Institute of Molecular and Cell Biology
A*STAR

SEMINAR ANNOUNCEMENT

We would like to invite you to attend this seminar hosted by A/Prof. Sudipto Roy:

Date: 8 August 2018, Wednesday
Time: 11:00AM – 12:00PM
Venue: Level 3, IMCB Seminar Room 3-46, Proteos, Biopolis

Speaker: Prof. Ming Ma, School of Life Sciences, Southwest University, Chongqing, China
Title: Ciliary mechanisms for cyst formation polycystic kidney disease

Autosomal dominant polycystic kidney disease (ADPKD) is one of the most common human monogenic diseases, affecting over 12 million people worldwide. ADPKD is characterized by defective tissue homeostasis resulting in active remodeling of nephrons and bile ducts to form fluid-filled sacs called cysts. The causal genes *PKD1* and *PKD2* encode transmembrane proteins polycystin 1 (PC1) and polycystin 2 (PC2), respectively. Together, the polycystins localize to the solitary primary cilium that protrudes from the apical surface of most kidney tubule cells and is thought to function as a privileged compartment that the cell uses for signal integration of sensory inputs. It has been proposed that PC1 and PC2 form a receptor-channel complex that detects external stimuli and transmit a local calcium-mediated signal, which may control a multitude of cellular processes by an as-yet unknown mechanism. Genetic studies using mouse models of cilia and polycystin dysfunction have shown that polycystins regulate an unknown cilia-dependent signal that is normally part of the homeostatic maintenance of nephron structure. ADPKD ensues when this pathway is dysregulated by absence of polycystins from intact cilia, but disruption of cilia also disrupts this signaling mechanism and ameliorates ADPKD even in the absence of polycystins. Elucidation of the components of pathway will define superior targets with a high likelihood for translation into therapies for this disease. I will discuss about the ciliary signaling molecules as candidates for the cyst promoting signals.