Role of Potassium Excretion and Percent Body Fat on Ethnic Differences in Plasma Aldosterone Levels

Jennifer Jones, PhD; Jung-Jun Park, PhD; Thomas Dowling, PhD; Dana Phares, PhD; Joon-Young Park, MS; Michael Brown, PhD

Objective: To determine whether plasma aldosterone (PA) levels differed between African American and White prehypertensives and if so, could the difference be explained by ethnicity-related variability in urinary K^+ and Na^+ excretion, body mass index (BMI), and percent body fat.

Design: Ethnic comparison

Setting: The University of Maryland College Park and the University of Maryland School of Pharmacy.

Participants: 61 (African American, $n=28$; White, $n=33$) prehypertensives (systolic blood pressure [SBP] 131 $\pm$ 10 mm Hg, diastolic blood [DBP] 85 $\pm$ 6 mm Hg).

Intervention: 6-week dietary stabilization and medication tapering period.

Main Outcome Measures: PA levels, Na^+ and K^+ excretion, blood pressure, and percent body fat and BMI.

Results: We saw no differences in SBP ($P=.36$) and DBP ($P=.54$) between the two ethnic groups. PA levels were lower in African Americans compared to Whites (62 $\pm$ 7 vs 107 $\pm$ 12 pg/mL, $P=.002$). 24-hour K^+ excretion was lower among African Americans compared to Whites (51 $\pm$ 7 vs 70 $\pm$ 4 mmol/day, $P=.002$). We saw no difference in percent body fat, BMI, SBP, or DBP between African Americans and Whites. After separately accounting for K^+ excretion and Na^+ excretion and BMI, plasma aldosterone levels remained significantly different between the two ethnic groups. After adjusting for percent body fat, PA levels were not significantly different between the two ethnic groups ($P=.06$).

Conclusions: The findings of the current study indicate that PA levels differ between African American and White prehypertensives and this difference may partly be due to ethnic variability in K^+ excretion and percent body fat. (*Ethn Dis*. 2006;16[suppl 4]:S4-10–S4-14)

Key Words: African Americans, K^+ Excretion, Plasma Aldosterone

From the Department of Kinesiology (JJ, JJP, DP, JYP, MB), University of Maryland, College Park, Maryland; Department of Pharmacy Practice and Science, University of Maryland Baltimore, Baltimore, Maryland (TD).

Address correspondence and reprint requests to Jennifer M. Jones, PhD; Department of Internal Medicine; Division of Hypertension; University of Texas Southwestern Medical Center; 5323 Harry Hines Blvd., J4.134; Dallas, TX 75390-8586; 214-648-9038; 214-648-7902 (fax); jennifer.jones@utsouthwestern.edu

Introduction

Aldosterone, a mineralocorticoid produced in the adrenal gland, is integrally related to blood pressure regulation. Historically, aldosterone's influence was believed to be restricted to the kidney, but evidence now shows aldosterone synthase gene (CYP11B2) expression and mineralocorticoid receptors in both vascular endothelial[1,2] and myocardial cells.[3–6] Research suggests that elevated levels of aldosterone can contribute to the pathogenesis of hypertension,[7] cardiovascular disease (CVD),[4,5,8,9] and end-stage renal disease.[5,10]

Debate continues regarding whether African Americans suppress the renin-angiotensin-aldosterone system (RAAS) more than Whites.[11–16] Lower urinary and plasma levels of aldosterone among normotensive and hypertensive African Americans have been reported.[14,16] Nevertheless, some have suggested that the difference in aldosterone levels between African Americans and Whites may be explained by differences in potassium (K^+) intake, a major stimulus of aldosterone biosynthesis.[14,17] Recently, adiposity has been shown to contribute to the variability in plasma aldosterone levels.[18,19] Despite this finding, few studies have investigated whether differences in adiposity contribute to the variability in aldosterone levels between African Americans and Whites.[20]

We do not know whether the difference in plasma aldosterone levels between African Americans and Whites is due to ethnicity-related differences in RAAS regulation or variability in other biological variables known to affect aldosterone biosynthesis. Clarifying this issue will improve understanding of the pathogenesis of hypertension, CVD, and end-stage renal disease in these two ethnic groups. Therefore, the purpose of this study was to determine whether plasma aldosterone levels differed between African American and White prehypertensives and if so, whether the difference could be explained by ethnicity-related variability in urinary K^+ and sodium (Na^+) excretion, body mass index (BMI), and percent body fat.

