Changes in Diet Among Adult Prevalent Kidney Stone Formers in the United States: The National Health and Nutrition Examination Survey, 1988–2018

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ABSTRACT

BACKGROUND/PURPOSE: Dietary factors greatly influence kidney stone disease (KSD). We aimed to examine the dietary pattern changes of adult stone formers (SF) in the United States from 1988 to 2018.

METHODS: We used weighted, multiple linear regression analyses to examine dietary data from prevalent SF in the National Health and Nutrition Examination Survey (NHANES) III and 2007-2018.

RESULTS: The study was divided into four periods: 1988–1994, 2007–2010, 2011–2014, and 2015–2018. The KSD prevalence rate increased from 5.5% in 1988–1994 to 10.8% in 2015–2018. No significant changes occurred in the intake of total calories, total protein, saturated fat, or sodium. Total daily fat intake increased significantly (78.9 g in 1988–1994 to 88.7 g in 2015–2018, p=0.03). Daily potassium intake decreased significantly (2,947 mg in 1988–1994 to 2,628 mg in 2015–2018, p<0.001). Daily calcium intake (795 mg in 1988–1994 to 950 mg in 2015–2018, p<0.001) and daily fluid intake (2,120 ml in 1988–1994 to 3,124 ml in 2015–2018, p<0.001) increased significantly. No effect modification by gender or age group was detected.

CONCLUSION: There were significant increases in polyunsaturated fat, calcium, and fluid intakes. However, dietary potassium intake was reduced. Overall, there were no statistically significant changes in dietary intakes of calories, protein, carbohydrates, fiber, saturated fat, and sodium.

KEYWORDS: Kidney stone; dietary habits; NHANES; potassium; fluid intake

INTRODUCTION

Kidney stone disease (KSD) is a prevalent and debilitating condition affecting millions globally.[1] It is caused by the complex interactions of dietary, genetic, and environmental factors that are not fully understood. However, specific nutrients and dietary patterns are implicated in stone formation and prevention.[2] For instance, high sodium intake has been linked to increased urinary calcium excretion and a consequent increase in the risk of calcium-based stone formation,[3] while high potassium intake has been associated with decreased urinary calcium excretion, reducing that risk.[4,5]

Current guidelines for the prevention of kidney stones recommend a diet rich in fruits, vegetables, and fluids, and restricted in sodium and animal proteins.[8] Nevertheless, adherence to those guidelines is variable among individuals. To better understand the rising prevalence rate of KSD, it is essential to examine the changes in dietary habits among stone formers. This study examines the data from the National Health and Nutrition Examination Survey (NHANES), a comprehensive dataset for evaluating dietary trends and their effects on health outcomes, to establish a temporal trend of dietary patterns among kidney stone formers from 1988 to 2018.

METHODS

Study Design and Population

This study uses NHANES data to compare the 1988–1994 cycle with the more recent 2007–2018 cycle. NHANES is a cross-sectional survey that combines self-reported data, physical examinations, and laboratory tests to provide a comprehensive picture of health and nutrition among the U.S. population.[9]

Inclusion and Exclusion Criteria

Participants with a self-reported history of kidney stones were identified based on responses to the NHANES dietary interview and medical history questions. Only those with complete dietary data and relevant covariate information were included. Individuals with incomplete data or those reporting conditions unrelated to kidney stones were excluded. **Table 1** shows the number of patients included and excluded from this study.

Dietary Assessment

Dietary intake was assessed using 24-hour dietary recall interviews conducted by trained interviewers. The dietary data were analyzed to evaluate adherence to kidney stone prevention guidelines, including sodium, potassium, and fluid intake. For the NHANES 2007–2018 cycles, dietary intake was based on the day-one recall.

Table 1. Study Population Inclusion and Exclusion Criteria

Study population	NHANES 1988–1994	NHANES 2007–2018
Total records	20,050	59,842
Exclude missing KSD‡ response	21	25,163
Exclude age < 20	1,225	0
Total remaining	18,804	34,679
Exclude missing dietary data	2,835	3,963
Total remaining	15,969	30,716
Exclude missing covariates	1,861	3,390
Total included	14,108	27,326
Total KSD only included	680	2,636

Data derived from NHANES III (1988–1994) and NHANES 2007–2018 cycles. KSD: Kidney Stone Disease.

