

Association of Dietary Patterns With Albuminuria and Kidney Function Decline in Older White Women: A Subgroup Analysis From the Nurses' Health Study

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Background: Dietary patterns have been linked to such chronic diseases as cardiovascular disease, but sparse data currently are available for associations between dietary patterns and microalbuminuria or kidney function decline.

Study Design: Subgroup analysis from a prospective observational cohort study.

Setting & Participants: Female participants in the Nurses' Health Study who had dietary pattern data from food frequency questionnaires returned in 1984, 1986, 1990, 1994, and 1998 and urinary albumin-creatinine ratios from 2000 (n = 3,121); estimated glomerular filtration rate (eGFR) change between 1989 and 2000 was available for 3,071.

Predictor: Prudent (higher intake of fruits, vegetables, legumes, fish, poultry, and whole grains), Western (higher intake of red and processed meats, saturated fats, and sweets), and Dietary Approach to Stop Hypertension (DASH)-style dietary patterns (also greater intake of vegetables, fruits, and whole grains).

Outcomes & Measurements: Microalbuminuria (albumin-creatinine ratio, 25-354 $\mu\text{g}/\text{mg}$) in 2000 and change in kidney function using eGFR between 1989 and 2000.

Results: After multivariable adjustment, the highest quartile of Western pattern score compared with the lowest quartile was associated directly with microalbuminuria (OR, 2.17; 95% CI, 1.18-3.66; *P* for trend = 0.01) and rapid eGFR decline $\geq 3 \text{ mL}/\text{min}/1.73 \text{ m}^2/\text{y}$ (OR, 1.77; 95% CI, 1.03-3.03). Women in the top quartile of the DASH score had decreased risk of rapid eGFR decline (OR, 0.55; 95% CI, 0.38-0.80), but no association with microalbuminuria. These associations did not vary by diabetes status. The prudent dietary pattern was not associated with microalbuminuria or eGFR decline.

Limitations: Study cohort included primarily older white women and generalizability of results would benefit from validation in nonwhites and men.

Conclusions: A Western dietary pattern is associated with a significantly increased odds of microalbuminuria and rapid kidney function decrease, whereas a DASH-style dietary pattern may be protective against rapid eGFR decline.

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INDEX WORDS: Albuminuria; dietary patterns; estimated glomerular filtration rate.

The presence of microalbuminuria and moderately decreased kidney filtration function are powerful predictors of cardiovascular disease¹⁻³ and mortality,^{3,4} but there are limited data about how diet, an important modifiable risk factor, might be associated with microalbuminuria or kidney function decrease. In particular, the influence of dietary patterns over time on the kidney is not well defined. Whereas traditional nutritional epidemiology has focused on individual nutrients or foods, perhaps their additive or interactive influence may be observed better when overall diet patterns are considered for incident chronic diseases. In addition to the ability to capture potential synergy between foods and nutrients, dietary patterns also may allow for easier translation into practical dietary advice because people eat many different foods in combination.⁵ Furthermore, classifying individuals according to their eating pattern can yield a larger contrast between exposure groups than analyses based on multiple single nutrients or foods, which can be influenced by collinearity.

One previously published study analyzed dietary patterns and albuminuria. The Multiethnic Study of Atherosclerosis (MESA) Study reported that a diet pattern rich in whole grains, fruit, and low-fat dairy foods was associated with lower albumin-creatinine ratios (ACRs).⁶ We therefore investigated the associations between dietary patterns and the presence of microalbuminuria or estimated glomerular filtration rate (eGFR) decline in

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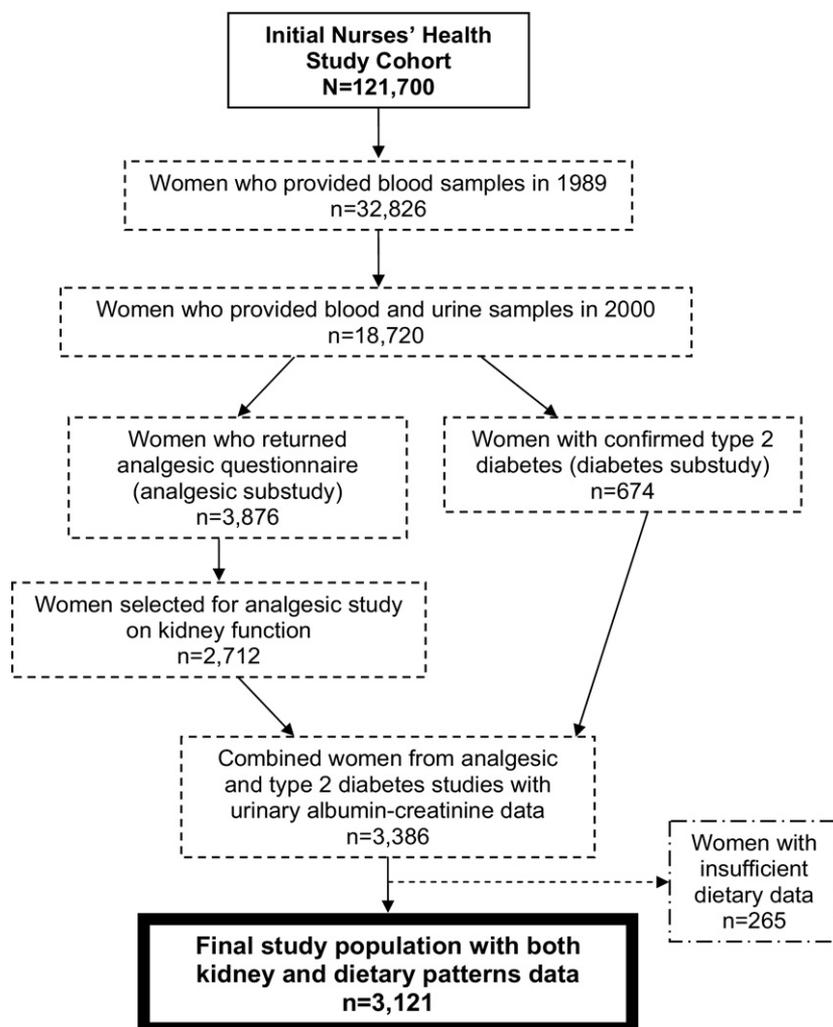


Figure 1. Inclusion and exclusion criteria for Nurses' Health Study women in this analysis of dietary patterns with albuminuria and kidney function decrease.

3,121 women participating in the Nurses' Health Study (NHS). We hypothesized that healthier eating patterns, measured using the prudent or DASH (Dietary Approach to Hypertension)-style dietary patterns, would be associated inversely, whereas the Western dietary pattern would be associated directly with microalbuminuria and eGFR decline.

METHODS

Study Design

The NHS was initiated in 1976 with the enrollment of 121,700 US female nurses aged 30-55 years. This cohort is followed up through mailed biennial questionnaires related to lifestyle factors and health outcomes. Between 1989 and 1990, a total of 32,826 participants provided blood samples that were shipped on ice by overnight delivery and stored at -130°C as previously described.⁷ In 2000, a total of 18,720 of these participants submitted second blood and spot urine specimens. Participants who did and did not return blood samples were similar in terms of demographics and lifestyle characteristics.

