**Abstract**

Alzheimer’s disease is a progressive neurodegenerative disorder that affects cognitive function and memory of the patient. It results from plaques formed by the abnormal cleavage of the Amyloid Precursor Protein (APP). Accumulation of this peptide triggers cell death in the neuronal cell population of central nervous system. The role of mitochondrial dysfunction as the trigger for neurodegeneration is investigated, as its malfunction or loss has been linked to loss of dendritic branches and alteration of dendritic spines. The *Drosophila* melanogaster eye serves as a model of Alzheimer’s disease, and the Gal4/UAS system is utilized to misexpress the gene in the photoreceptor neurons where high levels of the human Alzheimer’s gene are expressed.

**Drosophila as a Model: The Gal4/UAS Technique**

**Neurology Behind Alzheimer’s Disease**

**Drosophila Model: Expression of Aβ42 by GMR/GAL4**

**Drosophila Melanogaster Life Cycle**

**Role of Mitochondria in Neurodegeneration**

- Mitochondria assume central functions in the cell, including ATP production, calcium homeostasis, reactive oxygen species generation, and apoptotic signaling. Mitochondrial dysfunction, abnormal mitochondrial dynamics and degradation by mitophagy occur during the disease process, contributing to the onset and progression of Alzheimer’s disease.
- In AD, alterations in enzymes involved in oxidative phosphorylation, oxidative damage, and mitochondrial binding of Aβ and amyloid precursor protein have been reported.

**Conclusion**

The upregulation of the mitochondrial ribosomal protein S31 allowed for successful rescue of the Aβ42-mediated neurodegenerative phenotype. This promising data pushes the aim of the project to identify other components of mitochondrial function that are affected by the expression of the Aβ42 gene.