Reproducibility of Blood Pressure Responses to Dietary Sodium and Potassium Interventions

The GenSalt Study

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Abstract—Blood pressure responses to dietary sodium and potassium interventions vary among individuals. We studied the long-term reproducibility of blood pressure responses to dietary sodium and potassium intake. We repeated the dietary sodium and potassium interventions among 487 Chinese adults 4.5 years after the original dietary intervention. The identical dietary intervention protocol, which included a 7-day low-sodium feeding (51.3 mmol/d), a 7-day high-sodium feeding (307.8 mmol/d), and a 7-day high-sodium feeding with oral potassium supplementation (60.0 mmol/d), was applied in both the initial and repeated studies. Three blood pressure measurements were obtained during each of the 3 days of baseline observation and on days 5, 6, and 7 of each intervention period. The results from the 24-hour urinary excretion of sodium and potassium showed excellent compliance with the study diet. Blood pressure responses to dietary intervention in the original and repeated studies were highly correlated. For example, the correlation coefficients (95% confidence interval) for systolic blood pressure levels were 0.77 (0.73–0.80) at baseline, 0.79 (0.75–0.82) during low sodium, 0.80 (0.77–0.83) during high sodium, and 0.82 (0.79–0.85) during high sodium and potassium supplementation interventions (all P<0.0001). The correlation coefficients for systolic blood pressure changes were 0.37 (0.29–0.44) from baseline to low sodium, 0.37 (0.29–0.44) from low to high sodium, and 0.28 (0.20–0.36) from high sodium to high sodium plus potassium supplementation (all P<0.0001). These data indicate that blood pressure responses to dietary sodium and potassium interventions have long-term reproducibility and stable characteristics in the general population. *(Hypertension. 2013;62:499-505.)*

Key Words: blood pressure ■ potassium, dietary ■ reproducibility of results ■ sodium, dietary

High dietary sodium and low potassium intake have been associated with increased blood pressure (BP) in animal experiments, observational epidemiological studies, and randomized controlled clinical trials.1-4 However, BP responses to changes in dietary sodium and potassium intake vary considerably among individuals, also known as salt sensitivity and potassium sensitivity of BP.4-8 Salt sensitivity of BP has been associated with an increased risk of hypertension, cardiovascular disease, and premature death.3-11 Currently, 2 common methods have been used for the assessment of salt sensitivity: (1) BP response to changed dietary sodium intake and (2) BP response during a rapid intravenous saline loading followed by diuretic-induced salt and volume depletion.12-15 In general, the dietary approach is believed to be more clinically relevant and considered the gold standard for the characterization of salt sensitivity.

A few clinical studies have examined the short-term reproducibility of BP response to dietary sodium change and have reported inconsistent findings.16-21 All of the studies have a small sample size, and most have <30 study participants. Some studies did not assess participants’ compliance to the dietary sodium intervention. Because all previous studies examined the short-term reproducibility of salt sensitivity (<1 year), it is still unknown whether salt sensitivity is a reproducible and stable trait during the long term. In addition, the reproducibility of potassium sensitivity of BP has not been studied.

In the Genetic Epidemiology Network of Salt Sensitivity (GenSalt) study, we tested and retested BP responses to dietary sodium and potassium interventions among 487 participants 4 to 5 years apart. To the best of our knowledge, this is the first study to investigate the long-term reproducibility of salt sensitivity and potassium sensitivity of BP.

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Methods

Study Participants

The GenSalt study was performed in rural areas in northern China. The initial GenSalt study was performed from October 2003 to July 2005. The details of the study design and methods have been published elsewhere. In brief, community-based BP screening was performed among persons aged 18 to 60 years in the study villages to identify potential probands and their families. Probands with a 60-mmol potassium (potassium chloride) supplement daily. The average time between test and retest was 4.4 years. Institutional Review Boards or Ethics Committees at all participating institutions approved the study protocol. Written informed consent was obtained from each study participant. This study adheres to the principles of the Declaration of Helsinki and Title 45, US Code of Federal Regulations, Part 46, Protection of Human Subjects, revised on November 13, 2001 and effective December 2003, and all study procedures were in accordance with institutional guidelines.

