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# Elevation of Morning Blood Pressure in Sodium Resistant Subjects by High Sodium Diet

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The present study evaluated the response of blood pressure (BP) by dietary sodium in sodium resistant (SR) subjects. One hundred one subjects (mean age, 46.0 yr; 31 hypertensives) were admitted and given low sodium-dietary approaches to stop hypertension (DASH) diet (LSD, 100 mM NaCl/day) for 7 days and high sodium-DASH diet (HSD, 300 mM NaCl/day) for the following 7 days. On the last day of each diet, 24 hr ambulatory BP was measured. Morning systolic BP (SBP) and diastolic BP (DBP) were elevated after HSD in all subjects ($P < 0.01$), but daytime SBP and DBP were not changed ($P > 0.05$). In hypertensive subjects, morning DBP elevation was greater than daytime DBP elevation ($P = 0.036$), although both DBPs were significantly elevated after HSD. The augmented elevation of morning DBP in hypertensive subjects was contributed by the absolute elevation of morning DBP ($P = 0.032$) and relative elevation to daytime DBP ($P = 0.005$) in sodium resistant (SR) subjects, but not by sodium sensitive subjects. Although there was no absolute elevation, SR subjects with normotension showed a relative elevation of morning SBP compared to daytime SBP change after HSD ($P = 0.009$). The present study demonstrates an absolute and relative elevation of morning BP in SR subjects by HSD.

**Key Words:** Sodium; Hypertension; Sodium Resistance

## INTRODUCTION

High sodium intake is a well-known and important environmental factor for the development of hypertension and cardiovascular events. A reduction in dietary sodium intake reduces blood pressure (BP) and the long-term risk of cardiovascular events (1, 2).

However, reduction of sodium intake is not always effective, because BP response to high sodium intake is not uniform. Individuals showing a dramatic response to dietary sodium intake have been defined as having sodium sensitivity (SS) (3, 4). The prevalence of SS is higher in hypertensive subjects than in normotensives, showing a more exaggerated response to high sodium intake in hypertensive subjects (3-5). A long-term follow-up study revealed highest mortality in hypertensive subjects with SS, and lowest mortality in normotensive subjects with sodium resistance (SR) (6).

Diminished nighttime BP lowering and exaggerated morning BP elevation increase cardiovascular events (7-12). The disturbance of nocturnal BP decline by high sodium intake in hypertensive subjects with SS was attributed to a worsening mechanism of high sodium intake on cardiovascular events (13-15). However, it is questionable whether high sodium intake is safe for subjects with SR, because one study suggested possibly harmful effect of high sodium intake by showing an exaggerated response of morning BP surge by high sodium intake in hypertensive subjects with SR, but not with SS (16). Furthermore, no studies have been conducted in normotensive subjects.

The present study evaluated the effects of high sodium intake on morning BP response in subjects with SR.

## MATERIALS AND METHODS

The study design is described elsewhere (17, 18). In brief, 101 volunteers participated. Thirty one subjects were hypertensives and 70 subjects were normotensives. Subjects with stage 2 and



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3 hypertension (BP ≥ 160/100 mmHg) at the time of entry into the study, secondary hypertension, angina pectoris, myocardial infarction, congestive cardiac failure, stroke, diabetes mellitus, or chronic kidney disease (estimated glomerular filtration rate by the Modification of Diet in Renal Disease equation < 60 mL/min/1.73 $m^2$) were excluded. Participants were asked to maintain their usual dietary pattern and physical activity, and discontinue medications that affected BP or urinary electrolyte excretion, such as anti-hypertensive medications, from 2 weeks before hospitalization. After 1 day of the control diet, a low-sodium dietary approache to stop hypertension (DASH) diet (LSD; 100 mM NaCl/day) was given during the next 7 days. During the following 7 days, a high-sodium DASH diet (HSD; 300 mM NaCl/day) was given. All three meals were prepared by trained dietitians in the hospital kitchen. Dietary compliance was verified by two trained dieticians. Participants who could not consume > 90% of meals were dropped from the study; dietary compliance was 99.8%. All participants were hospitalized, their daily activity was monitored and their access to other food in addition to study meals was prohibited. During the last day of each dietary period, excretion of 24 hr urine sodium was measured. Twenty-four-hour ambulatory BP monitoring with an interval of 30 min was measured on the last day of each diet period using an automated, non-invasive oscillometric device (P6 Pressurometer; Del Mar Reynolds, Irvine, CA, USA) attached to the left upper arm. The study protocol and informed consent form were approved by the Institutional Review Board of Dongguk University Ilsan Hospital (IRB No. 2008-1-9), and written informed consent was obtained from every participants.

SS was defined as an increase in the 24 hr averaged mean BP (MBP) of > 4 mmHg ($P < 0.05$) in response to a HSD (17, 18). Daytime (average of readings between 1,000 and 2,000 hr), nighttime (average of readings between 0000 and 0600 hr) and morning (average of readings between 0630 and 0930 hr) BP were calculated. Morning surge of systolic BP (SBP) was calculated by subtracting the average of three SBP reading centered on the lowest nighttime reading from average of morning SBP readings between 0700 and 0900.

