**Week Colloquium**
Tuesday, 6/27/2017, 12:30pm, Billings Building – Rosedale Conference Room

"**Molecular mechanisms driving spontaneous recovery of function after CNS trauma**"

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**Research abstract:**
Axons in the adult central nervous system (CNS) fail to regenerate after spinal cord injury (SCI), therefore patients with severe SCI remain insensate and paralyzed below the level of the injury. To date, most research has sought to repair the damaged spinal cord by stimulating cut axons to regenerate across the injury site. However, this strategy has resulted in only incremental gains and has not been translated to the clinic. My laboratory focuses on the capacity of intact CNS circuitry to undergo structural plasticity to drive restoration of function post injury. Previously I showed that sprouting of intact motor (corticospinal tract and rubrospinal) and sensory (primary afferent) terminals mediated modest functional recovery after SCI. Critically, recovery was enhanced if the CNS was rendered more permissive via negating the inhibitory activity of proteins such as NogoA and the chondroitin sulfate proteoglycans. These data demonstrated the potency of intact pathways to restore function. Using transcriptional profiling, genetics, physiology, biochemistry, tissue culture and chronic two-photon in vivo imaging methodology, my laboratory seeks to identify and exploit the molecular mechanisms that drive structural plasticity in intact CNS neurons and develop new tools with which to specifically study these pathways to ultimately restore function after SCI.

**Recent publications:**
