**Weekly Colloquium**

Tuesday, 04/05/2016, 12:30pm, Billings Building – Rosedale Conference Room

“Neuroprotection and Regeneration of the Injured Spinal Cord”

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**Research Summary**

The goal of our laboratory is to study mechanisms underlying spinal cord injury (SCI) and develop novel repair strategies to improve anatomical reorganization and functional recovery in experimental models of SCI. Our long-term goal is to translate effective treatments from animal models to humans. To reach these goals, two lines of research are being conducted. First, we aim at acute neuroprotection by investigating novel targets that may play central roles in mediating progressive secondary degeneration in the spinal cord after injury. Our recent work suggests that phospholipase A2 (PLA2), a diverse family of phospholipid enzymes, may be such a molecule. We are currently studying mechanisms underlying PLA2-mediated spinal cord secondary injury including mitochondria dysfunction, and PLA2-RhoA/Rho Kinase interaction and signaling pathways. We are also testing agents that may block PLA2-RhoA/Rho Kinase-mediated cytotoxicity and cell death to enhance neuroprotection and recovery of function in animal models of SCI. A second line of research is to use cellular transplantation strategies to promote axonal regeneration through and beyond a lesion gap after SCI. SCI incurs disconnection of nerve fibers (called axons) and a successful repair strategy requires reconnection of these axons to their appropriate targets. Grafts of tissue engineered scaffolds seeded with different growth-supportive cells, such as Schwann cells (SCs), oligodendrocyte progenitor cells (OPCs), or immature astrocytes derived from embryonic stem cells, may provide a necessary cellular alignment and environment to guide and support axonal regeneration through the lesion gap and beyond. We combined cell-based therapy with other efficacious treatments including boosting the intrinsic regenerative capacity of injured CNS neurons, overcoming the inhibitory environment associated with the glial scar and CNS myelin, providing growth-promoting pathways along the course of axonal regeneration, and enhancing synaptic reconnection between regenerating axons and their targets. Ultimately, the final repair of the injured spinal cord may be achievable by combining an early phase of neuroprotection, a delayed phase of transplantation-mediated axonal regeneration, and vigorous rehabilitation strategies.

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**Recent Publications:**

