Neuroprotective Potential of Coconut (Cocos nucifera) Leaf Extract using Transgenic Caenorhabditis elegans

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The discovery and development of treatments for Alzheimer's disease (AD) and Inclusion Body Myositis (IBM) are continuous worldwide demands. Virgin Coconut oil, derived from the coconut fruit, has been proven to alleviate AD pathology, focusing on in vitro antioxidant and acetylcholinesterase (AChE) inhibitory properties. Cocos nucifera leaves are considered abundant yet underutilized and lesser-valued part of the tree. This study was conducted to evaluate the neuroprotective activities of Coconut leaf extract (CLE) by determining its ability to reduce ß-amyloid aggregates and delay paralysis which serves as benchmark data in discovering a potential treatment for neuronal and muscular degenerative diseases. CLE was screened for antioxidant and anti-AChE activities by a regulated research institution (RRI) and neuroprotective effects in two strains of transgenic Caenorhabditis elegans expressing amyloid-beta1-42 (Aß1-42). Based in the preliminary toxicity test, CLE exhibited low percentage mortality in the test organisms ranging from 1.86-4.19%. Moreover, surprisingly, in spite of its sub-optimal antioxidant activity and AChE inhibition – which would predict increased Aß1-42 aggregation and paralysis – CLE reduced the amount of Aß deposits by 30.31% in a dose-independent manner, and paralysis by 84.02% of the transgenic C. elegans expressing Aß1-42 after exposure to the extract. Phytochemical analysis done by an RRI detected the presence of glycosides, flavonoids and hydrolyzable tannins in CLE which are known to target ß-amyloid proteins. These findings suggest that CLE reduces Aß1-42 aggregation and delay paralysis via a non-radical scavenging and AChE-independent pathway in vivo indicating that CLE could be a potential treatment for AD and IBM.

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