High Salt Diet Sensitizes Cardiopulmonary Baroreflexes in Dahl Salt-Resistant Rats

RONALD G. VICTOR, DONALD A. MORGAN, PETER THOREN, AND ALLYN L. MARK

SUMMARY Compared with Dahl salt-resistant (R) rats, Dahl salt-sensitive (S) rats on a low salt diet have impaired cardiopulmonary baroreflex control of sympathetic nerve activity. The purpose of this study was to examine the sensitivity of cardiopulmonary baroreflex function in both strains of Dahl rats when they are challenged with a high salt diet. We studied Dahl R and S rats after 6 weeks of low and high salt diets. To assess cardiopulmonary baroreflex function, we measured decreases in splanchnic sympathetic nerve activity produced by increases in left ventricular end-diastolic pressure during graded volume expansion (dextran 75) after bilateral sinoaortic denervation. A given amount of infused volume produced comparable increases in left ventricular end-diastolic pressure in Dahl R rats on low and high salt diets but significantly greater decreases in sympathetic nerve activity in the high salt group than in the low salt group. Thus, the high salt diet augmented the sympathoinhibitory response to volume expansion in the Dahl R rats. In contrast, among the Dahl S rats equivalent increases in left ventricular end-diastolic pressure during volume expansion produced smaller sympathoinhibitory responses in the high salt group than in the low salt group. The gain of the cardiopulmonary baroreflex, expressed as the percentage decrease in sympathetic nerve activity per mm Hg increase in left ventricular end-diastolic pressure, was significantly increased by a high salt diet in Dahl R rats but tended to be decreased by a high salt diet in Dahl S rats. In conclusion, this study demonstrates that a high salt diet sensitizes the cardiopulmonary baroreflex in Dahl R rats but not in Dahl S rats. We suggest that this augmentation in the reflex restraint of sympathetic neural outflow in Dahl R rats on a high salt diet may exert a protective effect against the development of salt-induced hypertension. Conversely, the lack of this compensatory augmentation in baroreflex function in Dahl S rats might contribute to salt sensitivity. (Hypertension 8 [Suppl II]: II-21-II-27, 1986)

KEY WORDS • high salt diet • cardiopulmonary baroreflexes • sympathetic nerve activity • Dahl rats

Dahl salt-sensitive (S) rats develop hypertension when put on a high salt diet but remain normotensive on a rigorously low salt diet. In contrast, Dahl salt-resistant (R) rats remain normotensive with either a low or a high salt diet. Several studies indicate the importance of humoral and renal factors in the pathogenesis of salt-induced hypertension in Dahl S rats.1-4

In addition, there is increasing evidence that alterations in the neurogenic control of sympathetic nerve activity (SNA) and vascular resistance play an important part in Dahl salt-dependent hypertension.5-7 These alterations in neurogenic control relate in part to peripheral adrenergic8 and central neural mechanisms.9-12 Prehypertensive Dahl S rats on a low salt diet are reported to have reduced arterial13-15 and cardiopulmonary baroreflexes,16 as compared with Dahl R rats. This phenomenon is mediated primarily by a decreased afferent baroreceptor discharge and not by altered central neural integration.15,16 Since decreased arterial and cardiopulmonary baroreceptor function can be demonstrated in Dahl S rats before they develop hypertension or cardiac hypertrophy, these alterations may be genetically determined.

The differences in arterial baroreceptor function between Dahl R and S rats on a low salt diet are further accentuated when the rats are challenged with high salt. Thus, Ferrari and Mark17 have reported that a high salt diet enhances the afferent discharge of aortic baro-
receptors in Dahl R rats but inhibits afferent discharge in Dahl S rats.

The purpose of the present study was to examine the sensitivity of cardiopulmonary baroreflex (CPBR) function when Dahl R and S rats are challenged with a high salt diet. Our interest in this concept was prompted by increasing evidence that baroreceptor endings are sensitive to ionic and humoral influences.\(^\text{18-19}\) Accordingly, we speculated that CPBR function might also be influenced by humoral or ionic adjustments (or both) during dietary salt intake. Specifically, we hypothesized that in Dahl R rats a high salt diet sensitizes the CPBR. Increased CPBR gain during a high salt diet would facilitate this reflex restraint of SNA and thus help protect against salt-induced hypertension. We further hypothesized that Dahl S rats lack this protective augmentation in CPBR function.

