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Effects of Cadmium on Renal Function and Renin-angiotensin-aldosterone system in NaCl-Loaded Rats

Yung Kyu Kim
Department of Physiology, College of Medicine, Dongguk University, Kyungju 780-714, Korea

Acute cadmium exposure has been shown to increase sodium reabsorption in kidney through increase in aldosterone secretion in human and rodents. However, the antinatriuresis is not completely explained by hyperaldosteronism. Moreover, it is still controversial that the increase in plasma aldosterone concentration is mediated by the renin-angiotensin-aldosterone system (RAAS). In this study, the effects of cadmium on the renal sodium handling and RAAS in salt-loaded rats were examined.

Pathogen free male Sprague-Dawley rats were given free access to a standard diet and tap water or 0.3 M NaCl *ad libitum*. After 7 days pre-administration with salts, the animals were subcutaneously injected with 2 mg Cd in saline/kg/day or the same volume of saline (2 ml/kg/day) for more 3 days with continuing salt-loading. 24-hr urine was collected in a metabolic cages and measured sodium, potassium, and creatinine excretions. Plasma aldosterone concentration and renin activity were analyzed with radioimmunoassy.

The creatinine clearance in salt-loaded animals was significantly elevated by cadmium exposure. The plasma aldosterone concentration in salt-loaded animals was decreased by 50% of control. Cadmium dramatically reduced the plasma renin acitivity by 13% in salt-loaded group. Under these conditions, the fractional excretions of sodium and potassium were significantly diminished by cadmium exposure in salted loaded animals as in tap water-supplied animals.

These results support that the hyperaldosteronism induced by cadmium is not mediated by RAAS and strongly suggest that the there is, at least in part, direct effects of cadmium on the renal antinatriuresis not through aldosteone mediation.