Importance of the Renin System in Determining Blood Pressure Fall With Salt Restriction in Black and White Hypertensives

Feng J. He, Nirmala D. Markandu, Giuseppe A. Sagnella, Graham A. MacGregor

Abstract—Seventy-one white and 33 black patients with essential hypertension were studied while on a high sodium intake of 350 mmol/d for 5 days and low sodium intake of 10 mmol/d for 5 days. The fall in blood pressure on changing from the high sodium to the low sodium diet was 17/6 mm Hg in whites and 22/10 mm Hg in blacks. Compared with whites, black patients had a 7-mm Hg greater fall (P < 0.05) in systolic blood pressure and 4-mm Hg greater fall (P = 0.068) in diastolic blood pressure (adjusted for age and blood pressure on the normal diet) with similar changes in urinary sodium excretion. With sodium restriction, plasma renin activity rose from 0.65 to 3.03 ng · mL⁻¹ · h⁻¹ in whites, whereas in blacks it rose only from 0.3 to 1.28 ng · mL⁻¹ · h⁻¹ (P < 0.001 between blacks and whites). From the high to the low salt diet, plasma angiotensin II increased by 31 pmol/L in whites and by 12 pmol/L in blacks (P < 0.05 compared with whites), and plasma aldosterone rose by 499 pmol/L in whites and by 256 pmol/L in blacks (P < 0.01). Significant inverse correlations were obtained for all patients between the fall in systolic blood pressure from the high to low salt diet and the rise in plasma renin activity and angiotensin II, as well as the absolute level on the low salt diet. These results demonstrate that the larger fall in blood pressure with a reduction in salt intake in blacks is due at least in part to a less responsive renin-angiotensin-aldosterone system in blacks. (Hypertension. 1998;32:820-824.)

Key Words: renin-angiotensin system ▪ blood pressure ▪ sodium, dietary ▪ blacks ▪ whites

Although sodium restriction reduces blood pressure in many patients with essential hypertension, the mechanism through which it acts is not fully understood.¹⁻³ However, several studies have shown that the fall in blood pressure with sodium restriction is related to the blunting of the renin response that occurs in many patients with essential hypertension⁴⁻⁷; this was shown with the use of the angiotensin (Ang) II antagonist saralasin to explain at least part of the different patterns of blood pressure response.⁴ It has long been established that black patients with high blood pressure tend to have lower levels of plasma renin activity (PRA) and are claimed to have more salt-sensitive blood pressure responses, although evidence for this latter claim is not substantiated. To assess the blood pressure response to short-term alteration of sodium intake further and also to try to elucidate the mechanism behind that difference, we studied black and white patients with essential hypertension during intake of their normal diet, a high sodium diet, and a low sodium diet with measurements of the renin-angiotensin-aldosterone system.

Methods

Seventy-one white and 33 black patients with essential hypertension referred by local practitioners were studied. Patients had not received previous treatment or treatment had been stopped 3 months before the study. There were 33 male and 38 female whites (mean age, 49 years; range, 19 to 70 years) and 15 male and 18 female blacks (mean age, 44 years; range, 29 to 62 years). All patients gave informed consent. The study was approved by the local hospital ethics committee.

Patients were studied on their normal diet, on the fifth day of high sodium intake (350 mmol/d), and on the fifth day of low sodium intake (10 mmol/d). High sodium intake was achieved by supplementing the normal diet with 20 slow sodium tablets (200 mmol/d). A low sodium diet was provided by the Metabolic Unit kitchen. The potassium intake was not altered for either diet.

Blood pressure was measured in the same arm by nurses using semiautomatic ultrasonic sphygmomanometers (Arteriosonde) with attached recorders. The measurements were therefore free from observer bias. Supine and standing blood pressures were the mean of 5 readings taken at 1- to 2-minute intervals in the corresponding positions. Two 24-hour urine samples were collected during the last 2 days of each dietary period for measurement of sodium, potassium, and creatinine. Blood was also taken at the end of each diet period for measurement of electrolytes, potassium, and creatinine. Plasma Ang II was measured in 43 patients. Plasma volume was measured using ¹³¹I-albumin.

