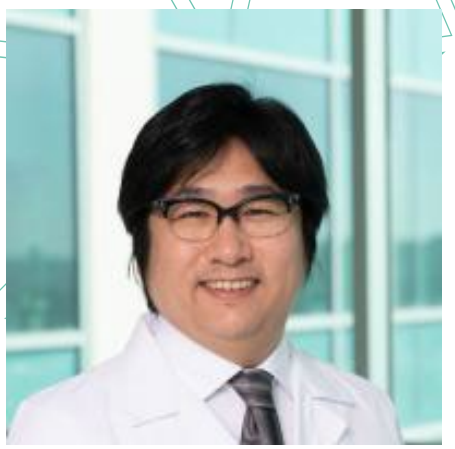
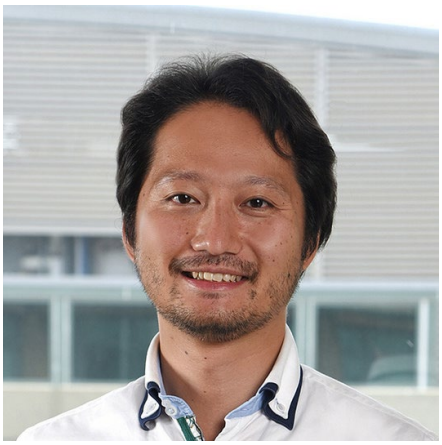
NEURO GLOBAL Seminar

Date:
4 July 2022 (Monday) 17:00-18:30



①Speaker
Takashi KITAMURA, Assistant Professor
Department of Psychiatry, University of Texas Southwestern Medical Center

Title:
Island cells and myself



②Speaker
Kei M IGARASHI, Associate Professor
Department of Anatomy and Neurobiology, School of Medicine, University of California, Irvine

Title:
Circuit mechanisms underlying Alzheimer's disease

Format: **Hybrid (Onsite & Online)**

Venue: **Middle Auditorium, Clinical Lecture Building [A21] 2F, Seiryo Campus** **[MAP]** https://www.tohoku.ac.jp/map/en/?f=SR_A21

Registration: **Refer to the message from the NGP office.**

●Neuro Globalプログラム生(Neuro Global Program Students)
【脳科学セミナーシリーズEx】/【先進脳科学セミナーシリーズEx】セミナー1ポイント
【Brain Science Seminar Series Ex】/【Advanced brain science seminar series Ex】 1 point
●医学系研究科(Graduate School of Medicine)
【医学履修課程】国際交流セミナー (アドバンスド講義科目)」(出席1回分)
【Medical Science Doctoral Course】 International Interchange Seminar (Advanced Lecture course) (It will be counted as 1 attendance.)
●生命科学研究科(Graduate School of Life Sciences)
【単位認定セミナー】単位認定セミナーとして2ポイントを付与します。
【Credit-granted seminar】 2 point will be granted to the students who will attend this seminar.

東北大学 Neuro Global 国際共同大学院プログラム事務局
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NEURO GLOBAL
Tohoku University

NEURO GLOBAL Seminar

①Speaker: Takashi KITAMURA, Assistant Professor

Abstract:

First topic: Medial entorhinal cortex (MEC) represents spatiotemporal cognitive variables. A unique topographical representation for space is recently found in the concerted activity of grid cells that fire at multiple locations with a regular hexagonal structure in MEC, called grid cell modules. While we previously demonstrated that CalB+ cells are clustered into patches arranged in MECII, it remains unknown whether CalB+ cell clusters may facilitate grid cell modules. In my talk, I present the molecular mechanisms for the formation of CalB+ cell clusters during development period and then examine the role of CalB+ cell clusters on the topographical functional modules of grid cell activity in MEC.
Second Topic: Remembering features of the self and monitoring the current states are crucial for self-recognition. However, neural mechanisms of self-recognition still remain unknown due to a limited availability of experimental animal models. Recently, we have demonstrated a mirror-induced visual self-recognition (MSR) in mice, which requires mice to remember visual features of their own heads as a reference and find the current status via a mirror reflection. In my talk, I present neural circuit mechanisms for how we remember the visual features of the self.

Selected article:

1) Terranova et al., ***Neuron***. 110(8):1416-1431 (2022)
2) Kitamura et al., ***Science***, 356, 6333, 73-78 (2017)
3) Kitamura et al., ***Science***, 343, 896, 896-901 (2014)
4) Kitamura T, et al., ***Cell***, 139, 4, 814-827 (2009)

Related Website:

https://www.kitamuralab.org/
https://researchmap.jp/gengehotaruika

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NEURO GLOBAL
Tohoku University

NEURO GLOBAL
Seminar

②Speaker: Kei M IGARASHI, Associate Professor

Abstract:

Alzheimer's disease (AD) currently affects more than 50 million people worldwide, but no cure exists. Although molecular and cellular mechanisms of AD are becoming clearer from recent studies using AD animal models, brain circuit mechanisms of how neurodegeneration of vulnerable neurons causes memory impairment in AD subjects are still unclear. This is a critical gap in knowledge in current AD research: If we can clearly identify such circuit mechanisms, we may be able to develop a therapeutic treatment to prevent the deterioration of memory circuits in AD patients. To fill this critical gap, my lab has been striving to elucidate circuit mechanisms of AD that cause memory impairment using *in vivo* circuit analysis and electrophysiological methods. We are focusing on the entorhinal cortex (EC), a brain region that receives input from multiple cortical regions and sends information to the hippocampus. Importantly, the EC is the earliest brain region that exhibits atrophy and activity loss in patients with early-stage AD. Using a novel amyloid precursor protein knock-in (APP-KI) AD mouse model (Saito, Saido et al., 2014), we recently found that a neuronal function that discriminates distinct environment, called “remapping”, is impaired in the memory circuit of entorhinal cortex and hippocampus (Jun et al., *Neuron* 2020). I will also share our results that another neuronal function for associative memory (Lee, Jun, Soma, Nakazono et al., *Nature*, 2021) shows a characteristic impairment in the AD mouse model. Finally, I would like to discuss insights from our results for identifying vulnerable cell types in AD in future studies.

Selected article:

Lee JY, Jun H, Soma S, Nakazono T, Shiraiwa K, Dasgupta A, Nakagawa T, Xie JL, Chavez J, Romo R, Yungblut Y, Hagihara M, Murata K, and Igarashi KM* (2021)
Dopamine facilitates associative memory encoding in the entorhinal cortex ***Nature*****,** 598:321-326
Jun H, Bramian A, Soma S, Saito T, Saido TC, Igarashi KM* (2020)
Disrupted Place Cell Remapping and Impaired Grid Cells in a Knockin Model of Alzheimer's Disease ***Neuron***, 107:1095-1112

Related Website: www.igarashilab.org

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