Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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# The Role of Urine-Derived Stem Cells in Diabetes-Induced Kidney Disease(mesangial cell) Models

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**Objectives :** Under hyperglycemic conditions, mesangial ((MES) cells promote fibrotic responses through excessive extracellular matrix (ECM) accumulation, enhanced inflammatory signaling, and activation of the TGF-β/Smad and Akt/mTOR/FOXO pathways. UDSCs are stem cells with high renal cell affinity and exhibit Klotho-mediated antifibrotic and antioxidative properties. In this study, we investigated co-culture of MES cells with UDSCs under hyperglycemic conditions might have protection as a novel therapeutic strategy for diabetic nephropathy.
**Methods :** Urine-derived stem cells (UDSCs) were obtained through American Type Culture Collection (ATCC). Human glomerular MES cells were cultured, and high-glucose conditions were established to model the hyperglycemic environment of diabetic nephropathy. MES cells were then co-cultured with UDSCs under high-glucose conditions. Pathological and molecular changes were assessed by analyzing key fibrotic signaling pathways, including SOX9, transforming growth factor-β (TGF-β), and Smad2/3, as well as activation of the Akt/mTOR/FOXO pathway (Akt, mTOR, FOXO) and the MAPK signaling pathway (Raf, ERK).
**Results :** MES cells exhibited characteristic spindle-shaped morphology and expressed α-SMA and fibronectin, confirming mesangial identity, while UDSCs showed a typical mesenchymal stem cell phenotype with high expression of CD73, CD90, CD105, CD146, SSEA-4, and CD24/CD133. Exposure of MES cells to high glucose established a reproducible hyperglycemia-induced injury model, characterized by structural alteration, hyperproliferation, and fibrotic changes, with 10 mM glucose identified as the optimal condition. Fig 1 UDSC co-culture under high-glucose conditions preserved MES cell morphology, suppressed abnormal proliferation and ECM accumulation, restored Akt/mTOR/FOXO protective signaling, and inhibited TGF-β/Smad and Raf/ERK fibrotic pathways Fig2
**Conclusions :** Notably, we demonstrate that UDSCs effectively attenuate MES cell injury by suppressing ECM accumulation, downregulating the TGF-β/Smad and Akt/mTOR pathways, and activating protective signaling mediated by FOXO and Klotho. These findings provide compelling scientific evidence supporting UDSCs as a novel and promising cell-based therapeutic candidate for DN.

Figure 1.png

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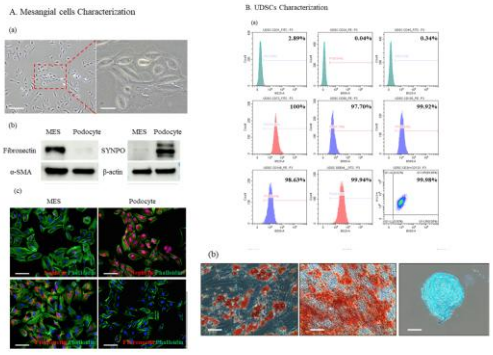

Figure 1-1 Characterization of MES cells (A) and UDSCs (B)

LM at 40x and 200x magnification. Scale bars: 50 μm and 10 μm,
IF staining Nephrin and Fibronectin (Red), Phalloidin (Green), DAPI(Blue)
at 100x magnification. Scale bar: 20 μm.

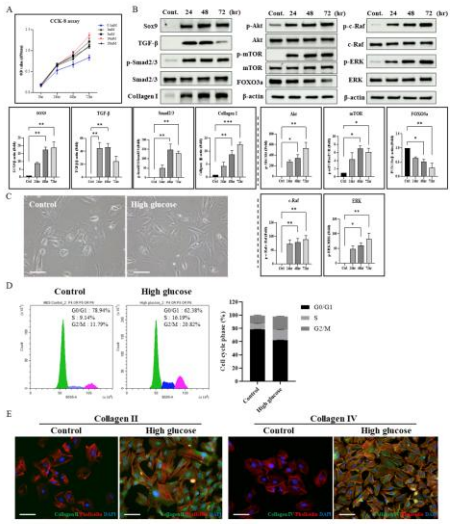

Figure 1-2. Establishment of a high glucose-induced mesangial cell injury model.

Figure 1.png