“Novel genes and pathways contributing to Alzheimer’s and related neurodegenerative diseases”

by

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Abstract

Alzheimer’s disease (AD) is an incurable and fatal brain-wasting disorder that causes memory loss and progressive dementia in the elderly. Although aging is the main risk factor for AD, several causal gene mutations and genetic risk factors linked to AD expression have been identified. However, the mechanism of action and significance of these risk factors to AD pathophysiology have yet to be fully explored. Here we present evidence describing known and novel genetic risk factors linked to AD and related tauopathies; including SNX27, SLC25A38 (appoptosin), SORLA, EphA4, Rps23r1 and TREM2. I will discuss their respective contribution to Aβ production and deposition in brain, tau-containing neurofibrillary tangles formation, synaptic failure and cognitive decline. I will also discuss our recent findings supporting appoptosin role as a pro-apoptotic molecule and the contribution of upregulated appoptosin expression to tau metabolism/neurofibrillary tangles formation in tauopathies such as progressive supranuclear palsy (PSP) and AD. In addition, I will present evidence describing new roles for appoptosin and Rps23r1 in synaptic function, plasticity and behavior. Finally, I will discuss novel roles for SNX27 and SORLA/EphA4 in preserving and protecting the structural and functional integrity of synapses against Aβ-mediated synaptoxicity in AD and Down syndrome. I will conclude by presenting new ideas and directions for drug discovery strategies aimed at regulating the expression and function of these novel genes and pathways in AD and tau-related disorders.

Date : 24 March 2017 (Friday)
Time : 4:00 p.m.
Venue : Lecture Theatre C
The Hong Kong University of Science & Technology
Clear Water Bay, Kowloon

(Host faculty: Prof. Jun Xia)

ALL ARE WELCOME!!