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# Particulate Matter Induces Early Renal Inflammation and Inflammasome Activation in Normal and CKD Rat Kidneys

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**Objectives :** Exposure to particulate matter (PM) has been associated with various systemic diseases, including chronic kidney disease (CKD). Although epidemiological studies suggest that PM exposure increases the risk of kidney dysfunction, the mechanisms linking PM exposure to renal injury remain poorly understood. In particular, whether PM can directly trigger early inflammatory and stress responses in normal kidneys has not been clearly established. This study investigated the effects of artificially manufactured PM (APM) on renal inflammation, oxidative stress, and inflammasome activation in normal rats and in a unilateral ureteral obstruction (UUO) model of CKD.
**Methods :** Male Sprague-Dawley rats were divided into four groups: normal control, APM exposure (5 mg/kg via intratracheal instillation administered three or five times), UUO, and UUO+APM. Renal injury was evaluated by measuring blood urea nitrogen (BUN), proteinuria, and renal histological changes, including macrophage marker ED-1 expression. Protein expression of endoplasmic reticulum (ER) stress markers (GRP78, ATF4, CHOP), inflammasome components (NLRP3, ASC, and cleaved caspase-1), oxidative stress markers (nitrotyrosine, SOD2, catalase, and GPx1), and fibrosis markers (F4/80, α-SMA, and osteopontin) was analyzed in kidney tissues at 9 or 14 days after APM exposure.
**Results :** In normal rats, APM exposure induced ER stress signaling, inflammasome activation, and oxidative stress imbalance, accompanied by increased expression of macrophage marker F4/80 and osteopontin. In UUO rats, renal injury was characterized by increased BUN levels, tubulointerstitial fibrosis, and upregulation of ER stress markers and inflammasome components. Exposure to APM further aggravated renal inflammation and macrophage infiltration, with increased ED-1 expression at both 9 and 14 days and enhanced expression of NLRP3 inflammasome and fibrosis markers at 14 days.
**Conclusions :** APM exposure induces renal inflammatory signaling, ER stress activation, oxidative stress imbalance, and NLRP3 inflammasome activation in both normal and CKD rat kidneys, suggesting that particulate matter may initiate early renal injury and exacerbate kidney damage in CKD.