Racial differences in potassium homeostasis in response to differences in dietary sodium in girls[1–3]

Cristina Palacios, Karin Wigertz, Berdine R Martin, Michelle Braun, J Howard Pratt, Munro Peacock, and Connie M Weaver

ABSTRACT

Background: Racial differences in the renal disposition of potassium may be related to mechanisms for the greater susceptibility to hypertension in blacks than in whites.

Objective: Our objective was to study the racial differences in the renin-angiotensin-aldosterone system and in potassium balance in black and white girls consuming a controlled diet that was low in potassium with 2 amounts of sodium intake (low compared with high).

Design: The studies reported here were performed in 40 black and 28 white girls, aged 11–15 y, under highly controlled metabolic conditions. The studies comprised 2 sessions of 20-d metabolic balance sessions, at 2 amounts of dietary sodium intake (58 and 170 mmol $\cdot$ L^{-1} $\cdot$ d^{-1}), in a crossover design and with a constant dietary potassium intake of 50 mmol $\cdot$ L^{-1} $\cdot$ d^{-1}. Repeated-measures analysis of variance was used to test for racial differences in potassium output and retention by sodium intakes.

Results: Thirty black and 20 white girls completed the study. Urinary potassium excretion was lower in blacks than in whites, regardless of sodium intake ($P < 0.05$), with no differences in fecal or sweat potassium excretion. Cumulative potassium retention was significantly higher in blacks while consuming the low sodium diet. Plasma aldosterone concentrations after upright posture were significantly lower in blacks than in whites but were similar when supine, as were urinary aldosterone excretion rates. On week 3, blood pressure, body weight, urinary volume, creatinine, and serum sodium and potassium were similar.

Conclusion: The well-known racial difference in urinary potassium excretion appears to be at least in part due to greater renal retention of potassium in black girls. *Am J Clin Nutr* 2010;91:597–603.

INTRODUCTION

Racial differences in the renal disposition of potassium may be related to mechanisms for the increased susceptibility to hypertension in blacks (1, 2). Blacks typically show a lower urinary excretion rate of potassium than do whites in both adults and children (3–7), even when consuming diets containing similar amounts of potassium (8–11). This racial difference in potassium homeostasis may be related to differences in the renin-angiotensin-aldosterone system. In comparison with whites, blacks have lower plasma renin activity (PRA) in both adults (12) and children (13) and are more likely to develop salt-sensitive hypertension (14), which is consistent with greater retention of sodium. Plasma aldosterone and urinary aldosterone excretion have also been reported to be lower in blacks than in whites (7, 15), which could be secondary to the increased retention of sodium, suppressing angiotensin II, the principal stimulus of aldosterone secretion. Lower aldosterone concentrations in blacks could also be related to a diet lower in potassium, because potassium is an important stimulus of aldosterone secretion (16).

Lower aldosterone production in blacks could also result from a diet lower in potassium and/or a diet higher in sodium. The mechanism could have relevance to hypertension risk in blacks. Therefore, we sought to study the racial differences in the renin-angiotensin-aldosterone system and in potassium balance in black and white girls while consuming a controlled diet low in potassium with 2 amounts of sodium intake (low compared with high). The combination of low potassium/high sodium intakes may further suppress the aldosterone production in black girls. The studies reported here were carried out under highly controlled metabolic conditions. Food intake and excretion rates, with complete 24-h collections of urine and feces over a period of 20 d under each condition (low and high sodium intakes) were carefully monitored during the entire study.

SUBJECTS AND METHODS

Subjects

Forty black and 28 white girls from Indiana aged 11–15 y participated in metabolic studies that consisted of two 20-d sessions in the summers of 1999 and 2000. Subjects were randomly assigned to receive a low-sodium diet (57 mmol $\cdot$ L^{-1} $\cdot$ d^{-1}) or a high-sodium diet (174 mmol $\cdot$ L^{-1} $\cdot$ d^{-1}) with the use of a randomized crossover design, in which intakes of protein, fat, magnesium, phosphorus, and potassium were fixed. Potassium

[1] From the Nutrition Program, Graduate School of Public Health, Medical Sciences Campus, University of Puerto Rico, San Juan, Puerto Rico (CP); the Department of Foods and Nutrition, Purdue University, West Lafayette, IN (KW, BRM, MB, and CMW); and the Department of Medicine, Indiana University School of Medicine and the VA Medical Center, Indianapolis, IN (JHP and MP).

