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Dietary patterns and mortality in a Chinese population¹⁻³

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ABSTRACT

Background: Limited research has examined the association between dietary patterns and mortality, especially in non-Western populations.

Objective: We examined the association of dietary patterns with all-cause mortality and cause-specific mortality in the Singapore Chinese Health Study, which included a unique ethnic population with strong Western and South Asian cultural influences.

Design: We conducted a prospective data analysis of the Singapore Chinese Health Study, which included 52,584 Chinese men and women (aged 45–74 y) who were free of diabetes, cardiovascular disease (CVD), and cancer at baseline (1993–1998) and followed through 2011 with 10,029 deaths. The following 2 major dietary patterns were identified by using a principal components analysis: a vegetable-, fruit-, and soy-rich (VFS) pattern and a dim sum– and meat-rich (DSM) dietary pattern. Pattern scores for each participant were calculated and examined with all-cause and cause-specific mortality risks by using a Cox proportional hazards regression.

Results: The VFS pattern was inversely associated with all-cause mortality and each cause-specific category (CVD, cancer, and respiratory) of mortality during the follow-up period. Compared with the lowest quintile of the VFS pattern, HRs for quintiles 2-5 for all-cause mortality were 0.90, 0.79, 0.80, and 0.75, respectively (*P*-trend < 0.0001). The DSM pattern was positively associated with CVD mortality in the whole population (HR for fifth quintile compared with first quintile: 1.23; 95% CI: 1.07, 1.40; P-trend = 0.001). Positive associations between the DSM pattern and cancer and all-cause mortality were only present in ever-smokers. In eversmokers, relative to the first quintile, HRs for quintiles 2-5 of the DSM pattern for all-cause mortality were 1.04, 1.04, 1.13, and 1.24, respectively (P-trend < 0.0001). Similarly, HRs for quintiles 2–5 for cancer mortality were 1.08, 1.03, 1.25, and 1.34, respectively (P-trend < 0.0001). The DSM pattern was not associated with respiratory mortality.

Conclusion: Dietary patterns are strongly associated with mortality in Chinese Singaporeans. *Am J Clin Nutr* 2014;100:877–83.

INTRODUCTION

A broad, growing, and evolving body of scientific inquiry on the association between dietary intake and health and disease has led to a recommendation to eat a dietary pattern that emphasizes a variety of plant-based foods (eg, vegetables, fruit, legumes, whole grains, nuts, and seeds), and deemphasizing processed food products, especially those with added sugars and that are high in meat (particularly processed meats) (1). Indeed, this type of dietary pattern is recognized as a major factor in the prevention and treatment of the major chronic diseases (2). Less described has been the association between diet and longevity.

The research that has examined dietary patterns with mortality has been limited to Western and East Asian populations (eg, Japanese) with little data from Southeast Asian populations (3– 11). Generally, these studies have suggested that a culturally appropriate version of the previously mentioned recommended dietary pattern was associated with lower risk of all-cause or cardiovascular disease (CVD)⁴ mortality. However, most studies were limited by a small sample size, short follow-up time, or the ability to adequately address important potential effect-measure modifiers (eg, education and smoking status).

The Singapore Chinese Health Study (SCHS) provides a unique population to examine dietary patterns and longevity. The culture has both Western and south Asian dietary influences. In addition to the Western dietary influences, mortality patterns of Singapore mirror those of Western countries, providing a unique population within which to study this topic (12). Thus, the goal of this study was to examine the associations between dietary patterns and all-cause mortality and cause-specific mortality in Singaporean Chinese. On the basis of the literature and previous studies from the SCHS, we hypothesized that a dietary pattern that emphasizes plant-based foods would be inversely associated with all-cause mortality and cause-specific mortality during follow-up, and a dietary pattern that deemphasizes plant-based foods and emphasizes more meat and processed foods in the diet would be positively associated with mortality during follow-up. We also hypothesized that smoking status may modify the dietmortality association as it has for other diet-disease relations in the SCHS and elsewhere (13-16).

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⁴ Abbreviations used: CVD, cardiovascular disease; DSM, dim sum– and meat-rich; SCHS, Singapore Chinese Health Study; VFS, vegetable-, fruit-, and soy-rich.