Dietary Intervention

The same dietary intervention protocol was applied to the initial study and the follow-up study. After a 3-day baseline observation, study participants received a low-salt diet (3 g of salt or 51.3 mmol of sodium per day) for 7 days and then a high-salt diet (18 g of salt or 307.8 mmol of sodium per day) for 7 days. During the first 2 intervention phases, potassium intake remained unchanged. In the final week, participants maintained a high-salt diet and took a 60-mmol potassium (potassium chloride) supplement daily. The average dietary sodium intake was 240 mmol per day in the study population. In previous studies of salt sensitivity, a dietary intake of 10 to 70 mmol sodium per day for the low-sodium intervention and a dietary intake of 180 to 345 mmol sodium per day for the high-sodium intervention were used. Although dietary salt intake was the same for all study participants, dietary total energy intake was varied according to their baseline energy intake. All foods were cooked without salt, and prepackaged salt was added to the individual study participant’s meal when it was served by the study staff. To ensure study participants’ compliance with the intervention program, they were required to have their breakfast, lunch, and dinner at the study kitchen under supervision of the study staff during the entire study period. Food consumption of study participants was carefully recorded at each meal by study staff members. Study participants were instructed to avoid consuming any foods that were not provided by the study. In addition, 3 timed urinary specimens (one 24 hour and 2 overnight) were collected at baseline and in each intervention phase to monitor participants’ compliance with dietary sodium and potassium intake. The overnight urinary excretions of sodium and potassium were converted to 24-hour values on the basis of formulas developed from study participants.

Data Collection

During a 3-day baseline examination of the initial study and the follow-up study, trained staff administered a standard questionnaire to collect information on demographic characteristics, medical history, and lifestyle risk factors (including cigarette smoking, alcohol drinking, and physical activity). Body weight and height were measured twice in light indoor clothing without shoes. Waist circumference was measured 1 cm above each participant’s naval during light breathing.

Three BP measurements were obtained each morning of the 3-day baseline examination and on days 5, 6, and 7 of each intervention period by the trained and certified observers using a random-zero sphygmomanometer according to a standard protocol. All sphygmomanometers were centrally calibrated using a standard method by a single technician at the Cardiovascular Institute of Chinese Academy of Medical Sciences in Beijing. In addition, BP observers participated in a centralized study training session on the use of the standardized protocol for BP measurement for both the initial study and the follow-up study. satisfactory performance was required during a written test on preparing study participants for BP measurement and selecting correct cuff sizes and while using standard techniques for BP measurement during a standardized videotape examination and during concordant measurements of BP with an instructor for certification as a study BP observer. To minimize measurement error, BP readings were obtained by the same BP observer using the same random-zero sphygmomanometer at approximately the same time each day for each study participant throughout the study. BP was measured with the participants in a sitting position after 5 minutes of rest. In addition, participants were advised to avoid alcohol, cigarette smoking, coffee/tea, and exercise for ≥30 minutes before their measurements. All BP observers were blinded to the dietary intervention. BP levels at baseline and during the intervention were calculated as the mean of 9 measurements during the 3-day baseline observation and on days 5, 6, and 7 of each intervention phase, respectively. Mean arterial pressure was calculated by adding one third of the pulse pressure to diastolic BP. BP responses were defined as follows: BP response to low sodium=BP on low-sodium diet – BP at baseline; BP response to high sodium=BP on high-sodium diet – BP on low-sodium diet; and BP response to potassium supplementation=BP on high-sodium diet with potassium supplementation – BP on high-sodium diet.