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[3] Address correspondence to C Palacios, Nutrition Program, Graduate School of Public Health, Medical Sciences Campus, University of Puerto Rico, San Juan, Puerto Rico 00935. E-mail: cristina.palacios@upr.edu.

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intake was set at 50 mmol · L^{-1} · d^{-1}, which is about half of the Dietary Reference Intake (17). Between sessions, there was a 2-wk washout period, in which subjects returned home and to their usual diet.

Applicants were excluded from the study if they were aged <11 or >15 y, their body mass index (BMI) was <15th or >85th percentile for age, or if there was a history of amenorrhea, pregnancy, eating disorders, or use of oral contraceptives or tobacco. The subjects were housed at Purdue University for the study periods. They were strictly supervised to ensure consumption of only the study diet and closely monitored with respect to sample collections. All subjects were studied under protocols approved by the Purdue University Use of Human Subjects Research Committee.

Measurements

Full details of the metabolic studies have been previously published (18). In brief, dietary potassium intake was estimated from 6 d of diet records before the study began and analyzed with the computer program Nutritionist IV Diet Analysis (First Databank Division, San Bruno, CA). Tanner score was assessed at baseline by pubertal development with the use of a self-assessment of breast and pubic hair stage (19). Postmenarcheal age was defined as months after menarche.

A 4-d-cycle menu, with 3 meals and 2 snacks, was designed for the study. Daily food portions were maintained at a constant amount and were equal for all subjects. Subjects were strictly supervised at all times to ensure compliance and to avoid consumption of other foods. The food and beverages were prepared with deionized water and weighed to the nearest 10th of a gram on digital scales. Duplicates of each of the day's meals were homogenized and analyzed due to variations of food batches.

Complete urine and fecal collections were performed in acid-washed containers for the 20 d of the study on a daily basis. Urine was pooled as 24-h samples, which were analyzed daily for creatinine to check compliance. The first 24-h urine collection was used to estimate usual potassium intake. Fecal samples were also pooled for each 24-h period, and completeness of collections was determined by using polyethylene glycol by turbidimetric assay (20). Balance was determined daily by the following equation:

$$\text{Daily potassium balance} = \text{daily potassium intake} - \text{daily potassium excretion (feces and urine)} \quad (1)$$

Whole-body sweat was collected after 14 d of acclimation and adaptation to the diet for 24 h by a whole-body scrub-down procedure during each session as previously described (21). Briefly, participants wore 100% cotton, long-sleeve shirts and long pants (Lands End, Oakham, United Kingdom) and 100% cotton socks. This suit and all the towels, washcloths, and pillowcases used for the study had been chemically treated previously. Before wearing the suit, subjects were rinsed thoroughly with a detergent solution and then dried with the chemically treated towel and dressed. Breathable paper outer garments and shoes (Fisher, Pittsburgh, PA) were worn to protect the clothing from contamination. Sweat loss from the scalp and hands was not collected. Subjects were asked to wash their hands as necessary during the test. Subjects collected facial sweat on the inner part of their suit. After 24 h, subjects were again rinsed with a chemically treated washcloth while inside a plastic bag and dried with a chemically treated towel. Potassium was analyzed from the body, clothing, pillowcase, washcloths, and towel rinses.

Blood was collected on day 20 of each diet period for analysis of PRA, aldosterone, and serum potassium and sodium. Blood samples were drawn at 0700 after subjects had been recumbent overnight and at 0900 after they sat for 1 h and then stood for 1 h. For measurement of urinary aldosterone, an aliquot of the 24-h urine sample collected on day 20 was used.

