Differences in 24-Hour Urine Composition between Black and White Women

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Black women are less likely to develop kidney stones and have greater bone mass than white women. However, little is known about racial differences in urine composition. Urine pH, volume, and 24-h urinary excretion of calcium, citrate, oxalate, uric acid, sodium, potassium, magnesium, phosphate, sulfate, and creatinine of 146 black women were compared with 330 white women in the Nurses' Health Study. All participants were postmenopausal non–stone formers. ANOVA was used to compare mean urinary values. Linear regression models were adjusted for age, body mass index, dietary intake, and urinary factors. On average, black women excreted 65 mg less urinary calcium ($P < 0.001$), 4 mg more oxalate ($P < 0.001$), 9 mEq less potassium ($P < 0.001$), 11 mg less magnesium ($P = 0.003$), 120 mg less phosphate ($P < 0.001$), and 3 mmol less sulfate ($P < 0.001$) per day than did white women. The urine pH of black women was 0.11 units higher ($P = 0.03$) and urine volume was 0.24 L less ($P = 0.001$). The urinary relative supersaturations of calcium oxalate ($P = 0.03$) and brushite ($P = 0.002$) were lower in black women. No other significant differences were observed. Differences in urinary calcium and pH persisted after multivariate adjustment and after exclusion of participants who were taking thiazide diuretics or those with diabetes. In conclusion, black women excrete less urinary calcium and have a higher urinary pH than do white women. These differences are not explained by differences in age, body mass index, or diet and may account for the lower incidence of both nephrolithiasis and osteoporosis in black women.

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Black women are less likely to develop kidney stones and have greater bone mass than their white counterparts. After adjustment for region of residence, age, and diuretic use, the odds ratio of prevalent nephrolithiasis in black compared with white women was 0.35 (1). The incidence of hip fracture among black women is approximately half that of white women (2–4), and studies have reported higher bone mass density in black women at most skeletal sites (5–9).

The underlying mechanisms of these racial differences are unclear. Previous data suggest that African Americans, Europeans of African descent, and black South Africans excrete lower levels of urinary calcium than do white individuals (6,10,11). In one study of stone formers, the frequency of hypercalciuria was 25% in black individuals compared with 67% in white individuals (12). Several small metabolic studies of black individuals who were on controlled diets demonstrated lower levels of urinary calcium than white control subjects (13–16), which in one trial was attributed to higher levels of circulating parathyroid hormone (PTH) (13). However, previous studies that compared urine composition of individuals of African descent with white individuals are limited. Population-based studies of urinary calcium excretion included only stone formers (12) or did not account for differences in body size or the dietary intake of sodium and animal protein (6,10,11), factors that have marked effects on urinary calcium excretion (17,18). Controlled metabolic studies that compared racial differences in urine composition included only male participants (19,20), yielded inconsistent results (19,20), or did not measure urinary factors that potentially are important to both bone and kidney stone disease, such as urinary pH and citrate (13–16).

To compare the urine composition of black and white women, we studied the urine pH, urine volume, and 24-h urinary excretion of calcium, citrate, oxalate, uric acid, sodium, magnesium, potassium, phosphate, sulfate, and creatinine in 146 black and 330 white women in the Nurses' Health Study (NHS). All participants were postmenopausal at the time of urine collection and had no history of kidney stones.

Materials and Methods

Study Population and Ascertainment of Race

In 1976, 121,700 female registered nurses who were between the ages of 30 and 55 enrolled in the NHS by completing and returning an initial questionnaire that provided detailed information on medical history, lifestyle, and medications. This cohort is followed by biennial mailed questionnaires. For the purposes of this study, any participant who identified herself as African-American in the 1992 questionnaire was assigned as African-American. Participants who identified themselves as Southern European, Scandinavian, other Caucasian, or any combination of these and did not also select African-American or Asian were assigned as Caucasian.

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Ascertainment of Other Covariates

Information on age was obtained on the baseline questionnaire. Thiazide use was determined in 1980 and 1982 and then every 6 yr until 1994, when biennial updates started. Information on hypertension and diabetes was obtained at baseline and then every 2 yr. The validity of these self-reported diseases has been documented (21,22). Information on weight and height was obtained on the baseline questionnaire. Self-reported weight was updated every 2 yr. Body mass index (BMI) was calculated as the weight in kilograms divided by the square of height in meters. Self-reported weight has been validated (23). Self-reported weights from 140 women were highly correlated with values that were obtained by technicians who visited the participants at home ($r = 0.97$) (23).

