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REVIEW

Pollen deposition in intrathoracic airways

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Pollen deposition in intrathoracic airways. M.N.B.M. Driessen, Ph.H. Quanjer.

ABSTRACT: The pathophysiological mechanisms of pollen-induced asthma have until now remained unclear, because pollen particles have generally been considered too large to penetrate into the lower respiratory tract. Such grains are therefore believed to be unable to induce an immunological response in the lower respiratory tract. There is evidence, however, that a small percentage of large particles (20–30 μm) may penetrate into the peripheral areas of the lung. It also appears that small airborne units of less than 5 μm with the same antigenic activity as pollen, may contribute to inhaled allergen burdens.

Pollen asthma probably results from a gradual cumulative effect of deposition of small amounts of allergen in lower airways, and is hence poorly correlated with daily pollen counts.

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It is generally assumed that larger particles such as grass pollen grains (about 30 μm) cannot penetrate into the lower airways; however, a number of pollinosis patients develop pollen asthma during the grass pollen season. The general view is that all pollen grains are trapped in the nose or oropharyngeal cavity and are then believed to only directly cause symptoms of rhinitis and conjunctivitis. An explanation of pollen asthma might be sought in reflex mechanisms or in the presence in the atmosphere of small fragments of pollen grains which are capable of penetrating into deeper levels of the respiratory tract.

The lungs and respiratory tract can be regarded as a system, which, in adults, is exposed to the outside world *via* a contact surface area measuring about 70 m^2. This exposure is limited by two factors: ventilation, which normally amounts to 10,000–20,000 *l* (10–20 m^3) per day, and the fact that the upper airways act as a filter and air conditioner, thus protecting the intrathoracic compartment. During the pollen season atmospheric air contains 250–1,000 pollen grains per m^3, so that individuals are exposed to some 2,500–20,000 pollen grains daily *via* the air they inhale. This corresponds with an estimated exposure to 0.1–10 $ng \cdot day^{-1}$ or <1 g $allergen \cdot yr^{-1}$ [1, 2].

The question of whether pollen can penetrate into the lower airways and directly cause an asthmatic reaction is generally answered in the negative, because it is assumed that virtually all particles with an aerodynamic diameter in excess of 10 μm are trapped in the upper airways [3–6]; pollen grains with wind pollination have a diameter of 10–100 μm, mostly between 20–30 μm [7, 8]. Another argument against deposition in intrathoracic airways is the fact that in asthma [9, 10] and pollinosis [11–13] there is no, or only a poor correlation between clinical symptoms and pollen exposure, while in the laboratory pollen inhalation does not lead to bronchoconstriction [5, 9].

We will briefly review relevant determinants of deposition of particles in the airways, experimental observations on the site of deposition and attempt to achieve a synthesis concerning the relationship between pollen exposure and clinical symptoms.

Determinants of particle deposition

Inhaled particles are deposited on the wall of the respiratory tract by impaction, sedimentation and diffusion [6, 14, 15]. In impaction, mass inertia plays a role, and consequently impaction is proportional to the linear velocity of a particle, its angle of deviation from the linear, and the square of its aerodynamic diameter (d^2). The concept of aerodynamic diameter entails correction for particle density as well as for size. Particles of an equal aerodynamic diameter have equal sedimentation and inertial behaviour. The aerodynamic diameter of spherical particles is proportional to the physical (microscopic) diameter and to the square root from the particle density in $g \cdot cm^{-3}$ [16].