Body weight was recorded daily by using a professional electronic scale (Health O Meter, Bridgeview, IL). Blood pressure was measured every other day while subjects were in a recumbent position by using a sphygmomanometer (Hawksely and Sons, Sussex, United Kingdom). Korotokoff sound 1 was used for systolic and sound 5 for diastolic. Blood pressure was recorded for each subject while supine by the same observer throughout the study at 0700. Lean body mass was determined by dual-energy X-ray absorptiometry (Lunar Corp, Madison, WI).

Dietary, urinary, fecal, and sweat potassium were measured by using atomic absorption spectrophotometry (5100 PC; Perkins Elmer, Waltham, MA). Unacidified urinary aliquots were measured for creatinine by a kinetic modification of Jaffe's colorimetric assay (Cobas Mira Plus; Roche Diagnostic Systems, Nutley, NJ). PRA was measured by using a Clinical Assays GammaCoat radioimmunoassay kit (Baxter Health Care, Cambridge, MA). Plasma and urinary aldosterone concentrations were measured by radioimmunoassay using kits from Diagnostic Products Corporation (Los Angeles, CA). Serum potassium and sodium were measured by using an automated colorimetric method (Cobas Mira Plus; Roche Diagnostic Systems).

Statistical methods

Means and SDs were calculated for each variable. Normality was tested by the Shapiro-Wilk test and by Q-Q plots. Nonnormally distributed variables (urinary potassium) were transformed to improve normality (square root). Student's *t* test was used to assess for differences in general characteristics between white and black girls. A repeated-measures analysis of variance (ANOVA) was performed to test for racial differences in urinary and fecal potassium excretion and for cumulative potassium retention measured daily for 20 d due to the 2 treatments. At the end of the study, a mixed-model ANOVA was used to assess the effects of the low- and high-sodium diets and race with regard to plasma and urinary aldosterone, PRA, serum sodium and potassium, weight, and blood pressure. Statistical significance was set at $P < 0.05$. Microsoft Excel for Windows 2000 (version 7.0; Microsoft Corporation, Redmond, WA) and the Statistical Analysis System (SAS Institute, Cary, NC) program were used for all statistical analyses. Values are expressed as means ± SDs.

RESULTS

Subjects

Thirty black and 20 white girls completed both sessions of the metabolic studies, whereas 8 white and 10 black girls completed only one session of the metabolic studies. Only the girls

TABLE 1
Baseline characteristics of subjects[1]

Characteristics	Blacks (n = 30)	Whites (n = 20)
Age (y)	12.5 ± 1.0	12.9 ± 0.9
Height (m)	1.57 ± 0.06	1.59 ± 0.08
Weight (kg)	52.7 ± 13.0	54.5 ± 14.4
Postmenarcheal age (mo)	5.5 ± 11.1	4.8 ± 9.5
Tanner score	4.0 ± 0.8	3.7 ± 0.8
BMI (kg/m^2)	21.1 ± 4.2	21.6 ± 5.0
Lean body mass (%)	74.1 ± 11.2	73.6 ± 10.0
Estimated dietary potassium intake (mmol · L^{-1} · d^{-1})	59.6 ± 6.2	64.9 ± 5.9
Estimated dietary sodium intake (mmol · L^{-1} · d^{-1})	121.5 ± 8.5	122.0 ± 7.5
Baseline urinary potassium (mmol · L^{-1} · 24 h^{-1})	36.5 ± 6.0	37.0 ± 5.3
Baseline urinary sodium (mmol · L^{-1} · 24 h^{-1})	96.5 ± 17.6	86.9 ± 9.7
Baseline systolic blood pressure (mm Hg)	106.1 ± 9.6	103.8 ± 10.1
Baseline diastolic blood pressure (mm Hg)	57.4 ± 11.7	56.4 ± 8.9

[1] All values are means ± SDs. No significant differences were observed between races ($P < 0.05$).

completing both sessions of the study were included in the analysis. Black and white girls had similar baseline characteristics (**Table 1**). Dietary potassium intake, estimated from 6 d of dietary records before the study began, was ≈60 mmol · L^{-1} · d^{-1}; this intake was similar between white and black girls. However, the first 24-h urine sample collected at baseline, which is a more reliable measure for estimating dietary intake, showed only ≈37 mmol · L^{-1} · d^{-1}. This was also similar between blacks and whites but was lower than with the balance diet administered during the study, which contained 50 ± 10 mmol potassium · L^{-1} · d^{-1} . The sodium content of the balance diet was 58.1 ± 2.3 mmol · L^{-1} · d^{-1} during the low intake of sodium and 170 ± 4.1 mmol · L^{-1} · d^{-1} during the high intake of sodium. These intakes were also much lower or higher than subjects' usual diets, respectively. Blood pressure was also similar between the 2 groups.