Methods

Screening

Participants between the ages of 50–75 years were recruited from the College Park, Maryland, and the District of Columbia metropolitan area via newspaper advertisements, radio public service announcements, direct mail, and health fairs. The institutional review board of the University of Maryland, College Park, approved the study. Participants underwent an initial telephone screening and were excluded from the study if they had a body mass index (BMI) >37 kg/m^2, liver disease, pulmonary disease, diabetes, or any form of CVD other than hypertension. Women were excluded if they were not postmenopausal for at least two years.

Women were also required to maintain their hormone replacement status (either on or not on hormone replacement therapy) for the entirety of the study. Potential participants on more than two antihypertensive medications were excluded.

Participants who met the initial study inclusion criteria provided written informed consent and were scheduled for two screening visits. Participants were excluded if they exhibited a fasting blood glucose >126 mg/dL or blood glucose >200 mg/dL two hours after an oral glucose tolerance test (OGTT). Participants were excluded if they had a glomerular filtration rate <60 mL/min/1.73 m^2, which was estimated by using the Modification of Diet in Renal Disease Study equation,[21] and serum creatinine levels >1.5 mg/dL; both ensured that they did not have evidence of renal disease. Body mass index (BMI) was verified, and three casual blood pressure measurements were taken on each arm by using a standard sphygmomanometer and JNC 7 guidelines.[22] Participants with an average systolic blood pressure (SBP) <120 or >159 mm Hg and/or diastolic blood pressure (DBP) <80 or >99 mm Hg were excluded from the study.

Dietary Stabilization/ Medication Tapering

Participants attended a six-week dietary class taught by a registered dietitian two times per week. The participants followed the American Heart Association (AHA) Step I diet (50%–55% of calories from carbohydrates, 30%–35% from fat, 20%–25% from protein, 350 mmol/day of cholesterol, and 3 g/day of salt)[23] for the entirety of the study. The participants were required to stay on the AHA diet and within 5% of their study entry body weight throughout the study. The participants completed periodic seven-day food records to ensure that they adhered to the diet, and these records were analyzed by the dietitian with Nutritionist IV software.

Participants using antihypertensive medications were tapered off of their medications during the six-week dietary period with written approval from their personal physicians. Participants needed to maintain their blood pressure between 120–159/80–99 mm Hg during the medication tapering process to be included in the study. Participants with SBP <120 or >159 mm Hg and/or DBP <80 or >99 mm Hg during the six-week dietary period were excluded from the study. Testing was conducted after the 6-week dietary stabilization period was completed.

Casual Blood Pressure Measurement

Casual blood pressure was measured in all participants on three separate days according to the JNC 7 guidelines.[22] Three blood pressure measurements were taken each testing day. The primary blood pressure outcome variable used in the data analysis was the average measurement of the blood pressures that fell within a 10% range of the mean of the three blood pressure measurements. If one of the blood pressure measurements did not fall within the mean, the two measurements that did were averaged and used as the primary outcome variable.

24-Hour Urine Collection

Participants underwent urine collection to measure 24-hour Na$^+$ and K$^+$ excretion. The participants were given two urine collection bottles in which to collect their urine over the 24-hour period, starting the morning (7 AM– 9 AM) that they received the containers and ending after their first urination the following morning. They were also given a cooler filled with ice to keep the urine cold. Participants returned the urine containers on the same morning that they made the final required urine collection. The urine was processed at the University of Maryland, College Park, Hypertension and Exercise Physiology Laboratory and sent to Quest Diagnostic Laboratories

(CLIA License 21D0218877) for analysis of Na$^+$ and K$^+$ excretion.

