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Food Access, Chronic Kidney Disease, and Hypertension in the U.S.

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Abstract

Introduction—Greater distance to full-service supermarkets and low income may impair access to healthy diets and contribute to chronic kidney disease (CKD) and hypertension. The study aim was to determine relationships among residence in a “food desert,” low income, CKD, and blood pressure.

Methods—Adults in the 2003–2010 National Health and Nutrition Examination Survey (N=22,173) were linked to food desert data (www.ers.usda.gov) by Census Tracts. Food deserts have low median income and are further from a supermarket or large grocery store (>1 mile in urban areas, >10 miles in rural areas). Weighted regression was used to determine the association of residence in a food desert and family income with dietary intake, systolic blood pressure (SBP), and odds of CKD. Data analysis was performed in 2014–2015.

Results—Compared with those not in food deserts, participants residing in food deserts had lower levels of serum carotenoids ($p<0.01$), a biomarker of fruit and vegetable intake, and higher SBP (1.53 mmHg higher, 95% CI=0.41, 2.66) after adjustment for demographics and income. Residence in a food desert was not associated with odds of CKD (OR=1.20, 95% CI=0.96, 1.49). Lower, versus higher, income was associated with lower serum carotenoids ($p<0.01$) and higher

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SBP (2.00 mmHg higher for income–poverty ratio 1 vs >3, 95% CI=1.12, 2.89), but also greater odds of CKD (OR=1.76 for income–poverty ratio 1 vs >3, 95% CI=1.48, 2.10).

Conclusions—Limited access to healthy food due to geographic or financial barriers could be targeted for prevention of CKD and hypertension.

Introduction

Chronic kidney disease (CKD) affects 26 million Americans with growing prevalence and impact nationally.^{1,2} Hypertension is a major risk factor for CKD and a leading national risk factor for morbidity and mortality.^{2,3} As a result, CKD and hypertension are deemed important public health priorities warranting surveillance in the U.S. by CDC.⁴ Both CKD and hypertension are associated with substantial ethnic/race, socioeconomic, and geographic disparities that suggest modifiable environmental risk factors that could be targeted.^{5–9} The roles of diet and food environments as contributors to chronic disease have gained increased attention in recent years,^{10–12} with dietary risk factors identified as the leading risk factor for death and disability in the U.S. and globally.^{2,13} It is increasingly recognized that adverse dietary patterns may increase the risk of CKD and hypertension.^{14–20} Although the exact mechanisms through which diet promotes CKD and hypertension are not known, a number have been proposed, including high sodium and phosphate content,^{21,22} increased diet acid load,^{23,24} and high intake of saturated fats.¹⁶

Adverse dietary patterns, such as higher intake of processed meats and fats and limited intake of fruits and vegetables, are common in lower-income neighborhoods in the U.S. and may be influenced by the local availability of affordable, healthy foods.^{10,12,25,26} The U.S. Department of Agriculture (USDA) has defined areas in the U.S. that are characterized by low income and limited access to large grocery stores or supermarkets as “food deserts.”²⁷ Although food deserts have been linked to some diet-related illnesses such as obesity, data on CKD and hypertension are lacking.^{10,12,27} Furthermore, the higher prices of healthy foods, such as fresh fruits and vegetables, also make low family income a substantial barrier to healthy food access.²⁸ Thus, this paper describes the associations of residence in a food desert and low family income with kidney function and hypertension using national data. The authors hypothesize that food deserts and poverty will associate with greater likelihood of CKD and higher blood pressure due to adverse impacts on diet.

Methods

Study Population

The study included adults aged ≥20 years who enrolled in the National Health and Nutrition Examination Survey (NHANES) between 2003 and 2010 and had information on Census Tract of residence (N=22,173).

Measures

The NHANES is a complex survey of the non-institutionalized U.S. population. Blood pressure was measured manually in triplicate by study physicians after 5 minutes at rest in the seated position. Blood and urine samples were obtained and tested for a variety of

analytes, including serum creatinine, phosphate, bicarbonate, carotenoids, and urine albumin to creatinine ratio (UACR). Dietary intake was ascertained through in-person 24-hour dietary recall interview utilizing the Automated Multiple Pass Method.²⁹ The majority (89%) of participants completed a second 24-hour dietary recall by phone within 3–10 days.

