Effects of Potassium on Blood Pressure in Salt-Sensitive and Salt-Resistant Black Adolescents

Dawn K. Wilson, Domenic A. Sica, Sydney B. Miller

Abstract—This study examined the effects of increasing dietary potassium on ambulatory blood pressure nondipping status (<10% decrease in blood pressure from awake to asleep) and cardiovascular reactivity in salt-sensitive and salt-resistant black adolescents. A sample of 58 normotensive (blood pressure, 101/57±9/4 mm Hg) black adolescents (aged 13 to 16 years) participated in a 5-day low sodium diet (50 mmol/24 h) followed by a 10-day high sodium diet (150 mmol/24 h NaCl supplement) to determine salt-sensitivity status. Participants showed a significant increase in urinary sodium excretion (24±19 to 224±65 mmol/24 h) and were identified as salt-sensitive if their mean blood pressure increase was ≥5 mm Hg from the low to high sodium diet. Sixteen salt-sensitive and 42 salt-resistant subjects were then randomly assigned to either a 3-week high potassium diet (80 mmol/24 h) or usual diet control group. Urinary potassium excretion significantly increased in the treatment group (35±7 to 57±21 mmol/24 h). At baseline, a significantly greater percentage of salt-sensitive (44%) compared with salt-resistant (7%) subjects were nondippers on the basis of diastolic blood pressure classifications (P<0.04). After the dietary intervention, all of the salt-sensitive subjects in the high potassium group achieved dipper status as a result of a drop in nocturnal diastolic blood pressure (daytime, 69 versus 67 mm Hg; nighttime, 69 versus 57 mm Hg). No significant group differences in cardiovascular reactivity were observed. These results suggest that a positive relationship between dietary potassium intake and blood pressure modulation can still exist even when daytime blood pressure is unchanged by a high potassium diet. (Hypertension. 1999;34:181-186.)

Key Words: sodium ■ potassium ■ blood pressure ■ blacks ■ adolescence

Blacks are at increased risk for developing essential hypertension (EH) compared with other ethnic groups.1 Thus, identifying precursors or markers of hypertension is important for prevention of EH in blacks. Ambulatory blood pressure (ABP) nondipping status (<10% decrease in blood pressure [BP] from awake to asleep) is one risk factor that may be modifiable in youth. Previous research from our laboratory has indicated that nondipping BP status is observable in normotensive black adolescents.2 Additional research has not been consistent in defining nondipping BP status. Regardless of the definitional differences of past studies, nondipping status has been consistently associated with the development and progression of end-organ disease such as stroke3,4 and left ventricular hypertrophy.6,7 Cardiovascular reactivity (CVR) (increases in BP in response to stress) may be another potentially modifiable risk factor of EH in youth. Although there is controversy concerning the predictive value of CVR,8,9 several prospective studies have shown that increased CVR in response to mental stress is predictive of the later development of EH.10,11

One potential strategy that may be effective in decreasing the risk of EH in black children is to increase K+ intake. Although previous research has suggested that modifying K+ intake may reduce BP, the results have been inconsistent across studies,12 in part because of nonhomogeneity in study populations and protocol designs. Despite these inconsistencies, research has consistently demonstrated that increasing K+ significantly lowers BP (systolic [SBP] and diastolic [DBP]) among salt-sensitive (SS) individuals (ie, hypertensives, blacks) who show increased BP in response to high Na+ intake.13-16 Results have been more mixed for studies examining the effects of K+ supplementation on BP under low Na+ conditions.17 However, little is known of the effects of increasing K+ on markers of EH in youth. Thus, the present study expands on past work by examining the effects of increasing dietary K+ on ABP nondipping status and CVR in SS versus salt-resistant (SR) black adolescents.

Methods

Subjects
The study protocol was approved by the Committee on the Conduct of Human Research of Virginia Commonwealth University. Written informed consent was obtained from each participant. Healthy black adolescents (aged 13 to 16 years) were recruited from schools and

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TABLE 1. Demographic Characteristics and Baseline Measurements