SUBJECTS AND METHODS

Study population

The design of the Singapore Chinese Health Study has been previously described (17). Briefly, the cohort was drawn from men and women, aged 45–74 y, who belonged to one of the major dialect groups (Hokkien or Cantonese) of Chinese in Singapore. Between April 1993 and December 1998, 63,257 individuals completed an in-person interview that included questions on usual diet, demographics, height and weight, use of tobacco, usual physical activity, menstrual and reproductive history (women only), medical history, and family history of cancer. Institutional review boards at the National University of Singapore, University of Pittsburgh, and the University of Minnesota approved this study.

Assessment of diet and covariates

A semiquantitative food-frequency questionnaire specifically developed for this population that assessed 165 commonly consumed dishes and food items was administered during the baseline interview to assess usual dietary intake of the previous year. During the interview, the respondent referred to accompanying photographs to select from 8 food-frequency categories (ranging from "never or hardly ever" to "2 or more times a day") and 3 portion sizes. The food-frequency questionnaire has subsequently been validated against a series of 24-h dietary recall interviews (17) as well as selected biomarker studies (18, 19). In conjunction with this cohort, the Singapore Food Composition Table was developed, which is a food-nutrient database that lists the amounts of 96 nutritive and nonnutritive components per 100 g raw food, cooked food, and beverages in the diet of Singaporean Chinese (17).

At the baseline interview, all lifestyle factors were collected by a self-report. Height and weight were used to calculate BMI (in kg/m^2) as weight divided by height squared. An inquiry on smoking habits included smoking status (never, former, or current smoker), and former and current (ie, ever) smokers were further asked for age when they started or quit smoking, the number of cigarettes per day, and the number of years of smoking (20). For physical activity, participants were asked the number of hours per week spent on moderate activities such as brisk walking, bowling, bicycling on level ground, tai chi or chi kung, and strenuous sports such as jogging, bicycling on hills, tennis, squash, swimming laps, or aerobics. The physical activity portion of the questionnaire was modeled after the European Prospective Investigation into Cancer and Nutrition study physical activity questionnaire, which has been shown to be valid and repeatable (21). The usual sleep duration was assessed by using the following question: "On average, during the last year, how many hours in a day did you sleep?" with response categories of ≤ 5 h, 6, 7, 8, 9, and ≥ 10 h.

Assessment of mortality

Information on the date and cause of death was obtained through linkage with the nation-wide registry of birth and death in Singapore. Up to 6 different International Classification of Diseases version 9 codes were recorded in the registry. The primary cause of death was used for analysis. The vital status of cohort participants was updated through 31 December 2011. Follow-up for mortality was considered virtually complete because of the linkage analysis and negligible emigration; only 47 subjects from this cohort were known to be lost to follow-up because of migration out of Singapore or for other reasons. Endpoints in our cause-specific analyses were deaths from CVD (codes 394.0–459.0), all cancers (140.0–195.8 and 199–208.9), and respiratory causes [such as pneumonia and influenza (480– 488) and chronic obstructive pulmonary disease (490–496)].

Statistical analysis

For the analysis, we excluded 1936 subjects with a history of invasive cancer (except nonmelanoma skin cancer) or superficial, papillary bladder cancer at recruitment because they did not meet study inclusion criteria. We further excluded subjects with a self-reported history of physician-diagnosed diabetes (n = 5469) or CVD (n = 2399) at baseline, plus 869 subjects who reported extreme sex-specific energy intakes (<600 or >3000 kcal for women; <700 or >3700 kcal for men). The analysis included 52,584 participants.

Dietary patterns were derived by using a principal components analysis with SAS software (version 9.2; SAS Institute Inc). All 165 foods and beverages, including alcohol, were first standardized to the same frequency per month unit before the principal components analysis method was applied, and factors were extracted. Factors were rotated orthogonally to maintain an uncorrelated state and improve interpretability, and a 2-factor solution was retained that was based on eigenvalues (>1.0), a scree plot, and factor interpretability. For comparability and interpretability of our results, we present factor loadings ≥ 0.20 even though values <0.20 were statistically significant because of the large sample size of the study. These variables are in alignment with those used in previous studies (22-26). Factor scores for each participant were calculated by multiplying the intake of the standardized food item by its respective factor loading on each pattern. Scores were linear variables and represented the weighted sum of all 165 food and beverage items. Participants were divided into quintiles by scores to indicate the amount their dietary intake corresponded with each pattern (ie, a higher score corresponded with greater conformity to the derived pattern). Factors were initially extracted by sex, dialect, and smoking status and were highly similar in loading structure and disease prediction to reported whole-cohort factors, and thus, factors derived from the overall cohort were used. Baseline and dietary characteristics were calculated for participants across quintiles of each dietary pattern score. For each study subject, person-years were counted from the date of the baseline interview to the date of death, date of last contact (for the few subjects who migrated out of Singapore), or 31 December 2011, whichever occurred first. HRs per quintile of dietary pattern score were estimated by using Cox proportional hazards regression models with SAS statistical software (version 9.2). There was no evidence that proportional hazards assumptions were violated as indicated by the lack of a significant interaction between dietary pattern scores and a function of the survival time in models. Tests for trends across dietary patterns scores were performed by assigning the median value of the quintile to the respective categories and entering this variable as a continuous variable into the models.

