Dopamine Effect On Na⁺,K⁺-ATPase activity In The Presence of FXYD2

Cobal, S.A., Simão, D. C., Ferreira, L.G.R., Barbosa, L.A.O., Cortes, V. F.

1, 2, 3, 4, 5 - Campus Centro Oeste Dona Lindu, UFSJ

The FXYD2 is expressed only in kidney medulla associated with Na⁺,K⁺-ATPase. The dopamine is a hormone responsible for an increase in sodium excretion, that involves the Na⁺,K⁺-ATPase inhibition. This work demonstrate the effects of dopamine on Na⁺,K⁺-ATPase in the presence of FXYD2. We extracted FXYD2 from native Na⁺,K⁺-ATPase from pig kidney preparation as described in (Arch. Biochem. Biophys. 505; 75, 2011) and the results were statistically evaluated with the Student’s t-Test, employing Sigma-Plot 8.0 program. We showed that dopamine can decrease Na⁺,K⁺-ATPase activity about 20% and the addition of FXYD2 reversed this inhibitory effect, increasing the activity of the enzyme by 71%. The phosphorylation of Na⁺,K⁺-ATPase by endogenous PKA and PKC decreased this enzyme activity up to 90%. PKC and PKA inhibitors (chelerythrine and H-89, respectively) also protect the Na⁺,K⁺-ATPase from dopamine inhibition and we didn’t observe any additional protection when the FXYD2 was added. We also observed that He-La cells viability in presence of dopamine (1µM) is reduced in 20 %, however, when FXYD2 is added, the cells viability were restored up to 15%. In this work we showed that the FXYD2 seems to have an important participation on hormonal signal by dopamine, because this peptide can reverse the Na⁺,K⁺-ATPase inhibition by this hormone. We also conclude that the FXYD2 disturbs PKA and PKC signal induced by dopamine. Now we will study the influence of Dopamine and FXYD2 in Na,K-ATPase activity from human He-La Cells and compare with the results obtained with purified Na⁺,K⁺-ATPase from pig kidney medulla.

Words Keys: Dopamine, FXYD2, Na⁺,K⁺-ATPase

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