To characterize geographic access to healthy foods, publicly available food desert data from the USDA Economic Research Service (www.ers.usda.gov) were linked based on 2000 U.S. Census Tracts by the Research Data Center of the National Center for Health Statistics. Food deserts were classified as low-income Census Tracts in which 33% of the population or 500 individuals reside >1 mile, if urban, or >10 miles, if rural, from the nearest supermarket or large grocery store based on data from 2006 by the USDA.

To characterize SES that may also impact the ability to purchase healthy foods, the authors used the self-reported family income indexed to the DHHS poverty threshold specific to the year, state, and family size (income–poverty ratio). Although food desert Census Tracts are characterized in part by low median income or high poverty rate, there was a range of family incomes represented among participants residing in food deserts (weighted percentage by income–poverty ratio: <1, 26%; 1–1.5, 16%; 1.5–2, 14%; 2–3, 16%; and >3, 29%).

Statistical Analysis

Characteristics were summarized using weighted distributions and frequencies to account for the sampling structure of NHANES. Dietary intake variables were averaged from both recall interviews, where available, but also included participants with only one dietary recall. Prior literature has suggested that lower dietary acid load and greater intake of fruits and vegetables may be associated with slower CKD progression.^{23,30} Fruits and vegetables are primarily base-producing foods and therefore lower dietary acid load. As indicators of dietary acid load, net endogenous acid production (NEAP) and potential renal acid load were calculated from intake.³¹ Serum carotenoids were utilized as unbiased biomarkers of fruit and vegetable intake, where available (survey years 2003–2006, $n=8,496$).^{32,33} Values were calibrated according to NHANES guidance, combined as the sum of alpha- and beta-carotene, beta-cryptoxanthin, lutein and zeaxanthin, and log-transformed. Differences were tested across groups using weighted chisquare or univariate linear regression.

To evaluate the relationship among food deserts, income–poverty ratio, blood pressure, and odds of CKD, weighted multivariable linear and logistic regression models were constructed. Systolic blood pressure (SBP) was the average of all available measurements. CKD was defined according to accepted criteria as estimated glomerular filtration rate (eGFR) <60 mL/minute/1.73m² or UACR >30 mg/g. GFR was estimated using the CKD Epidemiology Collaboration equation for serum creatinine³⁴ with recalibration of serum creatinine from the 2005–2006 survey according to NHANES guidance. Models included the covariates age, sex, race/ethnicity, food desert status, and income–poverty ratio; and interactions were tested between food desert status and income (income to poverty ratio 2 versus >2). Based on the hypothesis that lower fruit and vegetable intake may mediate the above associations, the effect of additional adjustment serum carotenoids was evaluated. Analyses were performed in 2014–2015 using Stata MP, version 13.

Sensitivity Analysis

Updated definitions of food deserts were released in 2013, incorporating alternate distance thresholds, accounting for the prevalence of vehicle access and using household and supermarket data from 2010. These definitions were based on 2010 Census Tracts, which were not available in the NHANES data, thus they were not used in the primary analyses. To explore the potential impact of this on our results, selected 2013 food desert definitions were merged to the study participants in Census Tracts whose boundaries had not changed substantially between 2000 and 2010 (www.census.gov, $n=15,673$). Classification according to alternative food desert definitions exhibited fair to moderate agreement with the primary definition (kappa statistic ranging from 0.26 to 0.45).

Results

The study included 22,173 participants representing 2,567 unique Census Tracts in the U.S., 259 of which were food deserts (Appendix Figure 1). The weighted characteristics of the participants generally reflected those of the adult U.S. population (Table 1). A minority of the participants in the study self-reported a history of CKD (2.1%), diabetes (8.2%) or hypertension (30.1%), although awareness is known to be low.^{3,35} Participants living in food deserts were more likely to be black or Hispanic ($p<0.001$), have a known history of diabetes ($p=0.02$) or CKD ($p=0.02$), and had higher average SBP, BMI, UACR, and eGFR (Table 1). Differences in baseline characteristics were similar according to categories of income–poverty ratio, but additionally included younger age, greater proportion of women, and increased prevalence of eGFR <60 mL/minute/1.73 m² among those with lower income–poverty ratio (Table 1).

Mean protein, potassium, sodium, calcium, and magnesium intake were lower, yielding higher NEAP, a measure of dietary acid load, in food deserts compared with non-food deserts ($p<0.05$ for each, Table 2). A similar pattern was observed in lower categories of income–poverty ratio (Table 2). Additionally, serum carotenoids, a biomarker of fruit and vegetable intake, were lower in participants living in food deserts ($p=0.007$) and those with lower income–poverty ratio ($p<0.007$, Table 2).