Two models were constructed to examine the association between dietary pattern score and risk of mortality during followup as follows: covariates included in model 1 were baseline age $(<50, 50-54, 55-59, 60-64, or \ge 65 y)$, year of interview (1993– 1995 or 1996-1998), dialect (Hokkiens compared with Cantonese), sex, and education (none, primary, and secondary or above); the second model included smoking (never, light, or heavy) (27), moderate activity (≥ 2 compared with < 2 h/wk), strenuous physical activity (≥ 1.5 compared with < 1.5 h/wk), sleep (6–8 compared with <6 or \geq 9 h/d), history of physiciandiagnosed hypertension (yes compared with no), total energy intake (kcal/d), and baseline BMI [in kg/m² as the original BMI and its quadratic term (BMI²)]. Analyses were conducted to test for interactions between diet patterns and age, sex, education, BMI, and smoking. Interactions with smoking and BMI were hypothesized to have a biological basis because of the biological enhancement of overall risk conferred by obesity and cigarette smoking, whereby the age-, sex-, and education-interaction tests were carried out to check for differential results because of varying distributions of dietary patterns and confounding factors. To reduce the potential bias because of nonreported preexisting disease or an underlying illness, participants who died within 3 to <5 y were excluded in sensitivity analyses.

RESULTS

Two main dietary patterns were derived from the principal components analysis. The first was pattern was named vegetable-, fruit-, and soy-rich (VFS). *See* Supplemental Table 1 under

"Supplemental data" in the online issue for factor loadings for this pattern ≥ 0.20 . Foods that loaded highly on this pattern were predominantly vegetables, fruit, and soy-based items. The second pattern was named dim sum– and meat-rich (DSM). *See* Supplemental Table 1 under "Supplemental data" in the online issue for foods that loaded ≥ 0.20 on the DSM pattern. A variety of foods, predominantly dim sum, fresh and processed meats and seafood, noodle and rice dishes, sweetened foods, and deep-fried foods, were prominent contributors to the pattern. Most dim sum foods are savory pastries such as steamed or deep-fried dumplings, filled buns, noodles, sweet pastries, and meat dishes.

Baseline characteristics of participants according to dietary pattern scores are shown in **Tables 1** and **2**. Higher VFS pattern– score participants were, on average, slightly younger, more educated, smoked less, and more active, and a greater proportion were women. Nutritionally, participants with a higher VFS score reported consuming less carbohydrate but more fiber, more dietary fat mainly from plant sources, and more protein. In contrast, participants with higher DSM scores were younger, more educated, smoked more, had less moderate physical activity but greater vigorous activity, and a greater proportion were men. Nutritionally, participants with higher DSM scores reported consuming less carbohydrate with a corresponding decrease in dietary fiber, more dietary fat mainly from animal sources, and more protein.

HRs for all-cause mortality and cause-specific mortality according to each pattern score are shown in **Table 3**. Overall, during \sim 793,948 person-years of follow-up there were 10,029 deaths. There was a strong, inverse association between a higher

Baseline demographic and lifestyle characteristics according to quintile of dietary pattern score: the SCHS¹

