for favour of posting
dated on: 2024/02/01

The Chinese University of Hong Kong
Department of Psychiatry
Schedule for February, 2024

Date	Time	Activity	Speaker(s)
Feb1		No Event	
Feb8	14:30-16:00	Psychotherapy Case Conference (MUL)# *Attachment trauma in family context - a case illustration*	Dr. Irene KAM
	16:00-17:00	Psychotherapy Supervision (MUL)#	
Feb15	14:30-16:30	Quality Assurance Meeting (SH)#/(TPH)#	
Feb22	14:30-16:00	Academic Lecture (MUL)* *Somatic Mobile Element Insertions in Human Brain Development and Neurodevelopmental Disorders* Registration: https://bit.ly/3vOQAju	Prof. Xiaowei ZHU Assistant Professor Department of Neuroscience City University of Hong Kong
Feb29	14:30-15:30	Research Seminar* *Neural correlates of impulsivity across early stages of a-synucleinopathy: a electroencephalography - functional Magnetic Resonance Imaging (EEG-fMRI) case-control study*	Ms. Siyi GONG Supervisor: Prof. YK WING Co-supervisors: Dr. Steven CHAU, Dr. Joanne HUANG, Dr. Yaping LIU
	15:30-16:30	Research Seminar* *Gut microbiota in major depressive disorders with and without Rapid Eye Movement Behavior Disorder: tracing a subtype of depression with underlying neurodegeneration* Registration: https://bit.ly/3OgiHP8	Ms. Yuhua YANG Supervisor: Prof. YK WING Co-supervisors: Dr. Joanne HUANG, Dr. Yaping LIU

Venue:	*Live video	#Closed meeting	@Non-CME Event	MUL	TPH	SH	1AL
				Seminar Room, Multi-centre, Tai Po Hospital, Tai Po, N.T.	Conference Room 1 G/F, Wing D Tai Po Hospital Tai Po, N.T.	Dining Room Ward 7AB Dept. of Psychiatry 7/F, Shatin Hospital Shatin, N.T.	Rm. 1005, Dining Room Ward 1AL, 1/F Tai Po Hospital Tai Po, N.T.

Please contact 2607-6025 two days before hand to arrange presentation equipment.

http://www.psychiatry.cuhk.edu.hk

ACADEMIC LECTURE

Dr. Xiaowei ZHU

Assistant Professor
Department of Neuroscience
City University of Hong Kong

22 Feb 2024 (THU)

14:30 - 16:00

Seminar Room, Multicentre, TPH & Zoom

Topic: Somatic Mobile Element Insertions in Human Brain Development and Neurodevelopmental Disorders

Abstract:

Mobile elements (MEs) are segments of DNA that can change genomic positions, leading to profound impacts on the structure and function of the host genome. Active MEs in the human genome can undergo retrotransposition to create novel mobile element insertion (MEI) mutations in both germline and somatic tissues. Somatic retrotransposition of active LINE1 elements occurs during human brain development and is hypothesized to contribute to neurodevelopmental disorders (NDDs) by affecting the expression level or the coding sequences of disease-associated genes. However, individual somatic MEIs are present in small proportions of cells at a given anatomical location, and thus far the overall mutation frequency and direct functional impacts remain elusive. In this talk, I will first address the MEI detection problem using a novel machine-learning algorithm and a digital PCR-based approach for validation. I will then discuss the findings from an investigation of a cohort of 130 donors, including 9 Schizophrenia, 19 Tourette Syndrome (TS), 59 Autism Spectrum Disorder (ASD), and 43 Healthy controls. Finally, I will focus on two cases, one with TS and the other with ASD, and discuss the likely scenario where somatic MEIs could contribute to the disease pathogenesis. With future in vivo experiments to tackle the direct functional consequences of somatic MEIs, I hope to reveal a new layer of biological regulation and possible etiology of neurodevelopmental disorders.

Biography:

Prof. Xiaowei Zhu obtained the BSc in the Special Class for the Gifted Young at the University of Science and Technology of China in 2002. Between 2002 and 2009, he studied bioinformatics and computational biology in Yale University, and received PhD with his research on mapping biological networks using genomic and proteomic approaches. Between 2010 and 2021, he joined the department of psychiatry and behavioral sciences at Stanford University, first as a postdoc and then as a research scientist. During this time, he participated in the Brain Somatic Mosaicism Consortium Network and led the research on the computational and functional analysis of somatic mutations in human brain development and neuropsychiatric disorders. Prof. Xiaowei Zhu joined City University of Hong Kong in 2022 as an assistant professor.

Prof. Zhu's main research interest lies in understanding the genetic basis of psychiatric disorders, specifically on the role of somatic retrotransposition of the highly repetitive mobile elements. He developed a novel machine learning based approach (RetroSom), and a novel experimental detection method (droplet-based full length PCR), that allowed, for the first time, to detect somatic L1 and Alu retrotransposition in standard whole-genome sequencing using bulk tissue. Prof. Zhu also led or participated in several other genomic research projects in psychiatry, including a transcriptome analysis comparing the expression patterns between major depression disorder patients and controls, as well as the chromatin structure alterations led by the chr22q11 deletion, which is associated with a significant increase of risk for psychiatric disorders. During his part time he won several top solutions in hackathons organized by the Silicon Valley Artificial Intelligence (SVAI) group, to study rare cancers with AI and genomics.

