Assessing the Effect of Atg1 Overexpression on Autophagy and Neurodegeneration in a Drosophila Parkinson's Disease Model Using qPCR

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Parkinson's disease (PD) is a neurodegenerative disorder characterized by the accumulation of misfolded a-synuclein proteins due to impaired autophagy. Enhancing autophagy represents a potential therapeutic strategy to reduce toxic protein aggregates. This study investigated whether overexpression of the Atg1 gene could restore autophagy and improve locomotor function in a Drosophila melanogaster PD model. Three groups were tested: wild-type controls, F1 flies expressing human a-synuclein (PD model), and F2 flies with Atg1 overexpression (PD + Atg1). Locomotion was assessed using a negative geotaxis assay with 15 flies per vial and five trials per group. Wild-type flies averaged 12 flies reaching an 8 mm line in 10 seconds, compared to 6 in the PD model and 10 in the PD + Atg1 group. One-way ANOVA revealed significant differences among groups ($F = 46$, $p < 0.01$). Tukey post hoc analysis showed the PD model was significantly impaired relative to wild-type and PD + Atg1 flies ($p < 0.01$). RNA was extracted using the RNeasy Qiagen kit, verified for purity (A260/280 ratios 1.98–2.11), and reverse transcribed to cDNA using a Bio-Rad reverse transcription kit. The cDNA was quantified through PCR using Atg1 and housekeeping gene Rpl32 primers. Gel electrophoresis was performed, and band fluorescence intensity indicated that Atg1 overexpression restored autophagy-related activity. qPCR was performed twice; however, CT values were not successfully obtained. These results suggest Atg1 overexpression majorly restores motor performance in a PD model, supporting its role in enhancing autophagy and reducing PD-related motor deficits, though quantitative data were not obtained and semi-quantitative analysis yielded consistent results indicating autophagy restoration by Atg1.

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