The NHS women in this investigation were participants in substudies of analgesic use and kidney function⁸ or type 2 diabetes and kidney function. Women in the analgesic study had submitted plasma

in 1989 and 2000 and were sent supplemental questionnaires to obtain detailed information regarding lifetime analgesic use. In total, 3,876 women returned the analgesic questionnaires. There were 2,712 women selected, with oversampling of those from the highest levels of lifetime analgesic consumption. For the type 2 diabetes substudy, we included 674 women who had submitted biological samples and had reported a diagnosis of diabetes that was confirmed using a diabetes supplemental questionnaire. The total number of women with diabetes was 694.

We included women who had cumulative average dietary pattern data available and who submitted a urine specimen in 2000 ($n = 3,121$; Fig 1). Most of these women ($n = 3,071$) also had plasma creatinine measured in samples collected in 1989 and 2000, which allowed us to examine eGFR change over 11 years.

Dietary Assessment

We used information collected from the 1984, 1986, 1990, 1994, and 1998 semiquantitative food frequency questionnaires (FFQs; Fig 2). The FFQs were designed to assess average food intake during the preceding year, and each has approximately 116 items. A standard portion size and 9 possible frequency-of-consumption responses, ranging from "never or less than once per month" to "6 or more times per day" were given for each food item. Total energy and nutrient intake was calculated by summing energy or nutrients from all foods. Previous validation studies in members of the NHS

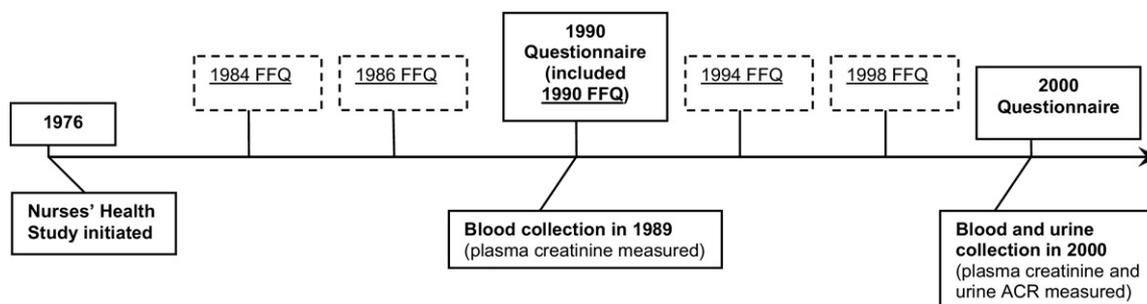


Figure 2. Timeline of questionnaire and biological sample data collection in the Nurses' Health Study for these analyses. Questionnaires are administered every 2 years beginning in 1976, but only questionnaire data for nondietary covariates from years used for this study (1990 and 2000) are shown. Food frequency questionnaires (FFQs) asking about diet during the previous 12 months were administered in 1984, 1986, 1990, 1994, and 1998. Abbreviation: ACR, albumin-creatinine ratio. Reproduced from Lin et al²⁸ with permission of the American Society of Nephrology.

showed a correlation coefficient of 0.66 between assessment using the FFQ and multiple weeks of food records completed during the preceding year.⁹

Diet Pattern Indexes

We have previously identified 2 major diet patterns using the statistical procedure factor analysis (principal components).¹⁰ Briefly, foods from the FFQ first were classified into 38 food groups based on similar nutrient profiles or culinary use. The principle components procedure identifies diet patterns based on correlations between these food groups. We also used an orthogonal rotation procedure that results in uncorrelated factors or patterns.¹¹ The factor score for each pattern was calculated by summing intakes of food groups weighted by their factor loadings.¹² Factor scores were standardized to have a mean of 0 and standard deviation of 1. Scores reflect how closely a participant's diet resembles each identified pattern, with higher scores representing closer resemblance.

Each woman received a factor score for each identified pattern. The first identified factor, which we called the prudent pattern, is characterized by higher intake of fruits, vegetables, whole grains, fish, and poultry. The second factor, which we called the Western pattern, is characterized by higher intake of processed and red meats, refined grains, sweets, and desserts. Factor scores generated using this approach are not correlated with each other. Factor analysis was conducted using SAS PROC FACTOR (SAS Institute Inc, www.sas.com).¹³

We also constructed the DASH score as previously detailed^{14,15} (range, 8-40 points) based on food and nutrients emphasized or minimized in the DASH diet¹⁶ focusing on 8 components: high intake of fruits, vegetables, nuts, legumes, low-fat dairy products, and whole grains and low intake of sodium, sweetened beverages, and red and processed meats.

Of note, 3 independent dietary patterns were derived based on a large number of correlated food items, which were aggregated into a small number of conceptually meaningful food patterns. Each individual receives a score for each pattern. There is only minor overlap between the Western and prudent patterns because we used an orthogonal rotation algorithm to derive the patterns (eg, correlations between them are close to zero).

Measurement of Urinary ACR

Urinary assays were performed on spot collections. Urinary creatinine was measured using a modified Jaffé method (coefficient of variation, 1.6%). Urinary albumin was measured using a solid-phase fluorescence immunoassay using the Hitachi 911 analyzer and Roche diagnostics reagents (www.roche.com) with a lower limit of detection

of 0.1 mg/L (coefficient of variation, 8.0%). Urinary ACR ≥ 25 $\mu\text{g}/\text{mg}$ was used to define the microalbuminuria threshold. This sex-specific cutoff value for women has been reported to approximate a urinary albumin excretion rate of ~ 30 mg/24 h,¹⁷ which classically is considered clinically relevant microalbuminuria. In this study, 177 women (5.7%) met the criterion for microalbuminuria (ACR, 25-355 $\mu\text{g}/\text{mg}$). There were 30 women with macroalbuminuria (ACR > 355 $\mu\text{g}/\text{mg}$; range, 393-6,234 $\mu\text{g}/\text{mg}$) who were excluded from the microalbuminuria analyses.

Measurement of Kidney Function Decrease

Plasma creatinine was analyzed using a modified kinetic Jaffé reaction (coefficient of variation, 10%). In 2007, repeated creatinine assays of 20 NHS plasma samples (with a range of 0.6-1.4 mg/dL) initially measured in 2000 showed a mean recalibration coefficient (new value/original value) of 0.97 and confirmed that plasma creatinine is stable for many years under our storage conditions.

Glomerular filtration was estimated using the 4-variable Modification of Diet in Renal Disease (MDRD) Study equation: $\text{GFR (mL/min/1.73 m}^2) = 186 \times [\text{PCr (mg/dL)}]^{-1.154} \times [\text{Age}]^{-0.203} \times [0.742 \text{ if female}] \times [1.21 \text{ if black}]$, in which PCr is plasma creatinine.¹⁸ An eGFR decline $\geq 30\%$ between 1989 and 2000 was determined a priori as a clinically significant change in kidney function and has been used in a previous analysis of kidney function decline in NHS participants.¹⁹ We also examined rapid eGFR decline, defined as ≥ 3 mL/min/1.73 m²/y, which has been used previously as a cutoff that reflects 3 times more rapid decrease than expected by normal aging.²⁰

Measurement of Covariates

Race and height initially were reported on the 1992 questionnaire. Other self-reported clinical and lifestyle variables, including weight, hypertension, smoking status, physical activity calculated using a weekly metabolic score, cardiovascular disease (angina, myocardial infarction, coronary artery bypass surgery, percutaneous coronary revascularization, or stroke), and blood pressure medication use were reported on the biennial questionnaires. Self-reported hypertension has been validated previously in a subset of this cohort through direct medical record review.²¹ In addition, we obtained self-reported blood pressure from the 1990 questionnaire.