**Methods**

Twenty-five Dahl R rats and 20 Dahl S rats were randomly assigned to a low salt (0.1% NaCl) or high salt (8.0% NaCl) diet 1 week after they had been weaned and were studied during the 7th week of the diet. The experiments conformed with the American Physiological Society’s guiding principles for animal experiments.

**Surgical Procedure and Hemodynamic Measurements**

For the studies of CPBR control of SNA, 21 Dahl R rats and 15 Dahl S rats were used. Each animal was anesthetized with urethan (1.2 g/kg, i.p.). Polyethylene catheters were inserted into a femoral artery to measure arterial pressure, into a vein to perform volume expansion, and into the left ventricle (through the right common carotid artery) to monitor left ventricular end-diastolic pressure (LVEDP). LVEDP was used as a measure of left heart filling pressure and the stimulus to cardiopulmonary baroreceptors.

Sinoaortic baroreceptors were denervated by cutting the aortic depressor nerves at their junction with the superior laryngeal nerve and by stripping the arterial walls in the carotid sinus region and painting them with 10% phenol. The cervical sympathetic chains and superior laryngeal nerves were also cut. The effectiveness of sinoaortic denervation was confirmed by the failure of phenylephrine-induced increases in mean arterial pressure (20–40 mm Hg) to elicit reflex decreases in the heart rate and in SNA.

The trachea was cannulated, and the animal was paralyzed with decamethonium bromide (0.3 mg/kg, i.v., and small supplements as needed) and ventilated artificially with O\(_2\)-enriched air. Tidal volume and respiratory rate were adjusted to maintain arterial blood pH between 7.35 and 7.45 throughout the experiment. Body temperature was maintained at 38 °C with a heating pad.

**Recording of Sympathetic Nerve Activity**

The abdomen was opened through a midline incision. The left greater splanchnic nerve was isolated and cut just proximal to the cardiac sympathetic ganglion in the abdomen. The central cut end of the splanchnic nerve was placed on a silver–silver chloride electrode and suspended in a pool of mineral oil for multiunit recordings of efferent SNA. The nerve action potentials were detected by a high-impedance probe and amplified 50,000-fold to 100,000-fold by a Grass (Quincy, MA, USA) band pass P511 amplifier with a band width of 100 and 3000 Hz. For monitoring during the experiment, the filtered neurogram was routed through a Tektronix oscilloscope (Chicago, IL, USA) and through an amplitude discriminator to an audio amplifier and loudspeaker. For permanent recording and analysis, the filtered neurogram was fed through a nerve-traffic analyzer (No. 706C, University of Iowa Bioengineering), which counted nerve spikes exceeding a threshold voltage set just above the noise level. The counter’s time bin was set at 1 second, so that the impulse frequency was displayed as the number of spikes collected each second (Hz) on a time-frequency histogram. This display was used to measure changes in SNA during graded volume expansion. Measured neural and hemodynamic variables were recorded continuously on an Beckman RM Dynograph recorder (Schiller Park, IL, USA) at a paper speed of 50 mm/min (50 mm/sec when LVEDP values were to be measured).

**Protocol**

After the experimental preparation had been completed and the hemodynamic and neural parameters had stabilized, graded volume expansion was performed by injection of dextran 75 into the femoral vein in 0.5-ml increments to a total of 5 ml. This protocol produced increases in LVEDP of at least 15 mm Hg in all animals. Increments in volume were administered over 15 to 30 seconds. Data were obtained during the peak response immediately after each increment in volume. After volume expansion, blood was withdrawn to restore preinfusion values of LVEDP. The vagi were then cut in the neck, and after 10 minutes, volume expansion and withdrawal were repeated. The animal was then killed (air embolus), and the noise level of the neurogram was determined by inspection of the oscilloscope display.

