

Nasal Hypersensitivity in Wood Furniture Workers

An Allergological and Immunological Investigation with Special Reference to Mould and Wood

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Occupational nasal allergies caused by moulds and wood dust were extensively studied in six wood furniture factories. The concentration of moulds, wood dust and endotoxins was registered and occasionally high values were found. *Paecilomyces* spec. was the most common mould. Answers to a special questionnaire showed that 16% (42/268) of the wood workers with a mean exposure time of 12 years had a history compatible with hypersensitivity in the upper airways associated with their work. Calculations based on skin prick tests and provocation tests with relevant allergens showed that the wood furniture workers had an incidence of allergy to moulds in 3% and to wood dust in 2%. Most of the workers (5/7) sensitive to moulds and/or woods were also skin prick sensitive to other allergens. No statistical difference concerning the presence of precipitating antibodies against mould and wood antigens could be registered between workers with discomfort and workers without symptoms.

Key words: mould allergy; nasal hypersensitivity; precipitation antibodies; wood allergy; wood workers.

Accepted for publication 24 April 1984

It has long been recognized that nasal obstruction and discharge commonly occur among wood furniture workers, and that these symptoms seem to be correlated to the concentration of wood dust in the working environment (3, 22). Epidemiologically, it has also been demonstrated that in wood furniture workers adenocarcinoma in the ethmoidal region is highly overrepresented (2, 10, 14).

In the course of investigations on 723 workers from 50 wood furniture factories in the county of Småland in southern Sweden, with regard to cellular changes in the nasal mucosa, we also noticed that some of them reported discomfort similar to allergic symptoms, such as sneezing, nasal obstruction and nasal discharge (33). With the aid of a questionnaire dealing with symptoms from the upper airways, we found a significantly higher inci-

dence ($P < 0.05$) of perennial nasal obstruction and discharge compared with a reference group not or only slightly exposed to wood dust. Furthermore, the number of upper respiratory tract infections was higher ($P < 0.05$) than in the non-exposed reference subjects (33). The purpose of this study was to investigate the possible immunological mechanisms underlying the nasal symptoms. The question of allergy to moulds and wood, for example, was of particular interest.

We therefore constructed a special questionnaire to be answered by the woodworkers, measured the concentration of fungus spores, wood dust and endotoxins in the air of the factories, and performed allergological and immunological tests with extracts of relevant antigens.

MATERIAL AND METHODS

Of the original 50 factories involved, five furniture industries were chosen at random and a parquet flooring factory was also included as in our earlier histological study (34). The factories were selected so as to differ with respect to geographical location, size, age, type of manufacturing and technological standard.

Questionnaire

A questionnaire specially designed for this occupational situation was used. Thirty-eight questions were asked concerning the general state of health and history of illness, including heredity (questions 1–3), conditions and time of exposure, including a definition of the most frequent kind of work performed (4–11), factors provoking suspected allergic symptoms (12–21), and descriptions of symptoms from the nose, eyes, lower airways and skin connected with the occupational environment (22–38). The investigation was carried out as a cross-sectional study. The questionnaire was given to all 298 workers on duty on the days of investigation. No clerks were included. Workers who reported symptoms of hypersensitivity in the questionnaire were selected for further investigations and were compared with a reference group from the same factories and exposed to wood dust, but free from symptoms.

Collecting of moulds

Analyses concerning quantities and types of moulds were performed on two different occasions, summer and winter, with 6 months in between. Air samples were taken during factory work, from the breathing zone of the workers. Air-borne moulds were collected by means of a Multistage liquid impinger (20 l/min model, A. W. Dixon and Co., London) for isokinetic sampling (15). The air was sucked into the top of a special glass bottle in which particles were collected by impaction in a flowing medium (3% malt extract, Difco).

A special pump for this equipment was used (Neuberger type MW 64-4, Bergman and Be-

ving, Stockholm, Sweden). The sampling fluid was dispersed onto malt extract agar plates. After incubation at room temperature for 5–7 days the colony-forming units (cfu) were counted, and representative mould colonies were identified on morphological diagnostic features. In addition, samples of wood dust were taken from the surfaces of the machines in the breathing zone for cultivation and identification of moulds.

