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# Role of air pollution in rhinitis

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## Abstract

Air pollution is an increasing global concern with serious health and economic impacts. Among its many effects, respiratory health is particularly vulnerable. As the first point of contact with inhaled pollutants, the nasal passages play a crucial role in airway defense, making rhinitis one of the key inflammatory conditions linked to environmental pollution. This review explores the relationship between air pollution and rhinitis, highlighting key pollutants such as particulate matter (PM, $PM_{2.5}$, $PM_{10}$), nitrogen dioxide ($NO_2$), sulfur dioxide ($SO_2$), and ozone ($O_3$), which contribute to airway inflammation, epithelial barrier dysfunction, immune system dysregulation, and epigenetic changes. Epidemiological studies demonstrate a strong association between pollutant exposure and increased prevalence, severity, and healthcare utilization for allergic rhinitis. However, there is limited research focusing on non-allergic rhinitis. Beyond health concerns, air pollution also imposes a significant economic burden due to rising healthcare costs and lost productivity. Effective mitigation strategies include air quality monitoring, indoor air filtration, policy interventions, and lifestyle modifications. Addressing pollution-related rhinitis requires a multidisciplinary approach involving public health initiatives, clinical management, and environmental policies to reduce exposure and improve patient outcomes. Additionally, limitations in current research are discussed, and further studies are recommended to fill existing knowledge gaps.

## Keywords

Rhinitis, allergic rhinitis, immunology, pollution, particulate matter, air pollutants

## Introduction

Air pollution poses a significant threat to both human health and the economy. The severity of air pollution is steadily increasing in several parts of the world, driven by human activities, including both indoor and outdoor sources, from domestic to industrial levels. Climate change exacerbates this issue, and conversely, pollution intensifies climate change, creating a vicious cycle.

Air pollution has detrimental effects on various aspects of human health, including multiple organ systems [1], and has been linked to increased mortality rates worldwide [2]. The impact on the human respiratory system is particularly evident and complex, with conditions such as asthma (lower airway) and rhinitis (upper airway) being prominent examples.

Environmental pollutants play a pivotal role in the progression of obstructive airway diseases, such as asthma and chronic obstructive pulmonary disease (COPD). Pollutants disrupt the airway epithelial barrier and impair mucociliary function, leading to chronic inflammation, airway remodeling, and increased susceptibility to infections. This is evident in higher exacerbation rates among asthma and COPD patients living in polluted regions [3, 4].

Upper respiratory tract symptoms, including runny nose, sneezing, nasal congestion, and cough, are significantly influenced by air pollution. As the first point of contact for airborne pollutants, the upper respiratory tract is particularly vulnerable to their harmful effects. Children exposed to higher levels of particulate matter (PM) showed a 10–11% higher prevalence of these symptoms compared to those in less polluted areas [5]. Additionally, over 51% of patients with allergic rhinitis (AR) reported air pollution as a trigger, and 81.1% associated their symptoms with climate change [6]. These exposures can impair daily functioning, reduce work productivity, and contribute to a growing economic burden. Understanding the interplay between air pollutants and the upper respiratory system is essential for developing effective prevention strategies and informing efforts at the policy level. This review aims to examine the role of air pollution in rhinitis by exploring its components, health impacts, underlying mechanisms, economic implications, and available mitigation strategies.

## Air pollution

Air pollution arises from both anthropogenic and natural sources. Anthropogenic sources include transportation, industrial activities, domestic fuel combustion, agriculture, and energy production. Transportation emissions come from vehicles such as cars, trucks, and airplanes, while industrial activities release pollutants through processes like power generation and manufacturing. Domestic fuel burning, especially biomass in developing countries and agricultural emissions from livestock rearing, fertilizer use, and deforestation further contribute to pollution.

Natural sources also contribute to air pollution, albeit typically to a lesser extent than anthropogenic sources. These include volcanic eruptions, dust storms, the resuspension of soil dust, wildfires, and the release of sea salt [7].

Key pollutants with proven health impacts include PM, carbon monoxide (CO), ozone ($O_3$), nitrogen dioxide ($NO_2$), and sulfur dioxide ($SO_2$) [8]. PM, a major concern due to its ability to penetrate deep into the respiratory tract, refers to tiny airborne particles categorized by size, such as $PM_{2.5}$ (≤ 2.5 μm) and $PM_{10}$ (≤ 10 μm).

A systematic review of global sources of PM ($PM_{10}$ and $PM_{2.5}$) identified five primary categories. Globally, urban ambient $PM_{2.5}$ pollution is attributed to transportation (25%), domestic fuel burning (20%), industrial activities (15%), other human activities (22%), and natural sources including soil dust and sea salt (18%) [9].

Transportation, including emissions from fuel combustion and wear of brake linings, clutch, and tires, is a significant contributor. The emitted particles are deposited onto the road and then re-suspended by vehicle traffic, along with crustal/mineral dust particles and road wear material. Vehicle age and maintenance significantly influence emission levels [10, 11].

Domestic fuel burning, including the combustion of wood, coal, and gas for cooking and heating, contributes significantly to PM levels. However, biomass burning (wood, dung) is a significant source of PM in many developing countries. Indoor air pollution from domestic fuel burning poses significant health risks to populations in these regions.

Industry encompasses emissions from diverse sectors, including petrochemical, metallurgic, ceramic, pharmaceutical, and IT hardware manufacturing. Variations exist across different industrial activities, and energy production remains a significant contributor due to fossil fuel combustion [11].

Natural sources can be significant contributors to atmospheric PM concentrations. Soil dust, primarily composed of elements abundant in the Earth's crust, is readily resuspended by local winds from fields or bare soils. Sea salt particles, commonly found in coastal areas, can be transported inland by wind or introduced through the application of road salt [9]. Dust storms involve the wind-driven movement of dust particles across large areas. Additionally, wildfires release smoke and PM generated from burning vegetation. The relative importance of these natural sources can vary considerably based on regional characteristics, climate conditions, and land use practices.

