Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
KSN SEOUL, KOREA 2026
The 46th Annual Meeting of the Korean Society of Nephrology
THE KOREAN SOCIETY OF NEPHROLOGY
Jun 11 (Thu) – 14 (Sun), 2026
COEX, Seoul, Korea

Abstract Type : Poster exhibition
Abstract Submission No.: A-0592
Abstract Topic : Basic Research

Particulate Matter Induces epithelial-to-mesenchymal transition in Renal Tubular Epithelial Cells via Oxidative and ER Stress Pathways

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Objectives : Particulate matter (PM) exposure has emerged as a potential risk factor not only for respiratory and cardiovascular diseases but also for kidney injury. However, the mechanisms linking PM exposure to renal pathophysiology remain poorly understood. Oxidative stress, endoplasmic reticulum (ER) stress, and epithelial-to-mesenchymal transition (EMT) are key mechanisms involved in renal injury. This study investigated whether PM induces EMT in renal tubular epithelial cells through oxidative and ER stress–related pathways.
Methods : Artificial particulate matter (APM; Korea Institute of Toxicology) was dissolved in DMSO. Normal rat kidney (NRK) tubular epithelial cells were exposed to APM to assess cytotoxicity, ROS generation, and the expression of oxidative stress, ER stress, NLRP3 inflammasome, and EMT-related proteins. Antioxidants (N-acetylcysteine, apocynin) and ER stress inhibitors (TUDCA, 4-PBA) were used for mechanistic analyses. In vivo, Sprague-Dawley rats inhaled APM for consecutive five days, and kidney tissues were analyzed for renal injury and molecular markers.
Results : APM exposure did not significantly affect LDH release or cell proliferation at concentrations up to 5 μg/mL but induced cytotoxicity at 10 μg/mL. APM markedly increased NOX4-dependent ROS generation (DCF-DA and MitoSOX staining), H_2O_2 production, and NADPH oxidase activity, and upregulated ER stress markers (GRP78, ATF4, ATF6, and CHOP). These changes induced EMT in NRK cells, characterized by decreased E-cadherin and increased α-SMA and fibronectin expression. Antioxidant pretreatment suppressed NOX4 expression, NLRP3 inflammasome (NLRP3, ASC, and cleaved caspase-1), and EMT marker alterations, while ER stress inhibition attenuated EMT. In vivo, APM inhalation increased ED-1–positive macrophage infiltration, nitrotyrosine expression, ER stress marker GRP78, and NLRP3 inflammasome components, accompanied by reduced antioxidant enzymes (catalase, GPx1, and SOD2).
Conclusions : APM induces EMT in renal tubular epithelial cells through NOX4-dependent ROS production and ER stress signaling, leading to NLRP3 inflammasome activation and suggesting that PM exposure may initiate inflammatory and fibrogenic responses in the kidney.