Body Composition

Total-body dual energy x-ray absorptiometry (model DPX-L, Lunar Corporation, Madison, Wis) was used to assess body composition. Participants were instructed to fast for 12 hours before the start of the test.

Measurement of Aldosterone

Blood samples for the measurement of plasma aldosterone were collected before the start of the OGTT. Participants were instructed to undergo a 12-hour fast before blood sample collection and to exclude all ibuprofen, aspirin, and antihistamines 48 hours before the OGTT.[24] The blood samples were collected after the participant was supine for 15–20 minutes.[25] Blood samples were collected in EDTA tubes and centrifuged at 3000 rpm for 20 minutes at 4°C, and the plasma was aliquoted into 1.5 ml microtubes and stored at −20°C.[26] Radioimmunoassay was used to measure plasma aldosterone levels (^{125}I Coat-A-Count Aldosterone kit, Diagnostic Products Corp. Los Angeles, Calif.) All samples from each participant were run in duplicate in the same assay. Four separate assays were completed to measure plasma aldosterone levels in all of the samples. The intra-assay coefficient of variation was 25%, and the inter-assay coefficient was 20%. The sensitivity of the assay was 8.8 pg/mL.

STATISTICAL ANALYSES

Statistical analyses were performed by using SPSS (version 11.0). Independent sample *t* tests were performed to compare subject characteristics and main outcome variables between African Americans and Whites. Analysis of covariance was performed to determine if plasma aldosterone levels were different between these ethnic groups after

Table 1. Subject characteristics

Variable	African American	White
Age (years)	57 ± 1 (n=28)	58 ± 1 (n=33)
Weight (kg)	82 ± 2 (n=25)	86 ± 3 (n=31)
BMI (kg/m²)	29 ± 1 (n=25)	29 ± 1 (n=31)
Body fat (%)	40 ± 2 (n=20)	36 ± 2 (n=32)
Serum creatinine (mg/dL)	1.1 ± .0 (n=26)	1.0 ± .0 (n=31)
GFR (mL/min/1.73 m²)	77 ± 3 (n=26)	75 ± 2 (n=31)
SBP (mm Hg)	129 ± 2 (n=21)	132 ± 2 (n=31)
DBP (mm Hg)	86 ± 1 (n=20)	85 ± 1 (n=28)

GFR=glomerular filtration rate; BMI=body mass index; SBP=systolic blood pressure; DBP=diastolic blood pressure.

Values are unadjusted means ± SE. * $P<.05$

adjusting for potential confounding factors. Pearson correlation analyses were also conducted within each ethnic group to determine relationships between plasma aldosterone levels, 24-hour K^+ and Na^+ excretion, BMI, and percent body fat. All values are reported as means ± standard errors. If an outcome variable was not randomly distributed or the variances were heterogeneous, these data were appropriately transformed. A *P* value <.05 was considered statistically significant.

RESULTS

Participant characteristics for both ethnic groups are shown in Table 1. A total of 61 (33 Whites and 28 African Americans) men and women completed the study. Both the African American and White participants were prehypertensive and had normal renal function.

The White participants were overweight (based on BMI) and had normal plasma aldosterone levels (20–230 pg/mL)[27] and their urinary K^+ excretion (22–160 mmol/day)[28] and Na^+ (52–380 mmol/day)[29] were within the normal range (Figures 1–3). No significant relationship was seen between plasma aldosterone levels and percent body fat, BMI, and K^+ and Na^+ excretion. The African American participants were also overweight and had normal plasma aldosterone levels, and their urinary K^+ and Na^+ excretions were normal (Figures 1–3). No significant relationship

was seen between plasma aldosterone levels and percent body fat, BMI, and K^+ and Na^+ excretion in the African American participants.