Statistical Analysis

The means and percentages of participants’ characteristics were calculated and compared between the initial and follow-up studies. The statistical significance of differences was examined by paired t-tests for continuous variables and χ² tests for categorical variables. Absolute BP levels and BP responses to dietary interventions were normally distributed. We used the Pearson correlation coefficient to assess the association of absolute BP levels during dietary interventions and BP responses to dietary interventions between the initial and repeated studies. In addition, partial correlation coefficients were calculated after adjusting for age, sex, field center, time intervals, and differences in room temperature between the initial and follow-up studies, as well as body mass index and BP at baseline examination. Additionally, Bland and Altman graphical approaches were used to test whether for each BP response variable, and the difference of the 2 measurements at the initial and follow-up studies varied in a systematic way against the mean of the 2 measurements. All analyses were performed using the SAS statistical package (version 9.2; SAS Institute, Cary, NC).

Results

The average age of the 487 participants was 39.7 years at the initial examination, and 49.3% of the participants were male. Mean body mass index, waist circumference, and BP levels increased significantly during the follow-up period (Table 1). The results from 24-hour urinary sodium and potassium excretions at baseline and during dietary interventions indicate excellent compliance with the dietary sodium and potassium interventions in both the initial and follow-up studies (Table 2).
BP changes during dietary interventions followed the same pattern in the initial study and the follow-up study. BP levels decreased from baseline to the low-sodium intervention, increased from the low-sodium intervention to the high-sodium intervention, and decreased again from the high-sodium intervention to the high-sodium plus potassium supplementation intervention (Table 3). Absolute BP levels during each intervention phase were highly correlated between the initial and follow-up studies. The correlation coefficients ranged from 0.79 to 0.82 for systolic BP, from 0.63 to 0.66 for diastolic BP, and from 0.72 to 0.76 for mean arterial pressure (all P<0.0001). After adjustment for multiple covariates, correlation coefficients did not change significantly.

Table 4 shows mean values and correlation coefficients for BP responses to low-sodium, high-sodium, and high-sodium plus potassium supplementation in the initial and follow-up studies. Figure 1 displays the scatterplots of BP responses to dietary interventions in the initial study versus those in the follow-up study. The correlation coefficients for BP responses to dietary interventions were moderate but highly significant. In general, the correlation coefficients for systolic BP responses were higher than those for diastolic BP responses, and the correlation coefficients for BP responses to the dietary sodium intervention were higher than those for BP responses to the dietary potassium intervention. The Bland–Altman plots did not show obvious relations between the differences and the means of BP responses from the initial and the follow-up studies (Figure 2).

### Discussion

In the present study, the correlations of BP responses to dietary sodium and potassium interventions between the 2 separate assessments 4.4 years apart are moderate and highly statistically significant. These data indicate that BP responses to changes in dietary sodium and potassium are not random phenomena but stable and reproducible human characteristics over a relatively long time period. These findings have potentially important clinical and public health implications. High dietary sodium intake is a major risk factor for hypertension. Population-based sodium reduction should be complemented by targeted sodium reduction among individuals who are more sensitive to sodium intake. In addition, the establishment of long-term reproducibility of salt sensitivity supports its pathogenic role in the pathogenesis of hypertension.

Salt sensitivity is a well-known phenomenon and has been observed in both hypertensive and normotensive subjects. Salt-sensitive subjects may be especially prone to developing hypertension compared with salt-resistant subjects. Older age, female sex, elevated baseline BP, and family history of hypertension have been associated with salt sensitivity. Many genetic variants have been identified to be associated with salt-sensitivity among different ethnicities.

A few studies have investigated the short-term reproducibility of salt sensitivity assessed using dietary sodium interventions. Most of these studies categorized study participants as salt sensitive or salt resistant using cutoffs of BP responses to dietary sodium change. The reported results of reproducibility of salt sensitivity varied greatly among studies. For example, Sharma et al used mean arterial pressure response to a reduced sodium intake (from 220 to 20 mmol/d) >3 mm Hg to define salt sensitivity and reported that the reliability of classification of salt sensitivity by the κ statistic was 0.87, implying a strong agreement between the 2 tests. However, Zoccali
et al\textsuperscript{21} reported a $\kappa$ statistic of 0.24 for the classification agreement between 2 tests using a BP change from a high-sodium to a low-sodium diet (from 170 to 40 mmol/d) >10% as the cutoff. Differences in the characteristics of subjects, magnitude and duration of dietary sodium change, subjects' compliance to dietary intervention, BP measurement method, and cutoff used for salt-sensitivity classification among studies may contribute to the inconsistent results about the reproducibility of salt sensitivity.