Determination of endotoxin content

The specific activity of endotoxins, produced by Gram negative bacteria, in the air of the factories was investigated on the same two occasions as concerning the moulds, by means of the *Limulus* amebocyte lysate (LAL) test (32). For air samples, a special pump with an air flow metre (MSA pump, 3 l/min; Ramström, Solna, Sweden) and a collector with filters with a pore size of 0.8 μm (Millipore, Bedford, Mass., USA) were used.

Determination of wood dust concentration

The total wood dust concentration in the air was measured according to a standard method used by the National Board of Occupational Safety and Health (4). The equipment was the same as that described above for endotoxins. Analyses were performed through weighing the filters before and after the measurement.

Preparation of mould and wood extracts

Mould extract. Ten extracts (four *Penicillium*-, two *Paecilomyces*-, two *Cladosporium*-, one *Aspergillus*- and one *Trichoderma* species) were prepared from the most common moulds isolated from the factories by growing them in glass bottles containing Sabouraud Liquid Medium (19) at 25°C for 1–2 weeks under continuous shaking (200 rpm). The broth was separated from the organism by filtration and the filtrate was dialysed against distilled water (2 $\times$ 24 H) in a dialysis tube which retained molecules greater than 10,000 daltons, and was thereafter freeze-dried and stored. For preparation of the aller-

gen extract for prick test, the freeze-dried mould extract was thawed and suspended in buffered saline solution (pH 7.0) to a final protein concentration of 0.5 mg/ml (Bio-Rad method (7)). The extracts were analysed by the Limulus (LAL) test for the determination of the pyrogen and endotoxin contents (32). No pyrogens or endotoxins were found. For sterilization the extracts were further filtrated through a 450-nm pore size membrane (Millipore Corp., Bedford, Mass., USA) and sterility was checked by aerobic and anaerobic cultivations. For the precipitation tests the freeze-dried mould extracts were rehydrated in distilled water and the protein content was determined. Seventeen different extracts from thermophilic Actinomycetes and different moulds* prepared in the same way were used as reference antigen preparations. These extracts are included in a standardized panel of antigen extracts used for serological screening of precipitating antibodies in hypersensitivity pneumonitis at the Mycology Section, National Bacteriological Laboratory (NBL), Stockholm.

Wood extract. Wood dust was prepared from seven types of woods (fir, oak, beech, birch, walnut, teak and mahogany), all obtained from the investigated factories. A sanding machine operating in exactly the same way as those in the furniture factories was used for preparing fresh wood dust. Extraction of allergens from the wood dust was accomplished in 0.1 M phosphate buffer (pH 7.5) by low speed centrifugation (4,000 rpm) for 15 min. The supernatant was then centrifuged ($40,000 \times g$; 90 min; 4°C) dialysed and freeze-dried in the same way as for the moulds. The protein, pyrogen and endotoxin content was determined, the dilution and sterilization was made and the sterility checked in the same way as for mould extracts.

Standardization of extracts

The special mould and wood extracts used for the allergological investigations were standardized with respect to milligrams of protein/ml. The stem solution contained 0.5 mg protein/ml. The commercial extracts used were manufactured and supplied by Allergologisk Laboratorium, Copenhagen (ALK) (dog, horse, cat, *Dermatophagoides pteronyssinus*) and Pharmacia, Sweden (birch, timothy, mugwort). These extracts were standardized with respect to histamine equivalent point (HEP) and biological units (BU) (1). The protein content in the commercial extracts used (five HEP units or 100,000 BU) corresponded well with the protein content in our specially prepared mould and wood extracts (dog 0.43 mg protein/ml, horse 0.23 mg protein/ml, cat 0.33 mg protein/ml, *Dermatophagoides* 0.24 mg protein/ml, birch 0.37 mg protein/ml, timothy grass 0.37 mg protein/ml, mugwort 0.52 mg protein/ml).

