Cancer Causes and Control **11**: 419–431, 2000.

Diet and lung cancer mortality: a 1987 National Health Interview Survey cohort study

Rosalind A. Breslow[1,*], Barry I. Graubard[2], Rashmi Sinha[3] & Amy F. Subar[4]
[1]*Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Division of Cancer Prevention and Control, Epidemiology and Health Services Research Branch, 4770 Buford Highway, MS K-55, Atlanta, GA, 30341, USA;* [2]*Division of Cancer Epidemiology and Genetics, Biostatistics Branch, National Cancer Institute, Bethesda, MD, USA;* [3]*Division of Cancer Epidemiology and Genetics, Nutritional Epidemiology Branch, National Cancer Institute, Bethesda, MD, 20892, USA;* [4]*National Cancer Institute, Division of Cancer Control and Population Sciences, Applied Research Branch, Bethesda, MD, 20892, USA (*Author for correspondence)*

Received 23 July 1999; accepted in revised form 3 January 2000

Key words: diet, lung cancer, mortality, National Health Interview Survey.

Abstract

Objectives: To study the association between diet and lung cancer mortality in the United States.
Methods: Records from 20,195 participants with usable dietary data in the 1987 National Health Interview Survey were linked to the National Death Index. Baseline diet was assessed with a 59-item food-frequency questionnaire. Food groups (fruits, vegetables, total meat/poultry/fish, red meats, processed meats, dairy products, breakfast cereals, other starches, added fats, and alcohol) were analyzed in cause-specific Cox proportional hazard regression models adjusted for age, gender and smoking.
Results: There were 158 deaths from lung cancer (median follow-up 8.5 years). Frequencies of meat/poultry/fish intake (relative risk [RR] (highest compared to lowest quartile) = 2.0; 95% confidence interval [CI] 1.2–3.5, p for trend [p] < 0.027), and red meat intake (RR = 1.6; CI 1.0–2.6, $p < 0.014$), were positively and significantly associated with lung cancer mortality. Specifically, the red meats, including pork (RR = 1.6; CI 1.0–2.7, $p < 0.028$), and ground beef (RR = 2.0; CI 1.1–3.5, $p < 0.096$) were associated with increased risk, although for ground beef the trend was not significant. Dairy products (RR = 0.5; CI 0.3–0.8, $p < 0.009$) were inversely associated with lung cancer mortality. There was no statistically significant association between intake of fruits and vegetables and lung cancer mortality.
Conclusions: In this nationally representative study, intake of red meats was positively associated with lung cancer mortality while intake of dairy products was inversely associated. While smoking is the major risk for lung cancer mortality, diet may have a contributory role.

Introduction

Lung cancer is the most common cause of cancer mortality in the United States [1]. While smoking is the undisputed primary risk factor [2], numerous studies suggest that diet may be etiologically important [3, 4].

The association between foods and lung cancer has been studied extensively for some foods and less extensively for others [4]. There are over 200 studies on the association between fruits, vegetables and lung cancer [4, 5]. There are considerably fewer studies on meats, fish, poultry, dairy products [4, 6–13], grains [8], and alcohol [4, 11, 14–19]. However, associations between these foods and lung cancer, both positive and inverse, are biologically plausible. For example, heterocyclic amines [20] found in cooked red meats [21–25] may increase risk.

The majority of studies on diet and lung cancer have used the case–control method; yet cohort studies are less subject to recall bias, which may be important in studies of the association [26]. Historically, few cohorts in the United States, and only one of them nationally representative [27], have provided data on diet and cancer. Thus, new cohorts created by linkage of the National Health Interview Surveys (NHIS) to the National Death Index (NDI) by the National Center for Health Statistics (NCHS), provide valuable opportunities for epidemiologists to study associations between risk factors (including diet) and cause-specific mortality (including cancer). The purpose of this study was to prospectively examine the association between diet and lung cancer mortality in a nationally representative cohort of 20,195 individuals who satisfactorily completed the 59-item food-frequency questionnaire in the 1987 NHIS.

Material and methods

Population

The National Health Interview Survey (NHIS), conducted by NCHS, is a continuing annual, cross-sectional, nationally representative, probability survey of the health of the United States civilian, non-institutionalized population [28]. Door-to-door, in-home interviews of non-institutionalized individuals are conducted by experienced Bureau of the Census interviewers.

The 1987 NHIS included a core questionnaire completed by 47,240 households containing 122,859 persons, and a Cancer Risk Factor Supplement (CRFS) developed and funded by the National Cancer Institute [29]. The CRFS consisted of two Supplements, Cancer Control (n = 22,043) and Cancer Epidemiology (n = 22,080). One adult, age 18 years or older, from each household completing the core questionnaire was randomly selected to receive either the Cancer Control or Cancer Epidemiology Supplement. Our study used data from the Cancer Epidemiology Supplement which included a food-frequency questionnaire (FFQ). The response rate for the core questionnaire was 95%. The response rate for the Cancer Epidemiology Supplement was 86.1%. Hispanics were over-sampled; Spanish translations were available.

Mortality link

Starting with the 1986 NHIS, each subsequent NHIS has been (and will continue to be) linked to the NDI. The data files are available on CD-ROMs (*National Health Interview Survey Multiple Cause-of-Death Public Use Data File*, June 1997). The NHIS-NDI linkage enables investigators to perform cohort studies having cause-specific mortality endpoints [30, 31]. Briefly, NHIS participants are matched to individuals reported as deceased in the NDI based on criteria including social security number and first name, social security number and last name, social security number and father's surname, or other combinations of birth date, name and initials. At the time of this study, records from the 1987 NHIS were matched to the NDI through 31 December 1995.

Dietary data

The 1987 NHIS Cancer Epidemiology Supplement included a 59-item food-frequency questionnaire (FFQ), developed by Block *et al.* [32], which queried participants about the number of times per day, week, month or year they consumed certain foods during the past year. The 59 items were selected based on contribution to nutrient intake in the US population, determined from 24-hour recall data collected in the second National Health and Nutrition Examination Survey (NHANES II, 1976–1980) [33, 34]. The 59-item FFQ was based on a longer 98-item FFQ validated in numerous subpopulations varying by gender, age and income [35–39]. Correlations between nutrients and multiple days of diet records were similar for both instruments [40].

Foods in the 1987 NHIS FFQ were listed as line-items (subsequently referred to as food-items). A food-item consisted of a single food (for example, oranges), or of several foods of similar nutrient content or usage (for example, hamburgers, cheeseburgers and meatloaf). We formed nine food groups (fruits, vegetables, meat/poultry/fish [and the subgroups red meats and processed meats], dairy products, breakfast cereals, starches, added fats, desserts, and alcohol) (see Appendix).

Data on food group and food-item intake were categorized (by quartiles of weekly intake frequency) to avoid assumptions about the parametric form of the dose–response relationships and to reduce potential influence by outlying dietary intake observations [41]. Food-items with high numbers of non-consumers (milks and cereals) were categorized into three levels; non-consumers, and consumers above and below their sample weighted median intake.

