

Pollen sensitization and allergy in children depend on the pollen load

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We investigated the influence of grass and birch pollen load on specific IgE response, skin prick test reactivity, and manifest allergic disorders of the eyes and respiratory tract. The study population was two groups of 200 children each, matched in sex and age, who were domiciled in areas with high and low pollen load, respectively. In the children from the area with high pollen load, the grass pollen load was 1.7 times higher than in the other and only specific IgE to grass pollen was found to be present more frequently (23.5% as against 12.5%; $P < 0.01$). In the same area, the birch pollen load was 2.8 times higher, which caused specific IgE in 10.5% as against 3.5% of the other group ($P < 0.01$) as well as positive skin prick test in 9.0% as against 3.5% ($P < 0.05$). Reports of subjective complaints were rare among the group with high pollen load but the total number did not amount to a statistically significant increase. There was no difference between the two groups concerning sensitization and allergy to house dust mite. This suggests that the influence of the pollen load is strongest on the development of specific IgE, and less on skin test reactivity and manifest allergies.

Key words: allergy; birch pollen; grass pollen; subclinical sensitization.

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In recent years, the risk factors that may have an impact on sensitization to aeroallergens and the incidence of allergic diseases have been main subjects of epidemiological investigations (7, 11, 17-20). Many questions arose from uncertain increases in the incidence of allergic diseases (8, 14). It has never been clearly understood why allergen-specific IgE to aeroallergens, which are the causes of allergic disorders, were found only in a few persons exposed to them. Allergic diseases were related to different causes, thus giving rise to a number of theories on their development (14). The causal relation with allergen load, the development of subclinical sensitization, and the onset of allergic disorders of the eyes and respiratory tract, however, remained unexplored.

The aim of the present study has been to investigate the incidence of subclinical sensi-

zation to aeroallergens, and manifest allergies in children from two areas with high quality air but different pollen load.

MATERIAL AND METHODS

The study was planned to investigate two groups of 200 children each, recruited from areas where birch and grass pollen load was high and low, respectively. From a target group of 759 children we obtained written informed consent from 75.1% parents and from these volunteers, 400 subjects were matched as to sex and age. The pollen load was measured in both areas by means of a Burkard pollen trap through continuous measurement and recording of 2-h mean values of pollen/m³ during a period of 3 years. Both areas had equal meteorological conditions and

Table 1.

Study subjects and pollen load		
	High pollen load	Low pollen load
Age (years)	11.5±0.5	11.5±0.5 NS
Active smokers	11 (5.5%)	9 (4.5%) NS
Days/year with daily mean > 50 grass pollen/m ³	14	10
Days/year with daily mean > 100 birch pollen/m ³	5	1

high quality air with regard to the air pollutants SO₂ (<0.22 mg/m³ in the last year) and NO₂ (<0.23 mg/m³ in the last year). Both areas are free of industries or other major sources of air pollution. The investigation was performed by the same team of investigators in the fall (after the pollen season) and under identical meteorological conditions.

A standardized pediatric questionnaire on personal and environmental conditions simplified for local requirements was submitted to the children's parents for joint evaluation (9). The complaints enquired about in the questionnaire were: usually cough with colds or otherwise (ATS-DLD-78-C, questions 14A or 14B), phlegm production with or without colds (questions 15A or 15B), and breathlessness (question 18A). Questions about conjunctivitis, rhinitis, cough, or breathlessness in spring, summer, or during the whole year were discussed with all test subjects before investigation.

Spirometry was performed with a pneumotachograph (Spiriflow, Jaeger/Würzburg), the patient in the sitting position with noseclip

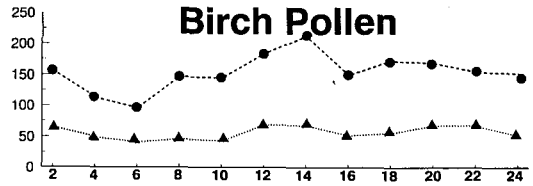


Fig. 2. Birch pollen counts given as sums of the 2-h mean values at the same time of day are represented by circles (area with high pollen load) and triangles (area with low pollen load).

applied. Forced vital capacity (FVC), forced expiratory volume in 1 s (FEV₁), and maximal expiratory flow at 50% expiration (MEF₅₀) were measured by means of standardized methods (1). From these results the percentages of FEV₁/FVC and MEF₅₀/FVC were calculated.