**Statistical analyses**

All statistical tests were two-sided, and the level of significance was set at 0.05. Continuous variables were expressed as the mean ± SEM and categorical variables were described by number and percentage in parentheses. Demographic and clinical characteristics were expressed by descriptive statistics for the 101 subjects, and stratified according to hypertensive and normotensive subjects. All categorical data were analyzed using Pearson's chi-square test. We performed a paired t-test to test the mean differences of BP, which were measured after HSD and LSD and Student's t-test to test the mean differences of continuous demographic data. Magnitude of SBP and diastolic BP (DBP) changes (expressed as ΔSBP and ΔDBP) were calculated by subtracting SBP and DBP after LSD from those after HSD, to compare relative changes of BP after HSD. Changes of nighttime and morning BP were compared to the changes of daytime BP by paired t-test. All outputs were produced using SPSS version 20 (SPSS, Chicago, IL, USA).

## RESULTS

Some of the demographic and clinical characteristics of the studied population have been described elsewhere (17, 18). BP after LSD and HSD are presented in the Tables (part of the BP data has been published elsewhere) (18). In all studied subjects, morning SBP and DBP were significantly elevated after HSD (Table 1). However, daytime SBP and DBP were not changed after HSD ($P > 0.05$). Hypertensive subjects showed significant elevation of all period SBP and DBP after HSD (Table 1). There were no changes of all period SBP and DBP in normotensive subjects after HSD (Table 1, $P < 0.05$). Subjects with SS also showed significant elevation of all period SBP and DBP after HSD ($P < 0.001$), irrespective of the presence of hypertension (Fig. 1). In contrast to the subjects with SS, there was a significant lowering of daytime SBP and DBP ($P < 0.005$) and no changes in morning SBP and DBP after HSD in the subjects with SR (Fig. 1). However, subgroup analysis revealed a significant elevation of morning DBP (from 84.6 ± 1.9 to 88.0 ± 1.7 mmHg,

**Table 1.** Blood pressure after one week of low and high sodium diet

| Blood pressure | | All (n = 101) | | | HT (n = 31) | | | NT (n = 70) | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | LSD | HSD | *P* value* | LSD | HSD | *P* value* | LSD | HSD | *P* value* |
| SBP | Daytime | 117.0 ± 1.1 | 117.7 ± 1.2 | 0.355 | 125.6 ± 1.7 | 129.0 ± 1.6 | 0.013 | 113.2 ± 1.1 | 112.7 ± 1.2 | 0.487 |
| | Nighttime | 107.7 ± 1.1 | 110.0 ± 1.3 | 0.007 | 115.3 ± 2.0 | 121.1 ± 1.8 | 0.001 | 104.3 ± 1.2 | 105.0 ± 1.3 | 0.438 |
| | Morning | 118.2 ± 1.2 | 120.8 ± 1.2 | 0.001 | 127.1 ± 1.7 | 132.6 ± 1.8 | < 0.001 | 114.3 ± 1.3 | 115.6 ± 1.1 | 0.137 |
| DBP | Daytime | 75.6 ± 0.9 | 76.3 ± 0.9 | 0.148 | 83.1 ± 1.4 | 85.0 ± 1.5 | 0.032 | 72.3 ± 0.8 | 72.4 ± 0.8 | 0.806 |
| | Nighttime | 68.4 ± 0.9 | 70.0 ± 0.9 | 0.004 | 74.4 ± 1.6 | 78.4 ± 1.5 | 0.001 | 65.8 ± 0.8 | 66.2 ± 0.9 | 0.415 |
| | Morning | 76.2 ± 0.9 | 78.0 ± 1.0 | 0.007 | 83.4 ± 1.3 | 87.8 ± 1.4 | < 0.001 | 73.0 ± 1.0 | 73.7 ± 1.0 | 0.418 |
| MBP | Daytime | 89.4 ± 0.9 | 90.1 ± 1.0 | 0.187 | 97.2 ± 1.4 | 99.7 ± 1.4 | 0.013 | 85.9 ± 0.8 | 85.8 ± 0.9 | 0.867 |
| | Nighttime | 81.5 ± 0.9 | 83.3 ± 1.0 | 0.003 | 88.0 ± 1.6 | 92.6 ± 1.6 | < 0.001 | 78.6 ± 0.9 | 79.1 ± 0.9 | 0.414 |
| | Morning | 90.2 ± 0.9 | 92.3 ± 1.0 | 0.001 | 97.9 ± 1.3 | 102.7 ± 1.4 | < 0.001 | 86.8 ± 1.0 | 87.6 ± 0.9 | 0.266 |

Data are expressed as a mean ± SEM. **P* value derived from paired t-test. NT, normotensive subjects; HT, hypertensive subjects; LSD, low sodium DASH diet; HSD, high sodium DASH diet; SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean blood pressure.


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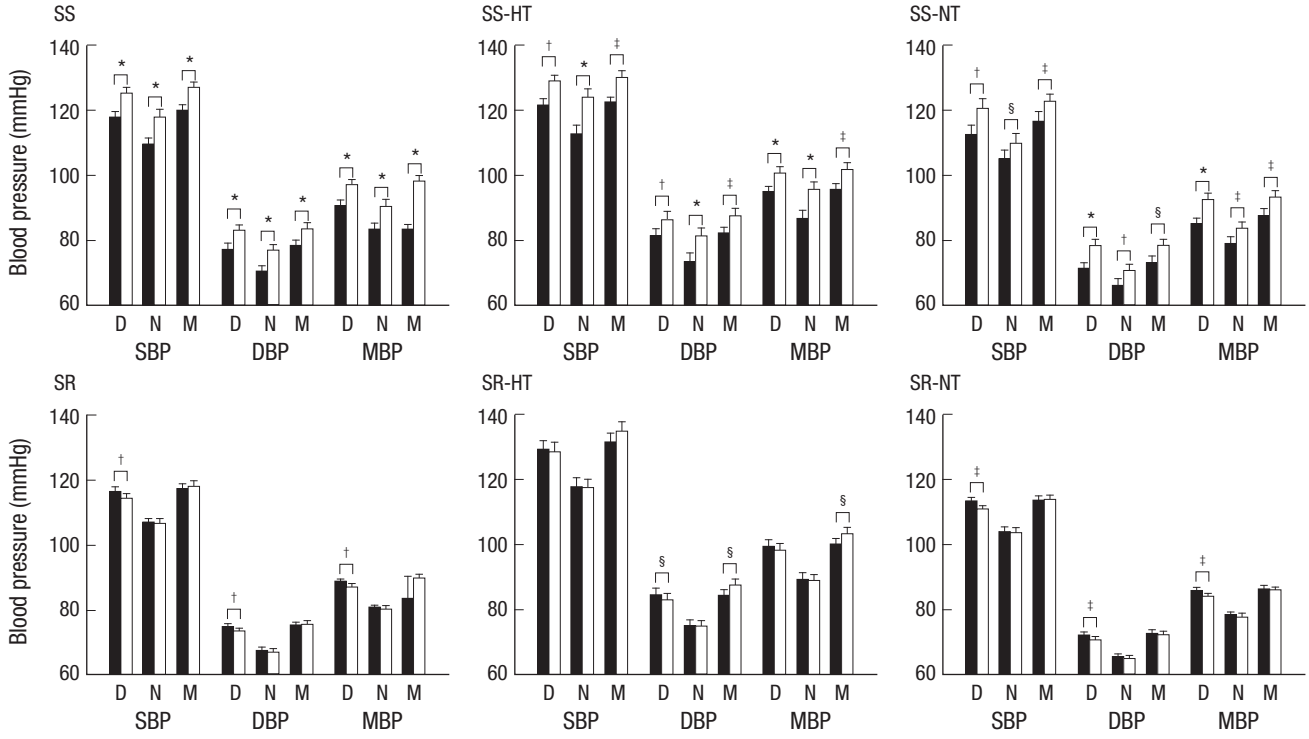


**Fig. 1.** Comparison of ambulatory blood pressure after one week of low sodium DASH diet (closed bar) and high sodium DASH diet (open bar). Study population was divided into two groups, subjects with sodium sensitivity (SS) and sodium resistance (SR). Each group was further grouped in hypertensive subjects with sodium sensitivity (SS-HT) and sodium resistance (SR-HT), and normotensive subjects with sodium sensitivity (SS-NT) and sodium resistance (SR-NT). Data are expressed as a mean±SEM. $^{*}P < 0.0001$; $^{\dagger}P < 0.001$; $^{\ddagger}P < 0.01$; $^{\S}P < 0.05$. *P* value was derived from paired t-test. D, daytime; N, nighttime; M, morning; SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean blood pressure.

*P* = 0.032) and significant lowering of daytime DBP (from 84.8 ± 2.0 to 83.4 ± 1.9 mmHg, *P* = 0.048) after HSD in hypertensive subjects with SR (Fig. 1). Normotensive subjects with SR showed a lowering of daytime BP and no change of morning BP (Fig. 1).

Magnitudes of BP changes (ΔBP) from LSD to HSD were compared to evaluate whether there was a significant difference in the relative changes of BP. The data is depicted in the Fig. 2. In all studied subjects, morning ΔSBP were significantly higher when compared to daytime ΔSBP (*P* = 0.016). Hypertensive subjects showed significantly higher morning ΔDBP (*P* = 0.036), when compared to daytime ΔDBP. However, morning ΔBP were not different from daytime ΔBP in normotensive subjects (Fig. 2, first row). Although there were significant elevations of BP after HSD, the magnitude of BP elevations in subjects with SS were not different between each time period, irrespective of the presence of hypertension (Fig. 2, second row). On the other hand, in the subjects with SR (Fig. 2, third low), daytime ΔBP were negative values (*i.e., BP lowering*), while morning ΔSBP and ΔDBP were positive values (*i.e., BP elevation*) with significant difference to daytime ΔBP (ΔSBP *P* = 0.001, ΔDBP *P* = 0.012). Hypertensive subjects with SR showed higher morning ΔDBP (*P* = 0.005) and marginally higher morning ΔSBP (*P* = 0.051) compared to daytime ΔDBP and ΔSBP, respectively. Normotensive subjects with SR showed significantly higher morning ΔSBP than daytime ΔSBP (*P* = 0.009). The direction of the morning SBP changes was opposite to the daytime SBP changes.