Analytic cohort

Of the 22,080 participants who completed the 1987 Cancer Epidemiology Supplement, 1494 were excluded from analyses because of coding, interviewer or participant errors on the FFQ, and 391 were excluded because of inadequate information to perform a mortality match with the NDI; thus, 20,195 participants had usable dietary data. Of these, 187 died of lung cancer as the underlying cause, between baseline (the quarter of the year 1987 during which the participant was interviewed) and 31 December 1995, (the latest NDI match date available at the time of this study). Thus, there were 187 cases and 20,008 non-cases.

To form the analytic cohort we excluded all participants dying within 1 year of baseline or who reported a diagnosis of lung cancer at baseline, as disease might bias dietary recall. Thus, an additional 29 cases (23 died within 1 year of baseline; six were alive 1 year after baseline but had stated a diagnosis of lung cancer at baseline), and 162 non-cases (140 died within 1 year of their baseline interview; 22 were alive at 1 year but had stated a diagnosis of lung cancer at baseline) were excluded, resulting in an analytic cohort of 20,004 individuals (158 cases and 19,846 non-cases; median follow-up = 8.5 years).

Statistical analysis

Covariates considered in analyses included the potential confounders of age at baseline interview (continuous), gender, and ever smoking as duration in years and packs per day (both continuous). Additional covariates included race (white non-Hispanic and other, black non-Hispanic, Hispanic), family income (< \$20,000 and ≥\$20,000 per year), region of the country (Northeast, Midwest, South, West) occupation (white-collar, blue-collar, unemployed), marital status (single, married), body mass index (continuous), and the number of days the respondent participated in social activities during the past year (continuous).

Sample weighted distributions of covariates within quartiles of food groups were determined. Analyses for sample weighted quartiles of intake were performed using Cox proportional hazard regression analysis to estimate relative hazards (henceforth referred to as relative risks) of lung cancer mortality. The response variable in the Cox models was time to lung cancer mortality from baseline [42]. Participants who died during follow-up, but not of lung cancer, were censored at time of death. Overall, there were 1852 deaths in the 20,195 participants between baseline and 31 December 1995. Participants who survived through follow-up were censored on 31 December 1995.

Analyses were performed for food groups using two models: model 1 included age; model 2 included age, gender, and smoking (packs per day and duration). Both models were run with and without the covariates race, family income, region of the country, occupation, marital status, body mass index, and social activities. If, in any model, there was a significant trend ($p < 0.05$) over quartiles of food group intake, further analyses were performed for food-items within that group. Analyses using models 1 and 2 were also performed individually for the food-items not included in food groups (see Appendix). Final models were performed with and without adjustment for total caloric intake (continuous). Tests of trend were performed by assigning a score to each quartile of intake (the sample weighted median intake within each quartile), including that score in a Cox regression model as an independent variable, and performing *t*-tests of the regression coefficients associated with the score to determine the level of significance of the trend. Restricted cubic regression splines were used to check for non-linearity in dose–response for food groups and food-items that had significant linear trends [43]; for spline analyses, continuous dietary variables were used.

Analyses were performed using SAS, version 6.12 (SAS Institute, Cary, NC) and SUDAAN v.7.5 (Research Triangle Institute, Research Triangle Park, NC) a statistical software package that allows inclusion of the complex sampling design of the NHIS in the analyses of statistical models, thus facilitating computation of design-based estimates of relative risks and their standard errors. Unless otherwise indicated, all analyses were weighted to the US population using sample weights from the 1987 NHIS. All tests of significance were two-tailed with the level of significance set at $p < 0.05$.

Results

The baseline, unweighted characteristics of the analytic cohort were determined. The median age of the analytic cohort was 40 years (range 18–87 years). Eighty-five percent of the participants were age 64 years or younger. There were 8363 men (41.8%) and 11,641 women (58.2%). Approximately 83% of the participants were non-Hispanic whites, 10% were non-Hispanic blacks, and 7% were Hispanic. Thirty-nine percent had more than a high school education. Approximately 47% were never smokers, 21% were former smokers, and 29% were current smokers.

Of the 158 cases, 90 were men and 68 were women. The mean duration of smoking in the male cases was 36 years; in the female cases mean duration was 25 years. In the non-cases duration was considerably less; 13 years for men and 9 years for women. The weighted mean age at mortality from lung cancer in the cases was 65.7 years (SD 13.4). In the US population in 1991 (midpoint between 1987 and 1995), mean age at mortality from lung cancer was 68.6 years (SD 10.7) (US Vital Statistics). Differences could be due to the fact that the

NHIS is conducted in a non-institutionalized population.

Table 1 describes the demographics of the cohort by quartiles of food group intake frequency. Intake of fruits, vegetables, cereals, and added fats increased with age, while intake of alcohol decreased. Relative to women, men more frequently consumed meat, dairy products, starches, and alcohol, and less frequently consumed fruits, vegetables, and breakfast cereals. Relative to participants who never smoked, ever-smokers more frequently consumed added fats and alcohol, and less frequently consumed fruits and breakfast cereals. Duration of smoking was longest for the most frequent consumers of added fats and alcohol.

Table 2 shows relative risks of lung cancer mortality based on weekly frequency of food group intake. Comparisons discussed are for the highest (Q4) *vs.* lowest (Q1) quartiles of intake. In models adjusted for age, gender, and smoking, the most frequent consumers of meat/poultry/fish had more than double the risk for lung cancer mortality of the least frequent consumers (p for trend < 0.027). The most frequent consumers of red meats had an approximate two-thirds increase in risk (p for trend < 0.014). In contrast, the most frequent consumers of dairy products had about half the risk (p for trend < 0.009). Consumers in the highest quartile of breakfast cereals intake had significantly reduced risk for lung cancer mortality compared to those in the lowest quartile, but the trend was not significant (p for trend < 0.094). There were no significant associations between other food groups and lung cancer mortality (including a dark green, yellow, and orange vegetables group that included sweet potatoes, or adding white potatoes to the vegetable group, both analyses run in response to a reviewer's query). Inclusion of the covariates race, family income, region of the country, occupation, marital status, body mass index, and social activities, or inclusion of only occupation and income, did not substantially alter results. Inclusion of calories as a continuous variable in models did not substantially alter results.

Table 3 presents the relative risks of lung cancer mortality for food-items within food groups that had significant trends. The most frequent consumers of pork had almost a two-thirds significant increase in risk (p for trend < 0.028). Consumers in the highest quartile of ground beef had almost a doubling of risk but the trend over quartiles was not significant (p for trend < 0.096). There were no significant findings for any other food-items within the meat/poultry/fish or dairy products groups or for food-items not included in food-groups (see Appendix). Inclusion of the covariates race, family income, region of the country, occupation, marital status, body mass index, and social activities, or inclusion of only occupation and income, did not substantially alter results. Inclusion of calories as a continuous variable in models did not substantially alter results.

