Weekly Colloquium
Tuesday, 03/29/2016, 12:30pm, Billings Building – Rosedale Conference Room

“Intermittent Hypoxia and Neonatal White Matter Injury - the Role of Mitochondria”

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Research Summary
Perinatal white matter injury (WMI) is the most common cause of life-long neurological handicap in premature infants. Over the past decade, pathological pattern of WMI has shifted away from extensive periventricular tissue loss, named cystic periventricular leukomalacia (PVL) toward non-cystic focal and diffuse white matter abnormality, the essence of which is axonal myelination defect. This evolution in clinical and pathological phenotypes of this disease implies changes in the main mechanisms of WMI from the mechanisms of cell death toward the mechanisms of maturational failure. My presentation is focused on the support of our novel Hypothesis, that mitochondrial bioenergetics dysfunction to support a proper growth and development is a fundamental mechanism of maturational failure of oligodendrocyte lineage cells and poor myelination. This hypothesis is being tested using our original model of non-cystic WMI produced by frequent intermittent hypoxic stress in neonatal mice. The nature of mitochondrial dysfunction is uncoupling of oxidative phosphorylation triggered by intermittent hypoxic stress. The primary biophysics of mitochondrial uncoupling is coupling of oxidative phosphorylation triggered by intermittent hypoxic stress. The primary biophysics of mitochondrial uncoupling is transient opening of the mitochondrial permeability transition pore to release matrix Ca2+. At the end of the presentation, a fundamental nature of the hypothesized concept is shown by the application of the same concept to the alveolar developmental arrest, the chronic lung disease of prematurity.

Recent publications:
