Rhinology, 23, 297-302, 1985

Nasal hypersensitivity in wood furniture workers

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SUMMARY

Occupational nasal allergies were 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. 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 to their work. Rhinomanometric investigations showed mucosal congestion in the workers with nasal discomfort and nasal clearance was pathologically slow in 54%. Histological studies revealed a high incidence of unciliated and metaplastic nasal epithelium. Skin prick tests and provocation tests with standard allergens and allergens prepared from the moulds and the wood dust in the environment 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 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.

It has long been recognized that nasal obstruction and discharge commonly occur among wood furniture workers (Andersen et al., 1977; Ruppe, 1973). Epidemiologically it has also been demonstrated that in wood furniture workers adenocarcinoma in the ethmoidal region is highly overrepresented (Engzell et al., 1978; MacBeth, 1965). 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.

Six wood furniture industries were chosen at random. A study was performed as a field study in the county of Småland where most of the furniture industries in Sweden are localized (Wilhelmsson et al., 1985). The factories were selected so as to differ with respect to geographical location, size, age, type of manufactoring and technological standard.

Paper presented at the 10th Congress of the European Rhinologic Society and 4th ISIAN, Nancy (France), August 1984.

A questionnaire with 38 questions, specially designed for this occupation and situation was used. The questionnaire was given to all 298 workers on duty on the days of investigation. No clerks were included. Ninety % of the workers answered the questionnaire properly. It was found that 42 of the 268 workers who answered, that is 16%, had a history compatible with a hypersensitivity 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. Another group of 14 workers, exposed to wood dust to about the same extent, but free from symptoms, served as controls, matched for sex and age. Furthermore another control group consisting of 25 healthy persons from the staff of the Department of Otolaryngology, Huddinge Hospital, was used.

The total wood dust concentration in the breathing zone was measured according to a standard method used by the National Board of Occupation, Safety and Health in Sweden (1979). For air samples, a special pump with an air flow meter and a collector with a millipore filter was used. The mean concentration of wood dust differed considerably between the different factories, ranging from 0.3 to 4.0 mg/m^2 air. The mean values was 2.0 mg/m^2 air, which should be compared with the Swedish threshold limit value of 4.0 mg/m^2.

The specific activity of endotoxins, produced by gramnegative bacteria, in the air of the factories was investigated by means of the limulus-amoebocyte-lysate (LAL) test (Wildfleur et al., 1974). The equipment on the field was the same as that described for determination of wood dust concentration. In two factories high endotoxin values, that is more than 0.30 $\mu g/m^2$ air, were found. Concerning the effects of endotoxins on the upper airways little is known, but in the lungs inspired air with an endotoxin concentration higher than 0.30 $\mu g/m^2$ air can after some hours breathing cause an inflammatory reaction accompanied by fever and bronchoconstriction (Rylander, 1982; Snella and Rylander, 1982). If this can act on the nasal mucosa as well, is not yet known.

It has also been claimed by Rylander (1982) that an epithelial damage to respiratory mucosa could facilitate an immunological reaction. We found in our nasal biopsies a high frequency of cuboidal metaplasia with loss of cilia (Wilhelmsson and Lundh, 1984) as well as very slow or absent nasal mucociliary clearance in more than 50% in the investigated wood workers (Wilhelmsson and Drettner, 1985). These factors could facilitate an absorption of allergens and thus also facilitate an immunological reaction (Wilhelmsson and Lundh, 1984).

Analyses concerning quantities and types of moulds were performed on two different occasions, summer and winter, with six months in between. Air samples were taken during factory work, from the breathing zone of the workers. Airborne moulds were collected by means of a multistage liquid impinger for isokinetic sampling (May, 1966).

Table 1. Environmental variables in the investigated factories.

factory no.	living mould spores/m^3 air December	June	endotoxins µg/m^3 air December	June	wood dust mg/m^3 air mean range
I	576	351	*0.0033–0.0053	0.0055–0.35	1.33 (0.35–2.99)
II	645,328	1,046	0.062	0.0030–0.0059	0.41 (0.36–0.52)
III	18,531	288	0.0029	0.002 –0.33	1.70 (0.83–2.76)
IV	788	177	0.0017–0.0044	0.0027–0.0030	4.03 (1.94–5.06)
V	166	306	0.0027–0.0036	0.0110	1.38 (0.30–2.96)
VI	2,564	2,588	0.0012	0.0023–0.0032	1.30 (0.36–3.23)

* Two different measurement points.

