Semaphorin1 may play a causative role in compensatory regrowth of the cricket auditory system following injury
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Nervous system development and repair are thought to depend on various factors, both extrinsic and intrinsic. Understanding the basic mechanisms that influence post-embryonic plasticity could contribute to our understanding of how nervous system plasticity aids in compensatory recovery following injury.

The auditory system of the cricket Gryllus bimaculatus provides a model in which to study postembryonic plasticity, specifically in dendrites. In many species, deafferentation, or the loss of electrical input, can cause dendritic retraction or cell death of the post-synaptic cells. The cricket auditory system has the unusual ability to compensate for this loss of input, forming new, functional synapses. Following unilateral deafferentation of the auditory interneurons in the cricket, post-synaptic dendrites grow across the midline of the prothoracic ganglion, a border they observe in normal development. This summer, my project attempted to assess the expression levels of the proteins involved in this process of compensatory recovery.

Several guidance molecules have been proposed to play a role in nervous system recovery following injury. Increased levels of semaphorin2a in the prothoracic ganglion have been shown to correlate with the compensatory regrowth observed in the cricket auditory system. Additionally, sema2 might play a more complex role in conjunction with a similar protein—semaphorin1. Recent evidence suggests that sema1 and sema2 might act synergistically, as receptors that modulate each other’s effects. We predict that the differential levels of these two proteins relative to each other might be responsible for modulating the guidance of dendrites in the observed compensatory regrowth.

Using Real Time quantitative PCR (RT-qPCR) we measured changing levels of semaphorin1 in the days following deafferentation. Deafferentation occurred within 1-2 days of the crickets’ final molt into adulthood and tissue samples were collected at various time points in the days following injury. Gene-specific primers (GSPs) targeted sema1 and β-actin (a housekeeping gene) in cDNA prepared from total RNA of the prothoracic ganglion. Previous anatomical characterization of this process has suggested the presence of a sexual dimorphism in this recovery process wherein females experience more immediate compensatory growth that soon tapers off while males show more gradual growth over time. Relative mRNA levels of Sema1 were significantly upregulated at 18 hours post-deafferentation in both males and females but maintained at this high level only in females. Two-way ANOVA revealed significant difference in means based on both sex and deafferentation, suggesting a role for Sema1 in the sex-differential compensatory recovery of the cricket auditory system. Following the aforementioned quantification of Sema1 mRNA levels, we hope to complete analysis that will address the synergistic interaction between Sema1 and Sema2a in the adult cricket. We aim to perform immunohistochemistry experiments in order to localize Sema1 and Sema2a proteins within the prothoracic ganglion. These experiments will inform mechanistic hypotheses surrounding compensatory recovery in the cricket CNS.

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