



Understanding the Molecular and Circuit Interactions Underlying Motor Learning in Normal and Disease Mouse Models

SEMINAR & VISITING SPEAKER SERIES WORLD WIDE NEURO PLATFORM

DATE

Tuesday, May 24, 2022
12:00 PM CST

WORLD WIDE NEURO LINK

This talk will be hosted on zoom:

<https://umanitoba.zoom.us/j/64221713635?pwd=eHlxRy9pWVBLOWI4eCtGdTV1SjFQdz09>

MEETING ID 642 2171 3635

PASSCODE 243454

SPEAKER

Dr. Simon Chen, PhD

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BIO

Simon completed his BSc in Cell Biology at the University of British Columbia (UBC). He continued his Ph.D. at UBC in Dr. Kurt Haas' lab, where he learned in vivo two-photon imaging and studied how synapse formation and neuronal activity direct the plasticity of functional neuronal networks in the developing brain of *Xenopus* tadpoles. He then moved to sunny San Diego where he did his postdoctoral research in Dr. Takaki Komiyama's lab. He acquired expertise in two-photon in vivo imaging in awake and behaving mice. He started his lab at the University of Ottawa in 2016, and he currently holds the Tier II Canada Research Chair position.

RESEARCH

Mammals exhibit an incredible amount of flexibility in motor control, which is believed to be due to the remarkable ability of brain circuits to rapidly undergo structural and functional plasticity to fluidly modifying body movements through learning. Disrupting these processes can often lead to impaired motor learning in both normal and diseased conditions. Our lab research focuses on bridging the gap between cellular and molecular signaling underlying the plasticity of neural circuits involved in motor skill learning. In the first part of the talk, I will present our current work, in which we revealed a critical role of a functional distinct NPAS4-expressing somatostatin-interneuron ensemble in motor learning, using chronic in vivo two-photon imaging in head-fixed behaving mice. In the second part of the talk, I will present a recently published work from the lab, in which we examined mice with a syntenic deletion of chromosome 16p11.2, a common copy number variation associated with ASD. We found 16p11.2 deletion mice display a delay in motor learning, which reminiscent of the motor learning-related deficits in children with ASD, without showing gross movement deficits. In addition, we identified a dysfunctional locus coeruleus noradrenergic (LC-NA) neuromodulatory system that leads to abnormal structural and functional changes in the motor cortex, which resulted in delayed motor learning in the 16p11.2 deletion mice.

OBJECTIVES

1. Understand the microcircuit interactions between excitatory and inhibitory neurons in the motor cortex
2. Examine the role of Npas4 transcription factor in regulating motor learning
3. Dissect neural circuits underlying the delayed motor learning in the 16p11.2 deletion model of Autism

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