Skin prick tests were performed by one experienced investigator by means of standardized skin prick test methods, using a wide range of solutions of common aeroallergens of which the following were further evaluated: *Dermatophagoides pteronyssinus*, *Dermatophagoides farinae*, grass pollen, birch pollen, negative control, and 1% histamine control (Pangram®/Abello) (6). The skin prick test was considered positive when the wheal was 2mm larger in diameter than that of the negative control 15 min after application.

Total IgE was determined by the Phadezym PRIST method (22). Allergen-specific IgE was determined for *Dermatophagoides pteronyssinus* (d1), grass pollen (g3), and birch pollen (t3) and considered positive when the value thus obtained was higher than 0.34 PRU (13).

The results with normal distribution are given as mean values ± SD and in the absence of normal distribution as median ± SEM (age, total IgE). For the statistical qualitative analysis of the results we used the chi-square test and for quantitative group comparisons the Kolmogoroff-Smirnoff test, respectively (21). Odds ratios (OR) for analyses of influence of smoking habits and gas stoves were given with 95% confidence intervals (CI) (15,16). A *P* value <0.05 was considered statistically significant.

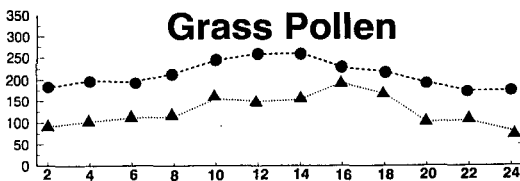


Fig. 1. Grass pollen counts given as sums of the 2 h mean values at the same time of day are represented by circles (area with high pollen load) and triangles (area with low pollen load).

RESULTS

The epidemiological data were identical in the two groups of sex- and age-matched study subjects (Table 1). The pollen load proved to be different in the two areas from which the study population was recruited. The sums of the annual 2-h mean values at the same time of day were 212 ± 31 and 127 ± 35 ($P < 0.001$) for grass pollen, and 154 ± 30 and 56 ± 10 ($P < 0.0001$) for birch pollen (Figs. 1 and 2). In addition to the grass pollen count, which was 1.7 times higher, the frequency of days with increased pollen load (daily mean value > 50 grass pollen/ m^3) recorded was significantly higher (Table 1). The same was true for birch pollen: in addition to the pollen load, which was 2.8 times higher, the frequency of days with increased pollen load (daily mean value > 100 birch pollens/ m^3) recorded was highly significantly higher (Table 1).

The two groups recruited from areas with different pollen load reported similar symptoms of conjunctivitis, rhinitis, cough with or without phlegm production, or breathlessness during the pollen season or the whole year (Table 2). Manifest allergies to grass pollen were confirmed in 16 cases (8%) from the area with high pollen load and in 13 cases (6.5%) with low pollen load, manifest allergies to birch pollen in seven cases (3.5%) and three cases (1.5%), and manifest allergies to house dust mite in 13 cases (6.5%) and 11 cases (5.5%), respectively (all NS). Both groups furnished identical lung function data and, further, showed no difference in the number of pathological spirometry findings (Table 3).

The results of the skin prick test showed positivity to birch pollen in 18 subjects from the area with high pollen load as against seven from that with low pollen load ($P < 0.05$) (Table 4). There was no significant difference between the two groups as to number of test subjects with positive skin prick test to grass pollen and house dust mite.

The total IgE levels did not differ in the two groups, nor did the number of subjects with increased IgE (> 100 kU/L). Specific IgE to grass pollen was identified in 47 subjects from the area with high pollen load as against 25 subjects from the area with low pollen load ($P < 0.01$); specific IgE to birch pollen was found in 21 as against seven subjects ($P < 0.01$). Statistically, there was no significant difference in specific IgE to *Dermatophagoides pteronyssinus*.

Active smoking did not influence sensitization to grass pollen (OR = 0.80; CI = 0.23–2.78) and to birch pollen (OR = 1.45; CI: 0.32–6.54). Other environmental factors were also similar in both areas and had no further impact on sensitization: passive smoking had no influence on sensitization to grass pollen (OR = 1.21; CI: 0.72–2.05) and birch pollen (OR = 1.24; CI: 0.57–2.70) and neither did gas stoves (OR = 1.46; CI: 0.84–2.53) and (OR = 1.11; CI: 0.47–2.58), respectively.