Dietary pattern	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5	P-trend
Vegetable-, fruit-, and soy-rich						
n	10,516	10,517	10,517	10,517	10,517	_
Age (y)	56.4 ± 8.1^2	56.2 ± 8.0	55.8 ± 7.8	55.4 ± 7.7	55.2 ± 7.7	< 0.0001
Sex (F) (%)	43.3	55.6	57.8	60.7	61.8	< 0.0001
Dialect (Hokkien) (%)	59.8	56.1	53.1	50.3	49.0	< 0.0001
Education (%)	22.6	26.1	29.6	32.5	36.6	< 0.0001
Smoking (ever) (%)	45.0	31.8	27.7	23.0	21.3	< 0.0001
Moderate activity (%)	10.1	14.1	16.7	17.7	19.8	< 0.0001
Vigorous activity (%)	10.9	9.1	8.9	8.9	9.7	0.018
BMI (kg/m ²)	22.8 ± 3.6	23.1 ± 3.6	23.0 ± 3.5	23.0 ± 3.5	23.0 ± 3.5	0.28
Sleep (h)	7.0 ± 1.2	7.0 ± 1.1	7.0 ± 1.1	7.0 ± 1.1	7.0 ± 1.1	0.25
Dim sum– and meat-rich						
п	10,516	10,517	10,517	10,517	10,517	_
Age (y)	58.3 ± 8.0	56.7 ± 7.9	55.6 ± 7.7	$54.8~\pm~7.8$	53.6 ± 7.2	< 0.0001
Sex (F) (%)	72.4	63.4	56.3	48.5	38.8	< 0.0001
Dialect (Hokkien) (%)	55.3	54.4	53.3	53.4	51.9	< 0.0001
Education $(\%)^3$	22.0	25.5	28.1	32.7	39.1	< 0.0001
Smoking (ever) (%)	17.5	25.2	29.8	35.1	41.3	< 0.0001
Moderate activity $(\%)^4$	18.9	16.0	15.3	14.4	13.8	< 0.0001
Vigorous activity (%) ⁵	5.4	7.0	8.9	11.4	14.9	< 0.0001
BMI (kg/m ²)	22.9 ± 3.6	23.0 ± 3.5	23.1 ± 3.5	23.0 ± 3.5	23.0 ± 3.5	0.78
Sleep (h)	6.9 ± 1.1	7.0 ± 1.1	7.0 ± 1.1	7.0 ± 1.1	7.0 ± 1.1	0.01

¹ SCHS, Singapore Chinese Cohort Study.

²Mean \pm SD (all such values).

³ Percentage of population with a secondary education or greater.

⁴ Moderate activity: ≥ 2 h/wk (percentage of subjects who reported this level).

⁵ Vigorous activity: ≥ 1.5 h/wk vigorous work or strenuous physical activity (percentage of subjects who reported this level).