Morning SBP surge of hypertensive subjects with SR was higher compared to hypertensive subjects with SS. However, there was no significant change of morning SBP surge by HSD in both hypertensive subjects with SR or SS from LSD (Table 2). In normotensive subjects, there was no difference of morning SBP surge between subjects with SR and SS. Although it was insignificant, normotensive subjects with SR showed a tendency of elevation in morning SBP surge after HSD (from 13.9 ± 1.4 to 16.0 ± 1.5 mmHg, *P* = 0.270).

In the present study population, the prevalence of SS increased to 48.5% from 27.7% when SS was defined as an increase in either of 24 hr averaged MBP or morning averaged MBP more than 4 mmHg in response to a HSD. In this new definition, 23 hypertensive (74.2%) and 26 normotensive (37.1%) had SS. From the original definition of SS, 21 subjects with SR (28.8%), 7 hypertensive subjects with SR (46.7%), and 14 normotensive subjects with SR (24.1%) had morning SS.

## DISCUSSION

Several studies reported the effect of high sodium intake on the

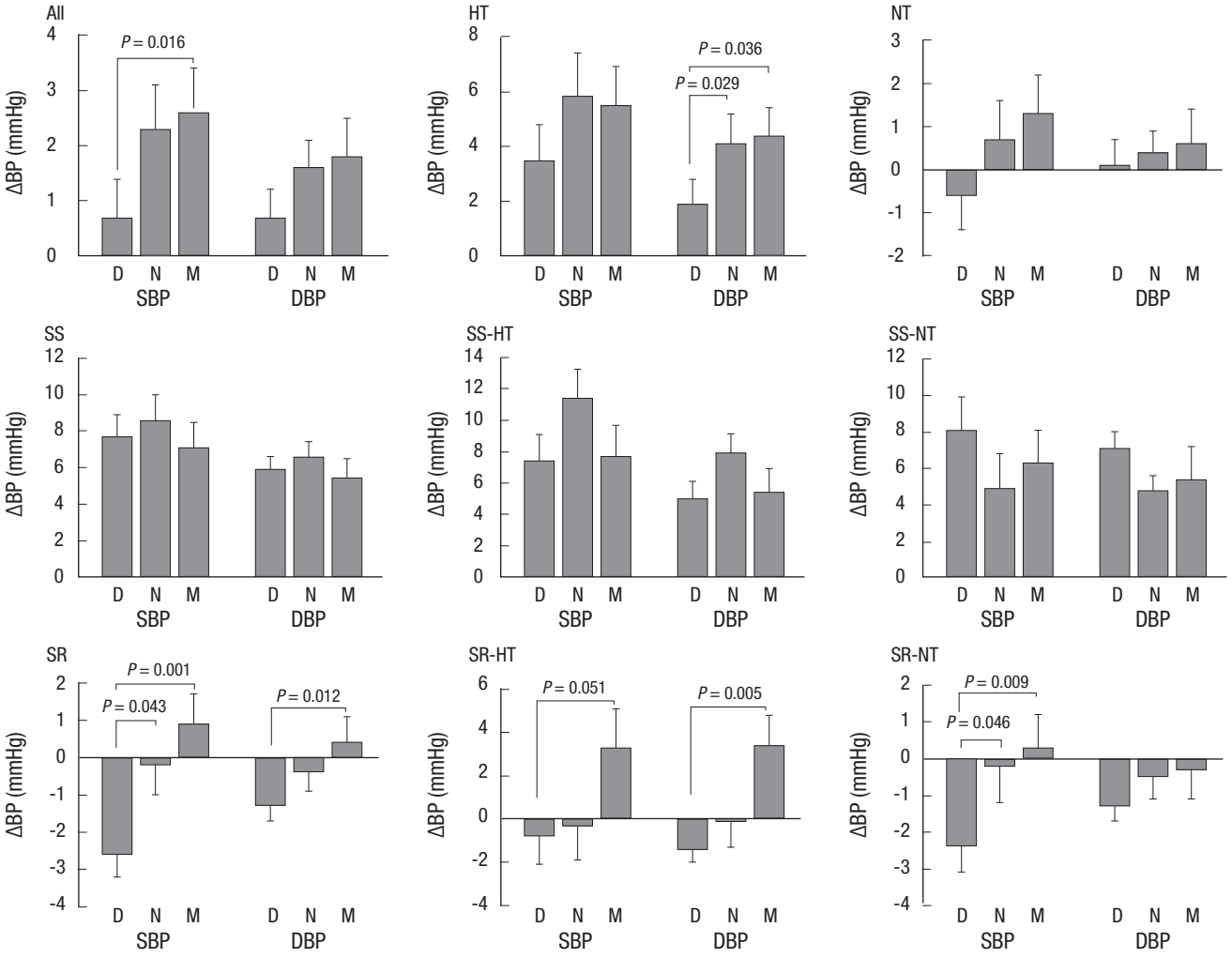


**Fig. 2.** Comparisons of daytime, nighttime and morning blood pressure changes. Data are expressed as a mean±SEM. *P* value derived from paired t-test of morning BP changes vs daytime BP changes, and nighttime BP changes vs daytime BP changes. ΔBP, magnitude of blood pressure change calculated by subtracting blood pressure after low sodium diet from that after high sodium diet; NT, normotensive subjects; HT, hypertensive subjects; SS, sodium sensitive subjects; SR, sodium resistant subjects; SS-HT, hypertensive subjects with sodium sensitivity; SS-NT, normotensive subjects with sodium sensitivity; SR-HT, hypertensive subjects with sodium resistance; SR-NT, normotensive subjects with sodium resistance; SBP, systolic blood pressure; DBP, diastolic blood pressure; D, daytime; N, nighttime; M, morning.

**Table 2.** Morning BP surge after low sodium and high sodium DASH diet

| Subjects | After LSD (mmHg) | After HSD (mmHg) | *P* value* |
|---|---|---|---|
| Hypertensive subjects | | | |
| SS (n = 16) | 14.5 ± 2.5 | 14.4 ± 2.6 | 0.989 |
| SR (n = 15) | 21.9 ± 3.7 | 22.9 ± 2.4 | 0.802 |
| *P* value† | 0.098 | 0.024 | |
| Normotensive subjects | | | |
| SS (n = 12) | 15.7 ± 3.1 | 15.2 ± 2.7 | 0.879 |
| SR (n = 58) | 13.9 ± 1.4 | 16.0 ± 1.5 | 0.270 |
| *P* value† | 0.822 | 0.615 | |

Data are expressed as a mean ± SEM. **P* value derived from paired t-test, comparison of SBP surge after HSD to after LSD; †*P* value derived from independent t-test, comparison SBP surge between SS and SR. SS, sodium sensitive subjects; SR, sodium resistant subjects; LSD, low sodium DASH diet; HSD, high sodium DASH diet.

elevation of morning BP and nighttime BP decline (13-16), addressing changes of circadian BP variation as the worsening mechanism of high sodium intake for the adverse cardiovascular events. The elevation of morning BP, (i.e., morning SBP surge and morning hypertension), is an important risk factor of cardiovascular events. Long-term prospective studies have demonstrated the strong and independent effects of morning BP surge and morning hypertension in the prediction of cardiovascular events (9, 11), outcomes (10), and stroke (12).