Table 1. Demographics of the 1987 National Health Interview Survey cohort (n = 20,004) by frequency of food group intake

	Quartiles (frequency of food group intake)[a]			
	Q1	Q2	Q3	Q4
Fruits				
Mean age (years)	38.5	40.8	44.1	47.8
Percentage male	53.9	50.0	45.6	41.1
Percentage ever smoked	61.2	51.3	47.8	42.9
Mean duration smoked (years)[b]	12.1	10.5	10.2	9.7
Vegetables				
Mean age (years)	40.0	42.5	43.7	45.2
Percentage male	50.1	49.1	47.0	44.5
Percentage ever smoked	52.9	50.5	50.6	49.2
Mean duration smoked (years)	10.6	10.7	10.7	10.6
Meat/poultry/fish				
Mean age (years)	47.5	44.5	41.8	38.2
Percentage male	38.9	44.1	48.7	57.8
Percentage ever smoked	49.5	51.2	51.6	50.9
Mean duration smoked (years)	11.5	11.2	10.4	9.6
Dairy products				
Mean age (years)	47.6	42.9	42.8	38.7
Percentage male	44.4	46.6	48.2	51.2
Percentage ever smoked	51.7	50.8	50.5	50.0
Mean duration smoked (years)	12.4	10.6	10.4	9.4
Breakfast cereals				
Mean age (years)	40.9	40.3	43.1	47.4
Percentage male	52.8	47.2	46.3	44.6
Percentage ever smoked	58.5	50.8	49.1	44.7
Mean duration smoked (years)	12.3	10.1	10.1	10.1
Other starches				
Mean age (years)	41.4	44.5	41.9	43.7
Percentage male	42.2	48.4	47.2	52.8
Percentage ever smoked	48.9	52.7	49.9	51.6
Mean duration smoked (years)	9.6	11.4	10.1	11.5
Added fats				
Mean age (years)	40.9	43.1	42.9	44.5
Percentage male	50.0	48.1	45.7	47.4
Percentage ever smoked	46.5	49.9	51.3	55.1
Mean duration smoked (years)	9.1	10.5	10.7	12.2
Alcohol				
Mean age (years)	48.7	42.6	39.5	39.8
Percentage male	37.1	34.2	47.4	70.2
Percentage ever smoked	39.9	46.9	52.0	64.5
Mean duration smoked (years)	9.6	10.0	9.7	13.2

[a] All p values for trend across quartiles were significant at $p < 0.006$ except for percentage ever smoked (vegetables, $p < 0.02$; meat/poultry/fish, $p < 0.29$, dairy products, $p < 0.56$) and mean duration smoked (vegetables $p < 0.98$).

[b] Duration ever smoked in total cohort.

Table 2. Relative risks (RR) and 95% confidence intervals (95% CI) for lung cancer mortality in the 1987 National Health Interview Survey cohort by frequency of food group intake[a]

	Quartiles of intake				p for trend[b]
	1	2	3	4	
Fruits					
Servings/week[c]	0–3.0	3.0–7.2	7.2–11.5	>11.5	
Median[d]	1.3	4.9	9.0	16.0	
No. of cases[e]	35	46	31	42	
Person-years	39,880	41,087	39,865	39,893	
RR	1.0	1.1	0.6	0.6	<0.030
MV RR (95% CI)	1.0 (referent)	1.2 (0.7–1.9)	0.8 (0.4–1.4)	0.9 (0.5–1.6)	<0.489
Vegetables					
Servings/week	0–5.2	5.2–8.9	8.9–13.6	>13.6	
Median	3.2	7.0	11.0	17.5	
No. of cases	36	38	49	35	
Person-years	40,478	41,474	41,078	41,088	
RR	1.0	1.0	1.2	0.8	<0.321
MV RR (95% CI)	1.0 (referent)	1.0 (0.6–1.8)	1.3 (0.8–2.0)	0.9 (0.5–1.5)	<0.786
Meat/poultry/fish					
Servings/week	0–3.7	3.7–5.5	5.5–7.6	>7.6	
Median	2.8	4.6	6.4	9.6	
No. of cases	35	43	38	42	
Person-years	39,894	40,821	40,147	40,770	
RR	1.0	1.6	1.5	2.4	<0.004
MV RR (95% CI)	1.0 (referent)	1.5 (0.8–2.5)	1.3 (0.8–2.0)	2.0 (1.2–3.5)	<0.027
Red meats					
Servings/week	0–2.3	2.3–4.2	4.2–6.6	>6.6	
Median	1.4	3.2	5.2	9.0	
No. of cases	39	29	44	46	
Person-years	40,580	41,661	40,690	41,228	
RR	1.0	0.9	2.1	2.4	<0.001
MV RR (95% CI)	1.0 (referent)	0.7 (0.4–1.2)	1.5 (0.9–2.4)	1.6 (1.0–2.6)	<0.014
Processed meats					
Servings/week	0–0.5	0.5–1.2	1.2–2.9	>3.0	
Median	0	0.7	2.0	4.5	
No. of cases	54	36	34	34	
Person-years	40,323	40,886	38,674	44,260	
RR	1.0	1.0	1.3	1.2	<0.405
MV RR (95% CI)	1.0 (referent)	0.8 (0.5–1.3)	1.0 (0.6–1.6)	0.8 (0.5–1.4)	<0.721
Dairy products					
Servings/week	0–3.0	3.0–7.0	7.0–10.0	>10.0	
Median	1.0	5.0	9.0	15.0	
No. of cases	51	50	33	22	
Person-years	39,138	46,437	36,161	40,562	
RR	1.0	0.9	0.7	0.5	<0.006
MV RR (95% CI)	1.0 (referent)	0.9 (0.5–1.4)	0.7 (0.4–1.2)	0.5 (0.3–0.8)	<0.009
Breakfast cereals					
Servings/week	0–0.5	0.5–2.5	2.5–6.0	>6.0	
Median	0	1.2	4.0	7.0	
No. of cases	49	32	35	41	
Person-years	40,022	41,967	39,257	41,312	
RR	1.0	0.5	0.5	0.4	<0.013
MV RR (95% CI)	1.0 (referent)	0.5 (0.3–0.9)	0.6 (0.4–1.1)	0.5 (0.3–0.9)	<0.094

Table 2. (Continued)

	Quartiles of intake				p for trend[b]
	1	2	3	4	
Other starches					
Servings/week	0–7.0	7.0–9.0	9.0–13.2	>13.3	
Median	4.7	8.0	10.5	16.7	
No. of cases	31	32	47	48	
Person-years	41,173	41,619	40,792	40,567	
RR	1.0	0.7	1.4	1.1	<0.344
MV RR (95% CI)	1.0 (referent)	0.6 (0.4–1.1)	1.2 (0.7–2.0)	0.9 (0.5–1.5)	<0.943
Added fats					
Servings/week	0–5.4	5.5–9.0	9.0–14.0	>14.0	
Median	3.0	7.0	10.0	16.0	
No. of cases	31	38	36	52	
Person-years	40,763	39,894	39,447	42,152	
RR	1.0	1.1	1.1	1.3	<0.381
MV RR (95% CI)	1.0 (referent)	1.1 (0.6–1.9)	1.0 (0.5–2.1)	1.2 (0.7–2.0)	<0.603
Alcohol					
Servings/week	0	0.02–0.5	0.5–4.4	>4.4	
Median	0	0.2	1.9	10.5	
No. of cases	52	23	32	50	
Person-years	47,779	31,894	41,061	41,026	
RR	1.0	0.9	1.2	2.1	<0.0001
MV RR (95% CI)	1.0 (referent)	0.7 (0.4–1.3)	1.0 (0.6–1.6)	1.3 (0.8–2.0)	<0.101