No difference was seen in percent body fat, BMI, or systolic or diastolic blood pressure between African Americans and Whites (Table 1). African Americans had plasma aldosterone levels that were 42% lower than Whites (Figure 1). Compared to Whites, 24-hour K^+ excretion was also significantly lower among African Americans (Figure 2); 24-hour Na^+ excretion did not differ between the two ethnic groups (Figure 3). After separately accounting for K^+ excretion and Na^+ excretion and BMI, plasma aldosterone levels remained significantly different between the two ethnic groups. However, after accounting for percent body fat, plasma aldosterone levels tended to be but were no longer significantly different (P=.06) between the two ethnic groups.

DISCUSSION

The purpose of this study was to determine whether plasma aldosterone levels differed between African American and White prehypertensives and if so, whether the difference could be explained by differences in urinary K^+ and Na^+ excretion, percent body fat, or BMI. The most important finding of the study was that the African Americans had plasma aldosterone levels that were 42% lower than those of the White

participants. The significant difference in plasma aldosterone levels between the two ethnic groups remained after accounting for K^+ and Na^+ excretion and BMI, three variables that affect aldosterone biosynthesis.

Studies have reported that lower K^+ intake can account for lower urinary and plasma aldosterone levels in African Americans compared to Whites.[14,17,30] Furthermore, some studies have found that the difference in plasma aldosterone levels between African Americans and Whites was eliminated when both groups were on a homogenous K^+ diet.[16,17] Recently, Suh et al found K^+ disposal to be lower among African American compared to White normotensives and suggested that this finding may reflect decreased cellular Na^+, K^+-ATPase activity in African Americans.[31] Suh et al also reported that plasma aldosterone levels were lower in the African Americans compared to the Whites, but these differences were eliminated after an intravenous K^+ infusion.[31] In the present study, K^+ excretion was lower among African Americans compared to the Whites and possibly contributed to their lower aldosterone levels. However, after accounting for K^+ excretion, plasma aldosterone levels remained significantly different between the two ethnic groups, indicating that lower K^+ excretion among African Americans may not be the single most important factor contributing to their lower plasma aldosterone levels.

Evidence has shown that compared to Whites, African Americans, both normotensive and hypertensive, excrete less Na^+ when Na^+ loaded and are more likely to be salt sensitive.[32,33] Additionally, the lower plasma aldosterone levels in African American hypertensives may be attributed to their tendency to retain Na^+.[11] Recently Grim et al reported no difference in Na^+ excretion between normotensive and hypertensive African Americans and French Canadians.[20] Despite this finding, African American
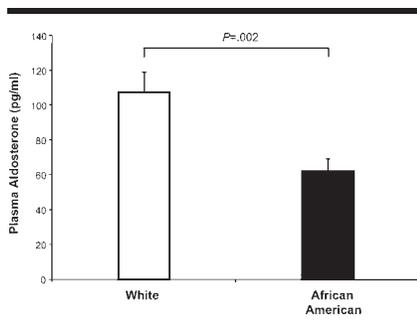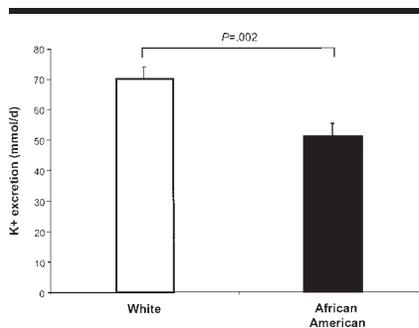
Fig 1. Plasma aldosterone levels in White and African American prehypertensives

Fig 2. 24-hour urinary K$^+$ excretion in White and African American prehypertensives

Fig 3. 24-hour urinary Na$^+$ excretion in White and African American pre-hypertensives

hypertensives and normotensives had lower supine plasma aldosterone levels compared to the French Canadians.[20] Fewer African Americans may exhibit the volume-dependent form of hypertension as has been suggested in the past.[34] Additionally, when the two ethnic groups are placed on similar Na$^+$ diets, volume dependency may contribute less to differences in aldosterone biosynthesis. In the present study, Na$^+$ excretion was not significantly different between the two ethnic groups, and when controlling for the influence of Na$^+$ excretion, plasma aldosterone levels remained significantly different between the two groups.