<table>
<thead>
<tr>
<th>Variables</th>
<th>SS</th>
<th>SR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size</td>
<td>16</td>
<td>42</td>
</tr>
<tr>
<td>Gender (men/women)</td>
<td>8/8</td>
<td>20/22</td>
</tr>
<tr>
<td>Family history of EH (+)</td>
<td>73%</td>
<td>74%</td>
</tr>
<tr>
<td>Parents married</td>
<td>56%</td>
<td>48%</td>
</tr>
<tr>
<td>Parental education</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Some high school</td>
<td>6%</td>
<td>10%</td>
</tr>
<tr>
<td>High school graduate</td>
<td>44%</td>
<td>31%</td>
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<tr>
<td>Some college</td>
<td>12%</td>
<td>21%</td>
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<tr>
<td>College graduate</td>
<td>38%</td>
<td>24%</td>
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<tr>
<td>Graduate school</td>
<td>0%</td>
<td>14%</td>
</tr>
<tr>
<td>Annual family income</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;$10 000 to $19 999</td>
<td>13%</td>
<td>22%</td>
</tr>
<tr>
<td>$20 000 to $49 999</td>
<td>50%</td>
<td>46%</td>
</tr>
<tr>
<td>$50 000</td>
<td>37%</td>
<td>32%</td>
</tr>
<tr>
<td>Age, y</td>
<td>14±1</td>
<td>56±10</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>58±9</td>
<td>14±1</td>
</tr>
<tr>
<td>Height, cm</td>
<td>162±9</td>
<td>163±8</td>
</tr>
<tr>
<td>Casual SBP, mm Hg</td>
<td>103±13</td>
<td>100±8</td>
</tr>
<tr>
<td>Casual DBP, mm Hg</td>
<td>58±6</td>
<td>57±4</td>
</tr>
<tr>
<td>Casual HR, bpm</td>
<td>72±7</td>
<td>75±18</td>
</tr>
</tbody>
</table>

Values are mean ± SD. All P=NS.

Procedures

The study procedures were in accordance with institutional guidelines. Subjects arrived at the Clinical Research Center, where each participant’s parent provided information on family history of EH, level of education, total annual family income, and parental marital status (Table 1). Each subject’s casual BP was measured by a trained technician with a Dinamap BP apparatus (model 8100; Critikon Inc) as outlined by the Second Task Force on Blood Pressure Control in Children. Before BP assessment, 1 BP measurement was obtained for each subject and was compared with a sphygmomanometer reading to ensure accuracy. Only subjects whose SBP was within ±5 mm Hg by these 2 methods were included in the study. After a 5-minute rest period, a total of 5 SBP, DBP, and heart rate (HR) measurements were taken, with a 30-second interval between readings. The average of these 5 measurements was used as the BP value for subsequent data analyses. Mean blood pressure (MBP) values were computed as SBP×2+DBP/3 and were used to define SS status. Subjects were classified as SS if they showed an increase in MBP of ≥5 mm Hg from the low Na+ diet to the high NaCl supplement diet; subjects who remained were classified as SR. Each participant was then randomly assigned to either a high K+ diet or a usual diet control group for 3 weeks. Subjects participated in a series of CVR tasks and wore an ABP monitor for a 24-hour period during a baseline ad libitum dietary period within 2 months after being classified as either SS or SR. At the completion of the program, subjects repeated the ABP and CVR protocols.

Dietary Protocols for Determination of SS

Low Na+ Diet

Subjects participated in a 5-day low Na+ diet (50 mmol/24 h) that has already been described in detail. Briefly, a trained technician gave each child and his/her parents guidelines for maintaining the low Na+ diet. Participants were told to eat fresh meats, fresh vegetables, and fresh fruits and to avoid fast foods or other prepackaged processed foods. They were also provided with an assortment of low Na+ foods to assist them in maintaining the diet. Each family was given sample breakfast, lunch, and dinner menus that met their individual food preferences. All adolescent participants were required to record their Na+ intake daily.

High Na+ Diet

Immediately after completing the 5-day low Na+ diet, subjects began a 10-day high Na+ diet in which, in addition to their regular diet, they received 150 mmol/24 h of NaCl supplement. All participants were instructed to distribute the NaCl supplement between their 2 heaviest meals and to take the supplements only on a full stomach.

CVR Testing

Subjects participated in a competitive video game to determine their CVR scores. After a 5-minute rest period, 4 resting SBP, DBP, and HR readings were taken by a trained technician. Next, each child participated in 3 video games (Atari “Breakout”). One BP and HR measure was obtained during each game. Each child then participated in the cold face stimulus procedure. Before the task, subjects relaxed for a 10-minute period. Next, 4 baseline SBP, DBP, and HR measurements were obtained by a trained technician. A plastic bag containing water and crushed ice (3°C to 5°C: 1.5 cups ice to 0.5 cup of water) was then placed across the subject’s forehead. After 30 seconds, 1 BP and HR measure was obtained. Average resting and stress BP and HR values were calculated for each CVR task. CVR was determined by calculating baseline-stress change scores for each task (video and cold face).