Major components of air pollution are also classified as greenhouse gases, and their elevated levels contribute to global warming, which, in turn, can further exacerbate air pollution. These gases include $O_3$, methane, nitrous oxide, carbon dioxide, CO, $SO_2$, and $NO_2$. They absorb infrared radiation (heat) emitted from the Earth's surface after it has absorbed sunlight. This absorbed energy is then partially re-radiated back toward the Earth's surface, warming the atmosphere and helping to maintain a temperature essential for sustaining life.

Since the industrial revolution in the mid-19th century, the levels of greenhouse gases have increased significantly, largely due to human activities such as fossil fuel combustion, deforestation, and industrial processes. As a result, the Earth's average temperature has risen by approximately 1°C [12]. Projections suggest that, if current trends persist, global temperatures could increase by 1.5°C by the middle of the 21st century.

The rise in global temperatures has already resulted in various climate change impacts, including more frequent and intense heatwaves, rising sea levels, altered precipitation patterns, and disruptions to ecosystems and agriculture. Moreover, climate change is associated with increased air pollution. Elevated temperatures can accelerate the formation of ground-level $O_3$, while climate-related phenomena such as dust storms and wildfires have direct and significant impacts on air quality. This creates a vicious cycle in which climate change worsens air pollution, and in turn, air pollution contributes to further climate change [7].

## Air pollution and AR

The impact of pollution on AR has been a significant focus of research worldwide, particularly in regions with substantial air quality challenges, such as many Asian countries. A number of epidemiological investigations have explored the association between air pollution and AR, its influence on healthcare utilization, and the resulting quality-of-life implications for affected individuals.

A systematic review and meta-analysis conducted in 2022 examined the link between air pollution exposure and AR, analyzing 35 observational studies from 12 countries. Despite the high heterogeneity among the studies, a significant association was identified between AR and air pollutants such as $PM_{2.5}$, $PM_{10}$, $NO_2$, $SO_2$, and $O_3$. Notably, no significant correlation was observed with CO. The pooled odds ratios ranged from 1.07 to 1.13, underscoring the health risks posed by these pollutants [13]. A national multicenter survey in China involving 40,279 young adults from eight cities highlighted home environment factors associated with asthma and AR. The study reported that 1.6% of participants had asthma, while 6.6% reported AR. Key risk factors associated with AR included high levels of urbanization, elevated $NO_2$ concentrations, proximity to heavy traffic, home redecoration, buying new furniture and the use of specific cooking fuels [14]. Similarly, growing investigations from Latin America also found a 43% increase in AR odds among individuals exposed to air pollutants compared to non-exposed individuals, highlighting the

urgent need for policies to reduce pollutant exposure and protect vulnerable populations [15]. Furthermore, a systematic review of eight studies, primarily conducted in China, Canada, and Germany, demonstrated a positive correlation between prenatal and early-life exposure to traffic-related air pollution and the subsequent development of AR in children [16].

Using data from the UK Biobank, a causal effect analysis investigated the relationship between pollution and AR in 379,488 participants initially free of rhinitis symptoms. Over a median follow-up period of 12.5 years, 3,095 cases of AR were identified. Significant associations were observed between AR risk and various pollutants, including $PM_{2.5}$, PM coarse, $PM_{10}$, $NO_2$ and nitrogen oxides ($NO_x$). The hazard ratios varied between 1.10–1.51. The study also found that individuals with both high pollution exposure and genetic predisposition faced the greatest risk of developing AR [17].

An Italian study assessed long-term exposure to air pollutants and the prevalence of respiratory and allergic symptoms in 14,420 participants. Findings revealed that $PM_{10}$ and $PM_{2.5}$ were associated with a 14–25% increase in rhinitis odds. $NO_2$ exposure correlated with a 6–9% increase in rhinitis odds [18]. A large French cohort study of 127,108 adults evaluated the association between long-term pollutant exposure and self-reported rhinitis. AR and non-AR (NAR) prevalence were 24.9% and 11.0%, respectively. There was an association between current rhinitis and exposure to pollutants, including $PM_{2.5}$, black carbon (BC), and $NO_2$. Both AR and NAR showed significant positive associations with pollutant exposure [19].

The effects of pollution on rhinitis symptoms and quality of life have been further elucidated through panel studies. For instance, a Swedish study on individuals with asthma and birch allergy demonstrated that $PM_{2.5}$ exposure significantly increased the odds of rhinitis or eye irritation and allergy medication usage [20]. Similarly, a panel study in China linked $PM_{2.5}$ and its constituents to elevated visual analog scale and rhinoconjunctivitis quality of life questionnaire scores. Nasal oxidative stress markers, such as increased malondialdehyde and decreased superoxide dismutase, further supported these findings [21]. Another investigation by the same research group assessed short-term exposure to a combination of pollutants ($PM_{2.5}$, $O_3$, $SO_2$, and $NO_2$) and its effects on AR patients. Results showed exacerbated nasal symptoms, higher rhinitis scores, and reduced antioxidant capacity in nasal lavage fluid [22].

These studies collectively highlight the critical role of air pollution in the pathogenesis and exacerbation of AR, underscoring the need for targeted interventions and mitigation strategies (Table 1).