**Data Analysis**

To evaluate the reflex effects of volume expansion and stimulation of cardiopulmonary receptors, LVEDP (mm Hg) and SNA (Hz) were measured under control conditions and after each increment in volume expansion. The maximal increases in LVEDP and corresponding decreases in SNA during volume expansion were averaged over 15 heart beats during the peak responses immediately after each increment in volume. The data were compiled and analyzed on an IBM personal computer (PCAarmonk, NY, USA). For each animal, the curve relating LVEDP and SNA over the complete volume expansion was displayed on an IBM-PC video screen. Maximal CPBR gain was defined as the slope of the regression equation for the first four infusion steps of the baroreflex curve and was expressed as the percentage decrease in SNA per mm Hg increase in LVEDP. The first four infusion steps (a
total of 2 ml of dextran) were used to calculate CPBR gain for two reasons: first, these steps resulted in a linear relationship between LVEDP and SNA in each animal, and second, they encompassed a physiological range of cardiac filling pressures. Values of LVEDP in mm Hg were also plotted against increments of infused volume in milliliters of dextran per 100 g of body weight. The data analysis compared values in the group of animals on a high salt diet and the group on a low salt diet for each strain; comparisons between the two strains are not included in this report. Statistical analysis was performed using unpaired t-tests and analysis of variance with the Bonferroni method for multiple comparisons. Differences with p values under 0.05 were considered significant. Results are expressed as means ± SE.

**Results**

**Base-Line Data**

The high salt diet did not alter values of mean arterial pressure or heart rate before volume expansion (after sinoaortic denervation) in Dahl R rats (Table 1). Mean arterial pressure recorded during anesthesia tended to be higher in Dahl S rats on high salt diet than in those on low salt diet, but these values were not significantly different (see Table 1). LVEDP before volume expansion was similar in Dahl S rats on low and high salt diets (5.4 ± 0.6 and 5.4 ± 0.7 mm Hg, respectively) but was significantly higher (p < 0.05) in Dahl R rats on a high salt diet (7.5 ± 0.5 mm Hg) than in those on a low salt diet (5.9 ± 0.6 mm Hg). Dahl R rats on a low salt diet were heavier than those on a high salt diet (220 ± 5 vs 198 ± 6 g, p < 0.05); a similar tendency was also seen in the Dahl S rats (216 ± 4 g for those on a low salt diet vs 199 ± 5 for those on a high salt diet, p = 0.10).

**Responses to Volume Expansion**

Volume expansion after sinoaortic denervation increased the mean arterial pressure and LVEDP and decreased SNA without causing significant changes in the heart rate (see Tables 1 and 2; Figures 1 and 2). Bilateral vagotomy abolished the decreases in nerve activity associated with volume expansion. Specifically, infusion of 2 to 3 ml of dextran after vagotomy produced average increases in LVEDP of 11 to 12 mm Hg but only insignificant changes in SNA (−6.6 ± 3.4% in Dahl R rats on low salt, −8.3 ± 5.0% in Dahl R rats on high salt, −9.3 ± 6.4% in Dahl S rats on low salt, and −2.5 ± 3.6% in Dahl S rats on high salt).

**Effects of Dietary Salt on Sympathetic Nerve and Pressor Responses to Volume Expansion**

In Dahl R rats, high salt diet augmented the sympathetic inhibitory responses to volume expansion over a wide range of LVEDP values (see Table 2 and Figure 2). Specifically, equivalent amounts of dextran in Dahl R rats on low salt diet (0.94 ± 0.03 ml/100 g) and high salt diet (1.01 ± 0.02 ml/100 g) caused comparable increases in LVEDP in the two groups (+10.0 ± 1.4 and +9.9 ± 1.4 mm Hg, respectively) but produced a greater decrease in SNA in the high salt group (-55 ± 5% vs -30 ± 4%, respectively; p < 0.05).

In Dahl S rats, the high sodium diet tended to attenuate the decreases in SNA produced by increases in LVEDP (see Table 2 and Figure 2). Dextran (0.92 ± 0.02 ml/100 g in the low salt group and 1.02 ± 0.03 ml/100 g in the high salt group) produced equivalent increases in LVEDP in the two groups (+8.1 ± 0.4 and +7.6 ± 0.7 mm Hg, respectively) but caused a smaller decrease in nerve activity in Dahl S rats on a high salt diet than in those on a low salt diet (−21 ± 5% vs −26 ± 3%, respectively).

Figure 3 shows the effects of dietary salt on CPBR gain, expressed as the percentage decrease in SNA per mm Hg increase in LVEDP. CPBR gain for Dahl R rats was −6.24 ± 0.49 in the high salt group versus −4.38 ± 0.37 in the low salt group (p < 0.01). Thus, the high salt diet significantly augmented CPBR gain in Dahl R rats. In contrast, CPBR gain tended to be lower in Dahl S rats on a high salt diet than in those on a low salt diet (−2.49 ± 0.47 vs −3.59 ± 0.44, respectively; p = 0.10).

