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Review

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Chinese Expert Consensus on the Impact of Ambient Air Pollution on Allergic Rhinitis and Recommendations for Mitigation Strategies

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ABSTRACT

Ambient air pollution poses a significant yet manageable threat to human health. The growing consensus on the impact of ambient air pollutants on allergic rhinitis (AR) emphasizes the importance of prevention, control, and treatment strategies. A multidisciplinary consensus development group was established to further standardize management strategies for AR in the presence of exposure to ambient air pollutants. The quality of the evidence and the strength of the recommendations were evaluated using the grading of recommendations, assessment, development, and evaluation (GRADE) system based on domestic and international relevant medical evidence. This consensus evaluates the effects of key air pollutants on public health in relation to AR, including the synergistic effects of air pollutants with meteorological conditions and aeroallergens. At the same time, the consensus provides recommendations for targeted therapeutic and preventive measures for AR under conditions of ambient air pollution, aiming to improve AR-related health outcomes. These recommendations aim to increase public and clinical awareness of the contribution of environmental factors to AR, and offer evidence-based insights for policymakers and regulators to establish informed ambient air quality standards.

Keywords: Air pollution; allergic rhinitis; prevention; treatment; allergens; air quality; public health

INTRODUCTION

Globally, over 99% of the population is exposed to unsafe levels of ambient air pollutants that exceed the World Health Organization (WHO) 2021 Air Quality Guidelines (AQG), with 80% of those affected living in low- and middle-income countries.[1] Substantial evidence shows that air pollution is a leading cause of morbidity and mortality across a broad spectrum of diseases, including respiratory, cardiovascular, neurological, and other multi-organ conditions.[2,3] According to the data from Global Burden of Diseases, ambient particulate matter (PM) ranked as the top contributor among 88 risk factors, responsible for approximately 8.0% of global disability-adjusted life-years in 2021.[3] In addition to its health toll, air pollution has been linked to high economic costs, primarily due to premature deaths and increased healthcare expenditures. In 2016, global economic losses from air pollution-driven deaths among older adults reached 2.4 trillion US dollars, accounting for 59% of the total pollution-related economic burden.[4] In China, ambient fine particulate matter ($PM_{2.5}$) pollution resulted in an estimated national economic loss of 248 billion US dollars in 2015, with provincial losses varying from 0.5% to 5.8% of the national baseline welfare fee due to differences in population density, economic development, and air quality.[5]

Allergic rhinitis (AR) is one of the most prevalent diseases, affecting 10%–40% of the population worldwide.[6] In China, AR prevalence varies from 10% to 24% across cities, substantially reducing quality of life and significant direct and indirect costs.[7,8] AR is a complex disorder influenced by both genetic and environmental factors. The close relationship between AR and environmental changes has been recognized since ancient

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times. In China, over 3,000 years ago, the book of "Rites" documented a link between the occurrence of AR and changes in the natural environment during the Western Zhou Dynasty.[9] Ambient air pollution is now acknowledged as a critical but controllable threat to public health and well-being, particularly with China's sustained and rapid industrial growth over the past three decades. Ambient air pollutants exposure, such as $PM_{2.5}$, PM less than 10 μm in diameter (PM_{10}), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3), and carbon monoxide (CO), has been shown to increase the incidence of AR and worsen nasal symptoms in patients with AR, thereby reducing the quality of life for individuals with AR.[10]

China has made significant improvements in ambient air quality over recent years, with $PM_{2.5}$ levels decreasing from an annual average of 72 μg/m^3 in 2013 to 30 μg/m^3 in 2023, following the implementation of a series of stringent measures.[11] However, these levels still exceed WHO guidelines, underscoring the need for continued efforts to reduce the health risks associated with air pollution. Despite plenty of literature on the impact of air pollution on AR, no comprehensive expert consensus has been established. Therefore, we have systematically reviewed the existing literature and integrated it with clinical experience to develop an expert consensus. It aims to provide clear guidance on the impact of ambient air pollution on AR and to offer practical recommendations for healthcare professionals and policymakers regarding its prevention and control.

METHODS

Members of the group of experts

This consensus working group consists of 30 members, including allergy specialists, otolaryngologists, ophthalmologists with expertise and clinical experience in the treatment of AR, public health experts with a special focus on the exposome, and environmental scientists who aims to fill this gap. The core leadership team supervised the project, defined its scope, formulated key clinical questions, coordinated literature reviews, and drafted the manuscript.

Literature retrieval

Literature search databases for this consensus included PubMed and China Knowledge Network. The search terms were "ambient air pollution," "allergic rhinitis," "mechanism," "climate change," "prevention" and "treatment." The search timeframe was from the establishment of the repository to September 2024. The included studies involved systematic reviews, meta-analyses, randomized controlled studies, cohort studies, case-control studies, and *in vitro* studies related to ambient air pollutants and AR. It also focused on retrieving relevant studies by Chinese scholars. At least two evaluators screened literature to determine inclusion by first excluding irrelevant literature by title and abstract, and examining the full text of literature that may meet the inclusion criteria.

Methods of consensus formation

This consensus used the grading of recommendations, assessment, development, and evaluation (GRADE) system to evaluate evidence levels, categorizing the quality of evidence into 4 grades (high, moderate, low, and very low), and the strength of recommendation into 2 grades (strong and weak). The quality of the evidence is only one of the factors determining the strength of the recommendation. Narrative reviews were conducted by a different panel of content experts for each topic, and an expert workshop was convened in

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which the participating experts discussed, voted, and proposed revisions that led to the final recommendations.

EPIDEMIOLOGY

Consensus opinion 1

Ambient air pollutants with significant human health impacts are mainly related to broad sources of $PM_{2.5}$, PM_{10}, O_3, NO_2, SO_2, and CO (Level of Evidence: High, Strength of Recommendation: Strong).

Consensus opinion 2

Exposure to ambient air pollutants not only increases the incidence of disease in AR-susceptible populations, but also exacerbates clinical symptoms (Level of Evidence: High, Strength of Recommendation: Strong).

Ambient air pollution

The WHO defines ambient air pollution as "contamination of the outdoor environment by any chemical, physical, or biological agent that modifies the natural characteristics of the atmosphere." Key air pollutants of public health concern–including PM, O_3, nitrogen oxides (NO_x), SO_2, and CO–are routinely monitored in China to evaluate the ambient air quality. These pollutants include primary pollutants directly emitted from various sources, such as industrial processes, transportation, residential combustion, biomass burning, construction, and natural sources and secondary pollutants formed in ground level by chemical reaction (**Fig. 1**).[12]

In 2021, the WHO updated its global AQG, providing recommendations on safe levels for $PM_{2.5}$, PM_{10}, O_3, NO_2, SO_2, and CO, with thresholds of 5 μg/m^3 (annual), 15 μg/m^3 (annual), 60 μg/m^3 (peak season), 10 μg/m^3 (annual), 40 μg/m^3 (24-hour), and 4 mg/m^3 (24-hour).[13] These new guidelines, informed by the high-quality evidence covering a vast body of research papers published up to 2018, reinforce the understanding that air pollution continues to pose severe health risks. According to the WHO data, more than 90% of the global population currently breathes air that exceeds the recommended AQG limits, with low- and middle-income countries experiencing the highest exposure levels.

Air pollution plays a major role in the incidence of diseases, as well as in morbidity and mortality rates. Globally, it was estimated that 4.51 million deaths were attributable to ambient air pollution in 2017, with 4.14 million related to PM exposure and 0.37 million to O_3 exposure.[14] In China, ambient air pollution accounted for an estimated 1.03 million deaths in 2017, including 0.85 million from ambient PM exposure and 0.18 million from ambient O_3 exposure.[15]

Effect of air pollutant exposure on AR

There is growing evidence that exposure to air pollutants not only increases the disease incidence in susceptible subpopulations but also exacerbates clinical symptoms. It is now well-documented that air pollution is a risk factor for AR development. A review of 35 studies conducted across countries in Europe and Asia revealed a stronger association between air pollution and AR prevalence in developing countries, compared to developed nations.[16] Furthermore, a meta-analysis found a positive association between AR prevalence and increased levels of ambient NO_2, $PM_{2.5}$, and SO_2,[17] supporting the notion that air pollution

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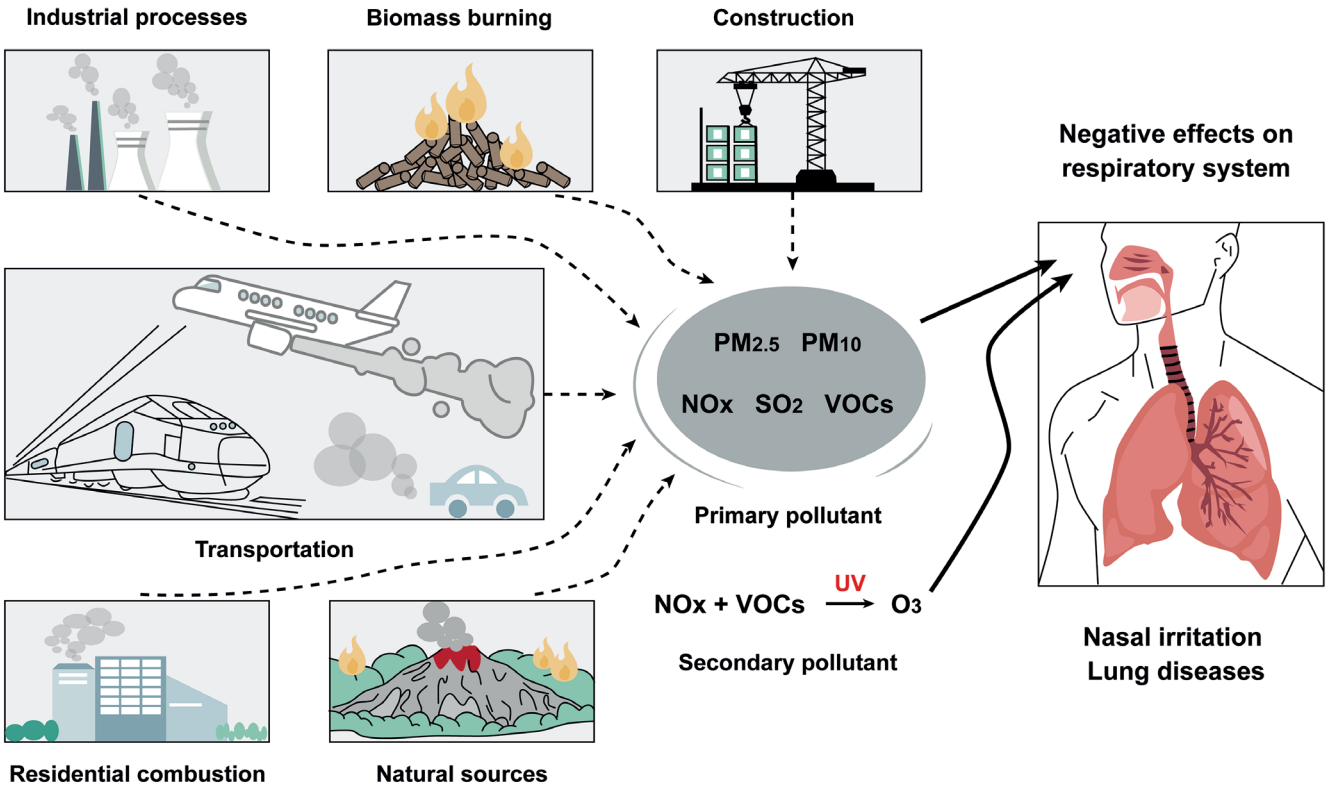