Potassium homeostasis

Urinary potassium excretion was similar between blacks and whites at baseline (first 24 h). However, after being exposed to the controlled diet, there was a significant increase in urinary potassium excretion in both races regardless of sodium intake (**Figure 1**). Repeated-measures ANOVA showed a significant difference in daily urinary potassium excretion between races, whereas black girls excreted significantly less urinary potassium from day 2 to the end of the study, regardless of sodium intake (Figure 1). The ratio of urinary potassium to urinary creatinine was constant throughout the study, which is consistent with a steady state condition. Fecal potassium excretion was similar between race groups and dietary sodium intake by repeated-measures ANOVA, which

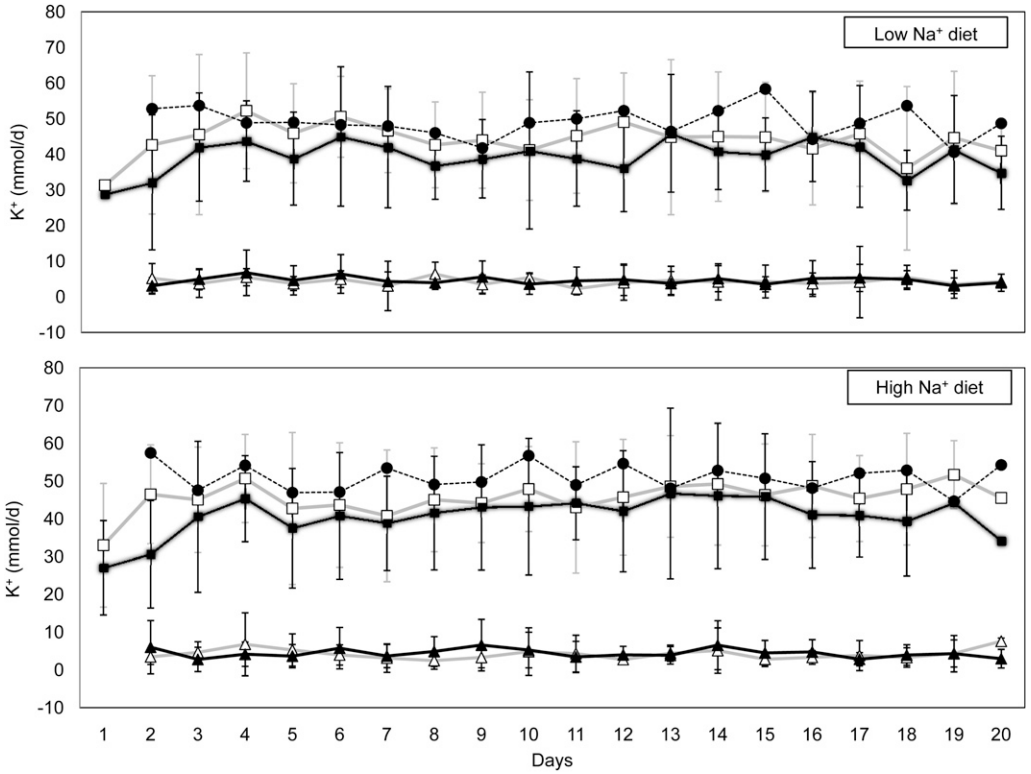

FIGURE 1. Mean (±SD) daily 24-h potassium (K^+) intake and urinary and fecal excretion in blacks and whites. Repeated-measures ANOVA showed that blacks excreted significantly less urinary potassium from day 2 to the end of the study compared with whites while consuming either the low- or high-sodium (Na^+) diet ($P < 0.05$). No significant differences were observed with fecal output. □, urinary output in whites; ■, urinary output in blacks; Δ, fecal output in whites; ▲, fecal output in blacks; ●, diet.

