

## Adriamycin 유도 신병증에서 A3 아데노신 수용체 차단이 신기능에 미치는 효과

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### Effects of Highly Selective Adenosine 3 Receptor Antagonist on Renal Function in Mice Model of Adriamycin Induced Nephropathy

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**Background:** Concentration of adenosine in normal kidney increases markedly during renal hypoxia and ischemia. Previous studies have reported that mice lacking renal A3 adenosine receptor (A3AR) show significant protection against acute kidney injury, such as ischemia-reperfusion injury and myoglobinuria-induced injury. A recent study reported that A3AR antagonist blocked the development and attenuated the progression of renal fibrosis. Adriamycin (ADR)-induced nephropathy model has developed podocyte injury, which resulted in severe proteinuria and progressive glomerulosclerosis. The aim of the this study is to investigate the mechanism and preventive effect of fibrosis in glomerular, tubular and interstitial tissue associated with highly selective adenosine 3 receptor antagonist (LJ1888) treatment in ADR-induced nephropathy

**Methods:** We designed three animal groups as following: 1) 6-week-old balb/c mice(control), 2) untreated with LJ1888 after injection of ADR (11 mg/kg), 3) treated with LJ1888 (10 mg/kg) for 2 weeks after 5 weeks of injection of ADR.

**Results:** Body weight was significantly decreased in both injection of ADR and treated with LJ1888 at 7 weeks. Proteinuria and micro-albuminuria were significantly increased after injection of ADR and significantly decreased in treated with LJ1888. Urinary excretion of nephrin was significantly increased at 7 weeks after injection of ADR and significantly decreased in treated with DA1229. Urine isoprostane and lipid peroxide, which reflect oxidative stress markers in kidney, were significantly increased after injection of ADR and significantly decreased in treated with LJ1888.

**Conclusion:** Our results suggest that renoprotective effects of LJ1888 in ADR-induced nephropathy may be associated with protective effect of podocyte injury. LJ1888 may be used as potential therapeutic agents in a variety of glomerular diseases inducing proteinuria.

**Key Words:** A3AR 억제제, Adriamycin 유도 신병증, 족세포  
A3AR antagonist, Adriamycin induced nephropathy, Podocyte