Role of brain Na^+ channels in the pressor mechanism of salt-sensitive hypertensive models.

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Summary

Changes in the renin-angiotensin system (RAS) mRNAs in the brain and the kidney of rats after administration of deoxycorticosterone acetate-salt (DOCA) and/or sodium chloride were assessed by using a competitive polymerase chain reaction method. Benzamil, a blocker of amiloride-sensitive sodium channels, was infused intracerebroventricularly or intravenously for 7 days in DOCA-salt or renal hypertensive rats, and the effects of benzamil on the brain RAS mRNAs were determined. Renin and angiotensin I-converting enzyme (ACE) mRNAs were not downregulated in the brain of rats administered with DOCA and/or salt, whereas these mRNAs were decreased in the kidney. Intracerebroventricular infusion of benzamil decreased renin, ACE, and angiotensin II type-1 (AT1) receptors mRNAs in the brain of DOCA-salt hypertensive rats, but not of renal hypertensive rats. The gene expression of the brain RAS, particularly renin and ACE, is regulated differently between the brain and the kidney in DOCA-salt hypertensive rats, and benzamil blockable brain sodium channels may participate in the regulation of the brain RAS mRNAs.