Results are reported as mean ± SEM. Changes in continuous variables within each group were analyzed by paired t tests. Comparisons between blacks and whites at baseline (on the normal diet) were made with unpaired t tests for continuous variables and χ² test for categorical data. Because of the unequal distributions of baseline characteristics between blacks and whites, we used a
multiple linear regression model for comparison between the 2 groups while adjusting for potential confounders. All statistical analyses were performed with Statistical Package for Social Science (SPSS).

Results

Baseline Characteristics

Table 1 shows the results obtained on the final day of normal sodium intake (before alteration of sodium intake) for black and white hypertensive patients. There were no significant differences between blacks and whites in gender, supine and standing systolic blood pressures, 24-hour urinary sodium and potassium excretion, plasma potassium, plasma volume, and total blood volume while subjects were on their usual diet. The blacks were, however, younger, slightly heavier, and had higher diastolic blood pressure than whites. The PRA on the normal diet was significantly lower in blacks than in whites (0.48 versus 1.08 ng·mL⁻¹·h⁻¹, P<0.001). Blacks also had lower plasma Ang II and aldosterone, but the differences did not reach statistical significance.

High to Low Sodium Intake

White Hypertensive Patients

On the fifth day of high sodium intake, supine blood pressure was 172/108±2/1 mm Hg with a 24-hour urinary sodium excretion of 280±14 mmol. After 5 days of the low sodium diet, blood pressure fell significantly to 155/102±2/1 mm Hg (urinary sodium excretion, 21±2 mmol/24 h). The average fall in blood pressure with this reduction in salt intake was 17/6 mm Hg (10.7% fall in systolic and 6.0% fall in diastolic). Changes in standing blood pressure during the study were similar to those found for supine blood pressure (Table 2). PRA on the high sodium intake was 0.65±0.07 ng·mL⁻¹·h⁻¹ and rose to 3.03±0.28 ng·mL⁻¹·h⁻¹ on the low sodium diet (P<0.001). Plasma Ang II increased by 31 pmol/L (P<0.001) and aldosterone increased by 499 pmol/L (P<0.001) with the change from the high to the low sodium diet. With sodium restriction there was a fall of 1.8 kg in body weight (P<0.001) and falls of 0.24 L in plasma volume (P<0.001) and 0.27 L in total blood volume (P<0.001).

Black Hypertensive Patients

On the fifth day of high sodium intake, supine blood pressure was 176/112±4/3 mm Hg with a 24-hour urinary sodium excretion of 271±22 mmol. After 5 days of the low sodium diet, blood pressure fell significantly to 154/102±4/2 mm Hg (urinary sodium excretion, 21±2 mmol/24 h). The average fall in blood pressure with the low salt diet was 22/10 mm Hg (14.3% fall in systolic and 10.0% fall in diastolic). Standing blood pressures showed falls similar to those of supine blood pressures after sodium restriction (Table 2). PRA on the high sodium intake was 0.30±0.04 ng·mL⁻¹·h⁻¹ and rose to 1.28±0.26 ng·mL⁻¹·h⁻¹ on the low sodium diet (P<0.01). Plasma Ang II increased by 12.2 pmol/L and aldosterone increased by 255.7 pmol/L (P<0.01) with the change from the high to the low sodium diet. With sodium restriction there was a fall of 1.9 kg in body weight (P<0.001) and falls of 0.23 L in plasma volume (P<0.001) and 0.28 L in total blood volume (P<0.001).

Differences Between Blacks and Whites

The Figure shows the changes in systolic blood pressure, PRA, and Ang II from the high to the low salt diet in black and white hypertensive patients. Compared with whites, black patients had a 7/4-mm Hg greater fall in supine blood pressure (systolic, P<0.05; diastolic, P=0.068; adjusted for age and supine blood pressure on the normal diet), with a similar reduction in urinary sodium excretion (Table 3). Blacks also showed a bigger fall in standing blood pressure than whites; however, the difference in the fall in standing blood pressure between blacks and whites did not reach statistical significance. After sodium restriction, PRA rose by 2.37 ng·mL⁻¹·h⁻¹ in whites, whereas in blacks it only increased by 0.97 ng·mL⁻¹·h⁻¹ (P<0.001 between blacks and whites). The average rises in plasma Ang II and aldosterone on change from the high to the low salt diet were significantly less in blacks than in whites (Table 2). After 5 days of the low salt diet, black patients showed reductions in body weight, plasma volume, and total blood volume similar to those of the whites. There were no significant differences between blacks and whites in the changes in plasma sodium, potassium, and creatinine and urinary potassium and creatinine excretion.
Correlations Between Blood Pressure Response and Other Variables