ABP Monitoring

After completing the CVR tasks, subjects participated in the ABP protocol. Each participant was seated, and the ABP recorder (Advanced Biosensor Inc) was applied and calibrated. Three readings from the ABP recorder were compared with a mercury column to determine proper functioning of the recorder. If the technician was unable to match 3 readings to within ±5 mm Hg for SBP, then another recorder was applied and calibrated. Participants recorded their actual awake and asleep times. The recorder was set to take readings at 15-minute intervals over 24 hours. Once the recorder had been removed, the BP results were examined, and if >25% of the measurements were artifacts or missing, the participant was asked to repeat the ABP recording.

Dietary K+ Intervention

The dietary intervention has been previously published in detail. Briefly, participants were randomly assigned to either a 3-week high K+ diet (80 mmol/24 h) or a usual diet control group. The intervention was divided into 4 weekly 1-hour classes that included a baseline week. The 3 general principles of the program were education, behavioral skills training, and feedback on performance on the basis of food records and 24-hour urine results. The control group also met each week for a 1-hour session to discuss the adequacy of their food intake records and urine collections. During the first week of the program, participants in both groups were taught how to accurately estimate and record their food intake by a registered dietician. At each group session, subjects were given a 7-day food record to complete for the following week. The focus of the second week of the program was to increase K+ intake to 3200 mg/24 h for the treatment group. K+ counter books, high K+ menus, and a list of foods that were high in K+ (ie, 300 to 600 mg per
serving) were provided to participants, and they were instructed to eat 6 to 8 servings of these foods per day. Subjects were encouraged to eat the high K+ foods throughout the study period. Participants were provided with foods each week that they had chosen from a list of high K+ foods (eg, roasted peanuts, fresh fruits, and vegetables). The third week of the intervention concentrated on eliminating barriers to increasing K+ consumption and discussing suggestions for substituting high K+ foods for low K+ junk foods. The fourth week of the program focused on generating a list of strategies that had been successful in helping individuals to increase their K+ consumption.

**Urinary Measures**

**Na+ and K+ Dietary Compliance**

To determine compliance, urinary sodium excretion (U\textsubscript{Na}V), urinary potassium excretion (U\textsubscript{K}V), and creatinine levels were determined by 24-hour urine collections. For determination of SS status, one 24-hour collection was performed on day 5 of the low Na+ diet. On days 8 and 9 of the high Na+ diet, 2 consecutive 24-hour collections were completed by each subject. Subjects who had a 24-hour urine volume ≥500 mL and excretion of creatinine ≥10 mg/kg per 24 hours met the criteria for an adequate urine collection. Subjects were considered compliant if their low Na+ diet U\textsubscript{Na}V was ≤50 mmol/24 h and their high NaCl diet U\textsubscript{Na}V was ≥165 mmol/24 h. Subjects whose U\textsubscript{Na}V was ≤50 mmol/24 h and who showed a 3-fold or more increase in their U\textsubscript{Na}V during the NaCl load were also considered compliant and were included in subsequent analyses. For determination of compliance with the high K+ program, the average of 3 baseline and the average of 3 treatment (or control) 24-hour urine collections were obtained. Urine samples were analyzed for Na+ and K+ with a NOVA 13 analyzer (NOVA Biomedical). Creatinine was measured with a Beckman Creatinine Analyzer 2 (Beckman Instruments, Inc).

**Results**

**Subject Characteristics**

Table 1 presents demographic and baseline characteristics for the sample. There were no significant group differences on demographic or baseline measures.

**Electrolyte Measures**

A series of 2×2×2 (SS versus SR; low Na+ versus high Na+; before versus after diet) repeated-measures ANOVAs were conducted to determine compliance with the low and high Na+ diets. As expected, there was a significant diet-by-time interaction (P<0.05). Subjects showed a significant increase in U\textsubscript{Na}V from the low to high Na+ intake (24±19 versus 224±65 mmol/24 h).

Table 2 shows U\textsubscript{Na}V, U\textsubscript{K}V, and urinary creatinine values for subjects categorized by SS status and dietary condition. A series of 2×2×2 (SS versus SR; K+ versus control group; before versus after diet) repeated-measures ANOVAs demonstrated a significant diet-by-time interaction for U\textsubscript{K}V (P<0.02). As expected, subjects in the K+ group showed a significant increase in U\textsubscript{K}V levels from before diet to after diet (35±7 to 57±21 mmol/24 h); in contrast, subjects in the control group showed no significant change in U\textsubscript{K}V levels from before diet to after diet. Subjects in the K+ group also showed significantly higher U\textsubscript{K}V levels after diet than did subjects in the control group.

