

# PARTICULATE AIR POLLUTION, BLOOD COAGULATION AND FIBRINOLYTIC FACTORS

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**Background and Aims:** Whether fine particulate matter (PM<sub>2.5</sub>) affects clinical thrombosis remains controversial, whereas an association between PM<sub>2.5</sub> and coronary heart disease (CHD) has been reported more consistently. We therefore investigated the cross-sectional association between individual-level short-term PM<sub>2.5</sub> exposure and blood hemostatic factors--blood markers of coagulation and thrombosis potential.

**Methods:** We used a Personal DataRam (pDR) to measure 24-hour individual-level real-time PM<sub>2.5</sub> 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 hemostatic factors were assessed and averaged. Linear regression models were used to assess the association between mean 24-hour PM<sub>2.5</sub> exposure and the mean hemostatic factors. 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<sub>2.5</sub> concentration was 14.33 (14.60) µg/m<sup>3</sup>. We did not observe significant associations between 24-hour mean PM<sub>2.5</sub> exposure and mean hemostatic factor levels. The regression coefficients (SE) per 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> were: -0.13 (0.78) % for antithrombin III, 1.95 (2.97) % for factor VIII, -2.79 (3.88) mg/dL for fibrinogen, 0.56 (0.54) IU/mL for plasminogen activator inhibitor, 0.06 (0.19) ng/mL for tissue plasminogen activator, and -0.13 (0.15) µg/mL for D-Dimer (all p-values > 0.05).

**Conclusion:** Low levels of 24-hour mean levels of PM<sub>2.5</sub> exposure were not associated with blood hemostatic factors. More studies on chronic PM exposure and hemostatic factors are needed to elucidate their role in the pathophysiologic effects of air pollution on deep vein thrombosis and CHD.