World Academy of Science, Engineering and Technology
International Journal of Nutrition and Food Engineering
Vol:13, No:01, 2019

Attenuation of Amyloid beta (Aβ) (1-42)-Induced Neurotoxicity by Luteolin

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Abstract : Being a neurodegenerative disorder, Alzheimer's disease (AD) affects a majority of the elderly demented worldwide. The key risk factors for AD are age, metabolic syndrome, allele status of APOE gene, head injuries and lifestyle. The progressive nature of AD is characterized by symptoms of multiple cognitive deficits exacerbated over time, leading to death within a decade from clinical diagnosis. However, it is revealed that AD originates via a prodromal phase that spans from one to few decades before symptoms first manifest. The key pathological hallmarks of AD brains are deposition of amyloid beta (Aβ) plaques and neurofibrillary tangles (NFT). However, the yet unknown etiology of the disease fails to distinguish mitochondrial dysfunction between a cause or an outcome. The absence of early diagnosis tools and definite therapies for AD have permitted recruits of nutraceutical-based approaches aimed at reducing the risk of AD by modulating lifestyle or be used as preventive tools during AD prodromal state before widespread neurodegeneration begins. The objective of the present study was to investigate beneficial effects of luteolin, a plant-based flavone compound, against AD. The neuroprotective effects of luteolin on amyloid beta (Aβ) (1-42)-induced neurotoxicity was measured using cultured human neuroblastoma BE(2)-M17 cells. After exposure to 20μM Aβ (1-42) for 48 h, the neuroblastoma cells exhibited marked apoptotic death. Co-treatment of 20μM Aβ (1-42) with luteolin (0.5-5μM) significantly protected the cells against Aβ (1-42)-induced toxicity, as assessed by the MTS [3-(4,5-dimethylthiazol-2-yl)-5-(3-carboxymethoxyphenyl)-2(4sulfophenyl)-2H-tetrazolium, inner salt; MTS] reduction assay and the lactate dehydrogenase (LDH) cell death assay. The results suggest that luteolin prevents Aβ (1-42)-induced apoptotic neuronal death. However, further studies are underway to determine its protective mechanisms in AD including the activity against tau hyperphosphorylation and mitochondrial dysfunction.

Keywords : Aβ (1-42)-induced toxicity, Alzheimer's disease, luteolin, neuroblastoma cells
Conference Title : ICNFF 2019 : International Conference on Nutraceuticals and Functional Foods
Conference Location : Sydney, Australia
Conference Dates : January 30-31, 2019