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Promoting Sustainable Kidney Health: The Asia-Pacific and Beyond

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Session Title : Chronic Kidney Disease 1
Session Topic : Advances in Understanding the Mechanisms and Therapies for Kidney Fibrosis
Date & Time, Place : June 14 (Fri) / 08:30-10:00 / Room 3 (GBR 104-105)

Understanding the Mechanism of Interstitial Fibrosis in the Kidney

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The number of patients with end stage kidney disease (ESKD) are increasing world-wide. While interstitial fibrosis (IF) is a common step for the progression to ESKD, therapeutic options for IF is still limited in clinical settings. Various mechanisms are proposed for the progression of fibrosis. Chronic inflammation is the one of the major player from early stage of kidney injury to ESKD. We have explored the role of inflammation with murine kidney injury models, especially focusing on the association between gut and kidney. In diabetic kidney disease, hyper glycemia induced leaky gut, which allows intestinal bacteria to translocate into circulation and kidney. Then, the bacteria induced systemic inflammation, such as the increased blood levels of IL-17, resulting in worsened kidney injury and fibrosis. We have also focused on the pathophysiological role of metabolites of gut microbiota. D-amino acids are one of the metabolites. Among them, we have highlighted that D-serine and D-alanine has reno-protective effects in acute kidney injury. Both D-serine and D-alanine protected tubular epithelial cells from hypoxia-related cell injury and induced proliferation after hypoxia. The administration of D-serine and D-alanine ameliorated kidney injury after the induction of ischemia/reperfusion in mice.

Based on these results, gut is deeply involved in the pathogenesis of progressive kidney injury. Also, controlling intestinal homeostasis may offer a novel therapeutic approach for kidney injury including fibrosis.

Keywords: