

Modulation of Ca²⁺-ATPase Activity on LLC-PK1 Cells: Angiotensin II Effect Via Luminal Membrane on SERCA and PMCA

Ferrão, F.M.¹, Axelband, F.¹, Lara, L.S.², Vieyra, A.¹, Lowe, J.¹

¹Instituto de Biofísica Carlos Chagas Filho, Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brazil; ²Instituto de Ciências Biomédicas, Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brazil.

Ang II is involved in maintaining the extracellular volume and blood pressure. A recent view is that Ang II is formed in the renal interstitial fluid and in proximal tubules at much higher concentrations than those existing in the vascular compartment. In these renal compartments, Ang II elicits Ca²⁺-dependent intracellular signalling that stimulates solute and fluid reabsorption. The aim of this work was to study the effect of Ang II on Ca²⁺-ATPase activity from a proximal tubule cell culture. The influence of luminal membrane on the two Ca²⁺ pumps, PMCA and SERCA, and the angiotensin receptors involved was also investigated. Ang II modulates the total Ca²⁺-ATPase in a biphasic manner when applied in the luminal side of LLC-PK1 cells for 30 min. This effect is mediated by the stimulation of SERCA activity but not of PMCA. Losartan and PD123319, AT₁ and AT₂ angiotensin receptors antagonists, revert the stimulation of SERCA activity. The time-course of Ang II effects indicate that there is a rapid (1 min) and persistent (up to 30 min) stimulus of SERCA. Altogether, the present data indicates that low concentrations of Ang II on luminal side of LLC-PK1 promotes the removal of intracellular Ca²⁺ by a sustained increase in SERCA activity, mediated by AT₁ and AT₂ receptors.

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