PM stands for particulate matter (also called particle pollution): the term for a mixture of solid particles and liquid droplets found in the air. Some particles, such as dust, dirt, soot, or smoke, are large or dark enough to be seen with the naked eye. Others are so small they can only be detected using an electron microscope.

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Particle pollution includes:

PM10 : inhalable particles, with diameters that are generally 10 micrometers and smaller; and

PM2.5 : fine inhalable particles, with diameters that are generally 2.5 micrometers and smaller.

Recent epidemiological research has shown that exposure to fine particulate pollution (PM2.5) is associated with a reduction in cognitive function in older adults. However, primary evidence comes from high-income countries, and no specific studies have been conducted in low and middle-income countries where higher air pollution levels exist.

To estimate the association between the exposure to PM2.5 and cognitive function in a nationally representative sample of older Mexican adults and the associated effect modifiers.

Data for this study were taken from the National Survey of Health and Nutrition in Mexico carried out in 2012. A total of 7986 older adults composed the analytical sample. Cognitive function was assessed using two tests: semantic verbal fluency and three-word memory. The annual concentration of PM2.5 was calculated using satellite data. Association between exposure to PM2.5 and cognitive function was estimated using two-level logistic and linear regression models.

In adjusted multilevel regression models, each 10 μ g/m3 increase in ambient PM2.5 raised the odds of a poorer cognitive function using the three-word memory test (OR = 1.37, 95% CI: 1.08, 1.74), and reduced the number of valid animal named in the verbal fluency test (β = -0.72, 95% CI: -1.05, -0.40). Stratified analyses did not yield any significant modification effects of age, sex, indoor pollution, urban/rural dwelling, education, smoking and other factors.

This study supports an association between exposure to PM2.5 concentrations and cognitive function in older adults. This is particularly relevant to low- and middle-income countries, which are marked by a rapid growth of their aging population and high levels of air pollution ¹⁾.

Air pollution is associated with the increased risk of metabolic syndrome. In this study, we performed inhalation exposure of mice fed normal chow or a high-fat diet to airborne fine particulate matters (PM2.5), and then investigated the complex effects and mechanisms of inhalation exposure to PM2.5 on hepatic steatosis, a precursor or manifestation of metabolic syndrome. Our studies demonstrated that inhalation exposure of mice fed normal chow to concentrated ambient PM2.5 repressed hepatic transcriptional regulators involved in fatty acid oxidation and lipolysis, and thus promoted hepatic steatosis. However, PM2.5 exposure relieved hepatic steatosis in high-fat diet-induced obese mice. Further investigation revealed that inhalation exposure to PM2.5 induced hepatic autophagy in mouse livers in a manner depending on the MyD88-mediated inflammatory pathway. The counteractive

effect of PM2.5 exposure on high-fat diet-induced hepatic steatosis was mediated through PM2.5induced hepatic autophagy. The findings from this study not only defined the effects and mechanisms of PM2.5 exposure in metabolic disorders, but also revealed the pleotrophic acts of an environmental stressor in a complex stress system relevant to public health ².

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