Fig. 1. Pollutants of major public health concern. These pollutants are emitted from various sources, such as industrial processes, transportation, residential combustion, biomass burning, construction and biomass burning are major sources of air pollutants, which are strongly associated with prevalence of respiratory diseases.
$PM_{2.5}$, particulate matter less than 2.5 μm in diameter; PM_{10}, particulate matter less than 10 μm in diameter; NO_x, nitrogen oxides; SO_2, sulfur dioxide; O_3, ozone; VOC, volatile organic compound; UV, ultraviolet.

contributes to the growing burden of allergic diseases.[18] A multi-city research in China found that a 10 μg/m^3 increase in concentrations of SO_2, NO_2, $PM_{2.5}$, PM_{10}, and maximum 8-hour moving average ozone ($O_3$8h) concentrations corresponded with increased daily AR outpatient visits by 7.69%, 2.43%, 1.84%, 1.55%, and 0.34%, respectively.[19] Each standard deviation increase in $PM_{2.5}$ and NO_2 concentrations was associated with a 10% and 11% increase in AR incidence, respectively.[20]

We further evaluated the existing literature on the correlation between ambient air pollution and AR symptom severity. Long-term exposure to ambient air pollution, such as PM_{10}, $PM_{2.5}$, and NO_2, was associated with increased rhinitis severity in two European cohorts.[21] A one-year panel study showed that patients with AR exposed to air pollutant mixtures ($PM_{2.5}$, O_3, SO_2, and NO_2) were more likely to have more severe nasal symptoms and worse quality of life.[22] During high pollen exposure, O_3 concentrations were associated with increased OR of rhinitis, and $PM_{2.5}$ concentrations were associated with increased ORs of rhinitis and increased use of allergy medication.[23] Higher levels of pollution have been found to lead to worse seasonal allergy symptoms in seasonal AR (SAR) patients.[24,25] In addition, cross-sectional study revealed that ambient air pollutants–CO, PM_{10}, and $PM_{2.5}$–negatively impacted discomfort and symptoms of rhinitis in obese children with AR compared to their non-obese counterparts.[26] These results all suggested that exposure to ambient air pollutants plays an important role in the development and exacerbation of AR.

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MECHANISMS

Consensus opinion 3

The mechanisms by which ambient air pollutants affect AR involve increased oxidative stress and epithelial barrier disruption, promotion of inflammation and dysregulation of the immune response, as well as effects on nasal mucosal gene expression and DNA methylation (Level of Evidence: Moderate, Strength of Recommendation: Strong).

Knowledge of the mechanisms of air pollution harm mainly includes increased oxidative stress and altered immune system response to allergic reactions, while genetic and epigenetic modifications also influence the response to air pollutants in patients with AR (**Fig. 2**).

