Poster Abstract Submission

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Research Title	In-Vitro Study of the Protective effect of Kuromanin Chloride Against Induced Excitotoxicity of Glutamate in Neuroblastoma Cells

Abstract:

Aim of the Study: Over-activation of the glutamatergic system can lead to glutamate toxicity and neurodegeneration. There isn't any available treatment to cure or neurodegenerative diseases or reverse the process so far. Neuroprotection broadly refers to preserve the structure and function of neurons from neuronal injuries and maybe achieved by several mechanisms such as free radical trapping, inhibition of apoptosis and preventing excitotoxicity. This study was designed to analyze the various cellular measures in response to anthocyanin (kuromanin chloride) in neuroblastoma cell line SH-SY5Y and its neuroprotective effects against glutamatergic excitotoxicity induced by L-glutamic acid monosodium (MSG). Methodology: SH-SY5Y cells were treated with various concentrations of kuromanin chloride (1µM, 10µM, 20µM, 30µM, 40µM, 50µM) for 24 hours prior to inducing excitotoxicity with 50mM of monosodium glutamic acid (MSG) for a period of 24, 48 and 72 hours. Cellular activities were assessed by MTT assay and lactate dehydrogenase assay, respectively. The expression of apoptotic genes BDNF, PPARGC1A, CASP3, CASP9, BRCA and BRCA1 and BRCA2 while GAPDH and Bcl-2 were used as endogenous loading control were measured by qRT-PCR. Further, morphology of cells treated with kuromanin chloride versus the cells exposed to glutamatergic excitotoxicity was also analyzed. Results: SH-SY5Y Cells viability increased with lower concentration of kuromanin chloride. Significant changes in cells toxicity were observed after 24 hours, while slight changes were observed after 48 and 72 hours. Neuronal survival gene BDNF and DNA damage response gene BRCA1 expression were significantly upregulated after treatment. Tumor suppressor genes BRAC1 and BRAC2 were highly expressed after treated with glutamate although significantly downregulated after treated with kromanin chloride. Apoptotic genes CASP3 and CASP9 expression were progressively downregulated after being treated with kromanin chloride. Conclusion: Our results indicate that kuromanin chloride can be a possible neuroprotective agent against neurodegeneration caused by excitotoxicity. This will pave the path for future studies to elucidate the precise molecular mechanism of neuroprotection exerted by kuromanin chloride. Furthermore, kuromanin chloride may be a putative neuro-protective agent that can be developed to a potential therapy to help protect neurons from neurodegeneration. Key Words: MSG, anthocyanin, LDH assay, MTT assay, qPCR, excitotoxicity, neuroprotection