TABLE 2

Dietary intake characteristics according to quintile of dietary pattern score¹

	Vegetable, fruit, and soy			Dim sum and meat		
	Quintile 1	Quintile 3	Quintile 5	Quintile 1	Quintile 3	Quintile 5
Total energy (kcal/d)	1373 ± 502	1503 ± 463	1876 ± 523	1245 ± 372	1481 ± 403	2078 ± 540
Carbohydrate (% of energy)	61.2 ± 7.9	59.5 ± 6.8	56.4 ± 6.9	63.2 ± 6.8	59.4 ± 6.7	54.6 ± 6.4
Total fat (% of energy)	22.4 ± 5.8	24.8 ± 5.1	28.2 ± 5.0	22.4 ± 5.4	24.7 ± 5.2	28.5 ± 5.0
Saturated fat (% of energy)	8.3 ± 2.6	8.8 ± 2.4	9.6 ± 2.5	7.6 ± 2.4	8.7 ± 2.3	10.5 ± 2.3
Monounsaturated fat (% of energy)	7.7 ± 2.1	8.4 ± 1.9	9.4 ± 1.9	7.4 ± 1.9	8.4 ± 1.9	9.7 ± 1.9
Polyunsaturated fat (% of energy)	4.1 ± 1.4	5.0 ± 1.6	6.1 ± 2.1	5.0 ± 2.1	5.0 ± 1.8	5.3 ± 1.6
Protein (% of energy)	14.3 ± 2.5	15.2 ± 2.3	15.8 ± 2.4	14.5 ± 2.6	15.2 ± 2.4	15.7 ± 2.2
Soy protein (% of energy)	1.0 ± 0.7	1.4 ± 0.8	2.1 ± 1.2	1.4 ± 1.1	1.5 ± 1.0	1.6 ± 0.9
Fiber (g/1000 kcal)	6.1 ± 1.9	8.2 ± 2.2	10.1 ± 2.6	9.4 ± 3.0	8.0 ± 2.5	7.4 ± 2.1
Animal fat (g/1000 kcal) ²	9.0 ± 3.8	8.4 ± 3.3	8.1 ± 3.2	5.9 ± 2.8	8.4 ± 2.8	11.2 ± 3.1
Plant fat $(g/1000 \text{ kcal})^3$	13.4 ± 3.8	16.5 ± 3.7	20.1 ± 4.2	16.5 ± 4.8	16.4 ± 4.4	17.3 ± 4.1
Rice (g/1000 kcal)	318.9 ± 104.3	277.3 ± 86.9	216.6 ± 78.0	306.2 ± 101.7	276.5 ± 91.8	226.7 ± 79.1
Noodles (g/1000 kcal)	38.2 ± 30.1	33.3 ± 24.7	31.6 ± 22.7	22.1 ± 21.8	36.8 ± 26.6	41.5 ± 24.3
Vegetables (g/1000 kcal)	45.3 ± 19.7	61.0 ± 22.3	104.7 ± 40.2	83.3 ± 42.5	71.6 ± 33.2	65.0 ± 27.7
Fruit and related juices (g/1000 kcal)	75.2 ± 69.3	132.1 ± 87.7	183.9 ± 110.9	147.1 ± 109.7	129.2 ± 96.9	122.2 ± 85.3
Soy foods/beverages (g/1000 kcal)	47.0 ± 37.7	68.4 ± 43.2	99.8 ± 59.3	66.6 ± 52.5	71.4 ± 50.4	74.5 ± 46.4
Red meat (g/1000 kcal)	20.9 ± 12.1	18.7 ± 10.7	17.2 ± 10.6	11.9 ± 9.2	18.9 ± 9.8	26.2 ± 11.2
Poultry (g/1000 kcal)	12.8 ± 9.8	12.8 ± 9.4	12.7 ± 10.0	7.7 ± 7.6	12.8 ± 9.0	17.1 ± 10.0
Fish (g/1000 kcal)	32.1 ± 17.5	36.2 ± 16.7	37.8 ± 18.5	35.4 ± 20.1	36.8 ± 17.4	34.8 ± 14.5
Dairy products (g/1000 kcal)	31.9 ± 62.5	46.3 ± 74.3	55.1 ± 72.7	67.0 ± 99.0	41.2 ± 66.7	33.2 ± 47.1
Cooking fats/oils (g/1000 kcal)	8.5 ± 3.0	10.5 ± 3.0	13.2 ± 3.6	10.3 ± 3.9	10.7 ± 3.5	11.1 ± 3.1
Coffee (cups/mo)	46.5 ± 40.8	34.8 ± 34.3	29.8 ± 32.1	23.0 ± 26.8	37.6 ± 35.8	45.6 ± 40.2
Black tea (cups/mo)	7.3 ± 18.5	6.4 ± 15.7	8.0 ± 17.4	3.7 ± 11.4	6.8 ± 16.2	11.0 ± 21.3
Green tea (cups/mo)	6.3 ± 20.3	9.3 ± 24.2	12.3 ± 26.9	7.1 ± 21.1	9.4 ± 24.6	11.2 ± 26.2
Soft drinks (drinks/mo)	4.4 ± 12.9	2.2 ± 7.7	1.9 ± 6.7	0.4 ± 2.0	1.9 ± 6.5	6.8 ± 15.2
Alcohol (drinks/wk)	1.9 ± 6.2	0.7 ± 3.3	0.7 ± 3.1	0.2 ± 1.1	0.8 ± 3.6	2.2 ± 6.3

¹All values are means \pm SDs. All *P*-trend < 0.0001 across all groups except for poultry in the vegetable-, fruit-, and soy-rich pattern (*P* = 0.76).

²Dietary fat from animal-based foods.

³Dietary fat from plant-based foods.

VFS dietary pattern score and all-cause mortality and each causespecific category of mortality after adjustment for demographic and lifestyle confounders. The strongest, graded association was observed with CVD mortality (quintiles 5 compared with 1) whereby the HR (95% CI) was 0.63 (0.56, 0.72). For the DSM pattern, a significant positive association was observed for allcause mortality and CVD mortality with a suggestive positive association with mortality that was due to cancer. The DSM pattern was not associated with increased risk of mortality because of respiratory causes during the follow-up period. There was no evidence that any of the associations between VFS and DSM dietary pattern scores and all-cause– and cause-specific–mortality categories differed by age, sex, education, or BMI. Furthermore, there was no evidence that an association with the VFS pattern differed by smoking status with any mortality outcome.

