

Respiratory symptoms following brief exposure to Cedar of Lebanon (*Cedra libani*) dust

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Summary

A limited study was carried out on six workers troubled by asthma and rhinitis after exposures to Cedar of Lebanon dust in a joinery works. Though five subjects were atopic they had had no major trouble working with a variety of woods hitherto. Cessation of exposure improved matters insofar as ventilatory capacity was within normal limits but some still noted wheeze and rhinitis periodically. Skin testing with wood extract was unhelpful as was serological testing (no specific precipitins were demonstrated) in elucidating the mechanism of the reaction. It is recommended that where a hazard exists in exposure to an organic dust the environmental hygiene requires attention and the atopic subject merits special monitoring.

Introduction

Woodworkers in their trade may be exposed to dusts derived from a wide range of woods, associated fungal material and spores and from substances used in wood treatment (Fillasier, 1967). Among the effects are respiratory complaints such as cough, wheeze, dyspnoea and tightness and soreness of the chest as well as rhinitis.

At a joinery works where Cedar of Lebanon had been machined on three occasions over the course of 3 months, six male workers were troubled by chest tightness, cough and dyspnoea after short exposures. A seventh worker, an asthmatic intolerant of Western Red Cedar, tolerated exposure to Cedar of Lebanon. The works was a specialist joinery works making wooden structures according to architects' special requirements, accustomed to handling a variety of exotic woods and equipped with ventilated woodworking machinery. The timber used had been grown in this country and delivered to the works as clean planks, untreated chemically and kiln dried. As a consequence of the drying process machining produced a quantity of fine floury dust that escaped the exhaust system and was identifiable by its pleasant pungent aroma.

Investigation

All subjects were examined in August 1970 when Cedar of Lebanon wood had not

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been worked for over a week. A history was taken, spirometry carried out (McDermott dry spirometer), prick testing done (control, grass pollens, *D. farinae*, *A. fumigatus* and Cedar of Lebanon) and blood taken from five subjects for study of serological effects of exposure. Skin extract for *D. farinae* was prepared in the Department of Clinical Immunology, Institute of Diseases of the Chest, the others were Bencard preparations. A positive prick test was taken as a weal of 1 mm diameter or larger, or a weal greater than that which might have been produced by the control. Prediction of $FEV_{1.0}$ was made using the formula $[Ht \text{ metres} \times 3.62 - \text{Age} \times 0.031] - 1.41$ litres (Cotes, 1965).

Case reports

(1) C.R., aged 48, had worked as a joiner since leaving school at age of 14 and had hitherto handled all woods with impunity except for *Mansonia* which provoked sneezing. There was no personal history of asthma, hay fever or eczema nor in parents or sibs. He had worked with Cedar of Lebanon for 1 day on the first occasion it appeared in the factory. During the working day he had no trouble but 4 hr after work sitting in a chair at home his chest felt 'rasping' and he was so short of breath that he was reluctant to talk. Overnight the symptoms cleared and he was not re-exposed until 2 weeks later when again in the evening of the first day of re-exposure the symptoms returned. Despite this he continued working with the wood for a further 2 days, the symptoms persisting all the time, severe enough to produce aversion to smoking. A week before examination he was exposed for a further 3 days during which he described constant chest soreness, dyspnoea and a productive cough with clear sputum. Symptomatic improvement did not occur until 2 days after cessation of this exposure.

Prick tests	Grass pollen	2.5%	- ve
	<i>A. fumigatus</i>	5%	- ve
	<i>D. farinae</i>	1 mg/ml	+ ve
	Cedar of Lebanon	5%	+ ve

($FEV_1 = 3.88$ l. [Predicted 3.5 l.]; $FVC = 4.73$ l.; $FEV_1/FVC = 82\%$).

(2) W.A., aged 54, had worked as a joiner since the age of 14 and had handled all woods with impunity apart from irritation of the eyes 20 years ago when working with walnut. No personal or family history of asthma, hay fever or eczema. One month before examination he first handled Cedar of Lebanon. After 2 hr work his chest felt tight but he carried on working for a second day, then going on holiday. On the day following his exposure symptoms slowly improved. A week previous to examination he again worked with Cedar of Lebanon for 2 days and gradually noticed tightness of the chest developing together with obstruction and discharge from the nose. On the day prior to examination his family doctor had given him nose drops and tablets (which might conceivably have interfered with skin testing).