**Table 1. Selected studies on air pollution and AR**

| Author | Year | Study design | Location | Population | Pollutant | Outcomes | Result |
|---|---|---|---|---|---|---|---|
| Rosario Filho et al. [15] | 2021 | Systematic review and meta-analysis | Latin America | 22 observational studies | $PM_{2.5}$ $PM_{10}$, $NO_2$, $O_3$ | Associations between air pollutant exposure and AR in Latin American countries (OR [95% CI]) | The OR for developing AR in exposed individuals was 1.43 [1.026, 1.980]. Among children and adolescents, the OR was 1.36 [1.051, 1.759]. |
| Wang et al. [14] | 2021 | Cross-sectional study | China | 40,279 young adults | $PM_{10}$, $NO_2$ | Associations between in-home exposures and AR (OR [95% CI]) | Key risk factors associated with AR were as follows:<br>- High levels of urbanization, with lower odds in suburban areas (0.57 [0.50, 0.65])<br>- Elevated $NO_2$ concentrations (1.17 [1.06, 1.3])<br>- Proximity to heavy traffic (1.36 [1.22, 1.51])<br>- Home redecoration (1.16 [1.01, 1.34])<br>- Buying new furniture (1.19 [1.07, 1.32]) |

**Table 1. Selected studies on air pollution and AR** (*continued*)

| Author | Year | Study design | Location | Population | Pollutant | Outcomes | Result |
|---|---|---|---|---|---|---|---|
| | | | | | | | - Use of specific cooking fuels; natural gas (1.34 [1.13, 1.58]); coal/wood (1.35 [1.07, 1.69]) |
| Savouré et al. [19] | 2021 | Cross-sectional study | France | 127,108 adults | $PM_{2.5}$, BC, $NO_2$ | Associations between long-term exposure to air pollutants and self-reported rhinitis (OR [95% CI]) | Associations between long-term pollutant exposure and self-reported rhinitis were shown.<br>Current rhinitis:<br>- $PM_{2.5}$ (1.13 [1.08, 1.17])<br>- BC (1.12 [1.07, 1.17])<br>- $NO_2$ (1.11 [1.06, 1.17])<br>AR:<br>- $PM_{2.5}$ (1.15 [1.09, 1.21])<br>- BC (1.15 [1.09, 1.21])<br>- $NO_2$ (1.13 [1.08, 1.19])<br>NAR:<br>- $PM_{2.5}$ (1.13 [1.10, 1.17])<br>- BC (1.09 [1.04, 1.14])<br>- $NO_2$ (1.11 [1.06, 1.16]) |
| Li et al. [13] | 2022 | Systematic review and meta-analysis | 12 countries (Asia, Europe, America) | 35 observational studies | $PM_{2.5}$, $PM_{10}$, $NO_2$, $SO_2$, $O_3$, CO | Associations between air pollutant exposure and AR (OR [95% CI]) | The OR for associations between air pollutants and AR were as follows:<br>- $PM_{2.5}$ (1.12 [1.05, 1.20])<br>- $PM_{10}$ (1.13 [1.04, 1.22])<br>- $NO_2$ (1.13 [1.07, 1.20])<br>- $SO_2$ (1.13 [1.04, 1.22])<br>- $O_3$ (1.07 [1.01, 1.12])<br>No significant association was observed between CO and AR. |
| Carlsen et al. [20] | 2022 | Cohort study | Sweden | 37 adults | $PM_{2.5}$, $NO_x$, $O_3$, birch pollen | Associations between AR and co-exposure to air pollutants and birch pollen (OR [95% CI]) | $PM_{2.5}$ exposure was associated with rhinitis or eye irritation and increased allergy medication usage during the birch pollen season in the multi-exposure model:<br>- Rhinitis or eye irritation (1.16 [1.02, 1.32])<br>- Allergy medication usage (1.25 [1.07, 1.46])<br>$NO_x$ and $O_3$ did not show an effect on rhinitis or eye irritation.<br>Exposure to $NO_x$ was associated with significantly reduced OR for allergy medication usage in multi-exposure models (0.74 [0.59, 0.92]). |
| Lu et al. [64] | 2023 | Systematic review | 3 countries (China, Canada, | 8 observational studies | $PM_{2.5}$, $PM_{10}$, $NO_2$, BC, CO, $O_3$ | Associations between prenatal and early-life | All studies showed a positive association between exposure to |

**Table 1. Selected studies on air pollution and AR** (*continued*)

| Author | Year | Study design | Location | Population | Pollutant | Outcomes | Result |
|---|---|---|---|---|---|---|---|
| | | | and Germany) | | | exposure to TRAP and AR in children | TRAP during pregnancy and the first year of life and the development of AR in children. |
| Luo et al. [17] | 2023 | Cohort study | UK Biobank | 379,488 adults | $PM_{2.5}$, PM coarse, $PM_{10}$, $NO_2$, $NO_x$ | Associations between long-term exposure to air pollutants and the risk of AR (OR [95% CI]) | Significant associations were observed between the risk of AR and various pollutants:<br>- $PM_{2.5}$ (1.51 [1.27,1.79])<br>- PM coarse (1.28 [1.06,1.55])<br>- $PM_{10}$ (1.45 [1.20, 1.74])<br>- $NO_2$ (1.14 [1.09, 1.19])<br>- $NO_x$ (1.10 [1.05, 1.15]) |
| Maio et al. [18] | 2023 | Cohort study | Italy | 14,420 adults | $PM_{2.5}$, $PM_{10}$, $NO_2$, $O_3$ | Associations between long-term exposure to air pollutants and AR | Association between long-term exposure to air pollutants and AR (combined condition: prevalence of AR, symptoms, use of medicines):<br>- $PM_{2.5}$ (1.17 [1.06, 1.30])<br>- $PM_{10}$ (1.16 [1.06, 1.26])<br>- $NO_2$ (1.07 [1.04, 1.10])<br>- $O_3$ (0.99 [0.87–1.13]) (not significant) |
| Li et al. [21] | 2023 | Cohort study | China | 49 adults | $PM_{2.5}$ and its constituents | Associations between short-term exposure to $PM_{2.5}$ and its constituents and oxidative stress markers, symptoms, and quality of life in AR | Exposure to $PM_{2.5}$ and its constituents was associated with elevated scores on VAS and RQLQ.<br>Increased levels of nasal oxidative stress markers were observed, including elevated MDA and reduced SOD levels. |
| Li et al. [22] | 2024 | Cohort study | China | 49 adults | $PM_{2.5}$, $NO_2$, $SO_2$, $O_3$ | Associations between short-term exposure to air pollutants and oxidative stress markers, symptoms, and quality of life in AR | An association was observed between exposure to the air pollutant mixture and rhinitis symptom scale scores. No significant associations were found with VAS or RQLQ.<br>Increased levels of nasal oxidative stress markers were observed, including elevated MDA and reduced SOD levels. |