The semiquantitative food frequency questionnaire (FFQ; first mailed in 1980) asked about the average use of more than 130 foods and beverages during the previous year. In addition, respondents provided information on the use of nutritional supplements, taken either alone or in multivitamin form. Subsequently, a version of this FFQ has been mailed to study participants every 4 yr. The reproducibility and the validity of the FFQ have been documented (24).

Nutrient intake was computed from the reported frequency of consumption of each specified unit of food and from US Department of Agriculture data on the content of the relevant nutrient in specified portions. The intake of supplemental vitamins and minerals in multivitamin or isolated form was determined by the brand, type, and frequency of reported use.

Urine Collection Procedure

Twenty-four-hour urine samples were collected in two cycles as part of an ongoing study to compare the urine composition of individuals with and without kidney stone disease. All participants in this study did not have a history of kidney stones at the time of urine collection. In the first cycle, which spanned from 1994 to 1999, we obtained one 24-h urine collection from 99 randomly selected non–stone-forming participants. In the second cycle, which started in 2001, we randomly selected additional non–stone-forming participants and requested two 24-h urine collections. In the first cycle, participants were ineligible when they were older than 65 yr or had a history of cancer or cardiovascular disease. In the second cycle, participants were ineligible when they were older than 75 yr or had a history of cancer (other than nonmelanoma skin cancer). In 2005, we requested 24-h urine collections from 400 randomly selected non–stone-forming black participants.

The 24-h urine collection participation and completion rates in white NHS I participants were 48 and 86%, respectively. The participation and completion rates in the black urine collection were 44 and 82%, respectively. There were no substantial differences between women who collected urine and women who did not according to age, BMI, and intake of dietary calcium, sodium, and animal protein. In this study, 335 white and 146 black participants provided at least one 24-h urine collection.

The 24-h urine collections were performed using the system provided by Mission Pharmacal (San Antonio, TX). We sent a kit that contained all of the necessary supplies, including a 4-L jug with a marker-impregnated sponge attached to the bottom, to participants who agreed to collect a 24-h specimen. The jugs also contained preservative to prevent bacterial growth. To assist in urine collection, female participants were sent specially designed "hats." Upon completion of the collection, participants poured samples into two small vials, one of which contained acid preservative. The vials were returned to Mission Pharmacal in a prepaid, self-addressed FedEx mailer. The concentration of the marker was used to calculate the total volume of the collection.

Analytic Procedures Used for the Urine Measurements

Calcium and magnesium were measured by an atomic absorption spectrophotometer. Creatinine, uric acid, citrate, and phosphorus were measured by a Cobas centrifugal analyzer. Oxalate was analyzed by ion chromatography. Sodium and potassium were determined directly by flame emission photometry. The urine pH was measured by a pH electrode. We previously sent blinded split samples to assess reproducibility; the coefficients of variation for all factors analyzed were <10%.

Statistical Analyses

In the primary analysis, we included participants who provided a single 24-h urine collection. When participants submitted more than one urine collection, we used the first sample. To exclude those with incomplete collections, we limited the analysis to participants whose 24-h urine creatinine values were >600 mg (five white participants were excluded using this criterion). Uric acid results were obtained after exclusion of participants with uric acid crystals noted in the 24-h urine collection (10 white and 15 black participants had uric acid crystals). In secondary analyses, we restricted the study to the 218 white and 132 black participants who submitted two 24-h urine collections. In these analyses, participants were included only when the creatinine value in each urine collection differed by <30% from the mean creatinine value of the two collections. Values for each urinary factor were obtained by calculation of the arithmetic mean of both collections.

ANOVA was used to compare the mean value of each urinary factor in white and black women. Multivariate linear regression was used to determine the relation between race and urinary factors after adjustment simultaneously for multiple covariates. Because residuals in the regression analyses approximated the normal distribution and sample size was relatively large, we did not transform non-normally distributed urinary factors. Variables that were included in the multivariate analyses were age (continuous); BMI (continuous); urinary pH; urine volume; and the 24-h urinary excretion of calcium, citrate, oxalate, uric acid, sodium, magnesium, potassium, phosphate, sulfate, and creatinine (all in quartiles).