Prick tests	Grass pollen	2.5%	- ve
	<i>A. fumigatus</i>	5%	- ve
	<i>D. farinae</i>	1 mg/ml	+ ve
	Cedar of Lebanon	5%	- ve

($FEV_1 = 2.85$ l. [Predicted 2.78 l.]; $FVC = 3.78$ l.; $FEV_1/FVC = 75\%$).

(3) H.E., aged 43, had been a woodworker since the age of 14 and had hitherto worked with many woods including Cedar of Lebanon without adverse effect. He gave a history of one episode of hay fever 15 years previously and complained of 'chestiness' attributed to smoking up to thirty cigarettes a day. He had no family history of asthma, hay fever or eczema. During the second run of Cedar of Lebanon working he was in the environs of the woodworking machinery for 2 days. The first day of work was unremarkable but he awoke the following morning with a tight chest and a severe unproductive cough. He continued to work the second day despite continuing symptoms. Symptoms slowly eased 2-3 days after the job ceased. A week prior to examination, though there had been a run of work, he had been little exposed and was unaffected.

<i>Prick tests</i>	Grass pollen	2.5%	-ve
	<i>A. fumigatus</i>	5%	-ve
	<i>D. farinae</i>	1 mg/ml	-ve
	Cedar of Lebanon	5%	-ve

(Absent for ventilatory function tests).

(4) H.F., aged 18, had worked as a joinery apprentice for 3 years uneventfully and had no personal or family history of asthma, eczema or hay fever. He first handled Cedar of Lebanon 2 months prior to examination when he worked on a planing machine for 3 days without adverse effect. On the following day he started sneezing and noticed nasal obstruction. Two weeks later while on holiday he felt tightchested and was short of breath when swimming. Subsequently he has sneezed, had a blocked nose and had tightness of the chest. Since the planing episode he had not handled the wood and had been able to pass through an area where it is handled without exacerbation of his condition.

<i>Prick tests</i>	Grass pollen	2.5%	+ve
	<i>A. fumigatus</i>	5%	-ve
	<i>D. farinae</i>	10 mg/ml	-ve
	Cedar of Lebanon	5%	-ve

(FEV₁ = 3.75 l. [Predicted 3.76 l.]; FVC = 4.20 l.; FEV₁/FVC = 89%).

(5) J.P., aged 24, had been a woodworker for 9 years with no adverse effect from wood previously though he had a history of 'hay fever'. There was a family history of one sib having 'hay fever'. At the time that Cedar of Lebanon was first being worked in the factory he worked in the environs without trouble until the evening of the second day when he developed a tight chest, severe cough and produced a lot of clear mucus. Later that night he had severe dyspnoea. He was off work for 2 weeks and was investigated at a chest clinic. Since returning to work he had worn a mask when handling the wood but merely walking through a workshop where Cedar of Lebanon had been sanded, without a mask, started him wheezing. At the time of examination he thought that he had recovered apart from periodic wheezing.

<i>Prick tests</i>	Control	+ve	(1 mm)
	Grass pollen	2.5%	-ve
	<i>A. fumigatus</i>	5%	-ve
	<i>D. farinae</i>	10 ml/ml	+ve (2 mm)
	Cedar of Lebanon	5%	-ve

(FEV₁ = 2.73 l. [Predicted = 3.57 l.]; FVC = 3.42 l.; FEV₁/FVC = 80%).

(6) S.T., aged 23, was a truck driver who assisted with wood machining in the factory. Previously he had worked as a driver, paint sprayer and degreasing tank operator without trouble. His history included hay fever, frequent sore throats, and 'colds' frequently going to his chest. There was no family history of atopy. On the second occasion that Cedar of Lebanon was worked he spent a day on the sanding machine without incident. He awoke, however, the next morning dyspnoeic, with a tight chest and was coughing severely enough to retch producing small quantities of yellow sputum. He was off work for 3 days. On a subsequent occasion he had worn a mask while holding timber being machined and had had no further trouble.

Prick tests	Grass pollen	2.5%	+ ve
	<i>A. fumigatus</i>	5%	- ve
	<i>D. farinae</i> 10 mg/ml		+ ve
	Cedar of Lebanon	5%	- ve

(FEV₁ = 3.82 l. [Predicted 4.03 l.]; FVC = 4.93 l.; FEV₁/FVC = 77%).

