Fine particulate matter control as a potential prevention measure for invasive pulmonary aspergillosis

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Invasive Pulmonary Aspergillosis (IPA) is a severe and progressive fungal lung diseases caused predominantly by *Aspergillus* species, with a highly infection rate among individuals with weakened immune system and serious pre-existing diseases. A recent study has estimated that over 2.1 million patients with specific life-threatening health problems developed invasive aspergillosis annually, with a crude mortality rate exceeding 85%, making it one of the deadliest fungal infections.[1] Despite increasing clinical awareness of its severity, early detection, control, and treatment of IPA remain challengeable. One key explanation lies in the incomplete understanding of its risk factors.

Traditionally, IPA has been recognised in immunocompromised patients, such as those receiving high dose corticosteroids, undergoing chemotherapy, or admitted to intensive care with critical illness. Recently, emerging risk factors include severe viral infections (e.g., COVID-19), exposure to environmental fungal spores, and certain climatic conditions have also been documented.[2,3] However, it remains unclear whether air pollution may contribute to the development of IPA. Given that fine particulate matter ($PM_{2.5}$) is ubiquitous and has an aerodynamic diameter similar to *Aspergillus* spores, controlling $PM_{2.5}$ concentration and exposure could represent an important, previously underappreciated avenue for the prevention of IPA.

Zhou and colleagues, in their recent publication in *eBioMedicine*, explored this hypothesis by analysing two large cohorts of patients with severe pneumonia between 2019 and 2024.[4] They assessed both short- and long-term pre-admission exposure to ambient $PM_{2.5}$ and found consistent associations with increased IPA risk, ranging from 11% to 23% for each 10 $\mu g/m^3$ increment in $PM_{2.5}$. These associations are generally linear. Interestingly, male patients showed greater susceptibility to the impacts of $PM_{2.5}$ than females, possibly attributing to potential sex heterogeneity in pathogenic effects of $PM_{2.5}$, prevalence of existing respiratory diseases, and intensity of critical care admission.[5,6] In addition, the team pointed out that *Aspergillus* plays a key role underlying the linkages between $PM_{2.5}$ and mortality due to pneumonia, proving important evidence for a plausible biological pathway.

This research carries important implications for clinical practice and public health. IPA remains a serious, and often fatal complication in patients with severe pneumonia and respiratory failure, often leading to delayed diagnosis and high mortality. The identification of ambient air pollution exposure as a pre-admission risk factor underscores the need for broader diagnostic vigilance and context-specific prevention approaches in highly polluted regions. Assessing patients' environmental exposure history could aid earlier suspicion, diagnosis, and perhaps even targeted antifungal surveillance. Incorporating environmental awareness into clinical protocols may provide clinicians with a more comprehensive perspective on infection risk and prevention. Moreover, the study reinforces the importance of air-quality control in reducing the burden of both chronic respiratory and acute infectious diseases. $PM_{2.5}$ control can therefore be regarded not only as an environmental strategy but also as an infection prevention measure, potentially protecting the most vulnerable hospital populations.

The paper also raises critical mechanistic questions that warrant further investigation. *Aspergillus* spores are commonly detected in decayed organic matters, such as soil, seeds and grains. However, it remains unclear whether ambient $PM_{2.5}$ can transport these spores, promote their inhalation deep into the alveoli, and directly lead to or amplify IPA infection. Future laboratory and field studies should explore the physical and biological interactions between $PM_{2.5}$ and fungal spores, as well as their causal effects on IPA.

While this study provides robust and new evidence on the associations between pre-admission ambient $PM_{2.5}$ and IPA, the health impacts of other air pollution remain unknown, such as specific $PM_{2.5}$ components, household $PM_{2.5}$, air quality in healthcare settings, and other air pollutants with a similar diameter. In addition, expanding this research to other vulnerable patients, such as those with cancers, HIV, chronic obstructive pulmonary disease and organ transplants, would help generalise and strengthen the clinical applicability of these results.

In summary, Zhou and colleagues have provided timely and compelling evidence that ambient fine particulate matter exposure increases the risk of IPA in

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patients with severe pneumonia. The research findings bridge environmental and clinical sciences, demonstrating that the inhalation of air pollutants may predispose at-risk individuals to fatal fungal infections. They also underscore the value of incorporating environmental exposure assessment into patient evaluation and hospital infection-control strategies, informing future revisions of IPA guidelines. Such research could unite environmental toxicology with fungal pathogenesis, offering a new research frontier on how inhaled pollutants modulate host–pathogen interactions.

Contributors

C.G. conducted the literature search, interpreted the findings, drafted and revised the paper, and secured funding. The author reads and approves the final manuscript.

Declaration of interests

The authors declare no conflict of interest.

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