$PM_{2.5}$: particulate matter (PM) with diameter ≤ 2.5 μm; $PM_{10}$: PM with diameter ≤ 10 μm; $NO_2$: nitrogen dioxide; $NO_x$: nitrogen oxides; BC: black carbon; $SO_2$: sulfur dioxide; $O_3$: ozone; CO: carbon monoxide; AR: allergic rhinitis; NAR: non-AR; OR: odd ratio; TRAP: traffic-related air pollution; VAS: visual analog scale; RQLQ: rhinoconjunctivitis quality of life questionnaire; MDA: malondialdehyde; SOD: superoxide dismutase

## Air pollution and NAR

While most studies on air pollution and rhinitis have focused on AR, they often fail to distinguish the specific effects of pollutants on NAR. As a result, evidence directly addressing NAR remains limited and less well-defined [23]. A cross-sectional study involving 1,839 elderly participants from a nationally representative U.S. cohort found that long-term exposure to $PM_{2.5}$ was associated with rhinitis symptoms,

based on medication use. However, this study did not distinguish between AR and NAR, limiting its specificity for NAR [24]. Complementing these findings, the previously mentioned French cohort study provided more targeted insights into NAR, reporting significant associations between exposure to $PM_{2.5}$, BC and $NO_2$ and the prevalence of NAR—albeit at a slightly lower level than that observed for AR [19]. Furthermore, a time-series study from China focusing on children under 18 years old, clinically identified as having NAR, demonstrated a positive association between NAR outpatient visits and levels of $NO_2$, $SO_2$, $PM_{10}$, $PM_{2.5}$, $O_3$, and CO [25]. The synergistic effect of air pollution and pollen exposure on NAR has also been observed. A study involving 2,411 participants in China found that symptom severity during the pollen season was significantly associated with exposure to $NO_2$, $SO_2$, $PM_{2.5}$, and $PM_{10}$. Notably, no such association was observed during non-pollen seasons, suggesting that pollutant exposure may amplify allergic-like symptoms even in non-allergic individuals, possibly through mechanism such as nasal hyperreactivity [26].

## Pathophysiological mechanisms of air pollution-induced rhinitis

### Air pollution-induced inflammation and injury

AR is characterized by the accumulation of inflammatory cells in the nasal tissue and secretions, along with structural changes in the mucosa, resulting from an IgE-mediated type I hypersensitivity response to inhaled allergens. The nasal epithelium serves as a pivotal physical barrier of mucosa, detecting and responding to microbes, allergens, and air pollutants, thereby regulating both innate and adaptive immune response. This barrier function relies on the integrity of complex intercellular junctions, which include apical tight junctions (TJs) and underlying adherent junctions. Epithelial cells also release chemokines, and cytokines, such as thymic stromal lymphopoietin (TSLP), interleukin (IL)-25 and IL-33, which act as alarmins, and are crucial in initiating and maintaining mucosal immunity [27]. Disrupting of epithelial integrity allows allergens to penetrate the epithelium and interact with inflammatory cells [28, 29]. Numerous studies have investigated the effect of air pollution on respiratory epithelial cells, revealing several mechanisms that underlie its harmful effects (Figure 1). For example, in vitro and ex vivo exposure of nasal epithelial cells to air pollutants, such as $PM_{2.5}$ or diesel exhaust particles (DEP), resulted in barrier dysfunction, increased epithelial permeability, and reduced transepithelial electrical resistance (Figure 2). This is due to the downregulation of junctional proteins such as claudin-1, occludin, and zonula occludens-1 (ZO-1), as well as the enhanced release of pro-inflammatory cytokines IL-8, tissue inhibitor of metalloproteinases-1 and TSLP with proinflammatory action [30–35]. Additionally, $PM_{2.5}$ exposure has been shown to cause nasal mucosal damage, loss of cilia, goblet cell metaplasia, and submucosal gland hypertrophy, leading to increased mucus secretion [36–38]. Animal models exposed to $PM_{2.5}$ exhibited local inflammatory responses characterized by immune cell infiltration and increased expression of pro-inflammatory cytokines (IL-1, IL-6, TNF-α), IL-4, IL-13, TGF-β1, and eosinophil chemokine eotaxin-1 [36, 39, 40]. Consequently, these effects may increase susceptibility to AR and exacerbate its symptoms, particularly in areas with high pollution levels.

