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Airborne particulate matter exposure induces renal tubular cell injury in vitro: The role of vitamin D signaling and the renin angiotensin system

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Objectives: Exposure to air pollution can interfere hormonal systems including the vitamin D endocrine system. This study examined the effects of airborne particulate matter (PM) on renal tubular cell injury *in vitro* and explPored the mechanisms underlying its effects.

Methods: Human proximal tubular epithelial (HK-2) cells were treated with PM with or without $1,25(OH)_2D_3$ (paricalcitol, 10 nMP) for 48 h. The dose- and time-dependent cytotoxicity of PM with or without paricalcitol was examined by cell counting kit-8 assay. Cell oxidative stress was assessed using commercial enzyme-linked immunosorbent assay kits. The protein expressions of vitamin D receptor (VDR), cytochrome P450(CYP)27B1, CYP24A1, renin, angiotensin converting enzyme (ACE), angiotensin II type 1 receptor (AT1), nuclear factor erythroid 2-related factor 2 (Nrf2), nuclear factor-kB (NF-kB) p65, tumor necrosis factor (TNF)-α, and interleukin (IL)-6 were determined.

Results: PM exposure decreased HK-2 cell viability in a dose- and time-dependent manner. The activities of superoxide dismutase and malondialdehyde in HK-2 cells increased significantly in the PM exposure group. PM exposure decreased VDR and Nrf2, while increasing CYP27B1, renin, ACE, AT1, NF-kB p65, TNF-α, and IL-6. The expressions of VDR, CYP27B1, renin, ACE, AT1, and TNF-α were all reversed by paricalcitol treatment. Paricalcitol also restored the cell viability of PM-exposed HK-2 cells.

Conclusions: Our findings indicate that exposure to PM induces renal tubular cell injury, concomitant with the alteration of vitamin D endocrine system and renin angiotensin system. Vitamin D could attenuate kidney damage following PM exposure by suppressing the renin angiotensin system and by inhibiting inflammatory response in part.