Morning hypertension is an absolute elevation of morning BP. A previous study, the only study to date that has evaluated the effect of high sodium intake on morning BP in hypertensive subjects with SR, showed no absolute elevation of morning MBP by high sodium intake, although there was a tendency of elevation (16). The present study demonstrates for the first time a significant elevation of morning DBP and MBP in hypertensive subjects with SR by high sodium intake. In addition, subgroup analysis showed that the augmented elevation of morning DBP in hypertensive subjects compared to daytime DBP el-


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evation was contributed by the elevation of morning DBP in hypertensive subjects with SR, but not by hypertensive subjects with SS. The greater elevation of morning BP indicates that high sodium intake may also be harmful in hypertensive subjects with SR.

Another striking feature of the present study is the different response of daytime and morning BP in normotensive subjects with SR by a HSD. In normotensive subjects with SR, daytime BP was significantly lowered after HSD. However, morning BP was not changed by a HSD despite lowering of daytime BP, indicating different response of BP to HSD depending on the period of circadian rhythm. Although there was no absolute change of morning BP after HSD, analysis revealed a relative elevation of morning SBP after HSD in normotensive subjects with SR. The magnitude of SBP changes was different with the opposite direction of morning SBP changes to that of daytime SBP changes, showing the relative elevation of morning SBP compared to change of daytime BP by high sodium intake in normotensive subjects with SR.

Morning surge of SBP is the relative elevation of morning BP to nighttime BP. The inconsistent result in prognostic significance of morning surge of SBP reflects the influence of nighttime SBP levels (9-11,19). Osanai et al. (16) reported an exacerbation of morning mean BP (MBP) surge by high sodium intake in hypertensive subjects with SR. There was no exacerbation of morning MBP surge in hypertensive subjects with SS (16). The present study, which evaluated morning SBP surge, showed higher morning SBP surge of hypertensive subjects with SR compared to hypertensive subjects with SS, consistent with a previous study (16). However we could not demonstrate a significant elevation of morning SBP surge by high sodium diet in hypertensive subjects with SR. Explanations for the differences in the results are different definitions of SS and the background DASH diet. The DASH diet attenuates the effect of sodium on the elevation of BP (20). Thus, it is possible that the effect of HSD to increase morning SBP surge in hypertensive subjects with SR may be abolished by DASH diet. In addition, morning DBP and MBP in hypertensive subjects with SR elevated with no changes of nighttime DBP and MBP by the HSD, suggesting the possibility of increase in morning DBP and MBP surge by this diet. However, we did not evaluate the morning surge of DBP and MBP, because there is no evidence for the prognostic significance of morning DBP and MBP surge. In addition there was an insignificant increase in morning SBP surge after HSD in normotensive subjects with SR.

