 (1.56-1.70)	P<0001
Adjusted model ^{5,6}	1.00	1.08 (1.03-1.12)	1.17 (1.12-1.22)	1.28 (1.23-1.34)	1.36 (1.30-1.43)	P<0001
<i>Cancer Mortality</i>						
Deaths	2134	1976	1784	1687	1348	
Basic model ⁴	1.00	1.07 (1.01-1.14)	1.15 (1.08-1.23)	1.34 (1.26-1.43)	1.42 (1.33-1.52)	P<0001
Adjusted model ^{5,6}	1.00	1.02 (0.96-1.09)	1.06 (1.00-1.14)	1.20 (1.12-1.28)	1.20 (1.12-1.30)	P<0001
<i>CVD Mortality</i>						
Deaths	1173	1155	1101	1027	900	
Basic model ⁴	1.00	1.15 (1.06-1.25)	1.32 (1.22-1.44)	1.54 (1.41-1.68)	1.82 (1.66-1.98)	P<0001
Adjusted model ^{5,6}	1.00	1.13 (1.04-1.23)	1.26 (1.16-1.37)	1.39 (1.27-1.52)	1.50 (1.37-1.65)	P<0001
<i>Accidental deaths</i>						
Deaths	129	97	74	76	61	
Basic model ⁴	1.00	0.86 (0.66-1.12)	0.77 (0.58-1.03)	0.96 (0.72-1.28)	1.01 (0.74-1.37)	P=0.88
Adjusted model ^{5,6}	1.00	0.85 (0.65-1.12)	0.75 (0.56-1.02)	0.92 (0.68-1.25)	0.94 (0.68-1.31)	P=0.88
<i>All other deaths</i>						
Deaths	1178	1187	1181	1058	961	
Basic model ⁴	1.00	1.18 (1.09-1.28)	1.41 (1.30-1.53)	1.58 (1.45-1.72)	1.91 (1.76-2.09)	P<0001
Adjusted model ^{5,6}	1.00	1.16 (1.07-1.26)	1.35 (1.24-1.47)	1.44 (1.32-1.57)	1.61 (1.46-1.76)	P<0001
White meat quintiles²						
Deaths	5006	4606	4469	4520	4675	
Basic model ⁴	1.00	0.87 (0.84-0.91)	0.81 (0.78-0.84)	0.78 (0.75-0.81)	0.76 (0.73-0.79)	P<0001

Women	Red meat quintiles ¹					P for trend
	Q1	Q2	Q3	Q4	Q5	
Deaths	1245	1132	1039	973	967	
Basic model⁴	1.00	1.13 (1.04-1.22)	1.25 (1.14-1.35)	1.41 (1.29-1.54)	1.69 (1.55-1.84)	P<0001
Adjusted model^{5,6}	1.00	1.08 (0.99-1.17)	1.15 (1.05-1.25)	1.24 (1.13-1.35)	1.38 (1.26-1.51)	P<0001
<u>Accidental deaths</u>						
Deaths	118	115	71	71	62	
Basic model⁴	1.00	1.22 (0.94-1.59)	0.91 (0.67-1.23)	1.10 (0.82-1.50)	1.18 (0.86-1.62)	P=0.52
Adjusted model^{5,6}	1.00	1.21 (0.93-1.57)	0.89 (0.65-1.21)	1.06 (0.78-1.45)	1.10 (0.80-1.53)	P=0.83
<u>All other deaths</u>						
Deaths	1265	1174	1101	1055	970	
Basic model⁴	1.00	1.16 (1.07-1.26)	1.32 (1.22-1.44)	1.54 (1.42-1.68)	1.72 (1.58-1.87)	P<0001
Adjusted model^{5,6}	1.00	1.11 (1.02-1.20)	1.22 (1.12-1.32)	1.35 (1.24-1.47)	1.39 (1.27-1.51)	P<0001

Total and cause specific mortality in women (n=223,390 deaths) among cohort of 545,653 in the NIH-AARP Diet and Health Study

- ¹Median red meat based on men and women (g/1000 kcal): 1st quintile 9.8; 2nd quintile 21.4; 3rd quintile 31.3; 4th quintile 42.8; 5th quintile 62.5.
- ²Median white meat based on men and women (g/1000 kcal): 1st quintile 9.5; 2nd quintile 18.4; 3rd quintile 27.4; 4th quintile 39.4; 5th quintile 64.6.
- ³Median processed meat based on men and women (g/1000 kcal): 1st quintile 1.6; 2nd quintile 4.4; 3rd quintile 7.4; 4th quintile 12.2; 5th quintile 22.6.

⁴Basic model: age (continuous); race (non-Hispanic white, non-Hispanic black, Hispanic/Asian/Pacific Islander/American Indian/Alaskan native or unknown); total energy intake (continuous).

⁵Adjusted model: Basic model plus education (less than 8 years or unknown, 8 to 11 years, 12 years (high school), some college, college graduate); marital status (married: yes/no); family history of cancer (yes/no) (cancer mortality only); body mass index (18.5-<25, 25-<30, 30-<35, ≥35 kg/m²); 31-level smoking history using smoking status (never, former, current), time since quitting for former smokers, and smoking dose, frequency of vigorous physical activity (never/rarely, 1-3 times/month, 1-2 times/week, 3-4 times/week, 5 or more times/week); alcohol intake (none, 0-<5, 5-<15, 15-<30, ≥30 servings/1000 kcal), vitamin supplement user (one or more supplement per month); fruit consumption (0-<0.7, 0.7-<1.2, 1.2-<1.7, 1.7-<2.5, ≥2.5 servings/1000 kcal); vegetable consumption (0-<1.3, 1.3-<1.8, 1.8-<2.2, 2.2-<3.0, ≥3.0 serving/1000 kcal).

⁶Hormone replacement therapy included in models for women.