

The Effect of Copper (II) Sulfate Pentahydrate Concentration on Locomotor Ability and Lifespan of Transgenic Alzheimer's Model *Drosophila melanogaster*

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Alzheimer's Disease (AD) is a progressive neurodegenerative disease believed to be caused by the abnormal breakdown of proteins creating toxic A β plaques. Copper is naturally occurring in the brain and is required for maintaining the health of neurons. Two conflicting theories on copper interaction with AD exist: 1. A β plaques absorb Cu $^{2+}$ ions, creating a Cu $^{2+}$ deficiency in neurons, decreasing neuronal function 2. Excess Cu $^{2+}$ in the brain binds to A β , increasing aggregation, causing an increase in neurodegeneration. This study researched the effects of Cu $^{2+}$ concentration on transgenic *Drosophila melanogaster* using a GAL4-UAS system expressing the human "Arctic" A β 42 mutation to determine if supplementation could decrease AD-associated effects. A dose-response study was conducted by adding 0, 1, 10, 100, 500, and 1000 μ M concentrations of CuSO $_4$.5H $_2$ O to Instant *Drosophila* Medium Blue. AD has progressive deterioration of movement coordination over time so a negative geotaxis assay was conducted on days 7, 8, and 9 of life to measure neurological function. Probability of survival was documented using Kaplan-Meier curves. Results showed that as Cu $^{2+}$ concentration increased, locomotor ability of Arctic flies increased significantly ($p < .05$), while that of wildtype and Gal4 controls generally decreased. Arctic flies had normalization of lifespan with no significant difference in lifespan existing between 10 μ M Arctic and 0 μ M wildtype flies ($p > .05$). These results imply that Cu $^{2+}$ supplementation had an ameliorative effect on neurological function and lifespan of transgenic *Drosophila melanogaster* suggesting that Cu $^{2+}$ supplementation may have an improving effect in AD patients.