Taking all patients together, the fall in systolic blood pressure with the low sodium diet was significantly correlated with age ($r=0.20, P<0.05$) and with the level of systolic blood pressure on the normal diet ($r=0.23, P<0.05$). There was also a significant inverse correlation between the fall in blood pressure with sodium restriction and the PRA (log-transformed values) on the low salt diet (systolic, $r=-0.29, P<0.01$; diastolic, $r=-0.24, P<0.05$). The fall in systolic blood pressure with the low salt diet was also significantly associated with the rise in PRA on going from the high to the low salt diet ($r=-0.28, P<0.01$). In the 43 patients who had plasma Ang II measured, the fall in systolic blood pressure with sodium restriction showed a significant inverse correlation with the log(Ang II) on the low salt diet ($r=-0.46, P<0.001$), as well as the rise in plasma Ang II on going from the high to the low sodium diet ($r=-0.46, P<0.01$).

Multiple regression analysis was carried out to further analyze the differences between blacks and whites in the blood pressure fall with sodium restriction in relation to the renin-angiotensin-aldosterone system. In a multiple linear regression model, the racial differences in blood pressure response to sodium restriction became less and not significant if adjusted for the log(PRA) on the low salt diet or the rise in PRA on going from the high to the low sodium diet. These results suggest that the differences in the fall in blood pressure with sodium restriction between blacks and whites is due at least in part to the different responses of the renin-angiotensin system, particularly because the fall in urinary sodium excretion and weight loss were similar in blacks and whites.

The fall in blood pressure with the low salt diet was also significantly correlated with the plasma aldosterone concentration on the low sodium diet (systolic, $r=-0.38, P<0.01$; diastolic, $r=-0.29, P<0.05$), as well as with the rise in plasma aldosterone on going from the high to the low sodium diet (systolic, $r=-0.41, P<0.001$; diastolic, $r=-0.26, P<0.05$). As expected, the rise in plasma aldosterone was significantly associated with the rise in plasma Ang II ($r=0.55, P<0.001$).

Correlation analyses were also carried out in blacks and whites separately. The subgroup analyses showed that most significant inverse correlations between the changes in blood pressure and PRA, Ang II, or aldosterone were present in whites alone but no significant correlations were found in the blacks alone, which is likely to be due to the small number of black patients and much smaller range of changes in PRA and Ang II in the blacks.

**Discussion**

In view of the fact that blacks were younger and had higher levels of blood pressure at entry to the study (on the normal diet), and the finding that age and entry blood pressure were