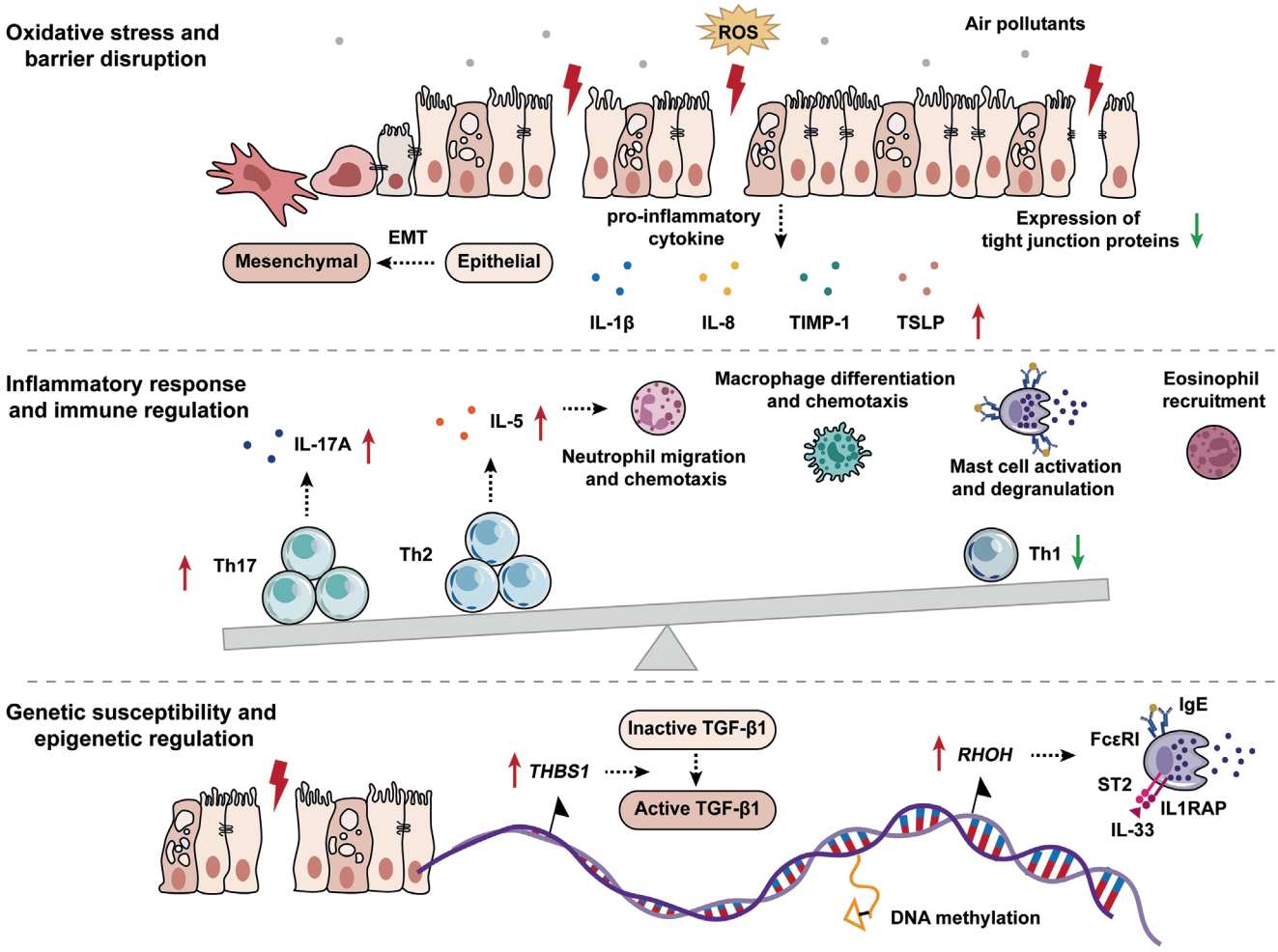

Fig. 2. Systemic mechanisms underlying the hazards of ambient air pollution. Exposure to ambient air pollutants leads to ROS overproduction, which initiates epithelial-to-mesenchymal transition, reduces the expression of tight junction proteins, and increases the release of proinflammatory cytokines (IL-1β, IL-8, TIMP-1, and TSLP). Ambient particles induce inflammation and shift immune function from Th1 to Th2, while enhancing Th17-mediated adaptive immune responses. In addition, air pollutants affect respiratory cilia function, mast cell activation, and proinflammatory TGF-β1 signaling by altering nasal DNA methylation and nasal gene (like *THBS1* and *RHOH*) expression levels.
ROS, reactive oxygen species; EMT, epithelial-mesenchymal transition; IL, interleukin; TIMP-1, tissue inhibitors of metalloproteases-1; TSLP, thymic stromal lymphopoietin; THBS1, thrombospondin 1; TGF-β1, transforming growth factor beta 1; RHOH, ras homolog family member H; FcεRI, Fc epsilon RI; IgE, immunoglobulin E; ST2, suppression of tumorigenicity 2; IL1RAP, IL-1 receptor accessory protein.

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Oxidative stress and barrier disruption

In a panel study, Li *et al.*[22] found that air pollutants such as $PM_{2.5}$, O_3, and SO_2 exposure contributed to AR exacerbation by increasing oxidative stress and reducing antioxidant capacity. Evidence suggests that air pollutants trigger the generation of reactive oxygen species (ROS), either by disrupting cellular redox signaling or upregulating endogenous ROS production.[27] Since the respiratory system is the primary route of exposure, oxidative stress initially affects the airways and lungs. It is generally accepted that oxidative stress results from excessive production of oxidants and depletion of antioxidants in the cell, leading to the overproduction of ROS, a process associated with mitochondrial dysfunction.[28] In the context of this mechanism, air pollutants appear to impair the function of structural cells, initiating epithelial-mesenchymal transition, a process that leads to dysfunction of the endothelial and epithelial cell barriers, impeding tissue repair and ultimately leading to fibrosis.[29,30]

In vitro studies have shown that black carbon, a byproduct from incomplete fuel combustion, exaggerates oxidative stress, significantly increases interleukin (IL)-1β expression, and decreases cell viability in human nasal epithelial cells (hNECs) from patients through the integrated pathway of ROS-NLRP3-Caspase-1-IL-1β signaling.[31] By extracting epithelial cells derived from nasal mucosal tissues of patients, it was found that exposure to $PM_{2.5}$ can compromise the nasal epithelial barrier by reducing the expression of tight junction proteins and increasing proinflammatory cytokine (IL-8, TIMP-1, and TSLP) release.[32] Furthermore, it was demonstrated in a mouse model that O_3 exposure induces immediate epithelial barrier damage, leading to protein leakage, epithelial cell desquamation, and release of the alarmin IL-33, which is followed by the recruitment of myeloid cells (especially neutrophils), thus exacerbating respiratory epithelial damage and inflammation.[33]

