BURKE MEDICAL RESEARCH INSTITUTE

Weekly Colloquium

Tuesday, 1/30/2018, 12:30pm, Billings Building – Rosedale Conference Room

"Influences of neuronal activity on pain and regeneration after spinal cord injury"

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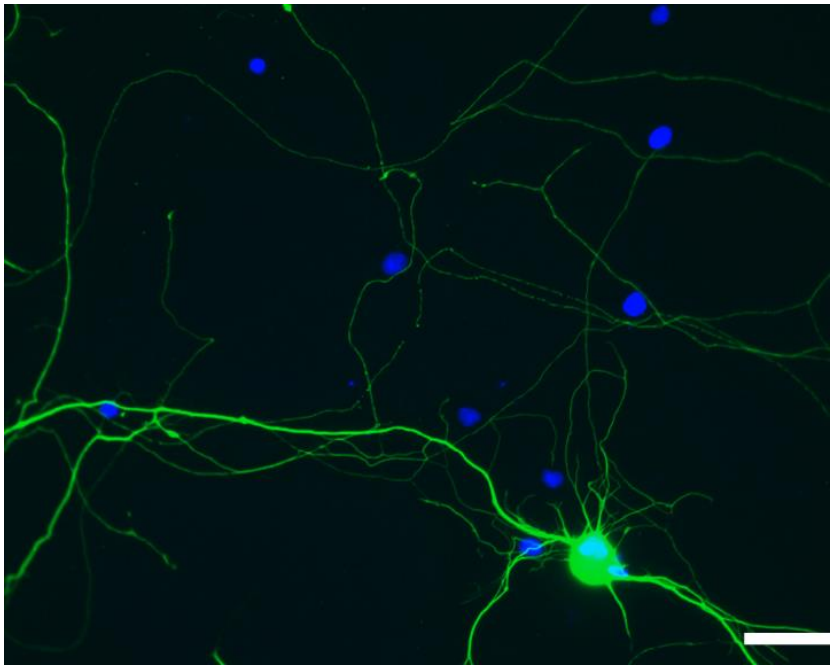
Research Summary:
The majority of my research is related to the modulation of plasticity and regeneration in the adult nervous system using animal models of traumatic CNS injury and in the past, neurodegenerative diseases, as well as in vitro assays of axonal growth and neuronal survival. The development of cell and gene therapy for diseases of the nervous system constitutes an important aspect of this work. In addition to translational studies, I am equally interested in the basic mechanisms underlying the capacity or inability of neurons to regenerate and maladaptive plasticity in the injured adult nervous system. Animal models used in current experiments include different lesions in the adult rat and mouse spinal cord, and the peripheral nervous system. Current studies are focused on the potential role of neural stem cells, biomaterials and neuronal activity as a means for spinal cord regeneration, and the structural and functional changes underlying the development of pain after spinal cord injury. The latter together with evaluation of bladder dysfunction and autonomic responses after injury have added more clinically relevant outcome measures to our animal models of spinal cord injury complementing a strong focus on cellular and molecular mechanisms.



Publications:

Fouad K, Bennett D, Vavrek R, **Blesch A** (2013). Long-term viral brain-derived neurotrophic factor delivery promotes spasticity in rats with a cervical spinal cord hemisection. Front Neurol: 4:187. PMID:24312075

Hou S, Nicholson L, van Niekerk E, Motsch M, **Blesch A** (2012). Dependence of regenerated sensory axons on continuous neurotrophin-3 delivery. J Neurosci: 32:13206 –13220.

Blesch A, Lu P, Tsukada S, Taylor Alto L, Roet K, Coppola G, Geschwind D, Tuszynski MH (2012). Conditioning lesions before or after spinal cord injury recruit broad genetic mechanisms that sustain axonal regeneration: superiority to cAMP-mediated effects. Exp Neurol: 235:162-173.