The high salt diet tended to decrease the pressor response to volume expansion in Dahl R rats, whereas it tended to augment the pressor response to volume expansion in Dahl S rats (see Table 1).

**Table 1. Arterial Pressure and Heart Rate Before and After Volume Expansion in Dahl Salt-Resistant (R) and Salt-Sensitive (S) Rats**

<table>
<thead>
<tr>
<th>Experimental group</th>
<th>Baseline values</th>
<th>Response to volume expansion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean arterial pressure (mm Hg)</td>
<td>Heart rate (beats/min)</td>
</tr>
<tr>
<td>Dahl R rats</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low salt</td>
<td>106 ± 8</td>
<td>412 ± 15</td>
</tr>
<tr>
<td>High salt</td>
<td>102 ± 8</td>
<td>419 ± 8</td>
</tr>
<tr>
<td>Dahl S rats</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low salt</td>
<td>104 ± 7</td>
<td>413 ± 20</td>
</tr>
<tr>
<td>High salt</td>
<td>121 ± 7</td>
<td>419 ± 14</td>
</tr>
</tbody>
</table>

Data are means ± SE for 12 Dahl R rats fed 0.1% NaCl, 11 Dahl R rats fed 8.0% NaCl, 6 Dahl S rats fed 0.1% NaCl, and 9 Dahl S rats fed 8.0% NaCl. Baseline values are those obtained in anesthetized rats after bilateral sinoaortic denervation. Responses to volume expansion represent changes after infusion of 2 ml of dextran. See text for discussion of data.
TABLE 2. Responses to Volume Expansion in Dahl Salt-Resistant (R) and Salt-Sensitive (S) Rats

<table>
<thead>
<tr>
<th>Infusion step</th>
<th>Low salt</th>
<th>High salt</th>
<th>Low salt</th>
<th>High salt</th>
<th>Low salt</th>
<th>High salt</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔVol (ml/100 g)</td>
<td>ΔLVEDP (mm Hg)</td>
<td>ΔSNAR (%)</td>
<td>ΔVol (ml/100 g)</td>
<td>ΔLVEDP (mm Hg)</td>
<td>ΔSNAR (%)</td>
<td>ΔVol (ml/100 g)</td>
</tr>
<tr>
<td>1</td>
<td>0.24 ± 0.01</td>
<td>2.4 ± 1.3</td>
<td>0.25 ± 0.01</td>
<td>1.7 ± 0.5</td>
<td>0.01 ± 0.01</td>
<td>0.7 ± 0.6</td>
</tr>
<tr>
<td>2</td>
<td>0.47 ± 0.01</td>
<td>4.6 ± 3.4</td>
<td>0.50 ± 0.01</td>
<td>4.4 ± 0.8</td>
<td>0.01 ± 0.01</td>
<td>0.8 ± 5.9</td>
</tr>
<tr>
<td>3</td>
<td>0.70 ± 0.02</td>
<td>6.4 ± 4.8</td>
<td>0.76 ± 0.01</td>
<td>7.2 ± 1.1</td>
<td>0.01 ± 0.01</td>
<td>1.1 ± 5.8</td>
</tr>
<tr>
<td>4</td>
<td>0.94 ± 0.03</td>
<td>10.0 ± 4.4</td>
<td>1.01 ± 0.02</td>
<td>9.9 ± 1.4</td>
<td>0.02 ± 0.02</td>
<td>1.4 ± 4.6</td>
</tr>
</tbody>
</table>

Data are means ± SE for 12 Dahl R rats fed 0.1% NaCl, 11 Dahl R rats fed 8.0% NaCl, 6 Dahl S rats fed 0.1% NaCl, and 9 Dahl S rats fed 8.0% NaCl. Vol = volume of dextran in milliliters per 100 g of body weight; LVEDP = left ventricular end-diastolic pressure; SNA = splanchnic sympathetic nerve activity.