Prick tests

Prick tests were performed with a lancet as described by Pepys (18) on the volar side of both forearms in two straight rows, using the concentration of the stock solution. A standard prick panel was used consisting of seven different commercial allergen extracts (see above) together with the 10 specially prepared mould extracts and the seven specially prepared wood extracts mentioned above, and also with histamine chloride (1 mg/ml). The skin reaction was recorded after 10 min and compared with the histamine reaction. The contour of the weal was outlined with a fine fibre tip pen and transferred to a paper by means of adhesive tape. The weal area was calculated as the sum of the greatest diameter and the diameter at right angles to it. A weal reaction stronger than or similar to the weal obtained with the histamine solution was regarded as strongly positive (3+). A moderate reaction, i.e. half of the histamine reaction, was graded as 2+, and a weak reaction, i.e. one quarter of histamine weal, as 1+. Skin reactions with a weal strength of less than 1+ were considered negative.

* *Microsporysphaera faeni*, *Thermoactinomyces vulgaris*, *Thermoactinomyces candidus*, *Aspergillus fumigatus*, *Aspergillus niger*, *Aspergillus nidulans*, *Aspergillus terreus*, *Aspergillus flavus*, *Mucor* sp., *Cladosporium herbarum*, *Alternaria iridis*, *Aureobasidium pullulans*, *Phoma herbarum*, *Trichoderma viride*, *Penicillium* sp., *Fusarium roseum*, *Helmintosporium* sp.

Prick tests were performed on two groups of workers occupationally exposed to wood dust, one group with nasal symptoms compatible with allergy and the other, the reference group, without such symptoms. The extracts were also tested on 25 non-allergic control persons from the hospital staff.

Provocation tests

Provocation tests with mould and/or wood extracts were performed on those workers who had reacted positively to moulds and/or wood dust extracts at skin prick tests. The extract which gave the strongest skin reaction was chosen for the provocation test. If similar reactions were registered for several allergens, the one chosen for the provocation was the one most prevalent in the occupational environment. The tests were done by instillation of increasing concentrations of 0.2 ml of the mould and wood extracts into the nostril, using a nasal speculum, and into the conjunctival sac. The test was begun, as a control, with 0.15 M NaCl followed by a 10-fold dilution of the skin prick test concentration. If no reaction was observed after 10 min the test was repeated with the stock solution. A provocation test was regarded as positive if observable symptoms occurred in the form of nasal obstruction, sneezing, and tearing and/or redness of the conjunctiva. Just one provocation was performed on the same occasion.

Immunoglobulin E

The serum level of immunoglobulin E (IgE) was measured by the Phadebas paper radioimmunosorbent test – PRIST (Pharmacia Diagnostics) at the Chemical Laboratory of Huddinge Hospital, Sweden.

Counterimmunoelectrophoresis

Serological tests for precipitating antibodies against wood extracts and mould extracts were performed by counterimmunoelectrophoresis (CIE) (25).

This technique is based on two electrokinetic

phenomena where antibodies are forced to move in a supporting gelmedium (agarose) at pH 8.6 towards the cathode by virtue of the electroendosmotic flow, while negatively charged antigen molecules at pH 8.6 move electrophoretically towards the anode. At the confrontation line the reagents form antigen-antibody complexes which precipitate in the presence of an adequate relationship between reagents.

One per cent agarose (Litex, Glostrup, Denmark) in 0.1 M veronal buffer, pH 8.6, was layered to a depth of 1 mm on 10 × 10 cm Gelbond plates. Wells 4 mm in diameter, set 4 mm edge to edge were cut in gel. Electrophoresis (90 min; 10 V/cm) was carried out with serum wells as the anode. The immune gels were washed in 0.01 M PBS, pH 7.0, to remove the non-reacting proteins, and were then stained with Coomassie Brilliant Blue R250 according to the method of Weeke (31) to obtain permanent documentation of the immune precipitates.

RESULTS

Questionnaire

The questionnaire was answered by 268 workers corresponding to 90% of the workers employed on that day in these factories. From the answers to the questionnaire, it was found that 42 (16%) of these 268 workers with a mean exposure time of 12 years (range 1–38 years) had a history compatible with a possible allergy in the upper airways connected with their work. By virtue of the shift system that was used in the factories we were able to perform a thorough allergological investigation in 23 of these 42 workers; 23 are referred to as the discomfort group. Another group of 14 workers, exposed to wood dust to about the same extent as those in the discomfort group but free from symptoms, served as controls, matched for sex and age, and was called the reference group.

