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PM_{2.5} induced Phenotype Transition of Renal Tubular Epithelial Cells via Oxidative Stress

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Objectives : The incidence and prevalence of chronic kidney disease (CKD) are rising worldwide. Recently, exposure to fine particulate matter, particularly PM_{2.5}, has been identified as a risk factor for CKD development and progression. However, it remains unclear whether PM_{2.5} exposure directly contributes to kidney disease. Epithelial-to-mesenchymal transition (EMT) of renal tubular cells is recognized as an early mechanism of renal injury. This study aims to investigate whether PM_{2.5} induces EMT in renal tubular epithelial cells through oxidative stress.

Methods : PM_{2.5} samples collected from Ulaanbaatar, Mongolia, were dissolved in DMSO using sonication. Normal rat kidney (NRK) tubular epithelial cells were treated with PM_{2.5} (2 & 5 µL/mL). EMT was evaluated by assessing morphological changes and analyzing the expression levels of E-cadherin, α-SMA, and vimentin via western blotting and immunostaining. Reactive oxygen species (ROS) generation was measured using DCF-DA and MitoSox staining. Additionally, RNA-seq was performed to identify differentially expressed genes in PM_{2.5}-exposed NRK cells.

Results : PM_{2.5} (2 and 5 µL/mL) did not affect LDH release or cell proliferation for up to 48 hours. However, PM_{2.5} induced EMT in NRK cells, as evidenced by morphological changes, decreased E-cadherin expression, and de novo expression of α-SMA and vimentin. PM_{2.5} also led to increased ROS production, as indicated by enhanced DCF-DA and Mito-Sox staining. RNA-seq analysis revealed significant alterations in gene expression related to EMT (16.7%), adipokine (15.9%), apoptosis (13.7%), and oxidative stress (12.8%). Among the differentially expressed genes, Lipocalin-2, Interleukin-11, and hyaluronan synthase-2 exhibited the highest fold changes (2.7-folds, 2.5-folds, and 2.0-folds, respectively) in PM_{2.5}-exposed NRK cells compared to control cells.

Conclusions : These findings suggest that PM_{2.5} induces EMT and oxidative stress in renal tubular epithelial cells, highlighting a potential causal link between fine dust and kidney disease. Furthermore, phenotype transition and oxidative stress may serve as therapeutic targets for PM_{2.5}-induced kidney disease.