This data article is concerned with chronic regulation of Na,K-ATPase by catecholamines. After a chronic treatment, inhibition of Na,K-ATPase activity was observed in cultures with dopamine, while a stimulation was observed in cultures treated with nor-epinephrine. Following a chronic incubation with guanabenz, an α adrenergic agonist, an increase in Na,K-ATPase α and β subunit mRNAs was observed. This data supports the research article entitled, “Renal proximal tubule Na, K-ATPase is controlled by CREB regulated transcriptional coactivators as well as salt inducible kinase 1” (Taub et al. 2015) [1].

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Experimental factors

Primary cultures of rabbit kidney proximal tubule cells treated with catecholamines and control

Experimental features

Rb\(^+\) uptake into intact cells was examined in triplicate, and standardized with respect to protein, to calculate nmoles of Rb\(^+\) uptake per mg protein; in Real-Time PCR, Ct values of Na,K-ATPase and GAPDH mRNAs were obtained from quadruplicate determinations, and used to calculate the relative increase in Na,K-ATPase in catecholamine treated and control cells.

Data source location

All analyses and experiments were performed in Buffalo, New York, USA

Value of the data

- This data will have an impact on therapies using catecholamines for blood pressure regulation.
- The data can be compared with other studies of transcriptional regulation of the genes encoding for each of these subunits.

1. Data

The data shown in this report measures changes both in Na,K-ATPase activity and Na,K-ATPase mRNA levels following a chronic incubation of renal proximal tubule cells with catecholamines.

2. Experimental design, materials and methods

2.1. Rubidium uptake studies

Primary cultures of rabbit kidney proximal tubule cells, were prepared as described previously [1,2]. Rabbits employed to obtain the primary cultures were used by procedures approved by the University at Buffalo Institutional Animal Care and Use Committee. The primary cultures were grown...

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Fig. 1. The effect of PGE\(_1\), norepinephrine and dopamine on transport. A. Primary RPT cells were incubated 30 min with either 280 nM PGE\(_1\), 1 \(\mu\)M norepinephrine or untreated (and +/- ouabain), followed by a 20 min uptake period with 1 mM \(^{86}\)Rb\(^+\). Uptake values are averages (+/- SEM) of ouabain-sensitive Rb\(^+\) uptake relative to the untreated control. The ouabain-sensitive component of Rb\(^+\) uptake was calculated by subtracting the Rb\(^+\) uptake observed in the presence of ouabain from total Rb\(^+\) uptake. The results were divided by the untreated control value. B. Primary RPT cells were incubated 30 min with either 10 \(\mu\)M dopamine +/- 5 \(\mu\)M monensin, or untreated (+/- 5 \(\mu\)M monensin). Uptake studies were conducted both in the presence and in the absence of 1 mM ouabain for each of the 4 conditions, followed by a 20 min uptake period with 1 mM \(^{86}\)Rb\(^+\). The ouabain-sensitive component of Rb\(^+\) uptake was determined as described in part A. *\(p<0.05\) relative to untreated Control.
in a 50:50 mixture of Dulbecco's Modified Eagle's medium and Ham's F12 (DME/F12) supplemented with 5 mg/ml bovine insulin, 5 mg/ml human transferrin and 5 \times 10^{-8} \text{M} \text{hydrocortisone (i.e. Medium RK-1)} [2]. ^{86}\text{Rb}^+ \text{uptake studies were conducted, as described previously, in K}^+ \text{-free DME/F12 supplemented with insulin, transferrin and indomethacin [3,4]. Agonists (PGE}_1, \text{norepinephrine, Fig. 1A, and dopamine, Fig. 1B) were added after a 30 min pre-incubation in this supplemented, K}^+ \text{-free DME/F12 (+/− 1 mM ouabain). Subsequently, agonists were added, followed by a 30 min incubation, the addition of }^{86}\text{Rb}^+ \text{(1 mM), and a 20 min uptake period. Uptake values were corrected for zero time uptake, and standardized with respect to protein. The ouabain-sensitive component of Rb}^+ \text{uptake was calculated, and divided by the values obtained with untreated controls (Fig. 1). Differences were considered significant if } p < 0.05 \text{ relative to the untreated control.}

2.2. Na,K-ATPase mRNA levels

Primary RPT cells were incubated for 4 days with either guanabenz (an \( \alpha \) adrenergic agonist), or isoproterenol (a \( \beta \) adrenergic agonist). Subsequently, RNA was purified, and cDNA was synthesized [4]. Specific cDNAs were amplified in a Bio-Rad iCycler. Ct values were calculated, and relative mRNA levels quantitated using GAPDH mRNA as an internal control. Primers designed using Primer-BLAST (NCBI), include primers for rabbit ATP1A1 (Assession number AF235024, ATP1A1_RABIT), TGACTCTCTGCTCTGAAG, CACAATGGAAGCGAAGTTATC; rabbit ATP1B1 (Assession number AF204927, ATP1B1_RABIT), ACTGGCAAGGAGATGGAAG, ATGGTGAGGTTGTTGAAACTG; and rabbit GAPDH (Assession number L23961, RABGLY3PHO), GCCCTCAATGACCACTTTGT, TCATGACAAGGTAGGGCTCC. Increases in the level of Na,K-ATPase \( \alpha \) and \( \beta \) subunit mRNA were observed in the presence of guanabenz, and were considered to be significantly elevated relative to the untreated control if \( p < 0.05 \) (Fig. 2).

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2015.12.013.

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