Neuroprotective effect of Apigenin through inhibition of GSk-3β and amelioration of Aβ pathology in a rat model of Alzheimer’s disease

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Glycogen synthase kinase-3 (GSK-3) is a critical molecule in Alzheimer’s disease (AD) modulating two histopathological hallmarks of AD: Amyloid beta (Aβ) plaques and neurofibrillary tangles composed of aberrant hyper phosphorylation of tau protein. This study was performed to investigate the protective effect of apigenin, through inhibition of GSK-3 and the involvement of this kinase in BACE1 expression and hyper phosphorylation of tau protein in a rat AD model. 15 nM of aggregated amyloid beta 25-35 was microinjected into the left lateral ventricle. Apigenin (50 mg/kg) was administered orally 45 min before Aβ injection and continued daily for three weeks. Immunohistochemistry and western blot analysis showed that apigenin significantly reduced hyper phosphorylation of tau level in the hippocampus. Real time PCR analysis revealed a significant inhibition of mRNA level of β secretase (BACE1) and GSK-3β. Apigenin had no effect on the level of GSK-3α. The results demonstrate that apigenin has protective effects against amyloid beta 25-35 by decreasing the expression of GSK-3β with the consequence of lower the hyper phosphorylation of tau protein and suppression of BACE1 expression.

Results and Conclusions:

Biography
Farnz Nikbakht obtaining her PhD Degree in Human Physiology from Shiraz University. She received an award from the Iran Ministry of Health and Education and spent six months at Flinders University, Adelaide, Australia for completing her research on degenerative diseases. Currently she is working as the Associate Professor of Department of Physiology, in Iran University of Medical Sciences; she has managed several research programs and has conducted the thesis of several master’s and PhD students in her Lab. Since 2010 she has directed a research team on Epilepsy and Alzheimer’s diseases fields in her lab. Her research leads to publishing several articles.