Double-Blind, Placebo-Controlled Trial of Potassium Chloride in the Treatment of Mild Hypertension

LAURA P. SVETKEY, WILLIAM E. YARGER, JOHN R. FEUSSNER, ELIZABETH DELONG, AND PAUL E. KLOTMAN

With the technical assistance of Toby S. Brown

SUMMARY Epidemiological and experimental data suggest blood pressure-lowering effects of dietary potassium. A randomized, double-blind clinical trial was used to assess blood pressure response to orally administered potassium, 120 mEq/day, and to placebo in 101 adults with mild hypertension. Blood pressure was measured with a random-zero sphygmomanometer every 2 weeks of this 8-week trial. Systolic blood pressure in the potassium-treated group decreased by 6.4 ± 13.7 (SD) mm Hg (p<0.025) compared with 0.11 ± 13.0 mm Hg in the placebo-treated group (p = 0.96). Diastolic blood pressure in the potassium-treated group decreased by 4.1 ± 8.3 mm Hg (p<0.05) compared with a 1.6 ± 6.5 mm Hg decrease in placebo-treated subjects (p = 0.09). Baseline blood pressure of potassium-treated subjects was unexpectedly higher than that of controls. After correcting for baseline variation, blood pressure still decreased 3.4/1.8 mm Hg more in potassium recipients than in placebo recipients (p = 0.14 and 0.24, respectively). Blood pressure decreased by 19/13 mm Hg in five blacks taking potassium versus a 1/0 mm Hg increase in seven blacks taking placebo. Compliance with the potassium regimen was 91.5% by pill count; only one subject discontinued treatment because of side effects. In conclusion, 120 mEq/day of microencapsulated potassium chloride was well tolerated in adults with mild hypertension. An antihypertensive effect of potassium cannot be ruled out despite the fact that there was no statistically significant difference between potassium-treated and placebo-treated subjects after adjustment for differences in baseline blood pressure. Systolic blood pressure was affected more than diastolic blood pressure. Blacks may be particularly sensitive to blood pressure-lowering effects of potassium chloride. A larger clinical trial stratified by baseline blood pressure and race is required. (Hypertension 9: 444-450, 1987)

KEY WORDS • hypertension • potassium • therapy

ADDISON1 stated in 1928 that “a potash [potassium] poor diet” causes hypertension and that “the giving of potassium chloride . . . is associated with a drop in blood pressure.” Subsequent experimental, epidemiological, and clinical data support this claim.2-34

In experimental models of renovascular, genetic, and salt-sensitive hypertension, high potassium intake has been shown to attenuate the development of hypertension.2-6 Epidemiological surveys similarly implicate potassium deficiency and suggest a protective effect of high potassium intake. An inverse relationship exists between blood pressure and dietary potassium when comparing both large populations7-9 and individuals within a population.8,10-12 Furthermore, movement from an area of low potassium intake to an area of high potassium intake is associated with a decrease in blood pressure.13-16

Evidence for a therapeutic effect of potassium in established hypertension has also been accumulating. Dietary and parenterally administered potassium lowers blood pressure in various animal models of hypertension.2,3,17-21 Clinical trials suggest that oral potassium has a blood pressure-lowering effect in normal humans with or without a family history of hypertension.
Patients and Methods

Patient Selection

Ambulatory hypertensive adults were recruited from the Duke Hypertension Center and the Durham Veterans Administration Medical Center Hypertension Clinic. Subjects were excluded from entry if history or physical examination revealed any of the following: a single diastolic blood pressure exceeding 114 mm Hg, prior episode of malignant hypertension or hypertensive encephalopathy, angina, myocardial infarction within the previous 6 months, congestive heart failure, arrhythmia, cerebrovascular accident, transient ischemic attacks, or the presence of a terminal illness. Secondary hypertension was excluded by history, physical examination, serum electrolyte levels, and measurements of renal function (plasma creatinine concentration, creatinine clearance, and complete urinalysis). Subjects who might be at risk from high potassium intake were also excluded: those with renal insufficiency or baseline serum potassium values greater than 5.0 mEq/L, those taking digitalis preparations, and those with chronic diarrhea or history of ulcer disease. Pregnant and nursing women were excluded.

