The Janus face of Ouabain in Na+, K+-ATPase and calcium signalling in neurons

Paula Kinoshita[1], Ana Orellana[2], Vinicius Nakao[1], Natacha de Souza Port´s[1], Luis Quintas[3], Elisa Kawamoto[1], and Cristoforo Scavone (GUEST EDITOR)[1]

[1]Universidade de São Paulo
[2]Universidade de Sao Paulo
[3]Universidade Federal do Rio de Janeiro

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Abstract

Na+,K+ATPase (NKA), a transmembrane protein essential for maintaining the electrochemical gradient across the plasma membrane, acts as a receptor for cardiotonic steroids (CTS) such as ouabain. CTS binding to NKA, triggers signalling pathways or inhibits NKA activity in a concentration-dependent manner, resulting in a modulation of Ca2+ levels, which are essential for homeostasis in neurons. However, most of the pharmacological strategies for avoiding neuronal death do not target NKA activity, due to its complexity and poor comprehension of the mechanisms involved in NKA modulation. The present review aims to discuss two points regarding the interplay between NKA and Ca2+ signalling in the brain: NKA impairment causing illness as well as neuronal death due to Ca2+ signalling and benefits to the brain by modulating NKA activity. These interactions play an essential role in neuronal cell fate determination and are relevant to finding new targets for the treatment of neurodegenerative diseases.

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