College of Science and Engineering

Department of Biomedical Sciences

Seminar

Neuron-glia Energy Metabolic Interactions via Monocarboxylate Transporter1 (MCT1) in CNS Health and Disease

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Date: 28 April 2016 (Thursday)

Time: 12:00nn – 1:30pm (Reception with light sandwiches starts at 11:45am. To facilitate the order of sandwiches, please register through email shulchan2@cityu.edu.hk.)

Venue: B6605, Academic 1, City University of Hong Kong

Language: English

Abstract

The central nervous system (CNS) consumes very high levels of energy, which represents approximately 20% of total amount of energy consumption of the whole organism, while representing only about 2% of body weight. Therefore, alterations of energy metabolism might cause serious neuronal dysfunction, neurodevelopmental defect, and neurodegeneration in certain neurological disorders. Glucose is the essential energy substrate for nerve function. Nonetheless, in certain conditions such as oxygen and glucose deficiency, or during the neonatal period, monocarboxylates, including lactate and ketone bodies, are consumed by the CNS as energy fuels.

Monocarboxylates are co-transported along with protons across the plasma membrane by monocarboxylate transporters 1 to 4 (MCTs1-4). The in vivo distribution and role of MCT1, a major CNS lactate transporter, has been identified by us and other colleagues. Supply of lactate by astroglia to active synapse as an energy fuel has been proposed for a decade as an "astroglia-neuron lactate shuttle hypothesis". Our novel discovery in mouse models, including MCT1 BAC reporter mouse and MCT1 knockout mouse, indicated the "energy metabolic coupling between myelin and axon" via oligodendroglial MCT1, which is necessary for maintaining axonal integrity and neuronal survival implicated in Amyotrophic Lateral Sclerosis pathogenesis. This finding suggested that neuron-glia energy metabolic coupling via MCT1 might be associated with the pathogenesis of many neurological diseases, including Multiple Sclerosis, Alzheimer's disease, and Parkinson's disease. We are now investigating the function of Purkinje neuron MCT1 in the neonatal cerebellum and its potential implication in Autism Spectrum Disorders as well as metabolic interactions between glioma and neuronal cells.

Biography

Dr Youngjin Lee received his Bachelor's and Master's degree from the School of Pharmacy at the Sungkyunkwan University in Korea. After working a few years as a clinical pharmacist, he moved to United States for studying neuroscience at the University of Alabama at Birmingham Medical Center, and he began to do research on glial biology in the central nervous system (CNS) under the guidance of Dr Michael Brenner. After completion of his PhD thesis in 2007, he joined Jeffrey Rothstein's lab in the school of medicine at the Johns Hopkins University for postdoctoral training in 2009, and he has investigated the role of glia in Amyotrophic Lateral Sclerosis pathogenesis before joining the Department of Biomedical Sciences at the City University of Hong Kong in 2015. During graduate- and postdoctoral- training, he significantly contributed to understanding the property and role of glia, including astroglia and oligodendroglia, in health and disease. He was a recipient of the Research Fellowship Award from the Health Fellowship Foundation in Korea (1996), and the W. Barry Wood, Jr. Research Award at the Johns Hopkins University in USA (2013). Dr Lee also was a principal investigator on Development Grant from Muscular Dystrophy Association (2011–2014).

**** All ARE WELCOME ****

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