The airways may be depicted as a ramifying system of tubes, which from the trachea down gives rise to approximately dichotomous divisions, in which the diameter of each daughter branch is about 0.8 times that of the mother branch. Hence, the velocity of particles diminishes progressively during the passage from trachea to alveoli as the total cross-sectional area increases. According to the morphometric data of WEIBEL

[17] concerning particle velocity at the 16th order of branches (*i.e.* at the level of the smallest lobular bronchi), the surface area relative to that of the trachea has increased by a factor of 73, so that the velocity is about 1% of that in the trachea. Owing to their higher mass, which varies as the cube of the diameter, nearly all particles with a diameter exceeding 10 μm are impacted onto the nasopharyngeal mucosa, whereas smaller particles can reach the tracheobronchial tree [6, 14, 15]. Such airborne particles may subsequently be removed by impaction at bifurcations, but as the velocity diminishes progressively, sedimentation and diffusion become more important. Because the average distance of particles from the wall becomes progressively less towards the alveoli, deposition by diffusion and sedimentation plays an ever-increasing role towards the periphery. Deposition at an alveolar level mainly involves particles of 0.5–5 μm, in intrathoracic airways mainly 5–10 μm; 50% of all particles of 0.5 μm are trapped in the lung parenchyma [6].

The reader is reminded of the fact that, whilst theory predicts that the number of larger particles which reach the intrathoracic airways is extremely small, the volume of a spherical particle varies as the third power of its diameter; hence a particle of 10 μm brings 8,000 times as much allergen into the lungs as one of 0.5 μm. The pollen diameter is 10–100 μm. Since only an occasional pollen grain can slip past the nasopharynx (no more than 1–2% in nasal breathing, but more in oral breathing [6]), and even then will be trapped in the largest intrathoracic airways, the question arises how pollen can ever give rise to asthmatic reaction.

Extrapulmonary mechanisms

In an effort to explain that pollen provocation unmistakably elicits reactive bronchial obstruction in some patients, a reflex arising from the nose or nasopharynx has been postulated. In dogs, electrical and mechanical irritation of the nasal mucosa can cause bronchoconstriction, which is abolished by vagotomy [18]. This is corroborated by the (slight) bronchoconstriction in healthy human volunteers ex posed to silica particles on the nasal mucosa, and the protective effect of atropine in these cases [19]. RALL *et al.* [20] also found bronchoconstriction after irritation of the nasal mucosa in dogs, but this was not elicited *via* the vagus nerve. In cats, NADEL and WIDDICOMBE [21] found that only mechanical irritation of the larynx (not of the nose) led to bronchoconstriction. On the other hand, there is the fact that in ragweed-sensitive asthmatics, despite marked local reactions, no bronchoconstriction could be produced by nasal application of ragweed (*Ambrosia*) [5, 9] and of histamine [5]. In view of this, it is less likely that reflexes should make a clinically relevant contribution, and absorption *via* the mucosa likewise seems to be of no importance.

Because inhalations of *Poa pratensis* gave rise to a late asthmatic reaction, although deposition in the lung was excluded, it has been suggested that allergen intake *via* the gastrointestinal tract might play a role [4].

Anaphylactic reactions to pollens collected by honey bees and used in health goods have been described [22]. Recently an anaphylactic reaction a few minutes after ingestion of sunflower honey, containing a large number of entomophilous pollens has been reported [23]. An argument against an asthmatic reaction induced *via* the gastrointestinal tract is the fact that ingestion of 5,000 pollen grains packed in a capsule elicited no reaction whatever in persons sensitive to the pollen in question [5].

The above data indicate that "pollen asthma" is caused by still unknown mechanisms, or that one of the mechanisms mentioned was mistakenly rejected. This question merits a closer look.

