Altered Skin Prick Test Reactivity and Histamine Release with Extracts from Pollen Exposed to Pollutants

P.		Thomas ^a
D.		Strube ^b
B.		Przybilla ^a

^aDermatologische Klinik und Poliklinik der Ludwig-Maximilians-Universität München und

^bGesellschaft für Strahlenschutz und Umweltforschung, Neuherberg, Deutschland

Key Words

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Histamine
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Correspondence to: Dr. Peter Thomas, Dermatologische Klinik und Poliklinik, Ludwig-Maximilians-Universität München, Frauenlobstrasse 9–11, D–80377 München (Germany)

Introduction

Both epidemiological studies and experimental data point to the role of environmental pollutants as one of the factors responsible for the increasing prevalence of allergic diseases [1, 2].

Exposure to air pollutants can lead to structural and functional alterations of the mucosa and modulation of the immune response. In addition, air pollutants may directly act upon pollen or airborne allergens and modulate their allergenic potency [3]. To further address this question, pollen of birch (B), rye (R) and ash tree (A) was exposed to NO₂, SO₂ or O₃. Subsequently, extracts were prepared and used at equal protein contents for skin prick tests and in vitro determination of histamine-releasing capacity.

Materials and Methods

Purified B, R or A pollen was commercially obtained. For pollutant exposure, pollen aliquots were kept in glass chambers (approximately 1 liter volume) for 4 h under continuous flow (2.5 l/min) of ambient air or ambient air containing 50,100,200 or 400 ppb NO₂, 900 ppb SO₂, or 300 ppb O₃. Subsequently, 105 pollen grains/ml were incubated in Tris-containing PBS (TCM buffer; pH 7.2) and extracted by gentle stirring for 1 h. After removal of the pollen, supernatants were tested for protein concentration by the Coomassie protein assay and adjusted to equal protein content for the respective pollen species after dilution. Cell suspensions containing basophils were obtained by dextran sedimentation outside the pollen season from peripheral blood of 8 individuals with seasonal rhinoconjunctivitis due to B (6/8) or R pollen (4/8), but not A pollen. Cells (2×10⁶/ml) were incubated in duplicate with the extracts of native/pollutant-exposed B, R or irrelevant A pollen for 30 min at 37°C. Histamine in the supernatants was measured by ELISA and expressed as the percentage of total histamine content after correction for spontaneous histamine release. Prick testing with the respective extracts (examiner blinded to the specification) was performed on the forearm of the 8 individuals. After 20 min, areas of wheal and flare response were documented on transparent tapes and

subsequently quantified by the use of Summa Graphics Bit-Pad Two. The Mann-Whitney U test was used for statistical evaluation.

Results and Discussion

Results obtained with extracts of B and R pollen are shown in figures 1 and 2. Extracts of A pollen, i.e. control tests, did not induce any relevant reactions in vivo or in vitro (data not shown). Preexposure of extracts of B and R pollen to air pollutants enhanced histamine release and skin prick test reactivity to a varying degree in the presence of IgE-type sensitization. Thus, naturally occurring pollutant exposure may enhance the effects of pollen allergens on clinical reactivity. As suggested in previous studies [4-6], an alteration of aeroallergen release and/or composition may account for this. The potential linkage between solubility and allergenicity of pollen proteins may act as an additional factor [7]. It still remains to be elucidated to what extent pollutant exposure also alters the release of proteins from pollen upon contact with mucosal surfaces.

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Fig.1. Flare (a) and wheal (b) response (mm²) upon skin prick testing with extracts from native/pollutant-exposed pollen (10 test series). – = median. Symbols denote individual test series.

Fig. 2. Histamine release by extracts from native/pollutant-exposed birch/rye pollen (8 test series). – = median. Symbols denote individual test series.

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 Exposure (ppb)
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