

Joint Physiology Lecture Series and Neurobiology/Ageing Programme Seminar Series



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"Managing toxic protein aggregates- how the proteasome communicates with autophagy."

Biography

I received undergraduate degree from National Taiwan University. I did my graduate study at Dr. Ron Evans' lab at Salk Institute and postdoc training at Dr. David Livingston's lab at Dana Farber Cancer Institute. We are primarily interested in the fundamental mechanism that drives physiological aging. We focus on the origin of pathological lesions prevalent in age-associated disease to enable reconstruction of aging in a physiological context and guide the effort toward therapeutic solutions.

Abstract

Accumulation of toxic protein aggregates is a dominant pathological feature in neurodegenerative disease. Although they are commonly ubiquitinated, protein aggregates are not processed by the proteasome. The prevailing model proposes that aggregated proteins are actively transported and concentrated to the inclusion bodies and subsequently processed by autophagy. I will discuss a novel mechanism through which the proteasome and autophagy communicate and coordinate to dispose of toxic protein aggregates.

Date: 13 March 2013 (Wednesday) // Time: 4.00pm // Venue: Centre for Life Sciences Seminar Room 2

Host: A/P Lim Kah Leong, Chairman (Programme's Steering Committee)