The role of semaphorin signaling in the cricket's auditory plasticity

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The role of semaphorin signaling between Sema1a.2 and Sema2a in the auditory plasticity of was examined. Double-stranded RNA (dsRNA) was injected and used to knockdown semaphorin 1a.2 and semaphorin 2 in 7th instar crickets. Physiology recordings quantified responses to auditory stimuli and backfill visualized morphology. Most of the summer focused on troubleshooting the visualization process. Analysis of physiology recordings indicated differences between un-injected and GFP-] v i] l [ ]as v and } duration of response under specific conditions. Future research will utilize these findings to determine if Z } } ( ] v i] } v o Z] l [} v] À v v µv v Z }] o } o u Z }] v] P vaudtory P u · o · plasticity.

Crickets exhibit an unusual compensatory plasticity in response to injury and are easy subjects to study. When cricket ears are removed via amputation, ascending neurons in the prothoracic ganglion (PTG), part of the central auditory system, sprout across the midline, a boundary they typically respect. The neural pathways reroute and connect to the connections originating in the opposite ear, limiting the disadvantages caused by trauma by re-establishing some ability to localize sound (Scholes, 2020). Proteins involved in this phenomenon have only recently been characterized in cricket. In previous studies, Semaphorins have been shown to guide the development of axons and dendrites which v u] o]] P v o ` · (} u Z v µ } v [o o } s from v other] À o] neurons. In developing grasshoppers, a closely related insect, this guidance is achieved by Semaphorin signaling through Plexin receptors. In the cricket, the Horch lab has characterized two distinct Semaphorin1 proteins, known as Sema1a.1 and Sema1a.2 (Horch et al., 2020). Sema1a.2 messenger RNA (mRNA) is expressed in adult thoracic ganglion, but the adult thoracic ganglion lacks appreciable expression of Plexin, the receptor for Sema1 proteins (Horch et al., 2020). In Drosophila melanogaster, Sema-1a is an important regulator of midline crossing, limiting when and where neurons can send axons across the midline. Via reverse Sema signaling, Sema-1a functions can also act as a receptor for secreted Sema2 proteins, causing a surprising type of reverse signaling. This promotes midline attraction and enables axons to cross the midline and form functional midline circuits (Hernandez-Fleming et al., 2017). Such mechanisms } µo } µv (} Z] l [µ] } · o]] · X } v } u completed by the Horch lab, we know that Sema2a is also present in the PTG (Fisher et al., 2018). Furthermore, predicted Sema1a.2 protein sequence was closely related to Drosophila melanogaster Sema1a (Horch et al., 2020). Therefore, it is possible that reverse Sema signaling guides the dendrites post-amputation when they cross the midline to form connections with the opposite side.

al., 2011). Sem1a.2, also present in the prothoracic ganglion, was considered the other target protein because in situ hybridization localized strong expression of Sema1a.2 in the prothoracic ganglion (Horch et al., 2020). Furthermore, significant downregulation of Sema1a.2 was observed 18 and 30 hours post

if reverse Sema signaling played a role in the midline crossing observed post deafferentation, 7th instar crickets were injected with both Sema2a dsRNA and Sema1a.2 dsRNA. P

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Bateman PW, Fleming PA. 2006. Sex and the single (-eared) female: leg function, limb autotomy and mating history trade-offs in field crickets (Gryllus bimaculatus). Biol Lett. 2(1):33 t35. doi:10.1098/rsbl.2005.0408.