Alzheimer’s Disease and Synaptic Injury

Professor Edward H. Koo
Department of Medicine and Physiology, National University of Singapore
Department of Neurosciences, University of California, San Diego, USA

Chaired by Prof Dean Nizetic, Lee Kong Chian School of Medicine, NTU

Date: 13 April 2016, Wednesday
Time: 4pm – 5pm
Venue: Learning Studio, Level 1, Experimental Medicine Building
Nanyang Technological University, 59 Nanyang Drive, Singapore 636921

Synopsis

Increasing evidence favours the concept that synaptic injury underlying cognitive deficits accompanies Alzheimer’s disease (AD). The amyloid hypothesis of AD posits that synapses are one of the initial sites of neuronal damage initiated by amyloid β-protein (Aβ), thus placing Aβ in a seminal role in AD pathogenesis. An interesting picture has emerged whereby neuronal activity augments processing of the amyloid precursor protein (APP) to enhance Aβ production and release, which in turn depresses synaptic activity and synaptic plasticity as well as causing synapse loss. However, while a number of plausible pathways have been proposed, the molecular mechanisms of Aβ-induced synaptic injury remain to be clearly elucidated. I will briefly review the activity dependent pathway of Aβ production and an APP-dependent mechanism of Aβ-initiated synaptic damage.