



## Association of Biomarkers of Systemic Inflammation with Organic Components and Source Tracers in Quasi-Ultrafine Particles

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The evidences regarding the air pollutant components and their sources responsible for associations between particle mass concentrations and human cardiovascular outcomes are still not adequate. In one of our previous investigations, we demonstrated the associations between circulating biomarkers of inflammation and mass concentrations of quasi-ultrafine particles  $\leq 0.25 \mu\text{m}$  in aerodynamic diameter ( $\text{PM}_{0.25}$ ) in a panel cohort study of 60 elderly subjects with coronary artery disease living in the Los Angeles Basin. The objective of our present study is to reassess the biomarker associations with  $\text{PM}_{0.25}$  using new particle composition data. Plasma interleukin-6 (IL-6) and soluble tumor necrosis factor- $\alpha$  receptor II (sTNF-RII) were used as the weekly biomarkers of inflammation ( $n=578$ ). Exposures included indoor and outdoor community  $\text{PM}_{0.25}$  constituents [polycyclic aromatic hydrocarbons (PAHs), hopanes,  $n$ -alkanes, organic acids, water-soluble organic carbon, and transition metals]. We analyzed the relation between biomarkers and exposures with mixed-effects models adjusted for potential confounders.

Indoor and outdoor PAHs (low-, medium-, and high-molecular-weight PAHs), followed by hopanes (vehicle emissions tracer), were positively associated with biomarkers, but other organic components and transition metals were not. sTNF-RII increased by 135 pg/mL [95% confidence interval (CI), 45–225 pg/mL], and IL-6 increased by 0.27 pg/mL (95% CI, 0.10–0.44 pg/mL) per interquartile range increase of 0.56 ng/m<sup>3</sup> outdoor total PAHs. Two-pollutant models of  $\text{PM}_{0.25}$  with PAHs showed that nominal associations of IL-6 and sTNF-RII with  $\text{PM}_{0.25}$  mass were completely confounded by PAHs. Vehicular emission sources estimated from chemical mass balance models were strongly correlated with PAHs ( $R=0.71$ ). We conclude that traffic emission sources of organic chemicals represented by PAHs are associated with increased systemic inflammation and explain the associations with quasi-ultrafine particle mass.