THE KOREAN SOCIETY OF NEPHROLOGY
APCN & KSN 2024
June 13(Thu) - 16(Sun), 2024 Coex, Seoul, Korea
Promoting Sustainable Kidney Health: The Asia-Pacific and Beyond

Abstract Submission No.: A-1179

Particulate Matter Exacerbates Kidney Damage by Increasing the CSF2, Inflammasomes, and Senescence

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Objectives : Air pollution is a widely recognized public health issue. The main components of air pollutants include particulate matter (PM), which comprises suspended solid or liquid particles and is a crucial indicator of air pollution. While the harmful effects of PM on the respiratory system are well-studied, the toxic mechanisms affecting the kidneys remain unclear. In this study, our main objective is to investigate the mechanisms underlying renal damage caused by exposure to PM.
Methods : We have addressed PM's kidney health risk using Next Generation Sequencing (NGS) transcriptomics datasets. The NGS results of PM exposure by Ingenuity Pathway Analysis (IPA) and Gene Set Enrichment Analysis (GSEA) compared with control. Furthermore, the transcriptional profiles of the CSF family in kidney tissues of chronic kidney disease (CKD) patients in the Gene Expression Omnibus (GEO) data repository were investigated. In the animal experiments, mice were situated close to busy urban roads for a realistic 3 months of exposure to ambient PM. The effects of PM in kidney cells were analyzed by western blotting.
Results : The NGS results using IPA and GSEA stimulation indicated that the CSF2 was the No. 1 putative candidate to be activated in response to PM exposure. We found that PM increased CSF2 expression in kidney cells. Furthermore, NLRP3 and NLRP6 inflammasomes were activated in kidney cells treated with PM. PM increased the senescence- and autophagy-related proteins. In a real-world of whole-body exposure to PM model for 3 months, PM in urban ambient air can affect kidney function and increase cystatin C and CSF2 in serum. PM groups induced inflammasomes-, senescence- and fibrosis-related proteins in kidney tissues.
Conclusions : These findings suggest that air pollution in urban areas may cause nephrotoxicity by increasing CSF2 and senescence and activating inflammasomes.