Inflammatory response and immune regulation

Ambient particles induce inflammation, shifting immune function from Th1 to Th2 responses and promoting IgE production.[34] A controlled human exposure study demonstrated that a positive correlation between ambient air pollutant levels and systemic inflammation.[35] Exposure of healthy nasal mucosal tissues to $PM_{2.5}$ initiated a proinflammatory immune response, which was accompanied by an increase in type 2 cytokines (*e.g.*, IL-5) and proinflammatory cytokines (IL-1β, IL-6, and IL-8). This response mirrors type 2 inflammation observed in AR patients with chronic inflammation of the upper airway mucosa.[36] This suggested that $PM_{2.5}$ exposure may induce exacerbations of airway disease and contribute to the development of established type 2 immune responses in the airways with repeated exposures over time. Combined exposure to ozone-aged environmental black carbon and pollen allergens may induce innate immunity and allergic inflammation in hNECs (from nasal epithelial mucosal samples of healthy individuals undergoing nasal surgery), including the regulation of neutrophil migration and chemotaxis, macrophage differentiation and chemotaxis, mast cell activation and phagocytosis, and a significant increase in the expression of inflammation-related genes *IL1B*, *CSF1*, and *FCER1G*.[37] In a mouse model of chronic allergy, it was found that exposure to ambient air pollution significantly increased the number of eosinophils in the nasal mucosa and that $PM_{2.5}$ may stimulate the innate immune system through the $PM_{2.5}$-Nod1-nuclear factor-κB axis, which triggers chronic allergic diseases.[38] Additionally, ambient air pollution enhances not only the type 2 immune response but also the Th17-mediated adaptive immune response.[39] Increased expression of IL-17A in the nasal mucosa may contribute to diesel exhaust particle (DEP)-mediated exacerbation of AR in house dust mite-sensitized murine AR model.[40]

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Genetic susceptibility and epigenetic regulation

An increasing body of evidence points to changes in DNA methylation as one of the main mechanisms by which air pollutants affect human biology and health, and gene expression differences in the nasal mucosa have been previously associated with criteria air pollutant exposure. The heritability of AR has been estimated to range from 33% to 91%.[41] Individuals with a strong genetic predisposition are at increased risk of developing AR when exposed to ambient air pollution, with even low pollutant exposure possibly triggering AR.[42] Multi-omics integration data revealed that air pollutants influence the development of AR by altering nasal DNA methylation and expression levels of nasal genes (*e.g.*, *THBS1* and *RHOH*) involved in respiratory ciliary function, mast cell activation, proinflammatory transforming growth factor (TGF)-β1 signaling, and the regulation of myeloid immune cell function.[43]

The mechanisms described above form the basis of the "epithelial barrier hypothesis," which explains how ambient air pollutants disrupt the epithelial barrier and interact with immune cells, contributing to AR pathology.[44] These mechanisms link environmental exposures to disease risk, and a better understanding of the molecular and genetic factors influenced by ambient air pollutants could guide the prevention, management, and treatment of AR.

SYNERGISTIC EFFECTS

Consensus opinion 4

Ambient air pollutants contribute to warmer temperatures and increased extreme weather events, and climate change simultaneously exacerbates air pollution exposure (Level of Evidence: High, Strength of Recommendation: Strong).

Consensus opinion 5

Ambient air pollutants and climate change affect the occurrence and progression of AR by increasing the exposure opportunity to aeroallergens (Level of Evidence: Moderate, Strength of Recommendation: Strong).

Climate change, as well as air pollution, are believed to be significant contributors to the global rise in allergic diseases.[45,46] Ambient air pollution, particularly from transportation and other human activities, not only directly affects human health but also contributes to global warming and increases the frequency of extreme weather events, changes that in turn modify the availability and distribution of plant- and fungal-derived allergens (**Fig. 3**).[46]

Since the Industrial Revolution, there has been a significant increase in atmospheric pollutants, such as greenhouse gases and aerosolized PM, produced by human activities, which have had a profound impact on the climate system. According to the Intergovernmental Panel on Climate Change, without significant reductions in greenhouse gas emissions, global warming could top 1.5°C sometime between 2030 and 2052, with a concomitant increase in the probability of extreme weather events (such as the heatwaves, droughts, storms, and flooding).[47] The Sixth Assessment Report (AR6) on climate change estimated a global mean surface air temperature (GSAT) increase of about 1.1°C between 2011 and 2020 due to greenhouse gases (GHGs) and O_3, compared to 1850–1900.[48] It is worth noting that climate change can simultaneously exacerbate ambient air pollution exposure and its resulting health risks. The representative concentration pathway 4.5, a GHG modeling trajectory based on global CO_2 reaching a peak around 2040, predicts that conditions such as

