DENGUE VIRUS INFECTION MODULATES MITOCHONDRIAL HEXOKINASE ACTIVITY IN NEUROBLASTOMA CELLS.

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Dengue virus (DV) infection affects thousands of people around the world and it is considered the major human arbovirosis. Severe encephalitis has been reported in severe disease and signs and symptoms vary from headache and clouded sensorium to convulsion, spasticity and coma. In culture, DV-infected cells show many apoptotic features and oxidative stress and mitochondrial dysfunction maybe involved. It has been described that the activity of mitochondrial hexokinase controls the production of reactive oxygen species, suggesting its importance during the course of DV infection. In this study we evaluated the effects promoted by DV infection on the activities of the enzymes hexokinase and catalase in a mouse neuroblastoma cell model N2A. Cells were infected and enzymes activities were determined after 24 hours. The majority of both hexokinase and catalase activities were detected in mitochondrial fraction. DV-infected cells presented a 1.5-fold increase in mitochondrial hexokinase activity. Accordingly, the activity of mitochondrial catalase increased 2-fold in infected cells. The increase in catalase activity indicates that DV infection promotes an enhancement in oxidative stress in N2A cells. Indeed, the increase in mitochondrial hexokinase activity corroborates the role of this enzyme as a defense mechanism against oxidative stress and possibly its involvement in mitochondrial dysfunction during the apoptotic process in N2A infected with DV.
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