### Interaction between air pollution and AR

Data suggest that environmental pollutants can interact with allergens, amplifying the allergic response in sensitized individuals [41]. Human challenge studies have demonstrated that nasal exposure to DEP increases local IgE production, the number of IgE-secreting cells, and the release of various cytokines [42, 43]. Interestingly, co-exposure to DEP and keyhole limpet hemocyanin (KLH) promoted the transition of humoral response to KLH-specific IgE production as opposed to the generation of IgG and IgA seen with KLH alone [44]. Similarly, co-challenge of DEP and purified ragweed allergen, Amb a 1, amplified localized T-helper (Th) 2 immune responses, significantly increasing ragweed-specific IgE and IgG4 levels, and elevating Th2 and proinflammatory cytokines (IL-4, IL-5, IL-6, IL-10, and IL-13) while reducing Th1 mediators [interferon (IFN)-γ] [45]. Animal studies confirmed that intranasal administration of DEP or $PM_{2.5}$ worsened allergen-induced AR by increasing nasal symptoms (e.g., sneezes, nasal rubs), boosting total and allergen-specific IgE production, and disrupting nasal mucosal TJs. This is likely due to the

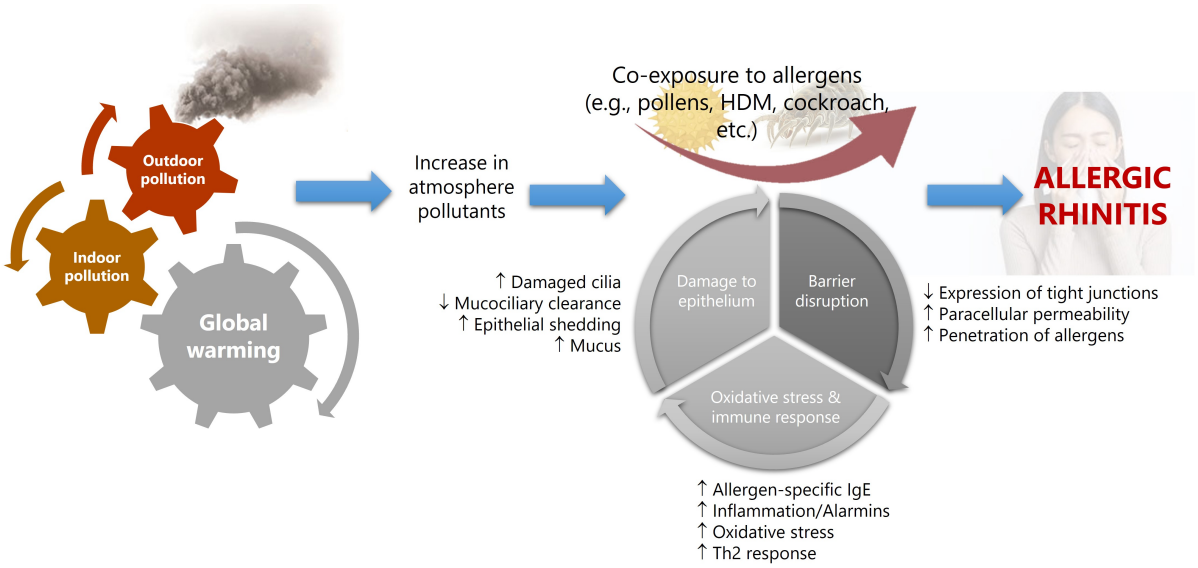


**Figure 1. Impact of air pollution on allergic rhinitis.** Th: T-helper. Modified from [27], CC BY. The dust mite illustration was generated by the authors using Google Gemini (an AI tool)

