

Témakör: Nincs kiválasztott témakör

The role of protein kinase D activation in response to oxidative stress-induced neuronal cell death

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Protein kinase D (PKD), a serine/threonine kinase is highly expressed in the mammalian brain and fulfills many roles in cellular physiology, including the modulation of signaling pathways activated in response to cellular stress. According to studies on non-neuronal cells, reactive oxygen species originating from the mitochondria during oxidative stress activate PKD's downstream signaling. Our knowledge on the relation between oxidative stress and PKD-mediated changes in neurons, however, is still limited. During the present work we have shown that PKD undergoes a rapid and transient activation upon H₂O₂ treatment in mouse primary cortical neuronal cultures. Activation of PKD was verified by the increased autophosphorylation level of PKD as well as by PKD FRET reporters. Using MTT assays, we have demonstrated that treatment with PKD-specific inhibitors significantly attenuated cell death induced by 24 hours of oxidative stress. The neuroprotective effect of the inhibitors was the most profound when neurons were preincubated for 2 hours before the onset of the H₂O₂ treatment. This can be explained by the finding that PKD inhibitors activate NFκB, which might help neurons withstand oxidative stress. This work was supported by OTKA K81934 to K.S.

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