

occupational and environmental lung disease

Occupational Asthma Caused by Oak Wood Dust*

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Three cases of occupational asthma caused by oak wood dust are presented. The diagnosis was confirmed by specific inhalation challenges, which induced a dual asthmatic reaction in two subjects and an atypical prolonged immediate reaction in one subject.

PC20=provocative concentration of methacholine causing a 20% change in FEV₁; PEFr=peak expiratory flow rate

A variety of wood species have been documented as causing work-related asthma.¹ To our knowledge, only one case of occupational asthma due to oak (*Quercus robur*) wood dust has been reported.² We describe specific asthmatic reactions to this wood species that developed in three workers.

CASE REPORTS

Case 1

A 43-year-old patient had been employed for 8 years for a company that produced mobile homes. At work, he was exposed to oak (principally), spruce, and jack pine wood dust daily. He was also exposed to melamine powder containing formaldehyde. He had started reporting symptoms of dyspnea, chest tightness, wheezing, and a productive cough 5 years previously. Four months before being referred, his symptoms got worse and he had to use an inhaled β_2 -adrenergic agent up to ten times per day. During the summer and winter holidays, he gradually improved and his asthma disappeared over a 5- to 7-day period. When he was assessed, besides inhaled β_2 -adrenergic agent, he also took a sustained-release theophylline derivative and inhaled beclomethasone (2,000 μ g/d). He reported having used short courses of oral steroids on four occasions in the previous 3 years. He had no medical history of atopy and had never smoked regularly. Skin testing done by the prick method with 15 common inhalants showed a positive reaction to mixed tree pollens only. Tests were negative with an extract of oak wood dust. Monitoring of peak expiratory flow rates (PEFRs) at work showed marked daily fluctuations with values which were down to a minimum value of 210 L/min when he experienced nocturnal awakenings due to asthma. These values improved progressively to a maximum value of 640 L/min 10 days after being away from work. The provocative concentration of methacholine causing a 20% change in FEV₁ (PC20) was 0.2 mg/mL with a baseline FEV₁ of 3.1 L or

82% predicted³ and the FEV₁-forced vital capacity ratio was 50% (predicted of 80%)³ when the subject was at work.

Specific inhalation challenges were carried out in the following way. Monitoring of FEV₁ during a day spent in the hospital laboratory did not show significant changes. The subject was returned to work with serial monitoring of PEFr. After 5 days spent at work, the subject started having asthmatic symptoms, requiring his inhaled β_2 -adrenergic agent, and PEFr values were down to a minimum value of 350L/min. The next day, a technician was sent to the workplace. Serial monitoring of FEV₁ at work under supervision showed a reduction of 25% 8 h after starting work. Consultation of the safety data sheet revealed that the subject was exposed to melamine powder which contained formaldehyde. Exposing the subject to formaldehyde in the hospital laboratory challenge room (the subject had to stay in the cubicle at a distance of 30 cm from a small recipient containing 100 mL of formaldehyde liquid) showed no significant changes in FEV₁ up to 8 h after the exposure ended. It was decided to expose the individual to the wood dusts used at work. Asking the subject to saw boards of oak, jack pine, and spruce for 3 h induced an immediate reaction (maximum fall in FEV₁ of 21%) followed by a late reaction (maximum fall in FEV₁ of 18% 7 h after ending exposure). Two days later, the subject was exposed specifically to oak wood dust, first generated by an aerosolization device during 2 h⁴ and then by asking the subject to tip the dust from one tray to another for 20 min.⁵ A maximum fall in FEV₁ of 20% was recorded 20 mins after the end of exposure with functional recovery and the appearance of a late reaction (maximum fall in FEV₁ of 32% 7 h after exposure ended [Fig 1]). Two weeks later, exposing the subject to wood dust of local species (spruce, balsam fir, and jack pine) for the same interval and in the same way as for oak wood dust did not cause any significant changes in FEV₁. To further confirm the specificity of the reaction, a control asthmatic subject (baseline PC20=0.5 mg/mL) was exposed to oak wood dust in the same manner and for the same interval as for subject 1. No significant changes (<10%) in FEV₁ were documented in the minutes and hours following exposure.

