

IgE-mediated allergy to wood dusts probably does not explain the high prevalence of respiratory symptoms among Swedish woodwork teachers

Åhman M, van Hage-Hamsten M, Johansson SGO. IgE-mediated allergy to wood dusts probably does not explain the high prevalence of respiratory symptoms among Swedish woodwork teachers.
Allergy 1995; 50: 559–562. © Munksgaard 1995.

A previous study revealed an increased occurrence of work-related respiratory complaints among Swedish woodwork teachers. For determination of whether an IgE-mediated mechanism was the cause of the symptoms, 127 woodwork teachers and 111 reference subjects (other school personnel) in Stockholm gave serum for analysis of total IgE, Phadiatop, and RAST to extract of sawdust from five commonly used Scandinavian woods (pine, birch, juniper, alder, and linden). The total serum-IgE level was similar in the woodwork teachers (geometric mean 35, range <2–1700 kU/l) and the reference group (34, <2–860 kU/l), and the frequency of positive Phadiatop was equal (29.9% and 31.5%, respectively). RAST was positive only in three cases, two reference subjects (birch and pine) and one woodwork teacher (alder). Hence, no correlation between exposure and sensitization to wood dust was found, and the results do not support the assumption that the complaints related to exposure to dusts from the examined Scandinavian woods are IgE mediated.

M. Åhman¹, M. van Hage-Hamsten²,
S. G. O. Johansson²

¹ Division of Occupational and Environmental Medicine/Department of Medicine at Karolinska Institute and Department of Occupational and Environmental Medicine, Huddinge Hospital, S-141 86 Huddinge; ² Department of Laboratory Medicine, Division of Clinical Immunology, Karolinska Hospital, S-171 76 Stockholm, Sweden

Key words: allergy; IgE; occupational exposure; RAST; wood dust.

Mats Åhman, MD
Department of Occupational and Environmental Medicine
Huddinge University Hospital
S-141 86 Huddinge
Sweden

Accepted for publication 10 March 1995

Diseases and complaints involving the skin and airways are common among wood-dust-exposed groups and have been reviewed by several authors (8, 10, 12, 20, 25). A previous study of 130 Swedish woodwork teachers found a high frequency of symptoms in the skin, eyes, nose, throat, and lower airways (27). The symptoms usually increased during working days and decreased during days off. Several of the symptoms were related to type of ventilation and dust-spreading factors. But individual factors, including smoking habits and a history of hay fever or asthma during adolescence, were associated with increased occurrence of symptoms. Measurements performed later in 40 workshops revealed moderate dust levels (total dust 0.23–1.18 mg/m³) and low terpene levels (0–6 mg/m³).

There may be various causes of the symptoms in

wood-dust-related complaints. This study examined whether IgE-mediated sensitization to wood dust could cause the symptoms. It has been postulated that allergic mechanisms are common in patients exposed to many different types of wood (15). But specific IgE antibodies to wood dust have been confirmed in only a limited number of woods and most often in tropical woods (12).

Among the woodwork teachers studied, Scandinavian woods were most commonly used, especially pine and, to some extent, alder, birch, juniper, and linden. Exotic woods such as teak, mahogany, and jelutong were used less. However, the respiratory complaints were not clearly related to the use of any specific wood. This study focused on the most common Scandinavian woods to detect whether an IgE-mediated mechanism caused the symptoms.

Material and methods

Subjects

All 139 woodwork teachers employed in the Stockholm public school system in 1991 were included in the study. Eleven declined to participate and one was excluded because he was a substitute teacher. Thus, 127 woodwork teachers (117 men and 10 women) participated, all of whom were also included in an earlier questionnaire study (27). The same reference group of 112 other school personnel participated. They were mainly other teachers, 103 men and eight women (one declined). They did not differ significantly from the woodwork teachers in school, age, sex, smoking habits, number of classes per week, or self-reported symptoms of atopic dermatitis, hay fever, and asthma during adolescence.

Blood samples

Venous blood samples were drawn from all subjects. The sera were kept frozen (-20°) until analyzed.

Total serum IgE determination and Phadiatop

Serum total IgE levels (Pharmacia CAP System IgE FEIA, Uppsala, Sweden) and Phadiatop (Pharmacia CAP System Phadiatop FEIA) were determined according to the manufacturer's instructions. The results were given in kU/l for the former test and expressed as positive or negative in the latter.

Determination of IgE antibodies

Extracts from five common Scandinavian woods were prepared from stored timber by sawing each of the following with a circular saw and collecting the sawdust with a vacuum cleaner:

1. pine (*Pinus sylvestris*)
2. birch (*Betula verrucosa*)
3. juniper (*Juniperus communis*)
4. alder (*Alnus glutinosa incana*)
5. linden (*Tilia cordata*).