Responses to graded volume expansion with intravenous injections of dextran 75 in 0.5-ml steps. Equivalent amounts of dextran produced comparable increases in LVEDP in Dahl R rats on low and high salt diets but greater reflex decreases in SNA in the high salt group. Equivalent amounts of dextran also produced similar increases in LVEDP in Dahl S rats on low and high salt diets. However, the high salt diet tended to attenuate the sympathoinhibitory response to volume expansion in the Dahl S rats.

*p < 0.05 for differences in responses between high and low salt groups of a given strain.

Effects of Dietary Salt on Increases in Left Ventricular End-Diastolic Pressure During Volume Expansion

Table 2 and Figure 4 show the increases in LVEDP during graded volume expansion in Dahl R and S rats on high and low salt diets. The increase in LVEDP (mm Hg) for a given increase in volume (milliliters of dextran per 100 g of body weight) did not differ significantly between the high and low salt groups in either the Dahl R rats (9.79 ± 1.14 and 9.86 ± 1.23, respectively) or the Dahl S rats (7.53 ± 0.80 and 8.44 ± 0.33, respectively).

![Figure 1](http://hyper.ahajournals.org/)

**Figure 1.** Segments of an original record from a Dahl salt-resistant rat (8.0% NaCl diet) showing the sympathoinhibitory response to stimulation of cardiopulmonary baroreceptors by graded volume expansion (i.e., dextran) after bilateral sinoaortic denervation. From top to bottom: arterial pressure (AP), heart rate (HR), left ventricular end-diastolic pressure (LVEDP), and sympathetic nerve activity (SNA) are displayed as a time-frequency histogram. Values of SNA expressed as a percentage of the control value are also shown. The panel labeled LVEDP is a tracing of left ventricular pressure recorded at high gain (0 to 60 mm Hg) to facilitate measurements of LVEDP, which are shown below the pressure tracings.
Discussion

The major new finding of this study is that a high salt diet markedly augments CPBR restraint of SNA in Dahl R rats. In contrast, a high salt diet in Dahl S rats does not augment but rather tends to decrease the effectiveness of CPBR inhibition of SNA. Thus, a high salt diet produces contrasting effects on CPBR function in the two strains of rats. The discussion below addresses the possible mechanisms and pathophysiological implications of these findings.

Augmented sympathoinhibitory responses during volume expansion in Dahl R rats fed high salt cannot be explained by arterial baroreceptors or excitatory sympathetic afferents, since the arterial baroreceptors were denervated and since volume expansion produced no significant changes in SNA after vagotomy. Thus, this phenomenon must involve cardiopulmonary vagal afferents. This action could be mediated by effects on one or more of the components of the reflex arc. These include central processing of afferent input, ganglionic transmission, and afferent mechanisms.

From the observations in this study we cannot determine which mechanisms are involved in the salt-induced changes in cardiac baroreflex function in Dahl R and S rats, but several observations from recent studies in our laboratory lead us to speculate that afferent mechanisms are probably responsible.

For example, a study from our laboratory found no...
The difference between R and S rats in terms of their sympathetic nerve responses to electrical stimulation of the central end of the aortic depressor nerve. Since the central pathways of arterial and cardiopulmonary baroreceptors are at least partly similar, the finding that central integration of responses to aortic nerve stimulation is normal in Dahl S rats suggests that alterations in cardiac baroreflexes in Dahl R rats might also reside at the afferent rather than central or ganglionic level. In this regard, we have identified impairment in both aortic baroreceptor and cardiac vagal afferent activity in prehypertensive Dahl S rats on a low salt diet. In addition, a high salt diet sensitizes arterial baroreceptor afferents in Dahl R rats. Specifically, increases in multifiber aortic depressor nerve activity produced by increases in arterial pressure were augmented by a high salt diet in Dahl R rats but were attenuated in Dahl S rats. These findings involving the effects of dietary salt on arterial baroreceptor discharge parallel the present findings regarding the effects of dietary salt on cardiac baroreflex control of SNA in Dahl rats. Thus, we speculate that the salt-induced augmentation of the cardiac baroreflex in Dahl R rats is mediated by sensitization of cardiac vagal afferents.

Could the effect of a high salt diet on cardiac baroreflex function in Dahl R rats have been caused simply by an increase in the mechanical stimulus? Baseline LVEDP was significantly higher in Dahl R rats fed high salt than in those fed low salt. However, it is difficult to explain our findings on the basis of a resetting of receptors, since the high salt diet altered the slope of the relationship between changes in LVEDP and SNA (see Figure 2).

