Title: The Role of Neuroglian/L1-CAM in Ras-Mediated Tumor Growth

Approximately 40.5% of individuals will face a cancer diagnosis in their lifetime. Nearly a third of these cancers attributable to oncogenic mutations in the KRAS gene. KRAS cooperates with the epidermal growth factor receptor (EGFR) to drive aggressive cancers that are resistant to existing therapeutic. How EGFR is regulated to promote oncogenic KRAS tumors remains unclear. Expanding our basic understanding of oncogenic Ras signaling has the potential to expose vulnerabilities that can be targeted to confront KRAS-driven cancers. Animal models, such as *Drosophila*, have been instrumental in elucidating fundamental signaling mechanisms. Through a candidate genetic screen approach, the lab found the cell adhesion molecules Neuroglian (Nrg) is required for oncogenic RAS-mediated tumor overgrowth in *Drosophila.* We propose that oncogenic RAS signaling transcriptionally stimulate Nrg. In turn, Nrg stabilizes EGFR to promote RAS tumor overgrowth and drug resistance.