In addition to the elevation of morning BP, diminished nighttime BP decline is related to the high frequency of strokes in hypertensive subjects (7), and cardiovascular mortality in the general population independent to the 24 hr BP (8). Uzu et al. (13) reported failure of nocturnal BP decline in hypertensive subjects with SS, but not in hypertensive subjects with SR. The authors also showed change of BP circadian rhythm from nondipper to dipper by sodium restriction, with the change being confined to the hypertensive subjects with SS (14). In the hypertensive subjects with SR, nocturnal BP decline was not affected by sodium restriction. In the present study, HSD attenuated nocturnal BP decline only in hypertensive subjects with SS (data not shown) which is consistent with the result of Uzu et al. (13) and Higashi et al. (15).

The evident BP change by high sodium intake in the present study adds to the weight of evidence of the detrimental effect of high sodium intake on cardiovascular health, which may be independent to the presence of SS. The striking changes of morning BP by HSD in subjects with SR indicate that SR is not a true resistance to high sodium intake, but is actually a misunderstanding because SS of previous studies was determined by casual BP measurement, not by 24 hr ambulatory BP measurement (3, 21). In the present study, the prevalence of SS increased when morning SS was included in the definition of SS. Likewise, the status of individual SS depends on the definition of SS. Thus, if we consider the different circadian response of BP to sodium intake, the results of the present and previous study (16) indicate that hypertensive subjects with SR are not safe from high sodium intake. Furthermore, although the implication of relative BP elevation on cardiovascular health is remained to be proven, it is possible that different changes of morning BP (i.e., relative elevation) may have a harmful effect in normotensive subjects with SR. Thus, sodium intake reduction without discrimination of the presence of SS is prudent as a general public approach.

The limitation of the present study is the small number of subjects including hypertensive subjects. However, previous studies did not include normotensive subjects (13-16). The present study included normotensive subjects, providing new information of normotensive subjects for the first time. The second limitation is the DASH diet, used as a background diet. The DASH diet was given for 2 weeks. This length of the DASH diet can lower BP (22) and may attenuate the effects of high sodium intake on BP (20). However, the present study showed an elevation of morning BP by high sodium intake. Although the BP differences were small, they were significant. The findings of the present study suggest that a DASH diet-only approach may not be enough to protect from cardiovascular events (20). Finally, the present study did not use a random order allocation of dietary schedule and did not examine the reproducibility of the SS test. There are controversies in the reproducibility of SS test. Several studies reported high rate of false-positive and -negative in the SS test (23, 24). However, other investigators showed SS as a reproducible phenomenon (25, 26). Although there exists a possibility of falsely classified SS or SR in the present study, elevation of morning BP in all studied subjects of the present study by HSD supports the recommendation for lower

sodium intake directed at the general population rather than SS individuals (23).

Despite these limitations and a need of further well designed studies in large populations, the present study demonstrates the harmful effect of high sodium intake by morning BP elevation on hypertensive subjects with SR and possibly on normotensive subjects with SR for the first time. The present results provide additional evidence of sodium intake reduction in general population. Furthermore, epidemiologic and large intervention studies (1, 2, 27) performed irrespective of the presence of hypertension and SS, demonstrated the beneficial effects of sodium intake reduction in the general population for the prevention of cardiovascular events. Thus, targeting a general population irrespective of the presence of hypertension and SS, which was insisted in a previous study (23), is an appropriate way to reduce cardiovascular mortality and morbidity by high sodium intake.

In conclusion, the present study shows an absolute and relative elevation of morning BP by high sodium intake in subjects with SR. This emphasizes the importance of sodium intake reduction in the general population.

## DISCLOSURE

JW Kim and HK Park are employees of Korea Food and Drug Administration. No conflicts of interest relevant to this article have been reported by MY Rhee, CY Lim, SJ Shin, SW Oh, YS Park, Cl Kim, and CY Park.

