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**Research Description:**

Our laboratory is focused on investigating how inflammatory mediators alter the sensitivity of the sensory neurons that initiate pain signaling. Our current work involves examining the role of small g proteins in growth factor-initiated transduction cascades and how these pathways alter the sensitivity of sensory neurons. We predominantly work with primary cultures of murine sensory neurons and measure the stimulus-evoked release of neuropeptide transmitters from these neurons as an indication of change in sensitivity. We manipulate the small g proteins and other components of the transductions cascades by pharmacological means when possible, but also use genetically altered mice, siRNA and viral infection to change expression of specific proteins in these pathways. One of the GAPs that we study is neurofibromin. There are reduced functional neurofibromin levels in people with the common human genetic disorder, neurofibromatosis type 1. The following are examples of specific projects available for an apprenticeship experience:

1) To determine if changes in expression of GTPase-activating proteins (GAPs) are causally related to the state of sensitization of mouse sensory neurons as measured by stimulus-evoked release of neuropeptide transmitters. This project uses mice with genetic mutations that cause decreased expression of GAPs, as well as, siRNA treatment to decrease GAP expression and viral infection to increase GAP expression.

2) To determine if the sensitizing actions of inflammatory growth factors and cytokines are altered by modulation of the expression of GAPs. This project uses stimulus-evoked release of neuropeptide transmitters from sensory neuronal cultures, as well as, models of pain behavior in the whole animal to examine the importance of GAPs in growth factor-mediated responses.

**Ten representative publications:**