Maneuver

This study was approved by the Institutional Review Boards and the Human Use Committees of Duke University Medical Center and the Durham Veterans Administration Medical Center. After written informed consent was obtained from all subjects, antihypertensive medication was discontinued. Subjects took no blood pressure medication during a 2-week washout interval. Baseline blood pressure off medication was then established by measurements performed weekly for 2 to 4 weeks. The diagnosis of hypertension was confirmed if the average seated diastolic blood pressure was 90 to 105 mm Hg on 2 consecutive weeks during this baseline period. (If this criterion was not reached within 4 consecutive weeks, subjects were disqualified from the study.) A computer-generated table of random numbers was used to assign subjects to the potassium chloride or placebo treatment groups. Study subjects received five potassium chloride capsules (Micro-K Extencaps; A. H. Robins Company, Richmond, VA, USA) three times daily (120 mEq/day) or an equal number of identical-appearing placebo capsules. Capsules were odorless and tasteless even if broken, so that subjects could not discover to which treatment group they belonged.

Personnel involved in the study were blinded to the assignment of subjects to active treatment or placebo group. A study monitoring committee reviewed the results of laboratory examinations on a daily basis. These data were not transmitted to the investigators during the course of the study, and the monitors had no direct contact with study subjects.

Potassium or placebo capsules were taken daily for 8 weeks. Subjects returned to the study sites every 2 weeks for evaluation. Subjects were asked not to alter their diet or activities in any way during the study. At each visit, three blood pressure measurements were recorded and the average value was considered to be the blood pressure for that day. All blood pressures were measured using a random zero sphygmomanometer (Hawksley and Sons, Lancing, Sussex, England) with the subject seated for 10 minutes before the readings. Diastolic blood pressure was recorded as the fifth Korotkoff sound. Blood pressure measurements and pulse were taken at the same time of day and by the same person at each visit. Subjects were advised not to smoke or eat for 30 minutes before each blood pressure reading. Weight was obtained and laboratory examinations (complete blood count, serum creatinine, electrolytes, triglyceride, cholesterol, hepatic enzymes, uric acid, albumin, and total protein) were performed on each visit. Stool guaiac was checked after 6 weeks of therapy. Subjects were questioned about possible side effects at each clinic visit. Compliance was assessed by pill count.

Termination Criteria

Criteria for removal from the study were average diastolic blood pressure on any clinic visit greater than 105 mm Hg, intolerable side effects, development of new target-organ damage, serum potassium greater than 5.5 mEq/L, serum creatinine greater than 1.5 mg/dl or doubled from baseline value, hemoglobin decreased by 2 g/dl, or evidence of gastrointestinal bleeding.
Statistical Analysis

The sample size (n = 100) was calculated to have an 80% power to detect a change in blood pressure of 5 mm Hg with significance at the 5% level (two-tailed). These calculations were based on an estimated standard deviation of multiple blood pressure readings in an individual of 10 mm Hg. A target sample size of 120 (60 in each treatment group) allowed for a 20% dropout rate. The actual dropout rate was 12.9%. Each blood pressure measurement consists of the average of three readings. Mean arterial pressure was calculated as \((2 \times \text{diastolic blood pressure}) + \text{systolic blood pressure})/3. Changes in blood pressure within each treatment group were analyzed using the paired t test. Comparisons between the two treatment groups were made with a linear regression model that adjusted for differences in baseline blood pressure. Additional hypothesis tests investigated the interaction between age, sex, race, weight, or compliance and the effect of treatment group on blood pressure. Differences in the incidence of side effects were assessed by standard chi-square tests. Other comparisons were made by Student's t test.

Results

A total of 116 hypertensive adults entered the study protocol and were randomly assigned to receive either potassium chloride (40 mEq t.i.d.) or placebo capsules for 8 weeks; 101 subjects completed the study. Fifty-nine subjects (mean age, 51.3 ± 12.3 years) received potassium chloride treatment. Of these, 76% were men and 89% were white. Initial serum potassium was 4.4 ± 0.4 mEq/L. Fifty-seven subjects (mean age, 50.8 ± 12.3 years) received placebo treatment. Of these, 72% were men and 83% were white. Their initial serum potassium was 4.5 ± 0.3 mEq/L. At the time of randomization there were no significant differences in body weight or pulse between the two groups, and 8 weeks of treatment did not affect these parameters (Table 1). Compliance was 91.5% in both groups.