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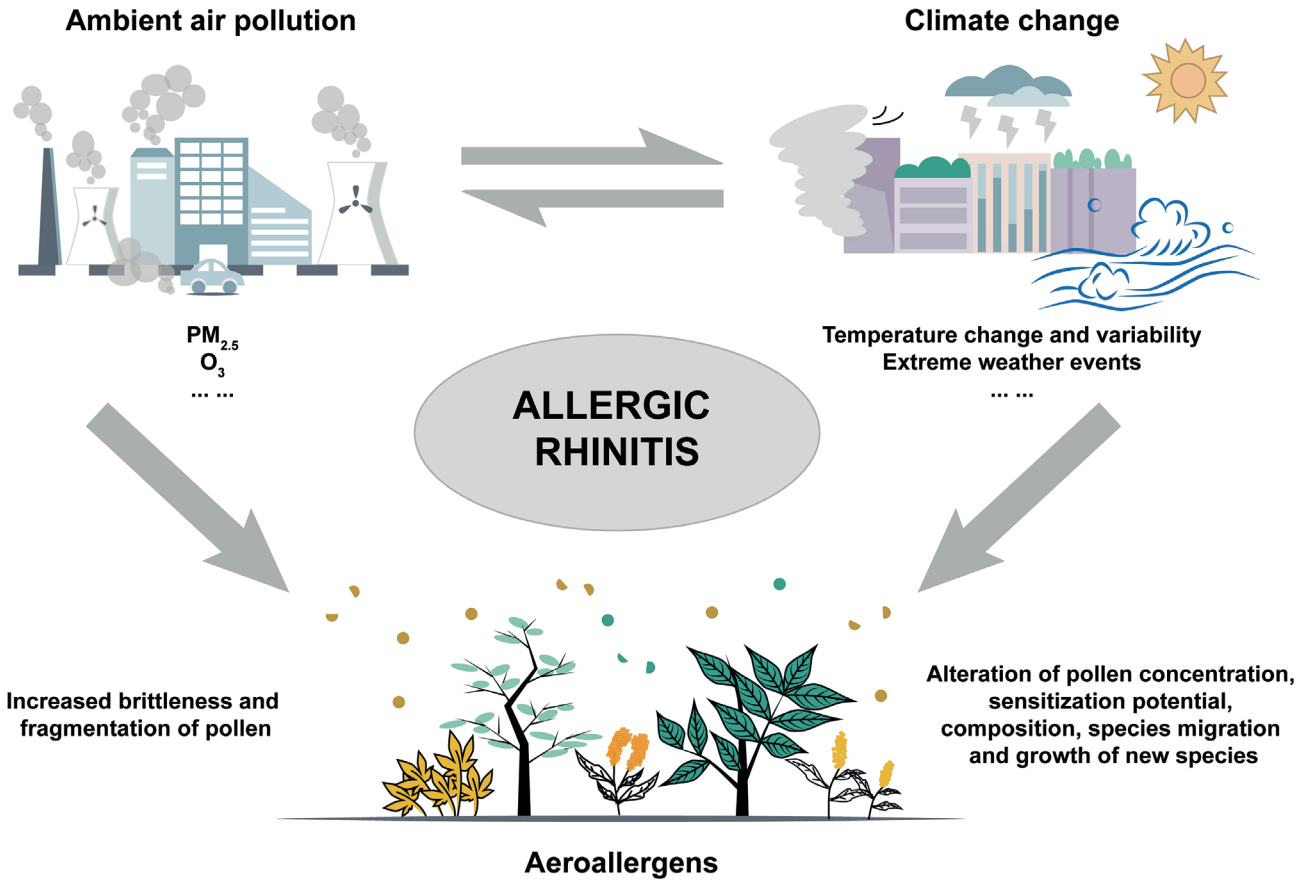

Fig. 3. Synergistic effects of air pollutants, meteorological conditions, and pollen on AR. Ambient air pollutants contribute to warmer temperatures and increased extreme weather events (such as the heatwaves, droughts, storms and flooding), and climate change simultaneously exacerbates air pollution exposure. Together, ambient air pollutants and climate change influence the onset and progression of AR by increasing the bioavailability of aeroallergens. $PM_{2.5}$, particulate matter less than 2.5 μm in diameter; O_3, ozone; AR, allergic rhinitis.

atmospheric stagnation and heat waves will contribute 68% to the climate-driven increase in $PM_{2.5}$ and 14% to O_3 in China.[49]

Rapid economic growth and urbanization have caused severe regional haze in China. Studies have shown that the concentrations of haze-causing air pollutants O_3, NO_x, and SO_2 are significantly associated with the daily number of grass pollen-positive patients and the concentration of grass pollens in the environment.[50] Oak pollen exposed to high levels of SO_2 or NO_2 becomes more fragile, leading to increased release of pollen cytoplasmic granules. This increased bioavailability of airborne pollen allergens has been linked to a rise in allergic disease incidence.[51] Climate change indirectly affects allergies by altering pollen concentrations, allergenic potential, composition, migration of species, and growth of new ones.[52] There is evidence that an increase in extreme weather events such as rain or humidity may induce hydration and fragmentation of pollen grains, while thunderstorms can cause pollens and molds to break up and their fragments to form a highly inhalable allergenic aerosol on the ground, which can trigger an asthma crisis in allergy sufferers.[53] Moreover, elevated ambient temperatures caused by air pollutants can result in an earlier start and prolonged duration of the tree/pollen season, thereby extending the exposure duration for patients with SAR.[54]

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In summary, the feedback loop between ambient air pollution and climate change creates optimal conditions for the increased incidence and prevalence of allergic diseases. Ambitious climate policies, a low-carbon energy transition, and stringent clean air policies are all necessary to significantly reduce the human health impacts of air pollution in China. Given the substantial role of increasingly intense extreme weather events in future climate-driven health risks from ambient air pollution in China, it is more appropriate for policymakers and the public to focus primarily on episodic periods of severe air pollution rather than average air quality alone.

