The Singapore Bioimaging Consortium (SBIC)
presents a seminar

on

"Disruption of adaptor protein 2μ (AP-2μ) in cochlear hair cells impairs vesicle eloading of synaptic release sites and hearing"

Speaker: **Dr Jung Sangyong**
Institute for Auditory Neuroscience
Max Planck Institute
Host : **Dr Han Weiping**
Date : **Monday, 15 February 2016**
Time : **3.00pm – 4.00pm**
Venue : **SBIC Seminar Room**
11 Biopolis Way
Level 2, Helios Building, Singapore 138667
(Please enter via Level 1)

Abstract

Disabling hearing impairment (HI) affects 360 million people worldwide, and prevalence increases with age. So far, no causal treatment is available for its most common form which is resulted from defect in hair cell or spiral ganglion neurons, called sensorineural HI. Active zones (AZs) of inner hair cells (IHCs) indefatigably release hundreds of vesicles per second, requiring each release site to replenish vesicles at tens per second. We report that the endocytic adaptor protein 2μ (AP-2μ) is required for release site replenishment and hearing. We show that hair-cell-specific disruption of AP-2μ slows IHC exocytosis immediately after fusion of the readily releasable pool of vesicles, despite normal abundance of membrane-proximal vesicles and intact endocytic membrane retrieval. Sound-driven postsynaptic spiking was reduced in a use-dependent manner and the altered interspike interval statistics suggested a slowed replenishment of release sites. Sustained strong stimulation led to accumulation of endosome-like vacuoles, fewer clathrin-coated endocytic intermediates and vesicle-depletion of the membrane-distal synaptic ribbon in AP-2μ-deficient IHCs, indicating a further role of AP-2μ in clathrin-dependent vesicle reformation on a timescale of many seconds. Finally, we characterized the interaction of AP-2 with its cargo otoferlin. We propose that binding of AP-2 to otoferlin facilitates replenishment of release sites via speeding clearance of exocytosed material, in addition to a role of AP-2 in endocytic recycling.

About the Speaker

Dr Jung Sangyong received his PhD in neuroscience in 2004 from Department of Physiology and Biophysics, school of medicine, Inha University, Korea. His research topic was "Effects of Caveolin-1 on the Na+-Ca2+ Exchanger Activity in Soma of Rat Cerebellar Purkinje Neuron" that was supervised by Dr Chang Kook Suh. Dr Jung

had been trained as a postdoctoral fellow in Department of Life Science, Pohang University of science and technology (POSTECH), Pohang, Korea since 2005 and was promoted to research assistant professor withinthe same institute in 2007. In POSTECH, research topic was that synaptic adhesion protein, neuroligin-1 (synaptic adhesion molecule) affected synaptic plasticity and the fear memory in amygdala. To study vesicle dynamics, Dr Jung came to Max-Planck-Institute for Experimentelle Medizin in Germany in 2009 with Korea government supported grant. His research topic was the measurement of calcium cooperativity on vesicle release in hippocampal neurons in simple autaptic culture system. He moved to Inner ear lab (present: Institute for Auditory Neuroscience) in 2012. Dr Jung studied ribbon synapse and hearing restoration in deafness mouse models with gene therapy.

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