Pollen deposition in lower airways

The first indications that pollen can indeed penetrate into the intrathoracic airways came from studies on guinea-pigs exposed to astronomical ragweed pollen concentrations in air (88,000–375,000 pollen·l^{-1}) [24]. Pollen grains were found in the trachea in five of the six guinea-pigs, in extrapulmonary bronchi in two, and in intrapulmonary bronchi in one of the animals. Since clearance of the airways requires 4–5 min in these animals, much of what was found in the trachea must have originated from peripheral airways. There had been direct deposition, therefore, but this amounted to less than 10^{-6} of the dose to which the animals had been exposed. In human volunteers, WILSON *et al.* [4], who carried out bronchial challenge tests by administering pollen to inhaled air, found no intrathoracic deposition of radioactively labelled *Poa pratensis* pollen (25 μm), but it seems doubtful whether the sensitivity of the collimators was adequate; moreover, there may have been aggregation of pollen during radio-labelling [25]. MICHEL *et al.* [25], on the other hand, leave little doubt that in humans pollen grains do penetrate into lower airways, but they too failed to demonstrate by radiological methods that radio-isotope-labelled pollen grains are in fact deposited in the lung after inhalation. Pollen grains were indeed found in tracheobronchial secretions after inhalation (a few to a few thousand pollen·ml^{-1} secretion). Lung resection specimens from five patients who had not undergone provocation by inhalation contained tracheobronchial secretions with an average of 14 pollen grains·g^{-1} of mucus, while the average amount found in lung tissue was 3 pollen grains·g^{-1}. As the pollen counts in each case were lower in the lung tissues than in the trachea, the suggestion that the pollen grains found came from environmental contaminants could be refuted. Moreover, the pollens were different when the different respiratory tract levels were studied. In the upper respiratory tract not only was a higher count found but also the pollen size was bigger. If the pollen had come from environmental contamination the same results should have been found whichever level was studied [26]. MICHEL and co-workers [25] also looked into the presence of pollen in animal lungs and airways. In animals kept indoors for two months prior to slaughter, pollen grains (up to 70 μm) were always

found, even in the periphery of the lung. The amounts of pollen found in the lungs of wild boars (15 pollen grains·g^{-1} of tissue) markedly exceeded those found in domestic animals kept indoors for two months (0.5 pollen grains·g^{-1} of tissue). The pollen species found corresponded well with the distribution in the atmosphere. These more recent observations indicate that considerable amounts of pollen can indeed penetrate far into the lung.

Deposition of pollen allergen

Fixation on whole pollen has long prevented a search for smaller particles with identical antigenic properties. The first experiments in this direction were performed by BUSSE *et al.* [27] who, using an Andersen sampler, demonstrated that airborne particles of less than 5 μm showed the same antigenic activity as pollen. On technical grounds their findings seemed dubious. However, these doubts were removed by SOLOMON *et al.* [28], who first sucked the air through a filter trapping particles larger than 5 μm, and then collected particles measuring 0.8–5 μm on a second filter. Extracts of this material, in which no pollen or microscopic pollen remnants were encountered, caused positive skin reactions in a patient sensitive to *Ambrosia*. With enzyme-linked immunosorbent assay (ELISA) techniques, moreover, the extracts gave positive reactions to *Ambrosia*. They concluded that in natural conditions the antigen can be leached out from the pollen and then transferred to other airborne particles. HABENICHT *et al.* [29] made similar observations with a filter that trapped particles up to 0.2 μm.

AGARWAL *et al.* [30] found antigenic activity originating from *Ambrosia* in air samples obtained before and after the pollen season. In a study performed in view of this [11], cascade impaction was used to trap particles in five fractions, whereupon an extract was prepared from each fraction. This extract was used to perform a radio-allergosorbent inhibition test (RAST) in relation to components of short ragweed (*Ambrosia eliator*), as well as skin tests in patients sensitive to *Ambrosia*. Significant antigenic activity was found even in extracts originating from particles of 0.3–1 μm. A finding in agreement with this was that in three ragweed-sensitive patients serial dilutions of each of the five fractions led to positive skin reactions. These findings show that there are airborne particles carrying allergens of dimensions much smaller than those of pollen, which can therefore readily penetrate into the lung and intrathoracic airways *via* ventilation. Their origin is still obscure. In nature, the allergen may be leached out from pollen and transferred to other airborne particles. Another possibility is that we are dealing with a different botanical material, which causes cross-reactions with the pollen antigen. A third possibility is that the components which give rise to pollinosis and bronchoconstriction originate not only from pollen but also from other parts of the plant. Allergens and rapidly released allergens are indeed found in all parts of the *Ambrosia* plant, not only during but also before and after the pollen season [11, 31, 32]. AGARWAL *et al.* [11] found large amounts of allergen before as well as during the flowering season, especially in the flowers of *Ambrosia eliator* but also, albeit to a lesser degree, in the other parts of the plant. Allergen from oak pollen is encountered in the atmosphere months after the flowering period [33].