Table 1
Environmental variables in the investigated factories

Factory No.	Living mould spores/m ³ air		Endotoxins µg/m ³ air		Wood dust mg/m ³ air	Age of building in years	Age of ventilation systems in years	No. of workers employed
	December	June	December	June				
I	576	351	0.0033–0.0053*	0.0055–0.35	1.33 (0.35–2.99)	26	8	129
II	645,328	1046	0.062	0.0030–0.0059	0.41 (0.36–0.52)	65	16	24
III	18,531	288	0.0029	0.002–0.33	1.70 (0.83–2.76)	29	29	46
IV	788	177	0.0017–0.0044	0.0027–0.0030	4.03 (1.94–5.06)	21	21	27
V	166	306	0.0027–0.0036	0.0110	1.38 (0.30–2.96)	20	1	120
VI	2564	2588	0.0012	0.0023–0.0032	1.30 (0.36–3.23)	6	6	65

* Two different measurement points.

Table 2

Results of the allergological examination concerning wood furniture workers with nasal symptoms and with positive prick tests against moulds

Test person No.	Positive provocation test to moulds + (0.5 mg protein/ml) ++ (0.05 mg protein/ml)	Total serum IgE kU/l	Positive prick tests to allergens other than moulds	History of obstructive lung disease	Years of employment
1	++	501	birch pollen, mugwort, dog, cat, <i>Dermatophagoides</i>	+	19
2	–	9	–	+	38
3	+	443	–	–	2
4	++	39	–	+	7
5	–	9	–	–	30
6	++	671	dog, horse, cat, <i>Dermatophagoides</i> mahogany	+	7
7	++	21	birch pollen, <i>Dermatophagoides</i>	+	8
8	–	102	birch pollen, timothy	+	12
9	–	24	birch pollen, birch wood, mugwort	–	36
10	–	17	–	–	17

– means negative.

Mould concentration

There were great differences in the amounts of living mould spores between the factories and also between different seasons. A high concentration of allergenic moulds was noted in winter in most of the factories. There was some correlation between the concentration of moulds and the age of the buildings (Table 1).

Totally, 12 different measurements of moulds in the environment of the factories were performed, and in five of the measurements *Paecilomyces* predominated. Other common moulds were *Penicillium*, *Cladosporium*, *Aspergillus* and *Trichoderma* spec.

There was a concordance between the predominant type of mould in the air and that in the wood dust in four of the six factories investigated.

Endotoxin concentration

In two factories, one of which also showed a high concentration of moulds (Table 1), high endotoxin values, i.e. more than 0.30 $\mu\text{g}/\text{m}^3$ air (23), were found in summertime. As a whole, however, there was no correlation between the

concentration of endotoxins and that of mould spores.

Wood dust concentration

The mean concentration of wood dust differed considerably between the different factories, ranging from 0.32–4.03 mg/m^3 air. The mean value was 2.0 mg/m^3 air, which should be compared with the Swedish Threshold Limit Value of 4.0 mg/m^3 (Table 1). There was no correlation between the wood dust concentration and the concentrations of moulds and endotoxins.

Mould allergy

Ten (44%) of the 23 subjects in the discomfort group (Table 2) and two (14%) of the 14 in the reference group were prick test positive to one or more of the mould extracts. All factories were represented. None of the staff control group was prick test positive. Of the total 12 prick test positive cases, 10 were positive to *Paecilomyces*, 5 positive to *Aspergillus* and *Cladosporium*, 4 positive to *Penicillium* and 1 positive to *Trichoderma*. None had positive reactions to all mould extracts. There was some but not com-

Table 3

Results of the allergological examination concerning wood furniture workers with nasal symptoms and with positive prick tests against wood dust. (Test persons No. 1 and 2 in this table correspond to test persons No. 6 and 9 in Table 2, respectively)

Test person No.	Positive provocation test to wood + (0.5 mg protein/ml) ++ (0.05 mg protein/ml)	Total serum IgE kU/l	Positive prick tests to allergens other than wood	History of obstructive lung disease	Years of employment
1	+	671	<i>Dermatophagoides</i> , <i>Cladosporium</i> , <i>Aspergillus</i>	+	7
2	+	24	birch pollen, mugwort, <i>Cladosporium</i> , <i>Paecilomyces</i>	—	36
3	+	49	—	—	4

— means negative.

plete concordance between the skin reactions to moulds of the same species but from different locations.