The GenSalt study has indicated that BP response to dietary sodium and potassium interventions are normally distributed among individuals.\textsuperscript{8} It would be arbitrary to categorize individuals into sensitive or resistant to sodium and potassium using specific cutoffs of BP responses to dietary

### Table 3. Blood Pressure Levels (mm Hg) During Dietary Interventions and Correlation Coefficients for the Initial Study vs the Follow-Up Study

<table>
<thead>
<tr>
<th>Blood Pressure, mm Hg</th>
<th>Initial Study</th>
<th>Follow-Up Study</th>
<th>CC* (95% CI)</th>
<th>Partial CC*† (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>SBP</td>
<td>118.1 (13.5)</td>
<td>123.3 (15.4)</td>
<td>0.77 (0.73–0.80)</td>
<td>0.79 (0.75–0.82)</td>
</tr>
<tr>
<td>DBP</td>
<td>74.3 (9.8)</td>
<td>80.4 (9.9)</td>
<td>0.65 (0.59–0.70)</td>
<td>0.68 (0.63–0.72)</td>
</tr>
<tr>
<td>MAP</td>
<td>88.9 (10.2)</td>
<td>94.7 (10.9)</td>
<td>0.72 (0.67–0.76)</td>
<td>0.75 (0.70–0.78)</td>
</tr>
<tr>
<td>Low-sodium intervention</td>
<td></td>
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</tr>
<tr>
<td>SBP</td>
<td>112.4 (12.7)</td>
<td>116.5 (13.6)</td>
<td>0.79 (0.75–0.82)</td>
<td>0.78 (0.74–0.81)</td>
</tr>
<tr>
<td>DBP</td>
<td>71.1 (9.4)</td>
<td>76.7 (9.2)</td>
<td>0.65 (0.59–0.70)</td>
<td>0.65 (0.59–0.70)</td>
</tr>
<tr>
<td>MAP</td>
<td>84.9 (9.6)</td>
<td>89.9 (10.7)</td>
<td>0.73 (0.68–0.77)</td>
<td>0.72 (0.68–0.76)</td>
</tr>
<tr>
<td>High-sodium intervention</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>SBP</td>
<td>118.2 (13.8)</td>
<td>123.7 (15.1)</td>
<td>0.80 (0.77–0.83)</td>
<td>0.77 (0.73–0.80)</td>
</tr>
<tr>
<td>DBP</td>
<td>74.3 (9.9)</td>
<td>80.1 (9.7)</td>
<td>0.63 (0.58–0.68)</td>
<td>0.63 (0.57–0.68)</td>
</tr>
<tr>
<td>MAP</td>
<td>89.0 (10.5)</td>
<td>94.6 (10.5)</td>
<td>0.73 (0.69–0.77)</td>
<td>0.71 (0.66–0.75)</td>
</tr>
<tr>
<td>Potassium Supplementation</td>
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</tr>
<tr>
<td>SBP</td>
<td>114.2 (13.6)</td>
<td>119.7 (14.5)</td>
<td>0.82 (0.79–0.85)</td>
<td>0.79 (0.75–0.82)</td>
</tr>
<tr>
<td>DBP</td>
<td>72.1 (9.4)</td>
<td>78.4 (9.6)</td>
<td>0.66 (0.61–0.71)</td>
<td>0.64 (0.59–0.69)</td>
</tr>
<tr>
<td>MAP</td>
<td>86.1 (10.0)</td>
<td>92.1 (10.3)</td>
<td>0.76 (0.72–0.80)</td>
<td>0.73 (0.68–0.77)</td>
</tr>
</tbody>
</table>

Blood pressure levels are given as mean (SD). CC indicates correlation coefficient; CI, confidence interval; DBP, diastolic blood pressure; MAP, mean arterial pressure; and SBP, systolic blood pressure.

\*All $P$ values for correlation coefficients are <0.0001.