Participants in food deserts spent less money at the grocery store in the last 30 days overall (geometric mean [–1SD, +1SD], \$290.51 [128.39, 657.33] vs \$333.40 [178.99, 621.01]; $p=0.001$). The pattern was similar with less money spent at the grocery store for participants with lower income–poverty ratio (geometric mean [–1SD, +1SD], \$287.63 [271.99, 304.17]; \$288.01 [268.72, 308.77]; \$277.97 [258.76, 298.60]; \$310.81 [289.68, 333.49] and \$367.43 [353.34, 382.08] for ratio 1, 1–1.5, 1.5–2, 2–3, and >3 , respectively, $p<0.001$). Fewer participants living in food deserts reported having fruit available in the home “always” or “most of the time” (80.3% vs 87.0% in non-food deserts, $p<0.001$). Similar results were observed among participants with lower income–poverty ratio for fruits (79.2% in lowest income category vs 89.7% in highest income category reporting available “always” or “most of the time,” $p<0.001$) and for dark green vegetables (71.6% in lowest income category vs 82.0% in highest income category reporting available “always” or “most of the time,” $p<0.001$). Participants living in food deserts rated their overall food security lower than those not in food deserts, with 17.2% reporting very low or low, 11.2% reporting marginal,

and 71.6% reporting full food security compared with 11.2% (very low or low), 6.8% (marginal), and 82.0% (full) in non-food deserts. Qualitatively larger differences in food security were observed according to income, with 36% reporting very low or low and 49% reporting full food security in the lowest income category versus 2% reporting very low or low and 96% reporting full food security in the highest category ($p<0.001$).

Residence in a food desert was associated with higher SBP in unadjusted models (Table 1), and after adjustment for age, sex, race/ethnicity, and income (SBP 1.53 mmHg higher in food deserts compared with non-food deserts, 95% CI=0.41 mmHg, 2.66 mmHg, Figure 1A). Additional adjustment for diabetes, eGFR, and proteinuria did not change the result (data not shown). SBP was higher among those with lower income–poverty ratio after adjustment (Figure 1A) and findings were similar among participants that were not using anti-hypertensive medications (Figure 1B). In stratified analyses, the relationship between residence in a food desert and SBP was stronger among participants with a family income more than two times the poverty threshold (p -interaction=0.06, Figure 1C). Serum carotenoids were robustly associated with higher SBP (Appendix Table 2). The relationship between food deserts and SBP was attenuated by 15% after adjustment for carotenoids and point estimates for income categories attenuated by 31%–37%. Residence in a food desert was not associated with odds of CKD in adjusted models overall (Figure 2), but was associated with 40% higher odds of CKD after adjustment among those whose labs were performed fasting (OR=1.40, 95% CI=1.11, 1.77). This result appeared to be related to a significant relationship with albuminuria among those with fasting samples (UACR >30 mg/g, OR=1.35, 95% CI=1.05, 1.73). The relationship between residence in a food desert and odds of albuminuria and CKD were similar when stratified by income–poverty ratio 2 versus >2 (p -interaction=0.6 and 0.9, respectively). Lower income–poverty ratio was associated with greater odds of CKD overall (Figure 2). Results were similar in the fasting subpopulation (data not shown).

Results using alternative food desert definitions revealed similar patterns of association (Appendix Table 1). Odds of CKD were higher among participants in food deserts according to most definitions, but SBP was not statistically higher in food deserts outside of the primary analysis.

Discussion

The authors found that adults living in Census Tracts classified as food deserts have higher SBP than adults in non-food desert Tracts in the U.S. A trend toward higher prevalence of CKD was also found in the primary analysis, but this was only significant among the fasting population who likely had more-reliable morning assessments of albuminuria.³⁶ Sensitivity analyses also supported a possible relationship between food deserts and CKD. By contrast, adults with lower family income had both higher SBP and higher odds of CKD. The authors propose that these associations may be due, at least in part, to adverse dietary intake and behaviors including decreased availability of fruits and vegetables that were observed among adults residing in food deserts and with lower income. This conclusion is supported by partial attenuation of these effects with adjustment for serum carotenoids, a biomarker of fruit and vegetable intake. Most prior studies of the neighborhood food environments have

focused on obesity and diabetes as opposed to other diet-dependent diseases, such as CKD and hypertension. The present findings strengthen the argument that diet and food access play a role in the disparate prevalence of hypertension and, possibly, CKD across communities.

