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Increased Renal Expression of ENaC and Na,K-ATPase in Rats with Puromycin Aminonucleoside-Induced Nephrotic Syndrome

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The present study was aimed to determine whether the regulation of renal sodium transporters is altered in nephrotic syndrome. Male Sprague-Dawley rats were used. The experimental group was treated with puromycin aminonucleoside (50 mg/kg, iv), while the control group was treated with vehicle. One week later, the protein expression of Na,K-ATPase, NHE3, BSC-1, TSC and ENaC were determined in the various segments of the kidney by Western blot analysis. The catalytic activity of Na,K-ATPase was also determined. The serum albumin level was significantly decreased in the experimental group. Accordingly, urinary sodium excretion was decreased, while urinary protein excretion increased. Plasma renin activity and aldosterone levels were not significantly altered. The expression of $\alpha 1$ subunit of Na,K-ATPase was increased in the cortex, outer medulla and inner medulla, while $\beta 1$ subunit increased in the outer medulla and inner medulla, not in the cortex. Accordingly, the catalytic activity of Na,K-ATPase was also increased in the cortex, outer medulla and inner medulla. The expression of α-subunit of ENaC was increased in the cortex, outer medulla and inner medulla. The expression of NHE3 was decreased in the cortex, while not changed in the outer medulla. The renal expression of BSC-1 and TSC-1 was not altered. An enhanced expression and activity of ENaC and Na,K-ATPase may in part account for the sodium retention in puromycin aminonucleoside-induced nephrotic syndrome.

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Increased expression of renal sodium transporters in rats with L-NAME induced chronic inhibition of nitri oxide synthesis

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The present study was aimed to determine whether endogenous nitric oxide (NO) plays a role in regulating the sodium transporters in the kidney. Male Sprague-Dawley rats were treated with N^G-nitro-L-arginine methyl ester (L-NAME, 100 mg/L drinking water) for 4 weeks. The group of rats supplied with tap water without drugs served as control. The protein expressions of Na,K-ATPase, NHE3, BSC-1, TSC and ENaC were determined in the kidney by Western blot analysis. The catalytic activity of Na,K-ATPase was also determined. Following the treatment with L-NAME, the systolic blood pressure measured on the day of experiment was significantly higher in the experimental group than in the control. Renal tissue levels of nitrite/nitrate were significantly decreased. The urinary excretion and fractional excretion of sodium were decreased, while creatinine clearance remained unaltered. The expression of $\alpha 1$ subunit of Na,K-ATPase and the catalytic activity of Na,K-ATPase were increased in the kidney. The expression of NHE3 was also increased. The expression of TSC was increased, whereas that of BSC-1 was not altered. The protein expression of α-subunit of ENaC was increased, while β- and γ-subunits of ENaC remain unaltered. These results suggest that endogenous NO system has a tonic inhibitory effect on the expression of Na,K-ATPase, NHE3, TSC, ENaC in the rat kidney.