CURRENT ROLE AT STANFORD

The main project of the Li lab is to elucidate the signaling pathways responsible for maintaining and initiating brain tumor growth. Previously the Li lab has identified an interesting protein, known as casein kinase 2, which plays an integral role in adult brain tumor growth. My goal is to expand on this initial finding and determine if casein kinase 2 could be a therapeutically relevant drug target in adult brain tumors and whether this protein plays a role in pediatric brain tumors. The role of the candidate would be the lead researcher on these projects, as well as managing and leading the undergraduate and medical students in the lab. In addition, I am responsible for maintaining the laboratory and assisting in grant writing.

HONORS AND AWARDS

- Benjamin F. Graham Jr. Scholarship, Grinnell College (2000 to 2001)
- Ruth L. Kirschstein National Research Service Award, NIH (2002 to 2007)
- Basic Research Fellowship, ABTA (8-2008 to 8-2010)
- Travel Fellowship School of Medicine Travel Grant, Stanford University School of Medicine (2010)

LINKS

- Li Lab Site: http://neurosurgery.stanford.edu/research/gordonli/

Publications

PUBLICATIONS

- **Casein kinase 2a regulates glioblastoma brain tumor-initiating cell growth through the β-catenin pathway.** *Oncogene*
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- **α-Glutamyl transferase 7 is a novel regulator of glioblastoma growth.** *BMC cancer*
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- **Casein Kinase 2: a novel player in glioblastoma therapy and cancer stem cells.** *Journal of molecular and genetic medicine : an international journal of biomedical research*
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- **The invasive nature of glioblastoma.** *World neurosurgery*
  Nitta, R. T., Li, G.
  2013; 80 (3-4): 279-280
Expression of epidermal growth factor variant III (EGFRvIII) in pediatric diffuse intrinsic pontine gliomas
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Epidermal Growth Factor Receptor Variant III Contributes to Cancer Stem Cell Phenotypes in Invasive Breast Carcinoma
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The role of the c-Jun N-terminal kinase 2-alpha-isoform in non-small cell lung carcinoma tumorigenesis
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Constitutive Activity of JNK2 alpha 2 Is Dependent on a Unique Mechanism of MAPK Activation
Nitta, R. T., Chu, A. H., Wong, A. J.
2008; 283 (50): 34935-34945

Evidence that Proteasome-Dependent Degradation of the Retinoblastoma Protein in Cells Lacking A-Type Lamins Occurs Independently of Gankyrin and MDM2
Nitta, R. T., Smith, C. L., Kennedy, B. K.
2007; 2 (9)

Stabilization of the retinoblastoma protein by A-type nuclear lamins is required for INK4A-mediated cell cycle arrest
2006; 26 (14): 5360-5372

A-type lamins regulate retinoblastoma protein function by promoting subnuclear localization and preventing proteasomal degradation
2004; 101 (26): 9677-9682