Secretion of Proinflammatory Eicosanoid-Like Substances Precedes Allergen Release from Pollen Grains in the Initiation of Allergic Sensitization

Abstract:
It is commonly believed that allergic sensitization starts when an allergen contacts the surface of an antigen-presenting cell in mucosal or skin epithelia. Most studies dealing with this aspect use allergen extracts as stimulus. Under natural exposure conditions, however, the bioavailability of allergen depends on allergen liberation from internal binding sites within the allergen carrier, e.g. pollen grains. In comparing total protein and major allergen release from timothy grass (Phleum pratense L.) pollen freshly collected on rural meadows or near high-traffic roads, there was a striking difference between the pollen, with higher allergen release rates from rural meadow pollen grains. Thus, allergen release does not explain the higher prevalence rates of atopic sensitization and disease observed in many epidemiological studies in children exposed to automobile exhaust. Therefore, other possible effectors from pollen grains were investigated. Pollen grains incubated in protein-free buffer were found to secrete significant amounts of eicosanoid-like substances, namely leukotriene (LT) B4-like and prostaglandin E2-like substances, in a pH-, time- and temperature-dependent fashion. The highest values of eicosanoid secretion were found in birch, grass and mugwort pollen, while pine (Pinus sylvestris L.) pollen showed only marginal eicosanoid-like secretion. Additionally, the release of
these substances was significantly higher from pollen which had been collected near roads with heavy traffic, indicating a stronger proinflammatory activity of these pollen grains. In order to investigate the effects of air pollutants, native pollen grains were exposed in a dose- and time-dependent fashion in a fluidized bed reactor to traffic-related pollutants, e.g. volatile organic compounds (toluene, m-xylene), leading again to a significant increase in the secretion of LTB4-like immunoreactivity, in contrast to exposure with sulfur dioxide. This finding opens a new dimension of understanding of the early events in allergic sensitization, indicating that proinflammatory effects of the allergen carrier, e.g. the pollen grain itself, can lead to activation of the mucosal membrane. These findings might help to also explain the higher prevalence rates of pollen allergy in areas with high automobile exhaust emissions. Furthermore, the allergenic ‘potency’ of various allergens has to be redefined at the allergen carrier level with regard to different stages of allergen and mediator release prior to the contact with the host’s immune system.

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