However, there was evidence for a multiplicative interaction between the DSM pattern and smoking for cancer mortality (P = 0.018). This finding drove similar results in all-cause mortality whereby there was evidence that the association between the DSM pattern and risk of all-cause mortality during follow-up also differed by smoking status (P = 0.05). These results are presented in **Table 4**. In never-smokers, there was no association between the DSM pattern and all-cause or cancer mortality, whereas in ever-smokers, there was a strong, graded, positive association (P < 0.0001) for both cancer and all-cause mortality. Last, there was no evidence any that associations differed on the exclusion of deaths ≤ 5 y after enrollment.

DISCUSSION

In this large study of Chinese Singaporeans, a higher VFSpattern score displayed a strong, inverse association with risk of all-cause mortality and cause-specific (CVD, cancer, and respiratory) mortality during the follow-up period. A higher score on the other main dietary pattern in the population, the DSM pattern, was positively associated with CVD mortality. Furthermore, participants with a history of smoking and a higher DSM-pattern score had a strong, positive association with cancer and all-cause mortality, whereas there was no association in participants who never smoked with these outcomes.

The composition of the VFS dietary pattern and its association with mortality has similarities with results in published literature on the topic. The unifying theme with Western populations and their dietary patterns that were associated with reduced risk of allcause mortality was an increased emphasis on vegetables, fruit, and grains (especially whole grains) with other main components that likely vary because of cultural influence and include legumes, nut and seeds, dairy, alcohol, vegetable oils, and fish (3-7). The VFS pattern was inversely associated with each cause-specific category of mortality in the SCHS, whereas the other studies that examined cause-specific categories of mortality were less consistent. Only one other study showed reduced risk with cancer (6) and "other causes," which may have included some respiratory causes similar to the respiratory-cause group in the SCHS (3). Two other studies that examined CVD mortality observed inverse associations (3, 6). Studies that were based in

DIET AND MORTALITY IN CHINESE

TABLE 3

HRs (95% CIs) of all-cause and cause-specific mortality according to dietary pattern score in Chinese men and women^I

Туре	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5	P-trend
Vegetable-, fruit-, and soy-rich pattern						
All cause						
No. of deaths/PY	2676/153,029	2233/158,657	1867/160,339	1719/160,892	1534/161,031	_
HR (95% CI) ²	1.00	0.86 (0.81, 0.91)	0.75 (0.71, 0.80)	0.75 (0.70, 0.80)	0.69 (0.65, 0.74)	< 0.0001
HR $(95\% \text{ CI})^3$	1.00	0.90 (0.84, 0.94)	0.79 (0.74, 0.84)	0.80 (0.75, 0.85)	0.75 (0.70, 0.80)	< 0.0001
Cancer						
No. of deaths/PY	973/153,029	874/158,657	732/160,339	698/160,892	625/161,031	_
HR (95% CI) ²	1.00	0.93 (0.84, 1.02)	0.81 (0.73, 0.89)	0.83 (0.75, 0.91)	0.77 (0.70, 0.85)	< 0.0001
HR (95% CI) ³	1.00	0.97 (0.89, 1.06)	0.86 (0.78, 0.95)	0.90 (0.81, 1.00)	0.84 (0.75, 0.94)	0.0009
CVD						
No. of deaths/PY	869/153,029	706/158,657	563/160,339	524/160,892	435/161,031	_
HR $(95\% \text{ CI})^2$	1.00	0.83 (0.75, 0.92)	0.70 (0.63, 0.78)	0.70 (0.63, 0.79)	0.61 (0.54, 0.68)	< 0.0001
HR $(95\% \text{ CI})^3$	1.00	0.85 (0.77, 0.94)	0.72 (0.64, 0.80)	0.73 (0.65, 0.82)	0.63 (0.56, 0.72)	< 0.0001
Respiratory						
No. of deaths/PY	489/153,029	338/158,657	308/160,339	266/160,892	235/161,031	—
HR $(95\% \text{ CI})^2$	1.00	0.72 (0.62, 0.82)	0.69 (0.60, 0.80)	0.66 (0.57, 0.77)	0.61 (0.52, 0.71)	< 0.0001
HR $(95\% \text{ CI})^3$	1.00	0.76 (0.66, 0.87)	0.75 (0.65, 0.87)	0.73 (0.62, 0.86)	0.68 (0.58, 0.81)	< 0.0001
Dim sum- and meat-rich pattern						
All cause						
No. of deaths/PY	2246/158,384	2076/159,536	1985/158,934	1917/158,629	1805/158,465	—
HR $(95\% \text{ CI})^2$	1.00	1.00 (0.94, 1.06)	1.04 (0.98, 1.10)	1.08 (1.01, 1.15)	1.11 (1.04, 1.19)	0.0001
HR $(95\% \text{ CI})^3$	1.00	0.98 (0.92, 1.04)	1.01 (0.95, 1.08)	1.06 (0.99, 1.13)	1.14 (1.06, 1.23)	< 0.0001
Cancer						
No. of deaths/PY	829/158,384	771/159,536	757/158,934	793/158,629	752/158,465	—
HR $(95\% \text{ CI})^2$	1.00	0.99 (0.90, 1.09)	1.04 (0.94, 1.14)	1.15 (1.04, 1.27)	1.17 (1.06, 1.30)	0.0001
HR $(95\% \text{ CI})^3$	1.00	0.95 (0.86, 1.05)	0.98 (0.89, 1.09)	1.08 (0.97, 1.19)	1.12 (0.99, 1.26)	0.013
CVD						
No. of deaths/PY	718/158,384	650/159,536	621/158,934	570/158,629	538/158,465	—
HR $(95\% \text{ CI})^2$	1.00	1.00 (0.90, 1.11)	1.05 (0.94, 1.17)	1.05 (0.93, 1.17)	1.09 (0.97, 1.23)	0.09
HR $(95\% \text{ CI})^3$	1.00	0.99 (0.89, 1.10)	1.06 (0.95, 1.18)	1.09 (0.97, 1.23)	1.23 (1.07, 1.40)	0.001
Respiratory						
No. of deaths/PY	376/158,384	372/159,536	331/158,934	269/158,629	288/158,465	_
HR (95% CI) ²	1.00	1.08 (0.93, 1.24)	1.06 (0.92, 1.23)	0.95 (0.81, 1.11)	1.15 (0.98, 1.35)	0.28
HR $(95\% \text{ CI})^3$	1.00	1.03 (0.89, 1.19)	1.01 (0.87, 1.17)	0.90 (0.76, 1.06)	1.15 (0.95, 1.38)	0.37