Systolic blood pressure was reported in 9 categories (<105, 105-114, 115-124, 125-134, 135-144, 145-154, 155-164, 165-174, and ≥ 175 mm Hg), and diastolic blood pressure was reported in 7 categories (<65, 65-74, 75-84, 85-89, 90-94, 95-104, and ≥ 105 mm Hg). A participant's blood pressure was defined as the middle systolic and middle diastolic value of the reported category. Many of these

Table 1. Demographic, Clinical, and Nutrient Characteristics of NHS Participants in 2000 Stratified by Western Pattern Diet Score

	All NHS (N = 3,121)	Cumulative Averaged Western Pattern Diet Score				P for Trend
		Q1 (n = 780)	Q2 (n = 780)	Q3 (n = 781)	Q4 (n = 780)	
Age (y)	67 (62, 73)	69 (63, 74)	67 (62, 73)	66 (61, 72)	65 (59, 71)	<0.001
White (%)	97.4	96.1	97.1	98.3	98.2	0.02
Hypertension (%)	54.1	53.1	44.9	43.4	42.2	<0.001
SBP in 1990 (mm Hg)	130 (120, 140)	120 (110, 130)	130 (120, 140)	130 (120, 140)	130 (120, 140)	<0.001
DBP in 1990 (mm Hg)	80 (70, 80)	80 (70, 80)	80 (70, 80)	80 (70, 88)	80 (70, 88)	<0.001
Diabetes (%)	23.1	17.1	22.8	23.8	28.6	<0.001
High cholesterol (%)	65.0	66.3	66.3	65.4	61.8	0.2
CVD (%)	6.0	6.4	6.0	5.4	6.0	0.9
Current smoker (%)	5.8	3.0	4.6	5.8	9.7	<0.001
Ever smoker (%)	52.8	52.8	54.6	51.5	52.2	0.6
Alcohol intake (g/d)	1.7 (0.2, 7.0)	1.9 (0.2, 7.1)	2.1 (0.4, 8.0)	1.9 (0.2, 7.4)	1.2 (0.0, 5.7)	0.2
Activity level (METs/wk)	11.4 (3.6, 25.2)	14.0 (4.3, 27.9)	12.7 (4.1, 26.0)	11.5 (4.0, 24.6)	8.1 (2.4, 20.8)	<0.001
BMI (kg/m ²)	26.4 (23.0, 30.2)	25.0 (22.3, 28.7)	26.4 (23.3, 30.0)	26.6 (23.2, 30.1)	27.5 (23.5, 32.0)	<0.001
Aspirin use lifetime (g/d)	748 (98, 3,169)	414 (33, 2,438)	748 (82, 3,006)	813 (98, 3,656)	975 (98, 3,656)	0.01
NSAID use lifetime (g/d)	60 (10, 1,000)	20 (10, 500)	50 (10, 900)	100 (20, 1,200)	100 (20, 1,500)	0.06
Acetaminophen use through 1999 (g/d)	98 (33, 1,138)	81 (33, 650)	98 (33, 98)	98 (33, 1,138)	260 (33, 2,438)	0.001
ACEi or ARB use (%)	21.3	18.7	20.3	23.9	22.2	0.06
Cholesterol-lowering medication (ever used by 2000, %)	28.8	32.2	27.3	28.6	27.2	0.1
Plasma Cr in 2000 (mg/dL)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.9
eGFR in 2000 (mL/min/ 1.73 m ²)	76 (65, 88)	76 (65, 88)	76 (65, 87)	76 (65, 88)	76 (66, 89)	0.9
Urinary ACR (μ g/mg)	3.4 (2.0, 6.2)	3.3 (2.1, 6.2)	3.3 (1.9, 6.0)	3.4 (1.9, 6.2)	3.4 (2.0, 6.1)	0.9
Dietary intake						
Calorie intake (kcal/d)	1,726 (1,468, 2,020)	1,413 (1,230, 1,626)	1,607 (1,408, 1,805)	1,800 (1,605, 1,996)	2,153 (1,923, 2,392)	<0.001
Total protein (g/d)	74 (68, 80)	76 (69, 83)	75 (69, 81)	73 (68, 79)	72 (66, 77)	<0.001
Animal protein (g/d)	53 (46, 59)	54 (47, 61)	53 (47, 59)	52 (46, 58)	51 (46, 58)	<0.001
Vegetable protein (g/d)	21 (19, 23)	22 (20, 25)	21 (19, 23)	21 (19, 23)	20 (18, 22)	<0.001
Total fat (g/d)	56 (50, 61)	49 (44, 55)	54 (50, 59)	57 (53, 62)	60 (56, 65)	<0.001
Animal fat (g/d)	30 (25, 34)	26 (22, 30)	29 (25, 33)	31 (27, 35)	33 (29, 37)	<0.001
Sodium (mg/d)	2,001 (1,801, 2,227)	1,907 (1,718, 2,116)	1,981 (1,781, 2,181)	2,033 (1,827, 2,262)	2,085 (1,888, 2,325)	<0.001
Beta-carotene (mg/d)	4,406 (3,281, 5,930)	5,763 (4,198, 7,522)	4,651 (3,499, 5,851)	4,031 (3,216, 5,344)	3,620 (2,732, 4,727)	<0.001
Median Western diet pattern score	-0.1 (-0.6, 0.4)	-0.9 (-1.1, -0.7)	-0.3 (-0.5, -0.2)	0.1 (0.0, 0.3)	0.9 (0.7, 1.3)	NA

Note: Results expressed as median (25th, 75th percentile), percentage, or median (percentage). Conversion factors for units: Cr in mg/dL to μ mol/L, $\times 88.4$; eGFR in mL/min/1.73 m² to mL/s/1.73 m², $\times 0.01667$.

Abbreviations: ACEi, angiotensin-converting enzyme inhibitor; ACR, albumin-creatinine ratio; ARB, angiotensin receptor blocker; BMI, body mass index; Cr, creatinine; CVD, cardiovascular disease; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; MET, metabolic equivalent; NA, not applicable; NHS, Nurses' Health Study; NSAID, nonsteroidal anti-inflammatory drug; Q, quartile; SBP, systolic blood pressure.

variables have been validated previously through direct medical record review.^{21,22}

Body mass index (BMI) was calculated as [weight (kg)/(height (m)²]. Questionnaire data collected closest to the year kidney function was measured (the 1988 questionnaire for eGFR decline and the 2000 questionnaire for urinary ACR) were used (Fig 2). Many of these variables have been validated previously through direct medical record review.^{21,22}

Participants report diabetes newly diagnosed by physicians on biennial questionnaires. We mailed a diabetes supplementary questionnaire to all women reporting diabetes to obtain further information about the date of diagnosis, symptoms, diagnostic tests, and treatment. We used National Diabetes Data Group criteria to define diabetes self-reported up to the 1996 biennial questionnaire²³; the American Diabetes Association diagnostic criteria for diabetes released in 1997 were used for incident cases of diabetes reported in 1998 and after.²⁴ Self-reported diagnosis of type 2 diabetes using the diabetes supple-

mentary questionnaire has been established as 98% accurate in a separate validation study through medical record review.²⁵ We considered a participant who was given a diagnosis of diabetes through the year 2000 as having diabetes.