Interestingly, family income showed qualitatively stronger relationships with diet, blood pressure, and CKD than living in a food desert. These results are consistent with prior studies that linked lower family income with albuminuria in NHANES III.³⁷ Recommended dietary patterns such as the Dietary Approaches to Stop Hypertension pattern are proven to lower blood pressure and may reduce risk of GFR decline,^{14,15} but are more expensive than less optimal diets.²⁸ Reduced family purchasing power may also impact risk of CKD and hypertension by limiting access to healthy food. However, residence in a food desert may have a stronger relationship with blood pressure when purchasing power is not limiting. Overall, the present data underscore the need to target these financial barriers to purchasing healthy food in addition to geographic barriers.

Although the reported associations among food deserts, dietary intake, CKD, and hypertension are modest, several sources of misclassification may have biased these results toward null.³⁸ Dietary recall interviews have several sources of misclassification, including recall inaccuracy and inability to account for daily variation in diet.³⁹ Additionally, this study's definition of food deserts, though accepted, may be a source of misclassification as indicated by the moderate concordance of the primary definition with alternative definitions in sensitivity analyses. Finally, individuals may often purchase food outside of their immediate neighborhoods of residence, such as neighborhoods in which they work or recreate. The authors did not have information on travel patterns to other neighborhoods. In light of these expected and challenging measurement limitations, use of a large generalizable source of data such as the NHANES allowed for detection of adverse associations between food deserts and disease that may be missed in smaller studies.

In addition, the current study adds to the existing literature linking low intake of fruits and vegetables and the resulting higher dietary acid load with hypertension.²⁴ In this study, NEAP, a measure of dietary acid load, was higher and serum carotenoids were lower in food deserts and among those with lower income. Furthermore, these measures were independently associated with SBP and prevalence of CKD. Lower intake of fruits and vegetables and the resulting increased acid load, lower potassium intake, or other metabolic effects may, in part, explain higher blood pressure observed in food deserts and among lower-income adults.

Contrary to the study hypothesis, residence in a food desert was associated with higher mean eGFR in univariate analyses. Although this may appear to refute the relationship between food access and CKD, albuminuria and blood pressure were increased in this group. Prior investigations have demonstrated robust relationships between albuminuria and risk of end-stage renal disease, particularly among African Americans who have a much greater risk of end-stage renal disease, advanced CKD, and albuminuria despite lower prevalence of mild reductions of eGFR than Caucasians.⁴⁰ For this reason, greater rates of albuminuria and

higher blood pressure in food deserts may indicate an early CKD-risk phenotype in which glomerular hyperfiltration or inaccuracies in eGFR estimation mask reduction in GFR.

Limitations

The results of this study are cross-sectional; therefore, the causal links among food access, income, blood pressure, and CKD should be interpreted cautiously. It is also important to note that neighborhood characteristics, such as geographic food access, have complex relationships with health outcomes in which true causal effects may emerge over generations through effects not only on individuals directly but also through influence on their peers, families, and other community resources. Other study designs including longitudinal studies and pre-post designs would better establish causality, but may be limited by an inability to sustain interventions long enough to assess their full impact. In addition, only adults were evaluated in this study. Exposure to adverse food environments in childhood may have even stronger effects on dietary behavior.⁴¹

Conclusions

This study reports higher blood pressure among adults residing in food deserts, as well as higher blood pressure and increased prevalence of CKD among adults with lower income, both of which are associated with adverse dietary intake and habits. Prior studies have linked area-level SES with an increased risk of CKD.^{42–44} The authors propose that lack of access to healthy foods may be one pathway explaining these relationships. Interventions targeting geographic food access and affordability should be tested to reduce disparities in blood pressure and CKD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Difference in mean systolic blood pressure according to residence in a food desert and income among (A) all participants; (B) participants not using medications for high blood pressure; and (C) among all participants stratified by income-poverty ratio vs. >2. Differences are adjusted using linear regression with the following covariates age, sex, race/ethnicity, food desert, income/poverty ratio. Differences in mean systolic blood pressure are represented by black squares. Bars represent 95% CI of the difference.

Figure 2.