### TABLE 2. Blood Pressure and Laboratory Data After 5 Days on Different Sodium Intake

<table>
<thead>
<tr>
<th>Variable</th>
<th>White</th>
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<tr>
<td></td>
<td>High Salt</td>
<td>Low Salt</td>
<td>Difference</td>
<td>High Salt</td>
<td>Low Salt</td>
<td>Difference</td>
<td>Adjusted Difference§</td>
<td>Adjusted Difference</td>
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<td>Adjusted Difference</td>
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<td>Supine blood pressure, mm Hg</td>
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<tr>
<td>Systolic</td>
<td>172.1 ± 2.5</td>
<td>155.4 ± 2.3</td>
<td>-16.6 ‡</td>
<td>175.7 ± 4.4</td>
<td>153.7 ± 3.8</td>
<td>-22.0 ‡</td>
<td>-0.16</td>
<td>0.417</td>
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<tr>
<td>Diastolic</td>
<td>107.7 ± 1.4</td>
<td>101.6 ± 1.4</td>
<td>-6.1 ‡</td>
<td>111.6 ± 2.6</td>
<td>101.5 ± 2.4</td>
<td>-10.1 ‡</td>
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<td>Standing blood pressure, mm Hg</td>
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<tr>
<td>Systolic</td>
<td>171.2 ± 2.4</td>
<td>152.6 ± 2.6</td>
<td>-18.6 ‡</td>
<td>173.2 ± 3.9</td>
<td>151.2 ± 3.7</td>
<td>-22.0 ‡</td>
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<tr>
<td>Diastolic</td>
<td>117.5 ± 2.0</td>
<td>107.5 ± 1.7</td>
<td>-9.99 ‡</td>
<td>121.5 ± 2.6</td>
<td>108.6 ± 2.6</td>
<td>-12.9 ‡</td>
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<tr>
<td>Weight, kg</td>
<td>72.6 ± 1.7</td>
<td>70.8 ± 1.7</td>
<td>-1.8 ‡</td>
<td>76.5 ± 2.0</td>
<td>74.6 ± 1.9</td>
<td>-1.9 ‡</td>
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<td>Urine</td>
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<tr>
<td>Sodium, mmol/24 h</td>
<td>279.7 ± 13.5</td>
<td>20.7 ± 1.7</td>
<td>-259.0 ‡</td>
<td>271.3 ± 22.5</td>
<td>21.1 ± 2.1</td>
<td>-250.2 ‡</td>
<td>12.48</td>
<td>0.638</td>
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<tr>
<td>Potassium, mmol/24 h</td>
<td>58.9 ± 2.3</td>
<td>59.0 ± 1.7</td>
<td>-0.82 ‡</td>
<td>53.8 ± 3.0</td>
<td>64.1 ± 1.8</td>
<td>-12.4 ‡</td>
<td>-3.40</td>
<td>0.419</td>
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<tr>
<td>Creatinine, mmol/24 h</td>
<td>11.1 ± 1.8</td>
<td>9.8 ± 0.6</td>
<td>-1.3</td>
<td>12.2 ± 1.3</td>
<td>11.9 ± 1.1</td>
<td>-0.3</td>
<td>0.55</td>
<td>0.844</td>
<td></td>
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<tr>
<td>Volume, mL/24 h</td>
<td>2134 ± 105</td>
<td>1341 ± 63</td>
<td>-792 ‡</td>
<td>2244 ± 163</td>
<td>1242 ± 79</td>
<td>-1002 ‡</td>
<td>-239</td>
<td>0.219</td>
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<tr>
<td>Plasma</td>
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<tr>
<td>PRA, ng/mL·L·h⁻¹</td>
<td>0.65 ± 0.07</td>
<td>3.03 ± 0.28</td>
<td>2.37 ‡</td>
<td>0.30 ± 0.04</td>
<td>1.28 ± 0.26</td>
<td>0.97 ‡</td>
<td>-1.72</td>
<td>0.000</td>
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<tr>
<td>Ang II, pmol/L (n=43)</td>
<td>193 ± 1.5</td>
<td>50.3 ± 6.3</td>
<td>31.0</td>
<td>137.2 ± 2</td>
<td>25.9 ± 5.5</td>
<td>12.2</td>
<td>-20.98</td>
<td>0.017</td>
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<tr>
<td>Aldosterone, pmol/L</td>
<td>307.2 ± 23.3</td>
<td>805.8 ± 65.8</td>
<td>498.5‡</td>
<td>336.5 ± 34.1</td>
<td>592.2 ± 76.5</td>
<td>255.7 ‡</td>
<td>-344.35</td>
<td>0.002</td>
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<tr>
<td>Sodium, mmol/L</td>
<td>140.7 ± 0.3</td>
<td>137.7 ± 0.4</td>
<td>-3.0 ‡</td>
<td>140.2 ± 0.4</td>
<td>137.5 ± 0.4</td>
<td>-2.67 ‡</td>
<td>0.59</td>
<td>0.327</td>
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<tr>
<td>Potassium, mmol/L</td>
<td>3.8 ± 0.06</td>
<td>3.9 ± 0.05</td>
<td>0.09</td>
<td>3.8 ± 0.06</td>
<td>3.9 ± 0.08</td>
<td>0.12</td>
<td>0.08</td>
<td>0.397</td>
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<tr>
<td>Creatinine, μmol/L</td>
<td>80.99 ± 2.05</td>
<td>89.7 ± 1.94</td>
<td>8.72 ‡</td>
<td>89.1 ± 2.8</td>
<td>100.6 ± 3.8</td>
<td>11.5 ‡</td>
<td>3.66</td>
<td>0.288</td>
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<tr>
<td>Plasma volume, L</td>
<td>2.81 ± 0.07</td>
<td>2.56 ± 0.06</td>
<td>-0.24 ‡</td>
<td>2.84 ± 0.08</td>
<td>2.61 ± 0.09</td>
<td>-0.23 ‡</td>
<td>0.03</td>
<td>0.689</td>
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<tr>
<td>Total blood volume, L</td>
<td>4.54 ± 0.11</td>
<td>4.27 ± 0.11</td>
<td>-0.27 ‡</td>
<td>4.49 ± 0.15</td>
<td>4.22 ± 0.16</td>
<td>-0.28 ‡</td>
<td>0.01</td>
<td>0.876</td>
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</table>