\*Adjusted for age, sex, field center, time interval between the initial study and the follow-up study, and differences in room temperature between the initial study and the follow-up study.

### Table 4. Blood Pressure Response (mm Hg) to Dietary Interventions and Correlation Coefficients for the Initial Study vs the Follow-Up Study

<table>
<thead>
<tr>
<th>BP Response, mm Hg</th>
<th>Initial Study</th>
<th>Follow-Up Study</th>
<th>CC (95% CI)</th>
<th>Partial CC* (95% CI)</th>
<th>Partial CC† (95% CI)</th>
<th>Partial CC‡ (95% CI)</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Response to low sodium</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>SBP</td>
<td>−5.7 (6.4)</td>
<td>−6.8 (7.4)</td>
<td>0.37 (0.29–0.44)</td>
<td>&lt;0.0001</td>
<td>0.34 (0.26–0.42)</td>
<td>&lt;0.0001</td>
<td>0.25 (0.17–0.34)</td>
</tr>
<tr>
<td>DBP</td>
<td>−3.2 (5.0)</td>
<td>−3.7 (4.9)</td>
<td>0.23 (0.15–0.32)</td>
<td>&lt;0.0001</td>
<td>0.22 (0.14–0.31)</td>
<td>&lt;0.0001</td>
<td>0.17 (0.08–0.25)</td>
</tr>
<tr>
<td>MAP</td>
<td>−4.0 (4.9)</td>
<td>−4.8 (5.2)</td>
<td>0.28 (0.19–0.36)</td>
<td>&lt;0.0001</td>
<td>0.27 (0.19–0.35)</td>
<td>&lt;0.0001</td>
<td>0.19 (0.10–0.27)</td>
</tr>
<tr>
<td>Response to high sodium</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>5.8 (5.5)</td>
<td>7.2 (7.6)</td>
<td>0.37 (0.29–0.44)</td>
<td>&lt;0.0001</td>
<td>0.32 (0.23–0.39)</td>
<td>&lt;0.0001</td>
<td>0.28 (0.20–0.36)</td>
</tr>
<tr>
<td>DBP</td>
<td>3.2 (5.1)</td>
<td>3.5 (5.1)</td>
<td>0.34 (0.26–0.42)</td>
<td>&lt;0.0001</td>
<td>0.26 (0.18–0.35)</td>
<td>&lt;0.0001</td>
<td>0.25 (0.17–0.34)</td>
</tr>
<tr>
<td>MAP</td>
<td>4.1 (4.8)</td>
<td>4.7 (5.5)</td>
<td>0.36 (0.28–0.43)</td>
<td>&lt;0.0001</td>
<td>0.29 (0.21–0.37)</td>
<td>&lt;0.0001</td>
<td>0.27 (0.18–0.35)</td>
</tr>
<tr>
<td>Response to potassium supplementation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>−4.0 (5.1)</td>
<td>−3.9 (5.4)</td>
<td>0.28 (0.20–0.36)</td>
<td>&lt;0.0001</td>
<td>0.24 (0.15–0.32)</td>
<td>&lt;0.0001</td>
<td>0.20 (0.11–0.29)</td>
</tr>
<tr>
<td>DBP</td>
<td>−2.2 (4.3)</td>
<td>−1.8 (3.9)</td>
<td>0.13 (0.04–0.22)</td>
<td>0.004</td>
<td>0.14 (0.05–0.23)</td>
<td>0.002</td>
<td>0.14 (0.05–0.23)</td>
</tr>
<tr>
<td>MAP</td>
<td>−2.8 (4.0)</td>
<td>−2.5 (3.9)</td>
<td>0.19 (0.10–0.27)</td>
<td>&lt;0.0001</td>
<td>0.18 (0.09–0.26)</td>
<td>&lt;0.0001</td>
<td>0.17 (0.08–0.25)</td>
</tr>
</tbody>
</table>

Blood pressure responses are given as mean (SD). BP indicates blood pressure; CC, correlation coefficient; CI, confidence interval; DBP, diastolic blood pressure; MAP, mean arterial pressure; and SBP, systolic blood pressure.

