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DISTINGUISHED SEMINAR

How to fly without wings - from flies to chronic kidney disease

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Date: Friday, 13 September 2024

Time: 16:00 – 17:00

Venue: Conference Room / Zoom
Hybrid
B1F, Faculty of Medicine Bldg. B

Eligibility: Academic Researchers and Students

Register here

The Wnt/ß-catenin system represents an evolutionary highly conserved signaling pathway that is of particular importance for morphogenesis and cell organization during embryogenesis. The system is usually suppressed in adult life, but can be re-activated in organ injury and tissue regeneration. In the absence of Wnt ligands, ß-catenin is subjected to proteasomal degradation to prevent excessive Wnt/ß-catenin signaling. Wnt glycoproteins engage the lipoprotein-related receptor protein 5/6 (LRP5/6)-Frizzled receptor complex leading to ß-catenin stabilization and T-cell factor (TCF)/lymphoid enhancer factor (LEF)-dependent gene expression. Wnt-deficient mice display severe kidney defects at birth. Moreover, Wnt/ß-catenin signaling has been documented to promote kidney fibrosis in chronic kidney diseases (CKD), tissue damage during acute kidney injury and cystic kidney diseases. The Wnt/ß-catenin pathway is tightly regulated e.g. by proteins of the Dickkopf (DKK) family. In particular, DKK3 is released by "stressed" tubular epithelial cells during kidney injury and is associated with short-term risk of kidney function loss after acute kidney injury and during CKD progression. It can be measured in urine using ELISA, where its concentration significantly correlates with subsequent loss of kidney function. Targeting the Wnt/ß-catenin pathway might thus represent a promising therapeutic strategy to prevent kidney injury and associated complications.

Hosted by Institute for the Advanced Study of Human Biology (WPI-ASHBi)

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