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$^1P<0.05$, $^1P<0.01$, $^1P<0.001$ high salt vs low salt.

§Adjusted differences refer to age-adjusted differences between blacks and whites in changes of variables from high to low salt diet.

||Adjusted P values represent comparisons between blacks and whites after adjustment for age.
significantly correlated with the fall in blood pressure with the low salt diet, we compared the racial difference in blood pressure response to sodium restriction by a multivariate model that included the potential confounders. Our results show that with a similar reduction in sodium intake, black hypertensive patients have a greater fall in systolic blood pressure with a smaller rise in PRA and Ang II compared with whites. The fall in systolic blood pressure with sodium restriction was significantly related to the PRA and Ang II on the low-salt diet, as well as the rise in PRA and Ang II on going from the high to the low sodium diet. Furthermore, in a multiple linear regression model, the racial differences in the fall in systolic blood pressure with sodium restriction became less and not significant if adjusted for the PRA on the low salt diet or the rise in PRA on going from the high to the low sodium diet. These results suggest that the greater fall in blood pressure with sodium restriction in hypertensive blacks is due at least in part to the decreased responsiveness of the renin-angiotensin system in blacks.

Compared with the whites, the black hypertensive patients also had a lower level of PRA at baseline on the normal diet as well as after low sodium intake. The implications of this observation may be 2-fold. First, the lower values of PRA may reflect a relative expansion of extracellular fluid volume and/or sodium balance in the black hypertensives. Thus, the blood pressure response to sodium and volume restriction would be greater. An alternative explanation is that the renin-angiotensin system protects against sodium and volume depletion and maintains vascular homeostasis during such situations; therefore, the black hypertensives who had a relatively less responsive renin-angiotensin system would have a greater permissive fall in blood pressure with the low salt diet. Evidence in support of this latter hypothesis was demonstrated in the study by Weinberger et al. when they compared 2 different tests for the assessment of blood pressure responses to sodium in the same individuals. The increases in PRA with the sodium and water depletion compared with the whites for the rises in PRA or Ang II from the high to the low salt diet adjusted for age.  

Our findings of racial differences in blood pressure and PRA responses to alteration of sodium intake are in agreement with those found in normotensive subjects. Luft et al. observed that normotensive blacks had higher blood pressures than whites after saline administration and had a greater suppression of PRA than whites 24 hours after saline. Kaplan et al. evaluated PRA responses after administration of 40 mg intravenous furosemide in 127 normotensive subjects and 363 patients with essential hypertension. They found that both hypertensive and normotensive blacks had significantly lower PRA after furosemide than did whites.