Statistical Analyses

For albuminuria analyses, cumulative averaging for each dietary pattern using available data for 5 FFQs (1984, 1986, 1990, 1994, and 1998) was performed for each participant and divided into quartiles as the primary exposure of interest. Similarly, for analyses of eGFR decline between 1989 and 2000, cumulative average dietary patterns from 1984, 1986, and 1990 were divided into quartiles. This modeling approach used dietary pattern exposures as measured up to the time of each outcome assessment (Fig 2). A cumulative average approach was chosen because it generally reflects long-term diet and also likely decreases measurement error from intraindividual variation over time.²⁶

Table 2. Demographic, Clinical, and Nutrient Characteristics of NHS Participants in 2000 Stratified by Prudent Pattern Diet Score

	All NHS (N = 3,121)	Cumulative Averaged Prudent Pattern Diet Score				P for Trend
		Q1 (n = 780)	Q2 (n = 780)	Q3 (n = 781)	Q4 (n = 780)	
Age (y)	67 (62, 73)	65 (60, 71)	66 (61, 72)	68 (63, 73)	69 (63, 74)	<0.001
White (%)	97.4	96.4	97.9	98.4	96.9	0.05
Hypertension (%)	54.1	51.4	56.3	55.3	53.5	0.2
SBP in 1990 (mm Hg)	130 (120, 120)	120 (120, 140)	130 (120, 140)	130 (120, 140)	130 (120, 140)	0.3
DBP in 1990 (mm Hg)	80 (70, 80)	80 (70, 80)	80 (70, 88)	80 (70, 80)	80 (70, 80)	0.6
Diabetes (%)	23.1	20.1	22.1	26.5	23.6	0.02
High cholesterol (%)	65.0	61.2	64.2	65.7	68.7	0.02
CVD (%)	6.0	4.7	6.8	5.8	6.5	0.3
Current smoker (%)	5.8	11.3	6.0	2.3	3.5	<0.001
Ever smoker (%)	52.8	54.1	55.4	52.2	49.4	0.09
Alcohol intake (g/d)	1.7 (0.2, 7.0)	1.6 (0, 7.6)	1.8 (0.2, 7.4)	2.0 (0.2, 7.4)	1.6 (0.2, 5.9)	0.01
Activity level (METs/wk)	11.4 (3.6, 25.2)	8.2 (2.3, 18.5)	10.2 (3.4, 22.1)	13.2 (4.0, 26.1)	16.2 (6.7, 32.4)	<0.001
BMI (kg/m ²)	26.4 (23.0, 30.2)	26.1 (23.0, 30.1)	26.1 (22.9, 30.1)	26.6 (23.0, 30.3)	26.3 (23.0, 30.4)	0.9
Aspirin use lifetime (g/d)	748 (98, 3,169)	650 (98, 2,454)	748 (98, 3,169)	731 (98, 3,656)	975 (98, 3,169)	0.9
NSAID use lifetime (g/d)	60 (10, 1,000)	60 (10, 900)	60 (10, 1,200)	60 (10, 900)	60 (10, 900)	0.9
Acetaminophen use through 1999 (g/d)	98 (33, 1,138)	98 (33, 1,138)	98 (33, 1,138)	98 (33, 1,138)	98 (33, 975)	0.4
ACEi or ARB medication use (%)	21.3	19.2	22.4	22.0	21.4	0.4
Cholesterol-lowering medication (ever used by 2000, %)	28.8	28.2	28.3	28.3	30.4	0.7
Plasma Cr in 2000 (mg/dL)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.02
eGFR in 2000 (mL/min/1.73 m ²)	76 (65, 88)	76 (66, 89)	75 (63, 86)	75 (64, 87)	77 (66, 90)	0.01
Urinary ACR (μg/mg)	3.4 (2.0, 6.2)	3.2 (1.9, 5.7)	3.3 (2.0, 5.8)	3.4 (2.1, 6.4)	3.6 (2.1, 6.7)	0.9
Dietary intake						
Calorie intake (kcal/d)	1,726 (1,468, 2,020)	1,474 (1,256, 1,798)	1,661 (1,458, 1,929)	1,750 (1,555, 2,005)	1,989 (1,708, 2,273)	<0.001
Total protein (g/d)	74 (68, 80)	70 (63, 75)	73 (68, 78)	75 (69, 81)	78 (72, 84)	<0.001
Animal protein (g/d)	53 (46, 59)	51 (44, 57)	53 (47, 59)	54 (47, 60)	54 (48, 61)	<0.001
Vegetable protein (g/d)	21 (19, 23)	19 (17, 21)	20 (19, 22)	22 (20, 23)	23 (21, 25)	<0.001
Total fat (g/d)	56 (50, 61)	60 (54, 65)	57 (52, 61)	55 (50, 59)	51 (46, 57)	<0.001
Animal fat (g/d)	30 (25, 34)	33 (29, 38)	31 (27, 35)	29 (25, 33)	27 (22, 30)	<0.001
Sodium (mg/d)	2,001 (1,801, 2,227)	1,994 (1,782, 2,230)	1,988 (1,790, 2,204)	2,013 (1,826, 2,214)	2,018 (1,806, 2,253)	0.4
Beta-carotene (mg/d)	4,406 (3,281, 5,930)	2,944 (2,215, 3,840)	3,888 (3,190, 4,840)	4,856 (4,054, 5,991)	6,239 (5,073, 7,822)	<0.001
Median prudent diet pattern score	-0.004 (-0.5, 0.5)	-0.8 (-0.3, -0.1)	-0.2 (-0.3, -0.1)	0.2 (0.1, 0.4)	0.9 (0.7, 1.3)	NA

Note: Results expressed as median (25th, 75th percentile), percentage, or median (percentage). Conversion factors for units: Cr in mg/dL to μmol/L, ×88.4; eGFR in mL/min/1.73 m² to mL/s/1.73 m², ×0.01667.

Abbreviations: ACEi, angiotensin-converting enzyme inhibitor; ACR, albumin-creatinine ratio; ARB, angiotensin receptor blocker; BMI, body mass index; Cr, creatinine; CVD, cardiovascular disease; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; NA, not applicable; NHS, Nurses' Health Study; NSAID, nonsteroidal anti-inflammatory drug; Q, quartile; SBP, systolic blood pressure.

Covariates included in adjusted models were determined from questionnaire data up to the nearest time of measurement of albuminuria or first eGFR.

