

# “MEGA” Symposium

(jointly organised by DBS, Xiamen University and Tsinghua University)

Wed, 8 February 2017 | 10am — 11.30am | DBS Conference Room 1



*Professor Shengcai Lin finished his B.Sc. at Xiamen University in 1984, and obtained his Ph. D from the University of Texas Medical Center at Dallas in 1991. He received his postdoctoral training at University of California San Diego. He was a principal investigator at the Institute of Molecular and Cell Biology (IMCB) from 1995-2001, and then stayed at Hong Kong University of Science and Technology from 2001-2006. He is currently a professor at the School of Life Sciences, Xiamen University. His recent major achievement is the elucidation of AMPK activation inside the cell as well as its implication in drug discovery.*



*Professor Peng Li finished her B.Sc. at Beijing Normal University in 1987, and obtained her Ph.D. from the University of California at San Diego in 1995. She then received her postdoctoral training at IMCB, Singapore and UT Southwestern Medical Center at Dallas. She was a principal investigator at IMCB from 1997-2002 and an assistant/associate professor in Hong Kong University of Science and Technology from 2003-2005. She is currently a professor at the School of Life Sciences, Tsinghua University and director of the division of life sciences, National Natural Science Foundation of China. She was elected Academician of the Chinese Academy of Sciences in 2015 and elected Fellow of TWAS in 2016. Her major scientific achievement is the elucidation of molecular mechanism and physiological relevance of LD fusion.*

## Mechanism for AMPK activation in glucose sensing

**LIN Sheng-Cai**

Principal Investigator for Regulatory Biology Group, Xiamen University

Metabolic homeostasis is maintained by various cellular sensors, including the master kinases AMPK (AMP-activated protein kinase) and mTORC1. The mechanism for AMPK activation had been mostly studied through biochemical or pharmacological approaches. We found that AXIN, a scaffold protein critical for Wnt signaling, serves as a bridge for the upstream activating kinase LKB1 to form complex with, and activate AMPK. Most surprisingly the lysosomal protein complex v-ATPase-Ragulator, essential for activation of mTORC1, is also required for AMPK activation. Under glucose starvation, the v-ATPase-Ragulator complex becomes accessible to AXIN/LKB1 for AMPK activation. Concurrently, mTORC1 dissociates from the lysosome and becomes inactivated. We have thus revealed a switch between catabolism and anabolism. We have found the signaling route for the anti-diabetic drug metformin in AMPK activation, revealing that metformin depends on the v-ATPase/Ragulator-based lysosomal pathway for AMPK activation and concomitant repression of mTORC1. Most recent work demonstrating that AMPK is a glucose sensor will also be presented.

## The Biology and Mechanism of Lipid Storage

**LI Peng**

Professor, School of Life Sciences, Tsinghua University

Neutral lipids are major energy source stored under energy excess condition and released in the form of FAs under starvation condition. Excessive lipid storage leads to the development of obesity and fatty liver disease. Lipid droplets (LDs) are dynamic subcellular organelles responsible for lipid storage and intracellular lipid homeostasis. We have shown that CIDE family proteins are LD and ER-associated proteins and are important regulatory factors for lipid storage in adipocytes, hepatocytes, mammary epithelial cells and skin sebocytes. CIDE family proteins are highly enriched at LD-LD contact sites (LDCS) and promote atypical form of LD fusion by initiating a directional lipid transfer from smaller to larger LDs. We have identified several other factors that control LD fusion and elucidated their detail biochemical and biophysical mechanism. General implication of lipid metabolism in the development of metabolic diseases will also be discussed.

All are welcome — No registration required