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Promoting Sustainable Kidney Health: The Asia-Pacific and Beyond

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Organelle Cross Talk on Kidney Cell Fate

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In recent years, the accumulated data of multi-omics analysis has revealed that metabolic disorders in kidney cells contribute to kidney damage and, consequently, to the pathogenesis of kidney disease. It has become a consensus that improving these disorders could be a target for drug development. In particular, alterations in the metabolic product profiles, estimated by metabolome analysis, resulting from metabolic disorders, accompany acute and chronic phenotypic changes in renal cells: functional impairment, adaptive responses, and repair reactions. This alteration ultimately determines kidney cell fate: cell cycle (senescence), cell death, and pro-inflammatory/fibrotic responses. In previous papers, including ours, the mechanisms by which renal metabolic abnormalities occur and how they can be controlled have been focused on. We are studying "organelle stress and organelle crosstalk," which are closely related to metabolic alterations, in order to understand the impact of the link between metabolism and organelle on determining kidney cell fate. Our recent results demonstrated that the rapid decline of kidney function in DKD patients is correlated with lipid metabolic alteration within tubules, which causes tubular organelle stress (mitochondrial stress and endoplasmic reticulum stress) and the alteration of their organelle interaction. These represent a new research field focusing on organelle homeostasis in kidney disease.

Keywords: ER stress, metabolic alteration, organelle contact site, tubular damage, diabetic kidney disease