[a] RR values are adjusted for age; multivariate RR (MV RR) are adjusted for age, gender, smoking duration (years), and packs per day smoked.
[b] p-Values for trend were obtained using weighted sample medians of quartiles as quartile scores.
[c] Ranges of intake are for multivariate models. Ranges for models adjusted for age only are similar. The overlap in ranges for quartiles is due to rounding.
[d] Sample weighted median intake within quartiles for multivariate models. Median values for models adjusted for age only are similar.
[e] Missing dietary data explains numbers fewer than 158 cases.

Smoking duration was a significant variable in all models for food groups and food-items. The non-age-adjusted relative risk of ever smoking for a duration of 40 years was 12.9 in this relatively young cohort with a high proportion of women; the age-adjusted relative risk of ever smoking was approximately 4.7. The number of packs smoked per day was not significant in any models. There were no significant interactions between smoking duration and food groups except alcohol ($p < 0.026$). Among non-smokers the relative risk of lung cancer mortality in the highest compared to the lowest quartile of alcohol intake was 2.3 (CI 1.1–4.6). Among those who smoked for 25 years the RR in the highest *vs.* lowest quartile of alcohol intake was 1.4 (CI 0.9–2.1), and among those who smoked for 50 years the relative risk was 0.9 (CI 0.5–1.2). Restricted cubic regression splines were not significant for any of the food groups or foods significantly associated with lung cancer mortality, indicating no statistical evidence of a non-linear dose–response relationship in the log relative risks. Although our power to perform analyses by gender was low due to small numbers of cases, we performed separate analyses of significant food groups and fruits and vegetables and tested for interactions between gender and these food groups. None of the interactions was significant at $p < 0.05$.

Discussion

In this nationally representative cohort, frequent intake of the meat/poultry/fish food group, specifically intake of red meats (pork, and to a lesser extent, ground beef), was positively associated with lung cancer mortality. Frequent intake of dairy products was inversely associated with risk.

A family of pyrolysis products is produced in red meats cooked at high temperature [20–25]. These highly mutagenic compounds initiate lung cancer in animals [20]. We did not have details on meat cooking practices in our cohort. However, it is known that red meats, in particular beef and pork, are frequently cooked at high temperatures to high levels of doneness. Data from Sinha *et al.* [23, 24] and Byrne *et al.* [25] suggest that beef and

Table 3. Relative risks (RR) and 95% confidence intervals (95% CI) for lung cancer mortality in the 1987 National Health Interview Survey cohort by frequency of selected food-item intake[a]

	Quartiles of intake				*p* for trend[b]
	1	2	3	4	
Meat/poultry/fish food group					
Fried fish or fish sandwiches					
Servings/week[c]	0	0.02–0.2	0.2–1.0	>1.0	
Median[d]	0	0.2	0.5	1.0	
No. of cases[e]	55	31	21	51	
Person-years	50,126	40,127	28,460	45,122	
RR	1.0	0.8	0.9	1.1	<0.441
MV RR (95% CI)	1.0 (referent)	0.7 (0.4–1.2)	0.8 (0.5–1.4)	1.0 (0.6–1.5)	<0.758
Ground beef (hamburgers, cheeseburgers, or meatloaf)					
Servings/week	0–0.5	0.5–1.0	1.2–2.0	>2.1	
Median	0.2	1.0	2.0	3.0	
No. of cases	55	56	21	26	
Person-years	48,416	53,609	31,542	30,210	
RR	1.0	1.5	1.0	2.3	<0.021
MV RR (95% CI)	1.0 (referent)	1.4 (0.9–2.3)	1.0 (0.6–1.8)	2.0 (1.1–3.5)	<0.096
Beef, such as steaks and roast					
Servings/week	0–0.5	0.5–1.0	1.0–1.9	>2.0	
Median	0.1	0.5	1.0	2.0	
No. of cases	34	23	49	52	
Person-years	37,277	28,050	50,096	48,456	
RR	1.0	1.0	1.4	1.8	<0.010
MV RR (95% CI)	1.0 (referent)	0.9 (0.4–1.8)	1.1 (0.7–1.7)	1.3 (0.8–2.1)	<0.122
Pork, such as pork chops or roasts					
Servings/week	0–0.02	0.04–0.2	0.2–1.0	>1.0	
Median	0	0.2	0.5	1.0	
No. of cases	40	34	35	49	
Person-years	39,797	42,649	35,983	45,320	
RR	1.0	0.9	1.4	2.0	<0.003
MV RR (95% CI)	1.0 (referent)	0.9 (0.5–1.5)	1.3 (0.8–2.2)	1.6 (1.0–2.7)	<0.028
Fried chicken					
Servings/week	0	0.02–0.4	0.5–1.0	>1.0	
Median	0	0.2	0.5	1.0	
No. of cases	45	24	30	59	
Person-years	41,389	33,560	31,370	57,572	
RR	1.0	0.8	1.5	1.5	<0.032
MV RR (95% CI)	1.0 (referent)	0.75 (0.4–1.3)	1.1 (0.6–1.8)	1.2 (0.8–1.8)	<0.203
Chicken or turkey, baked or broiled					
Servings/week	0–0.2	0.2–0.5	0.5–1.0	>1.1	
Median	0	0.2	1.0	2.0	
No. of cases	41	54	31	32	
Person-years	40,710	41,283	44,269	37,484	
RR	1.0	1.3	0.8	0.8	<0.165
MV RR (95% CI)	1.0 (referent)	1.5 (0.9–2.3)	0.9 (0.6–1.5)	1.1 (0.6–2.0)	<0.653
Bacon					
Servings/week	0	0.02–0.5	0.5–1.0	>1.1	
Median	0	0.2	1.0	2.3	
No. of cases	52	21	40	45	
Person-years	47,357	27,523	51,462	36,910	
RR	1.0	0.9	1.1	1.5	<0.041
MV RR (95% CI)	1.0 (referent)	0.8 (0.5–1.5)	0.9 (0.5–1.5)	1.2 (0.7–1.9)	<0.396
Sausage					
Servings/week	0	0.02–0.2	0.2–1.0	>1.0	
Median	0	0.1	0.2	1.0	
No. of cases	69	8	32	47	

Table 3. (Continued)