The wood dust was frozen (-20°) until preparation. A 1:25 w/v extract was prepared in 0.15 M NaCl with 0.02% NaN_3 at room temperature for 16–18 h. The extracts were dialyzed three times overnight in a dialysis tube which retained molecules larger than 6–8 kDa, centrifuged for 10 min (6600 g), and filtered through an 0.8- μm Millex-HA filter (Millipore, Bedford, MA, USA). A total of 50 μl undiluted extract was coupled per cyanogen bromide-activated paper disk (6). The RAST analyses were then performed as usual with 50 μl undiluted serum added to each disk for incubation during 3 h at room temperature. The disks were then washed three

times, incubated overnight with ^{125}I -labeled rabbit antihuman IgE (Phadebas RAST anti-IgE tracer, Pharmacia Diagnostics AB). The binding was determined in a multigamma counter (LKB, Sweden). The disks gave no nonspecific uptake of labeled anti-IgE when tested against nonatopic serum samples up to 1000 kU/l. A positive result was defined as a value ≥ 0.35 kU/l. As a positive control, we used a serum known to have IgE antibodies to a common Swedish wood, spruce. This serum was positive in a varying degree (0.6–6.6 kU/l) to all five wood dusts.

Statistics

The *t*-test and Fisher's exact test were used for comparison of differences between the groups in frequency of background data. Logistic regression was used when evaluating the importance of smoking habits, sex, atopy, and exposure to wood dust for the complaints.

Results

The total serum-IgE level was similar between the woodwork teachers (geometric mean 35, range <2 –1700 kU/l) and the reference group (geometric mean 34, range <2 –860 kU/l). A rather high figure of positive Phadiatop tests was obtained both among the woodwork teachers (29.9%) and among the referents (31.5%). The difference between the groups was not significant.

Three subjects (one woodwork teacher and two referents) had positive RAST to different Scandinavian woods. The woodwork teacher, employed as such for 21 years, had a positive RAST to alder (0.4 kU/l). One reference subject had positive RAST to birch (1.8 kU/l) and the other to birch (2.0 kU/l) and pine (0.4 kU/l). All three subjects were also positive to Phadiatop. Their total serum-IgE levels varied between 57 and 690 kU/l. Neither reference subject had previous occupational exposure to wood dust, but one was exposed for 1 h weekly during leisure time.

The occurrence of symptoms from skin, eyes, nose, throat, and lower airways remained elevated among the woodwork teachers also after adjustment for occupation, smoking habits, sex, and Phadiatop result (Table 1). Atopic subjects (defined as having a positive Phadiatop) had a raised rate of occurrence of itching and tears in the eyes. But atopy was not otherwise significantly associated with the complaints.

Discussion

Respiratory complaints among groups exposed to wood dust have previously been reported; for ex-

Table 1. Occurrence of some symptoms in eyes, upper and lower airways, and skin. Odds ratios (OR) and, in parentheses, 95% confidence interval were calculated by multiple logistic regression with adjustment for being woodwork teacher, current smoker, female, and atopic subject. ORs for smoking and female sex are not presented here; see previous questionnaire report (28)

Symptoms (from)	Woodwork teacher	Atopy*
Eye itching	4.3 (1.8–10.4)	2.5 (1.1–5.3)
Tears in eyes	2.6 (1.1–5.9)	2.3 (1.1–5.0)
Nasal obstruction	12.2 (5.8–25.7)	0.8 (0.4–1.5)
Runny nose	4.5 (2.0–9.9)	1.8 (0.9–3.7)
Sneezing	5.8 (2.9–11.7)	1.9 (0.99–3.7)
Bronchial irritation	10.0 (3.8–26.7)	0.9 (0.4–1.9)
Wheezing	4.0 (1.2–12.8)	1.1 (0.4–3.2)
Chronic bronchitis	12.1 (2.8–53.2)	0.8 (0.3–2.2)

* Defined as positive Phadiatop.

ample, among joiners, cabinet-makers, and other industrial woodworkers. In these groups, an elevated rate of occurrence of rhinitis (1, 13, 14, 19, 23, 26) and chronic bronchitis (2, 16) has been observed. In a previous report, we have shown that the picture of respiratory complaints among woodwork teachers was in accordance with that of other wood-dust-exposed groups, and the symptoms were related to work-environmental factors (27).

The selection of other school personnel as referents was an attempt to check other school environmental factors; for example, other irritants, sick building syndrome, and stress factors. In the woodwork teachers, the symptoms showed a work-related pattern with an increase of symptoms during the working week and improvement when off work. Reference subjects with complaints showed such a work-related pattern at a lower frequency.

