Acta Physiologica Hungarica, Volume 83 (4), pp. 323–332 (1995)

Effect of streptozotocin-induced diabetes on kidney Na^+/K^+-ATPase[1]

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Received April 10, 1995
Accepted May 30, 1995

The maximal capacity of low affinity ouabain binding sites in kidney medulla was found to be increased by 20 ± 3.8% after 2 weeks, and by 35 ± 4.5% in 4 weeks diabetes. However, in kidney cortex no similar changes could be detected. Western blot analysis of Na^+/K^+-ATPase subunits in kidney medulla indicated a significant enhancement of both the α_1 and β_1 subunit in two and four weeks diabetic rats (α_1: 35 ± 3.1, 51 ± 5.8% and β_1: 31.3 ± 5.2 and 43.2 ± 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 ± 0.59 and 2.89 ± 0.81 times increased in two and four weeks diabetic state, respectively. There was no change in the α_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 Na^+/K^+-ATPase α_1 and β_1 subunit protein and mRNA levels. Our data indicate a good correlation between changes in low affinity ouabain binding capacity and the level of α_1 isoform in diabetic rats, and suggest an important role of the b subunit in the regulation of enzyme quantity.

Keywords: streptozotocin-induced diabetes, Na^+/K^+-ATPase, α_1 and β_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 Na^+ and water reabsorption,

[1]This paper is dedicated to the memory of Professor Tibor Kovács (1929–1994)

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0231-424X / 95 / $5.00