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Increased Apical Targeting of ENaC Subunits in Rats with Inhibited Type 2 11β-hydroxysteroid Dehydrogenase by Glycyrrhizic Acid

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Background : The present study examined the hypothesis that an altered expression and/or apical membrane targeting of epithelial sodium channels (ENaC) may account for the increased sodium reabsorption and hypertension in rats treated with glycyrrhizic acid.

Methods : Adult male Wistar rats were used. They were supplemented with glycyrrhizic acid in the drinking water (3 g/L) for 8 days. Control rats were supplied with normal tap water. The protein expression of 11βHSD2, ENaC subunits and aquaporin-3 in the kidney was determined by immunoblotting and immunocytochemistry.

Results : The renal expression of αENaC was significantly increased in the experimental group compared with that in the control, while that of βENaC and γENaC was not significantly changed. Immunoperoxidase microscopy demonstrated increased targeting of αENaC, βENaC and γENaC in the late distal convoluted tubule (DCT2), connecting tubule, and cortical and medullary collecting duct segments. Immunolabelling of aquaporin-3 and Na,K-ATPase was increased in the collecting duct. However, the expression of 11β-hydroxysteroid dehydrogenase was not significantly changed in the cortex and outer medulla.

Conclusion : These results suggest that the glycyrrhizic acid-induced sodium retention and hypertension may be causally related with increased expression and increased apical targeting of ENaC in DCT2, connecting tubule and collecting ducts.