The underlying mechanisms may be various, and nonspecific irritation and specific sensitization have often been discussed. Our aim was to determine whether IgE-mediated allergy could be the cause of the symptoms.

Screening for atopy was performed by Phadiatop. This test has a high sensitivity and specificity for clinically diagnosed inhalant allergy (9, 18, 24). A total of 29.9% of the woodwork teachers and 31.5% of the reference subjects had a positive Phadiatop. The figures are increased compared to other population studies (20–25% positive Phadiatop) (18, 22). But Freidhoff et al. (11) used a skin prick test for establishing atopy and found that the frequency of atopy was significantly higher in adult men (36%) than in women (20%). In a study population of 2076 individuals, Vervloet et al. (22) also reported that the percentage of a positive Phadiatop was significantly higher in men than in women. This could be an explanation of the high frequency of atopy in our study, because 90% of the subjects were men.

The questionnaire study (27) indicated that self-reported symptoms of hay fever, asthma, and atopic

dermatitis during adolescence were associated with an increased risk of several work-related complaints in eyes, airways, and skin. But self-reported atopic symptoms may be misclassified, leading to unreliable conclusions. Because the prevalence of IgE antibodies against common inhalant allergens (as measured with Phadiatop) was equal in our groups, this test could not confirm the importance of atopy for the complaints in the woodwork teachers – with the exception of eye symptoms.

Despite the high frequency of atopy, only one woodwork teacher had a positive RAST to a Scandinavian wood (alder), and two reference subjects had IgE antibodies to birch and pine. Hence, no correlation between exposure and sensitization to wood dust could be stated. The complaints related to exposure to dusts from the examined Scandinavian woods were probably not IgE mediated.

Specific IgE antibodies to wood determined by a positive radioallergosorbent test (RAST) have been presented in some reports; for example, reports on the following:

- western red cedar (*Thuja plicata*) where a positive RAST for one allergen, plicatic acid, was detected (21)
- African zebrawood (*Microberlinia*) (4)
- obeche (*Triplachiton scleroxylon*) (5).

But in many patients with such wood-dust-related rhinitis/asthma, no positive RAST was found (3, 5, 7, 17, 21).

Skin prick tests (SPT) for wood dusts were not performed in our study. Regarding SPT to wood dusts in occupationally exposed groups, several studies have reported a rather low frequency of positive SPT to wood-dust extract (5, 26). In the study of Carosso et al. (5), both SPT and RAST to wood-dust extracts were done in 90 woodworkers and 53 reference subjects. Only one of 12 subjects with positive SPT to wood-dust extract had a positive RAST (to obeche dust). Wilhelmsson et al. (26) performed SPT with seven wood extracts on 23 woodworkers with nasal symptoms consistent with hypersensitivity and 14 nonallergic reference persons. They found a positive reaction in only three woodworkers and one reference subject. Kanerva & Vaheri (15) reported 30 cases of occupational rhinitis with positive nasal challenge with wood dust and found that only 13 had a positive SPT to wood-dust extract. It can be argued that SPT is a better method than RAST for detecting an IgE-mediated reaction to wood-dust allergens. But if the extracts used in SPT contain irritants, false-positive *in vivo* tests would be obtained. Because there are no commercial SPT and RAST available, it is difficult to compare the studies. Furthermore, negative RAST results could be achieved if the assay lacked the allergens or contained too low a concentration. Since, on the one

hand, we obtained positive RAST in three teachers and in the positive control and, on the other hand, negative RAST against nonatopic serum samples with high IgE levels, we consider that our tests specifically detected subjects with IgE antibodies against at least some wood-dust-related allergens.

The results from our study suggest that most respiratory complaints from teachers exposed to wood dust are caused by irritation rather than allergy.

Acknowledgments

The Swedish Work Environment Fund (91-0220), the Swedish Asthma and Allergy Association, and the Swedish Medical Research Council supported this study.

