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NEUROSCIENCE & BEHAVIOURAL DISORDERS

NNRIS Bench to Bedside Seminar Series

Date: **21 May 2021 (Friday)**

Time: **12:00pm – 1:00pm**

Zoom Details: **https://nus-sg.zoom.us/j/85479284427?pwd=S0FuZkhIdldFWjh2K1IraFc2V1BUdz09**
Meeting ID: 854 7928 4427
Passcode: 578013
Note: Please rename your login name to include your institute to facilitate admission

Moderator: **Assoc Prof Hyunsoo Shawn Je**
Neuroscience & Behavioural Disorders Programme, Duke-NUS

IMAGING MOLECULAR STRUCTURE, DYNAMICS, AND PLASTICITY OF DENDRITIC SPINES

Dr Jun Nishiyama
Assistant Professor
Neuroscience & Behavioural Disorders Programme
Duke-NUS Medical School

Abstract:
Our brain functions depend on proper connections between tens of billions of neurons. These connections or synapses are disrupted in many neuropsychiatric disorders. However, detailed molecular mechanisms underlying synaptic function and dysfunction are largely unknown. In this talk, I will introduce studies to probe endogenous proteins and image signal transduction at a single-synapse resolution using CRISPR/Cas9-mediated genome editing and various optical techniques. I will discuss how these advanced tools can help for the deeper understanding of molecular regulation of synapse.

Biography:
Jun Nishiyama is an assistant professor at Duke-NUS Medical School. He obtained M.D. and Ph.D. from the University of Tokyo, completed his residency in psychiatry, and performed his postdoctoral studies at Max Planck Florida Institute. He pioneered in vivo genome editing in the brain and received Japan Neuroscience Society Young Investigator Award in 2018 and Singapore NRF fellowship in 2020.

THE ROLE OF APOE4 ON INFLAMMATORY CHANGES IN THE APPKINL-G-F ALZHEIMER'S DISEASE MOUSE MODEL

Mr Calvin Cheah Chee Hoe
Research Assistant
Neuroscience and Mental Health Faculty
Lee Kong Chian School of Medicine
Nanyang Technological University

Abstract:
Alzheimer's disease (AD) is the most common dementia contributing to high socioeconomic burden. Despite decades of research efforts, the intervention strategies of AD were not significantly improved. This can be in part attributed to the inappropriate mouse models used in AD preclinical studies. Recently, Saido et al. generated an APP knock-in (KI) mouse model using the APP construct containing the Swedish (NL), Iberian (G) and Arctic (F) mutations (APPKINL-G-F). This KI mouse model exhibits accelerated amyloidosis without overexpressing APP, as well as other AD phenotypes such as gliosis, an advantage to over other mouse models in resembling clinical cases. To study for potential biomarkers during pre-AD symptomatic stages, we utilized qPCR and immunohistochemistry to unveil the neuroinflammation-associated changes at an early age of 3 months. APOE4 transgenic mouse was used to generate an APPKI:APOE4 double mutant to identify the effect of the APOE4 gene on the AD preclinical mouse model. We found that the 3-month old APPKINL-G-F mouse displayed significantly higher glia density and activation, whereas APOE4 seems to have a protective effect over the observed gliosis. However, both mouse models displayed clustered cell death as compared to none in control, suggesting non-inflammatory causes of cell death. These findings suggest that the mouse models play an important role in AD biomedical research, such as biomarker research whereby AD phenotypes were observed at early age.

Biography:
Mr. Calvin Cheah graduated from the International Medical University (Malaysia) with an honours in Biomedical Science before beginning his research career in Singapore. He is currently a research assistant in NTU-Lee Kong Chian School of Medicine, under the supervision of Assoc. Prof. Eyleen Goh to study the cellular and molecular changes of various neurodegenerative diseases.

All are welcome. No registration is required.