College of Science and Engineering | Department of Biomedical Sciences

Seminar

Genetic Mechanisms underlying Homeostatic Plasticity of Inhibitory Transmission

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Date: 2 Jun 2016 (Thursday)

Time: 12:30 pm – 2:00 pm (Reception with light sandwiches starts at 12:15 pm. To facilitate the order of sandwiches, please register through email shulchan2@cityu.edu.hk.)

Venue: P4302, Academic 1, City University of Hong Kong

Language: English

Abstract

Extreme and persistent alterations in network activity can occur as a consequence of associative plasticity or in disease states. In response to fluctuating activity levels, homeostatic mechanisms adjust excitatory and inhibitory transmission to maintain an appropriate degree of excitability to enable proper information processing. Inhibitory transmission in the cortex, mediated by GABAergic interneurons, downregulates in response to reduced activity levels. At the synaptic and circuit level, what are the signaling and genetic mechanisms that control this homeostatic process? TrkB, a major receptor for the brain-derived neurotrophic factor, can regulate interneuron anatomy and physiology, but its role in GABAergic homeostatic plasticity remains elusive. Downstream of TrkB, glutamic acid decarboxylase 67 (GAD67) is known for its role in GABA synthesis; it is unclear, however, whether it is involved in regulation of inhibitory synaptic strength. In this presentation, I will spend most of my time discussing how I dissected the molecular and physiological underpinnings of homeostatic plasticity of GABAergic neurons using a combination of mouse genetics, optogenetics and electrophysiology.

In the second part of my talk, I will discuss some of the ideas I have for my future research. My overarching goal is to understand how specific neuron-types are recruited during information processing and how they can be affected in disease states. These are the questions I would like to address: How are different forms of information extracted by neural circuits during olfactory processing? How does the interaction of excitatory and inhibitory neurons mediate sensory learning? How are different cell types affected by diseases such as Alzheimer's and epilepsy? I believe that, by understanding the connectivity and plasticity of neural circuits, we can develop better and safer neurotherapeutics.

Biography

Dr Lau received his Bachelor of Technology degree (Biomedical Science) with First Class Honours from the University of Auckland in 2001. He received his Master of Science in 2004 and PhD in 2007 from the Albert Einstein College of Medicine (New York) under the guidance of Suzanne Zukin and Michael Bennett. His graduate thesis work focused on the cellular and molecular mechanisms of glutamate receptor regulation in synaptic plasticity. Having attended the Neural Systems and Behavior at Marine Biological Laboratory, Woods Hole, Dr Lau decided to pursue systems neuroscience research in the lab of Venkatesh Murthy at Harvard University. His postdoctoral work surrounded the theme of genetic regulation of homeostatic plasticity at the synaptic and circuit level. In recognition of Dr Lau's work, he received a NARSAD Young Investigator Award in 2010. His teaching of an undergraduate tutorial entitled "Synapses: molecules, networks and behavior" earned him a Certificate of Distinction in Teaching from Harvard in 2014. Dr Lau joined CityU as an Assistant Professor in March 2016.

**** All ARE WELCOME ****

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