Table 1 also demonstrates that dropout rates were comparable in the two treatment groups. Five (8%) potassium-treated and 10 (17%) placebo-treated subjects discontinued the study drug before completing the protocol (p = 0.10). Of the five subjects who discontinued potassium, one subject dropped out due to side effects (nausea, flatulence, constipation, and decreased energy), three subjects did not return to the clinic but when contacted gave no specific reason for discontinuing the study, and one subject dropped out after randomization but before receiving study drug. Of the 10 patients who discontinued placebo, two subjects dropped out because of side effects (flatulence and increased anxiety in one and epigastric discomfort in the other) and three subjects did not return to the clinic and gave no reason for discontinuing the study. Of the remaining five dropouts in the placebo group, three subjects were removed because their blood pressure exceeded 105 mm Hg, one subject experienced unexplained polycythemia, and one subject was advised by his personal physician to stop taking the study drug despite an average diastolic blood pressure of 92 mm Hg.

Side effects were generally mild (Table 2). Abdominal pain and "gas" were more common in the potassium-treated group (18 vs 9%, p = 0.18, and 20 vs 10%, p = 0.22, respectively), but, as noted, only one subject stopped taking potassium because of gastrointestinal symptoms.

No subject was removed from the study because of altered renal function (creatinine >1.5 mg/dL), evidence of gastrointestinal bleeding (guaiac positive stool or decreased hemoglobin), or hyperkalemia (serum potassium >5.6 mEq/L), although mean serum potassium increased slightly in the potassium-treated group (from 4.4 ± 0.4 to 4.7 ± 0.4 mEq/L; p = 0.0004 by paired t test). No significant changes in other laboratory parameters were observed during the 8-week study.

In both groups, change in blood pressure over the 8 weeks of observation was correlated with baseline blood pressure; that is, the higher the blood pressure before a subject received the study drug, the greater the decrease in blood pressure in that subject, regardless of treatment-group assignment. Because of this effect and the unexpected difference in initial systolic blood pressure between the two groups (147.5 ± 13.1 mm Hg in the potassium-treated group vs 142.1 ± 14.3 mm Hg in the placebo-treated group), comparisons between treatment groups are reported both as mean changes in blood pressure in each treatment group and as residual changes after including the effect of baseline blood pressure in a linear regression model. Blood pressures at baseline and after 8 weeks of treatment are presented in Table 3. Only subjects completing the full 8-week protocol are included (n = 101), although three subjects receiving placebo were removed from the trial as a result of an increase in blood pressure.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Potassium (n = 59)</th>
<th>Placebo (n = 57)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretreatment</td>
<td>Posttreatment</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>83.8 ± 14.4</td>
<td>84.6 ± 16.8</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>76.9 ± 14.1</td>
<td>74.8 ± 12.9</td>
</tr>
<tr>
<td>Compliance (%)</td>
<td>91.5 ± 9.5</td>
<td>91.5 ± 6.3</td>
</tr>
<tr>
<td>No. of dropouts</td>
<td>5(8)</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD. Percentage of dropouts is shown in parentheses.
Potassium in the treatment of hypertension

TABLE 2. Incidence and Consequence of Side Effects During 8 Weeks of Therapy with Potassium or Placebo

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Potassium</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal pain (cramps, indigestion, or heartburn)</td>
<td>11 (18)</td>
<td>5 (9)</td>
</tr>
<tr>
<td>Change in bowel habits (diarrhea, soft stool, frequent stool, or constipation)</td>
<td>6 (10)</td>
<td>8 (14)</td>
</tr>
<tr>
<td>“Gas” (belching or flatulence)</td>
<td>12 (20)</td>
<td>6 (10)</td>
</tr>
<tr>
<td>Lethargy</td>
<td>1 (2)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>1 (2)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Skin rash</td>
<td>0 (0)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Palpitations</td>
<td>0 (0)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Headache</td>
<td>2 (3)</td>
<td>3 (5)</td>
</tr>
<tr>
<td>Dizziness</td>
<td>0 (0)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Dropout due to side effects</td>
<td>1 (2)</td>
<td>2 (3)</td>
</tr>
</tbody>
</table>