Another possibility is that the high salt diet altered cardiac distensibility or systemic venous compliance. However, equivalent increases in infused volume, expressed in milliliters per 100 g of body weight, produced comparable increases in LVEDP in Dahl R rats on high and low salt diets. These observations suggest that the augmented sympathoinhibitory response to volume expansion in Dahl R rats on the high salt diet does not result from a greater mechanical stimulus but instead probably reflects sensitization of cardiac vagal afferents.

The mechanisms responsible for salt-induced sensitization of the CPBR in Dahl R rats are unknown. In view of the work on the ionic sensitivity of baroreceptors, one possible explanation might involve salt-induced changes in the ionic environment of the vagal afferent endings in the heart. However, this is unlikely, since a high salt diet does not alter the concentrations of sodium and potassium in the serum or in the arterial walls of Dahl rats. A second possibility relates to baroreceptor sensitization by humoral factors released during high salt intake. For example, high salt diet has been reported to increase secretion of a natriuretic hormone that inhibits Na⁺,K⁺-ATPase could be involved in the baroreceptor-sensitizing effect of a high sodium diet.

Another humoral substance that might be involved is atrial natriuretic factor. Although it does not alter Na⁺,K⁺-ATPase, there is increasing evidence that atrial natriuretic factor exerts neural as well as renal and direct vascular effects. Two recent studies demonstrate that it acts in the heart to stimulate cardiopulmonary vagal afferent discharge and thereby inhibits SNA. Since levels of atrial natriuretic factor are reportedly increased during a high salt diet, this substance could be involved in the sensitization of the CPBR during a high salt diet in Dahl R rats.

Why does a high salt diet enhance CPBR function in Dahl R rats but not in Dahl S rats? One possible explanation is that Dahl S rats release less of a sensitizing humoral factor in response to a high salt diet. In the Sabra strain, for example, plasma concentrations of a ouabain-like natriuretic factor tend to be lower in hypertension-prone rats than in hypertension-resistant rats. An alternative explanation is that a primary (i.e., genetic) abnormality in the cardiopulmonary baroreceptors may render the Dahl S rats unresponsive to a baroreceptor-sensitizing agent.

The concept that long-term changes in dietary salt may alter CPBR function is not new. Takishita and Ferrario reported that long-term severe sodium depletion augmented the tonic inhibitory influence of cardiac vagal afferents on arterial baroreflex control of renal nerve activity in dogs. This effect was attributed to a central redistribution of blood volume (i.e., an increased mechanical stimulus) or to a change in central neural integration. The distinctive feature of our study of the Dahl strain is the suggestion that long-term changes in dietary salt might alter the discharge properties of baroreceptor endings (i.e., a sensitizing, not a mechanical, effect).

Our studies were performed after sinoaortic baroreceptor denervation to remove the buffering influence of arterial baroreceptors and permit the study of CPBR function. In this experimental situation, volume expansion produced significant increases in arterial pressure, presumably by increasing cardiac output. We have previously demonstrated that interruption of vagal afferents augments the pressor responses to volume expansion. This indicates that CPBR inhibition of SNA helps to buffer the pressor response to volume expansion. In this regard, it is interesting to note that the pressor responses to volume expansion were greater in Dahl S rats on a low salt diet than in Dahl R rats on a similar diet. Of even greater interest, high salt diet tended to attenuate the pressor responses to volume expansion in Dahl R rats (see Table 1), whereas high salt intake tended to augment the responses to volume expansion in Dahl S rats. These trends may be explained by contrasting effects of the high salt diet on cardiopulmonary vagal afferent discharge in the two strains. We speculate that high salt intake sensitizes cardiopulmonary vagal afferents in Dahl R rats and thereby tends to attenuate pressor responses to volume expansion; high salt intake tends to desensitize the
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CPBR in Dahl S rats and thus tends to augment the pressor responses to volume expansion.

In conclusion, this study demonstrates that a high salt diet sensitizes the CPBR in Dahl R rats but not in Dahl S rats. We suggest that the augmentation in the reflex restraint of efferent SNA in Dahl R rats on a high salt diet may exert a protective effect against the development of salt-induced hypertension. Conversely, the lack of this compensatory augmentation in baroreflex function in Dahl S rats might contribute to salt sensitivity.

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References


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