平成 30 年 4 月 2 日
April 2, 2018

大学院学生各位
To All Graduate Students

平成 **30** 年度

基盤医学特論　開講通知

Information on Special Lecture Tokuron 2018.4-2019.3

Title: Mechanisms underlying regional vulnerability and glial contribution in ALS and FTD

Teaching Staff: Shuo-Chien LING, Ph.D.

Assistant Professor, Department of Physiology

Yong Loo Lin School of Medicine, National University of Singapore

Time and Date: April 20 (Fri) 2018, 18:00-19:30

Room : The Research Institute of Environmental Medicine, North Building, Seminar room N201

Language: English

Abstract

Common genetic loci and pathological signatures have unified two seemingly different adult-onset neurodegenerative diseases, amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD), which affect predominantly the motor system and cognition, respectively. In particular, mutations in TDP-43 and FUS causal for both diseases coupled with the pathological TDP-43 and FUS inclusions present in the neurons and glia of ALS and FTD patients indicate that TDP-43 and FUS dysfunctions trigger ALS and FTD pathogenesis.

Here, we use mouse genetics and genomic methodologies to address two key questions: (1) why certain population of neurons are more susceptible and selectively degenerated, and (2) how glial dysfunctions may contribute to the disease pathogenesis. In the former, we hypothesized that FUS-mediated miRNA deregulation may, in part, accounts for the motor and cognitive deficits observed in ALS and FTD and uncover novel miRNA-RNA relationships that may be underlying the selective motor neuron death. In the latter, we address the physiological functions of TDP-43 in oligodendrocytes and demonstrate that TDP-43 is required for oligodendrocyte survival and functions.

References: Sun, S.***,** Ling, S.-C***.**, et al. (2015), ALS-causative mutations in FUS/TLS confer both gain- and loss-of RNA-processing functions by altered association with SMN and U1-snRNP. ***Nat Commune***, 6:6171.

Ling S.-C**.**, et al. (2010), ALS-associated mutations in TDP-43 increase its stability and promote TDP-43 complexes with FUS/TLS. ***Proc Natl Acad Sci U S A.***, 107: 13318-13323

Contact: Koji Yamanaka, Neuroscience and Pathobiology, RIEM. (Ext: 3867) No registration required.
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