Comparison studies between obese and lean individuals have reported elevated plasma aldosterone levels among those who are obese.[18,19] In the current study, the mean BMI for both African Americans and Whites classified them as overweight, not obese. In both ethnic groups, no association was found between plasma aldosterone levels and BMI. In addition, BMI did not account for the difference in plasma aldosterone levels between the two ethnic groups.
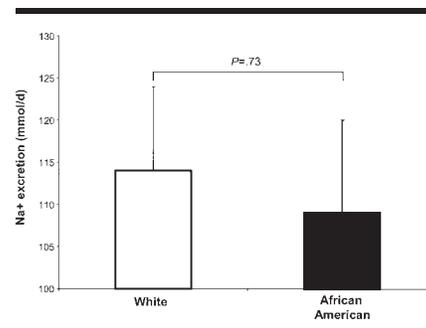
In the present study, after accounting for percent body fat, the difference in plasma aldosterone levels between the two ethnic groups was no longer significant, indicating that variability in percent body fat may have contributed to the plasma aldosterone difference. The increased aldosterone levels among those with greater fat mass may be related to increased renin, angioten-

sin converting enzyme, angiotensin II type 1-receptor gene expression,[35] and fatty acid oxidation.[36] However, in the current study, the African Americans had a greater percent body fat but lower plasma aldosterone levels compared to the Whites. These findings disagree with the suggestion that greater fat mass is associated with greater aldosterone levels. One possible explanation is that we did not investigate whether the ethnic groups differed in fat distribution. Studies have found a correlation between visceral adiposity and plasma aldosterone levels.[18,19] African Americans have been reported to have less visceral adipose tissue compared to Whites, which may contribute to their lower plasma aldosterone levels.[37,38] Therefore, the current findings should not be discounted because of the need for further investigation into whether fat distribution differences may have contributed to the ethnic difference in plasma aldosterone levels.

The lower aldosterone levels among the African Americans compared to the Whites does not necessarily suggest that the RAAS has less influence on disease pathogenesis compared to Whites. Grim et al reported that in African Americans, systolic and diastolic blood pressure correlated with plasma aldosterone levels, but this correlation was not found in the White participants.[20] Additionally, angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers are effective in lower-

ing the blood pressure of hypertensive African Americans.[39,40] These findings may indicate that even if circulating levels of angiotensin II and aldosterone are low, they may still contribute to certain disease processes.

The comparatively lower aldosterone levels among African Americans may be due to other factors not measured in the current study, such as CYP11B2 expression and levels of other steroid hormones involved in aldosterone biosynthesis. Further research is needed to investigate whether CYP11B2 mRNA levels or corticosterone levels, the precursor to aldosterone, may be lower among African Americans compared to other groups. Lower deoxycorticosterone levels, the corticosterone precursor, have been reported among young African American normotensives compared to Whites.[41]

In conclusion, the findings of the current study indicate that plasma aldosterone levels differ between African American and White prehypertensives. Differences in K$^+$ excretion and percent body fat may have partly contributed to the ethnic differences in plasma aldosterone levels, but further research is necessary to uncover whether ethnic variability in the molecular mechanisms leading to aldosterone biosynthesis are also important.

REFERENCES

1. Hatakeyama H, Miyamori I, Fujita T, Takeda Y, Takeda R, Yamamoto H. Vascular aldosterone. Biosynthesis and a link to angiotensin II-induced hypertrophy of vascular smooth muscle cells. *J Biol Chem.* 1994;269(39):24316–24320.

2. Takeda R, Hatakeyama H, Takeda Y, et al. Aldosterone biosynthesis and action in vascular cells. *Steroids.* 1995;60(1):120–124.