	Quartiles of intake				*p* for trend[b]
	1	2	3	4	
Person-years	63,957	11,360	46,649	41,291	
RR	1.0	0.6	0.8	1.3	<0.092
MV RR (95% CI)	1.0 (referent)	0.6 (0.3–1.4)	0.7 (0.4–1.2)	1.0 (0.6–1.6)	<0.604
Hot dogs					
Servings/week	0	0.02–0.2	0.2–0.7	>0.7	
Median	0	0.1	0.2	1.0	
No. of cases	76	9	42	31	
Person-years	52,885	15,096	52,317	43,412	
RR	1.0	0.6	0.8	1.0	<0.777
MV RR (95% CI)	1.0 (referent)	0.5 (0.3–1.1)	0.7 (0.5–1.1)	0.8 (0.5–1.4)	<0.730
Ham or lunch meats					
Servings/week	0–0.1	0.1–1.0	1.0–2.0	>2.1	
Median	0	0.5	1.0	4.0	
No. of cases	51	34	43	29	
Person-years	39,870	41,159	47,934	34,795	
RR	1.0	1.0	1.2	1.2	<0.393
MV RR (95% CI)	1.0 (referent)	0.9 (0.5–1.4)	0.9 (0.6–1.6)	0.9 (0.5–1.6)	<0.977
Dairy products food group					
Cheese or cheese spreads					
Servings/week	0–0.5	0.5–1.9	2.0–3.0	>3.0	
Median	0	1.0	2.0	7.0	
No. of cases	57	25	50	26	
Person-years	43,497	30,085	49,510	40,815	
RR	1.0	0.7	0.9	0.6	<0.105
MV RR (95% CI)	1.0 (referent)	0.6 (0.4–1.1)	0.8 (0.5–1.4)	0.6 (0.3–1.1)	<0.124
Whole milk, not including on cereal[f]					
Servings/week	0	0.02–7	>8.0		
Median	0	4.0	14.0		
No. of cases	102	49	6		
Person-years	101,524	52,427	9602		
RR	1.0	1.4	1.0		<0.394
MV RR (95% CI)	1.0 (referent)	1.1 (0.7–1.6)	0.7 (0.3–1.7)		<0.603
2% milk, not including on cereal[f]					
Servings/week	0	0.02–7.0	>8.0		
Median	0	4.0	14.0		
No. of cases	117	36	5		
Person-years	107,571	46,702	9330		
RR	1.0	0.9	0.5		<0.101
MV RR (95% CI)	1.0 (referent)	0.9 (0.6–1.4)	0.5 (0.2–1.4)		<0.169
Skim milk, 1% milk, or buttermilk, not including on cereal[f]					
Servings/week	0	0.02–5.0	>5.6		
Median	0	1.0	7.0		
No. of cases	132	14	11		
Person-years	134,336	14,919	14,238		
RR	1.0	1.0	0.6		<0.132
MV RR (95% CI)	1.0 (referent)	1.1 (0.6–2.0)	0.8 (0.4–1.6)		<0.500

[a] RR values are adjusted for age; multivariate RR (MV RR) are adjusted for age, gender, smoking duration (years), and packs per day smoked.

[b] *p*-Values for trend were obtained using weighted sample medians of quartiles as quartile scores.

[c] Ranges of intake are for multivariate models. Ranges for models adjusted for age only are similar.

[d] Sample weighted median intake within quartiles of multivariate models. Median values for models adjusted for age only are similar.

[e] Missing dietary data explains numbers fewer than 158 cases.

[f] Due to large numbers of non-consumers, a referent category of non-consumers was formed with consumers divided into two groups at the sample weighted median value of intake.

pork, cooked well-done and/or at high temperatures, are major sources of heterocyclic amines in the US diet [21, 22]. Our findings of associations between pork, ground beef intake and lung cancer mortality are thus consistent with literature on heterocyclic amines. Our null findings for the steaks and roasts food-items may be due to the grouping of these two types of meats, generally cooked by very different techniques, into a single food-item. Roasts are generally cooked at moderate temperatures by roasting and generally contain non-detectable levels of heterocylic amines; on the other hand, steaks may be cooked by techniques that can produce high levels of these mutagens [24].

Alternatively, dietary fat or cholesterol content might explain an effect of red meat. *In-vivo* studies demonstrate that dietary fat promotes lung carcinogenesis [4, 44]. However, epidemiologic evidence for these associations is inconsistent [4, 9, 10, 13, 45–49], perhaps due to confounding by energy intake. Our food-based study did not attempt to disentangle the effects of fat and energy. However, we found no association between high fat or high cholesterol foods and lung cancer mortality in this study. Inclusion of total calories, poorly captured in a 59-item FFQ, in food group models did not substantially alter our results.

Our results for red meat are consistent with reports of some case–control studies [50–54]. The results of our cohort study differ from the results of other cohort studies [6–8, 10–13] (Table 4). However, comparisons between the results of cohort studies are complicated by differing definitions of meat and differing categorizations of meat intake frequency. Other than our study, no cohort studies reported significantly increased risk of lung cancer associated with meat intake. However, three studies [6, 7, 11] (including one of lung cancer mortality) [6], reported point estimates of 1.3, suggesting the possibility of increased risk.

Our finding that dairy products were associated with reduced lung cancer mortality is interesting. Unfortunately, high numbers of milk non-consumers (for example, 16,000 for skim milk) limited statistical power to detect differences between different types of milk (whole, 2%, 1% or skim). Dairy products contain non-nutritive compounds with anticarcinogenic activity including conjugated linoleic acid, sphingomyelins, and butyric acid [55]. Milk is rich in pre-formed vitamin A (retinol), calcium and vitamin D. Retinol, necessary for cell differentiation, has been inconsistently associated with reduced risk of lung cancer [4]. In some epidemiologic studies, calcium and vitamin D have been associated with reduced risk of colorectal cancer [4], but evidence of an effect on lung cancer is lacking [4, 51]. Milk may contain phytochemicals [55], but epidemiologic evidence to substantiate a protective effect from this source is lacking.

There are few epidemiologic data on the association between dairy products and lung cancer (Table 4). Kvale *et al.* reported decreased risk for lung cancer for a subgroup of men with squamous cell lung cancer [11]; other studies have shown no association [6, 7, 9, 10, 12, 56, 57] or increased risk [51, 53, 58, 59]. One study [51] found elevated risk with intake of whole milk and decreased risk with intake of reduced fat milk. Comparison between our results and the results of other cohort studies on dairy products is not straightforward as the NHIS FFQ questions on milk intake specifically excluded milk on breakfast cereals. Thus, milk frequency was undoubtedly underestimated. Although there is no way to accurately impute the frequency of milk on cereal, we thought that frequency of cereal consumption was our best available surrogate for total frequency of milk intake. The Pearson correlation coefficient between dairy products and breakfast cereals was $r = 0.20$ ($p < 0.0001$). We created a new variable to include in a Cox model; intake frequency of the diary products group plus intake frequency of the breakfast cereals group. The RR for this surrogate total milk variable was 0.5 for the highest compared to lowest quartile (CI 0.3–0.8), similar to our original estimate.

