The Singapore Bioimaging Consortium (SBIC)
presents a seminar

on

"Dynamic interaction between Aβ plaques and immune cells in the brain of AD animal models"

Speaker: **Prof Inhee Mook-Jung**
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Host : **Prof Chang Young-Tae**
Date : **Friday, 5 February 2016**
Time : **4.00pm – 5.00pm**
Venue : **SBIC Seminar Room**
11 Biopolis Way
Level 2, Helios Building, Singapore 138667
(Please enter via Level 1)

Abstract

Pathological hallmarks of Alzheimer's disease (AD) include extracellularly accumulated amyloid β (Aβ) plaques and intracellular neurofibrillary tangles in the brain. Activated microglia, brain-resident macrophages, are also found surrounding Aβ plaques. The brain of AD mouse models revealed that Aβ plaque formation is completed by the consolidation of newly generated plaque clusters in vicinity of existed plaques. However, the dynamics of Aβ plaque formation, growth and the mechanisms by which microglia contribute to Aβ plaque formation are unknown. In the present study, we confirmed how microglia are involved in Aβ plaque formation and their growth in the brain of 5XFAD mice, the Aβ-overexpressing AD transgenic mouse model, and performed serial intravital two-photon microscopy (TPM) imaging of the brains of 5XFAD mice crossed with macrophage/microglia-specific GFP-expressing CX3CR1$^{GFP/GFP}$ mice. Also, we examined how innate immune cells including neutrophil, which is the most abundant type of white blood cell, infiltrates into the brain and migrates to amyloid plaques in the brain of 5XFAD mice using TPM. Our results show that chronic Aβ deposition attracts neutrophils from the blood vessels in AD animal model mice, suggesting that immune cell responses might be involved in the progress of AD. Understanding the interaction between immune cells and senile plaques might provide new insights into pathophysiology and potential treatments of AD.

About the Speaker

Professor Inhee Mook-Jung has been working on molecular pathogenesis of Alzheimer's disease (AD). Her major interests are (1) how does mitochondrial dysfunction contribute to AD pathogenesis, (2) how does glucose metabolism change affect cellular physiology including sugar modification on protein, (3) how do neuron and glia talk each other to maintain neuronal activity and brain circuits and (4) identification of blood biomarker for AD. To examine these phenomena, in vivo analysis of changes in brain pathology including Abeta plaques and glial cells using two photon microscopy in various AD animal models was challenged. Also, massive proteomics and bioinformatics were used to examine the specific proteins and genes for AD pathophysiology. To identify blood biomarker for AD, the plasma from PiB-PET positive and negative subjects were collected and analyzed. She has published more than 130 SCI papers and served as editor including Journal of Alzheimer's disease and Experimental Molecular Medicine.

--- Admission is free and all are welcome ---