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Butanol extract of Ecklonia cava prevents production and aggregation of beta-amyloid, and reduces beta-amyloid mediated neuronal death.

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Abstract

Beta-amyloid (Aβ) is a major pathogenic peptide for Alzheimer's disease (AD) and is generated by the processing of amyloid precursor protein (APP). The Aβ monomers aggregate into oligomeric and fibrillar forms which have been implicated as the toxic species inducing the neuronal dysfunction. Brown algae Ecklonia cava is known for its anti-oxidant and anti-inflammatory functions. Therefore, we tested the effect of E. cava extract on the production and aggregation of Aβ peptides. The butanol extract of E. cava reduced Aβ secretion from HEK293 cells expressing APP with Swedish mutation and increased soluble APPα and C-terminal fragment-α (CTFα), of which activity was similar to BACE (β-site of APP cleaving enzyme) inhibitors. Furthermore, the extract inhibited Aβ oligomerization, particularly mid-size oligomer formation, confirmed by the ultrastructural morphology. Congo red, thioflavin T assays, and electron microscopy showed that the extract inhibited Aβ fibril formation effectively. Finally, the extract protected primary cortical neurons from various Aβ-induced cell deaths, especially oligomer-induced death. Although further study is needed to test the effectiveness of the extract in vivo, our results demonstrate, for the first time, that the butanol extract of E. cava could be used as an anti-Aβ agent for AD therapeutics.