The Effect of Alpha-tocopherol on Locomotor Ability of the Drosophila melanogaster Mutant for Glutathione S-transferase Theta 3

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Over 200,000 children in the United States live with Juvenile Arthritis (JA), an autoimmune condition that causes chronic inflammation as well as muscle and tissue atrophy. The human GSTT1 (hGSTT1) gene, part of the Glutathione S-Transferase Theta series, has been linked to this muscle erosion causing locomotor deficiencies. Since current JA treatments can be neurotoxic, it is necessary to identify safer and less toxic treatments. alpha-tocopherol (Vitamin E) is an antioxidant known to protect and repair muscle tissue. This study examined whether alpha-tocopherol supplementation could rescue the locomotor deficiency associated with flies expressing UAS-RNAi against GstT3, the Drosophila ortholog of hGSTT1, and whether there is functional conservation between the human and fly gene by conducting a genetic cross to rescue the motor delay by overexpressing human GSTT1. Flies knocked down for GstT3 in muscle tissue were raised on 0, 20, 200, 1,000, and 2,000μM alpha-tocopherol diets and tested using a negative geotaxis assay. Performance of the knockdowns increased significantly as the alpha-tocopherol increased ($p<.0001$), but plateaued between 1,000 and 2,000μM, suggesting a saturation effect. The overexpressed hGSTT1 flies performed significantly better than knockdowns and indistinguishably from wild type at all alpha-tocopherol concentrations ($p>.05$), demonstrating functional conservation between hGSTT1 and GstT3. Since functional conservation was established, future research should assay muscle breakdown in alpha-tocopherol treated versus untreated flies and investigate how GstT3 interacts with immune pathways to better treat JA.

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