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Neuroprotective Role of Vitamin D in Primary Cortical Neuronal Cultures

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Abstract:

A role of vitamin D in brain development and function has been gaining support over the last decade. There are several lines of the evidence that suggest that vitamin D may have a neuroprotective role. The administration of vitamin D or its metabolites has been shown to reduce the neurological injury and or neurotoxicity in a variety of animal systems. Physiological concentration of calcidiol and calcitriol is between 30-50 nM in blood whereas the physiological concentration in brain is found to be 10 pM. This vitamin can cross the BBB and bind to the nuclear D3 receptors in the brain. Preliminary studies in our laboratory indicated that when isolated primary neuronal cells derived from rat's brain were pre-treated with vitamin D and then followed by the induced oxidative stress with hydrogen peroxide. It resulted in greater neuroprotection by up regulating the gene expression of glutathione. The research in our laboratory looked at various metabolic markers of oxidative stress and apoptosis, and found that pretreatment of neurons for up to six days with vitamin D3 can significantly reduce the lipid peroxidation as well as protecting genomic DNA oxidation by hydrogen peroxide. A number of criteria for looking at cell viability and proliferation in the presence of vitamin D3 treatments were employed and it again showed better protection of the neurons from induced oxidative stress. Suggesting its potential role as a neuro therapeutics agent. Our results highlight the importance of taking vitamin D3 supplements from an early age which could prevent the development of various neurodegenerative diseases.

Methods:

Primary neuronal cultures from cerebral cortex were set up from neonatal from 6 to 7 days old Wister Rat's brain. Different doses of [1,25(OH)2D3] that ranges from 0 to 1 µg/ml was added to the culture medium and the cells were cultured in its presence for upto 24 h to 120 h.

The effect of induced extracellular oxidative stress was measured by subjecting these cultured cells with 0.5 mM H_2O_2 for 2 h, prior to the collection of condition medium and the cell pellet for the biochemical assay. The control and H_2O_2 treated cultures were maintained in similar culture conditions for similar periods of time without any [1, 25(OH) 2D3] treatments.

Result:

The optimum concentration of the [1, 25(OH) 2D3] for treatment of primary cortical neuronal cultures was found to be 0.25 µg/ml by Trypan exclusion assay and MTT assay. Pre-treatments of cultured neuronal cells with 0.25 µg/ml of [1, 25(OH) 2D3] caused significantly increased levels of the reduced glutathione, accompanied by a similar increase in the enzyme levels of GST, to neutralize the induced oxidative stress by H_2O_2. The level of Lipid peroxidation was significantly higher in the cells treated with the H_2O_2 alone. But it was completely reversed in the neuronal cultures pre-treated with [1, 25(OH) 2D3]. The levels of Catalase enzyme also significantly reduced (≥ 0.05) in the [1, 25(OH) 2D3] pre-treated neuronal cultures.

Conclusion:

We concluded that the systemic treatment of the primary neuronal cultures with [1, 25(OH) 2D3] gave better protection to the neurons against the induced oxidative stress, as shown by quantitative measurements of various biomarkers of oxidative stress. This study also suggested that Vitamin D is vital for the growth, survival, and proliferation of the neurons and hence it has a potential therapeutic role against various neurodegenerative diseases.

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