Yan Liu et al., J Neurol Neurophysiol 2014, 5:5
http://dx.doi.org/10.4172/2155-9562.S1.017

OMICS Group Conferences
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3rd International Conference and Exhibition on
Neurology & Therapeutics
September 08-10, 2014 Hilton Philadelphia Airport, USA

Glycogen body metabolism is required for neurons against stresses

Yan Liu and **Yin Wang**
George Washington University, USA

Mammalian cells reserve energy glucose in glycogen in two forms, the commonly known soluble glycogen particles primarily stored in tissues of liver and muscle, and the less known insoluble glycogen granules/bodies (GBs) existing in almost all cells. The GB synthesis is led by muscle glycogen synthase (GS1) activated allosterically by glucose-6-phosphate (G6P) regardless of its inhibitory phosphorylation by GSK3, acting as a deep energy store for cells on demands. Although GBs exist in neurons in few amounts at physical state but are induced rapidly largely by stimulators or stresses that elevate intracellular G6P via increasing glucose uptake or inhibiting glycolysis. Stresses, such as energy deprivation and endoplasmic reticulum perturbation, rapidly induce GB synthesis and reserve for subsequent degradation to provide endogenous glucose and metabolites for neurons' survival and recovery from stresses. The GB degradation takes place in cytosol by a protein assembly that consists at least of four key enzymes, the laforin and malin in a complex, and the glycogenolytic enzymes glycogen debranching enzyme 1 (AGL1) and brain isoform glycogen phosphorylase (GPBB). Deficiency of either the dual phosphatase laforin encoded by EPM2A or the E3 ubiquitin ligase malin encoded by NHLRC1 causes progressive neurodegenerative epileptic Lafora disease (LD) with undegradable pathogenic insoluble GBs called Lafora bodies (LBs) largely accumulated in neurons and most other tissues. Laforin dephosphorylates and malin degrades the GS1 on GBs in concert with GPBB and AGL1 that break glycogen down. Demonstration of molecular mechanisms underlying GB metabolism lays a substantial biochemical base to know LD and develop drugs targeting GB pathogenesis.

Biography

Yan Liu graduated with a PhD in Biochemistry from Dalian Medical University in 1992, China, and was trained as Postdoctorate in the Department of Laboratory and Molecular Medicine at Kagoshima University in Japan in 1998. She continued her studies in Molecular Cell Biology as a Postdoctoral Fellow and as an Adjunct Assistant Professor in the Department of Pathology of Medical School, Ohio State University, since 2001 to 2006. She became a Research Assistant Professor in the Department of Surgery of Medical School, the University of Michigan in 2006. She joined the faculty of Children's National Medical Center in 2013.

ynliu@cnmc.org