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Changes of Renal Acid-Base Transporters in Response to Ammonium Chloride Loading in Rats

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The present study was aimed to determine whether there is an altered regulation of renal acid-base transporters following acute and chronic acid loading. Male Sprague-Dawley rats were used. Metabolic acidosis was induced by gavage feeding of ammonium chlroride (10 mEq/kg of body wt) in a volume of water equal to 2.5% of body weight for 2 days (acute) and 7 days (chronic). The serum and urinary pH and bicarbonate were measured. The protein expression of acid-base transporters (NHE3, Na-K+ ATPase, H+-ATPase, AE-1) was measured by immunoblotting. In acute stage, there were decreases in serum bicarbonate in acid-loading rats compared with the control (28±2.5 vs. 20±0.6, $p<0.05$) and pH (7.45±0.01 vs. 7.35±0.01, $p<0.05$). Accordingly, urinary pH decreased (7.4±0.5 vs. 6.5±0.3, $p<0.05$). The protein expression of NHE3, Na-K+ ATPase, H+-ATPase, and AE-1 was not changed. In chronic state, there were no changes in serum bicarbonate (22±2.3 vs. 22±3.2, p=NS) and pH (7.43±0.05 vs. 7.44±0.04, p=NS), while urinary pH was decreased (8.9±0.6 vs. 6.7±0.3, $p<0.05$) in acid-loading rats. The protein expression of NHE3 and H+-ATPase was increased in the renal cortex of chronic acid-loading rats. These results suggest that a failure of modifying the expression of acid transporters despite an increased acid-loading may contribute to the development of acidosis in the acute stage. The subsequent increased expression of NHE3 and H+-ATPase may then play a role to promote acid secretion in the later stage of acid-loading, counteracting the development of metabolic acidosis.

Key Words: 산증, 수송체, ammonium chloride
Acidosis, Transporter, Ammonium chloride