Table 2. Characteristics of Participants with KSD by Survey Period

		NHANES 1988–1994 n = 676	NHANES 2007–2018 n = 2,636
Age (year), mean (SE)		53.4 (0.8)	53.7 (0.4)
Male sex, % (n)		58.5 (408)	55.2 (1472)
Race, % (n)	Non-Hispanic White	87.2 (457)	76.7 (1460)
	Non-Hispanic Black	3.6 (83)	5.7 (340)
	Other	9.2 (136)	17.7 (836)
BMI (kg/m2), mean (SE)		27.8 (.30)	30.5 (.18)
Diabetes, % (n)		8.5 (71)	22.5 (691)
HbA1c (%), mean (SE)		5.6 (.05)	5.9 (.03)
Hypertension, % (n)		37.7 (266)	48.7 (1389)
SBP (mm Hg), mean (SE)		128.9 (.85)	124.5 (.40)
DBP (mm Hg), mean (SE)		76.9 (.61)	71.5 (.39)
Cardiovascular disease, % (n)		11.7 (115)	12.1 (403)
Smoking, active, % (n)		23.7 (133)	19.3 (517)

Values are expressed as weighted means (SE) or %, unweighted n. P-values test for differences between time periods without assuming a linear association with time. SE: standard error; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; cardiovascular disease includes histories of myocardial infarction, congestive heart failure, and/or ischemic stroke.

Covariates

Covariates included age, gender, race/ethnicity, BMI, and cardiovascular risk factors, including self-reported diabetes mellitus, hypertension, stroke, myocardial infarction, and congestive heart failure. Hemoglobin A1c (HbA1c) and blood pressure measurements were also examined as covariates.

Statistical Analysis

Descriptive statistics were used to summarize the demographic and clinical characteristics of the two NHANES surveys. Dietary trends over time were analyzed by multivariable linear regression, adjusting for age, gender, race/ethnicity, BMI, and cardiovascular risk factors. Unadjusted and adjusted means and 95% confidence intervals (CIs) were reported for 1988–1994 and three four-year periods for 2007–2018.

Product terms were included in the models to assess the effect of measure modification by gender and age (65+ vs. <65) when the main effect of the time period was statistically significant at alpha = 0.05. All analyses were performed using survey procedures in Stata MP 18 (Stata Corp, College Station, TX), including strata, primary sampling units, and survey weights. Exam weights and day one dietary recall weights were used for NHANES 1988–1994 and 2007–2018, respectively. Weights were adjusted when combining cycles as outlined in NHANES analytic guidelines.

Ethical Considerations

Ethical approval is not applicable to this article as it utilizes publicly available, de-identified data from NHANES. The National Center for Health Statistics Research Ethics Review Board approved the NHANES protocol. All participants provided informed consent for their data to be used in research.

RESULTS

Study Population

The prevalence of KSD increased from 5.6% in 1988–1994 (95% CI: 4.9–6.4) to 10.8% in 2015–2018 (95% CI: 9.9–11.9). **Table 2** outlines the characteristics of the study population. The cohort included 3,316 participants with a history of kidney stones across NHANES cycles. Non-White race, mean BMI, prevalent diabetes, mean HbA1c, and prevalent hypertension all increased between surveys. Mean SBP, mean DBP, and current smoking all decreased between surveys.

Values are expressed as weighted means (SE) or %, unweighted n. P-values test for differences between time periods without assuming a linear association with time. SE: standard error; cardiovascular disease includes histories of myocardial infarction, congestive heart failure, and/or ischemic stroke.

