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In Vitro Analysis of Klotho-Expressing Urine-Derived Stem Cells for Treating High Glucose-Induced Mesangial Cell Dysfunction

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Objectives : Diabetic nephropathy (DN) is a leading cause of chronic kidney disease (CKD), characterized by mesangial cell hypertrophy and fibrosis resulting from prolonged exposure to high glucose. Although urine-derived stem cells (UDSCs) have demonstrated therapeutic potential in various renal injury models, their role in DN—particularly in combination with the antioxidant protein Klotho—remains incompletely defined. This study investigated the protective effects and underlying signaling mechanisms of Klotho-expressing UDSCs on high glucose-induced mesangial cell hypertrophy and fibrosis in an in vitro model.
Methods : Human mesangial cells (HMCs) were cultured under high glucose (HG, 30 mM) conditions to simulate the diabetic milieu. UDSCs were isolated from human urine and transfected with a Klotho-expressing plasmid. HMCs were subsequently co-cultured with either wild-type UDSCs or Klotho-expressing UDSCs. Cell proliferation, hypertrophic changes, and expression of fibrotic markers (TGF-β1, fibronectin, and collagen IV) were assessed. Activation of the PI3K/Akt and MAPK signaling pathways was analyzed to elucidate the mechanism of action.
Results : HG conditions significantly increased HMC proliferation and hypertrophy, accompanied by upregulation of key fibrotic markers. Co-culture with Klotho-expressing UDSCs effectively attenuated these pathological changes, demonstrating superior inhibition of mesangial expansion and extracellular matrix (ECM) accumulation compared with wild-type UDSCs alone. Specifically, Klotho-expressing UDSCs significantly reduced protein levels of TGF-β1, fibronectin, and collagen IV in HG-induced HMCs. These renoprotective effects were associated with suppression of HG-induced phosphorylation of Akt and ERK1/2, indicating modulation of PI3K/Akt and MAPK signaling. Secretion of Klotho into the culture medium was confirmed, highlighting its
Conclusions : Klotho-expressing UDSCs effectively mitigate high glucose-induced mesangial hypertrophy and fibrosis by modulating the PI3K/Akt and MAPK signaling pathways. These findings suggest that Klotho augmentation of UDSCs represents a promising cell-based therapeutic strategy for diabetic nephropathy.