We found no association between fruit and vegetable intake and lung cancer mortality in this study. Some [7, 60–62], but not all [11, 63], cohort studies and the majority of case–control studies [4] have reported significant protective effects of fruits and vegetables. Given the incompleteness of the FFQ assessment of fruit and vegetable intake (six questions on fruits and vegetables, omitting commonly consumed items such as bananas, green beans, corn, and peas) our data are not comparable to data from other studies, the majority of which had more comprehensive assessments. We found a statistically significant interaction between alcohol, smoking, and lung cancer mortality. Of three cohort studies that considered an interaction between drinking and smoking, two [17, 18] found none while the other [15] reported greatest risk of lung cancer at the extreme upper levels of both drinking and smoking. Our study found no difference in lung cancer mortality across levels of drinking for individuals with long smoking duration (25 and 50 years); instead, mortality was greatest among non-smokers at the highest level of drinking. Taken together, the results of these four studies are inconsistent and do not suggest that an interaction between alcohol and smoking is of major importance.

The major strengths of our study were that prospectively collected dietary data were obtained from a

Table 4. Cohort studies of meats, dairy products, and lung cancer incidence and mortality (all adjusted for smoking)

Study	Population	Follow-up/ endpoint	Subjects (Case/non-case)	Frequency	RR (95% CI) [p for trend]	
Chow *et al.*, 1992 [6]	White men, age 35+, policy holders with Lutheran Brotherhood Insurance Society (United States)	20 years/ mortality	219/17,633	Meat (*times/week*)[a]		
				>10.7 *vs.* <2.3	1.3 (0.7–2.3)	
				Dairy (*times/week*)[a]		
				>20.3 *vs.* <6.6	0.8 (0.4–1.4)	
Fraser *et al.*, 1991 [7]	California Seventh-day Adventists, non-Hispanic men and women, age 25–80+	6 years/incidence	61/34,198	Meat/fish/poultry (*times/week*)		
				>2 *vs.* ≤2	1.31 (0.52–3.28)	
				Milk (*times/week*)		
				≥7 *vs.* <7	0.88 (0.37–2.12)	
Knekt *et al.*, 1991 [8]	Finnish men, age 20–69 – random sample providing diet history in mobile clinic screening study	20 years/incidence	117/4,538	(referent = high tertile)	Non-smokers	Smokers
				Meat (*low tertile*)	0.88 [0.74]	0.75 [0.47]
				Dairy (*low tertile*)	0.46 [0.35]	0.88 [0.24]
Kvale *et al.*, 1983 [11]	Three groups of Norwegian men (general population sample, family members of patients in a case–control study)	11.5 years/incidence	81 (with smoking data)/ 10,602 had histologically verified tumors (50 squamous or small cell)		All	Squamous
				Meats (*times/week*)[a]		
				≥2.9 *vs.* <1.4	1.33 [0.41]	1.03 [0.95]
				Milk (*glasses/day*)		
				≥4 *vs.* <4	0.64 [0.17]	0.32 [0.01]
Shekelle *et al.*, 1991 [12]	Men employed by Western Electric Company, age 40–55	24 years/incidence	57/1,878		(*data not shown*)	
				Beef	n.s.	
				Pork	n.s.	
				Whole milk	n.s.	
				Cheese	n.s.	
Veierod *et al.*, 1997 [13]	Men and women attending Norwegian health screening, age 16–56	11 years/incidence	153/51,452	Main meals with meat (*times/week*)		
				≥5 *vs.* ≤2	0.9 (0.5–1.6)	
				Frankfurters (*times/week*)[a]		
				≥0.7 *vs.* ≤0.1	2.1 (1.0–4.4)	
				Milk (use)		
				Skim *vs* whole	0.5 (0.3–0.9)	
				None *vs* whole	0.4 (0.1–1.4)	
Wu *et al.*, 1994 [10]	Postmenopausal women in Iowa Women's Health Study, median age 61	6 years/incidence	Women: 212/34,491	Meat	(*data not shown*)	
				Red meat	n.s.	
				Preserved meat	n.s.	
				Dairy products	n.s.	
				Milk	n.s.	

[a] Intake frequencies reported as times per month were converted to times per week to facilitate comparison of results.

nationally representative cohort. In addition, detailed data on smoking were available, making it possible to control for confounding and to study effect modification. The limitations of our study included having a brief dietary assessment tool administered at a single point-in-time and having a cohort followed-up for a relatively short duration (median of 8.5 years). In addition, although the FFQ has been validated for nutrients [40], it has not been validated for foods.

The results of our study may have differed from those of other cohort studies listed in Table 4 because of differences in populations and design. Unlike these other cohort studies, ours was performed in a sample representative of the US population. Our study also had an excellent response rate. Some of the other cohort studies had smaller numbers of lung cancer cases [7, 11, 12], which would lower their power. In addition, duration of follow-up differed in each cohort study, ranging from 6 years [7, 10] to 24 years [12]. The endpoint of our study was lung cancer mortality. In the other cohort studies, with one exception [6], the endpoint was incidence. However, survival from lung cancer is short, with incidence closely paralleling mortality [1]. Thus, we would not have expected this fact to account for differences between the results of our study of diet and lung cancer mortality and other studies of diet and lung cancer incidence. It should be noted that all of the cohort studies referenced in Table 4, and ours, adjusted for smoking.

In conclusion, red meats, in particular pork and ground beef, were associated with increased lung cancer mortality, while dairy products were associated with reduced risk. There have been few cohort studies of these associations. Further studies are needed to confirm our results and to elucidate possible mechanisms.

Acknowledgements

We thank James Cucinelli and Lisa Kahle of Information Management Systems, Silver Spring, MD, for assistance with programming and Drs Matt McKenna and Ralph Coates for comments.

Appendix: Composition of food groups formed from food-items on the 1987 National Health Interview Survey Food Frequency Questionnaire

Food groups

Fruits
Oranges
Grapefruit
Orange juice or grapefruit juice
Apples or apple sauce
Cantaloupe in season

Vegetables[a]
Beans, such as baked, pinto, kidney beans, or chili, not including green beans
Tomatoes, including in salad
Broccoli
Carrots or mixed vegetables containing carrots
Coleslaw, cabbage, or sauerkraut
Green salad
Spinach
Mustard greens, turnip greens, or collards

Dark green, yellow, orange vegetables
Broccoli
Carrots or mixed vegetables containing carrots
Spinach
Mustard greens, turnip greens, or collards
Sweet potatoes or yams

Meat/poultry/fish
Fried fish or fish sandwiches
Ground beef (hamburgers, cheeseburgers, or meatloaf)
Beef, such as steaks or roasts
Beef stew or potpie with vegetables
Pork, such as pork chops or roasts
Liver, including chicken liver
Fried chicken
Chicken or turkey, baked, stewed or broiled
Bacon
Sausage
Hot dogs
Ham or lunch meats

