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METABOLIC ACIDOSIS INFLUENCES ON RENAL SODIUM HANDLING IN CADMIUM-INTOXICATED RATS

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It has been reported that antinatriuresis is induced by acute cadmium intoxication. However, the mechanisms related to the increase in renal sodium reabsorption by cadmium exposure is not clear yet although it has been suggested that the elevated aldosterone might involve in this process. Thus, we evaluated the changes in urinary electrolyte excretion in chronic metabolic acidosis (CMA)-induced and cadmium-exposed rats and their relations on the renal function since the activities of renal sodium-hydrogen exchanger (NHE) are stimulated by CMA.

Pathogen free male Sprague-Dawley rats were given free access to a standard diet and tap water. CMA was induced by drinking 0.28 M NH4Cl ad libitum for two weeks. Then cadmium intoxication was induced by subcutaneous injection with 2 mg Cd in saline/kg/day for 3-4 days. Control group was administrated with the same volume of saline (2 ml/kg/day). Renal function were analyzed with 24-hr urine and plasma collected at the end of experiments.

Cadmium reduced the fractional excretions of sodium and potassium without significant changes in urine pH as can be seen at previous studies. However, CMA itself significantly elevated the fractional excretions of sodium and potassium. These might be resulted from the inhibition of activity of renal proximal tubular reabsorption. Especially, the potassium fractional excretion was much greater than in control. Interestingly, cadmium adminstration to CMA reduced the fractional excretion of potassium, but elevated the sodium fractional excretion.

These results demonstrate that the processes of sodium and potassium reabsorption were stimulated by cadmium in either CMA or not. Nevertheless, the effectiveness of cadmium on the sodium and potassium reabsorption in CMA was less than in its control. This study suggests that the processes of renal sodium reabsorption stimulated by cadmium are inhibited by CMA and may not be related to the NHE. (This study was supported in part by Korea Science & Engineering Foundation, R02-2000-00113)

keyword : Cadmium, Metabolic acidosis, Renal function