1 Pollen Grains Contain and Release Not Only Allergens, but Also Eicosanoid-Like Substances with Neutrophil Chemotactic Activity: A New Step in the Initiation of Allergic Sensitization?


Summary

Pollen grains as allergen carriers are the elicitors of the most common allergic diseases, namely seasonal allergic rhinoconjunctivitis („hay fever“), extrinsic bronchial asthma and other immediate-type allergic diseases.

All of them have increased in prevalence dramatically during the last decades. It is common belief that pollen themselves are inert and act by release of protein allergens in humid conditions on the mucosal surface, where the process of sensitization starts through the recognition of the allergen by an antigen-presenting cell. All studies dealing with the mechanism of this early phase of sensitization have used isolated allergens (from extracts or recombinant technology) as stimulus. However, under natural exposure conditions, the bioavailability of allergen depends upon allergen liberation from internal binding sites within the allergen carrier, here the pollen grain. It is not the soluble allergen, but rather the pollen grain as a particle which comes into contact with the body’s surface on the skin or the mucosa. We have shown earlier that the release of allergen from pollen grains can be modulated by external factors such as gaseous or particulate air pollutants.

We now have found that pollen grains from different plants (Timothy grass = Phleum pratense, birch = Betula alba) secrete significant amounts of eicosanoid-like substances in protein-free buffer solution in a pH, time and temperature-dependent fashion. When pure pollen grains were incubated together with suspensions of human polymorphonuclear leucocytes (PMN), these cells assembled around the pollen grains and showed signs of activation and mediator release leading to destruction of the pollen grain. Leukotriene B4-like activity secreted differed between pollen species with highest values for grass and birch pollen and significantly lower values for pine pollen (Pinus silvestris). Furthermore there was a significant modulatory effect from traffic-related pollutants, e.g. volatile organic compounds (VOCs) leading to a significant increase in secretion of LTB4-like activity from pollen grains.

This finding opens a new dimension in understanding the early events in allergic sensitization indicating that pro-inflammatory effects of the allergen carrier itself (the pollen grain) induce activation of cellular constituents of the host. We propose to call this phase the „initiation“ of allergic sensitization. The differences in allergenic „potency“ of various allergens may be explained by these new findings independent of allergen release from pollen. It also may be helpful in understanding so far unexplained differences in allergy prevalence associated with automobile exhaust exposure.

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J. Ring, H. Behrendt (Eds)
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Introduction

Allergic diseases (namely allergic rhinoconjunctivitis, bronchial asthma, atopic eczema) have increased in prevalence during the last decades world-wide; the causes for this increase are not known. Among hypothetical concepts under discussion, lack of adequate stimulation of the immune system, improved hygiene, socio-economic factors (“life style“) as well as influence of environmental pollutants have gained substantial public and scientific attention [1, 3, 6, 10, 11, 14, 16, 18, 22–25].

Allergies are among those few diseases in which environmental factors of both natural and anthropogenic origin have been identified as causal in the disease both in the development of sensitization as well as in the elicitation and aggravation of disease symptoms. We have shown earlier that outdoor air pollution differs strikingly in quality – not only in quantity – between former Eastern and Western European countries, whereby the modern type air pollution – characterized by organic compounds, nitrogen oxides, fine particles and ozone – was associated in multivariate regression analyses with increased prevalence of IgE-mediated sensitization and atopic disease [3, 10, 18]. Pollen grains collected from industrial regions with high polycyclic aromatic hydrocarbon load in West Germany, not in East Germany were shown to be agglomerated with fine airborne particles leading to morphological changes of the pollen surface and altered allergen release [1, 2]. Thus the bioavailability of pollen allergens may be influenced by interaction between pollen and air pollutants in the atmosphere [4]. It is common belief that allergic sensitization starts with the contact between the allergen and the surface of the antigen-presenting cell at the level of the mucosa or the skin. Most studies dealing with this aspect have used allergen extracts as stimulus. Under natural exposure conditions, however, the bioavailability of allergens depends on allergen liberation from internal binding sites within the allergen carrier (e.g. pollen grains) [4, 7, 13, 21]. Here we report the surprising finding that pollen grains themselves liberate substances with pro-inflammatory activity under humid conditions.

Allergen Liberation from Pollen Grains

Pollen grains are multicellular male gametothytes of both angiosperms and gymnosperms. Anemophilous pollen from gymnosperms (wind-transported) like grass pollen are sealed in a double-layered wall with an outer layer (exine) (containing the lipophilic sporopollenin) and a softer inner layer (intine) enclosing the cytoplasm with subcellular organelles, starch granules and polysaccharide particles [21]. Pollen allergens are bound within the pollen grain around organelles, P-particles and starch granules, some in the cytoplasmic matrix. The liberation of allergens from intracellular binding sites is the prerequisite for allergic sensitization in a pre-disposed individual. The most important stimulus for allergen liberation is humidity [4, 7, 8, 20, 21]; under humid conditions, allergens are rapidly released from pollen grains in pH and temperature-dependent fashion through small caniculi connecting the inner surface of the intine with the outer pollen surface.