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Asiatic acid protects differentiated PC12 cells from A β 25–35-induced apoptosis and tau hyperphosphorylation via regulating PI3K/Akt/GSK-3 β signaling

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Asiatic acid protects differentiated PC12 cells from A β ₂₅₋₃₅-induced apoptosis and tau hyperphosphorylation via regulating PI3K/Akt/GSK-3 β signaling

Running title: AA benefits A β ₂₅₋₃₅-treated differentiated PC12 cells

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Declaration of conflict of interest: None.

Abstract

Amyloid β (A β) peptide can cause neurotoxicity in Alzheimer's disease (AD). The main purpose of the present study is to investigate the protective role of asiatic acid (AA) against A β ₂₅₋₃₅-induced neurotoxicity in neuronally differentiated PC12 cells. Differentiated PC12 cells were pretreated with 5, 10 or 20 μ M AA before treatment with 20 μ M A β ₂₅₋₃₅. The viability and apoptosis of differentiated PC12 cells were determined by MTT assay and Annexin V-FITC/PI double staining, respectively. The mitochondrial membrane potential (MMP) of differentiated PC12 cells was analyzed by JC-1 staining. The expression levels of proteins were detected by western blot analysis. We found that AA significantly increased the viability of differentiated PC12 cells but attenuated the mitochondria-mediated apoptosis dose-dependently when challenging with A β ₂₅₋₃₅. Besides, the results of western blot analysis showed that AA

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