Wilcoxon rank sum and χ^2 tests were used to test for differences between groups as appropriate. Primary analysis was performed with outcomes of microalbuminuria using a sex-specific category of ACR of 25-355 μg/mg or presence of eGFR decline ≥30% or ≥3 mL/min/1.73 m²/y. Exposures of interest were quartiles of each dietary pattern, with the lowest quartile as the referent category. Logistic regression was used to model associations between quartiles of diet scores and presence of microalbuminuria, eGFR decline ≥30%, or decline ≥3 mL/min/1.73 m²/y between 1989 and 2000. In all analyses of microalbuminuria and eGFR decline, adjustment for alcohol intake and eGFR in 1989 did not influence results; therefore,

these covariates were not included in multivariable-adjusted models.

All analyses were performed using SAS software, version 9.1. This study was approved by the Partners' Healthcare Brigham and Women's Hospital Human Research Committee Institutional Review Board.

RESULTS

Study Participants and Dietary Pattern Assessment

Characteristics of these 3,121 women in 2000 are listed in Tables 1-3. Median age was 67 years, 97% were white, 54% had hypertension, and 23% had diabetes. The Western and prudent dietary patterns

Table 3. Demographic, Clinical, and Nutrient Characteristics of Participants in the NHS in the Year 2000 Stratified by DASH-Style Pattern Score

	All NHS (N = 3,121)	Cumulative Averaged DASH-Style Pattern Score				P for Trend
		Q1 (n = 780)	Q2 (n = 780)	Q3 (n = 781)	Q4 (n = 780)	
Age (y)	67 (62, 73)	64 (61, 73)	66 (61, 73)	68 (63, 73)	70 (64, 74)	<0.001
White (%)	97.4	96.3	98.0	98.0	97.1	0.08
Hypertension (%)	54.1	56.5	56.3	56.7	48.3	0.001
SBP in 1990 (mm Hg)	130 (120, 140)	130 (120, 140)	130 (120, 140)	130 (120, 140)	120 (120, 140)	0.05
DBP in 1990 (mm Hg)	80 (70, 80)	80 (70, 88)	80 (70, 88)	80 (70, 80)	80 (70, 80)	<0.001
Diabetes (%)	23.1	24.6	24.1	26.1	20.3	0.04
High cholesterol (%)	65.0	65.0	63.3	66.1	66.4	0.6
CVD (%)	6.0	6.8	6.3	6.6	5.3	0.6
Current smoker (%)	5.8	11.6	6.0	3.3	2.2	<0.001
Ever smoker (%)	52.8	56.3	57.7	50.3	48.4	<0.001
Alcohol intake (g/d)	1.7 (0.18, 7.0)	1.4 (0, 6.7)	2.0 (0.3, 8.0)	1.6 (0.2, 6.5)	1.8 (0.2, 6.5)	0.03
Activity level (METs/wk)	11.4 (3.6, 25.2)	7.7 (1.9, 17.7)	9.8 (3.1, 22.3)	12.7 (4.4, 27.0)	17.7 (7.7, 32.7)	<0.001
BMI (kg/m ²)	26.4 (23.0, 30.2)	27.1 (23.5, 31.4)	27.1 (23.5, 31.4)	26.3 (23.0, 30.5)	24.9 (22.3, 28.4)	<0.001
Aspirin use lifetime (g)	748 (98, 3,169)	813 (98, 3,169)	748 (98, 3,169)	569 (98, 2,844)	748 (81, 3,169)	0.4
NSAID use lifetime (g)	60 (10, 1,000)	60 (10, 1,200)	60 (10, 1,200)	60 (10, 900)	50 (10, 760)	0.2
Acetaminophen use lifetime (g)	98 (33, 1,138)	163 (33, 1,463)	98 (33, 1,235)	98 (33, 975)	98 (33, 650)	0.001
ACEi or ARB medication use (%)	21.3	22.9	22.3	23.9	17.2	0.004
Cholesterol-lowering medication (ever used by 2000, %)	28.8	30.9	27.9	30.4	28.0	0.4
Plasma Cr in 2000 (mg/dL)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)	0.03
eGFR in 2000 (mL/min/1.73 m ²)	76 (65, 88)	77 (65, 89)	75 (64, 88)	75 (64, 86)	77 (66, 89)	0.2
Urinary ACR (μ g/mg)	3.4 (2.0, 6.2)	3.4 (1.9, 6.2)	3.6 (1.9, 5.6)	3.4 (2.1, 6.8)	3.6 (2.1, 6.5)	0.7
Dietary intake						
Calorie intake (kcal/d)	1,726 (1,468, 2,019)	1,560 (1,301, 1,902)	1,685 (1,447, 1,978)	1,745 (1,496, 2,019)	1,876 (1,628, 2,157)	<0.001
Total protein (g/d)	74 (68, 80)	71 (65, 77)	73 (67, 79)	75 (69, 81)	77 (70, 83)	<0.001
Animal protein (g/d)	53 (46, 59)	52 (46, 58)	53 (47, 59)	53 (47, 59)	53 (46, 60)	0.1
Vegetable protein (g/d)	21 (19, 23)	19 (17, 21)	20 (19, 22)	22 (20, 23)	23 (21, 25)	<0.001
Total fat (g/d)	56 (50, 61)	61 (56, 65)	57 (53, 61)	54 (50, 59)	49 (45, 54)	<0.001
Animal fat (g/d)	30 (25, 34)	34 (30, 39)	31 (27, 35)	29 (25, 33)	25 (22, 29)	<0.001
Sodium (mg/d)	2,001 (1,801, 2,227)	2,084 (1,893, 3,276)	2,031 (1,831, 2,274)	1,964 (1,788, 2,165)	1,926 (1,730, 2,111)	<0.001
Beta-carotene (mg/d)	4,406 (3,281, 5,930)	3,109 (2,327, 4,087)	4,037 (3,225, 5,226)	4,619 (3,730, 6,011)	5,979 (4,833, 7,531)	<0.001
Median DASH-style pattern score	25 (22, 27)	20 (18, 21)	23 (22, 24)	26 (25, 27)	29 (28, 31)	NA

Note: Results expressed as median (25th, 75th percentile), percentage, or median (percentage). Conversion factors for units: Cr in mg/dL to μ mol/L, $\times 88.4$; eGFR in mL/min/1.73 m² to mL/s/1.73 m², $\times 0.01667$.

Abbreviations: ACEi, angiotensin-converting enzyme inhibitor; ACR, albumin-creatinine ratio; ARB, angiotensin receptor blocker; BMI, body mass index; Cr, creatinine; CVD, cardiovascular disease; DASH, Dietary Approach to Hypertension; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; MET, metabolic equivalent; NA, not applicable; NHS, Nurses' Health Study; NSAID, nonsteroidal anti-inflammatory drug; Q, quartile; SBP, systolic blood pressure.

had a weak but statistically significant inverse correlation ($r = -0.07$; $P < 0.001$). DASH score correlated directly with the prudent pattern ($r = 0.76$; $P < 0.001$) and inversely with the Western pattern ($r = -0.30$; $P < 0.001$). Cumulative average dietary pattern scores highly correlated comparing 1990 with 1998 values (all $r > 0.94$; $P < 0.001$), suggesting that dietary patterns were relatively unchanged in these women over time. Participant characteristics stratified by quartiles of dietary pattern scores are listed in Tables 1-3.