All P values are two tailed. We calculated 95% confidence intervals (CI) for all estimates that were obtained in the linear regression analyses. All data were analyzed by using SAS software, version 9.1 (SAS Institute, Cary, NC). The research protocol for this study was reviewed and approved by the institutional review board of Brigham and Women's Hospital.

Results

Characteristics of white and black participants are displayed in Table 1. Black women were older, had a higher BMI, were more likely to have hypertension and diabetes, and were more likely to use thiazide diuretics than their white counterparts. On average, black participants had lower average daily intakes of animal protein and total calcium.

The mean values of each urinary factor by race are shown in Table 2. Per 24-h period, black participants excreted 189 mg more creatinine ($P < 0.001$), 65 mg less calcium ($P < 0.001$), 2 mg more oxalate ($P = 0.06$), 9 mEq less potassium ($P < 0.001$), 11 mg less magnesium ($P = 0.003$), 120 mg less phosphate ($P < 0.001$), and 3 mmol less sulfate ($P < 0.001$) than white participants. The urine pH of black participants was 0.11 units higher ($P = 0.03$) and the urine volume was 0.24 L less ($P = 0.001$) than that in their white counterparts. The urinary relative supersaturations of calcium oxalate ($P = 0.03$) and brushite ($P = 0.002$) were lower in black participants.

Table 1. Mean demographic and dietary factors in white and black women[a]

Parameter	White (n = 330)	Black (n = 146)
Age (yr)	65.8 (65.9; 58.5 to 73.5)	67.5 (67.9; 61.0 to 73.0)
BMI (kg/m^2)	26.1 (25.1; 20.8 to 32.8)	29.3 (28.2; 23.2 to 35.9)
Hypertension (n [%])	150 (45.5)	105 (71.9)
Diabetes (n [%])	26 (7.9)	21 (14.4)
Thiazide use (n [%])	38 (11.5)	39 (26.7)
Hormone replacement	177 (53.6)	46 (31.5)
Therapy (n [%])		
Supplement use (n [%])		
multivitamins	201 (60.9)	87 (59.6)
calcium	211 (63.9)	74 (50.7)
vitamin D	111 (33.6)	69 (47.3)
Dietary factors		
animal protein (g/d)	50 (49; 33 to 68)	44 (44; 30 to 60)
total calcium (mg/d)	1297 (1179; 634 to 2061)	1029 (857; 447 to 1829)
total vitamin D (IU/d)	448 (449; 116 to 783)	440 (407; 88 to 906)
% calories from		
fat	29.0	27.8
carbohydrates	53.4	57.5
protein	18.1	16.7

[a]Data are means (median; 10th to 90th percentile range) except where noted.

Table 2. Mean 24-h urinary excretion in white and black women[a]

Parameter	White (n = 330)	Black (n = 146)	P
Creatinine (mg)	1036 (1017; 775 to 1326)	1225 (1199; 953 to 1528)	**<0.001**
Calcium (mg)	183 (170; 73 to 302)	118 (99; 32 to 240)	**<0.001**
Oxalate (mg)	28 (26; 18 to 40)	30 (29; 20 to 41)	0.06
Citrate (mg)	686 (657; 332 to 1012)	653 (598; 259 to 1056)	0.28
Uric acid (mg)	448 (435; 281 to 662)	450 (417; 284 to 639)	0.93
Sodium (mEq)	137 (127; 72 to 204)	142 (139; 79 to 207)	0.30
Potassium (mEq)	64 (61; 39 to 94)	55 (54; 33 to 80)	**<0.001**
Magnesium (mg)	104 (98; 63 to 152)	93 (89; 51 to 151)	**0.003**
Phosphate (mg)	743 (706; 465 to 1040)	623 (592; 357 to 881)	**<0.001**
Sulfate (mmol)	17 (17; 10 to 26)	14 (13; 8 to 22)	**<0.001**
pH (U)	6.08 (6.07; 5.42 to 6.71)	6.19 (6.25; 5.40 to 6.91)	**0.03**
Volume (L)	2.03 (1.96; 1.14 to 2.96)	1.79 (1.68; 0.91 to 2.80)	**0.001**
RSS CaOx	1.24 (1.07; 0.48 to 2.18)	1.06 (0.82; 0.30 to 2.25)	**0.03**
RSS brushite	1.09 (0.79; 0.20 to 2.23)	0.80 (0.53; 0.11 to 1.77)	**0.002**
RSS uric acid	1.12 (0.81; 0.18 to 2.65)	1.03 (0.60; 0.13 to 3.00)	0.41

[a]Data are means (median; 10th to 90th percentile range). Statistically significant P values are in boldface. RSS, relative supersaturation.