Synthesis

The originally raised question - whether pollen can penetrate into the airways - can be answered in the affirmative. An estimated 1–2% of airborne particles with a diameter exceeding 10 μm reach the lung [25]. In oral breathing this percentage may increase. The poor correlation between exposure and asthmatic reaction may be based on the small numbers of particles entering the lung, and on differences between individuals in the filter function of the airways and in bronchial responsiveness. A factor contributing to the poor correlation between symptoms and pollen counts is that airborne particles can have a respirable form both within and outside the pollen season, as was demonstrated for *Ambrosia*. The amount of allergen contained in minute particles (especially if resulting from aqueous elution of pollen in nature) is several orders of magnitude smaller than the amount present in pollen. This is why PLATTS-MILLS and co-workers [34, 35] assumed that pollen-allergic reactions in the lung result from pulmonary pollen deposition. Their assumption is that a few grains daily are deposited on widely different sites in the airway. Each separate grain is insufficient to cause a generalized bronchial obstruction, but the pollen inhaled day after day gives rise to a protracted inflammatory response at an increasing number of sites in the airway. A corroborating fact is that bronchial hyperreactivity diminishes when allergens are avoided [36–38], and that this hyperreactivity parallels the pollen season in pollen allergy [39–46], although this has not been confirmed by all investigators [47–49]. As a result of the gradually cumulative effect of exposure, the correlation between clinical symptoms of bronchial obstruction and daily pollen counts will be vague in most cases.

Most of the information about pollen deposition derives from animal studies. Hence, further evidence that the findings are relevant for human pathophysiology of asthma is called for. As the radio-isotopic method was until now not sufficiently sensitive to detect pollen particles in the respiratory tract [4, 25], we suggest that such experiments should be repeated with special caution for particle aggregation during radio-labelling of the pollen grains. Further innovative application of filters and high speed impingers, as well as of immunoassays, can increasingly clarify the clinical role of airborne units below 5 μm in size. Furthermore, studies which employ personal samplers, and which take into account cumulative *versus* daily exposure to a number of allergen particles and total amount of allergen could throw further light on the idea proposed by PLATTS-MILLS and

co-workers [34, 35], that it is the number of particles deposited cumulatively on airways rather than daily exposure which elicits pollen asthma.

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Dépôt de pollen dans les voies aériennes intrathoraciques. M.N.B.M. Driessen, Ph.H. Quanjer.

RÉSUMÉ: Jusqu'à ce jour, les mécanismes physiopathologiques de l'asthma induit par les pollens sont restés peu clairs car les particules de pollen ont été considérées comme trop grandes pour pénétere dans le tractus respiratoire inférieur. On pense dès lors que ces grains sont incapables d'induire une réponse immunologique dans l'arbre respiratoire inférieur. Toutfois, il est démontré qu'un petit pourcentage de particules de grandes tailles (20 à 30 μm) peut pénéter dans les zones périphériques du poumon. Il apparaît également que de petites unités en suspension dans l'air, d'un calibre inférieur à 5 μm et porteuses d'une activité antigénique semblable à celle du pollen, pourraient contribuer à la charge allergénique inhalée.
L'asthme au pollen résulte probablement d'une effet cumulatif graduel du dépôt de petites quantités d'allergènes dans les voies aériennes inférieures et dès lors, en faible corrélation avec les décomptes quotidiens de pollens.
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