Percentage of subjects is shown in parentheses.

above the 105 mm Hg limit (including these three subjects in the analysis did not significantly alter the results). Systolic blood pressure fell in the potassium-treated group by 6.4 ± 13.7 mm Hg (p = 0.025) and by 0.11 ± 13 mm Hg in the control group (p = 0.96). Diastolic blood pressure decreased by 4.1 ± 8.3 mm Hg in potassium-treated subjects (p = 0.05), and by 1.6 ± 6.5 mm Hg in the placebo-treated subjects (p = 0.09). Mean arterial pressure decreased by 4.8 ± 9.1 mm Hg in potassium recipients (p = 0.0005) compared with a decrease of 1.1 ± 7.7 mm Hg in placebo recipients (p = 0.32). After adjusting for baseline blood pressure using a linear regression model, there was still a 3.4 mm Hg greater decrease in systolic blood pressure (p = 0.14) and a 1.8 mm Hg greater decrease in diastolic blood pressure (p = 0.24) in potassium recipients than in subjects taking placebo.

Response was defined as a 10 mm Hg or greater decrease in blood pressure. Thirty-four percent (20) of potassium-treated compared with 16% (9) of placebo-treated subjects had a systolic blood pressure response while 24% (14) and 9% (5), respectively, had a diastolic response. Tests for interaction did not suggest that potassium was more or less effective in any group defined by age, sex, weight, severity of hypertension, or degree of compliance. However, Table 4 demonstrates that in the five blacks receiving potassium blood pressure decreased by 19 ± 24/13 ± 10 mm Hg compared with a 1 ± 7/0 ± 6 mm Hg increase in blood pressure in seven blacks taking placebo. Table 4 also shows that potassium-treated and placebo-treated blacks were similar in age and baseline plasma potassium levels. Body weight was initially higher in the potassium-treated blacks (p = 0.10), but it did not change in either group after 8 weeks of treatment.

Discussion

Several nutritional factors, such as sodium, calcium, magnesium, and fat, as well as potassium, have been linked to the pathogenesis and treatment of high blood pressure. Previous reports suggest that potassium balance may be related to blood pressure in two different ways. Potassium deficiency may be etiologically linked to the development of high blood pressure, and potassium supplementation may lower blood pressure in persons with established hypertension. The former effect of potassium, if confirmed, would encourage widespread nutritional interventions aimed at preventing hypertension. The latter effect suggests a possible role for dietary or pharmacological potassium in the treatment of hypertension.

Dietary potassium intake is linked to hypertension by population surveys in which the incidence of hypertension and its cardiovascular consequences are inversely related to the amount of dietary potassium consumed. Furthermore, movement from an area of high potassium intake to an area of low potassium intake is associated with increases in blood pressure. These associations generally are attributed to differences in sodium intake. However, in rural Japan, where sodium consumption is uniformly high, hypertension is less common in a village of apple farmers, where potassium intake is also high, than in a village with similar sodium but lower potassium intake. In general, persons who consume large quantities of potassium have lower blood pressure than persons with similar sodium intake but lower potassium consumption. In the United States, where hyper-

TABLE 3. Systolic, Diastolic, and Mean Arterial Pressure Response to 8 Weeks of Treatment with Potassium or Placebo

