News Release

Tin in PM2.5 May Worsen Cedar Pollinosis: A New Association Between Environmental Pollution and Allergies

Key Points

- In patients with Japanese cedar pollinosis, tin (Sn) contained in PM2.5 was found to potentially accumulate in the nasal cavity more readily than in healthy individuals.
- Nasal tin concentrations in Japanese cedar pollinosis patients were approximately 3-4 times higher than in non-allergic individuals and showed a significant correlation with the severity of nasal symptoms.
- In an allergic rhinitis mouse model, tin from PM2.5 was captured in the nasal cavity 2-3 times more than in non-allergic mice. As a result, the amount of tin reaching the lungs was reduced by 30-40%.
- The nasal mucosa of allergic rhinitis model mice exhibited excessive mucin production, suggesting that this increase in mucin may contribute to tin accumulation in the nasal cavity.
- These findings indicate that the widely accepted notion that “PM2.5 passes through the nose and reaches the lungs” may not necessarily apply to individuals with allergic rhinitis.
- This study highlights the role of tin in PM2.5 and provides evidence that air pollution may exacerbate allergic rhinitis, such as cedar pollinosis, while offering scientific data to support future environmental policies and health risk assessments.

Summary

A research group from the Department of Environmental and Occupational Health, Graduate School of Medicine, Nagoya University, including graduate student Nishadhi Delgama (co-first author), Lecturer Akira Tasaki (co-first author), and Professor Masashi Kato (corresponding author), in collaboration with Fukui University and Nagoya City University, has revealed that tin (Sn) contained in PM2.5 may exacerbate symptoms of allergic rhinitis, such as Japanese cedar pollinosis.

In this study, the researchers investigated the effects of tin in PM2.5 on Japanese cedar pollinosis symptoms using both human participants and animal

models. An epidemiological survey involving 44 pollinosis patients and 57 healthy individuals showed that: (1) nasal tin concentrations in allergy patients were 3-4 times higher than those in healthy individuals, and (2) nasal tin levels had association with the severity of self-reported symptoms. Furthermore, experiments using an allergic rhinitis mouse model demonstrated that tin exposed to the nasal cavity via aerosols similar to PM2.5 was captured by mucus components, specifically mucin, in the nasal mucosa, suggesting a potential mechanism for symptom aggravation.

This study represents the first report indicating that tin, a component of air pollution, may worsen allergic rhinitis. Previously, it was generally assumed that "PM2.5 passes through the nasal cavity and reaches the lungs" in healthy individuals; however, in individuals with allergic rhinitis, tin derived from PM2.5 may not pass through the nasal cavity, remaining there for extended periods and potentially contributing to symptom worsening.

Although the link between air pollution and allergic rhinitis has been suggested in past studies, scientific evidence regarding which specific pollutants influence allergic rhinitis has been limited. These findings not only identify one pollutant that may exacerbate allergic rhinitis but also provide scientific data that could inform future environmental policies.

The results of this study have been published online in the international journal *Allergy* on December 2, 2025.

Research Background

The prevalence of Japanese cedar pollinosis has been reported to reach approximately 50% in Japan, and it is often referred to as a national disease. In recent years, air pollutants have been suspected of negatively affecting pollinosis, but scientific evidence remains limited. The research group previously reported that lead (Pb), an air pollutant, acts as a factor that exacerbates allergic rhinitis (J Allergy Clin Immunol 2021). In the present study, the researchers investigated how tin (Sn), a chemically related element to Pb, affects allergic rhinitis.

Research Results

The research team measured tin (Sn) concentrations in nasal lavage fluid and serum from 44 patients with pollinosis and 57 healthy individuals, and found the following.

1. Nasal Sn concentrations in patients with pollinosis during the pollen season were 3-4 times higher than those in healthy individuals.
2. Nasal Sn levels were significantly correlated with the severity of allergic symptoms.
3. When a comparable amount of Sn was administered intranasally to mice with experimental allergic rhinitis, allergic symptoms were significantly exacerbated.

These findings provide the first evidence that exposure to Sn, an air pollutant, may worsen pollinosis.

These results raise several new questions. Why do patients with pollinosis have higher nasal Sn concentrations than healthy individuals despite breathing the same air? How does Sn aggravate allergic rhinitis? Sn generally exists in the atmosphere as suspended particulate matter or fine particulate matter, including PM2.5. To address these questions, the research team incorporated Sn into aerosols similar to PM2.5 and exposed model mice via inhalation. The results showed the following.

1. Sn accumulation in the nasal cavity of allergic rhinitis model mice increased by two to three times.
2. In contrast, Sn deposition in the lungs decreased by 30-40%.

This distribution differs from previous knowledge in healthy individuals, where PM2.5 is thought to pass through the nasal cavity and reach the lungs. This is the first evidence that, in individuals with allergic rhinitis, PM2.5 is captured in the nasal cavity, reducing lung exposure.

Why, then, is Sn in PM2.5-like aerosols retained in the nasal cavity? Detailed analyses combining histopathology and elemental imaging revealed the following.

1. Allergic rhinitis model mice exhibited increased nasal mucus production, primarily composed of mucin.
2. Sn showed a high degree of co-localization with mucin in the nasal cavity, with a co-localization rate of 67 percent.
3. Exposure to Sn further enhanced mucin production.

These findings suggest a novel mechanism in allergic rhinitis in which exposure to Sn promotes disease onset, induces excessive mucus production, leads to

retention of Sn in the nasal cavity, and ultimately exacerbates allergic symptoms. This mechanism provides new insights into how air pollution may aggravate pollinosis and other forms of allergic rhinitis.

Research Summary and Future Perspective

This study not only demonstrates that Sn, a component of air pollution, acts as a factor that can exacerbate allergic rhinitis, but also suggests a novel possibility: in individuals with allergic rhinitis, Sn contained in PM2.5 is more likely to accumulate in the nasal cavity.

These findings establish a more direct link between air pollutants and allergic rhinitis. They contribute not only to understanding the pathophysiology of allergic rhinitis (medical significance) but also provide fundamental data for risk assessment of air pollution and the establishment of environmental standards (environmental significance). Furthermore, this research could inform environmental policies by supporting a reevaluation of the health risks of air pollution with a focus on allergic rhinitis (societal significance).

Publication

Journal: Allergy
Title: Emerging Risk: Intranasal Tin Exacerbates Allergic Rhinitis in Humans and Mice.
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DOI: 10.1111/all.70180

Japanese ver.
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