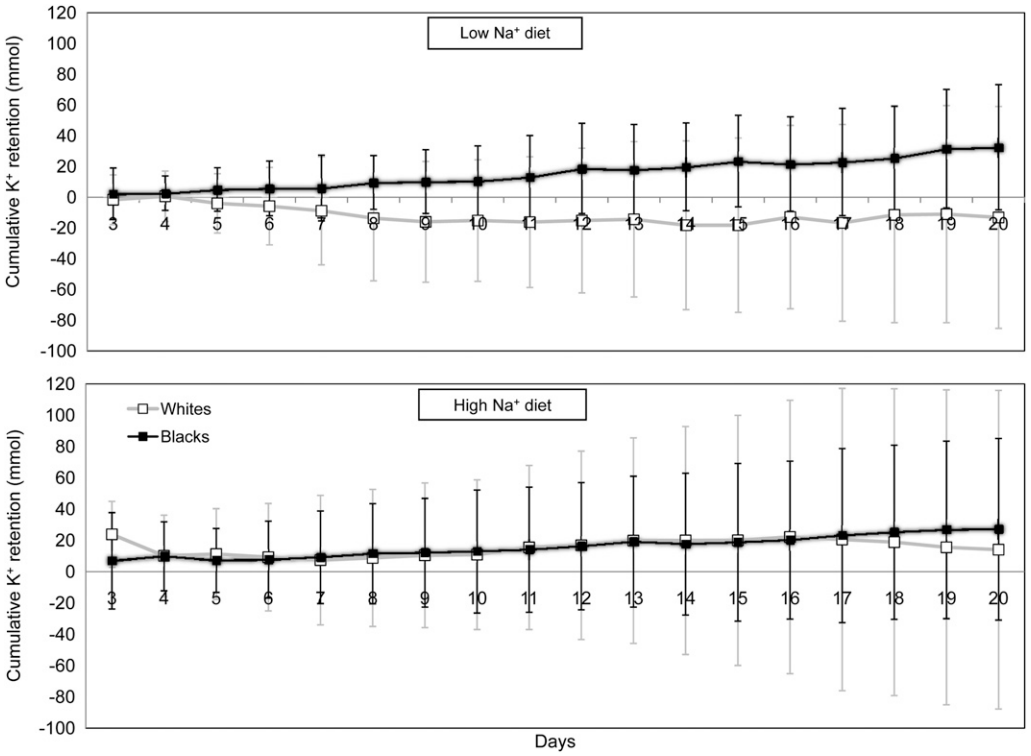

FIGURE 2. Mean (±SD) cumulative potassium (K^+) retention during the study in black and white girls on low- and high-sodium (Na^+) diets. Repeated-measures ANOVA showed that blacks had greater cumulative potassium retention while consuming the low-sodium diet than did whites ($P < 0.05$). No racial differences were observed for either group while consuming the high-sodium diet.

only represented 8–11% of dietary potassium intake (Figure 1). Cumulative potassium retention is shown in **Figure 2**. Repeated-measures analysis showed a significant interaction between race and sodium intake. Therefore, the analyses were run separately by sodium intake. Potassium retention was significantly higher in the black girls while consuming the lower-sodium diet compared with whites, but no racial differences were seen while consuming the higher-sodium intake (Figure 2). In addition, no differences were observed in cumulative potassium retention between the low and high sodium intakes.

As previously reported (21), a single 24-h, whole-body, total sweat potassium output was measured by using a whole-body detailed scrub procedure after 2 wk of acclimation to the study diet. We showed that there were no racial differences in sweat potassium or sodium output (**Table 2**). In addition, potassium sweat output was not affected by dietary sodium, and it represented ≈10% of dietary potassium intake.

