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Antibody Receptor Signaling from Spinal Cord Glial Cells Promotes Neuropathic Pain

Dr. Michael Lacagnina is a postdoctoral fellow with Dr. Peter Grace in the Department of Symptom Research at the University of Texas MD Anderson Cancer Center. He received his PhD at Duke University under the mentorship of Dr. Staci Bilbo, where he examined the role of neuroinflammatory signaling involved in opioid drug reinforcement. Dr. Lacagnina's current research has centered on delineating neuroimmune mechanisms in the spinal cord and brain that contribute to the manifestation of chronic pain. He is particularly interested in uncovering the role that astrocytes and microglia play in shaping the emergence of pain after injury, and how targeting these glial cells may offer new therapeutic strategies for treating the symptoms of pain.

Abstract: A hallmark of nerve injury is increased neuroimmune signaling from glial cells in the spinal cord, which can lead to enhanced activity of nociceptive neural circuits. However, it remains unclear what mechanisms control glial cell activation in the spinal cord following nerve injury. In this talk, we will present evidence that neuroimmune-mediator production by spinal cord glial cells is facilitated by activation of Fc gamma receptors (FcγRs), the receptors for immunoglobulin G (IgG) antibodies. Furthermore, inhibiting this signaling axis has the potential to arrest neuropathic pain behaviors. This raises the possibility that glial FcγRs may be targets for novel treatments to alleviate suffering from neuropathic pain.

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