Microalbuminuria

The 177 women (5.7%) who met the criterion for microalbuminuria (ACR, 25-355 μ g/mg) were more

likely to be older and have hypertension, diabetes, cardiovascular disease, higher BMI, and lower activity levels (Tables 1-3). In age- and energy-adjusted models, the Western pattern diet was associated directly with microalbuminuria (odds ratio [OR], 3.55 [95% confidence interval (CI), 2.03-6.20] for the fourth vs first quartile), whereas DASH score was associated inversely with microalbuminuria (OR, 0.53 [95% CI, 0.33-0.84] for the fourth vs first quartile). There was no association between the prudent diet pattern and microalbuminuria. After multivariable adjustment, the association between Western diet and microalbuminuria remained significant (OR, 2.17 [95% CI, 1.18-3.96] for the fourth vs first quartile), but not the DASH-style diet (Table 4). BMI, diabetes, and

Table 4. Odds Ratios for Microalbuminuria by Quartiles of Diet Pattern Scores

	Q1	Q2	Q3	Q4
Western				
Age and energy intake adjusted	1.00 (reference)	1.29 (0.81-2.05)	1.51 (0.91-2.51)	3.55 (2.03-6.20)
Multivariable ^a	1.00 (reference)	1.11 (0.68-1.81)	1.12 (0.66-1.92)	2.17 (1.18-3.96)
Prudent				
Age and energy intake adjusted	1.00 (reference)	0.98 (0.63-1.53)	1.11 (0.71-1.74)	0.94 (0.58-1.52)
Multivariable ^a	1.00 (reference)	0.89 (0.57-1.42)	1.05 (0.66-1.67)	0.97 (0.58-1.61)
DASH-style				
Age and energy intake adjusted	1.00 (reference)	0.82 (0.54-1.23)	0.72 (0.47-1.10)	0.53 (0.33-0.84)
Multivariable ^a	1.00 (reference)	0.80 (0.52-1.23)	0.77 (0.49-1.21)	0.71 (0.44-1.14)

Note: Microalbuminuria defined as an albumin-creatinine ratio of 25-355 $\mu\text{g}/\text{mg}$. The 95% confidence intervals are shown in parentheses.

Abbreviations: DASH, Dietary Approach to Hypertension; Q, quartile.

^aAdjusted for age, hypertension, body mass index, physical activity (METs/wk), energy intake, cigarette smoking, diabetes, cardiovascular disease, and angiotensin-converting enzyme-inhibitor/angiotensin receptor blocker medication use (alcohol intake and estimated glomerular filtration rate did not influence results and were removed).

physical activity level appeared to be the major confounders in the association between dietary patterns and microalbuminuria.

Stratifying analyses by diabetes status also did not meaningfully change results. When the outcome of $\text{ACR} \geq 25 \mu\text{g}/\text{mg}$ was examined, results also were not meaningfully changed.

eGFR Decline

There were 346 (11.3%) women who experienced an eGFR decline $\geq 30\%$ between 1989 and 2000; this reflected a median increase in plasma creatinine level of 0.33 mg/dL. There were 230 (7.5%) women with eGFR decline $\geq 3 \text{ mL}/\text{min}/1.73 \text{ m}^2/\text{y}$, representing a median eGFR decline rate of 3.8 $\text{mL}/\text{min}/1.73 \text{ m}^2/\text{y}$ and a median increase in plasma creatinine level of 0.34 mg/dL.

There were no significant associations between the prudent pattern and eGFR decline. After multivariable adjustment, the Western pattern was not significantly associated with eGFR decline $\geq 30\%$ (Table 5). DASH score maintained a significant inverse association with eGFR decline $\geq 30\%$ after adjustment (OR, 0.55 [95% CI, 0.38-0.80] comparing top with bottom quartiles). Results were not meaningfully different when adjusted for analgesic medication use, high cholesterol level or lipid-lowering medication use, or diabetes duration (Table 3). These associations for Western diet and DASH score did not vary by baseline eGFR $< 80 \text{ mL}/\text{min}/1.73 \text{ m}^2$ or diabetes status because all *P* values for interaction terms were nonsignificant. Results using $\geq 3 \text{ mL}/\text{min}/1.73 \text{ m}^2/\text{y}$ eGFR decline were virtually identical except that the highest quartile of the Western pattern remained statistically significantly associated after multivariable adjustment (OR, 1.77; 95% CI, 1.03-3.03).

Our results for kidney function decrease were unchanged when the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation was used to estimate GFR²⁷ or if change in weight or BMI was included as a covariate in the adjusted models.

DISCUSSION

Our data suggest that dietary patterns are associated with microalbuminuria and kidney function decrease in middle-aged and older women. Women in the highest quartile of the Western pattern had a significant 2-fold increased odds of having microalbuminuria and experiencing more rapid eGFR decline $\geq 3 \text{ mL}/\text{min}/1.73 \text{ m}^2/\text{y}$. Moreover, a DASH-style pattern appears to decline risk by $>40\%$ for eGFR decline $\geq 30\%$ over 11 years.

We previously reported that higher dietary intake of animal fat was associated with the presence of microalbuminuria, whereas higher sodium intake was associated directly and higher beta-carotene intake was associated inversely with faster eGFR decline over 11 years.²⁸ The present study provides additional information regarding decline in kidney function and dietary patterns that may be interpreted more easily by the general public.

The lack of association with the prudent pattern with renal outcomes despite its correlation with the DASH pattern may be due to the different weights given to food groups used to derive each score. Our results suggest that DASH score may better reflect food groups most relevant to microalbuminuria and eGFR decline. Of note, we do not believe the DASH-style diet is merely a surrogate for low sodium intake because we did not find significant associations of DASH scores with 24-hour urinary sodium excretion in a subset of $\sim 1,200$ NHS 1 women with these

Table 5. Odds Ratios for eGFR Decline $\geq 30\%$ by Quartiles of Diet Pattern Scores

	Q1	Q2	Q3	Q4
Western				
Age and energy intake adjusted	1.00 (reference)	1.37 (0.98-1.93)	1.84 (1.29-2.64)	1.95 (1.27-2.97)
Multivariable ^a	1.00 (reference)	1.22 (0.87-1.73)	1.57 (1.08-2.28)	1.48 (0.95-2.30)
Multivariable + analgesic medication use ^b	1.00 (reference)	1.22 (0.86-1.72)	1.52 (1.04-2.20)	1.40 (0.90-2.19)
Multivariable + high cholesterol or lipid-lowering drug	1.00 (reference)	1.23 (0.87-1.73)	1.57 (1.08-2.26)	1.46 (0.94-2.28)
Multivariable + diabetes duration	1.00 (reference)	1.22 (0.86-1.72)	1.58 (1.09-2.29)	1.46 (0.94-2.28)
Prudent				
Age and energy intake adjusted	1.00 (reference)	1.44 (1.05-1.97)	1.06 (0.76-1.48)	0.78 (0.53-1.13)
Multivariable ^a	1.00 (reference)	1.43 (1.04-1.98)	1.07 (0.76-1.51)	0.81 (0.55-1.19)
Multivariable + analgesic medication use ^b	1.00 (reference)	1.44 (1.04-1.98)	1.10 (0.78-1.56)	0.82 (0.56-1.21)
Multivariable + high cholesterol or lipid-lowering drug	1.00 (reference)	1.45 (1.05-2.00)	1.09 (0.77-1.54)	0.84 (0.57-1.23)
Multivariable + diabetes duration	1.00 (reference)	1.44 (1.04-1.98)	1.07 (0.76-1.51)	0.81 (0.55-1.19)
DASH-style				
Age and energy intake adjusted	1.00 (reference)	0.87 (0.64-1.18)	0.79 (0.58-1.09)	0.51 (0.36-0.72)
Multivariable ^a	1.00 (reference)	0.86 (0.63-1.17)	0.79 (0.57-1.09)	0.55 (0.38-0.80)
Multivariable + analgesic medication use ^b	1.00 (reference)	0.88 (0.65-1.21)	0.82 (0.60-1.13)	0.57 (0.39-0.83)
Multivariable + high cholesterol or lipid lowering drug	1.00 (reference)	0.86 (0.63-1.18)	0.79 (0.58-1.09)	0.55 (0.38-0.79)
Multivariable + diabetes duration	1.00 (reference)	0.87 (0.64-1.18)	0.79 (0.58-1.09)	0.55 (0.38-0.80)

