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.

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