There were great differencies in the amounts of living mould spores between the factories and also between different seasons (Table 1). A high concentration of allergenic moulds was noted in winter in most of the factories. Normal concentrations of mould spores in private houses, determined by the same method as was used in this study, are 40–500 spores/m^2 of air. In houses damaged by moulds the spore concentration can rise to 15.000/m^2 air (Holmberg, 1984).

Paecilomyces was the most prevalent kind of mould in this industrial environment and is known to be allergenic and associated with wood but not common in private houses. Other common moulds were penicillium, cladosporium, aspergillus and trichoderma species.

The wood and mould extracts for prick tests were prepared by extracting allergens from mycelia and wood dust respectively, by help of buffered saline solution in an ultracentrifuge. Small molecules, that is less than 10 000 daltons, were dialysed away. The extracts were standardized according to mg protein/ml solution. The stem solution had 0.5 mg protein/ml. This concentration was chosen because it is similar to that of the commercial extracts used in the screening allergological tests on the workers.

Allergological investigations were performed as prick tests and those who were prick test positive were challenged in the eye and the nose with conventional methods. There were 10 out of 23 in the group with symptoms who had a reaction to one or several moulds in the environment and half of them were also clearly positive at challenge (Table 2). Three of this five workers were also positive to other allergens. Four of these ten had a high IgE value (> 100 kV/l). In the non-discomfort group there were two workers who were prick test positive but none was positive at challenge. In a control group consisting of 25 persons from the staff of our hospital there was nobody prick test positive for the moulds investigated.

Concerning the wood dust extracts, 3 out of 23 workers were prick test positive and all 3 were also positive at nasal challenge (Table 3). Two of them also had a reaction to other allergens. One worker in the non-discomfort group had a reac-

Table 2. Results for wood furniture workers with nasal symptoms and with positive prick tests against moulds.

test person no.	positive test to moulds + (0.5 mg protein/ml) + + (0.05 mg protein/ml)	total serum IgE kU/l	positive prick tests to other allergens than moulds
1	+ +	501	birch pollen, mug-worth, dog, cat, Dermatophagoides
2	–	9	–
3	+	443	–
4	+ +	39	–
5	–	9	–
6	+ +	671	dog, horse, cat, Dermatophagoides, mahogany
7	+ +	21	birch pollen, Dermatophagoides
8	–	102	birch pollen, timothy
9	–	24	birch pollen, birch wood, mug-worth
10	–	17	–

tion on prick test but was negative at challenge. No reaction was seen in the hospital staff control group.

Serological test for precipitating antibodies against wood extracts and mould extracts were performed by counter-immunoelectrophoresis (CIE). Concerning moulds, precipitation reactions were most often produced by paecilomyces, in 32%, which was the most predominating kind of mould in these factories. On the other hand no difference could be observed between the discomfort and non-dis-

Table 3. Results for wood furniture workers with nasal symptoms and with positive prick tests against wood dust. (Test person no. 1 and 2 in this table correspond to test person no.6 and 9 in Table II 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 other allergens than wood
1	+	671	Dermatophagoides, Cladosporum Aspergillus
2	+	24	birch pollen, mug-worth, Cladosporum Paecilomyces
3	+	49	–

comfort groups which both had the same exposure conditions. From this finding one could conclude that the occurrence of precipitating antibodies is an indication of exposure to the kinds of moulds in question but it does not correlate to symptoms.

Concerning precipitating antibodies to wood dust extracts we got contradictory results. Some extracts, as mahogany, gave precipitating reactions with sera from all groups of subjects. Certain kinds of wood as oak and teak often gave precipitates but others as birch rarely produced any precipitation in any of the subjects. These results are thus difficult to evaluate yet, but there was, however, no significant difference between the groups in this aspect.

CONCLUSIONS

It could be concluded that the study showed a high frequency of cuboidal metaplasia in the nasal mucosa, which also automatically means loss of cilia and impaired nasal clearance. Further it was found that the levels of wood dusts in the environment were on the whole below the threshold limit value; sometimes very high concentrations of moulds were found and in certain cases high concentration of endotoxins.