References

- ANDERSEN HC, ANDERSEN I, SØLGAARD J. Nasal cancers, symptoms and upper airway function in woodworkers. *Br J Ind Med* 1977; 34: 201-7.
- BROOKS SM, EDWARDS JJ, HENDERSON EDWARDS F. An epidemiologic study of workers exposed to western red cedar and other wood dusts. *Chest* 1981; 80 (Suppl.): 30S-32S.
- BUSH RK, CLAYTON D. Asthma due to Central American walnut (*Juglans olanchana*) dust. *Clin Allergy* 1983; 13: 389-94.
- BUSH RK, YOUNG J, REED C. Asthma due to African zebra wood (*Microberlinia*) dust. *Am Rev Respir Dis* 1978; 117: 601-3.
- CAROSSO A, RUFFINO C, BUGIANI M. Respiratory diseases in wood workers. *Br J Ind Med* 1987; 44: 53-6.
- CESKA M, ERIKSSON R, VARGA JM. Radioimmunosorbent assay of allergens. *J Allergy Clin Immunol* 1972; 49: 1-9.
- CHAN-YEUNG M, DESJARDINS A. Bronchial hyperresponsiveness and level of exposure in occupational asthma to western red cedar (*Thuja plicata*). *Am Rev Respir Dis* 1992; 146: 1606-9.
- ENARSON DA, CHAN-YEUNG M. Characterization of health effects of wood dust exposures. *Am J Ind Med* 1990; 17: 33-8.
- ERIKSSON NE. Allergy screening with Phadiatop® and CAP Phadiatop® in combination with a questionnaire in adults with asthma and rhinitis. *Allergy* 1990; 45: 285-92.
- FLECHSIG R, NEDO G. Review: hazardous health effects of occupational exposure to wood dust. *Ind Health* 1990; 28: 107-19.
- FRIDHOFF IR, MEYERS DA, BIAS WB, CHASE GA, HUSSAIN R, MARSH DG. A genetic-epidemiologic study of human immune responsiveness to allergens in an industrial population. I. Epidemiology of reported allergy and skin-test positivity. *Am J Med Genet* 1981; 9: 323-40.
- GOLDSMITH DF, SHY CM. Respiratory health effects from occupational exposure to wood dusts. *Scand J Work Environ Health* 1988; 14: 1-15.
- HOLMSTRÖM M, WILHELMSSON B. Respiratory symptoms and pathophysiological effects of occupational exposure to formaldehyde and wood dust. *Scand J Work Environ Health* 1988; 14: 306-11.
- HOLNESS DL, SASS-KORTSAK AM, PILGER CW, NETHERCOTT JR. Respiratory function and exposure-effect relationships in wood dust-exposed and control workers. *J Occup Med* 1985; 27: 501-6.
- KANERVA L, VAHERI E. Occupational allergic rhinitis in Finland. *Int Arch Occup Environ Health* 1993; 64: 565-8.
- LI D, YUAN L, YI S, JIANG Z. Effects of wood dust exposure on respiratory health: cross-sectional study among farmers exposed to wood dust. *Am J Ind Med* 1990; 17: 84-5.
- MALO JL, CARTIER A. Occupational asthma caused by exposure to ash wood dust (*Fraxinus americana*). *Eur Respir J* 1989; 2: 385-7.
- MATRICARDI PM, NISINI R, PIZZOLO JG, D'AMELIO R. The use of Phadiatop® in mass-screening programmes of inhalant allergies: advantages and limitations. *Clin Exp Allergy* 1990; 20: 151-5.
- PISANIELLO DL, TKACZUK MN, OWEN N. Occupational wood dust exposures, lifestyle variables, and respiratory symptoms. *J Occup Med* 1992; 34: 788-92.
- TATKEN RL, BROWNING CA. Health effects of exposure to wood dust: a summary of the literature. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Standards Development and Technology Transfer, Cincinnati, Ohio, June 1987.
- TSE KS, CHAN H, CHAN-YEUNG M. Specific IgE antibodies in workers with occupational asthma due to western red cedar. *Clin Allergy* 1982; 12: 249-58.
- VERVLOET D, HADDI E, TAFFOREAU M, LANTEAUME A, KULLING G, CHARPIN D. Reliability of respiratory symptoms to diagnose atopy. *Clin Exp Allergy* 1991; 21: 733-7.
- WERNER U. Zur Einwirkung von Holzstäuben und Reizgasen auf den oberen Atemtrakt. *Z Ges Hyg Grenzgeb* 1979; 25: 290-3.
- WEVER AMJ, WEVER-HESS J, VAN SCHAYCK CP, VAN WEEL C. Evaluation of the Phadiatop® test in an epidemiological study. *Allergy* 1990; 45: 92-7.
- WHITEHEAD LW. Health effects of wood dust - relevance for an occupational standard. *Am Ind Hyg Assoc J* 1982; 43: 674-8.
- WILHELMSSON B, JERNUDD Y, RIPE E, HOLMBERG K. Nasal hypersensitivity in wood furniture workers. *Allergy* 1984; 39: 586-95.
- AHMAN M, SÖDERMAN E, CYNKIER I, KOLMODIN-HEDMAN B. Work-related respiratory problems in industrial arts teachers. *Int Arch Occup Environ Health* 1995; 67: 111-18.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.