TABLE 2
Systolic and diastolic blood pressure, body weight, urinary volume, creatinine and sodium excretion, and serum and sweat-output sodium and potassium in black and white girls during week 3 of the study period

	Low-sodium diet		High-sodium diet	
Measurement	Blacks (n = 30)	Whites (n = 20)	Blacks (n = 30)	Whites (n = 20)
Systolic blood pressure (mm Hg)	102.0 ± 8.9	100.0 ± 8.7	100.4 ± 8.3	100.8 ± 7.7
Diastolic blood pressure (mm Hg)	54.7 ± 10.6	54.5 ± 10.0	56.4 ± 11.2	56.2 ± 9.9
Body weight (kg)	52.6 ± 25.3	53.0 ± 34.0	53.0 ± 25.0	53.4 ± 35.0
Urinary volume (mL)	1269.8 ± 419.9	1252.8 ± 326.6	1295.9 ± 365.6	1347.3 ± 312.3
Urinary creatinine (mg/d)	880.7 ± 218.2	842.7 ± 204.4	874.7 ± 272.6	854.1 ± 195.1
Urinary sodium (mmol · L^{-1} · d^{-1})	66.7 ± 10.6	73.6 ± 12.8	130.3 ± 21.0	158.8 ± 15.4[1]
Sweat potassium (mmol · L^{-1} · d^{-1})[2]	5.41 ± 1.96	5.42 ± 1.83	5.55 ± 2.39[3]	5.69 ± 2.46
Sweat sodium (mmol · L^{-1} · d^{-1})[2]	3.96 ± 1.87	4.28 ± 2.30	5.43 ± 2.06	7.50 ± 7.31
Serum sodium (mEq/L)[4]	140.9 ± 2.37	142.2 ± 1.50	140.8 ± 3.07	143.3 ± 2.86
Serum potassium (mEq/L)	4.46 ± 0.25	4.38 ± 0.31	4.44 ± 0.38	4.32 ± 0.37

[1] Significantly different from blacks on the same diet, $P < 0.05$ (ANOVA).
[2] Data from reference 21.
[3] Significantly different from the low-sodium diet, $P < 0.05$ (ANOVA).
[4] Data for 27 girls were eliminated due to an instrument error that resulted in physiologically high concentrations.

PRA and plasma aldosterone

Results for 8 white and 15 black girls for plasma aldosterone and PRA have been previously reported (18), and in this report we add the results of an additional 12 white and 15 black girls. Plasma aldosterone (**Figure 3**) and PRA (**Figure 4**) showed the expected increases after 2 h of upright posture, from 0700 to 0900 ($P < 0.0001$), and with the consumption of the low-sodium diet in both whites and blacks ($P < 0.001$). There was a lesser increase in plasma aldosterone in blacks compared with whites with the upright posture on either the low- or the high-sodium diet ($P < 0.05$). Postural increments (Δ) of plasma aldosterone were also significantly lower on the low-sodium diet in blacks (12.9 ± 9.55 ng/dL) compared with whites (19.5 ± 8.47 ng/dL) and on the high-sodium diet in blacks (8.4 ± 6.79 ng/dL) compared with whites (12.3 ± 6.25 ng/dL) ($P < 0.05$). These changes were significantly reduced by the high-sodium diet. Postural increments (Δ) of PRA were only significantly lower on the low-sodium diet in blacks ($2.4 \pm 1.20\ ng \cdot L^{-1} \cdot s^{-1}$) compared with whites ($3.2 \pm 1.29\ ng \cdot L^{-1} \cdot s^{-1}$) ($P < 0.05$) but similar between races on the high-sodium diet (2.3.±1.89 and 2.2.±1.25 $ng \cdot L^{-1} \cdot s^{-1}$ in blacks and whites, respectively). The ratio of Δaldosterone/ΔPRA was lower in blacks (5.45) than in whites (6.08) on the low-sodium diet and even lower on the high-sodium diet (3.69 and 5.48 in blacks and whites, respectively).

Urinary aldosterone

Urinary aldosterone excretion rates were 9.9 ± 4.7 μg/24 h and 10.8 ± 3.3 μg/24 h while consuming the low-sodium diet in blacks and whites, respectively. These rates significantly decreased while consuming the high-sodium diet to 5.78 ± 3.9 μg/24 h and 5.9 ± 2.3 μg/24 h in blacks and whites, respectively. No racial differences were observed. No significant correlation was found between urinary aldosterone and urinary potassium in any of the groups.