In our material we found that 3% of the wood furniture workers had an allergy against moulds in the upper airways, proved by nasal challenge. Corresponding figure for wood dust allergy was 2%. Both these figures are most probably elevated compared with the general population.

ZUSAMMENFASSUNG

Berufsbedingte Nasenallergien wurden in sechs Holzmöbelindustrien studiert. Die Konzentrationen von Schimmel, Holzstaub und Endotoxinen wurden registriert und manchmal hohe Werte wurden gefunden. Paecilomyces war die gewöhnlichste Schimmelsorte.

Ein besonderes Frageformular zeigte, dass 16% (42/268) der Holzarbeiter - mit einer durchschnittlichen Expositionzeit von 12 Jahren - hatten eine Krankengeschichte vereinbar mit Überempfindlichkeit in den oberen Luftwegen die direkt zu ihrer Arbeit assoziiert werden konnte. Rhinomanometrische Untersuchungen zeigten Schleimhautanschwellungen bei Arbeitern mit Nasenbeschwerden und die reinhaltende Cilieaktivität war bei 54% pathologisch langsam. Histologische Studien erweisen ein hohes Vorkommen von nicht cilieirtem und metaplastischem Nasenepithel.

Haut- und Provokationsteste mit Standardallergenen und Allergenen von Schimmelarten und Holzstaub aus den aktuellen Milieun zeigten, dass die Holzmöbelindustriearbeiter Zeichen für Nasenallergien gegen Schimmel in 3% und gegen Holzstaub in 2% hatten. Die meisten Arbeiter, die empfindlich gegen Schimmel und/oder Holzstaub waren, waren auch Hauttestempfindlich gegen andere

Allergene. Kein statistischer Unterschied in Bezug auf präzipitierende Antikörper gegen Schimmel und Holzstaubantigen konnte zwischen Arbeitern mit Beschwerden und Arbeitern ohne Symptome registriert werden.

REFERENCES

1. Andersen HC, Andersen I, Solgaard J. Nasal cancers, symptoms and upper airway function in wood workers. Br J Ind Med 1977; 34:201-7.
2. Arbetarskyddsstyrelsen (National Board of Occupational Safety and Health), Stockholm, 1979: Metod no 1010: Sampling of total dust and respirable dust (Swedish).
3. Engzell U, Englund A, Westerholm P. Nasal cancer associated with occupational exposure to organic dust. Acta Otolaryngol (Stockh) 1978; 86:436-42.
4. Holmberg K. Airborne moulds in buildings. (In press).
5. MacBeth R. Malignant disease of paranasal sinuses. J Laryngol 1965; 79:592-612.
6. May KR. Multistage liquid impinger. Bact Rev 1966; 30:559-70.
7. Ruppe K. Erkrankungen und Funktionsstörungen am Atemtrakt bei Werktätigen der holzverarbeitenden Industrie. Z Ges Hyg 1973; 19:261-4.
8. Rylander R. Airborne endotoxins in the industrial environment (Swedish). Läkartidningen 1982; 79:3456-8.
9. Rylander R. Airborne micro-organisms in different occupational environments with risk of disease (Swedish). Acta Societas Medicorum Succonoc Hygia (Stockh) 1982; 96:218.
10. Snella MC, Rylander R. Lung cell reactions after inhalation of bacterial lipopolysaccharides. Eur J Respir Dis 1982; 63:550-7.
11. Wildfleur A, Heymer B, Schliefer KH, Haferkamp O. Investigations on the specificity of the Limulus test for detection of endotoxin. Appl Microbiol 1974; 28:867-71.
12. Wilhelmsson B, Lundh B. Nasal epithelium in wood workers in the furniture industry. A histological and cytological study. Acta Otolaryngol (Stockh) 1984; 98:321-34.
13. Wilhelmsson B, Drettner B. Nasal problems in wood furniture workers. A study of symptoms and physiological variables. Acta Otolaryngol (Stockh). 1984; 98:548-55.
14. Wilhelmsson B, Jernudd Y, Ripe E, Holmberg K. Nasal hypersensitivity in wood furniture workers. An allergological and immunological investigation with special reference to mould and wood. Allergy. 1984; 39:586-96.

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