PARTICULATE AIR POLLUTION AND INFLAMMATORY MARKERS

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Background and Aims: The inflammatory effect of fine particulate matter ($PM_{2.5}$) is one of the potential mechanisms linking PM pollution and cardiopulmonary outcomes. We examined the association between individual level 24-hour $PM_{2.5}$ concentration and blood inflammatory markers in a community dwelling sample.
Methods: We used a Personal DataRam (pDR) to measure 24-hour individual-level real-time $PM_{2.5}$ exposures in 105 middle-aged nonsmokers. Two blood samples were collected from each participant, one immediately before and one immediately after the 24-hour study period. Concentrations of several inflammation markers were assessed and averaged. Linear regression models were used to assess the association between mean 24-hour $PM_{2.5}$ exposure and the mean inflammation markers. Age, race, gender, relative humidity, temperature, and participant's chronic disease status were adjusted for in the regression models.
Results: The participants (mean [SD] age: 56 [8] years) tended to be female (60%) and white (74%). The 24-hour mean (SD) personal $PM_{2.5}$ concentration was 14.33 (14.60) $\mu g/m^3$. We did not observe significant associations between 24-hour mean $PM_{2.5}$ exposure and mean inflammation markers. The regression coefficients (SE) per 10 $\mu g/m^3$ increases in $PM_{2.5}$ were: -0.02 (0.02) g/dL for albumin, 81 (217) ng/mL for C-reactive protein (CRP), -0.56 (0.13) pg/mL for interleukin-1 α (IL-1 α), -0.28 (0.30) pg/mL for interleukin-1 β (IL-1 β), -19 (56) pg/mL for macrophage migration inhibitory factor (MIF), -1.66 (5.37) pg/mL for tumor necrosis factor-α (TNFα), 100 (73) pg/mL for tumor necrosis factor soluble receptor I (TNFsRI),and 0.02 (0.09) x $10^3/mm^3$ for white blood cell count (WBC), respectively (all p-values > 0.05).
Conclusion: Currently low levels of 24-hour mean $PM_{2.5}$ exposure are not associated with blood inflammation markers. More studies on the chronic exposure and inflammatory responses are needed to elucidate the sub-acute and chronic effects of PM on systemic inflammation as a mechanistic link between PM and cardiopulmonary disease.