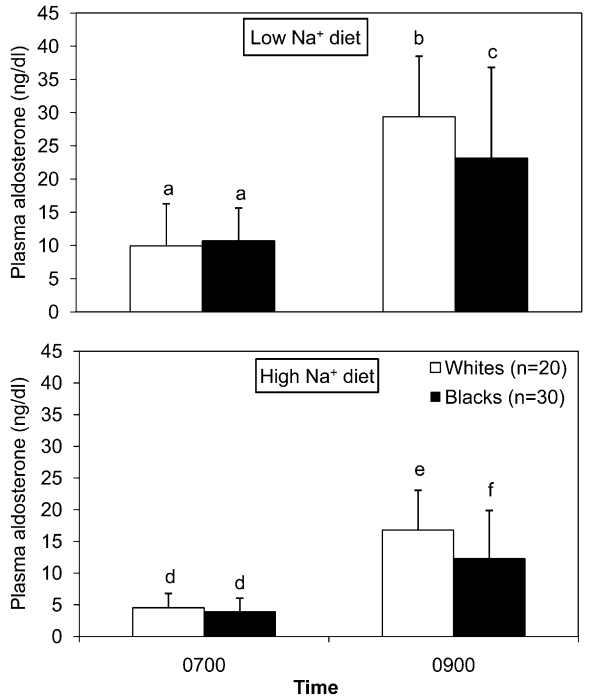

FIGURE 3. Mean (±SD) plasma aldosterone concentrations in black and white girls at the end of the 3-wk study period on low- and high-sodium (Na^+) diets. Blood samples were drawn at 0700 after subjects had been recumbent overnight and at 0900 after they had been upright for 2 h. Data points with different letters are significantly different ($P < 0.05$, ANOVA).

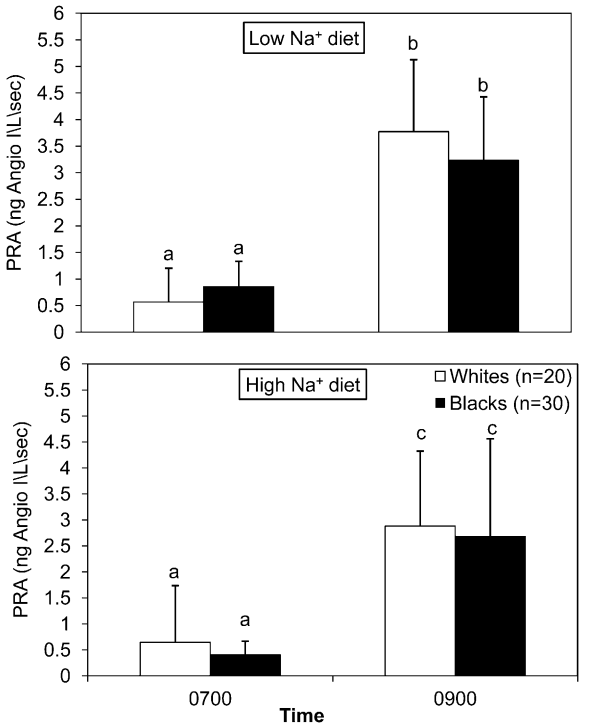

FIGURE 4. Mean (±SD) plasma renin activity (PRA) in black and white girls at the end of the 3-wk study period on low- and high-sodium (Na^+) diets. Blood samples were drawn at 0700 after subjects had been recumbent overnight and at 0900 after they had been upright for 2 h. Data points with different letters are significantly different ($P < 0.05$, ANOVA). Angio, angiotensin.

Blood pressure, weight, urinary volume, creatinine and Na^+ excretion, and serum data

There were no racial differences in blood pressure, body weight, urinary volume, urinary creatinine, serum sodium, or serum potassium on week 3 (Table 2). In addition, no significant differences were observed in these measurements due to the low- or high-sodium diets. Body weight and systolic blood pressure significantly decreased from baseline to the end of the study in all the girls, probably due to a higher physical activity during the study, because this was an active summer camp. In addition, urinary volume significantly increased from baseline to the end of the study in all girls (data not shown). This was in response to an increase in liquid consumption during the study to compensate for the summer heat and the increase in physical activity. However, no significant differences were observed between races or sodium intakes in these changes. We observed significant differences in urinary sodium excretion, whereas blacks excreted significantly less urinary sodium while consuming the high-sodium diet compared with whites, as we have previously described (18).

