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Particulate Matter induces Epithelial-to-Mesenchymal Transition by Oxidative and Endoplasmic Reticulum Stress in Normal Rat Kidney Epithelial Cells

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Objectives : Particulate matter (PM) exposure has been linked to respiratory, cardiovascular, and kidney diseases. However, its direct role in kidney disease remains unclear. Oxidative stress, endoplasmic reticulum (ER) stress, and epithelial-to-mesenchymal transition (EMT) are key mechanisms for renal injury. This study investigated the effects of artificially manufactured PM (APM) on renal tubular epithelial cells.
Methods : APM (provided by the Korea Institute of Toxicology) was dissolved in DMSO via sonication. Normal rat kidney epithelial (NRK) cells were treated with APM at 1, 2, and 5 μg/mL. Cytotoxicity and proliferation were assessed using LDH release and MTS assay. EMT was evaluated by morphological changes and expression of EMT markers (E-cadherin, α-SMA, and fibronectin) using western blotting, qPCR, and immunostaining. Reactive oxygen species (ROS) generation was measured using DCF-DA and MitoSOX staining. Antioxidants [N-acetylcysteine (NAC), 20 mM; apocynin, 100 uM] and ER stress inhibitors [tauroursodeoxycholic acid (TUDCA), 1 mM, 4-phenylbutyric acid (4-PBA), 5 mM] were used to examine their effects on EMT, NOX1/4 expression, and NLRP3 inflammasome activation in APM-exposed NRK cells at 24 or 48 hours.
Results : APM (1, 2, and 5 μg/mL) exposure for up to 24 hours did not alter LDH release or cell proliferation. APM induced EMT of NRK cells from 2 μg/mL shown as morphological alterations, decreased E-cadherin expression, and de novo expressions of α-SMA and fibronectin. Treatment with antioxidants (NAC and apocynin) and ER stress inhibitors (TUDCA and 4-PBA) mitigated APM-induced EMT, which was accompanied with reductions in NOX1/4 expression and NLRP3 inflammasome activation (NLRP3, ASC, and cleaved caspase-1) at 24 and 48 hours.
Conclusions : These findings suggest that APM induces EMT via oxidative and ER stress in NRK cells, highlighting a potential mechanism for air pollution-induced kidney disease.