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**Supplement Table 1.** Percent decline of nocturnal mean blood pressure after each diet

| Subjects | % decline of BP after LSD | % decline of BP after HSD | Difference of % decline of BP between LSD and HSD | *P** |
|---|---|---|---|---|
| All (n = 101) | 8.7 (0.6) | 7.5 (0.6) | 1.3 (0.7) | 0.0552 |
| SS (n = 28) | 8.0 (1.3) | 6.8 (1.2) | 1.2 (1.4) | 0.3781 |
| SR (n = 73) | 9.0 (0.7) | 7.7 (0.7) | 1.3 (0.7) | 0.0884 |
| *P*† | 0.4875 | 0.5290 | | |
| Hypertensives (n = 31) | 9.5 (1.1) | 6.9 (1.2) | 2.5 (1.0) | 0.0181 |
| SS (n = 16) | 8.8 (1.8) | 4.9 (1.7) | 3.8 (1.7) | 0.0344 |
| SR (n = 15) | 10.2 (1.2) | 9.1 (1.6) | 1.1 (1.1) | 0.3092 |
| *P*† | 0.4999 | 0.0841 | | |
| Normotensives (n = 70) | 8.4 (0.8) | 7.7 (0.7) | 0.7 (0.8) | 0.3933 |
| SS (n = 12) | 7.1 (2.1) | 9.4 (1.3) | - 2.3 (1.9) | 0.2522 |
| SR (n = 58) | 8.7 (0.8) | 7.3 (0.8) | 1.3 (0.9) | 0.1455 |
| *P*† | 0.4289 | 0.3047 | | |

Data are expressed as a mean (SEM). **P* value derived from paired t-test of nocturnal mean blood pressure decline after low sodium diet and high sodium diet; †*P*-value derived from comparison by Student's t-test of percent decline of mean blood pressure between sodium sensitive subjects and sodium resistant subjects. LSD, low sodium DASH diet; HSD, high sodium DASH diet; SS, sodium sensitive subjects; SR, sodium resistant subjects.

**Supplement Table 2.** Number of non-dippers after each diet period

| Subjects | After LSD (%) | *P* value | after HSD (%) | *P* value |
|---|---|---|---|---|
| All (n = 101) | | | | |
| SS (n = 28) | 17 (60.7) | 0.8683 | 21 (75.0) | 0.2068 |
| SR (n = 73) | 43 (58.9) | | 45 (61.6) | |
| Hypertensives (n = 31) | | | | |
| SS (n = 16) | 9 (56.3 ) | 0.8705 | 13 (81.3) | 0.0443 |
| SR (n = 15) | 8 (53.3) | | 7 (46.7) | |
| Normotensives (n = 70) | | | | |
| SS (n = 12) | 8 (66.7) | 0.6822 | 8 (66.7) | 0.9391 |
| SR (n = 58) | 35 (60.3) | | 38 (65.5) | |

*P* value from chi-square test. LSD, low sodium DASH diet; HSD, high sodium DASH diet; SS, sodium sensitive subjects; SR, sodium resistant subjects.


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**Supplement Table 3.** Differences of daytime, nighttime, and morning blood pressure changes

| | | All | | NT | | HT | | SS | | SR | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Mean (SEM) | *P* value* | Mean (SEM) | *P* value* | Mean (SEM) | *P* value* | Mean (SEM) | *P* value* | Mean (SEM) | *P* value* |
| ΔSBP | Daytime | 0.7 (0.7) | | -0.6 (0.8) | | 3.5 (1.3) | | 7.7 (1.2) | | -2.0 (0.6) | |
| | Nighttime | 2.3 (0.8) | | 0.7 (0.9) | | 5.8 (1.6) | | 8.6 (1.4) | | -0.2 (0.8) | |
| | Morning | 2.6 (0.8) | | 1.3 (0.9) | | 5.5 (1.4) | | 7.1 (1.4) | | 0.9 (0.8) | |
| ΔDBP | Daytime | 0.7 (0.5) | | 0.1(0.6) | | 1.9 (0.9) | | 5.9 (0.7) | | -1.3 (0.4) | |
| | Nighttime | 1.6 (0.5) | | 0.4 (0.5) | | 4.1 (1.1) | | 6.6 (0.8) | | -0.4 (0.5) | |
| | Morning | 1.8 (0.7) | | 0.6 (0.8) | | 4.4 (1.0) | | 5.4 (1.1) | | 0.4 (0.7) | |
| ΔMBP | Daytime | 0.7 (0.5) | | -0.1 (0.6) | | 2.4 (0.9) | | 6.5 (0.7) | | -1.6 (0.4) | |
| | Nighttime | 1.8 (0.6) | | 0.5 (0.6) | | 4.6 (1.2) | | 7.2 (1.0) | | -0.3 (0.6) | |
| | Morning | 2.0 (0.6) | | 0.8 (0.7) | | 4.8 (1.0) | | 5.9 (1.1) | | 0.5 (0.7) | |
| SBP | Nighttime ΔSBP-Daytime ΔSBP | 1.6 (0.8) | 0.0634 | 1.3 (1.1) | 0.2290 | 2.3 (1.4) | 0.1174 | 0.9 (2.0) | 0.6553 | 1.9 (0.9) | 0.0426 |
| | Morning ΔSBP-Daytime ΔSBP | 1.9 (0.8) | 0.0158 | 1.9 (0.9) | 0.0504 | 2.1 (1.5) | 0.1680 | -0.6 (1.7) | 0.7062 | 2.9 (0.9) | 0.0011 |
| DBP | Nighttime ΔDBP-Daytime ΔDBP | 0.9 (0.5) | 0.0997 | 0.3 (0.6) | 0.6245 | 2.1 (0.9) | 0.0287 | 0.7 (1.1) | 0.5379 | 0.9 (0.6) | 0.1186 |
| | Morning ΔDBP-Daytime ΔDBP | 1.1 (0.6) | 0.0711 | 0.5 (0.7) | 0.4863 | 2.5 (1.1) | 0.0361 | -0.5 (1.3) | 0.6974 | 1.8 (0.7) | 0.0115 |
| MBP | Nighttime ΔMBP-Daytime ΔMBP | 1.1 (0.6) | 0.0630 | 0.6 (0.7) | 0.3993 | 2.2 (1.0) | 0.0313 | 0.7 (1.3) | 0.5687 | 1.2 (0.7) | 0.0611 |
| | Morning ΔMBP-Daytime ΔMBP | 1.4 (0.6) | 0.0206 | 0.9 (0.7) | 0.1775 | 2.4 (1.1) | 0.0423 | -0.6 (1.3) | 0.6614 | 2.1 (0.6) | 0.0012 |

