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Neuroprotective effect of a plants extracts complex ACG

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식물추출물 복합제 ACG의 신경보호 효과

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Objectives

Alzheimer's disease (AD) is associated with brain shrinkage, and localized loss of neurons, mainly in the hippocampus and basal forebrain. Two microscopic features are characteristic of the disease, extracellular beta amyloid (Aβ) plaques and intraneuronal neurofibrillary tangles. *Aralia elata* Seem (AE; Arariaceae) has effect of analgesia, hypoglycemia, anti-inflammation and antioxidant activity. *Chaenomeles sinensis* (CS; Rosaceae) has been applied to the treatment of paralysis, torticollis and dyspepsia. Glycyrrhizae Radix (GR), the root of *glycyrrhiza uralensis* (Lycophodiaceae), has been used in Chinese medicine. The three ethanol extracts of the young shoots of AE, GR and fruits of CS were mixed in the same ratio and we named it ACG in the present study. The present study aims to investigate the neuroprotective effect of ACG using *in vitro* cultured neurons and *in vivo* experimental animals.

Materials and Methods

○ Materials

Ethanol extract of three plants complex ACG (AE, CS and GR), Aβ (25-35), SD rats, ICR mice.

○ Methods

Cerebral cortical neuronal cells were cultured from 16-day-old fetuses and cerebellar granular neuronal cells were cultured from 8-day-old rats. Cortical neurons were incubated with 10 μM of Aβ (25-35) for 36 h. Cerebellar granular neurons were incubated in hypoxic conditions (< 2% O_2, 5% CO_2, glucose free) for 24 h and further incubated in serum-free DMEM medium for 6 h. Cell viability was measured by 3-[4,5-dimethylthiazole-2-yl]-2,5-diphenyl-tetrazolium bromide (MTT) assay. Memory impairment was produced by intracerebroventricular (i.c.v) microinjection of 15 nmol Aß (25-35) and examined using passive avoidance test in ICR mice.

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Results

Both ACG (10 and 30 μg/ml) and every single extract, AE, CS and GR, of ACG inhibited 10 μM Aß (25-35)-and hypoxia-induced neuronal cell death. In *In vivo*, chronic treatments of ACG (7 days, p.o.) protected memory impairment induced by Aß (25-35). In conclusion, the present study suggests the role of this preparation as a promising therapeutic for neurodegenerative diseases like AD and stroke.

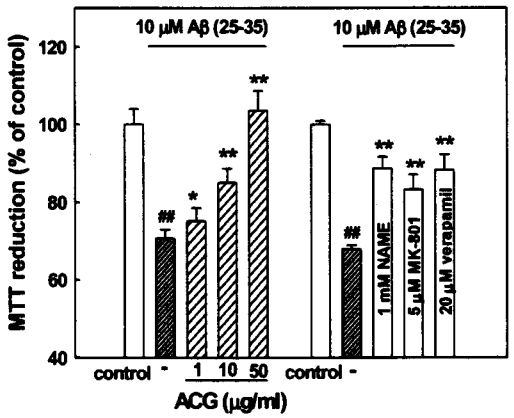

Fig. 1. Inhibitory effect of ACG on Aß (25-35)-induced cell death in cultured cortical neurons.

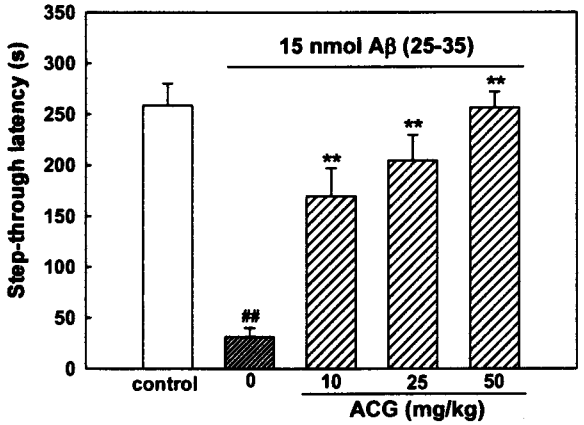

Fig. 2. Inhibitory Effect of ACG on 15 nmol Aß (25-35)-induced memory impairment in passive avoidance test.