Translational Regulation of *Drosophila* Notch

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ABSTRACT

The Notch protein is a highly conserved and fundamentally crucial part of higher eukaryotes. It has a strong hand in dictating signaling, cell-fate, and development. As well, Notch has been implicated in the pathogenesis of Alzheimer's and other neurogenic defects. In *Drosophila,* fully translated Notch is a 300 kDa transmembrane receptor protein which is comprised of several domains. In neurons of *Drosophila*, however, a truncated form of Notch is found. This Notch isoform, which is missing the intracellular C-terminus, does not show the ability for signaling. It is currently unknown what Notch's functions in neurons are, despite evidence pointing toward its continued importance. Research suggests that this truncation in Notch is not due to the normal means by which proteins are modified in eukaryotes, such as alternative splicing or protein cleavage. Evidence of a stem-loop structure within the Notch gene may indicate that a frameshift of the RNA produces an alternative stop site, causing the truncated product to form. The research contained herein attempts to elucidate how Notch is translationally processed. Several sets of vectors which contain various mutations in the frameshift site have been constructed. A leader sequence and tandem epitope tags were incorporated into the cloned vectors as well. Lastly, these vectors will be transfected into *Drosophila* S2 cells and differential protein synthesis will be observed by means of western blot assays. A better understanding of Notch's translational regulation may lend to a more complete understanding of its function in the neurons of *Drosophila*.