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Unlocking the Role of Satellite Glial Cells in Sensory Neuron Function in Healthy and Disease States

Research in my laboratory focuses on elucidating the principles and mechanisms by which peripheral nervous system neurons regenerate, and to identify therapeutic targets to improve neuronal recovery following axon injury. To understand why regeneration occurs in the peripheral but not the central nervous system, our lab studies a unique cell type that spans both systems: sensory neurons of the dorsal root ganglia. The cell bodies of sensory neurons are located in the dorsal root ganglion, a structure that sits just outside the spinal cord. These sensory neurons have a unique pseudo-unipolar morphology with a single axon which bifurcates within the ganglion; one axon proceeds along peripheral nerves and the other proceeds centrally along the dorsal root into the spinal cord. Importantly, the peripheral axon has a much greater regenerative capacity than the central axon.

Using this system, we have discovered epigenetic, transcriptional, and translational pathways employed by peripheral neurons to increase their growth capacity. While we continue to study the signaling pathways elicited in sensory neurons, we recently turned our attention to the possibility that other cells residing in dorsal root ganglia contribute to the nerve repair process. We focused on the glial cells that envelop the sensory neuron soma, known as satellite glial cells (SGC). We discovered that the Fasn-PPARalpha signaling pathway in SGC contribute to nerve repair and this pathway is conserved across rodent and human SGC. In our recent studies we found that SGC receives signals from macrophages after nerve injury that contribute to promote axon regeneration. Our recent studies suggest that the endothelin signaling pathways in SGC contribute to age-dependent axon regenerative decline.

While we continue our studies on mechanism nerve repair, we have also recently been interested in understanding how SGC contribute to sensory dysfunction in mouse models of autism. Together, our studies point to the importance of better understanding the contribution of DRG resident cells in nerve injury responses, which may pave the way for improved efficiency in translating discovery into new treatments of nerve injury and disease.

Keynote presenter