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Effect of Epinephrine on Na+/glucose Cotransporter's Activity in Renal Proximal Tubule Cells

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Backgroud : Epinephrine has known to be a very important factor in the regulation of renal sodium excretion. However, the effect of epinephrine on Na+/glucose cotransporter was not fully elucidated. Thus, we examined effect of epinephrine on α-methyl-D-glucopyranoside (α-MG) uptake and its related signal pathways in the primary cultured rabbit renal proximal tubule cells (PTCs).

Methods : PTCs were grown in D-MEM/F-12 supplemented with insulin, transferrin and hydrocortisone. α-MG uptake, AA release, PGE2 assay, cAMP assay and western blotting were performed.

Results : Epinephrine inhibited α-MG uptake in a time- and dose-dependent manner and also decreased SGLT1 and SGLT2 protein level. Both phentolamine and propranolol completely prevented epinephrine-induced inhibition of α-MG uptake. The epinephrine-induced inhibition of α-MG uptake was blocked by SQ-22536 or myristoylated PKA inhibitor amide 14-22 and epinephrine increased the intracellular cAMP content. In western blotting analysis, epinephrine increases phosphorylation of p44/42 and p38 MAPKs and PD 98059 or SB 203580 blocked the effect of epinephrine. In addition, epinephrine increased AA release and PGE2 production and effects of epinephrine on α-MG uptake and AA release were blocked by staurosporine and bisindolylmaleimide I or mepacrine and AACOCF3. Indeed, epinephrine translocated PKC or cPLA2 from cytosol to membrane fraction.

Conclusion : Our data suggests that epinephrine partially inhibits the α-MG uptake through PKA, PKC, MAPKs, cPLA2 pathways in the PTCs.