Multivariate linear regression results comparing the differences in urinary factors by race are displayed in Table 3. After adjustment for age, BMI, and quartile of each urinary factor, black participants excreted 40.9 mg less calcium (95% CI 21.8 to 60.0), 10.2 mEq less potassium (95% CI 6.6 to 13.9), and 119.1 mg less phosphate (95% CI 75.3 to 162.8) each day than white participants. Black participants excreted 74.9 mg more citrate (95% CI 5.1 to 144.7) than did white participants, and urine pH in black participants was 0.21 units higher (95% CI 0.09 to 0.32). Black participants excreted 2.4 mg more oxalate (95% CI −0.06 to 4.8). No association was observed between race and urinary citrate excretion until after adjustment for urinary potassium, calcium, and sulfate.

Restriction of the analyses to participants with two urine

Table 3. Multivariate adjusted differences in 24-h urinary excretion between black and white women[a]

Parameter	Difference	95% CI
Calcium (mg)	**−40.9**	−60.0 to −21.8
Oxalate (mg)	2.4	−0.06 to 4.8
Citrate (mg)	**74.9**	5.1 to 144.7
Uric acid (mg)	7.3	−21.9 to 36.5
Sodium (mEq)	10.1	−2.0 to 22.2
Potassium (mEq)	**−10.2**	−13.9 to −6.6
Magnesium (mg)	−3.3	−11.7 to 5.0
Phosphate (mg)	**−119.1**	−162.8 to −75.3
Sulfate (mmol)	−1.0	−2.1 to 0.2
pH (U)	**0.21**	0.09 to 0.32
Volume (L)	−0.13	−0.30 to 0.04

[a]White race is the referent. Differences are adjusted for age, body mass index, urinary creatinine, and all other urinary factors. Statistically significant values are in boldface. CI, confidence interval.

collections yielded similar results, except that differences in urinary oxalate increased and became statistically significant. Black participants excreted 30 mg and white participants excreted 26 mg/d oxalate ($P < 0.001$). After adjustment for age, BMI, and quartile of each urinary factor, black participants excreted 3 mg more oxalate (95% CI 0.8 to 5.2) than did white participants.

Exclusion of participants who were taking thiazide diuretics resulted in slightly higher mean urinary calcium levels in both white (187 mg) and black participants (124 mg) but did not change the relation between race and urinary calcium excretion. Exclusion of participants with a history of diabetes did not materially change the results. Additional adjustment for smoking, hypertension, use of hormone replacement therapy, and the intake of calcium and animal protein also did not change the results.

Discussion

We observed numerous and marked differences in the urine composition of black as compared with white women. Black women excreted significantly less urinary calcium, potassium, magnesium, phosphate, and volume than did white women but had higher urinary pH. Although black women had marginally higher urinary oxalate, the lower urinary calcium resulted in lower relative urinary supersaturations of both calcium oxalate and brushite.

Differences in body size, diet, and frequency of thiazide use are unlikely to account for the markedly lower levels of urinary calcium that were excreted by the black participants in our study. Previous studies described a positive association between BMI and urinary calcium (18,25), but black participants in our study had higher BMI than did their white counterparts. Lower sodium and animal protein intake also results in decreased urinary calcium (17), but 24-h urinary sodium levels in black women were higher than those in white women, and the racial difference in urinary calcium excretion persisted after adjustment for urinary sulfate, a reliable marker of animal protein intake (26). Of note, black women had a lower intake of calcium than did white women, despite similar levels of vitamin D intake. However, the racial difference in urinary calcium persisted after adjustment for calcium intake. Furthermore, even if black women absorbed a diminished fraction of intestinal calcium relative to white women (estimates of intestinal calcium absorption in healthy individuals vary but approximate 20% [27]), the magnitude of the difference in calcium intake is unlikely to account for the marked difference in urinary calcium excretion. Finally, the racial difference in urinary calcium excretion remained after exclusion of participants who were taking thiazide diuretics.