Registration is required. For enquiries, please contact pci-event-app@cuhk.edu.hk or 26076025.
Please display the registration name for joining the Zoom lecture.

REGISTER NOW

Research Seminar

Date: 29 Feb 2024 (THU)
Time: 14:30 - 16:30
Venue: Zoom

Register Now

Ms. Siyi GONG
Supervisor: Prof. YK WING
Co-supervisors: Dr. Steven CHAU, Dr. Joanne HUANG, Dr. Yaping LIU

Topic: Neural correlates of impulsivity across early stages of α-synucleinopathy: a electroencephalography - functional Magnetic Resonance Imaging (EEG-fMRI) case-control study

Abstract:
Impulsivity, as a multidimensional construct, refers to actions that are poorly conceived, prematurely expressed with unduly risk and often inappropriate to the situation. Impulsivity levels, as measured by a variety of questionnaires and tasks, have been reported to change in patients with early-stage Parkinson's disease (PD), which were initially thought to be primarily associated with dopaminergic agonists. Impulsive compulsive disorders (ICDs), as the most severe end of impulsivity, affect 3.5-44.7% in medicated PD patients with further impairment of patients' quality of life and increase in the caring burden of caregivers. Nevertheless, several studies also suggested that an altered level of impulsivity occurred in early drug-naïve PD patients and probably among patients with idiopathic rapid eye movement sleep behavior disorder (iRBD), a prodromal stage of α-synucleinopathy. Thus, gaining insight into the underlying mechanism of impulsivity could assist with better management of ICD symptoms in PD patients, and unveil potential impulsivity-related pathological changes in early α-synucleinopathy.
Our previous research observed a complex construct of altered impulsivity at which early drug naïve PD patients and iRBD patients had a lower level of risk taking but, interestingly, higher level of reflection impulsivity, indicating altered impulsivity at the early stage of α-synucleinopathy. However, few studies have demonstrated the underlying neural substrates in early α-synucleinopathy. Therefore, we aimed to investigate the functional neural signatures in relation to this altered impulsivity in the following four groups — control subjects, iRBD patients, early medicated PD patients, and early drug naïve PD patients using a combination of simultaneous electroencephalography and functional Magnetic Resonance Imaging (EEG-fMRI) method. The potential finding of the study may help to develop a better understanding of the neural circuitry of altered impulsivity and their management at the early stage of α-synucleinopathy.

Ms. Yuhua YANG
Supervisor: Prof. YK WING
Co-supervisors: Dr. Joanne HUANG, Dr. Yaping LIU

Topic: Gut microbiota in major depressive disorders with and without Rapid Eye Movement Behavior Disorder: tracing a subtype of depression with underlying neurodegeneration

Abstract:
Major depressive disorder, a highly prevalent psychiatric illness with a significant global burden, is characterized by its diverse clinical presentations. Characterized by dream enacting behaviors and loss of muscle atonia, idiopathic REM sleep behavior disorder (iRBD) is known as a prodromal stage of α-synucleinopathy. RBD features are frequently observed in patients with depression and antidepressants use (especially serotonin selective reuptake inhibitors, SSRI). Recent findings showed that depression is associated with a higher risk for neurodegeneration including α-synucleinopathy and dementia. Particularly, our recent data found that MDD patients with RBD occurred in about 9% of MDD in psychiatric clinic and were familially predisposed to α-synucleinopathy. Thus, it is likely that MDD comorbid with RBD may represent a variant of iRBD as well as a distinct subtype of depression with an underlying risk of neurodegeneration. Nevertheless, the intricate relationship between depression and neurodegeneration in terms of their pathophysiological mechanisms remains unclear. Growing attention has been paid to the role of gut-brain axis in the pathogenesis of depression and neurodegeneration. Gut dysbiosis has been considered an important biomarker of both depression and α-synucleinopathy per se. The gut microbiota plays a crucial role in regulating neurotransmitters and metabolites that can impact brain function and mood. Depression is also characterized by a higher abundance of proinflammatory species, which can influence the stress response and inflammatory processes. Our recent data also suggested that there was a distinct gut dysbiosis in patients with iRBD, which closely interacted with their depressive symptoms. Thus, there will be a need for further understanding of the gut microbiota of the subjects with MDD with RBD so as to determine whether it represents a specific subtype of depression predisposed to neurodegeneration as well as their relationship to that of MDD only subjects.
Therefore, we conducted a case-control study in an established clinical cohort consisting of three groups, patients with MDD+RBD, MDD only and healthy control. We aim to identify the variations in gut microbiota between two different subtypes of MDD (with and without RBD) and their correlation with clinical biomarkers. This study could help understand the potential role of gut microbiota in the pathogenesis of MDD and synucleinopathy neurodegeneration and explore the microbial functions which could contribute to depression and neurodegenerative processes. Moreover, we may able to find the key gut microbiota differentiating MDD subtypes and targets for intervention toprevent neurodegeneration in individuals with depression.

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