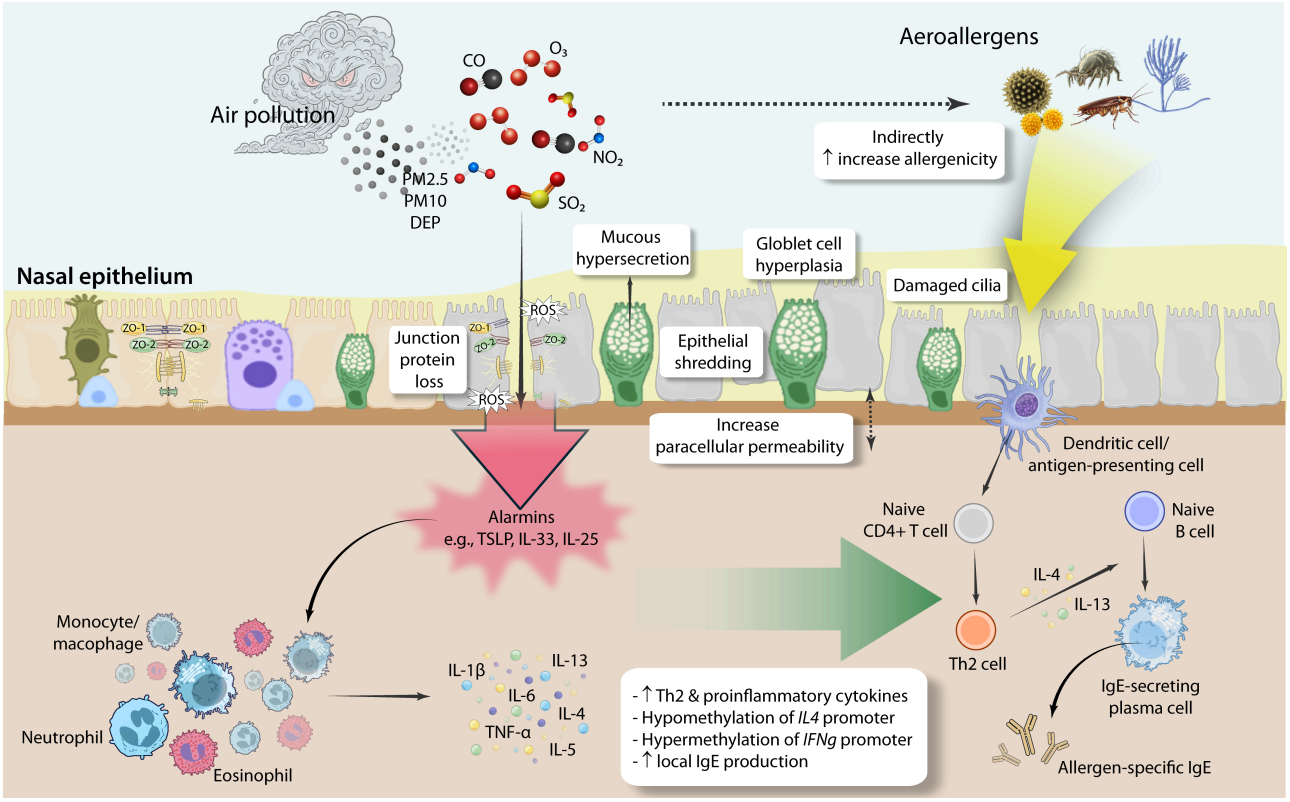


**Figure 2. Potential molecular mechanisms by which air pollution may exacerbate allergic rhinitis.** PM: particulate matter; DEP: diesel exhaust particles; TSLP: thymic stromal lymphopoietin; IL: interleukin; TNF: tumor necrosis factor; Th: T-helper; IFN: interferon. Figure 2 was created using Adobe Illustrator. The authors used Google Gemini (an AI tool) to generate the dust mite, air pollution, and cockroach illustrations. In addition to the elements mentioned above, the other illustrations of Figure 2 were adapted from NIAID NIH BIOART Source (https://bioart.niaid.nih.gov/) and Servier Medical Art (https://smart.servier.com/), licensed under CC BY 4.0

decreased expression of nasal mucosal ZO-1, increased oxidative stress, goblet cell hyperplasia, mucus hypersecretion, and an imbalance in Th1/Th2 cytokines, along with enhanced eosinophil chemotaxis [46–51]. Oxidative stress induced by inhaling DEP intensified allergic inflammation, resulting in an accumulation of inflammatory cells (neutrophils and eosinophils) and increased cytokine production (IL-5, IL-6, and IL-8) [52]. In addition, inflammatory biomarkers like eosinophil cationic protein (ECP) and monocyte chemotactic protein (MCP)-1 are elevated [53]. The severity of this inflammatory response varies

depending on individual health status and the specific environmental cues involved [54]. In line with previous findings, a double-blind, placebo-controlled study involving individuals with and without AR demonstrated that DEP exposure led to increased eosinophil activation, elevated eotaxin-1 and ECP levels, and heightened viral loads after virus inoculation [55, 56]. Subsequent research further revealed that DEP prolonged eosinophil activation through reduced natural killer cell activity, suggesting a potential mechanism underlying the amplification of allergic responses [57].

### Epigenetic alterations from air pollution exposures

Environmental factors can also influence epigenetic alterations, such as DNA methylation, histone modifications, and non-coding RNA changes, which are usually modifications that lead to changes in gene expression without affecting the DNA sequence. Recent research has linked epigenetic changes in immune-related genes, particularly those involved in Th1/Th2 responses or regulatory T cell differentiation, to the development of allergic diseases after exposure to environmental pollution [58, 59]. For example, reduced DNA methylation in the promoter of *IL4* gene has been observed, potentially leading to its increased expression in response to higher BC exposure [60]. Conversely, exposure to secondhand smoke and ambient air pollution has led to significant increases in DNA methylation and reduced expression of IFN-γ in effector T cells and Foxp3 in regulatory T cells in children [61]. Similarly, inhaled DEP augmented hypomethylation of the *IL4* gene promoter and hypermethylation of the *IFNg* promoter in helper T cells from mice sensitized intranasally to the fungus allergen *Aspergillus fumigatus*, leading to a shift of immune program towards Th2 polarization [51]. The magnitude of this demethylation was particularly pronounced in IgE-sensitized children, especially those atopic to cockroach allergens [60]. Furthermore, maternal and in utero exposure to air pollution has already been associated with aberrant fetal lung development and immune function, contributing to susceptibility to respiratory disease and atopy [62–66]. This partially attribute to epigenetic modifications, as certain mechanisms are known to be capable of transferring through multiple subsequent generations in several conditions [67–70], yet the impact of transgenerational epigenetic inheritance from air pollution on AR remains unexplored.

### Neuronal mechanisms

In addition, environmental pollution can trigger rhinitis through a neurogenic mechanism mediated by the trigeminal nerve. This mechanism likely involves mast cell degranulation, epithelial cell activation, central autonomic reflexes, and local axon reflexes [71]. The peripheral respiratory reflex to irritant exposure manifests as rhinorrhea, nasal obstruction, sneezing, coughing, and laryngospasm [41], which, in combination with allergens, can further amplify the allergic response.

### Indirect effects of air pollution on rhinitis

Interestingly, some air pollutants may indirectly influence AR by affecting the production and composition of allergens at their sources. For instance, research in Germany revealed more birch allergen (Bet v 1) and immunomodulatory pollen-associated lipid mediators (PALMs), which can promote Th2 cells to stimulate IgE production, per pollen grain produced by birch trees exposed to higher $O_3$ concentrations than those not exposed to $O_3$ [72]. Allergenic extracts from highly exposed trees exhibited greater allergenicity, eliciting larger wheal reactions in skin prick tests in birch pollen-allergic patients in comparison to those from less exposed trees. Notably, pollen collected near high-traffic roads also released higher abundance of eicosanoid-like substances as well, suggesting a stronger proinflammatory activity of these pollen grains [73]. Pollutions such as $NO_2$ and $O_3$ can also directly interact with allergens, damage the pollen envelopes facilitating the release of allergenic substances, and increase their immunogenicity and stability through oxidation or nitration [74]. Several studies have demonstrated that nitrated fungal spores and nitrated pollen from birch, ragweed, and hornbeam increased allergic sensitization, have a heightened capacity to activate T cells and induce IgE production, contributing to increased allergic sensitization [72, 75–78].