3. Heymes C, Garnier A, Fuchs S, et al. Aldosterone-synthase overexpression in heart: a tool to explore aldosterone's effects. *Mol Cell Endocrinol.* 2004;217(1–2):213–219.

4. Satoh M, Nakamura M, Saitoh H, et al. Aldosterone synthase (CYP11B2) expression and myocardial fibrosis in the failing human heart. *Clin Sci (Lond).* 2002;102(4):381–386.

5. Struthers AD, Macdonald TM. Review of aldosterone- and angiotensin II-induced target organ damage and prevention. *Cardiovasc Res.* 2004;61(4):663–670.

6. Bonvalet JP, Alfaidy N, Farman N, Lombes M. Aldosterone: intracellular receptors in human heart. *Eur Heart J.* 1995;16(suppl N):92–97.

7. Vasan RS, Evans JC, Larson MG, et al. Serum aldosterone and the incidence of hypertension in nonhypertensive persons. *N Engl J Med.* 2004;351(1):33–41.

8. Katada J, Meguro T, Saito H, et al. Persistent cardiac aldosterone synthesis in angiotensin II type 1A receptor-knockout mice after myocardial infarction. *Circulation.* 2005;111:2157–2164.

9. Keidar S, Kaplan M, Pavlotzky E, et al. Aldosterone administration to mice stimulates macrophage NADPH oxidase and increases atherosclerosis development: a possible role for angiotensin-converting enzyme and the receptors for angiotensin II and aldosterone. *Circulation.* 2004;109(18):2213–2220.

10. Greene EL, Kren S, Hostetter TH. Role of aldosterone in the remnant kidney model in the rat. *J Clin Invest.* 1996;98(4):1063–1068.

11. He FJ, Markandu ND, Sagnella GA, MacGregor GA. Importance of the renin system in determining blood pressure fall with salt restriction in Black and White hypertensives. *Hypertension.* 1998;32(5):820–824.

12. He J, Klag MJ, Appel LJ, Charleston J, Whelton PK. The renin-angiotensin system and blood pressure: differences between Blacks and Whites. *Am J Hypertens.* 1999;12(6):555–562.

13. Helmer OM, Judson WE. Metabolic studies on hypertensive patients with suppressed plasma renin activity not due to hyperaldosteronism. *Circulation.* 1968;38(5):965–976.

14. Pratt JH, Jones JJ, Miller JZ, Wagner MA, Fineberg NS. Racial differences in aldosterone excretion and plasma aldosterone concentrations in children. *N Engl J Med.* 1989;321(17):1152–1157.

15. Sagnella GA. Why is plasma renin activity lower in populations of African origin? *J Hum Hypertens.* 2001;15(1):17–25.

16. Fisher ND, Gleason RE, Moore TJ, Williams GH, Hollenberg NK. Regulation of aldosterone secretion in hypertensive Blacks. *Hypertension.* 1994;23(2):179–184.

17. Langford HG, Cushman WC, Hsu H. Chronic effect of KCl on Black-White differences in plasma renin activity, aldosterone, and urinary electrolytes. *Am J Hypertens.* 1991;4(5, pt 1):399–403.

18. Goodfriend TL, Egan BM, Kelley DE. Aldosterone in obesity. *Endocr Res.* 1998;24(3–4):789–796.

19. Goodfriend TL, Kelley DE, Goodpaster BH, Winters SJ. Visceral obesity and insulin resistance are associated with plasma aldosterone levels in women. *Obes Res.* 1999;7(4):355–362.

20. Grim CE, Cowley AWJr, Hamet P, et al. Hyperaldosteronism and hypertension: ethnic differences. *Hypertension.* 2005;45(4):766–772.

21. Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med.* 1999;130(6):461–470.

22. Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA.* 2003;289(19):2560–2572.

