Central auditory interneurons in the cricket prothoracic ganglion exhibit an unusual compensatory response to injury. While the dendrites of the auditory neuron AN2 normally respect the midline, in response to deafferentation, AN2 sprouts new dendrites which cross the midline to form connections with contralateral sensory afferents. The factors driving this compensatory response remain unknown. It is unclear as to whether it is the loss of physiological activity from or the loss of physical connection with sensory neurons that drives AN2's formation of new synaptic contacts. The ultimate goal of this project is to determine whether sensory activity deprivation is sufficient to induce AN2's regenerative response. This goal can be achieved by blocking the cricket ear with wax and physiologically screening for the formation of contralateral connections. Previous studies have confirmed the existence of deafferented AN2's compensatory contralateral connections by demonstrating that deafferented AN2 responds to contralateral acoustic stimulation. This test for regeneration relies on the assumption that AN2 in the intact animal lacks a response to contralateral stimulation. My research sought to test the validity of the latter assumption.

The responses of the cervical connective, which contains AN2's ascending axon, to contralateral acoustic stimulation were recorded in the intact animal. Contralateral stimulation elicited action potentials of comparable amplitude to AN2's ipsilaterally elicited spikes, raising the possibility that the contralaterally responsive cell is AN2. To shed light on the identity of the contralaterally responsive cell, I have previously explored its physiological characteristics, revealing that it shares certain response traits with AN2. The identity of the contralaterally responsive cell, however, remains unknown. We hope to ultimately identify the cellular origin of the contralateral response via morphological analysis. My research this summer thus focused on perfecting the technique of intracellularly injecting fluorescent dye into auditory neurons and visualizing via confocal microscopy. Future studies may use this technique to visualize the morphology of the contralaterally responsive cell and to determine whether it is AN2. This determination will clarify whether intact AN2 exhibits a response to contralateral stimulation or whether AN2's contralateral response only arises following deafferentation. The latter result would confirm the assumption of previous studies that AN2's contralateral responsiveness is a valid test for regeneration. Identifying a valid indicator of regeneration is essential to the investigation of our primary research question regarding whether activity deprivation is sufficient to induce regeneration.

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