DISCUSSION

In 2 separate, highly controlled, 20-d metabolic studies in black and white girls, we found that urinary potassium excretion

was significantly lower in black girls while consuming either a low- or a high- sodium diet with potassium intake held constant at 50 mmol · L^{-1} · d^{-1}. Potassium retention was significantly higher in black girls when consuming the low-sodium diet only, which was due to lower urinary potassium excretion, because there was no racial difference in fecal excretion. As previously reported (22), sweat potassium output was similar between race groups, which, combined with fecal potassium excretion, represented ≈20% of dietary potassium intake. Furthermore, as previously shown, sweat potassium output during exercise (collected from arm bags and patches in different parts of the body after 30 min of exercise) was also similar in black and white girls (22).

Others have also reported potassium retention in black adults (8–10, 23, 24), but none of the earlier studies was performed by using a completely controlled metabolic design that included food analyses and complete urinary, fecal, and sweat collections. In the present study, we found significant potassium retention in black girls compared with white girls when consuming a low-sodium diet. Similar to our study, Luft et al (8) found greater potassium retention in black adults than in white adults when consuming a very-low-sodium diet. The potassium retention decreased with salt loading in both whites and blacks, which is a kaliuretic response to sodium loading that the authors attributed to increased sodium delivery to the distal nephron and/or physical displacement of potassium from intracellular stores by sodium. In the present study, with salt loading there was no cumulative potassium retention in either black or white girls.

In adolescents, Wilson et al (25) reported a significant reduction in urinary potassium excretion in adolescent blacks while consuming a high-sodium diet compared with a low-sodium diet studied with an outpatient protocol. In our study, for which the environment was rigorously controlled, we found similar urinary potassium excretion in black girls while consuming either the low or high intakes of sodium. Wilson et al (25) attributed the differences observed in black girls regardless of the intake of sodium in their diet due to an increase in intakes of fruit and vegetables while consuming the low-sodium diet. In their study, diet was not completely controlled, and fecal output was not measured. In the present study, each food given during the study was weighed, and subjects were required to consume all foods in the diet. In addition, fecal output was collected daily. Therefore, under a completely controlled environment, black girls excreted significantly less urinary potassium than did white girls regardless of the intake of sodium in the diet.

Plasma aldosterone concentrations were significantly lower in blacks than in whites while in an upright posture, although not when subjects were supine, and this was regardless of sodium intake. Urinary excretion rates of aldosterone were also similar in blacks and whites, and again with both dietary sodium intakes. Thus, the present results do not fully support earlier studies showing aldosterone concentrations and excretion rates to be lower in blacks (7, 15). This discrepancy could potentially be explained by multiple factors, which could include the lower potassium intake by subject of the present study, or it may reflect the fact that dietary intakes, of potassium in particular, were identical, more so than in previous studies.

Our results suggest that the well-recognized lower urinary excretion of potassium in blacks does not result solely from differences in dietary potassium. We hypothesize that there is a significant reduction in renal potassium secretion, possibly related to lower aldosterone concentrations, although we found, at best, small differences in these concentrations between race groups.

Hypertension (26) and its complications (27, 28) are more common in blacks than in whites. An understanding of the factors contributing to the greater susceptibility of blacks has important public health implications. Insights into mechanisms for hypertension in general can potentially be gained from the observed differences between blacks and whites. The present study suggests that racial differences in potassium handling in girls are best explained by differences in renal output and less by differences in diet.

The authors' responsibilities were as follows—CMW, BRM, JHP and MP: contributed to the study design; CMW, JHP, and MP: responsible for obtaining funding; CP, KW, BRM and MB: contributed to data collection and on-site study management; CP: contributed to data analysis; and CP, CMW and JHP: contributed to manuscript preparation. None of the authors had a conflict of interest.

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