In summary, air pollution can exacerbate rhinitis through multiple interrelated mechanisms. Air pollutants compromise nasal epithelial barrier, increase proinflammatory cytokine production, disrupt

immune regulation and affect neurogenic pathways, leading to enhanced allergen penetration and inflammation. These amplify IgE-mediated responses, promote Th2 polarization, and worsen AR symptoms via oxidative stress, immune cell infiltration, and epithelial damage. Additionally, air pollution can cause epigenetic modifications that alter immune gene expression, potentially increasing allergic susceptibility across generations. Pollutant-altered allergen properties further contribute to heightened allergic responses and disease severity.

## Economic impact

Pollution imposes substantial economic burdens through productivity losses and increased healthcare costs. In low- to middle-income countries, pollution-related diseases reduce gross domestic product (GDP) by up to 1.9% annually, while healthcare expenditures account for 3.5–7% of total spending [79].

For individuals with AR, pollution exacerbates symptoms, leading to increased outpatient visits and medical expenses. For example, a study in Beijing (China) tracking 33,599 AR outpatient cases from 2014 to 2019 found that a 10 $\mu g/m^3$ increase in $PM_{2.5}$, $PM_{10}$, $NO_2$, and $SO_2$ corresponded to rises in outpatient visits by 1.24%, 0.79%, 3.05%, and 5.01%, respectively [80]. Similar trends were observed in Lanzhou, China, where multiple pollutants, including $PM_{2.5}$, $PM_{10}$, $SO_2$, $NO_2$, $O_3$, and CO, significantly correlated with healthcare utilization [81].

Pollution also reduces workplace productivity by exacerbating symptoms such as nasal congestion, sneezing, and fatigue, leading to absenteeism or decreased performance. Indirect costs from reduced productivity often surpass direct healthcare expenses, making rhinitis a significant economic concern [82].

Regarding healthcare expenditures, most pollution-focused AR studies are based in Asian regions. A time-series analysis in Seoul, South Korea (2016–2019), reported that increases in $PM_{10}$, $NO_2$, and CO levels were associated with rises in medical costs for AR, with each standard deviation increase in pollutant levels linked to a 6.22% to 11.27% increase in medical expenses [83]. In Beijing (China), data from a tertiary hospital (2013–2015) further demonstrated that $PM_{2.5}$ significantly increased healthcare costs related to respiratory diseases and was associated with increased $PM_{2.5}$ levels [84].

In Taiwan (China), analysis of national health insurance data (2006–2012) revealed that a one-unit reduction in the Air Quality Index could reduce respiratory-related outpatient expenditures by approximately 74 million USD annually. This corresponds to a potential 8% reduction in national health spending attributed to air pollution [85].

In terms of indirect costs, most existing research has addressed general AR rather than pollution-induced cases specifically. A global cross-sectional study using data from the MASK-air application (2005–2023) showed that poor AR control, as assessed by a visual analogue scale, led to work impairment in up to 60.7% of reported weeks. During these periods of poor control, productivity loss translated to indirect costs ranging from USD 65.7 purchasing power parities (PPP) in Brazil to USD 693.6 PPP in Iceland. Although the study was not limited to pollution-induced AR, it provides a valuable approximation of the broader economic burden. An online tool is available to estimate annual country-specific productivity losses related to AR [86]. In comparison, a multi-country study conducted in several Asian nations estimated that the indirect costs for patients with inadequately managed AR and urticaria (2015–2016) reached up to USD 105.4 billion annually, equating to USD 1,137–2,195 per patient from absenteeism and presenteeism. Again, while these findings do not isolate pollution-induced cases, they underscore the significant productivity losses associated with poorly controlled AR [87].

## Mitigation strategies for pollution-related AR

Several measures have been shown to reduce rhinitis symptoms and minimize exposure to air pollution.

### Avoidance and reduced exposure

Monitoring air pollution is vital to safeguarding vulnerable populations. Localized air quality monitoring tools provide critical information, enabling individuals to limit their exposure to harmful pollutants. Public

education campaigns should raise awareness about pollution's health impacts, promote behavioral changes to reduce exposure, and encourage compliance with regulatory guidelines to lower pollutant levels. Addressing key risk factors, such as tobacco smoke, indoor biomass fuel usage, and poorly planned urban developments, can significantly mitigate exposure risks [54]. Personal protective measures, such as nasal filters or high-quality masks, offer an additional layer of defense. Although nasal mucosal barriers show potential, more evidence is needed to support their widespread use.

### Air filtration and ventilation

High-efficiency particulate air (HEPA) filters are effective in removing particles as small as 0.3 microns, significantly reducing allergic and infectious triggers of respiratory diseases [88, 89]. Regular ventilation of indoor spaces, combined with air filtration, minimizes indoor pollutant concentrations, curbs mold growth, and reduces indoor $NO_2$ levels. Additionally, preventive measures like managing indoor dampness and limiting carpets can lower exposure to allergens like house dust mites and molds [90].

### Medical management

For patients with pollution-exacerbated AR, treatment plans should incorporate environmental triggers. Nasal corticosteroids, non-sedating antihistamines, and saline irrigation are commonly recommended to alleviate symptoms [54]. Allergen immunotherapy may also benefit patients with severe, pollution-induced allergic symptoms.

### Lifestyle adjustments

Lifestyle modifications can significantly reduce individual contributions to air pollution while improving health outcomes. Strategies include reducing the use of motorized transportation, avoiding outdoor activities during high-pollution periods, and utilizing mobile health tools such as air quality alerts, pollen calendars, and symptom trackers [91, 92]. Additional efforts to minimize carbon footprints, such as limiting meat consumption, reducing air travel, increasing the use of recyclable materials, and avoiding household irritants like incense, candles and domestic sprays, can further benefit environmental and respiratory health [91].