Five (50%) of the 10 prick test positive subjects of the discomfort group were positive at the nasal and eye challenge (Table 2), but none of the reference group showed this result. Three of the five had a serum IgE-level higher than 100 kU/l. Four of them had also slight symptoms of obstructive lung disease.

The CIE-tests to moulds from our own panel revealed that 65% (15/23) of the workers in the discomfort group had precipitates to one or several of the moulds and 64% (9/14) in the reference group. In the staff control group the incidence was 9% (2/22).

Two cases with a history of allergic alveolitis found in the questionnaire showed negative reactions to our mould allergens but showed positive reactions to the NBL panel (*Microspora faeni* and *Cladosporium herbarum* respectively).

The precipitation reactions observed at CIE were most commonly evoked by *Paecilomyces* (32%), the most frequent kind of mould found in the factories. There was no difference between the discomfort and the reference group concerning the precipitation reactions to *Paecilomyces* (35% (8/23) and 29% (4/14) respectively). In the staff control group the corresponding frequency was 9% (2/22).

Wood dust allergy

Three (13%) of the 23 workers in the discomfort group and one (7%) of the 14 in the reference group were found to be prick test positive to the wood dust extracts. These workers came from different factories. Kinds of wood giving positive prick tests were oak, mahogany, beech, birch and teak. All three prick test-positive persons with nasal discomfort were also positive at nasal challenge tests with extracts of oak, teak and birch respectively (Table 3). The only person from the reference group with a positive prick test reaction showed a negative result at nasal and eye challenge tests.

One of the persons with a positive result at prick tests and nasal challenge tests against

wood also had asthma, and in addition showed a positive reaction to prick and provocation tests against moulds, and had a high serum level of IgE (671) (Table 3). Of some interest was the finding that one worker was prick test positive to both birch pollen and birch wood.

Concerning the precipitating antibodies to wood allergens, it was found that all of the sera tested (from the discomfort group, reference group and the staff group) showed precipitating lines to some wood allergens as mahogany. Other kinds of wood as oak and teak often gave precipitins while other types of wood as birch, beech and fir seldom gave any reaction. There was, however, no significant difference between the groups concerning precipitins to any kind of wood.

DISCUSSION

Allergic inflammatory disease (allergic alveolitis) from moulds in the lower airways is known to occur in the saw mill industry (5, 35). Reports on occupational rhinitis in wood manufacturing industries are very sparse (11). The frequency of rhinitis caused by moulds is estimated to about 0.5% in rhinitis patients attending an allergological clinic in Stockholm (29).

From the answers to our specially designed questionnaire, we found that 16% of the wood workers in the factories examined had a history compatible with an occupational nasal hypersensitivity, while many more were inconvenienced by wood dust in a more unspecific way. In earlier studies (33) it was found that among heavily exposed wood furniture workers, 20% had nasal hypersecretion and 40% had perennial nasal obstruction.

The amounts of living mould spores in the different factories varied considerably but the highest concentrations of allergenic moulds were seen in winter time, which contrasts with the normal situation (21). There was also a clear tendency for newer industrial buildings with modern ventilation equipment to show lower concentrations of mould spores and wood dust particles in the air. On the other hand, there was not such a clear tendency

concerning the amount of endotoxins in the environmental air.

Normal concentrations of mould spores in private houses, determined by the same method as was used in this study, are 40–500/m³ air (12). In houses damaged by moulds the spore concentration can rise to 15,000/m³, but will hardly reach a value as high as that we found in one of the factories in this study, namely 645,000/m³. There are reasons to suspect that the risk of sensitization in this environment is high. Concerning endotoxins, little is known about their effects on the upper airways, but in the lungs inspired air with an endotoxin concentration higher than 0.30 µg/m³ can after some hours breathing cause an inflammatory reaction (23, 27) accompanied by fever and bronchoconstriction. No exact threshold limit values above which such symptoms will appear are, however, available. The response is considered to be a question of the individual hyperreactivity state in the airways caused, among other things, by smoking and allergies. Such hyperreactivity state, however, can be further increased by endotoxins, while the mucosal receptors registering irritation in the airways are reported to become more sensitive by e.g. coli endotoxins (26). It has also been claimed that an epithelial damage of the mucosa in the bronchi would facilitate an immunological reaction (24) and these reports increased our interest for the simultaneous registration of endotoxins, wood dust and mould spores in the air of the factories. Epithelial metaplasia with absence of cilia is very common in the nasal mucosa of these wood workers (33), giving an anatomical basis for similar effects in the nose. We were not, however, able to support this theory. Thus, there was no correlation between the concentration of endotoxins, moulds and wood dust in the factories investigated, and a high frequency of mould hypersensitivity was not found in the two factories with high concentration of endotoxins.