Red meats
Hamburgers, cheeseburgers, or meatloaf
Beef, such as steaks or roasts
Pork, such as pork chops or roasts
Bacon
Sausage

Processed meats
Ham or lunch meats
Hot dogs

Dairy products
Cheese or cheese spreads, not including cottage cheese
Whole milk or drinks made with whole milk, not including on cereal
2% milk or drinks made with 2% milk, not including on cereal
Skim milk, 1% milk, or buttermilk, not including on cereal

Breakfast cereals
Cooked cereals such as oatmeal
Highly fortified cereals like Product 19, Total, or Most
High-fiber cereals such as bran, granola, or shredded wheat
Other cold cereals such as Rice Krispies or corn flakes

Appendix (Continued)

Other starches
Rice
Spaghetti, lasagna, or pasta with tomato sauce
White bread, rolls, or crackers, including sandwiches, bagels and so forth
Dark breads such as whole wheat, rye, or pumpernickel
Corn bread, corn muffins, corn tortillas, or grits

Added fats
Salad dressing or mayonnaise, including on sandwiches
Butter on bread, rolls, or vegetables
Margarine on bread, rolls, or vegetables

Desserts
Ice cream
Doughnuts, cookies, cake or pastry
Pie
Chocolate candy

Alcohol
Beer (during past year)
Wine (during past year)
Liquor (during past year)

Food items not included in groups
Other fruit juices or fortified fruit drinks
Eggs
Peanuts or peanut butter
Salty snacks such as chips or popcorn
Sugar in coffee, tea or on cereal
Milk or cream in coffee or tea
Soda or soft drinks with sugar

[a] The vegetable group was analyzed with/without white potatoes, baked, boiled or mashed, and French fries or fried potatoes.

References

1. Landis SH, Murray T, Bolden S, Wingo PA (1999) Cancer Statistics, 1999. *CA Cancer J Clin* **49**: 8–31.
2. US Department of Health Education and Welfare (1964) *Smoking and Health*. Public Health Service Publ. no.1103. Washington, DC: Government Printing Office.
3. Willett W (1995) Diet, nutrition, and avoidable cancer. *Environ Health Perspect* **103**: 165–170.
4. *Food, Nutrition and the Prevention of Cancer: a global perspective* (1997) World Cancer Research Fund/American Institute for Cancer Research. Washington, DC: American Institute for Cancer Research.
5. Ziegler RG, Mayne ST, Swanson CA (1996) Nutrition and lung cancer. *Cancer Causes Control* **7**: 157–177.
6. Chow WH, Schuman LM, McLaughlin JK, *et al.* (1992) A cohort study of tobacco use, diet, occupation, and lung cancer mortality. *Cancer Causes Control* **3**: 247–254.
7. Fraser GE, Beeson WL, Phillips RL (1991) Diet and lung cancer in California Seventh-day Adventists. *Am J Epidemiol* **133**: 683–693.
8. Knekt P, Jarvinen R, Seppanen R, *et al.* (1991) Dietary antioxidants and the risk of lung cancer. *Am J Epidemiol* **134**: 471–479.
9. Knekt P, Seppanen R, Jarvinen R, *et al.* (1991) Dietary cholesterol, fatty acids, and the risk of lung cancer among men. *Nutr Cancer* **16**: 267–275.
10. Wu Y, Zheng W, Sellers TA, Kushi LH, Bostick RM, Potter JD (1994) Dietary cholesterol, fat, and lung cancer incidence among older women: the Iowa Women's Health Study (United States). *Cancer Causes Control* **5**: 395–400.
11. Kvale G, Bjelke E, Gart JJ (1983) Dietary habits and lung cancer risk. *Int J Cancer* **31**: 397–405.
12. Shekelle RB, Rossof AH, Stamler J (1991) Dietary cholesterol and incidence of lung cancer: the Western Electric Study. *Am J Epidemiol* **134**: 480–484.
13. Veierod MB, Laake P, Thelle DS (1997) Dietary fat intake and risk of lung cancer: a prospective study of 51,452 Norwegian men and women. *Eur J Cancer Prev* **6**: 540–549.
14. Gordon T, Doyle JT (1987) Drinking and mortality. The Albany Study. *Am J Epidemiol* **125**: 263–270.
15. Klatsky AL, Armstrong MA, Friedman GD (1981) Alcohol and mortality: a ten-year Kaiser-Permanente experience. *Ann Intern Med* **95**: 139–145.
16. Kono S, Ikeda M, Tokudome S, Nishizumi M, Kuratsune M (1986) Alcohol and mortality: a cohort study of male Japanese physicians. *Int J Epidemiol* **15**: 527–532.
17. Pollack ES, Nomura AM, Heilbrun LK, Stemmermann GN, Green SB (1984) Prospective study of alcohol consumption and cancer. *N Engl J Med* **310**: 617–621.
18. Potter JD, Sellers TA, Folsom AR, McGovern PG (1992) Alcohol, beer, and lung cancer in postmenopausal women. The Iowa Women's Health Study. *Ann Epidemiol* **2**: 587–595.
19. Thun MJ, Peto R, Lopez AD, *et al.* (1997) Alcohol consumption and mortality among middle-aged and elderly U.S. adults. *N Engl J Med* **337**: 1705–1714.
20. Layton DW, Bogen KT, Knize MG, Hatch FT, Johnson VM, Felton JS (1995) Cancer risk of heterocyclic amines in cooked foods: an analysis and implications for research. *Carcinogenesis* **16**: 39–52.
21. Sugimura T (1985) Carcinogenicity of mutagenic heterocyclic amines formed during the cooking process. *Mutat Res* **150**: 33–41.
22. Wakabayashi K, Nagao M, Esumi H, Sugimura T (1992) Food-derived mutagens and carcinogens. *Cancer Res* **52**: 2092s–2098s.
23. Sinha R, Knize MG, Salmon CP, *et al.* (1998) Heterocyclic amine content of pork products cooked by different methods and to varying degrees of doneness. *Food Chem Toxicol* **36**: 289–297.
24. Sinha R, Rothman N, Salmon CP, *et al.* (1998) Heterocyclic amine content in beef cooked by different methods to varying degrees of doneness and gravy made from meat drippings. *Food Chem Toxicol* **36**: 279–287.
25. Byrne C, Sinha R, Platz EA, *et al.* (1998) Predictors of dietary heterocyclic amine intake in three prospective cohorts. *Cancer Epidemiol Biomarkers Prev* **7**: 523–529.
26. US Preventive Services Task Force (1996) *Guide to Clinical Preventive Services*, 2nd edn. Baltimore: Williams & Wilkins.
27. Cox CS, Mussolino ME, Rothwell ST, *et al.* (1997) Plan and operation of the NHANES I Epidemiologic Followup Study, 1992. *Vital Health Stat* **1**: 1–23.
28. National Center for Health Statistics (1985) The National Health Interview Survey Design, 1973–1984, and Procedures, 1975–1983. Series 1, Number 18, DHHS Publ. No. (PHS) 85-1320. Hyattsville, MD: Department of Health and Human Services, pp. 815–1320.
29. National Center for Health Statistics, Schoenborn CA, Marano M (1988) Current Estimates from the National Health Interview Survey: United States, 1987. Vital and Health Statistics, Series 10, No. 166. DHHS Publ. No. (PHS) 88-1594. Washington, D.C.: Government Printing Office, pp. 1–233.
30. Patterson BH, Bilgrad R (1986) Use of the National Death Index in cancer studies. *J Natl Cancer Inst* **77**: 877–881.