A combination of these strategies often proves most effective in mitigating pollution-related AR. Addressing this complex issue demands robust collaboration and clear communication across various sectors. Public health authorities are crucial in providing the overarching framework through comprehensive surveillance, widespread education initiatives, and effective policy advocacy. Environmental regulators play a vital role by focusing on controlling and substantially reducing pollution at its sources. Healthcare professionals hold the primary responsibilities of accurately diagnosing, effectively managing, and thoroughly educating individuals affected by this condition. Strong collaboration among these sectors is essential for developing and implementing comprehensive solutions that safeguard public health and significantly improve the quality of life for those suffering from pollution-induced rhinitis.

## Limitations and research gaps

The majority of available studies, including meta-analyses, support an association between air pollution and rhinitis symptoms or prevalence. However, some conflicting evidence still exists in the literature [23, 93], contributing to uncertainty in the strength and consistency of this association. Several key limitations likely underline the heterogeneity in findings across different geographic regions. First, most air pollution research focuses on lower airway diseases (such as asthma or COPD), while the upper airways, especially rhinitis, receive comparatively less attention. Second, the heterogeneity of rhinitis phenotypes may result in differential immunological responses to pollutants. For instance, NAR is a prevalent but understudied subtype that may respond differently to environmental triggers. As previously mentioned, there is a significant lack of targeted research exploring NAR in the context of air pollution. Third, the difficulty in distinguishing rhinitis phenotypes poses a major challenge. Clinical tools for phenotyping, such as the nasal provocation test for identifying local AR, are not widely accessible in routine clinical practice. This lack of

diagnostic clarity introduces a potential confounding factor, especially in studies aiming to isolate the impact of pollutants on NAR populations. Fourth, geographical and environmental differences significantly affect study outcomes. Variability in pollutant sources, urbanization levels, pollen exposure, and climatic conditions across regions may partly explain the inconsistencies in findings [94]. While it is generally assumed that pollutant exposure is more intense in developing countries, studies show that prevalence rates of rhinitis remain high in both developed and developing settings. Meta-analyses have revealed a consistent trend toward positive associations between air pollution and AR across regions and income levels [13].

Specifically, higher associations were observed in developed countries for pollutants such as $PM_{2.5}$, $PM_{10}$, $SO_2$, $O_3$, and CO, although statistical significance was only reached for $NO_2$, which showed a higher effect size in developing countries. These findings suggest that in addition to pollutants, other contextual factors such as lifestyle patterns, traffic density, and climate conditions may amplify the risk of AR in developed countries [95].

To resolve some of these uncertainties, nasal challenge studies using controlled exposure to specific pollutants could provide valuable mechanistic insights. This method is analogous to allergen exposure chambers, which have advanced our understanding of AR. Previous studies using DEP for nasal challenge have demonstrated that DEP can induce oxidative stress, exacerbate allergic inflammation, and promote nasal hyperresponsiveness, while also enhancing neoallergen sensitization as previously described [96].

However, it is critical to note that while DEP is a major component of PM, it does not represent the full spectrum of PM sources. Attention to $PM_{2.5}$ is based on particle size (≤ 2.5 μm), encompassing a wide range of chemical compositions and sources, including industrial emissions, biomass burning, and road dust. The true molecular and immunological impact of $PM_{2.5}$ on the nasal mucosa in humans remains poorly understood. There is a pressing need for studies that use defined PM components or representative PM mixtures as provocation substances in controlled human exposure models. Current evidence largely relies on environmental monitoring (e.g., area-based or personal exposure data), which may not accurately reflect individual-level effects. So far, only a limited number of studies have employed $PM_{2.5}$ directly as a nasal challenge substance in human models [97, 98].

## Conclusion

A growing body of evidence highlights the significant role of air pollution in the development and worsening of rhinitis. Pollutants not only trigger and aggravate symptoms but also contribute to chronic inflammation and immune system dysfunction, posing a substantial global health burden. Given the economic and societal consequences, targeted interventions ranging from air quality regulations to personal protective measures are crucial in reducing the impact of pollution on respiratory health. Future research should focus on improving pollution exposure assessments, understanding long-term health effects, and developing innovative treatment strategies for pollution-related rhinitis. Addressing this challenge requires coordinated efforts among public health authorities, environmental policymakers, and healthcare professionals to implement effective prevention and management strategies.

## Abbreviations

AR: allergic rhinitis

BC: black carbon

CO: carbon monoxide

COPD: chronic obstructive pulmonary disease

DEP: diesel exhaust particles

IFN: interferon

IL: interleukin

KLH: keyhole limpet hemocyanin

NAR: non-allergic rhinitis

$NO_2$: nitrogen dioxide

$NO_x$: nitrogen oxides

$O_3$: ozone

PM: particulate matter

$SO_2$: sulfur dioxide

Th: T-helper

TSLP: thymic stromal lymphopoietin

# Declarations


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During the preparation of this work, the authors used Google Gemini (AI tool) to generate the dust mite illustration in Figure 1, and the dust mite, air pollution and cockroach illustration in Figure 2. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the final presentation in the publication. In addition to the elements mentioned above, the other illustrations of Figure 2 were adapted from NIAID NIH BIOART Source (https://bioart.niaid.nih.gov/) and Servier Medical Art (https://smart.servier.com/), licensed under CC BY 4.0.


## Author contributions

DK: Conceptualization, Writing—original draft. NL: Conceptualization, Visualization, Writing—original draft. PT: Conceptualization, Writing—review & editing.

## Conflicts of interest

Pongsakorn Tantilipikorn, who is the Editorial Board Member and Guest Editor of *Exploration of Asthma & Allergy* had no involvement in the decision-making or the review process of this manuscript. Dichapong Kanjanawasee, who is the Guest Editor of *Exploration of Asthma & Allergy* had also no involvement in the decision-making or the review process of this manuscript.

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Not applicable.

## Consent to participate

Not applicable.

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