

# Effect of streptozotocin-induced diabetes on kidney $\text{Na}^+/\text{K}^+$ -ATPase<sup>1</sup>

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The maximal capacity of low affinity ouabain binding sites in kidney medulla was found to be increased by  $20 \pm 3.8\%$  after 2 weeks, and by  $35 \pm 4.5\%$  in 4 weeks diabetes. However, in kidney cortex no similar changes could be detected. Western blot analysis of  $\text{Na}^+/\text{K}^+$ -ATPase subunits in kidney medulla indicated a significant enhancement of both the  $\alpha_1$  and  $\beta_1$  subunit in two and four weeks diabetic rats ( $\alpha_1$ :  $35 \pm 3.1$ ,  $51 \pm 5.8\%$  and  $\beta_1$ :  $31.3 \pm 5.2$  and  $43.2 \pm 6.8\%$ , respectively). However, kidney cortex showed no significant change in any condition tested. In diabetes we could detect a significant change only in the medulla in case of the b subunit mRNA transcript, which showed  $1.69 \pm 0.59$  and  $2.89 \pm 0.81$  times increased in two and four weeks diabetic state, respectively. There was no change in the  $\alpha_1$  subunit mRNA abundance. Insulin treatment of diabetic animals did not result in a complete reversal of diabetes-induced changes in ouabain binding capacity or in the amount of  $\text{Na}^+/\text{K}^+$ -ATPase  $\alpha_1$  and  $\beta_1$  subunit protein and mRNA levels. Our data indicate a good correlation between changes in low affinity ouabain binding capacity and the level of  $\alpha_1$  isoform in diabetic rats, and suggest an important role of the b subunit in the regulation of enzyme quantity.

**Keywords:** streptozotocin-induced diabetes,  $\text{Na}^+/\text{K}^+$ -ATPase,  $\alpha_1$  and  $\beta_1$  subunit, mRNA, kidney

Human and experimental diabetes is accompanied by an early, prominent and persistent renal hypertrophy [6, 22]. However, there are some evidence in experimental animal models of diabetes that early insulin treatment can prevent or reverse renal hypertrophy [15]. Nephromegaly has been associated with a renal hyperfunction (increased glomerular filtration rate of  $\text{Na}^+$  and water reabsorption,

<sup>1</sup>This paper is dedicated to the memory of Professor Tibor Kovács (1929-1994)

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