

ASHBi SEMINAR

Mechanisms of Necroinflammation and Nephron Loss

Lecturer: **Andreas Linkermann M.D., Ph.D.**

Deputy director of Nephrology and Heisenbergprofessor,
Division of Nephrology, University Hospital Carl Gustav Carus
at the Technische Universität Dresden, Dresden, Germany



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Time **16:00 – 17:10 [JST]**

Venue **Zoom Online Meeting***



*Register via the right QR code

Abstract

Understanding nephron loss is a primary strategy for preventing CKD progression. Death of renal tubular cells may occur by apoptosis during developmental and regenerative processes. However, during AKI, the transition of AKI to CKD, sepsis-associated AKI, and kidney transplantation ferroptosis and necroptosis, two pathways associated with the loss of plasma membrane integrity, kill renal cells. This necrotic type of cell death is associated with an inflammatory response, which is referred to as necroinflammation. Importantly, the necroinflammatory response to cells that die by necroptosis may be fundamentally different from the tissue response to ferroptosis. Although mechanisms of ferroptosis and necroptosis have recently been investigated in detail, the cell death propagation during tubular necrosis, although described morphologically, remains incompletely understood. Here, we argue that a molecular switch downstream of tubular necrosis determines nephron regeneration versus nephron loss. Unraveling the details of this "switch" must include the inflammatory response to tubular necrosis and regenerative signals potentially controlled by inflammatory cells, including the stimulation of myofibroblasts as the origin of fibrosis. Understanding in detail the molecular switch and the inflammatory responses to tubular necrosis can inform the discussion of therapeutic options.

Organizer : Graduate School of Medicine

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

Contact: Prof. Motoko Yanagita

[E-mail] kidney2011@kuhp.kyoto-u.ac.jp