**ABP Measures**

The data from the ABP recordings were edited according to previously published standards. Average awake and asleep

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**TABLE 2. Electrolyte Measurements Before and After the High K+ Dietary Program**

<table>
<thead>
<tr>
<th>Variables</th>
<th>SS K+ Group</th>
<th>SR K+ Group</th>
<th>SS Controls</th>
<th>SR Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size</td>
<td>9</td>
<td>17</td>
<td>7</td>
<td>25</td>
</tr>
<tr>
<td>Baseline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>U\textsubscript{Na}V, mmol/d</td>
<td>141±45</td>
<td>148±42</td>
<td>155±36</td>
<td>140±32</td>
</tr>
<tr>
<td>U\textsubscript{K}V, mmol/d*</td>
<td>36±7</td>
<td>35±8</td>
<td>35±13</td>
<td>36±10</td>
</tr>
<tr>
<td>Creatinine, mg (kg d)</td>
<td>22±3</td>
<td>23±4</td>
<td>23±3</td>
<td>25±3</td>
</tr>
<tr>
<td>Post-Program</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>U\textsubscript{Na}V, mmol/d</td>
<td>127±32</td>
<td>149±50</td>
<td>145±45</td>
<td>150±36</td>
</tr>
<tr>
<td>U\textsubscript{K}V, mmol/d*</td>
<td>56±26</td>
<td>58±19</td>
<td>48±31</td>
<td>37±12</td>
</tr>
<tr>
<td>Creatinine, mg (kg d)</td>
<td>23±3</td>
<td>22±4</td>
<td>26±6</td>
<td>25±4</td>
</tr>
</tbody>
</table>

Values are mean±SD. *P<0.05, K+ group-by-time interaction.

SBP, DBP, and MBP values were then determined for each subject on the basis of the subject’s self-report of awake and asleep times. A series of \chi² analyses were performed to examine dipper and nondipper BP status as a function of SS versus SR status and treatment group. Subjects were classified as dippers or nondippers separately for each measure (SBP, DBP, MBP); they were classified as dippers if their BP showed a >10% decrease from awake to asleep and as nondippers if their BP showed a <10% decrease from awake to asleep.

Figure 1 shows the percentage of dippers versus nondippers for SS and SR subjects at baseline and after treatment for the K+ group. At baseline, a significantly greater percentage of nondippers were SS compared with SR for DBP classifications (P<0.05). After treatment, all of the SS subjects who were originally classified as nondippers reverted to a normal dipping status because of a drop in nighttime DBP (nighttime
DBP, 69±7 versus 57±6 mm Hg; daytime DBP, 69±5 versus 67±5 mm Hg). Comparison subjects (n=3) (Figures 1 and 2) did not show a similar drop in nighttime or daytime DBP (nighttime DBP, 64±1 versus 61±5 mm Hg; daytime DBP, 69±1 versus 71±8 mm Hg).

Figure 2 shows the percentage of dippers versus nondippers for SS and SR subjects at baseline and after treatment for the control group. Again, a significantly greater percentage of nondippers were SS compared with SR (P<0.05) at baseline for DBP classifications. At the end of the program, only 1 of 4 SS subjects (25%) in the control group who was originally a nondipper reverted to a normal dipping status (Figure 2). There were no significant effects for SBP or MBP dipping status classifications. Follow-up analyses also indicated that there were no significant differences across groups in terms of weight change or sleep duration from before to after K+ intake.

CVR Measures
A series of 2x2x2 (SS versus SR; K+ versus control; before versus after diet) repeated-measures ANOVAs were conducted for CVR change scores. No significant group differences were found.

Discussion
ABP has been shown to be a better predictor of cardiovascular mortality than standard BP measures obtained in the physician’s office.23 The characteristics of an ABP profile have also been shown to be predictive of target-organ damage in EH among normotensive and hypertensive adults. For example, patients who are characterized as nondippers show a more frequent history of stroke4,5 and left ventricular hypertrophy.6,7 The present study demonstrated that 30% of a healthy cohort of black adolescents were classified as nondippers and that a significant proportion of these nondippers were SS. This prevalence rate of nondipping is consistent with an independent study published by our laboratory2 and with adult studies that indicate a prevalence rate of nondipping ranging from 33% to 40% among normotensive populations.24,25 These findings are also consistent with observations by Harshfield et al,22 who showed that black children had higher levels of DBP during sleep than white children. Our findings in conjunction with these other studies suggest that ABP nondipping is prevalent and may be an important precursor or marker for identifying youth who are at risk for developing EH and cardiovascular disease in early adulthood.

After increasing dietary K+, all of the SS subjects who were nondippers achieved a dipping status. This change in dipping status was due to a drop in nighttime DBP and not to a change in daytime DBP. In contrast to the K+ group, SS subjects in the control group did not show a significant reversal in nondipping status from before treatment to after treatment.

