# *Weekly Colloquium*

Tuesday, 09/19/2017, 12:30pm, Billings Building – Rosedale Conference Room

## *"The Axonal Injury Response: Friend and Foe"*


***Trent Watkins, PhD***
***Assistant Professor, Department of Neurosurgery***
***Baylor College of Medicine***
***Houston, TX***


**Recent publications:**


Larhammar M, Huntwork-Rodriguez S, Jiang Z, Solanoy H, Sengupta Ghosh A, Wang B, Kaminker JS, Huang K, Eastham-Anderson J, Siu M, Modrusan Z, Farley MM, Tessier-Lavigne M, Lewcock JW, **Watkins TA**. Dual leucine zipper kinase-dependent PERK activation contributes to neuronal degeneration following insult. Elife. 2017 Apr 25;6. pii: e20725. doi: 10.7554/eLife.20725. PubMed PMID: 28440222; PubMed Central PMCID: PMC5404924.

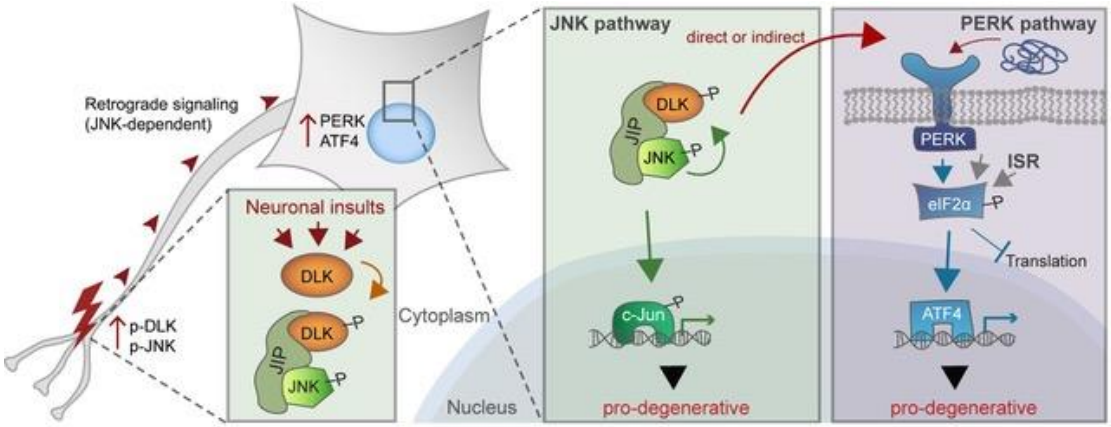
**Watkins TA**, Wang B, Huntwork-Rodriguez S, Yang J, Jiang Z, Eastham-Anderson J,Modrusan Z, Kaminker JS, Tessier-Lavigne M, Lewcock JW. DLK initiates a transcriptional program that couples apoptotic and regenerative responses to axonal injury. Proc Natl Acad Sci U S A. 2013 Mar 5;110(10):4039-44. doi: 10.1073/pnas.1211074110. Epub 2013 Feb 19. PubMed PMID: 23431164; PubMed Central PMCID: PMC3593899.

Huntwork-Rodriguez S, Wang B, **Watkins T**, Ghosh AS, Pozniak CD, Bustos D, Newton K, Kirkpatrick DS, Lewcock JW. JNK-mediated phosphorylation of DLK suppresses its ubiquitination to promote neuronal apoptosis. J Cell Biol. 2013 Sep 2;202(5):747-63. doi: 10.1083/jcb.201303066. Epub 2013 Aug 26. PubMed PMID: 23979718; PubMed Central PMCID: PMC3760612.



**Research Abstract:**

Neurons encounter a variety of insults that profoundly impact the function of the nervous system, from trauma (e.g., spinal cord injury) to neurodegenerative pathology (e.g., Alzheimer's disease). The fate of injured neurons depends on their ability to respond and adapt. My laboratory aims to understand the mechanisms and consequences of neuronal injury signaling. This understanding will drive the development of new therapeutic strategies to enhance repair pathways and reduce pathological responses. Axonal damage provides an invaluable system for these investigations, providing: (1) distinct examples of the strikingly contrasting outcomes of injury signaling, from functional axon regeneration to extensive neurodegeneration; (2) technical simplicity of both in vitro and in vivo models, including genetic manipulation; and (3) application to persistent challenges in neurology and neurosurgery. Our studies have identified the Dual Leucine-zipper Kinase (DLK) as a master regulator of both regenerative and apoptotic responses to optic nerve damage and of multiple axonal stress response pathways, including JNK and PERK signaling. Manipulation of DLK therefore represents an appealing approach for exploiting the intrinsic neuronal injury response for therapy.



For more information contact: dwhite@burke.org

Burke Medical Research Institute
*Academic Affiliate of Weill Cornell Medicine*
785 Mamaroneck Avenue, White Plains, NY 10605 | T. 914.597.2551 | burke.weill.cornell.edu