| | | SS-NT | | SS-HT | | SR-NT | | SR-HT | |
|---|---|---|---|---|---|---|---|---|---|
| | | Mean (SEM) | *P* value | Mean (SEM) | *P* value | Mean (SEM) | *P* value | Mean (SEM) | *P* value |
| ΔSBP | Daytime | 8.1 (1.8) | | 7.4 (1.7) | | -2.4 (0.7) | | -0.8 (1.3) | |
| | Nighttime | 4.9 (1.9) | | 11.4 (1.8) | | -0.2 (1.0) | | -0.3 (1.6) | |
| | Morning | 6.3 (1.8) | | 7.7 (2.0) | | 0.3 (0.9) | | 3.3 (1.8) | |
| ΔDBP | Daytime | 7.1 (0.9) | | 5.0 (1.1) | | -1.3 (0.4) | | -1.4 (0.6) | |
| | Nighttime | 4.8 (0.8) | | 7.9 (1.2) | | -0.5 (0.6) | | -0.1 (1.2) | |
| | Morning | 5.4 (1.8) | | 5.4 (1.5) | | -0.3 (0.8) | | 3.4 (1.4) | |
| ΔMBP | Daytime | 7.4 (0.9) | | 5.8 (1.1) | | -1.7 (0.5) | | -1.2 (0.7) | |
| | Nighttime | 4.8 (1.1) | | 9.1 (1.3) | | -0.4 (0.7) | | -0.1 (1.0) | |
| | Morning | 5.7 (1.6) | | 6.1 (1.5) | | -0.2 (0.8) | | 3.4 (1.4) | |
| SBP | Nighttime ΔSBP-Daytime ΔSBP | -3.2 (3.0) | 0.3145 | 4.0 (2.4) | 0.1266 | 2.2 (1.1) | 0.0463 | 0.5 (1.3) | 0.6910 |
| | Morning ΔSBP-Daytime ΔSBP | -1.8 (2.7) | 0.5231 | 0.2 (2.2) | 0.9210 | 2.6 (1.0) | 0.0087 | 4.1 (1.9) | 0.0505 |
| DBP | Nighttime ΔDBP-Daytime ΔDBP | -2.3 (1.4) | 0.1190 | 2.9 (1.4) | 0.0539 | 0.8 (0.7) | 0.2223 | 1.3 (1.2) | 0.3038 |
| | Morning ΔDBP-Daytime ΔDBP | -1.7 (2.2) | 0.4680 | 0.3 (1.6) | 0.8331 | 1.0 (0.7) | 0.1988 | 4.8 (1.4) | 0.0047 |
| MBP | Nighttime ΔMBP-Daytime ΔMBP | -2.6 (1.7) | 0.1422 | 3.3 (1.6) | 0.0580 | 1.3 (0.8) | 0.1037 | 1.1 (1.1) | 0.3328 |
| | Morning ΔMBP-Daytime ΔMBP | -1.7 (2.1) | 0.4206 | 0.3 (1.6) | 0.8524 | 1.5 (0.7) | 0.0360 | 4.6 (1.4) | 0.0049 |

**P* value derived from paired t-test, morning BP changes vs daytime BP changes, nighttime BP changes vs daytime BP changes. NT, normotensive subjects; HT, hypertensive subjects; SS, sodium sensitive subjects; SR, sodium resistant subjects; SS-HT, hypertensive subjects with sodium sensitivity; SS-NT, normotensive subjects with sodium sensitivity; SR-HT, hypertensive subjects with sodium resistance; SR-NT, normotensive subjects with sodium resistance; SBP, systolic blood pressure, DBP, diastolic blood pressure, MBP, mean blood pressure; ΔSBP, changes of systolic blood pressure (systolic blood pressure after high sodium DASH diet – systolic blood pressure after low sodium DASH diet); ΔDBP, changes of diastolic blood pressure (diastolic blood pressure after high sodium DASH diet – diastolic blood pressure after low sodium DASH diet); ΔMBP, changes of mean blood pressure (mean blood pressure after high sodium DASH diet – mean blood pressure after low sodium DASH diet).