<table>
<thead>
<tr>
<th>Blood pressure (mm Hg)</th>
<th>Potassium (n = 54)</th>
<th>Placebo (n = 47)</th>
<th>Adjusted difference</th>
<th>Adjusted p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretreatment</td>
<td>Posttreatment</td>
<td>Pretreatment</td>
<td>Posttreatment</td>
</tr>
<tr>
<td>Systolic Decrease</td>
<td>147.5 ± 13.1</td>
<td>141.1 ± 13.0</td>
<td>142.1 ± 14.3</td>
<td>142.0 ± 13.0</td>
</tr>
<tr>
<td></td>
<td>6.4 ± 13.7 (p = 0.025)</td>
<td></td>
<td>0.11 ± 13.0 (p = 0.96)</td>
<td></td>
</tr>
<tr>
<td>Diastolic Decrease</td>
<td>95.2 ± 3.2</td>
<td>91.1 ± 8.3</td>
<td>94.1 ± 3.0</td>
<td>92.4 ± 6.3</td>
</tr>
<tr>
<td></td>
<td>4.1 ± 8.3 (p = 0.05)</td>
<td></td>
<td>1.6 ± 6.5 (p = 0.09)</td>
<td></td>
</tr>
<tr>
<td>Mean arterial Decrease</td>
<td>112.6 ± 5.5</td>
<td>107.8 ± 8.5</td>
<td>110.1 ± 5.3</td>
<td>108.9 ± 6.9</td>
</tr>
<tr>
<td></td>
<td>4.8 ± 9.1 (p = 0.0005)</td>
<td></td>
<td>1.1 ± 7.7 (p = 0.32)</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD. The p values in parentheses represent paired t test comparisons within each treatment group. Adjusted differences are residual differences between the two treatment groups after adjusting for the effect of baseline blood pressure on change in blood pressure. Adjusted p values represent comparisons between the two treatment groups after adjusting for the effect of baseline blood pressure.
tension in blacks is more common and is associated with more severe target-organ damage than in whites, at least one large population survey shows that blacks ingest both less sodium and less potassium than whites.9

Animal models of hypertension also provide support for a role of potassium in the pathogenesis of hypertension.24 For instance, salt-sensitive rats on a high potassium, high sodium diet do not manifest the same degree of hypertension as similar rats on a low potassium, high sodium diet.2,4 Similarly, in the two-kidney, one clip model of renovascular hypertension, potassium attenuates the degree of blood pressure elevation that develops after renal artery clipping.1 In humans as well, potassium may decrease the hypertensive response to increased sodium intake.36,46

These experimental and epidemiological data suggest that potassium deficiency is etiological in hypertension and, conversely, that increased potassium intake protects against the development of hypertension. If these findings are confirmed, steps could be taken to increase the potassium intake of the population as a whole.

A related issue is the potential therapeutic role of potassium in persons with established hypertension. Potassium supplementation was associated with a small but statistically significant decrease in blood pressure in two double-blind, placebo-controlled crossover trials.25,29 A blood-pressure-lowering effect of potassium was found in these studies despite the small numbers of subjects studied (20 and 23, respectively), small doses of supplemental potassium (64 mEq/day in both studies), and the statistical hazards of crossover study design. In addition, Kaplan et al.30 reported improved blood pressure control in 16 hypertensive subjects with diuretic-induced hypokalemia when potassium was replete. However, the general applicability of the latter finding is unclear because of the small number of highly selected subjects studied. Potassium may have other effects on cardiovascular risk, independent of effects on blood pressure. Tobian et al.47 recently reported that stroke-prone spontaneously hypertensive rats fed a high potassium diet have a markedly decreased incidence of cerebrovascular events that occurs without any demonstrable effect on blood pressure.

Our study was designed to determine whether oral potassium supplementation is an efficacious and feasible therapeutic modality in an unselected group of adults with essential hypertension. After 8 weeks of 120 mEq per day of supplemental potassium or placebo, blood pressure decreased by an average of 6.4 ± 13.7/4.1 ± 8.3 mm Hg in the potassium-treated group, as contrasted with a negligible decrease in blood pressure in the placebo-treated group. An antihypertensive effect of potassium cannot be ruled out despite the fact that there were no statistically significant differences between treatment groups after adjusting for differences in baseline blood pressure. While small, the effect on mean blood pressure (a decrease of 4.8 ± 9.1 mm Hg compared with a decrease of 1.1 ± 7.7 mm Hg in the control group) may be clinically important. Rose33 has suggested that the prevalence and incidence of hypertension in a population are directly related to the mean blood pressure in that population. Furthermore, the small excess cardiovascular risk in the very large subset of the population with mild hypertension is translated into a large number of hypertension-related morbid events because that subset is so large. Therefore, Rose35 postulates that decreasing the mean blood pressure in a large population by as little as 3 mm Hg will reduce the number of persons with mild hypertension and therefore substantially reduce the number of people at excess risk of cardiovascular morbidity and mortality. Widespread increase in potassium consumption in this country might lead to a reduction in mean blood pressure of at least 3 mm Hg.