Our data are the first to indicate that increasing dietary K+ reverses nondipping status in SS subjects but has no effect on daytime BP. These findings in part support other investigations that have shown beneficial effects of increasing K+ intake on BP responses in SS populations. For example, Fujita and Ando13 showed that SS hypertensives who were given a K+ supplement (96 mmol/24 h) while on a high Na+ diet showed significantly greater decreases in MBP after 3 days compared with nonsupplemented hypertensive patients. Svetkey et al15 demonstrated a significant drop in SBP and DBP after 8 weeks of K+ supplementation (120 mmol/24 h versus placebo) among mildly hypertensive patients. Siani et al14 reported similar findings in hypertensive patients who were given K+ supplements (48 mmol/24 h) for 15 weeks. In one of the most dramatic studies by Obel,16 mildly hypertensive black males who received a K+ supplement (64 mmol/24 h versus placebo) for 16 weeks showed a drop in supine BP (SBP, 175±10 to 133±10 mm Hg; DBP, 100±3 to 83±4 mm Hg), whereas no change was detected in the placebo group. Research with animal models has also shown that increasing K+ reduced pressor responses to saline infusions in SS Dahl rats.26 Results have been more mixed for studies examining the effects of K+ supplementation in normotensive and white populations who show a lower prevalence of SS.27-29 Our data are also consistent with a recent meta-analysis by Whelton et al,12 which concluded that low K+ intake may play an important role in the genesis of high BP and that increasing K+ should be considered a recommendation for prevention and treatment of EH. However, to the best of our knowledge, our study is the first to show changes in ABP nondipping status resulting from an increase in K+ intake.

Several potential mechanistic pathways may explain how increasing K+ intake reverses nondipping BP status.30 One potential pathway involves K+-induced natriuresis. For example, a number of studies have strongly suggested that changes in dietary K+ alter Na+ balance, such that K+ restriction results in Na+ retention and K+ supplementation leads to greater natriuresis.31,32 Our study did not examine the
pattern of natriuresis, and further investigations are needed to
determine whether greater natriuresis may in part explain the
reversal in nondipping status shown in the present study.
Linas\(^{30}\) has also suggested that the effect of K\(^+\) on \(U_{Na,V}\), plasma
volume, and mean arterial pressure could be evidence
for a K\(^+\)-mediated vasodilator effect on BP. If nondippers are
characterized by heightened sympathetic nervous system
activity and increased peripheral resistance during sleep
hours, this K\(^+\)-mediated vasodilatory effect could explain the
reversal in nondipping status observed in the present study. In
support of this hypothesis, a number of studies have demon-
strated that the local arterial infusion of K\(^+\) decreases forearm
vascular resistance and increases forearm blood flow in a
dose-dependent fashion.\(^{33–35}\) For example, Fujita and Ito\(^{33}\)
observed that intrabrachial arterial infusions of KCl increased
forearm blood flow and decreased forearm vascular resis-
tance in a group of normotensive subjects. Research has also
shown that K\(^+\) supplementation given in combination with a
high Na\(^+\) diet suppressed the increase in catecholamines
that typically occurs in response to Na\(^+\) loading.\(^{36}\)

The present study has several limitations. Because a
crossover design was not used, intrasubject variability is
unknown for the individuals who were in the treatment group.
Recently, Mochizuki et al\(^{37}\) reported that 16% of hypertensive
patients in their study switched from a dipping to nondipping
status, whereas 13% of the patients who were nondippers
switched to a dipper status over a 2-day period. Our labora-
tory has reported that 18% of black adolescents were not
consistently classified as nondippers across 2 time periods.\(^{2}\)
However, in the present study, a randomized control design
was used, and therefore equal numbers of subjects in the
treatment versus control group should have demonstrated this
change in dipping status regardless of treatment. In contrast,
our data demonstrated that SS subjects in the treatment group
changed from nondippers to dippers at a greater rate than SS
subjects in the control group. Further research is needed to
replicate these results in a larger sample of subjects.

In conclusion, although K\(^+\) supplementation has been
advocated as a means for decreasing daytime BP, our data
extend past research by further demonstrating that nighttime
BP and associated nondipping status are reversible with K\(^+\)
supplementation. These data also suggest that a positive
relationship exists between dietary K\(^+\) intake and BP modu-
lation although daytime BP and CVR responses were un-
changed by the dietary intervention. Further investigations
are needed to better define the physiological mechanisms
underlying the reversal of nondipping status in SS subjects
who are administered high K\(^+\) diets.

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