

CENTER FOR EXCELLENCE in the NEUROSCIENCES



S E M I N A R S E R I E S

Enduring Interneuronopathy in the Prefrontal Cortex of Young Adult Offspring Exposed to Ethanol In Utero

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Monday, August 17th 2015

12:00-1:00 p.m.

Alfond 113
Biddeford Campus

Lunch will be Provided

Hosted by: Edward Bilsky, Ph.D.

Sponsored by: Center for Excellence in the Neurosciences



Dr. Alex Skorput graduated from UNE in 2004 with a BS in medical biology, and recently received a PhD in experimental and molecular medicine from Dartmouth College. He will share his work examining the role of abnormal GABAergic interneuron migration in the developmental etiology, and enduring consequences, of in utero ethanol exposure on the cerebral cortex.

Cortical processing depends upon a balance of synaptic inhibition and excitation within the intracortical circuit. While comprising only 20% of the total neuronal population, GABAergic interneurons provide local synaptic inhibition and pace cortical rhythmus essential for cognition. These cells migrate tangentially from subcortical germinal zones to reach the developing cerebral cortex during embryonic brain development. Abnormalities in this migratory path may skew the inhibitory/excitatory balance within intracortical circuits. Diseases in which a portion of their etiology is attributable to such aberrant migration are termed "interneuronopathies" and include epilepsy, schizophrenia, and autism spectrum disorder. Fetal Alcohol Spectrum Disorders (FASD) are the result of in utero exposure of the developing brain to ethanol. These disorders are hallmarked by deficits in executive function, which include attention, impulse inhibition, and behavioral flexibility. These higher order cognitive processes depend upon proper functioning of the prefrontal cortical circuit. While the teratogenic effects of ethanol on the developing brain have been recognized for decades, in utero ethanol exposure remains the leading cause of developmental disability in the US. The developmental etiology of FASD suggests that the associated deficits arise due to atypical cortical development. However, the mechanisms by which this occurs are poorly understood. In this seminar Dr. Skorput will present anatomical, electrophysiological, and behavioral data suggesting a role for interneuronopathy in the etiology of FASD, and describe mechanistic studies aimed at elucidating therapeutic avenues for its attenuation.