Abbreviations: eGFR, estimated glomerular filtration rate; DASH, Dietary Approach to Hypertension; NSAIDs, nonsteroidal anti-inflammatory drugs; Q, quartile.

^aAdjusted for age, hypertension, body mass index, physical activity (METs/week), energy intake, cigarette smoking, diabetes, cardiovascular disease, and angiotensin-converting enzyme-inhibitor/angiotensin receptor blocker medication use (alcohol intake and estimated glomerular filtration rate did not influence results and were removed).

^bWe mailed a supplementary questionnaire in 1999 to collect detailed information about the current use of each of the 3 analgesic medication classes (aspirin, nonsteroidal anti-inflammatory drugs, and acetaminophen), including frequency in days per month, tablets per day, tablet dosage, brand, and indication for current use. The questionnaire also asked about total consumption in 2 periods: the past 10 years and before 1990. The total number of tablets taken in those 2 periods was collected in 11 categories: none, 1-100, 101-500, 501-1,000, 1,001-1,500, 1,501-3,000, 3,001-5,000, 5,001-10,000, 10,001-15,000, 15,001-20,000, and 20,001 or more. We used the combined total from the 2 periods by adding the midpoints of the categories. We converted number of tablets to lifetime intake in grams by multiplying the total number of tablets (the midpoint of each category) by the most common dosage of each analgesic (aspirin and acetaminophen, 325 mg; NSAIDs, 200 mg).

data.²⁹ Although dietary protein (particularly red meat) intake may potentially affect plasma creatinine concentrations, we previously have ascertained that all nutrient intake, including total and subtypes of protein, varied by $\leq 16\%$ over time in this cohort of women.²⁸ Therefore, changes in dietary protein intake are unlikely to explain change in eGFR.

We hypothesize that inflammation might be one possible pathophysiologic link between dietary patterns and microalbuminuria. A number of studies have reported significant direct associations between markers of inflammation and higher albuminuria. For example, in a recent publication from the National Health and Nutrition Examination Surveys 1999-2004, each 1-mg/dL increase in C-reactive protein (CRP) level was associated independently with a 1.02 (95% CI, 1.01-1.02; $P = 0.0003$) OR for the presence of microalbuminuria in this large nationally representative cohort.³⁰ Furthermore, the highest tertile of ICAM-1 (inter-cellular adhesion molecule 1), a vascular endothelial transmembrane glycoprotein upregulated by inflammation, also previously has been associated independently with the development of incident

sustained microalbuminuria (OR, 1.67; P for trend = 0.03) in patient with type 1 diabetes.³¹

Previous work on dietary patterns and inflammation reported that the Western diet pattern showed significant direct correlations between CRP, ICAM-1, and VCAM-1 (vascular cell adhesion molecule 1) levels in multivariable models that included adjustment for BMI in NHS women,³² as well as Health Professionals Follow-Up Study (HPFS) men.³³ The strong associations between Western pattern and inflammatory markers may explain the significant direct association of the Western dietary pattern with microalbuminuria. In these previous studies, the prudent pattern was not associated with inflammatory marker levels after multivariable adjustment, which may be consistent with the lack of association between the prudent pattern and microalbuminuria. No data currently appear to be available for DASH score and markers of inflammation.

An investigation in the MESA that included almost 5,000 ethnically diverse men and women similarly has reported that a dietary pattern rich in whole grains, fruit, and low-fat dairy foods was associated with

lower urinary ACR (20% lower ACR across quintiles, P for trend = 0.004), whereas nondairy animal-based food intake was associated directly (11% higher ACR across quintiles, P for trend = 0.03).⁶ The MESA cohort also has reported that diets high in whole grains, fruits or vegetables, and fish are associated inversely with markers of inflammation, including CRP and soluble ICAM-1 levels, whereas a diet pattern high in fats and processed meats was associated directly with markers of inflammation, including CRP.³⁴ Data from other cohorts provide external validation for our findings regarding diet patterns, inflammation, and albuminuria in the NHS cohort.

There are no published data for dietary patterns and eGFR decline, but recent investigations have suggested that markers of inflammation,³⁵ including CRP,³⁶ are associated with faster eGFR decline. Therefore, because inflammatory biomarkers have been proposed to be potential mediators for associations observed between diet and cardiovascular disease,³⁷ we propose that inflammation also may be a factor in associations between diet and eGFR decline.

Notable strengths of this investigation include the relatively large number of women with data for both albuminuria and eGFR decline. Change in eGFR was assessed during an 11-year period, and repeated measures of diet intake over 14 years were performed. The substantial numbers of covariates, most of which have been validated extensively in this large and well-established longitudinal cohort, are additional assets in these analyses.

Limitations of this study include the predominant white population of older women; therefore, results may not necessarily be generalizable to men or non-white populations. However, similar results in the analysis of dietary patterns and albuminuria in the ethnically diverse MESA cohort would suggest that the associations may not vary substantially by race or ethnicity.⁶ In addition, no data for change in urinary ACR were available in our participants, and albuminuria analyses are cross-sectional. Markers of inflammation are not available in this subcohort of women. The presence of residual confounding also is possible, as in any observational study. Measurements of glycemic control to define glucose intolerance or prediabetes were not available for most of these women, although we conservatively considered a participant with a diagnosis of diabetes up to 10 years after the initial blood draw to address this issue. The possibility of survival bias is present because women who died before 2000 would not have been included in this study; however, we would expect this to bias results toward the null, whereas statistically significant associations between dietary patterns and microalbuminuria and eGFR decline were observed.

In conclusion, a Western pattern diet was associated with a 2-fold higher OR for microalbuminuria and increased risk of rapid eGFR decline (≥ 3 mL/min/1.73 m²/y). A DASH-style diet was associated with an almost 50% declined risk of eGFR decline. Therefore, diets higher in fruits, vegetables, and whole grains, but lower in meat and sweets, may be protective against eGFR decline. Future directions of interest include validation of these findings in other cohorts and examining how individual foods might influence microalbuminuria and eGFR decline.

