A7

Altered renal expression of aquaporin-2 water channels in rats treated with reserpine

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Background. The present study was aimed at exploring whether sympathetic nervous system plays a regulatory role in the expression of aquaporin (AQP) water channels in the kidney.

Methods. To deplete the endogenous sympathetic neural activity, rats were treated with reserpine (1 mg/kg, i.p.). One day thereafter, the abundance of AQP1-3 proteins was determined in different regions of the kidney. Adenylyl cyclase activity evoked by argine vasopressin (AVP) was also determined.

Results. Following the treatment with reserpine, systolic blood pressure was significantly decreased. The expression of AQP2 proteins was decreased in the cortex, outer medulla, and inner medulla. Neither the expression of AQP1 nor that of AQP3 was significantly altered. The adenylyl cyclase activity evoked by AVP was not altered following the treatment with reserpine.

Conclusions. These results suggest that the sympathetic nervous system has a tonic stimulatory effect on the expression of AQP2 water channels in the kidney.

A8

Increased renal expression of aquaporin water channels in rats chronically inhibited of nitric oxide synthesis

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Background. The present study was aimed at exploring whether endogenous nitric oxide has a regulatory role in the regulation of aquaporin (AQP) water channels in the kidney.

Methods. Sprague-Dawley rats were chronically inhibited of nitric oxide (NO) synthesis by the treatment with *N*G-nitro-L-arginine methyl ester (L-NAME, 100 mg/L drinking water) for 4 weeks. The abundance of AQP1-3 proteins was determined in different regions of the kidney. AQP2 targeting was assessed by comparing the magnitudes of its expression in the membrane-enriched fraction and in the cytoplasmic fraction using the differential centrifugation.

Results. Following the treatment with L-NAME, systolic blood pressure was significantly increased, while tissue cGMP levels in the kidney was decreased. The expression of AQP2 and AQP3 proteins was increased in the cortex, outer medulla, and inner medulla, being the former more prominent. However, the expression of AQP1 was not significantly altered. By cotreatment with hydralazine, the blood pressure was normalized, while the expression of AQP2 and AQP3 was persistently increased. The expression of AQP2 proteins in the membrane fraction increased in parallel with that in the cytoplasmic fraction in the inner medulla by the L-NAME treatment, suggesting a preserved targeting. The adenylyl cyclase activity evoked by AVP was significantly augmented in the inner medulla following the treatment with L-NAME. Supplementation with L-arginine during the whole period of L-NAME ingestion prevented the changes in adenylyl cyclase activity, and AQP2 and AQP3 expression.

Conclusions. These results suggest that the endogenous NO/cGMP system tonically inhibits the expression of AQP water channels that are regulated by AVP/cAMP pathway in the kidney.