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Promoting Sustainable Kidney Health: The Asia-Pacific and Beyond

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The Injury mechanism and possible treatment options for podocytes in CKD

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It has recently become clear that initial glomerular injury affects glomerular visceral epithelial cells (also called as podocytes), making them important target cells for progression of chronic kidney disease (CKD) and end-stage kidney disease. Podocytes are injured in many human kidney diseases including diabetic nephropathy, minimal change disease, focal segmental glomerulosclerosis, membranous nephropathy, and lupus nephritis. Podocytes serve as the final barrier to urinary protein loss through the formation and maintenance of foot-processes and an interposed slit-diaphragm (SD). Chronic podocyte injury may cause podocyte loss from the glomerular basement membrane (GBM), which leads to glomerulosclerosis. There are reported numerous mechanisms for podocyte loss including detachment related cell death, progtammed apoptotic cell death, programmed non apoptotic cell death, immune-reactive cell death and other type of cell death including mitotic catastrophe-induced cell death. However, the underlying mechanism remains unclear. In this talk, I would like to show the molecular mechanism of podocyte loss and discuss about potential drugs, which may affect podocyte function and integrity, against the mechanism.

Keywords: CKD, podocyte, podocyte injury, glomerulosclerosis