Dietary Intake Trends

Tables 3, 4, and 5 present changes in dietary intake among kidney stone formers between NHANES 1988–1994 and NHANES 2007-2018. There were no significant changes in total calorie or total protein intake [**Table 3**]. Total daily fat intake increased significantly from 78.9 g in 1988-1994 to 88.7 g in 2015-2018 (p=0.03). Specifically, the mean intake of saturated fat rose from 26.6 g to 29.3 g, though this increase was not statistically significant (p=0.089 in Model 3) [**Table 3**]. There were no significant changes in total sodium intake [**Table 4**]. Significant changes were observed in potassium and calcium intake [**Table 4**]; specifically, daily potassium intake decreased from 2,947 mg in 1988–1994 to 2,628 mg

Table 3. Change in Macronutrient Intake Among Participants with KSD Over Time

Intake, day 1 recall	1988–1994, n = 680	2007–2010, n = 882	2011–2014, n = 819	2015–2018, n = 935	P
Total calories (kcal)					
Unadjusted	2105.1 (2014.5–2196.7)	2140.3 (2039.0–2241.7)	2069.8 (1977.9–2161.7)	2166.6 (2058.5–2274.7)	0.6
Model 1	2169.1 (2079.5–2259.8)	2300.5 (2187.0–2414.1)	2239.6 (2111.8–2367.3)	2299.7 (2163.7–2435.8)	0.5
Model 2	2158.5 (2068.8–2248.2)	2183.7 (2091.7–2275.7)	2130.8 (2027.8–2233.7)	2189.1 (2075.0–2303.1)	0.4
Model 3	2170.5 (2078.9–2262.1)	2176.8 (2084.6–2269.0)	2120.6 (2018.3–2222.8)	2178.9 (2064.0–2293.9)	0.5
Protein (g)					
Unadjusted	79.6 (75.2–84.0)	80.5 (76.3–84.7)	79 (75.9–82.1)	83.4 (77.0–89.7)	0.66
Model 1	82.2 (77.7–86.7)	87.2 (81.3–93.1)	86.2 (80.9–91.5)	88.9 (80.0–97.8)	0.72
Model 2	82.2 (77.7–86.7)	82.5 (78.3–86.6)	81.9 (78.0–85.7)	84.5 (77.8–91.1)	0.70
Model 3	82.8 (78.2–87.4)	82.2 (78.0–86.5)	81.6 (77.8–85.5)	84.1 (77.3–91.0)	0.81
Total fat (g)					
Unadjusted	78.9 (74.8–83.1)	82.9 (78.5–87.2)	80.8 (76.9–84.8)	88.7 (83.5–93.9)	0.031
Model 1	79.9 (75.6–84.3)	86.4 (81.4–91.5)	84.9 (79.3–90.6)	91.5 (84.4–98.5)	0.020
Model 2	79.7 (75.4–84.0)	83.4 (79.4–87.5)	82.3 (77.6–86.9)	88.7 (83.0–94.4)	0.0098
Model 3	80.1 (75.7–84.4)	83.1 (79.0–87.1)	81.8 (77.2–86.5)	88.2 (82.4–93.9)	0.020
Saturated fat (g)					
Unadjusted	26.6 (24.8–28.3)	27 (25.5–28.5)	26.4 (24.9–27.8)	29.3 (27.3–31.2)	0.11
Model 1	26.9 (25.1–28.7)	28.2 (26.4–29.9)	27.7 (25.6–29.7)	30.2 (27.8–32.6)	0.099
Model 2	26.8 (25.0–28.5)	27.2 (25.8–28.6)	26.8 (25.1–28.4)	29.2 (27.2–31.3)	0.070
Model 3	26.9 (25.1–28.7)	27 (25.6–28.4)	26.6 (24.9–28.2)	29 (27.0–31.0)	0.089

Values are expressed as weighted means (95% CI). Multivariable linear regression models included the following covariates: Model 1: Age in years, gender, race/ethnicity, BMI. Model 2: Model 1 + diabetes, hypertension, cardiovascular disease, smoking. Model 3: Model 1 + HbA1c, SBP, DBP, cardiovascular disease, smoking. KSD: Kidney Stone Disease.

Table 4. Change in Micronutrient Intake Among Participants with KSD Over Time

Intake, day 1 recall	1988–1994, n = 680	2007–2010, n = 882	2011–2014, n = 819	2015–2018, n = 935	P
Sodium (mg)					
Unadjusted	3424.7 (3230.8–3618.5)	3594.5 (3424.8–3764.2)	3405.5 (3237.2–3573.9)	3634.3 (3413.3–3855.3)	0.19
Model 1	3505.0 (3312.6–3697.3)	3821.3 (3615.3–4027.4)	3654.2 (3422.4–3886.0)	3820.8 (3528.2–4113.3)	0.18
Model 2	3494.9 (3299.7–3690.0)	3634.8 (3469.7–3799.9)	3482.8 (3292.6–3673.0)	3644.7 (3408.7–3880.7)	0.17
Model 3	3504.1 (3307.3–3700.9)	3629.9 (3465.4–3794.4)	3476.7 (3283.1–3670.2)	3637.8 (3400.4–3875.1)	0.18
Potassium (mg)					
Unadjusted	2946.7 (2808.4–3085.1)	2760.1 (2604.5–2915.6)	2567.9 (2460.2–2675.6)	2627.8 (2477.0–2778.5)	<0.001
Model 1	2902.2 (2759.2–3045.3)	2768 (2589.3–2946.7)	2606 (2465.8–2746.3)	2620.4 (2441.7–2799.0)	0.0058
Model 2	2903.4 (2761.6–3045.2)	2734.4 (2578.2–2890.5)	2579.3 (2461.0–2697.6)	2589 (2439.0–2739.1)	0.0069
Model 3	2917,8 (2776.9–3058.7)	2726.6 (2568.7–2884.4)	2571.6 (2454.7–2688.4)	2577.6 (2424.9–2730.3)	0.0022
Calcium (mg)					
Unadjusted	795.1 (745.9–844.3)	914.1 (872.7–955.5)	928.6 (871.4–985.8)	950.4 (892.0–1008.9)	<0.001
Model 1	813.2 (762.2–864.3)	964.2 (907.7–1020.8)	979.9 (910.1–1049.7)	993.2 (920.5–1065.9)	<0.001
Model 2	815.7 (764.7–866.7)	928.9 (884.1–973.6)	949.1 (888.0–1010.1)	961.1 (900.7–1021.5)	<0.001
Model 3	822.5 (770.8–874.1)	925.9 (881.3–970.5)	947.4 (888.1–1006.6)	956.8 (897.0–1016.6)	<0.001

Values are expressed as weighted means (95% CI). Multivariable linear regression models included the following covariates: Model 1: Age in years, gender, race/ethnicity, BMI. Model 2: Model 1 + diabetes, hypertension, cardiovascular disease, smoking. Model 3: Model 1 + HbA1c, SBP, DBP, cardiovascular disease, smoking. KSD: Kidney Stone Disease.

Table 5. Change in Fluid Intake Among Participants with KSD Over Time

Intake, day 1 recall	1988–1994, n = 680	2007–2010, n = 882	2011–2014, n = 819	2015–2018, n = 935	P
Fluid (ml)					
Unadjusted	2120.4 (2006.0–2234.9)	3035 (2875.9–3194.1)	2863.3 (2759.2–2967.4)	3123.6 (2995.7–3251.5)	<0.001
Model 1	2173.3 (2045.7–2300.9)	3166.8 (3004.4–3329.3)	2986.7 (2851.2–3122.2)	3241.7 (3089.4–3394.0)	<0.001
Model 2	2160.0 (2032.1–2287.9)	3047.5 (2903.9–3191.2)	2862.7 (2756.7–2968.6)	3118.3 (2996.0–3240.7)	<0.001
Model 3	2146.8 (2016.1–2277.4)	3041.1 (2898.7–3183.6)	2856 (2749.8–2962.3)	3115.2 (2990.6–3239.8)	<0.001

Values are expressed as weighted means (95% CI). Multivariable linear regression models included the following covariates: Model 1: Age in years, gender, race/ethnicity, BMI. Model 2: Model 1 + diabetes, hypertension, cardiovascular disease, smoking. Model 3: Model 1 + HbA1c, SBP, DBP, cardiovascular disease, smoking. KSD: Kidney Stone Disease.

in 2015–2018 (p<0.001), and daily calcium intake increased significantly from 795 mg in 1988–1994 to 950 mg in 2015–2018 (p<0.001), a decrease in potassium and an increase in calcium. Daily fluid intake increased significantly from 2,120 ml in 1988–1994 to 3,124 ml in 2015–2018 (p<0.001) [**Table 5**]. No effect modification by gender or age group was detected.