31. Horm JW, Wright RA (1993) A new national source of health and mortality information in the United States. *Proc Am Stat Assoc* 256–261.
32. Block G, Hartman AM, Dresser CM, Carroll MD, Gannon J, Gardner L (1986) A data-based approach to diet questionnaire design and testing. *Am J Epidemiol* **124**: 453–469.
33. Block G, Dresser CM, Hartman AM, Carroll MD (1985) Nutrient sources in the American diet: quantitative data from the NHANES II survey. II. Macronutrients and fats. *Am J Epidemiol* **122**: 27–40.
34. Block G, Dresser CM, Hartman AM, Carroll MD (1985) Nutrient sources in the American diet: quantitative data from the NHANES II survey. I. Vitamins and minerals. *Am J Epidemiol* **122**: 13–26.
35. Sobell J, Block G, Koslowe P, Tobin J, Andres R (1989) Validation of a retrospective questionnaire assessing diet 10–15 years ago. *Am J Epidemiol* **130**: 173–187.
36. Block G, Woods M, Potosky A, Clifford C (1990) Validation of a self-administered diet history questionnaire using multiple diet records. *J Clin Epidemiol* **43**: 1327–1335.
37. Mares-Perlman JA, Klein BE, Klein R, Ritter LL, Fisher MR, Freudenheim JL (1993) A diet history questionnaire ranks nutrient intakes in middle-aged and older men and women similarly to multiple food records. *J Nutr* **123**: 489–501.
38. Block G, Thompson FE, Hartman AM, Larkin FA, Guire KE (1992) Comparison of two dietary questionnaires validated against multiple dietary records collected during a 1-year period. *J Am Diet Assoc* **92**: 686–693.
39. Coates RJ, Eley JW, Block G, *et al.* (1991) An evaluation of a food frequency questionnaire for assessing dietary intake of specific carotenoids and vitamin E among low-income black women. *Am J Epidemiol* **134**: 658–671.
40. Block G, Hartman AM, Naughton D (1990) A reduced dietary questionnaire: development and validation. *Epidemiology* **1**: 58–64.
41. Willett W (1998) *Nutritional Epidemiology*, 2nd edn. New York: Oxford University Press.
42. Korn EL, Graubard BI, Midthune D (1997) Time-to-event analysis of longitudinal follow-up of a survey: choice of the time scale. *Am J Epidemiol* **145**: 72–80.
43. Durrleman S, Simon R (1989) Flexible regression models with cubic splines. Stat Med **8**: 551–561.
44. Scholar EM, Violi LA, Newland J, Bresnick E, Birt DF (1989) The effect of dietary fat on metastasis of the Lewis lung carcinoma and the BALB/c mammary carcinoma. *Nutr Cancer* **12**: 109–119.
45. Byers TE, Graham S, Haughey BP, Marshall JR, Swanson MK (1987) Diet and lung cancer risk: findings from the Western New York Diet Study. *Am J Epidemiol* **125**: 351–363.
46. Goodman MT, Kolonel LN, Yoshizawa CN, Hankin JH (1988) The effect of dietary cholesterol and fat on the risk of lung cancer in Hawaii. *Am J Epidemiol* **128**: 1241–1255.
47. Hankin JH, Zhao LP, Wilkens LR, Kolonel LN (1992) Attributable risk of breast, prostate, and lung cancer in Hawaii due to saturated fat. *Cancer Causes Control* **3**: 17–23.
48. Jain M, Burch JD, Howe GR, Risch HA, Miller AB (1990) Dietary factors and risk of lung cancer: results from a case–control study, Toronto, 1981–1985. *Int J Cancer* **45**: 287–293.
49. Swanson CA (1997) Dietary fats and lung cancer risk among women: the Missouri Women's Health Study. *Cancer Causes Control* **8**: 883–893.
50. Deneo-Pellegrini H, De Stefani E, Ronco A, Mendilaharsu M, Carzoglio JC (1996) Meat consumption and risk of lung cancer; a case–control study from Uruguay. *Lung Cancer* **14**: 195–205.
51. Mettlin C (1989) Milk drinking, other beverage habits, and lung cancer risk. *Int J Cancer* **43**: 608–612.
52. Sinha R, Kulldorff M, Crandall J, Brown CC, Alavanja MCR, Swanson CA (1999) Fried, well done red meat and risk of lung cancer in women. *Cancer Causes Control* **9**: 621–630.
53. Sankaranarayanan R, Varghese C, Duffy SW, Padmakumary G, Day NE, Nair MK (1994) A case–control study of diet and lung cancer in Kerala, South India. *Int J Cancer* **58**: 644–649.
54. Suzuki I, Hamada GS, Zamboni MM, Cordeiro PB, Watanabe S, Tsugane S (1994) Risk factors for lung cancer in Rio de Janeiro, Brazil: a case–control study. *Lung Cancer* **11**: 179–190.
55. Parodi PW (1997) Cows' milk fat components as potential anticarcinogenic agents. *J Nutr* **127**: 1055–1060.
56. Kalandidi A, Katsouyanni K, Voropoulou N, Bastas G, Saracci R, Trichopoulos D (1990) Passive smoking and diet in the etiology of lung cancer among non-smokers. *Cancer Causes Control* **1**: 15–21.
57. Koo LC (1988) Dietary habits and lung cancer risk among Chinese females in Hong Kong who never smoked. *Nutr Cancer* **11**: 155–172.
58. Axelsson G, Liljeqvist T, Andersson L, Bergman B, Rylander R (1996) Dietary factors and lung cancer among men in west Sweden. *Int J Epidemiol* **25**: 32–39.
59. Goodman MT, Hankin JH, Wilkens LR, Kolonel LN (1992) High-fat foods and the risk of lung cancer. *Epidemiology* **3**: 288–299.
60. Hirayama T (1986) Nutrition and cancer – a large scale cohort study. *Prog Clin Biol Res* **206**: 299–311.
61. Kromhout D (1987) Essential micronutrients in relation to carcinogenesis. *Am J Clin Nutr* **45**: 1361–1367.
62. Steinmetz KA, Potter JD, Folsom AR (1993) Vegetables, fruit, and lung cancer in the Iowa Women's Health Study. *Cancer Res* **53**: 536–543.
63. Shibata A, Paganini-Hill A, Ross RK, Henderson BE (1992) Intake of vegetables, fruits, beta-carotene, vitamin C and vitamin supplements and cancer incidence among the elderly: a prospective study. *Br J Cancer* **66**: 673–679.