Review article

Interaction Between Air Pollutants and Pollen Grains: The Role on the Rising Trend in Allergy

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Abstract

Asthma and allergic diseases cases have risen in recent decades. Plant pollen is considered as the main aeroallergen causing allergic reactions. According to available data, urban residents experience more respiratory allergies than rural residents mainly due to the interaction between chemical air pollutants and pollen grains. This interaction can occur through several mechanisms; chemical pollutants might facilitate pollen allergen release, act as adjuvants to stimulate IgE-mediated responses, modify allergenic potential, and enhance the expression of some allergens in pollen grains. This review focuses on the most recent theories explaining how air pollutants can interact with pollen grains and allergens.

Keywords: Allergens, Chemical air pollutants, pollen, respiratory allergy, Urban air pollution.

Introduction

Cases of asthma and allergic diseases have increased in recent decades (1-3). Airborne allergens derived from plant pollen are considered as the main aeroallergens and a major cause of type I hypersensitivities such as asthma and allergic rhinitis (4). Previous studies indicate that aeroallergens are responsible for about 63%, and pollen for more than 92%, of allergic rhinitis cases, (5, 6).

Considering the crucial role of natural resources in modulating daily life stress and violence, green spaces have been widely cultivated worldwide. However, despite the undeniable advantages for public health, some problems have arisen from urban planting. The most serious issue might be the growing trend in sensitization to pollen, especially in urban settings (7, 8); in fact, people living near heavy traffic are affected with pollen-induced respiratory allergies more than those in rural districts (9). The sudden rise in environmental pollutant levels due to industrial development and urban motor vehicle traffic has affected air quality and consequently, the severity and mortality from allergic diseases (10). Some evidence suggests that air pollution might cause new cases of asthma as well (9, 11).

The effect of air pollutants on respiratory allergies depends on a combination of factors including components and concentrations of environmental pollutants, exposure duration, ventilation, climatic conditions, and the interaction between pollutants and pollen (Table 1) (9, 12). Regarding the last issue, air pollutants might damage the pollen cell wall, facilitating allergen release into the environment and penetration into the lower respiratory tract (13-15). These pollutants can interact with allergen-carrying small particles, which pass through the airway and cause asthma symptoms in predisposed subjects (16). Also, the allergenic potential of allergens could be enhanced through contact with chemicals. Furthermore, some components of air pollutants serve as adjuvants and stimulate IgE-mediated allergic responses (17). Finally, a number of publications appearing in recent years considered air pollution as a stressor that increases the expression of some allergens in pollen grains (17-19).

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The aim of this review is to present the most recent proposed mechanisms through which air pollutants can interact with pollen grains and allergens.

Species	Effect of pollutants on pollen	Reference
Zinnia elegans	Accumulation of new particles on pollen surface, lower protein content of pollen grains	(13)
Phleum pratense	Increased release of pollen cytoplasmic granules	(14)
Cupressus arizonica	Increased fragility of pollen exine, increased allergenicity	(20)
Poa annua L.	Increased release of pollen cytoplasmic granules, increased allergenicity	(19)
Platanus orientalis	Presence of new particles on the pollen surface, higher total protein content in pollen, more intensive proteins in pollen extracts, higher Pla or 1 levels	(21)
Lolium perenne	Increased in vitro and in vivo allergenicity, increased total protein content	(22)
Cupressus arizonica	Increased in vivo allergenicity, increased expression of Cup a 3 allergen	(17)
Betula pendula	Increased the allergenicity of Bet v 1a allergen through the formation of nitrotyrosine residues	(23)
Acer negundo	Increased allergenicity of pollen	(24)
Ambrosia artemisiifolia L.	Increased in vitro allergenicity of pollen	(15)
Betula pubescens	Increased allergenicity thorough induction of IL-5 expression	(25)
Betula	NO2 induces nitration of allergen and favors TH2 responses, leading to the higher allergenicity of pollen in urban areas	(26)
Aspergillus fumigatus	Increased allergenicity of pollen via nitration of allergen	(27)
Lolium perenne	Binding of the major grass pollen allergen, Lol p I, and thus its concentrating in polluted air	(28)
Phleum pratense	Decreased allergen content, decreased IgE recognition	(29)

Table 1. Studies on the effects of air pollutants on pollen allergens

Interaction between air pollutants and allergen-carrying particles

Pollen grains should be considered not only as allergen carriers; they can elicit allergic responses in sensitized individuals as well. In fact, people are more likely to be exposed to allergen-releasing particles, such as pollen grains or granules, than to pure allergens (16, 30). Pollen grains are 15-40 µm in size; thus, they cannot penetrate into the lower regions of the respiratory tract. Hence, the asthma symptoms frequently observed in pollen-allergic patients are most likely due to the presence of pollen-derived particles, which are smaller than pollen grains and can induce asthma-related symptoms. Interestingly, air pollutants bind to these particles and exacerbate allergic disorders. Moreover, pollen grains release biologically active lipids, which activate immune cells in vitro (16).

Cell wall damage, allergen release, and distribution in the environment

The interaction between air pollutants and pollen grains might damage the pollen cell wall, increasing the amount of allergens released into the environment. Experiments on *Platanus orientalis* pollen performed by Lu et al. revealed new kinds of particles on the pollen surface after exposure to a mixture of pollutant gasses (21). Chehregani et al. confirmed the accumulation of sub-micronic particles on the surface of *Zinnia elegans* pollen grown in the presence of pollutants, which resulted in an increase in allergen release into the environment and increased pollen allergenicity (13). This group suggested that air pollutants can elicit allergic reactions, but the allergenic potential might be enhanced after binding to pollen grains. Morphologic changes in pollen grains induced by air pollutants have also been observed. Scanning electron microscopic (SEM) investigation by Shahali et al. showed that the fragility of pollen exine is increased two weeks after microsporangia dehiscence (20). Therefore, allergens bound to pollen release faster and absorb to the airway mucus easier than unbound allergens, leading to induction of allergic responses. In addition, the release of pollen cytoplasmic granules (PCGs) has been shown to increase after pollen exposure to chemical compounds (14, 19). Such findings indicate this mechanism as a reason for the recent increase in allergic diseases.