MANAGEMENT

Consensus opinion 6

Wearing facemasks, using air purifiers, practicing green modes of transportation, increasing vegetation cover, and modifying diet can help reduce exposure to ambient air pollutants and thus prevent AR (Level of Evidence: Moderate, Strength of Recommendation: Strong).

Consensus opinion 7

Conventional treatments such as intranasal corticosteroids, second-generation selective histamine H1 receptor antagonists, and nasal irrigation can help reduce the disease load in AR patients with combined exposure to ambient air pollutants (Level of Evidence: Moderate, Strength of Recommendation: Strong).

Preventive and therapeutic measures are crucial in managing the development and exacerbation of AR in sensitive populations exposed to air pollutants (**Fig. 4**).

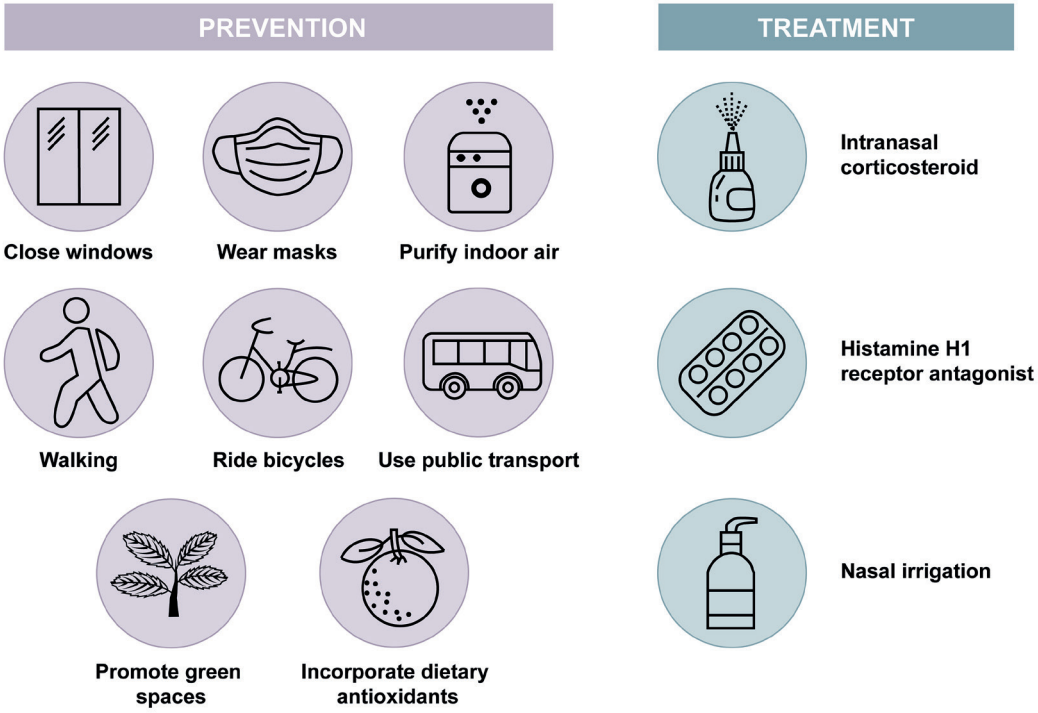

Fig. 4. Preventive and therapeutic measures against exposure to air pollutants. These measures include avoidance of exposure to air pollution, community efforts to reduce air pollutants, increased planting of green vegetation, consumption of antioxidant-rich diets, and the application of targeted allergic rhinitis treatments.

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Prevention of air pollution in patients with AR

Avoid exposure to air pollution

Reducing exposure to air pollutants is an effective strategy for preventing AR. During periods of high ambient air pollution, it is advisable to wear facemasks outdoors, keep windows closed indoors, and use an air purifier. The results of the randomized crossover trial demonstrated that after exposure to PM, the facemask-using group had lower levels of serum cytokines (IL-6, IL-10, IL-13, IL-17A, interferon-γ, and tumor necrosis factor-α) and effectively reduced levels of inflammation and oxidative stress compared to the control group.[55] Evaluations of both individuals and populations in Beijing showed that wearing facemasks reduced outdoor $PM_{2.5}$ exposure by 70%–81%, but the fact that people spent most of their time indoors resulted in much lower overall reductions.[56] Several studies have shown that the use of facemasks effectively reduces allergen exposure in sensitive individuals and significantly improves nasal and ocular symptoms in AR patients.[57] Measures to avoid indoor exposure to ambient air pollutants are necessary because of the strong air exchange between indoors and outdoors. A randomized crossover study demonstrated that indoor air purification reduced $PM_{2.5}$ levels by approximately 47.53%, and patients required less medication.[58] The use of air purifiers with high-efficiency particulate air filters significantly lowered indoor $PM_{2.5}$ concentrations and reduced symptoms and medication requirements for AR patients.[59]