In our study, we aimed to reduce dietary sodium intake to 10 mmol/d. However, on the fifth day of the low salt diet, the 24-hour urinary sodium excretion was 20.7 mmol for the whites and 21.1 mmol for the blacks. This may imply that the actual dietary sodium intake was greater than expected, or the patients had not reached equilibrium for the whites and 21.1 mmol for the blacks. This may reflect a relative expansion of extracellular fluid volume and/or sodium balance in the black hypertensives. Thus, the blood pressure response to sodium and volume restriction may be greater. An alternative explanation is that the renin-angiotensin system protects against sodium and volume restriction, and/or sodium balance in the black hypertensives. Thus, the blood pressure response to sodium and volume restriction would be greater. An alternative explanation is that the renin-angiotensin system protects against sodium and volume depletion and maintains vascular homeostasis during such situations; therefore, the black hypertensives who had a relatively less responsive renin-angiotensin system would have a greater permissive fall in blood pressure with the low salt diet. Evidence in support of this latter hypothesis was demonstrated in the study by Weinberger et al. when they compared 2 different tests for the assessment of blood pressure responses to sodium in the same individuals. The increases in PRA with the sodium and water depletion induced by the low salt diet and furosemide in the first study predicted (correlated with, $P<0.001$) the blood pressure response to the low salt diet in the second study, which was conducted 3 months later.


differences in changes in systolic blood pressure, PRA, and Ang II from the high to the low salt diet adjusted for age and systolic blood pressure on the normal diet; $**P<0.001$ compared with the high salt diet; $*$ $P<0.05$, $**P<0.001$ blacks vs whites for the rises in PRA or Ang II from the high to the low salt diet adjusted for age.

**TABLE 3. Differences Between Blacks and Whites in Changes in Blood Pressure From High to Low Salt Intake**

<table>
<thead>
<tr>
<th>Regression Model</th>
<th>Supine Blood Pressure, mm Hg</th>
<th>Standing Blood Pressure, mm Hg</th>
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<tr>
<td></td>
<td>Systolic</td>
<td>Diastolic</td>
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<tr>
<td>Univariate</td>
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<tr>
<td></td>
<td>$-5.4 (-11.6, 0.8)$</td>
<td>$0.0875$</td>
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<tr>
<td>Multivariate</td>
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<td></td>
<td>$-6.6 (-12.8, -0.3)$</td>
<td>$0.0403$</td>
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</table>

Values are blacks minus whites, with 95% confidence interval and 2-sided P values.

*Adjustments made for age and blood pressure on the normal diet.

Changes in systolic blood pressure, PRA, and Ang II from the high to the low salt diet in black and white hypertensive patients. $*P<0.05$ blacks vs whites adjusted for age and systolic blood pressure on the normal diet; $**P<0.01$, $***P<0.001$ compared with the high salt diet; $*$ $P<0.05$, $**P<0.001$ blacks vs whites for the rises in PRA or Ang II from the high to the low salt diet adjusted for age.
200 mmol/d in the other trial1) and different duration of the 2 studies (5 days and 4 weeks, respectively).

The data from the study by Luft et al14 also suggested that potassium balance might influence the responses in blood pressure that occurred with sodium loading. However, in our study potassium was unlikely to account for the racial difference in blood pressure responses to alteration of sodium intake because neither the changes in urinary potassium excretion on going from the high to the low salt diet nor in plasma potassium were different between blacks and whites.

In the present study, hypertensive blacks had a slightly lower plasma aldosterone than whites on the normal diet. With sodium restriction, blacks had a significantly smaller rise in plasma aldosterone than whites. This is likely due to the lower PRA and Ang II in the blacks, recognized by the rise in plasma aldosterone than whites. This is likely due to the lower PRA and Ang II in the blacks, recognized by the rise in plasma aldosterone with the low salt diet and the rise in Ang II. The finding of the racial difference in aldosterone secretion was in agreement with other studies.15,16 Furthermore, some studies also showed that the lower level of plasma aldosterone and the blunted responses to further stimulation (upright posture and Ang II infusion) in blacks were independent of renin status.15,16

To demonstrate more directly the importance of the response of the renin system in determining the blood pressure fall that occurs with salt restriction, a subgroup of the patients in our study (22 whites and 7 blacks) were also infused with saralasin, a competitive inhibitor of Ang II, on the fifth day of the low sodium diet.4 The fall in blood pressure with saralasin was inversely correlated with the fall in blood pressure with salt restriction in essential hypertension. BMJ. 1981;283:94–97.

Racial differences in aldosterone secretion were largely modulated by the reactivity of the renin-angiotensin-aldosterone system.

References
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