23. Krauss RM, Eckel RH, Howard B, et al. AHA Dietary Guidelines: revision 2000: a statement for healthcare professionals from the Nutrition Committee of the American Heart Association. *Circulation.* 2000;102(18):2284–2299.

24. Cartledge S, Lawson N. Aldosterone and renin measurements. *Ann Clin Biochem.* 2000;37(pt 3):262–278.

25. Stewart PM. Mineralocorticoid hypertension. *Lancet.* 1999;353(9161):1341–1347.

26. Kley HK, Schlaghecke R, Kruskemper HL. Stability of steroids in plasma over a 10-year period. *J Clin Chem Clin Biochem.* 1985;23(12):875–878.

27. Plouin PF, Chatellier G, Guyene TT, Vincent N, Corvol P. Recent advances in the clinical study of the renin system. Reference values and conditions of validity. *Presse Med.* 1989;18(18):917–921.

28. *Quest Diagnostics.* 2005, available at http://cas2.questdiagnostics.com/scripts/webdos.wls?MGWLPN=QDCWS0212&wlapp=DOS&OrderCode=734X&SITE=18&SearchString=P%2A&tmradio=title.

29. *Quest Diagnostics.* 2005, available at http://cas2.questdiagnostics.com/scripts/webdos.wls?MGWLPN=QDCWS0212&wlapp=DOS&OrderCode=838X&SITE=18&SearchString=S%2A&tmradio=title.

30. Urinary and serum electrolytes in untreated Black and White hypertensives. Veterans Administration Cooperative Study Group on Antihypertensive Agents. *J Chronic Dis.* 1987;40(9):839–847.

31. Suh A, DeJesus E, Rosner K, et al. Racial differences in potassium disposal. *Kidney Int.* 2004;66(3):1076–1081.

32. Luft FC, Miller JZ, Grim CE, et al. Salt sensitivity and resistance of blood pressure. Age and race as factors in physiological responses. *Hypertension.* 1991;17:I-102–I-108.

33. Luft FC, Rankin LI, Bloch R, et al. Cardiovascular and humoral responses to extremes of sodium intake in normal Black and White men. *Circulation.* 1979;60(3):697–706.

34. Jamerson KA. Rationale for angiotensin II receptor blockers in patients with low-renin hypertension. *Am J Kidney Dis.* 2000;36:S24–S30.

35. Gorzelniak K, Engeli S, Janke J, Luft FC, Sharma AM. Hormonal regulation of the human adipose-tissue renin-angiotensin system: relationship to obesity and hypertension. *J Hypertens.* 2002;20(5):965–973.

36. Goodfriend TL, Ball DL, Egan BM, Campbell WB, Nithipatikom K. Epoxy-keto derivative of linoleic acid stimulates aldosterone secretion. *Hypertension.* 2004;43(2):358–363.

37. Bacha F, Saad R, Gungor N, Janosky J, Arslanian SA. Obesity, regional fat distribution, and syndrome X in obese Black versus White adolescents: race differential in diabetogenic and atherogenic risk factors. *J Clin Endocrinol Metab.* 2003;88(6):2534–2540.

38. Kanaley JA, Giannopoulou I, Tillapaugh-Fay G, Nappi JS, Ploutz-Snyder LL. Racial differences in subcutaneous and visceral fat distribution in postmenopausal Black and White women. *Metabolism.* 2003;52(2):186–191.

39. Weir MR, Saunders E. Renin status does not predict the anti-hypertensive response to angiotensin-converting enzyme inhibition in African Americans. Trandolapril Multicenter Study Group. *J Hum Hypertens.* 1998;12(3):189–194.

40. Weir MR, Smith DH, Neutel JM, Bedigian MP. Valsartan alone or with a diuretic or ACE inhibitor as treatment for African American hypertensives: relation to salt intake. *Am J Hypertens.* 2001;14(7, pt 1):665–671.

41. Pratt JH, Rebhun JF, Zhou L, et al. Levels of mineralocorticoids in Whites and Blacks. *Hypertension.* 1999;34(2):315–319.