

Using *Drosophila melanogaster* to Study Alzheimer's-Related Amyloid Proteins

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Amyloid proteins are the key proteins in Alzheimer's disease. In humans, elevated levels of Amyloid Precursor Proteins (APP) can lead to a buildup of Amyloid-B plaques, which ultimately reduces neural transduction efficiency. While APP is human specific, *Drosophila* has an analogue protein, Amyloid Precursor Protein-Like (APPL), which functions identically. Additionally, neural activity in both *Drosophila* and humans is regulated by the NOTCH cell-signaling pathway. In a past study, certain chromatin-regulating sequences, namely Tip60, have been shown to alter the NOTCH Pathway and cause undifferentiated cells and degraded egg chambers in *Drosophila*. The present study examined the effect of the Tip60 gene on Amyloid protein levels in adult *Drosophila Melanogaster* neural tissue. Specifically, flies containing an actin marker were crossed with either flies containing a Tip60 RNAi sequence or wild-type flies. For analysis, *Drosophila* was examined morphologically for size differences and its neural tissue analyzed with DAPI staining to observe nuclei. In flies expressing Tip60 RNAi, male flies were significantly ($p < 0.05$) smaller than Wild-Type flies. In neural tissue, *Drosophila* expressing RNAi had large amounts of Amyloid-B plaques in neural tissue in the DAPI stain. In Wild-Type flies, there was little to no Amyloid-B plaques in neural tissues. Additionally, the nuclei in the Wild-Type flies were larger and more defined than those in the specimens expressing Tip60 RNAi. Thus, Tip60 does have an effect on Amyloid protein activity in *Drosophila Melanogaster*.