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싸이클로스포린신독성동물모델에서 PKCK2의 역할

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The Role of Protein Kinase CK2 (PKCK2) in the Cyclosporine Induced Nephropathy in Rats

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Background: Protein kinase CK2 (PKCK2) has multiple, overlapping roles in induction of apoptosis. Apoptosis might to be a common pathway of renal injury caused by nephrotoxic drug or injury. We evaluated the role of PKCK2 in the Cyclosporin A (CsA) induced nephropathy in rats by inhibition of PKCK2.

Methods: Male Sprague Dawley rats fed with low sodium diet were divided into four treatment groups: control (0.9% saline injection), CsA (15 mg/kg/day by s.c.), CsA+emodin (CsA plus emodin 20 mg/kg/day by s.c.), and emodin only. The expression levels of apoptosis-associated factors were examined by western blot analysis. Expression of PKCK2 was also assayed.

Results: Overexpression of PKCK2 was noted in CsA treatment. Emodin, low molecular weight PKCK2 specific inhibitor, prevented the up-regulation of phosphorylated p53 and the activation of caspase 3, 7, and 8 induced by CsA treatment. In addition, emodin prevented CsA-induced increased expression of Bax/Bcl-2 ratio. Emodin prevented the up-regulation of PKCK2 induced by CsA treatment, indicating that its apoptotic-preventing activity was mediated via PKCK2.

Conclusions: Our findings indicate that PKCK2 may play a major role in apoptotic injury in CsA induced nephropathy in rat models.

Key Words: 세포사멸, 싸이클로스포린, PKCK2
Apoptosis, Cyclosporine, PKCK2