The Role of Semaphorins in the Compensatory Dendritic Growth of *Gryllus bimaculatus*

Monique Lillis, Class of 2017

In most animals, including humans, the central nervous system (CNS) is able to grow and change during development, but once the animal reaches adulthood the nervous system loses this ability. Therefore, if the CNS of most animals is injured, the neurons cannot regrow in order to recover from the damage. *Gryllus bimaculatus* are one of the few species that has a CNS that can adapt to an injury. As shown in the figure below, in a normal cricket the auditory system is set up such that information from the ear is sent by Nerve-5 to auditory interneuron-2 (AN-2) and then up to the brain. When nerve-5 is removed, a process known as deafferentation, the dendrites of AN-2 grow across the midline and form new functional synapses with the contra-lateral nerve-5.

The Horch lab believes that proteins from the semaphorin family are involved in the midline crossing and dendritic branching seen post-deafferentation. These proteins are involved in the development of the central nervous system, and we believe that after the high stress of losing auditory input, the semaphorins change how they are expressed in order to allow the dendrites of AN-2 to change their structure. Previous research supported this showing that Sema1a is downregulated by 18 hours post deafferentation and Sema2a is downregulated by 5 days post deafferentation.

I want to understand if Sema1a, Sema2a or both are sufficient to cause the changes seen in the dendritic arbor of AN-2. Therefore, this summer I targeted Sema1a to see if the downregulation of it alone could induce the modifications. I first synthesized double-stranded RNA (dsRNA) against Sema1a, and injected it into 7th instar crickets. Once inside the body dsRNA in conjunction with other proteins is used to break down the mRNA for Sema1a, thereby preventing the translation of the mRNA into the Sema1a protein. After injections I waited until crickets became adults and then backfilled AN-2 and nerve-5 with dye so that I could look at their structures under the confocal microscope. Thus far the images that have been visualized had nerve-5 backfilled but not AN-2, which could be an issue with how the tissue was prepared. I made changes to our preparation and will visualize more in the near future. If Sema1a is able to cause morphological changes in the AN-2 dendritic arbor, then I expect I would see dendrites crossing the midline where they usually do not. If Sema1a does not have a causative affect then I expect to see the control structure of AN-2.

In the future, I will use dsRNA to knockdown Sem2a and visualize any differences, and then use it to knockdown both Sema1 and Sem2a at the same time in order to assess if they work in conjunction to cause the modifications of the AN-2 dendritic arbor. These experiments are aimed to help create an understanding of how neurons can grow and change in adult systems. Semaphorins are particularly interesting to study because they are well conserved through species and are not only present in invertebrates but also vertebrates and viruses. Therefore, if semaphorins are involved in the compensatory dendritic growth of crickets, it will help us understand the role they play in humans.

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