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Development of combined therapy for Alzheimer's disease: A mechanistic study

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Alzheimer's disease (AD) is one of most devastating diseases affecting elderly people, Amyloid-β (Aβ) accumulation and the downstream pathological events such as Tau phosphorylation play critical roles in ADpathogenesis. Edaravone is marketed for acute ischemic stroke, has been proved for its capacity of inhibiting Aβ aggregation and attenuating Aβ-induced oxidation *in vitro*. According to MTT assay, EDA has shown strong protection effect against cytotoxicity induced by CuSO4 and H2O2 on SY5Y695 cells. In the neurite outgrowth assay, the cortex neuron isolated from C57 pups were treated with 1uM Aβ42 in the presence of different concentration of EDA, data shows the neurite length of EDA 3uM group increased to 30% to the control group and two folders high then the Aβ only group. The PI staining apoptosis assay also indicated that cells treated with EDA in differently concentration significantly reduced the death caused by CuSO4.In addition, P25/35 ratio is also changed in EDA treatment group, in the 3uM EDA group, P35 expression is significant increase while P25 decease 2 folders. Acetylcholinesterase (AChE) are enzymes that hydrolyze the neurotransmitter acetylcholine (ACh) to acetate and choline, the AChE is often found to be highly active in AD pathology, according the AchE activity assay, the pilot result shows suppression effect of EDA on AchE activity *in vitro*.

The ectodomain of p75 neurotrophin receptor (p75NTR-ECD) has been suggested to play important roles in regulating beta-amyloid (Aβ) deposition and in protecting neurons from the toxicity of soluble Aβ. Thus, we injected EDA and P75ECD as a combination to treat AMY mice (AD model animal), we expect to see this combination can alleviates Alzheimer's disease-type pathologies and cognitive deficits.

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