Abstract

Particulate matter (PM)-associated metals can contribute to adverse cardiopulmonary effects following exposure to air pollution. The aim of this study was to investigate how variation in the **composition** and size of ambient PM collected from two distinct regions in Mexico City relates to toxicity differences. Male Wistar Kyoto rats (14 wk) were intratracheally instilled with chemically characterized PM10 and PM2.5 from the north and PM10 from the south of Mexico City (3 mg/kg). Both water-soluble and acid-leachable fractions contained several metals, with levels generally higher in PM10 South. The insoluble and total, but not soluble, fractions of all PM induced pulmonary damage that was indicated by significant increases in neutrophilic inflammation, and several lung injury biomarkers including total protein, albumin, lactate dehydrogenase activity, and γ-glutamyl transferase activity 24 and 72 h postexposure. PM10 North and PM2.5 North also significantly decreased levels of the antioxidant ascorbic acid. Elevation in lung mRNA biomarkers of inflammation (tumor necrosis factor [TNF]-α and macrophage inflammatory protein [MIP]-2), oxidative stress (heme oxygenase [HO]-1, lectin-like oxidized low-density lipoprotein receptor [LOX]-1, and inducibile nitric oxide synthase [iNOS]), and thrombosis (tissue factor [TF] and plasminogen activator inhibitor [PAI]-1), as well as reduced levels of fibrinolytic protein tissue plasminogen activator (tPA), further indicated pulmonary injury following PM exposure. These responses were more pronounced with PM10 South (PM10 South > PM10 North > PM2.5 North), which contained higher levels of redox-active transition metals that may have contributed to specific differences in selected lung gene markers. These findings provide evidence that surface chemistry of the PM core and not the water-soluble fraction played an important role in regulating in vivo pulmonary toxicity responses to Mexico City PM.