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PM2.5 induced Phenotype Transition of Renal Tubular Kidney Cells via Oxidative Stress

Hyun-Jung Kang, **Dal-Ah Kim**, Bo-Kyeong Park, Duk-Hee Kang
Department of Internal Medicine-Nephrology, Ewha Womans University School of Medicine, Korea, Republic of

Objectives: The incidence and prevalence of chronic kidney disease (CKD) are increasing worldwide. Recently, exposure to air pollution, especially particulate matter$_{2.5}$ ($PM_{2.5}$) was newly identified to be a potential risk factor for CKD, however there are no studies on whether exposure to $PM_{2.5}$ is a direct cause of CKD occurrence and exacerbation. Epithelial-to-mesenchymal transition (EMT) of tubular cells is one of the early mechanisms of progression of renal disease. Therefore, the identification of association between fine dust and EMT may allow the revealing casualty toward CKD.

Methods: Fine dust (Ulaanbaatar, Mongolia) collected by $PM_{2.5}$ filter was dissolved in DMSO by sonication. Renal tubular kidney cells (NRK) were treated with dissolved $PM_{2.5}$ (2 & 5 μL/mL). EMT was evaluated by morphological changes of NRK cells and the expressions of E-cadherin, α-SMA, and vimentin after the stimulation with $PM_{2.5}$ and TGF-β (5 ng/mL) by WB and immunostaining. ROS generation was assessed by DCF-DA and MitoSox staining. RNA-seq analysis (Ebiogen, Korea) was performed to investigate which upregulated/downregulated-genes are associated with $PM_{2.5}$-induced phenotype transition in NRK cells.

Results: 2 and 5 μL/mL $PM_{2.5}$ did not alter LDH release and cell proliferation up to 48 hours of exposure. $PM_{2.5}$ induced EMT of NRK cells assessed by morphologic changes associated with a decreased E-cadherin expression and de-novo expression of α-SMA and vimentin. $PM_{2.5}$ also increased DCF-DA and Mito-Sox staining. RNA-seq analysis demonstrated the differences in gene expression related to EMT (16.7%), adipokine (15.9%), apoptotic process (13.7%), and oxidative stress (12.8%). Among them, lipocalin 2 (LCN2), Interleukin-11 (IL-11), and hyaluronan synthase 2 (HAS2) expression showed the highest fold difference (2.7-folds, 2.5-folds, and 2.0-folds, respectively) between control and $PM_{2.5}$-treated NRK cells.

Conclusions: This data suggest that exposure to $PM_{2.5}$ induced EMT and oxidative stress in NRK cells, which may suggest the causative role of fine dust exposure and the development of renal disease.