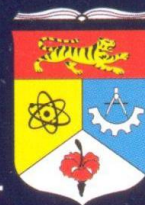


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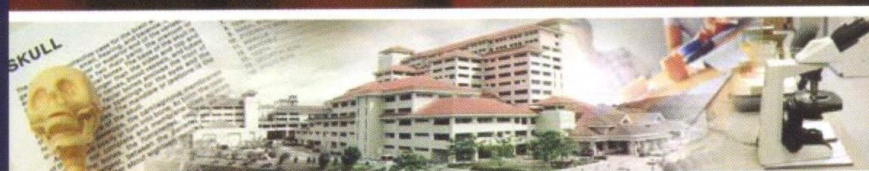
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SOD TRANSFECTED CELLS EXHIBIT CHANGES SIMILAR TO INCREASED OXIDATIVE STRESS CONDITIONS

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Background:

Superoxide dismutase (SOD) is an antioxidant enzyme which catalyses the conversion of oxygen free radicals to hydrogen peroxide. The hydrogen peroxide is further metabolised to water by the action of either catalase (Cat) or glutathione peroxidase (GPx). An imbalance between the enzymes, for example, the presence of an extra SOD gene in Down syndrome has been postulated as a possible cause for the neurodegeneration observed. In order to understand further the role of SOD in neurodegeneration, the present study aims to evaluate the characteristic of SOD-transfected versus normal neurons in terms of its oxidative DNA damage, apoptotic markers and cell cycle progression.

Material and Methods:

Neuron and SOD-transfected neurons (a gift from Dr Coral Sanfeliu, Institute of Biological Sciences, Barcelona, Spain) were incubated and the apoptosis rate determined using propidium iodide staining and Annexin V-FITC assay. DNA damage was assessed using comet assay. G0/G1, S and G2/M phases in cell cycle were evaluated from propidium iodide staining using flow cytometry. Caspase-8 and -9 activities were determined using Flowcytometric Caspases Activity kit and protein expressions (ATM kinase, ATR, p53, p73, Bax and Bcl2) of both cells were determined by Western Blotting.

Results:

Results showed that the presence of an extra copy of SOD gene increases apoptosis, DNA damage and the number of cells arrested in G0/G1 phase of the cell cycles which mirrors changes associated with increased oxidative stress conditions. There was upregulation of apoptotic pathway related proteins i.e ATM kinase, ATR, p53 and p73.

Conclusion:

These present findings indicate that overexpression of SOD increased oxidative stress related changes in cells which may lead to cell damage and death.

Keywords:

SOD, Neuron, oxidative stress, apoptosis