There was a further worker, H.S., who had worked with wood for 22 years. His history included summer-time hay fever, and asthma for 10 years following 1 month's heavy exposure to Red Cedar (*Thuja plicata*). There was a strong family history of atopy. He had not been troubled by Cedar of Lebanon though exposed on three occasions for a total of 6 days. Prick tests were positive for grass pollens and negative for Red Cedar and Cedar of Lebanon.

Serological examinations

No specific precipitins were demonstrated in the five subjects examined with standard extract of wood dust (Coca solution) and antigen-antibody precipitation tests using 1.5% ion agar double diffusion technique carried out in five of the subjects studied. One was not available for serological examination as he was being investigated for an unrelated complaint.

Discussion

It has been suspected or confirmed in several instances of pulmonary disease in workers in contact with timber that fungal contamination is the important allergen (e.g. Cohen *et al.*, 1967—Sequoia; Emanuel, Lawton & Wenzel, 1962—Maple Bark). In this instance the timber being machined was clean, had not been chemically treated, but had been kiln dried, a process that removed outer contamination from the timber and infection by fungus.

The pattern of asthma coming on initially after cessation of work and subsequently occurring at work presented in five subjects: a sixth (W.A.) experienced an attack at work shortly after commencing exposure. In the experience of the author these patterns are not uncommon in occupational asthma arising in association with such diverse materials as isocyanates, thermal breakdown products of polyurethane, aromatic amines mixed with chloraniline and proteolytic enzyme dusts.

Sosman *et al.* (1969) in their study of four patients sensitized to wood subjected them to provocation with alcoholic extracts of wood. They described an immediate reaction with quick recovery in the 'oak' sensitized subject, delayed reactions with

delayed recovery in the two 'cedar' sensitized subjects and a combined response in the 'mahogany' sensitive subject.

Whether the principal complaint was of cough or wheezing dyspnoea varied from subject to subject, and for an individual varied from time to time, much as happens with asthma not occupationally determined (extrinsic and intrinsic).

When subjects were examined Cedar of Lebanon was not in use and they were free of symptoms: tests of ventilatory function show no gross deficit.

Defining an 'atopic' subject as one showing an immediate skin reaction to one or more common allergens on prick testing (restricting reagents to grass pollens, *D. farinae* and *A. fumigatus*), then all the subjects save H.E. would classify as atopic. Only one subject, P.F., had a convincing family and personal clinical history of atopy.

Of special interest clinically and theoretically is the case of H.F. who had no clinical or family history of atopy though on prick test he was positive to grass pollens, where complaints of paroxysmal chest tightness and rhinitis have persisted despite cessation of exposure to Cedar of Lebanon. Similarly in the case of J.P., an atopic subject sensitive to *D. farinae*, with a personal and family history of hay fever, periodic wheezing has persisted after cessation of exposure. While it would have been of interest to study the effects of bronchial provocation with wood extracts, testing was not carried out.

Skin sensitivity could only be demonstrated in one subject (C.R.) who had a small immediate reaction and it was not possible to demonstrate circulating precipitins in any of the subjects. The history of respiratory symptoms strongly suggests an allergic phenomenon but the precise mechanism in this case must remain undeciphered. Assuming an allergic process, caution must be observed before concluding that Cedar of Lebanon has greater potency than other woods on the basis of the short time of exposure to it that preceeded symptoms as no information is available as to the relative doses of various wood dusts inhaled.

Sosman *et al.* (1969) found their patients to be skin-test negative for common allergens and the specific wood extracts and detected circulating precipitating antibody to the wood responsible for the adverse response. They were confident that there was an immunological basis for the phenomena they observed but were unable to explain the exact mechanism.

The susceptibility of the 'atopic' subject to develop immediate Type I (Anaphylactic) reactions is recognized (Pepys, 1969) but whether the atopic subject is hypersusceptible to Cedar of Lebanon dust (the precise mechanism of intolerance to which is unknown) cannot be determined from this study. However, it would be a wise precaution in the present state of knowledge to give special consideration to atopic subjects before exposing or continuing to expose them to sensitizing hazards such as hard wood dust even where the precise allergic mechanism is in doubt. Regular medical surveillance of both atopic and non-atopic workers should be considered when the hazard is high as for say, *B. subtilis* derivative (Greenberg, Milne & Watt, 1970).

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