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Effective simultaneous inhibition of aging and fibrosis using anti-senescence therapy in renal tubular cells

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Objectives : Effective treatment for chronic kidney disease remains a challenge due to various obstacles. Recently, studies on aging in glucose-induced kidney injury have been conducted, leading to investigations of anti-senescence therapy as a potential approach to prevent renal damage. This study aims to report the effects of the anti-senescence agent, trametinib, on glucose-induced kidney injury.
Methods : The study was conducted using HK-2 cells, a human proximal tubular cell line. Changes in oxidative stress, epithelial-to-mesenchymal transition (EMT), and senescence markers were evaluated after glucose treatment, and the effects of trametinib treatment on these changes were compared.
Results : Glucose-treated HK-2 cells exhibited fiboblast-like changes in morphology, with increased expression of senescence markers such as SA-β-gal and Sudan Black B staining. However, treatment with trametinib effectively preserved the epithelial morphology and significantly reduced the expression of senescence markers. Glucose increased the protein expression of fibronectin, p16, and CDK4 while decreasing the expression of E-cadherin. These alterations were effectively suppressed by trametinib treatment. Additionally, ROS levels measured by DCFDA staining were elevated after glucose treatment but were reduced by trametinib. Glucose treatment also enhanced the expression of senescence-associated secretory phenotype markers, including CDKN2a, TNF-α, and TGF-β, while trametinib successfully inhibited these changes.
Conclusions : Glucose induces ROS-mediated EMT and senescence in human proximal tubular cells. Trametinib effectively suppresses these processes, suggesting its potential as a therapeutic option for glucose-induced kidney injury.