DISCUSSION

The object of the present study was to investigate the influence of pollen load on subclinical sensitization and manifest allergies. Specific IgE reactivity, skin prick test reactivity, and respiratory health may be influenced by many

Table 2.

Subjective complaints of study subjects

	High pollen load <i>n</i> (%)	Low pollen load <i>n</i> (%)	
Conjunctivitis	10 (5.0)	7 (3.5)	NS
Rhinitis	11 (5.5)	8 (4.0)	NS
Cough	25 (12.5)	21 (10.5)	NS
Cough with phlegm	3 (1.5)	3 (1.5)	NS
Breathlessness	15 (7.5)	14 (7.0)	NS

Table 3.

Lung function data

	High pollen load	Low pollen load	
FVC (% pred.)	98.9 $\pm$ 11.7	99.0 $\pm$ 10.4	NS
FEV ₁ (% pred.)	98.2 $\pm$ 11.4	98.2 $\pm$ 10.7	NS
FEV ₁ /FVC (%)	85.0 $\pm$ 5.5	84.8 $\pm$ 5.1	NS
MEF ₅₀ /FVC (%)	105.2 $\pm$ 20.7	105.7 $\pm$ 20.1	NS
FEV ₁ < 80% pred.	6 (3.0%)	4 (2.0%)	NS
MEF ₅₀ /FVC < 75%	11 (5.5%)	13 (6.5%)	NS

Table 4.
Allergological results

	High pollen load <i>n</i> (%)	Low pollen load <i>n</i> (%)	
Positive skin prick test:			
grass pollen	35 (17.5)	28 (14.0)	NS
birch pollen	18 (9.0)	7 (3.5)	<i>P</i> < 0.05
<i>D. pt.</i>	14 (7.0)	19 (9.5)	NS
Total IgE (kU/l)	59±7	56±8	NS
Total IgE (> 100 kU/l)	72 (36.0)	73 (36.5)	NS
Specific IgE (> 0.34 PRU):			
grass pollen (g3)	47 (23.5)	25 (12.5)	<i>P</i> < 0.01
	<9/18/11/9>	<8/7/5/5>	
birch pollen (t3)	21 (10.5)	7 (3.5)	<i>P</i> > 0.01
	<2/11/6/2>	<3/3/1/0>	
<i>D. pt.</i> (d1)	24 (12)	19 (9.5)	NS
	<6/6/7/5>	<7/7/4/1>	

Numbers of positive Phadezym RAST classes are given in parentheses
<class 1/class 2/class 3/class 4>

factors (3, 4). We precluded air pollution as a major influence by demonstrating that both areas had good air quality.

The grass pollen load measured in both areas was enough to provoke allergic symptoms, but already a 1.7 increase resulted in a significantly higher frequency of increased specific IgE development. The skin prick test or manifest allergies, however, remained largely uninfluenced.

The birch pollen load was relatively low in the one area and 2.8 times higher in the other. This difference in pollen load caused not only a significantly higher frequency of increases in specific IgE but also a statistically stronger significant frequency of skin prick test sensitization. The frequency of manifest allergies recorded was only slightly, but not significantly, higher depending on the high pollen load.

Sensitization to house dust mite was only explored to preclude highly atopic conditions in the two areas. Therefore the load of house dust mite allergens was not measured, but was suspected to be similar because of similar indoor conditions. Thus, it was not surprising that the children of both areas showed no difference in sensitization to house dust mite or in any lung function data.

Even though specific IgE was found to be the most sensitive parameter for monitoring the aeroallergen load, total IgE, which may be influenced by many factors, had no significance for our study (2, 3; 5, 19). Recent studies showed that air pollution might be an important but not the only factor for sensitization to aeroallergens (12, 19). In epidemiological studies, therefore, specific IgE should be determined, especially when slight influences are suspected. Since skin test sensitization is known to be a risk factor for the development of bronchial asthma and allergic rhinitis (10), this may also hold true for specific IgE sensitization to aeroallergens.

