23) Effect on Na^+ , K^+ ATPase Activity of Erythrocytes after Acute and Chronic Salt Loading in Normotensive WKY Rats.

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Introduction

It is well established from the variety of experimental or clinical investigations that dietary sodium intake is important in the development of some types of experimental and human essential hypertension. It is also well-known that there is a heterogeneity with respect to susceptibility to sodium. Some hypertensives and borderline hypertensives are salt-sensitive, since blood pressure increases in response to excess salt loading. However, mechanisms of rise in blood pressure caused by excess salt have not been clarified. According to experimental study in salt sensitive rats, an inherited disturbance in sodium excretion might lead to a compensatory rise in the circulating level of Na^+ , K^+ ATPase inhibitor, which, while returning sodium balance towards normal, might also cause hypertension. In this study, using non salt-sensitive, Wistar-Kyoto rats (WKY), Na^+ , K^+ ATPase activity of erythrocytes and blood pressure were examined after acute and chronic salt loading to clarify the involvement of endogenous Na^+ , K^+ ATPase inhibitor in salt resistancy.

Materials and Methods

Fifteen male Wistar-Kyoto rats (WKY) at the age of 3 months were randomly divided into 3 groups, 5 of each. The first group was given 1% sodium chloride in drinking water (chronic salt loading group - A group). The second group was given tap water, and given 1% sodium chloride for last 3 days (acute salt loading group - B group). The third group was given tap water throughout the experiment (control group - C group). After 7 months all rats were checked for blood pressure by tail-pulse-pickup method, body weight and 24-hour water or salt water intake and sodium excretion (by flamephotometry) were measured while they were housed in metabolic cages. After sacrificed kidney weights and Na^+ , K^+ ATPase activity of erythrocytes. Ghosts from erythrocytes were prepared by the method of Dodge et al. (ARCHS BIODHEM BIOPHS 100: 119-123). Na^+ , K^+ ATPase activity was measured according to the method previously reported (Mori et al.: JPN HEART J 24: 1983).

Results and Discussion

There was no inter-group difference in blood pressure and in body weight except for B group in which body weight was significantly increased after acute salt loading ($p < 0.05$). Na^+ excretion in 24-hour urine was significantly increased compared with C group, but there were no difference between A and B groups. Kidney weights were significantly increased in A group ($p < 0.05$) but not increased in B group. Na^+ , K^+ ATPase activity of erythrocytes was significantly reduced in B ($p < 0.01$) but not in A group.

In salt resistant rats, during acute sodium loading when sodium load might exceed normal renal function to excrete sodium, relative inadequacy in excreting sodium might lead to a compensatory rise in circulating Na^+ , K^+ ATPase inhibitor which enhances sodium excretion through the kidney to equilibrate sodium balance. Sustained excessive sodium intake induces functional hypertrophy of kidneys, which increases renal functional capacity to excrete sodium, and then circulating Na^+ , K^+ ATPase inhibitor returns to normal. Thus, in salt resistant rats, the role of Na^+ , K^+ ATPase inhibitor to excrete more sodium under salt loading might be only temporarily of functional importance in sodium excretion.