OR of chronic kidney disease and albuminuria according to residence in a food desert (A) and income (B). Chronic kidney disease is defined as estimated GFR <60 ml/min/1.73m² or urine albumin to creatinine ratio (UACR) >30 mg/g. Albuminuria is defined by UACR >30 mg/g. ORs are adjusted using logistic regression with the following covariates age, sex, race/ethnicity, food desert, income/poverty ratio. Black square represents odds ratio estimates and bars represent 95% CI.

Table 1

Patient Characteristics According to Food Desert Status and Income

Mean ± SD or %	All (n=22,173)	Food desert Yes (n=2,053)	No (n=20,102)	p- value	1 (n=4,095)	1-1.5 (n=3,230)	1.5-2 (n=2,275)	2-3 (n=3,244)	>3 (n=7,573)	p- value ^a
Demographics										
Age (years)	46.7 ± 14.5	45.6 ± 17.4	46.7 ± 14.2	0.13	41 ± 18	48 ± 19	48 ± 18	48 ± 15	47 ± 11	<0.001
Female sex (%)	51.9%	51.1%	51.9%	0.40	57.2%	56.5%	53.8%	51.7%	49.1%	<0.001
Race/ethnicity				<0.001						<0.001
Hispanic	12.4%	18.5%	11.9%		26.7%	21.0%	16.4%	11.3%	5.5%	
Non-Hispanic white	70.3%	50.8%	71.8%		48.3%	57.1%	62.6%	71.6%	81.5%	
Non-Hispanic black	11.3%	27.1%	10.1%		18.7%	14.7%	15.5%	12.2%	7.4%	
Other	6.0%	3.6%	6.2%		6.2%	7.3%	5.6%	4.9%	5.7%	
Income-poverty ratio	33.8%	54.6%	32.2%	<0.001	—	—	—	—	—	—
Self-reported medical history										
Hypertension	30.1%	31.2%	30.0%	0.44	27.9%	33.3%	32.5%	32.5%	28.8%	<0.0
Self-reported kidney disease	2.1%	3.0%	2.0%	0.02	2.8%	3.5%	3.0%	2.7%	1.1%	<0.001
Diabetes	8.2%	10.2%	8.0%	0.02	9.4%	10.3%	10.9%	8.9%	6.2%	<0.001
Laboratory/Exam										
Systolic bp (mm Hg)	122 ± 15	124 ± 19	122 ± 15	0.001	121 ± 20	124 ± 21	124 ± 18	123 ± 15	121 ± 12	0.001
Diastolic bp (mm Hg)	70 ± 11	71 ± 13	70 ± 11	0.40	69 ± 14	69 ± 14	69 ± 12	69 ± 11	71 ± 9	<0.001
BMI (kg/m ²)	28.5 ± 5.6	29.3 ± 7.2	28.5 ± 5.5	<0.001	28.8 ± 8.0	28.8 ± 7.0	28.7 ± 6.3	28.8 ± 5.5	28.3 ± 4.5	0.04
Waist circumference (cm)	97.7 ± 13.6	98.9 ± 15.9	97.7 ± 13.4	0.07	97.2 ± 17.4	98.0 ± 16.5	98.2 ± 14.9	98.4 ± 13.3	97.6 ± 11.3	0.19
eGFR (ml/min/1.73m ²)	94.4 ± 18.7	97.5 ± 23.5	94.1 ± 18.3	0.001	102 ± 24	95 ± 26	95 ± 22	93 ± 19	93 ± 14	<0.001
eGFR <60 (%)	6.8%	7.7%	6.7%	0.22	4.9%	10.7%	9.3%	8.3%	5.1%	<0.001
UACR (mg/g) ^b	7.8 (3.2, 19.3)	8.7 (2.8, 26.6)	7.8 (3.2, 18.8)	0.01	8.8 (2.6, 30.0)	9.6 (2.9, 32.1)	8.8 (3.1, 24.6)	8.1 (3.3, 19.8)	7.0 (3.5, 13.8)	<0.001
UACR 30 mg/g	9.2%	12.0%	8.9%	<0.001	11.8%	13.4%	11.7%	9.5%	6.7%	<0.001
Serum phosphorus (mg/dl)	3.79 ± 0.47	3.80 ± 0.56	3.79 ± 0.47	0.46	3.80 ± 0.59	3.76 ± 0.58	3.76 ± 0.52	3.80 ± 0.47	3.79 ± 0.39	0.13
Serum bicarbonate (mEq/L)	25.0 ± 1.86	25.0 ± 2.2	25.0 ± 1.8	0.83	24.7 ± 2.3	24.8 ± 2.3	24.9 ± 2.1	25.0 ± 1.9	25.1 ± 1.5	<0.001

^a p-value from global F-test or Chi-square^b presented as geometric mean (−1SD, +1SD)

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eGFR, estimated glomerular filtration rate; UACR, urine albumin to creatinine ratio; PIR, ratio of family income to the local poverty line

Note: Boldface indicates statistical significance ($p<0.05$).