Case 2

A 50-year-old man had been employed for 24 years as a coffin maker. He reported a 10-year history of chest tightness, cough, and wheezing related to workplace exposure. He was exposed to oak and obeche (*Triplochiton scheroxylon*). Despite treatment with inhaled β_2 -adrenergic agent and beclomethasone (2,000 mg/d), his asth-

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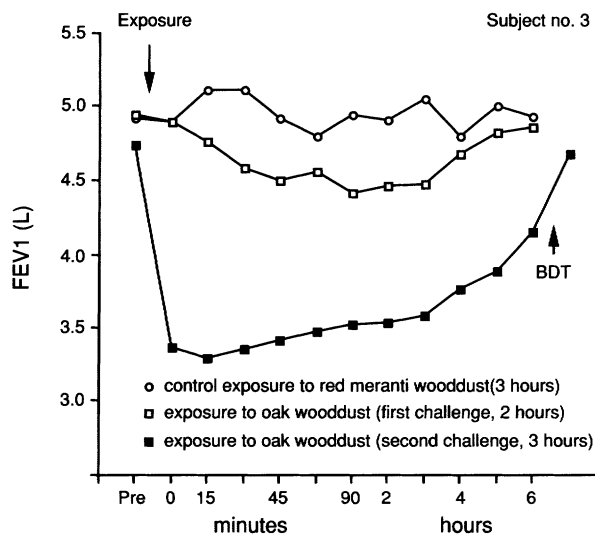
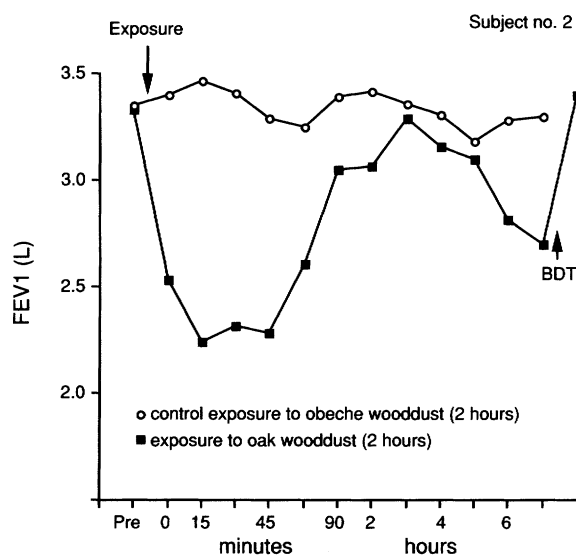
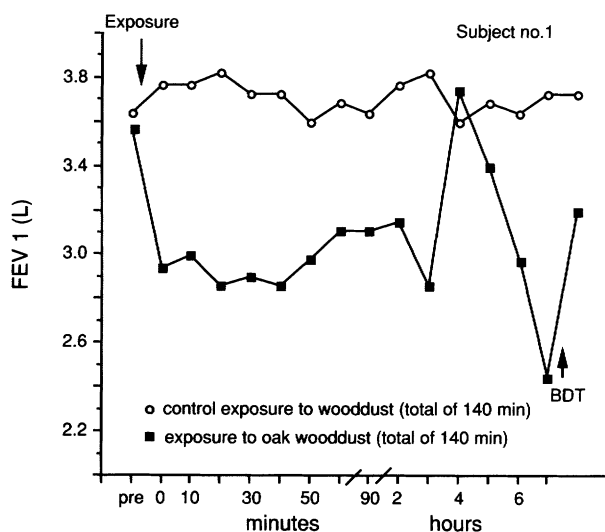


FIGURE 1. Results of inhalation challenges for the three subjects. BDT, inhaled bronchodilator (salbutamol, 200 µg).

matic condition progressively worsened and he had to use several courses of oral steroids a year. He reported no medical history of atopy and had never smoked. Skin prick tests were negative for common inhalant allergens as well as for an extract of oak dust. Specific inhalation challenges were carried out in the laboratory while the subject was away from work for 3 weeks. Baseline spirometry showed a forced vital capacity of 5.57 L (122% predicted) and an FEV₁ of 3.25 L (92% predicted). A control test with exposure to fir dust for 1 h by asking the subject to tip the dust from one tray to another⁵ did not show significant changes in FEV₁. Baseline histamine PC₂₀ assessed at the end of the control day was 0.44 mg/mL. On the next day, exposure to oak dust for a total of 2 h resulted in an immediate reaction (maximum fall in FEV₁ of 33%) followed by a late response (maximum fall of 19% at 6 h postexposure [Fig 1]). On the next morning, nonspecific bronchial hyperresponsiveness was significantly increased (histamine PC₂₀=0.04 mg/mL) as compared with the control day value. One month later, challenging the subject with obeche for 2 h did not elicit significant changes in FEV₁ (Fig 1).