¹ Definition of outcomes: CVD (International Classification of Diseases version 9 codes: 394–459; n = 2626), cancer (International Classification of Diseases version 9 codes: 140–239; n = 3279); respiratory (eg, influenza, pneumonia, and chronic obstructive pulmonary disease; International Classification of Diseases version 9 codes: 480–488 and 490–496; n = 1289). CVD, cardiovascular disease; PY, person-years follow-up.

²Model 1 was adjusted for age, sex, dialect, education, and year of interview.

³Model 2 was adjusted as for model 1 and for smoking, moderate and vigorous activity, sleep, BMI, history of hypertension (except for cancer), and energy intake.

Japanese populations focused on CVD mortality, and patterns that emphasized vegetables, fish, dairy, and soy were inversely associated with CVD mortality risk (8, 9). The study in Chinese women observed an inverse association between a higher fruit-rich (also vegetable) dietary pattern and total mortality (10), whereas there was no association in the population from Bangladesh (11).

Few of these studies reported on a corresponding less-healthy dietary pattern similar to the DSM pattern in the SCHS. The unifying theme in studies that did report on this type of pattern and the SCHS DSM pattern was an emphasis on meat and refined, sweetened, and fried foods. In Western populations, there was no association in 2 studies (5, 7), no report in 2 studies (4, 6), and strong, positive association between a higher Western pattern score and all-cause mortality and each cause-specific category in the Nurses' Health study (3). In Japanese populations, this pattern was positively associated with CVD mortality in one study (8) but not in another study (9). Similarly, there was no

association between this type of pattern and mortality in Chinese women (10). Note, compared with the current study, other studies that were based in Asian populations all used lessextensive dietary assessments and had shorter follow-up times. Other than the Nurses' Health study (3), none of the studies included extensive dietary and confounder assessments, nor did they have large sample sizes or long-term follow up. Results relating to the DSM pattern from the SCHS were similar to the results from the Western pattern from the Nurses' Health study except that we observed that findings for cancer depended on smoking status, which also affected point estimates for all-cause mortality.

