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Protective effect of a plants extract complex (SSB) against Amyloid β Protein (25-35)-induced neurotoxicity

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Amyloid β Protein (25-35)에 의해 유도된 신경독성에 대한 식물 추출물 복합제 (SSB)의 보호효과

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Objectives

Alzheimer's disease (AD) is a neurodegenerative disorder characterized by progressive loss of cognitive ability and by neuropathological features including senile plaque, neurofibrillary tangles and neuronal loss in selective brain regions. Amyloid β protein (Aβ) or Aβ peptide fragments have been suggested to play an important role in the pathogenesis of AD. Aβ-induced neurotoxicity is accompanied by increase of cytosolic Ca^{2+} concentration ($[Ca^{2+}]_c$) and generation of reactive oxygen species(ROS). In the present study, we investigated the protective effect of ethanol extract of plants extract complex (SSB) against Aβ (25-35)-induced neurotoxicity in cultured neurons and memory impairment in mice.

Materials and Methods

Materials

SSB (three plants extract complex including *Aralia Cordata*), Beta amyloid protein (Aβ) (25-35), SD rats, ICR mice

Methods

Neuronal cells, cultured from 16-day-old fetus of SD rats, were treated by Aβ (25-35). Viability of cultured neurons was measured by 3-[4,5-dimethylthiazole-2-yl]-2,5-diphenyl-tetrazolium bromide (MTT) assay and Hoechst 33342 staining. Aβ (25-35)-induced elevation of the $[Ca^{2+}]_c$ and generation of reactive oxygen species (ROS) were measured by fluorescence dyes using laser scanning confocal microscopy. Aβ (25-35)-induced memory impairment in mice was examined using passive avoidance test.

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Results and Discussion

SSB (1-30 μg/ml) inhibited Aβ (25-35)-induced elevation of $[Ca^{2+}]_c$, ROS generation and neuronal cell death. These results suggest that SSB may ameliorate Aβ (25-35)-induced neuronal cell death by interfering $[Ca^{2+}]_c$ increase and inhibiting ROS generation. Chronic administration of SSB (10-50 mg/kg, 8days) markedly improved memory impairment induced by intracerebralventricular injection of Aβ (25-35) in mice without affecting general motor function. In conclusion, the present study provides the pharmacological basis of SSB as a promising agent for the treatment of neurodegeneration in AD.

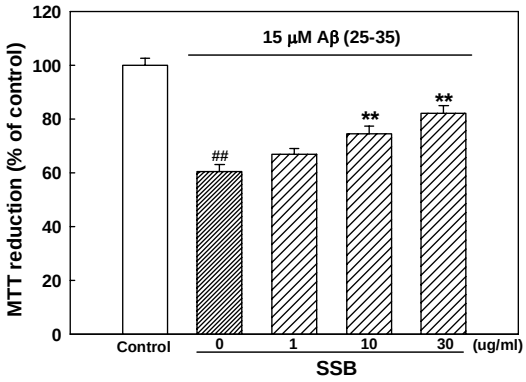

Fig 1. Protective effect of SSB against Aβ (25-35)-induced neuronal cell death measured by MTT assay.

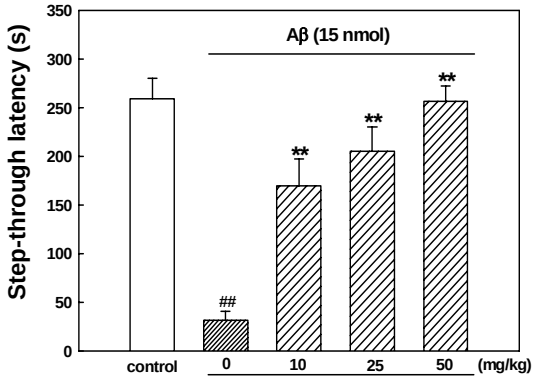

Fig 2. Protective effect of SSB against Aβ (25-35)-induced memory impairment in passive avoidance test.