# Brain-tissue nNOS activities and nNOS immunohistochemical reactivities in Dahl salt hypertensive rats.

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## Summary

We have demonstrated that intraperitoneal administration of 7-nitroindazole or intracerebroventricular injection of S-methyl-L-thiocitrullin markedly increases tonic sympathetic discharge, especially sympathetic discharge generated before baroreflex-mediated inhibition, in Dahl salt-sensitive (DS) hypertensive rats. In the present study, we have focused on the brain-tissue nNOS activities in the brainstem, diencephalons and cerebellum, and also nNOS immunohistochemical reactivities in the brainstem of Dahl salt hypertensive rats.

**Methods and Results:** DS and Dahl salt-resistant (DR) rats were fed a regular salt (0.4% NaCl) or a high salt (8% NaCl) diet for 4 weeks. After the sodium load, nNOS activities of the brain stem, diencephalons and cerebellum were determined by the citrulline method. The DS hypertensive rats showed significant increase in nNOS activity of the brain stem but did not of the diencephalons or cerebellum. Brain-tissue nNOS activities at 2-week-old, 4-week-old, 8-week-old and 12-week-old ages were compared between in normotensive DS and DR rats. However, there was no significant difference at any age or in any parts of brain between the two rat lines. Immunohistochemical reactivities of nNOS were examined in the brainstem of hypertensive and normotensive DS rats by the modified ABC method. nNOS was strongly expressed in the neuronal cells of several nuclei in the brainstem of both hypertensive and normotensive Dahl rats. More than 4 nuclei in the brainstem showed nNOS reactivities. The total number of nNOS-reactive cells was larger in hypertensive rats than in normotensive rats.

**Conclusion**: nNOS neurons in the brainstem but not in the diencephalons or cerebellum may be up-regulated in salt-sensitive hypertensive rats. The up-regulated nNOS neurons may strongly suppress tonic sympathetic discharge, generated before baroreflex-mediated inhibition, in hypertensive Dahl rats. The neuronal NO-mediated suppression mechanism may be markedly enhanced in salt-induced hypertension. The genetic differences between DS and DR rats do not produce any significant difference in brain-tissue nNOS expressions without sodium load or hypertensive load.