This study identifies subgroups in which further investigation of the effects of potassium on blood pressure is warranted. The effects of potassium on systolic blood pressure in the entire group and on systolic and diastolic blood pressure in blacks are particularly intriguing. Thirty-four percent of subjects had a 10 mm Hg or greater decrease in systolic blood pressure while taking supplemental potassium, as contrasted with 16% of subjects taking placebo. Because of the high prevalence of isolated systolic hypertension in the elderly, this subgroup might be particularly susceptible to an effect of potassium on systolic blood pressure. In addition, the dramatic decrease (19 ± 24/13 ± 10 mm Hg) in blood pressure in the small number of blacks taking potassium suggests that this subgroup may be particularly sensitive to the blood pressure-lowering effects of potassium. Although these subgroups were

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**Table 4. Effects of 8 Weeks of Potassium or Placebo Therapy in Black Patients**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Potassium (n = 5)</th>
<th>Placebo (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretreatment</td>
<td>Posttreatment</td>
</tr>
<tr>
<td>BP (mm Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>156 ± 28</td>
<td>137 ± 14</td>
</tr>
<tr>
<td>Diastolic</td>
<td>98 ± 3</td>
<td>85 ± 12</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>48 ± 14</td>
<td>—</td>
</tr>
<tr>
<td>Plasma K⁺ (mEq/L)</td>
<td>4.3 ± 0.2</td>
<td>—</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>88 ± 15</td>
<td>87 ± 16</td>
</tr>
</tbody>
</table>

Values are means ± SD.
not specifically addressed by this study, our data suggest that future research should investigate the effect of supplemental potassium on hypertension in these two hypertensive groups.

Three placebo recipients were prematurely removed from the study protocol because of progression of hypertension to diastolic blood pressures in excess of 105 mm Hg. No potassium-treated subject exhibited this degree of hypertension during the study. High potassium intake has been reported to protect humans from the progression of hypertension. In the present study, however, the sample size was too small and the observation period too short to draw any conclusions about this issue.

The present study demonstrated that short-term ingestion of 120 mEq of potassium per day in the form of microencapsulated potassium chloride is feasible. Over 90% of potassium doses were taken, despite suggestions in the medical literature that this type of intervention is not acceptable to patients. In addition, potassium intake of 170 to 270 mEq daily (50–150 mEq in the average American diet plus 120 mEq in supplemental potassium) was well tolerated. Clinically evident irritation of gastric mucosa did not occur, nor was occult gastrointestinal bleeding detected. The incidence of gastrointestinal side effects and dropout rates were comparable in the two treatment groups. Serum potassium levels increased by an average of 0.23 ± 0.46 mEq/L in subjects taking potassium but remained within the normal range. No other laboratory abnormalities could be attributed to potassium ingestion.

In summary, previous uncontrolled clinical trials and small crossover studies have suggested that potassium chloride lowers blood pressure. The current study, performed in a randomized, double-blind fashion in a large group with well-documented mild essential hypertension, does not conclusively establish potassium as an antihypertensive agent. However, despite the lack of a statistically significant difference in blood pressure between treatment groups after adjusting for baseline pressures, a blood pressure-lowering effect of potassium cannot be ruled out. Systolic blood pressure may be particularly sensitive to blood pressure-lowering effects of potassium and should be investigated further. This study also suggests that the effect of potassium on blood pressure of blacks with essential hypertension needs further investigation. The effect of potassium on blood pressure in essential hypertension and the role of potassium in the pathogenesis of hypertension have broad implications for our understanding and management of high blood pressure. At this time it would be premature to make recommendations concerning the treatment or prevention of high blood pressure with dietary or pharmacological potassium. A larger clinical trial stratified a priori by baseline blood pressure and race, or limited to a particular subgroup such as systolic hypertension in the elderly, is required to resolve the question of therapeutic utility. Epidemiological surveys as well as prospective studies will determine whether public health efforts to increase dietary potassium intake might prevent the development of hypertension.

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