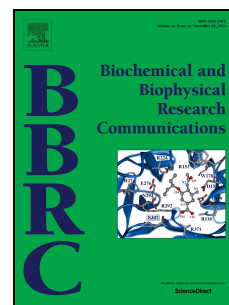


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AntagomiR-613 protects neuronal cells from oxygen glucose deprivation/re-oxygenation via increasing SphK2 expression

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Abstract. Oxygen glucose deprivation (OGD)/re-oxygenation (OGDR) causes damages to neuronal cells. Sphingosine kinase 2 (SphK2) expression could exert neuroprotective functions. Here, we aim to induce SphK2 expression via inhibiting the anti-SphK2 microRNA: microRNA-613 ("miR-613"). In both SH-SY5Y neuronal cells and primary murine hippocampal neurons, transfection of the miR-613's specific inhibitor, antagomiR-613 ("antamiR-613"), induced miR-613 depletion and SphK2 expression. Reversely, forced over-expression of miR-613 caused SphK2 downregulation in SH-SY5Y cells. OGDR-induced cytotoxicity in neuronal cells was largely attenuated by antamiR-613. SphK2 is required for antamiR-613-induced actions in neuronal cells. SphK2 knockdown (by targeted-shRNAs) or inhibition (by its inhibitor ABC294640) almost completely abolished antamiR-613-mediated neuroprotection against OGDR. Further studies showed that OGDR-induced reactive oxygen species (ROS) production, lipid peroxidation, and DNA damages in SH-SY5Y cells were largely attenuated by antamiR-613, but were intensified by miR-613 expression. Taken together, we conclude that antamiR-613 protects neuronal cells from OGDR probably via inducing SphK2 expression.

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