Table 2

Dietary Intake and Biomarkers According to Food Desert Status and Income

Mean ± SD	Food desert			Family income to poverty ratio					p-value ^d	
	All	Yes	No	p-value	1	1–1.5	1.5–2	2–3		>3
Macronutrients										
Energy (kcal)	2,123 ± 759	2,072 ± 879	2,127 ± 750	0.16	2,079 ± 1,048	2,024 ± 927	2,054 ± 815	2,098 ± 745	2,182 ± 610	<0.001
Protein (g)	83.0 ± 31.7	79.4 ± 37.7	83.3 ± 31.2	0.03	78.6 ± 41.7	77.4 ± 38.4	80.1 ± 33.5	82.0 ± 31.5	86.4 ± 25.9	<0.001
Carbohydrates (g)	257 ± 98	253 ± 109	258 ± 97	0.38	261 ± 135	256 ± 120	252 ± 105	255 ± 95	258 ± 79	0.22
Total fat (g)	80.3 ± 34.8	77.7 ± 39.8	80.5 ± 34.4	0.13	75.8 ± 46.1	74.4 ± 42.5	76.8 ± 35.7	80.1 ± 34.9	83.8 ± 28.5	<0.001
Selected micronutrients										
Phosphorus (mg)	1,352 ± 509	1,284 ± 601	1,358± 501	0.02	1,268± 661	1,264 ± 612	1,301 ± 533	1,334 ± 503	1,413 ± 420	<0.001
Sodium (mg)	3,472 ± 1,358	3,315 ± 1,524	3,484 ± 1,343	0.02	3,268 ± 1,820	3,236 ± 1,594	3,318 ± 1,396	3,458 ± 1,298	3,635 ± 1,136	<0.001
Potassium (mg)	2,715 ± 975	2,544 ± 1117	2,728 ± 963	0.002	2,488 ± 1,277	2,509 ± 1,147	2,599 ± 1,009	2,681 ± 932	2,852 ± 808	<0.001
Calcium (mg)	942 ± 456	884 ± 507	946 ± 452	0.02	866 ± 564	889 ± 549	896 ± 459	932 ± 457	988 ± 386	<0.001
Magnesium (mg)	295 ± 114	270 ± 121	296 ± 113	<0.001	269 ± 142	267 ± 130	276 ± 111	288 ± 107	313 ± 97	<0.001
PRAL (mEq)	13.8 ± 17.5	14.5 ± 20.7	13.7 ± 17.2	0.39	14.9 ± 21.4	13.5 ± 20.9	14.0 ± 18.3	13.7 ± 17.6	13.7 ± 14.8	0.15
NEAP (mEq)	58.6 ± 19.0	60.7 ± 23.6	58.4 ± 18.7	0.03	62.2 ± 25.3	59.8 ± 24.4	59.2 ± 20.8	58.2 ± 20.7	57.5 ± 14.8	<0.001
Dietary Biomarkers ^b										
Total carotenoids (ug/dl) ^c	39.2 (24.0, 64.3)	35.8 (21.2, 60.5)	39.5 (24.2, 64.5)	0.007	34.4 (19.1, 61.9)	36.1 (20.2, 64.2)	37.6 (22.0, 64.3)	37.3 (23.4, 59.3)	42.3 (27.5, 65.0)	<0.001

^a p-value from global F-test^b Only available in participants from NHANES 2003–2006 (n=8,496)^c presented as geometric mean (–1SD, +1SD)

PRAL, potential renal acid load; NEAP, net endogenous acid production

Note: Data from 20,150 adult NHANES participants who completed dietary intake interview. Some participants declined to report income or provide enough detail for classification according to poverty to income ratio, therefore 18,699 could be classified by income. SD may more accurately reflect underlying population SD for macronutrients than micronutrients due to greater day-to-day variation in the latter.

Note: Boldface indicates statistical significance ($p < 0.05$).