Near Infrared Light Photobiomodulation and C. longa Mitigates the Expression of Mutant Amyloid-Beta Precursor Protein Pathway in D. melanogaster

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This study examined the effects of a treatment protocol for Alzheimer's Disease (AD) consisting of near infrared light (NIR) and C. longa. Mutated D. melanogaster, engineered to express the myc-tagged C99 fragment of A-BETA precursor protein (APP) and human tau, were subjected to NIR and C. longa separately (mono treatments) and in combined form (combined treatment) alongside non-mutated controls. To investigate the effect of AD progression on treatment efficacy, two groups were created: (1) mutated D. melanogaster treated 2 ± 1 days and (2) mutated D. melanogaster treated 5 ± 1 days post disease onset. Mutated D. melanogaster were exposed to NIR one hour a day for 12 days, while receiving C. longa as part of their daily diet. A driving mechanism of AD pathology, the interaction between mutated GAMMA-secretase and the C99 fragment of APP was evaluated using western blot analysis. Results from the assays evidenced the highest concentration of disaggregated C99 fragments in mutated D. melanogaster exposed to the combined treatment. This suggests the combined treatment was a more effective inhibitor of mutant γ-secretase and, consequently, led to fewer A-BETA aggregates and plaques, than the mono treatments. AD symptomatology was assessed by investigating flight patterns/behavior (i.e. mean speed, displacement, and angular velocity) of mutated D. melanogaster and analyzed using CTRAX. Results showed a significant ($p < 0.05$) decrease in symptoms in the combined treatment protocol when compared to either mono treatments. Findings also showed the combined treatment to be more efficacious when used at earlier disease onset than a later stage. This study warrants further investigation into combined treatment protocols to target the pathological mechanisms of AD and slow down progression.