F-D3

Enhanced Activity of Natriuretic Peptide and Nitric Oxide Systems in the Kidney of Rats Inhibited of 11β HSD2

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Background : It has been known that an inhibition of type 2 11β-hydroxysteroid dehydrogenase (11β HSD2) results in renal sodium retention and hypertension due to inappropriate activation of mineralocorticoid receptors (MR) by glucocorticoids. Natriuretic peptides (NP) and nitric oxides (NO) have been implicated in the regulation of urinary sodium excretion. The present study was aimed to examine whether there is an altered regulation of NP and NO systems in the kidney following an inhibition of 11β HSD2.

Methods : Male Sprague-Dawley rats were treated with glycyrrhizic acid (3 g/L) for 3 weeks. Plasma renin activity (PRA) and serum aldosterone levels were determined by radioimmunoassay. The expression of NPs was determined by real-time polymerase chain reaction, and that of nitric oxide synthase (NOS) isozymes was determined by Western blot analysis. The activity of guanylyl cyclase was also determined by the amount of cGMP generated in response to atrial natriuretic peptide (ANP) or sodium nitroprusside (SNP).

Results : In the experimental group, systolic blood pressure measured by the tail-cuff method was significantly increased. PRA and serum aldosterone levels were decreased. The mRNA expression of 11β HSD2 was decreased while that of MR increased. The expression of ANP and C-type natriuretic peptide mRNA was increased in the kidney, although that of natriuretic peptide receptor-A and -C mRNA was not changed. The protein expression of NOS isozymes was increased. The cGMP production provoked by either ANP or SNP was not changed.

Conclusion : The enhanced activity of NP and NO systems may play a compensatory role in hypertension induced by inhibition of 11β HSD2.