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REFERENCES

1. Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. *N Engl J Med*. 2004;351(13):1296-1305.
2. Anavekar NS, McMurray JJ, Velazquez EJ, et al. Relation between renal dysfunction and cardiovascular outcomes after myocardial infarction. *N Engl J Med*. 2004;351(13):1285-1295.
3. Wachtell K, Ibsen H, Olsen MH, et al. Albuminuria and cardiovascular risk in hypertensive patients with left ventricular hypertrophy: the LIFE Study. *Ann Intern Med*. 2003;139(11):901-906.
4. Solomon SD, Lin J, Solomon CG, et al. Influence of albuminuria on cardiovascular risk in patients with stable coronary artery disease. *Circulation*. 2007;116(23):2687-2693.
5. Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. *Curr Opin Lipidol*. 2002;13(1):3-9.
6. Nettleton JA, Steffen LM, Palmas W, Burke GL, Jacobs DR Jr. Associations between microalbuminuria and animal foods, plant foods, and dietary patterns in the Multiethnic Study of Atherosclerosis. *Am J Clin Nutr*. 2008;87(6):1825-1836.
7. Schulze MB, Hoffmann K, Manson JE, et al. Dietary pattern, inflammation, and incidence of type 2 diabetes in women. *Am J Clin Nutr*. 2005;82(3):675-684; quiz 714-675.
8. Knight EL, Stampfer MJ, Hankinson SE, Spiegelman D, Curhan GC. The impact of protein intake on renal function decline in women with normal renal function or mild renal insufficiency. *Ann Intern Med*. 2003;138(6):460-467.
9. Salvini S, Hunter DJ, Sampson L, et al. Food-based validation of a dietary questionnaire: the effects of week-to-week variation in food consumption. *Int J Epidemiol*. 1989;18(4):858-867.
10. Fung TT, Willett WC, Stampfer MJ, Manson JE, Hu FB. Dietary patterns and the risk of coronary heart disease in women. *Arch Intern Med*. 2001;161(15):1857-1862.
11. Kim JO, Mueller CW. *Factor Analysis: Statistical Methods and Practical Issues*. Newbury Park, CA: Sage Publications Inc; 1978.
12. Kleinbaum DG, Kupper LL, Muller KE. Variable reduction and factor analysis. *Applied Regression Analysis and Other Multivariable Methods*. Boston, MA: Duxbury Press; 1988:718-738.

13. Institute IS. *SAS/STAT User's Guide*. 6th ed. Cary, NC: SAS Institute; 1989.
14. Fung TT, Chiuvè SE, McCullough ML, Rexrode KM, Logroscino G, Hu FB. Adherence to a DASH-style diet and risk of coronary heart disease and stroke in women. *Arch Intern Med*. 2008;168(7):713-720.
15. Taylor EN, Fung TT, Curhan GC. DASH-style diet and risk of incident kidney stones. *J Am Soc Nephrol*. 2009;20(10):2253-2259.
16. *Your Guide to Lowering Your Blood Pressure With DASH*. 1st ed. Bethesda, MD: National Institutes of Health, National Heart, Lung, and Blood Institute; 2006.
17. Warram JH, Gearin G, Laffel L, Krolewski AS. Effect of duration of type I diabetes on the prevalence of stages of diabetic nephropathy defined by urinary albumin/creatinine ratio. *J Am Soc Nephrol*. 1996;7(6):930-937.
18. Stevens LA, Coresh J, Greene T, Levey AS. Assessing kidney function—measured and estimated glomerular filtration rate. *N Engl J Med*. 2006;354(23):2473-2483.
19. Curhan GC, Knight EL, Rosner B, Hankinson SE, Stampfer MJ. Lifetime nonnarcotic analgesic use and decline in renal function in women. *Arch Intern Med*. 2004;164(14):1519-1524.
20. Rifkin DE, Shlipak MG, Katz R, et al. Rapid kidney function decline and mortality risk in older adults. *Arch Intern Med*. 2008;168(20):2212-2218.
21. Hu FB, Willett WC, Colditz GA, et al. Prospective study of snoring and risk of hypertension in women. *Am J Epidemiol*. 1999;150(8):806-816.
22. Fung TT, Rexrode KM, Mantzoros CS, Manson JE, Willett WC, Hu FB. Mediterranean diet and incidence of and mortality from coronary heart disease and stroke in women. *Circulation*. 2009;119(8):1093-1100.
23. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. National Diabetes Data Group. *Diabetes*. 1979;28(12):1039-1057.
24. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care*. 1997;20(7):1183-1197.
25. Shai I, Schulze MB, Manson JE, et al. A prospective study of soluble tumor necrosis factor-alpha receptor II (sTNF-RII) and risk of coronary heart disease among women with type 2 diabetes. *Diabetes Care*. 2005;28(6):1376-1382.
26. Hu FB, Stampfer MJ, Rimm E, et al. Dietary fat and coronary heart disease: a comparison of approaches for adjusting for total energy intake and modeling repeated dietary measurements. *Am J Epidemiol*. 1999;149(6):531-540.
27. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. *Ann Intern Med*. 2009;150(9):604-612.
28. Lin J, Hu FB, Curhan GC. Associations of diet with albuminuria and kidney function decline. *Clin J Am Soc Nephrol*. 2010;5:836-843.
29. Taylor EN, Stampfer MJ, Mount DB, Curhan GC. DASH-style diet and 24-hour urine composition [published online ahead of print September 16, 2010]. *Clin J Am Soc Nephrol*.
30. Kshirsagar AV, Bombardieri AS, Bang H, et al. Association of C-reactive protein and microalbuminuria (from the National Health and Nutrition Examination Surveys, 1999 to 2004). *Am J Cardiol*. 2008;101(3):401-406.
31. Lin J, Glynn RJ, Rifai N, et al. Inflammation and progressive nephropathy in type 1 diabetes in the diabetes control and complications trial. *Diabetes Care*. 2008;31(12):2338-2343.
32. Lopez-Garcia E, Schulze MB, Fung TT, et al. Major dietary patterns are related to plasma concentrations of markers of inflammation and endothelial dysfunction. *Am J Clin Nutr*. 2004;80(4):1029-1035.
33. Fung TT, Rimm EB, Spiegelman D, et al. Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk. *Am J Clin Nutr*. 2001;73(1):61-67.
34. Nettleton JA, Steffen LM, Mayer-Davis EJ, et al. Dietary patterns are associated with biochemical markers of inflammation and endothelial activation in the Multi-Ethnic Study of Atherosclerosis (MESA). *Am J Clin Nutr*. 2006;83(6):1369-1379.
35. Bash LD, Erlinger TP, Coresh J, Marsh-Manzi J, Folsom AR, Astor BC. Inflammation, hemostasis, and the risk of kidney function decline in the Atherosclerosis Risk in Communities (ARIC) Study. *Am J Kidney Dis*. 2009;53(4):596-605.
36. Tonelli M, Sacks F, Pfeffer M, Jhangri GS, Curhan G. Biomarkers of inflammation and progression of chronic kidney disease. *Kidney Int*. 2005;68(1):237-245.
37. Lopez-Garcia E, Hu FB. Nutrition and the endothelium. *Curr Diab Rep*. 2004;4(4):253-259.