SEMINAR

Department of Biological Sciences

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Control of Lipid Droplet growth by CIDE Proteins

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Disruption of lipid homeostasis often results in the development of metabolic disorders including obesity, diabetes, atherosclerosis and fatty liver formation. Lipid droplets (LD) are the major sub-cellular organelle responsible for lipid homeostasis. However, the molecular components that determine LD biogenesis, growth and interactions with other cellular organelles are poorly understood. CIDE family proteins consisting of Cidea, Cideb and Fsp27 (Cidec) are expressed in adipose tissues, liver, kidney and intestine that are closely related to energy homeostasis. Animals with deficiency in Cidea, Cideb, and Fsp27 all display lean phenotypes with higher energy expenditure, and are resistant to diet-induced obesity and insulin resistance. CIDE proteins are LD-associated and their deficiency in adipocytes and hepatocytes results in the accumulation of small sizes LDs and lower lipid storage. We observed that Fsp27 and Cidea are highly enriched at a particular sub-LD location: LD-LD contact sites (LDCS). Once enriched at LDCSs, Fsp27 and Cidea initiate a rapid lipid exchange among contacted LD pairs and a directional lipid transfer from smaller to larger LDs, resulting in the merging of smaller LDs and formation of large LDs. I will discuss our recent progress on the molecular mechanism of Fsp27-mediated LD growth.

Artwork by Ann Nee, DBS, NUS