DISCUSSION

This study examines dietary trends among kidney stone formers over 30 years of NHANES data to show how medical guidelines, specialist recommendations, and cultural shifts may have impacted their nutritional habits. The findings reveal significant changes in several critical dietary components, which may help guide future stone prevention strategies.

A concerning trend is the decreased potassium intake from 2947.6 mg to 2627.8 mg. Low potassium intake is a known risk factor for kidney stones.[4,5] Decreased potassium levels increase potassium reabsorption in the proximal tubule in part through K/H exchangers, with consequent increase in urine acidity. It, in turn, leads to decreased bicarbonate absorption and lowering of intracellular pH, which increases citrate reabsorption.[6] Decreased urinary citrate increases kidney stone risk.[7] Potassium also plays a crucial role in stone prevention by reducing urinary calcium excretion[8] and, in alkali-containing potassium salts, counterbalancing acid production from sulfur-containing amino acids in protein.[5,11] Indeed, a population study has indicated that low dietary potassium intake was associated with an increased risk of prevalent kidney stone disease.[4] In contrast, calcium intake has increased from 795.5 mg to 950.4 mg, aligning with dietary recommendations for kidney stone prevention in patients with enteric hyperoxaluria. Calcium's beneficial effects include decreasing phosphate and oxalate gastrointestinal absorption by binding these elements in the gut,[12] with low-calcium diets associated with increased hyperoxaluria.[13] It is of note, however, that certain conditions like idiopathic hypercalciuria and hyperparathyroidism can lead to increased urinary calcium excretion regardless of dietary intake.[14,15] Furthermore, a low-calcium diet is recommended for stone prevention in cases of enteric hypercalciuria. The relationship between calcium supplementation and stone risk remains a topic of debate, particularly if supplements are taken between meals, when they may be associated with increased urinary calcium excretion without affecting oxalate.[12] Due to the limitations of the NHANES data structure, we could not retrieve information regarding baseline hypercalciuria or hyperoxaluria.

Fluid intake dilutes urine, which is essential for kidney stone prevention.[16] Therefore, it is a positive highlight that it had the highest increase among dietary trends, from 2123 ml to 3123.6 ml. On the other hand, total fat intake increased from 78.9 g to 88.7 g, and saturated fat from 26.6 g to 29.3 g, mirroring the increased prevalence of obesity in the United States. Although the effect of fat in kidney stone formation is ill-defined, obesity is a well-established risk factor, partly by lowering urinary pH and reducing ammoniagenesis, increasing the risk of uric acid stones, and also due to its association with increased urinary calcium and oxalate excretion.[17,18]

In summary, the results are mixed when comparing dietary trends of prevalent KS formers with medical guidelines. While the improvement in fluid intake indicates better adherence to recommendations, the decline in potassium and increase in fat intake suggest otherwise. These changes reveal inconsistent compliance with medical guidelines and may be influenced more by broader population dietary changes and lifestyle than actual medical recommendations.

When considering this study, we acknowledge its limitations. It is based on self-reported data with potential for recall bias. The dietary assessment does not capture daily dietary variations and may not fully represent long-term habits. Finally, the study lacks data on baseline hypercalciuria and hyperoxaluria, specific liquid intake, medication and supplement use, and stone composition, which further limits our analysis.

PRACTICAL APPLICATION

This study highlights critical dietary trends among kidney stone formers. It is encouraging to note an increase in fluid intake conforming to preventive guidelines, while the

decreased potassium intake raises concerns. The increased dietary fat intake is also concerning, particularly in increasing obesity prevalence, although its impact on KS risk has not been well defined. The findings presented here highlight the need for better strategies to educate and change populational habits.

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