Increasing allergenicity through interaction with air pollutants

Air pollutants can modify pollen allergenicity through interaction with allergens by various mechanisms (16). Armentia et al. compared the allergenicity of samples gathered from different areas during the pollination period and found the in vitro and in vivo allergenicity of grass pollen was greater in urban than in rural areas (22). In addition, in skin prick tests, the in vivo allergenicity of *Cupressus arizonica* pollen extracts was greater from polluted than from unpolluted sites. (17). Also, in vitro allergenicity of *Ambrosia artemisifolia* pollen grown near heavy traffic was greater than that of pollen from rural districts as measured by Ghiani et al., using IgE reactivity of sera from people sensitized to this pollen (15). This group suggested that the higher allergenicity of pollen from polluted sites might contribute to higher allergen levels in these areas or conformational transformations induced by post-translational modifications. Others have suggested that the modified allergenicity is due to induction of previously unexpressed proteins in exposed pollen reacting with patients' sera (19, 25). For example, the high prevalence of an allergen in subjects allergic to *Cupressus arizonica* was observed in people living in Tehran (20). As this allergen was not observed in the available commercial extracts used for diagnostic experiments, optimization of standard allergen preparations seems necessary, taking into account the variability of allergenic components for each region.

In another study, the ability of nitrated and non-nitrated Bet v 1a allergen was analyzed for its ability to induce proliferation and cytokine release from spleen cells of sensitized BALB/c mice, and its ability to elicit IgE production (23). It was found that that NO_2, a major traffic-related air pollutant, enhanced the allergenicity of Bet v 1a and its affinity for IgE through the formation of nitrotyrosine residues.

Other studies also found that NO_2 induces allergen nitration and favors T helper 2 (Th2) responses, leading to greater allergenicity of pollen in polluted areas and increased risk for human health (24-26). Furthermore, Yona reported that the allergenicity of *Conidia* increased by 2–5 fold for exposure times up to 12 hours, but decreased as time elapsed, probably because of protein deamidation. This observation suggested the presence of higher-potency allergens in the environment mostly during the first 12 hours of exposure to air pollutants (27).

Adjuvant effects of air pollutants

Several air pollutants act as adjuvants through binding to allergens and stimulating IgE synthesis, resulting in exacerbation of asthma symptoms (16, 31-33). Previous studies considered diesel exhaust particles (DEPs), which aggravate allergic reactions and even cause sensitization to a new allergen, as the most important adjuvant in the environment (34-37). In addition, some research indicated that pollen grains can induce activation and maturation of dendritic cells. These observations implicate pollen not only as an allergen carrier, but also as an adjuvant in triggering immune responses (16, 38).

Induction of allergenic protein expression

Recent research has focused mainly on the impact of environmental pollutants on the expression of allergenic proteins. In a study by Armentia et al, total protein content was found to be greater in pollen gathered from urban than from rural settings (22). This finding was similar to those of Lu et al., who observed increased protein content in pollen extracts after exposure to pollutant gasses (21). Also, this group found increased levels of the *Platanus orientalis* major allergen Pla or 1 in exposed pollen. In contrast, other studies found lower distribution (7) or reduced protein content in pollen grains from urban environments (13). Chehregani et al. suggested that the observed decrease in protein content is probably due to the release of pollen material though the damaged pollen exine (13). Moreover, Rogeireux et al. pointed out that the decline in pollen allergen levels caused by gaseous pollutants could be due to their alteration induced by post-translational modifications, resulting in loss of recognition by IgE (29).

Recently, the effect of air pollution, as an environmental stressor, on allergens categorized as pathogenesis-related (PR) proteins, has attracted much attention (12). These proteins are part of a plant defense system that plays a key role in protecting plants from undesirable conditions, such as pathogen attacks, drought, and freezing

temperature (39-48). To date, few studies have examined the impact of air pollution on these proteins. For instance, Cortegano et al. reported that Cup a 3 allergen, a PR protein family member (PR5), was more abundant in polluted than in unpolluted conditions (17), a finding confirmed by Suarez-Cervera in 2008 (18). These studies noted that cypresses increase allergen expression as a defense strategy against pollution, which can also affect pollen allergenicity. In addition, Aina et al. reported the presence of previously unknown allergenic proteins, including a PR3 class I kinase, in cadmium-exposed, but not in other, pollen samples (19). Air pollution is a serious public health issue (9). During the past few decades, a growing body of evidence points to an association between urbanization and the rising trend in allergic diseases, suggesting air pollution as a major cause for this trend. Studies have shown that people living in urban areas experience more severe and prevalent allergic symptoms than those in rural areas. As pollen is an important source of outdoor allergens, pollen allergy is frequently studied to characterize the relationship between air pollution and respiratory allergy (9). Based on the data presented, chemical compounds might aggravate allergic responses by disseminating allergens out of pollen via pollen cell wall damage, acting as adjuvants, modifying pollen allergenicity, intensifying allergen expression, and inducing new allergens. To prevent further economic and health costs related to allergic diseases, municipal governments must make urgent decisions regarding pollution and urban planting.

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