Paecilomyces, which was the most prevalent kind of mould in this industrial environment, is known to be allergenic and associated with wood.

After challenge with mould allergens we

found that five (22%) persons of the 23 with upper-airway discomfort had nasal hypersensitivity to mould. Assuming that the incidence of nasal allergy to moulds was the same in the 19 non-tested night shift workers with occupational symptoms, it could be calculated that the incidence of nasal allergy to moulds would be 3% (9/268) in the wood furniture workers. In the factories where the shift system was used, the workers changed shift every month, so this estimation should be reasonable.

A high frequency of precipitating antibodies against moulds was found in the wood-workers, both those with (65%) and those without (64%) upper-airway discomfort, as compared with the staff reference group (9%). This might lead to the conclusion that the sensitization to mould can occur in this environment, but that there is no correlation between nasal and pulmonary symptoms and the occurrence of precipitating antibodies.

Investigations dealing with nasal wood dust allergies have been few (6, 11) compared with those concerning symptoms from the lower airways caused by wood dust (13, 20, 28, 30). No epidemiological studies concerning nasal wood dust allergy exist to our knowledge. In our study we found that three (13%) of 23 with nasal discomfort were allergic to certain kinds of wood dust, both exotic (teak) and non-exotic (oak, birch).

If it is presumed, as in the case of moulds, that the incidence of allergy to wood dust was the same in the 19 non-tested night shift workers, this would mean that 2% (6/268) of the workers in the investigated furniture industries were allergic to wood dust.

No conclusions could be drawn concerning the wood dust precipitin reactions, as nearly all investigated sera showed some precipitation lines when tested to wood antigens. The precipitation could be elicited by high concentrations of substances other than antigens. It could be comparable to a pseudoimmune-reaction as described in connection with byssinosis (9). Like us, other groups (6, 8) have failed to develop serological tests that are helpful in the diagnosis of wood dust allergies, but others (11, 30) have shown precipitation lines using

aqueous wood dust extracts on wood dust-exposed workers.

Thus, it is still controversial whether precipitating antibodies are of importance in some types of nasal allergy (16). We have not come closer to a solution of this question, and further research is needed.

In conclusion we can say that nasal perennial hypersensitivity is common among workers in the wood industries examined (16%, 42/268) and considerably exceed the frequency seen in the Scandinavian population in general (17). Allergies to moulds and wood dust can thus explain one third of these discomforts (5%, 13/268) but, in the rest of the cases other causes, such as unspecific hyperreactivity, might explain the symptoms. In this respect also toxic metabolites from moulds i.e. mycotoxins should be considered.

Mould and wood dust allergies seem to preferably strike persons who are allergic to several allergens such as pollens and animal epithelium.

For this reason the frequency of mould and wood allergy in the factories is mainly dependent on the number of atopic workers. However, we can say, because of the finding of high concentrations of moulds, wood dust as well as endotoxins at certain measurements, it is obviously of utmost importance to minimize the exposure of the workers to potentially biologically active material such as wood dust, moulds and bacterial endotoxins. In our experience the best way to achieve this is to use modern, effective ventilation equipment in this industrial environment. We do not consider it realistic to recommend that the workers wear masks except in special situations when the exposure is extremely heavy.

ACKNOWLEDGEMENTS

The analysis of the endotoxins was made by Professor Ragnar Rylander, the Department of Environmental Hygiene, University of Gothenburg, Sweden and he is gratefully acknowledged.

This work was supported by grants from the Swedish Work Environment Fund (project No. 80-0310) and the Swedish National Association against Heart and Chest diseases.

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