

عنوان مقاله:

Kalirin-γ plays the neuroprotective role in Neuro-2A cells injured by oxygen-glucose deprivation and reperfusion through Rac1 activation

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نویسندگان:

Jian Xu - Department of Neurosurgery, The Affiliated Hospital of Qingdao University, Qingdao ۲۶۶۵۰۰, China

Yanxiu Chen - Department of Neurology, Liaocheng People's Hospital, Liaocheng ۲۵۲۰۰۰, China

Zeyu Wu - Department of Neurosurgery, The Affiliated Hospital of Qingdao University, Qingdao ۲۶۶۵۰۰, China

Yihe Dou - Department of Neurosurgery, The Affiliated Hospital of Qingdao University, Qingdao ۲۶۶۵۰۰, China

Peng Lun - Department of Neurosurgery, The Affiliated Hospital of Qingdao University, Qingdao ۲۶۶۵۰۰, China

Peng Sun - Department of Neurosurgery, The Affiliated Hospital of Qingdao University, Qingdao ۲۶۶۵۰۰, China

خلاصه مقاله:

Objective(s): The study explored the neuroprotective role of Kalirin-γ (Kal-γ) in Neuro-2A cells after oxygen-glucose deprivation and reperfusion (OGD/R) treatment. **Materials and Methods:** The study used an OGD/R model of mouse Neuro-2A neuroblastoma cells in vitro. Cells were transfected with pCAGGS-Kal-γ to up-regulating kal-γ. Then cell proliferation and apoptosis were respectively analyzed by Trypan blue exclusion method and flow cytometry. To examine the involvement of Rac1, cells were treated with Rac1-GTP inhibitor NSC۲۳۷۶۶ before treatment with OGD/R. Expressions of Bax, Bcl-2, Rac1, and down-stream targets of Rac1 were analyzed by Western blot. **Results:** Kal-γ significantly decreased OGD/R induced cell apoptosis ($P<0.01$), but no significant effects were observed on cell proliferation. Kal-γ increased the expressions of apoptosis-related protein of Bcl-2 and Rac1, but decreased the expression of Bax in Neuro-2A cells stimulated to OGD/R. Rac1 was activated by Kal-γ due to the increased levels of its down-stream targets, p-p38 and p-PAK1. NSC۲۳۷۶۶ reduced the anti-apoptotic effect of Kal-γ as the enhanced apoptotic cell rate and increased Bax/Bcl-2 ratio. **Conclusion:** These findings suggest that the protective effects of Kal-γ against OGD/R injury in Neuro-2A cells were dependent in a Rac1 activation signaling.

کلمات کلیدی:

Anti-apoptosis, Kalirin-γ, Neuroprotection, OGD/R, Rac1

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