

Short-term exposure to haze pollution predisposes healthy volunteers to nasal inflammation

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In recent years, air pollution has become a severe environmental problem. Epidemiological studies suggest that environmental particulate matter (PM) exposure is associated with an increase in upper respiratory tract diseases. The aim of the study was therefore to investigate the impact of a short-term increase in ambient PM_{2.5} levels on the nasal patency of young healthy subjects. Thirty-three healthy university students living in downtown Beijing were recruited and assessed for nasal symptoms, nasal patency, upper and lower respiratory tract nitric oxide (NO), and inflammatory mediators and neuropeptides in their nasal secretions before and during a 5-day episode of haze pollution with ambient PM_{2.5} levels rising to $> 100\mu\text{g}/\text{m}^3$. Slight symptoms of nasal congestion, irritation, dryness or secretion were reported by varying numbers of participants during the haze pollution episode. Objective measures of nasal patency demonstrated that nasal airway resistance was significantly increased, while nasal cavity volume and minimum cross-sectional area significantly decreased from baseline levels during the haze pollution episode. Similarly, the concentrations of nasal NO and exhaled NO, eotaxin, IL-5, CCL17, IL-8, substance P, nerve growth factor, and vasoactive intestinal peptide in the nasal secretions were significantly increased from baseline values during the haze pollution episode. In contrast, IFN- γ , IL-10, TGF- β , and neuropeptide Y decreased significantly from baseline values after exposure to the haze pollution. Short-term haze pollution, comprising increased levels of PM_{2.5} $>100\mu\text{g}/\text{m}^3$, may lead to nasal inflammation in healthy subjects, by promoting Th2-predominant immune reactions and release of parasympathic neurotransmitters.

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