We cannot exclude relative vitamin D deficiency in black participants as the potential cause of lower urinary calcium excretion. Indeed, data from other populations show that serum 25-hydroxyvitamin D levels may be lower in black women (28). However, proposed mechanisms for the lower urinary calcium excretion that was observed in black women also should account for previous data demonstrating higher bone mass in black as compared with white women (5–9). For this reason, it is unsatisfactory to attribute low levels of urinary calcium solely to relative vitamin D deficiency or to reduced calcium intake. Several small metabolic studies suggested that black individuals may exhibit an isolated skeletal resistance to the actions of PTH. One trial that included 12 black and 14 white individuals who were consuming a controlled diet showed similar levels of serum calcium and phosphate but higher levels of serum PTH, higher levels of 1,25-dihydroxyvitamin D, and lower levels of serum 25-hydroxyvitamin D and urinary calcium in black participants (13). Another metabolic trial demonstrated decreased serum bone resorption markers in black compared with white individuals after intravenous infusion of PTH (29).

Ours is the first large-scale study to demonstrate higher urinary pH in black compared with white women. The reason for the lower urinary pH in white women is unclear. Factors that are associated with low urine pH in healthy individuals include higher weight, older age, and greater dietary acid intake (30–33). Because white women in our study had lower

BMI and were younger than the black women, differences in body size and age do not explain the lower urinary pH. Furthermore, the difference in urine pH persisted after adjustment for BMI, 24-h urinary creatinine excretion, and age. Because we adjusted our analyses for urinary potassium and sulfate, urinary factors that correlated highly with levels of dietary alkali and acid (34), respectively, it is unlikely that dietary differences accounted for the lower urinary pH in white women. Finally, our results did not change after we adjusted for the intake of dietary animal protein and potassium.

Lower urinary pH in white women could be explained by a relative defect in renal ammonium production, as described in individuals with insulin resistance (35), or by an increase in titratable acid excretion. Although we did not measure renal ammonium excretion, the black participants in the study had higher BMI than did white participants, and there is little reason to think that the white participants in our study were more insulin resistant. We are unaware of any literature suggesting a relative decrease in renal ammonium synthesis in white compared with black individuals. Of note, recent data suggest that individuals with diabetes may produce more organic acid than a group of normal volunteers (36), and it is intriguing to speculate whether there are racial differences in organic acid production.

Although purely hypothetical, an increased production of organic acid in white compared with black women could unify the urinary pH and calcium findings in our study. Chronic metabolic acidosis results in increased renal calcium excretion (37–39), perhaps by downregulating renal calcium transport proteins (40). Higher levels of organic acid production in white *versus* black women may result in increased renal calcium wasting, larger negative calcium balance, and greater concomitant reductions in bone mass.

The limitations of our study deserve mention. An important limitation is the lack of biochemical parameters that would allow for a full characterization of calcium metabolism and acid-base status. For example, we do not have data on serum PTH levels, serum vitamin D levels, or serum bicarbonate. We also did not measure urinary ammonium or titratable acids and therefore cannot measure renal net acid excretion. Another limitation is that weight, height, and dietary intake were self-reported. However, self-reported weight, height, and diet have been validated in these cohorts. Other potential sources of error include over- and undercollection of 24-h urine samples. However, there is little reason to believe that black individuals, as a group, would be more likely to submit over- or undercollections. In addition, we adjusted our analyses for the 24-h excretion of urinary creatinine. Finally, our study included only postmenopausal women. The racial differences that we observed may not apply to men or to younger women.

Conclusion

We observed marked differences in 24-h urine composition between black and white women with no history of kidney stones. Lower urinary calcium excretion in black women resulted in lower relative urinary supersaturation of calcium salts and may account for the lower rates of nephrolithiasis that have been observed in this racial group. Because lower urinary calcium excretion and higher urinary pH in black women could not be explained by differences in age, BMI, or diet, there may be an intrinsic physiologic difference in calcium and acid-base metabolism that contributes to the higher incidence of both kidney stones and osteoporosis in white women.

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Disclosures

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