EXPOSURE TO AMBIENT FINE PARTICULATE MATTER ALTERS CEREBRAL BLOOD FLOW IN THE ELDERLY: THE MOBILIZE BOSTON STUDY

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Background and Aims: Previous studies suggest that short-term elevations in ambient fine particulate matter (PM$_{2.5}$) may increase arterial blood pressure. However, the consequences of these systemic effects on the tightly-regulated cerebral circulation have not been examined. Accordingly, we evaluated the association between PM$_{2.5}$ and cerebral hemodynamics in a cohort of community-dwelling seniors.

Methods: We repeatedly assessed cerebral hemodynamics at rest and in response to blood pressure changes associated with standing from a seated position according to a standardized protocol in 424 participants (757 measurements) from the MOBILIZE Boston Study. We measured blood flow velocity in the middle cerebral artery using transcranial Doppler ultrasound and mean arterial blood pressure (MAP) non-invasively with a Finapres device. We calculated cerebral vascular resistance as the ratio of MAP to blood flow velocity. PM$_{2.5}$ levels were measured at a nearby monitoring site. We used linear mixed effects models with random subject intercepts to evaluate the association between hemodynamic parameters and mean PM$_{2.5}$ levels 1 to 28 days earlier adjusting for age, race, medical history, meteorologic covariates, day of week, temporal trends, and season.

Results: An interquartile range increase (3.0 µg/m$^3$) in mean PM$_{2.5}$ levels over the previous 28 days was associated with a 6.9% (95% confidence interval [CI]: 2.3%, 11.8%; p=0.003) higher cerebral vascular resistance and a 6.7% (95% CI: 3.4%, 9.9%; p<0.001) lower cerebral vascular blood flow at rest. Results at shorter PM$_{2.5}$ averaging times were qualitatively similar but weaker. Upon standing from a seated position MAP, cerebral vascular blood flow and resistance decreased, as expected. These responses were not associated with PM$_{2.5}$ levels.

Conclusions: In this cohort of community-dwelling seniors, exposure to PM$_{2.5}$ was associated with alterations in cerebral blood flow. If replicated, these novel findings suggest a mechanism by which ambient air pollution might increase the risk of acute cerebrovascular events.