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Magnesium elevation affects fate determination of primary cultured adult mouse neural progenitor cells via ERK/CREB activation

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Adult neurogenesis, which is the generation of functional neurons from neural precursors, occurs throughout life in restricted anatomical regions in mammals. Numerous studies have demonstrated a correlation between the level of hippocampal neurogenesis and cognition, whereas dysfunction of neurogenesis contributes to some pathological processes including Alzheimer's disease, Parkinson's disease, and other degenerative diseases. Magnesium is the fourth most abundant ion in mammals, and its elevation in the brain has been shown to enhance memory and synaptic plasticity *in vivo*. The substantial synapto-protective effects of magnesium elevation in the brain have also been demonstrated in a mouse model of Alzheimer's disease. However, the effects of magnesium on fate determination of aNPCs, which are vital processes in neurogenesis, remain unknown. NPCs isolated from the dentate gyrus of adult C57/BL6 mice were induced to differentiate in a medium with varying magnesium concentrations (0.6, 0.8, and 1.0 mM) and extracellular signal-regulated kinase (ERK) inhibitor PD0325901. The proportion of cells that differentiated into neurons and glial cells was evaluated using immunofluorescence. Quantitative real-time polymerase chain reaction and Western blot methods were used to determine the expression of β -III tubulin (Tuj1) and glial fibrillary acidic protein (GFAP). The activation of ERK and cAMP response element-binding protein (CREB) was examined by Western blot to reveal the underlying mechanism. Magnesium elevation increased the proportion of Tju1-positive cells and decreased the proportion of GFAP-positive cells. Also, the expression of Tuj1 was upregulated, whereas the expression of GFAP was downregulated. Moreover, magnesium elevation enhanced the activation of both ERK and CREB. Treatment with PD0325901 reversed these effects in a dose-dependent manner. This study showed that magnesium elevation effected fate determination of adult neural progenitor cells (aNPCs) and the possibly via ERK-induced CREB activation.

Biography

Jun Liu has completed his PhD and MD from Sun Yat-Sen University and Post-doctoral studies from Kansas University School of Medicine. He is a Doctorial Supervisor and serves as the Vice-Director of Department of Neurology, Sun Yat-Sen Memorial Hospital, Sun Yat-Sen University. He has published more than 40 papers in reputed journals (Biomaterials, JBC, CNS Neurosci.) and has been serving as an Editorial Board Member of BioMed Research International, World Journal of Neurology, J Nasopharyngeal Carcinoma, BMC Ophthalmology.

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