The Effects of Ginkgo biloba Extract EGb 761 and Trial Drug LY450139 Semagacestat on the Alzheimer's Beta Amyloid Protein: A Comparative Study for the Treatment of Beta Amyloid Plaques Using C. elegans as a Model Organism

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Alzheimer's disease (AD) is a neurodegenerative disorder classified by the loss of neuronal tissue in key areas of the brain and by the accumulation of beta-amyloid plaques in toxic oligomers. This study focuses on preventing the accumulation of these proteins by targeting the amyloid cascade hypothesis. It was hypothesized that Ginkgo biloba extract EGb 761 would eliminate or reduce that amount of beta-amyloid oligomers. It was further hypothesized that LY450139 would also reduce or eliminate the amount of beta-amyloid oligomers. Transgenic Caenorhabditis elegans were used as a model organism. C. elegans were treated with either Ginkgo biloba leaf extract EGb 761 or gamma secretase inhibitor LY450139 (Semagacestat) and stained with congo red for analysis. Fluorescence microscopy showed that in transgenic C. elegans, ginkgo biloba extract EGb 761 reduced the amount of beta-amyloid oligomers by 64.13% and LY450139 increased the amount of beta-amyloid oligomers by 1919.64% in transgenic worms but both treatments increased the amount of beta-amyloid oligomers in wild type worms by 26.70% under Ginkgo biloba EGb 761 and by 872.84% under LY450139. These results suggest that Ginkgo biloba could be a potential treatment for beta-amyloid oligomers.

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