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REFERENCES

1. American Thoracic Society. Standardization of spirometry - 1987 update. *Am Rev Respir Dis* 136, 1285-1298, 1987.
2. Barbee RA, Brown WG, Kaltenborn W, Halonen M. Allergen skin-test reactivity in a community population sample: correlation with age, histamine skin reactions,

- and total serum immunoglobulin E. *J Allergy Clin Immunol* 68, 15-19, 1981.
3. Barbee RA, Halonen M, Lebowitz M, Burrows B. Distribution of IgE in a community population sample: correlations with age, sex, and allergen skin test reactivity. *J Allergy Clin Immunol* 68, 106-111, 1981.
 4. Bericano FA, Crespo M, Bao CG, Alvarez FV. Serum levels of total IgE in non-allergic children. Influence of genetic and environmental factors. *Allergy* 42, 267-283, 1987.
 5. Burrows B, Halonen M, Lebowitz MD, Knudson RJ, Barbee RA. The relationship of serum immunoglobulin E, allergy skin tests, and smoking to respiratory disorders. *J Allergy Clin Immunol* 70, 199-204, 1982.
 6. Dreborg S. Skin tests used in type I allergy testing. *Allergy* 44, Suppl. 4, 1989.
 7. Dupal HD, Horak F, Türk R. Häufigkeit der Inhalationsallergie bei Schulkinder im ländlichen Raum im Vergleich zum Großstadtgebiet. *Laryngol Rhinol Otol (Stuttg)* 61, 648-650, 1982.
 8. Eaton KK. The incidence of allergy - has it changed? *Clin Allergy* 12, 107-110, 1982.
 9. Ferris Jr BG. Epidemiology standardization project. *Am Rev Respir Dis* 118, 1-120, 1978.
 10. Hagy GW, Settupane GA. Risk factors for developing asthma and allergic rhinitis. A 7-year follow-up study of college students. *J Allergy Clin Immunol* 58, 330-336, 1976.
 11. Horak F. Manifestation of allergic rhinitis in latent-sensitized patients. A prospective study. *Arch Otorhinolaryngol* 242, 242-249, 1985.
 12. Ishizaka T, Koizumi K, Ikemori R, Ishiyama Y, Kushibiki E. Studies of prevalence of Japanese cedar pollinosis among the residents in a densely cultivated area. *Ann Allergy* 58, 265-270, 1987.
 13. Johansson SGO, Bennich H, Berg T. In vitro diagnosis of atopic allergy. III. Quantitative estimation of circulating IgE antibodies by the radioallergosorbent test. *Int Arch Allergy Appl Immunol* 41, 443-451, 1971.
 14. Leskowitz S, Salvaggio JE, Schwartz HJ. An hypothesis for the development of atopic allergy in man. *Clin Allergy* 2, 237-246, 1972.
 15. Mantel N. Chi-square tests with one degree of freedom; extensions of the Mantel-Haenszel procedure. *J Am Stat Assoc* 58, 690-700, 1963.
 16. Mantel N, Haenszel W. Statistical aspects of the analysis of data from retrospective studies of diseases. *J Natl Cancer Inst* 22, 719-748, 1959.
 17. Massicot JG, Cohen SG. Epidemiologic and socioeconomic aspects of allergic diseases. *J Allergy Clin Immunol* 78, 954-958, 1986.
 18. Peat JK, Britton WJ, Salome CM, Woolcock AJ. Bronchial hyperresponsiveness in two populations of Australian schoolchildren. III. Effect of exposure to environmental allergens. *Clin Allergy* 17, 291-300, 1987.
 19. Popp W, Zwick H, Steyrer K, Rauscher H, Wanke T. Sensitization to aeroallergens depends on environmental factors. *Allergy* 44, 572-575, 1989.
 20. Ruffin J, Liu MYG, Sessoms R, Banerjee S, Banerjee UC. Effects of certain atmospheric pollutants (SO₂, NO₂ and CO) on the soluble amino acids, molecular weight and antigenicity of some airborne pollen grains. *Cytobios* 46, 119-129, 1986.
 21. Sachs L. *Angewandte Statistik*. 6th ed., Springer, Berlin 1984.
 22. Zetterstrom O, Johansson SGO. IgE concentration measured by PRIST in serum of healthy adults and in patients with respiratory allergy: A diagnostic approach. *Allergy* 36, 537-547, 1981.

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