The overall nutritional profile of the 2 respective patterns and the interrelation between the bioactive constituents of the numerous foods is the hypothesized rationale for the observed associations (28). Indeed, the overall dietary pattern may be the most fundamental and germane unit of measurement of diet and health (28). The finding that the association between a dietary

TABLE 4

HRs (95% CIs) of DSM pattern with cancer and all-cause mortality according to smoking status in Chinese men and women: the $SCHS^{1}$

	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5	P-trend
All cause						
Never-smokers						
No. of deaths/n	1504/8679	1104/7865	964/7385	787/6826	636/6170	_
HR (95% CI)	1.00	0.94 (0.86, 1.01)	1.02 (0.93, 1.10)	1.01 (0.92, 1.11)	1.04 (0.93, 1.16)	0.27
Ever-smokers						
No. of deaths/n	742/1837	972/2652	1021/3132	1130/3691	1169/4347	_
HR (95% CI)	1.00	1.04 (0.95, 1.15)	1.04 (0.94, 1.14)	1.13 (1.03, 1.25)	1.24 (1.12, 1.38)	< 0.0001
Cancer						
Never-smokers						
No. of deaths/n	559/8679	399/7865	372/7385	307/6826	245/6170	_
HR (95% CI)	1.00	0.88 (0.77, 1.00)	0.97 (0.85, 1.11)	0.94 (0.81, 1.09)	0.93 (0.78, 1.11)	0.59
Ever-smokers						
No. of deaths/n	270/1837	372/2652	385/3132	486/3691	507/4347	_
HR (95% CI)	1.00	1.08 (0.92, 1.27)	1.03 (0.88, 1.21)	1.25 (1.07, 1.46)	1.34 (1.13, 1.59)	< 0.0001

¹Model was adjusted for age, sex, dialect, education, year of interview, moderate and vigorous activity, sleep, BMI, history of hypertension (all-cause mortality), and energy intake. For DSM diet pattern \times smoking for cancer mortality, *P*-interaction = 0.018; for DSM diet pattern \times smoking for all-cause mortality, *P*-interaction = 0.05. DSM, dim sum- and meat-rich; SCHS, Singapore Chinese Cohort Study.

pattern with a poorer nutritional profile and cancer mortality is heightened in smokers is plausible because of the pleiotropic effects smoking and dietary intake have on biological systems of humans (29, 30). Broadly, smoking alters the metabolism, thereby creating dysfunction in cellular activity and a procarcinogenesis state (29). This state would plausibly create the environment in which a poor dietary pattern may have a carcinogenic effect because of the limited ability of the biological system to adapt and maintain homeostasis in the presence of excess metabolic perturbations generated by a poor diet (16). In addition, results of previous analyses that examined the patterns (VFS and DSM) from the SCHS cohort with different chronicdisease outcomes also support the findings (13, 31-33). Last, to our knowledge, the results related to respiratory mortality are a novel contribution to the literature. Because diet is linked to overall respiratory function, pneumonia, and chronic obstructive pulmonary disease (34, 35), we hypothesized that these mortality outcomes may have been relevant to our cause-specific analysis, especially because diet in the SCHS has been linked to nonmalignant respiratory symptoms (32).

Strengths of the current study included the large, Chinese population with a unique overall diet and ample events and follow-up time. Another particular strength was the use of a foodfrequency questionnaire that was specifically developed and validated in this population. Other strengths included the high participant response rate, detailed collection of data through faceto-face interviews, thorough assessment of potential lifestyle and demographic confounders, very low level of loss of participants to follow-up, and nearly complete mortality assessment with objectively obtained records on time and cause of death. Limitations included some amount of measurement error with the dietary assessment, although this limitation would most likely have resulted in a nondifferential misclassification with respect to mortality and a likely underestimation of risk. The self-report of other lifestyle-related data may have also resulted in some misclassification and residual confounding in our models. A repeated assessment of dietary intake and other lifestyle factors would have allowed us to examine changes in respect to mortality.

In conclusion, dietary patterns are unique to the populations they are derived from, but consistencies across populations and cultures suggest that patterns with higher intake of plant-based foods, such as vegetables, fruit, soy and other legumes, whole grains, and nuts and seeds are associated with increased longevity. In contrast, patterns with higher intakes of meat and processed meat and sweetened, fried, and refined foods are associated with decreased longevity. Results from this study highlight these patterns in a unique population comprised of Chinese in Southeast Asia, and they also suggest smoking may be an important consideration when examining diet because of its influence on biological pathways relevant to health.

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