\*Adjusted for age, sex, field center, time interval between the initial study and the follow-up study, and differences in room temperature between the initial study and the follow-up study (model 1).

\†Adjusted for variables in model 1 plus baseline body mass index in the initial study (model 2).

\‡Adjusted for variables in model 2 plus baseline blood pressure in the initial study.
sodium or potassium interventions. In the present study, we used BP responses to dietary sodium and potassium interventions as continuous traits to determine whether BP responses to dietary sodium and potassium interventions at the initial and follow-up studies are correlated 4.4 years apart. This approach should provide a more objective appraisal of the reproducibility of BP responses to dietary sodium and potassium interventions.

In the present study, the absolute BP levels during baseline and dietary intervention phases were highly correlated between the initial and follow-up studies. The correlation coefficients for absolute systolic BP and mean arterial pressure levels

Figure 1. Scatterplots of blood pressure responses to dietary interventions in the initial and follow-up studies. A–C, Blood pressure response to the low-sodium intervention. D–F, Blood pressure response to the high-sodium intervention. G–I, Blood pressure response to the high-sodium plus potassium supplementation intervention. Shaded areas show the 95% confidence interval for mean predicted values. DBP indicates diastolic blood pressure; MAP, mean arterial pressure; and SBP, systolic blood pressure.

Figure 2. Bland and Altman plots of blood pressure responses to dietary interventions in the initial and follow-up studies. A–C, Blood pressure response to the low-sodium intervention. D–F, Blood pressure response to the high-sodium intervention. G–I, Blood pressure response to the high-sodium plus potassium supplementation intervention. The x axis represents the mean of 2 measurements from the initial and follow-up studies, and y axis represents the difference between those measurements from the initial and follow-up studies. The middle solid line represents the mean difference, and the upper and lower dash lines represent the limits of agreement (mean±2 SD), respectively. DBP indicates diastolic blood pressure; MAP, mean arterial pressure; and SBP, systolic blood pressure.
during interventions were $\approx 0.8$. Similar findings have been reported in a previous study.\textsuperscript{20} However, the correlation coefficients for BP responses to dietary interventions were more moderate. The relatively weak correlation for BP responses is most likely because of their greater random errors. BP responses were calculated as the differences in BP measurements before and after intervention.

The correlation coefficients of BP responses to potassium supplementation were smaller than those to dietary sodium interventions before and after intervention. Responses were calculated as the differences in BP measurements before and after intervention.

Better understanding of the pathogenesis and identification of sodium (potassium) sensitive individuals may help the risk prediction and treatment of hypertension and related cardiovascular diseases.

Perspectives

Our study indicates that BP responses to dietary sodium and potassium interventions have long-term reproducibility and stable characteristics in the general population. Future studies should identify simple and convenient biomarkers for the classification of sodium (potassium) sensitivity in human subjects. Better understanding of the pathogenesis and identification of sodium (potassium) sensitive individuals may help the risk prediction and treatment of hypertension and related cardiovascular diseases.

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Disclosures

None.

References


**Novelty and Significance**

**What Is New?**
- The present study identified a moderate and highly statistically significant correlation between 2 separate assessments of blood pressure response to a dietary sodium intervention taken 4.4 years apart.
- The present study identified a moderate and highly statistically significant correlation between 2 separate assessments of blood pressure response to dietary potassium supplementation taken 4.4 years apart.

**What Is Relevant?**
- These data indicate that blood pressure responses to changes in dietary sodium and potassium are not random phenomena but stable and reproducible human characteristics during a relatively long time period.
- These findings provide scientific evidence for targeted sodium reduction among individuals who are more sensitive to sodium intake.
- In addition, the establishment of long-term reproducibility of salt sensitivity supports its pathogenic role in the pathogenesis of hypertension.

**Summary**

Our study indicates that blood pressure responses to dietary sodium and potassium interventions have long-term reproducibility and stable characteristics in the general population. Better understanding of the pathogenesis and identification of sodium and potassium sensitive individuals may help with risk prediction and treatment of hypertension and related cardiovascular diseases.
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