Collective actions

Collective actions to reduce air pollution are critical to improving public health. Vehicle emissions are a major source of air pollution, and public awareness campaigns can help promote green transportation methods, such as walking, cycling, and using public transport. These efforts are essential in reducing air pollution levels. For instance, during the 2008 Beijing Olympics, alternative traffic strategies were implemented, resulting in significant reductions in O_3 and $PM_{2.5}$ levels, which improved air quality and reduced AR incidence.[60] The transition from fuel-powered vehicles to new energy vehicles (*e.g.*, electric or hybrid) may also reduce fossil fuel use and improve air quality, thereby lowering the incidence of AR.[61]

Promoting green spaces

Increasing vegetation coverage in residential areas can help mitigate air pollution by removing PM, CO, CO_2, SO_2, NO_2, and O_3. Green spaces are particularly important for preventing the occurrence of AR.[62] An Austrian study found that AR may be linked to higher NO_2 levels, which were significantly reduced in areas with greater vegetation coverage, potentially lowering AR risk.[63] Notably, some woody plants (*e.g.*, hazel, birch, pine, *etc.*) and herbaceous plants (*e.g.*, mugwort, goosefoot, ragweed, *etc.*) are the main sources of seasonal allergens in China.[64] The effects of green space and air pollution on respiratory health of pollen allergy patients are complex. Air pollutants contribute to more severe allergy symptoms, whereas exposure to green space may mitigate the severity of tree pollen allergy symptoms, particularly when the density of allergenic trees is low.[65] Although findings on the benefits of green spaces are mixed, overall, increasing green vegetation cover is a valuable strategy for preventing AR and reducing its symptoms.[66]

Dietary measures

Epidemiologic data suggest that dietary intake of exogenous antioxidants may reduce the prevalence of atopic diseases.[67] Exogenous antioxidants are found in many foods, particularly fruits and vegetables. Common sources include citrus fruits (rich in vitamin C), nuts (rich

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in vitamin E), and carrots (containing β-carotene). Dietary antioxidants may counteract oxidative stress and inflammation caused by air pollutants. Stronger inverse association between dietary total antioxidant capacity and incident sensitization to inhalant allergens was observed among children with low exposure to traffic-related NO_x.[68] A fiber-rich diet attenuated airway hyperresponsiveness in male mice caused by O_3 exposure in a mouse model of allergic airway disease.[69] Until further epidemiologic and causal findings are available, dietary modifications in the context of ambient air pollution exposure may help prevent AR.

Treatment of AR exacerbated by air pollution

To date, given the lack of specific indicators to distinguish AR exacerbated by ambient air pollution, and the absence of pharmacologic therapies specifically targeting this subgroup of AR, the management of patients with AR exacerbated by ambient air pollution should follow guidelines. A number of potential therapeutic strategies have been identified by evaluating treatment efficacy in AR patients under environmental conditions that simulate ambient air pollution. One study demonstrated the effectiveness of intranasal corticosteroids in alleviating rhinitis symptoms during periods of severe air pollution. Ten days of budesonide treatment significantly improved total nasal symptoms and nasal itching/sneezing in patients with AR triggered or worsened by air pollution.[70] Moreover, second-generation, selective histamine H1 receptor antagonists have been reported to alleviate SAR symptoms during DEP and allergen co-exposure.[71,72] Additionally, nasal irrigation may serve as an adjunct treatment by cleansing dust particles and air pollutants from the nasal cavity, enhancing mucociliary function, reducing mucosal edema, promoting blood circulation, and aiding in repairing the damaged nasal mucociliary epithelium.[73] In the future, further large cohort and multicenter studies in the Chinese population are needed to clarify the efficacy of conventional drug therapy in reducing the disease load in AR patients with combined exposure to ambient air pollutants. More importantly, new therapeutic targets should be developed based on the identified mechanisms of cellular damage and signaling pathways induced by pollutant exposure.

CONCLUSIONS

Ambient air pollution plays a crucial role in the onset, acute exacerbation, and immune dysregulation associated with allergic diseases, attracting significant attention worldwide. In the future, the interactions of different ambient air pollutants still need to be explored in depth to clarify their combined effects on the risk of developing allergic diseases at different exposure levels and whether there is a threshold effect. China is a vast country with large differences in air quality, climatic conditions, and vegetation types in different regions, all of which may affect the effects of air pollutants on AR. Therefore, comparative studies should be strengthened in different regions, especially those with significant differences in air quality, in order to gain a more comprehensive understanding of the effects of air pollutant exposure on allergic diseases. Furthermore, China's large population necessitates greater attention to the precise prevention and control strategies tailored to populations particularly vulnerable to air pollutants, such as children, older adults, outdoor workers and people with chronic respiratory diseases. Simultaneously, effective prevention and control measures targeting environmental exposures are essential to minimize disease risk in vulnerable populations and alleviate clinical symptoms. Strengthening air quality monitoring and early warning systems, reducing air pollutant emissions, enhancing public health awareness and protective measures, as well as strengthening medical care and disease management, require joint efforts of government departments, medical practitioners, and the public.

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More importantly, the development of new therapeutic targets is underway, focusing on the molecular mechanisms and susceptibility genes identified in response to pollutant exposure.

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