Case 3

A 36-year-old man had been working for 20 years as a carpenter in a company manufacturing doors and window frames. Three years before being seen, he started experiencing rhinorrhea, sneezing, chest tightness, wheezing, and cough at work. He used principally oak and red meranti (*Shorea* species), but his symptomatology was specifically related to oak dust exposure. The subject took an inhaled bronchodilator when necessary, and he was symptom-free on weekends and holidays. Eight months before evaluation, he was relocated outside of the workshop. There, he was only occasionally exposed to oak, and his symptoms almost completely subsided. He had no medical history of atopy and had never smoked. Skin prick tests gave a positive reaction to *Dermatophagoides pteronyssinus* and a negative reaction to an extract of oak dust. Specific inhalation challenges were performed according to the same protocol as that used for subject 2.⁵ Results are shown in Figure 1. Baseline spirometry showed a forced vital capacity of 6.57 L (121% predicted) and an FEV₁ of 4.92 L (110% predicted). Exposure to fir wood dust for 1 h on a control day did not elicit significant changes in FEV₁. Baseline histamine PC₂₀ was 4.57 mg/mL at the end of the control day. The next day, the subject was exposed to oak wood dust, which did not induce significant changes in FEV₁ (maximal fall in FEV₁ of 10%). Reassessment of histamine PC₂₀ at 6 h postexposure however, revealed a significant increase in nonspecific bronchial responsiveness (PC₂₀ of 0.31 mg/mL) as compared with baseline value. The next day, a 3-h exposure to oak dust was followed by a 30% fall in FEV₁ with a progressive recovery over the next hours. On the next day, exposing the subject to red meranti for 3 h did not cause a bronchial response.

DISCUSSION

In this study, work-related asthma was first documented in case 1 through serial monitoring of PEFR and recording FEV₁ under the supervision of a technician at work. These procedures could not be used with the remaining two subjects because we were not allowed to send them back to their workplace. In all three subjects, the diagnosis of occupational asthma due to oak wood dust was demonstrated using specific inhalation challenges in the laboratory which induced a dual asthmatic reaction in two subjects and an atypical prolonged immediate reaction in one subject. The specificity of these reactions was supported by the fact that exposure to other wood species present at the

workplace did not cause significant changes in FEV₁.

There have been reports of asthma caused by various wood dusts, among which western red cedar asthma has been the most extensively studied.¹ To our knowledge, only one case of occupational asthma due to oak wood dust has been so far described.² In this case, specific inhalation challenges elicited an isolated immediate reaction. The authors postulated that the reaction was due to a hypersensitivity mechanism, since precipitating antibodies to an alcohol extract of oak were shown. In our cases, specific inhalation challenges provided convincing evidence that bronchial responses to oak wood dust were not due to a nonspecific irritant effect. Indeed, two of the cases (cases 1 and 2) developed dual reactions which are more consistent with a specific "sensitizing" (not necessarily IgE-mediated) mechanism. Such reactions have been consistently described after exposure to common inhalants and occupational agents and much more inconsistently after exercise or exposure to non-specific agents such as hypotonic and hypertonic solutions. Furthermore, the dual reaction in case 2 was associated with a significant increase in nonspecific bronchial hyperresponsiveness to histamine. Such changes in nonspecific bronchial responsiveness have been documented after asthmatic reactions caused by common inhalant allergens and various occupational sensitizing agents.⁶ In case 3, the increase in nonspecific bronchial responsiveness even preceded the occurrence of an asthmatic reaction, as previously reported in one subject challenged with red cedar.⁷ Skin prick tests with oak dust extract were negative. This finding is in agreement with most studies which failed to detect specific IgE, probably because the responsible agents are low molecular weight compounds acting as haptens. Wood dusts contain various low molecular weight acids, the most important one being abietic acid. Even when specific IgE antibodies had been identified, *eg*, to the complex (plicatic acid-human se-

rum albumin) in the case of plicatic acid contained in red cedar, its relevance to human disease seems questionable.⁸ Other immunologic (T-lymphocyte mediated) and nonimmunologic (activation of complement, generation of anaphylatoxins) pathogenic mechanisms can therefore be incriminated.⁹

Considering the large number of workers employed in the wood industry throughout the world, the prevalence and pathophysiologic mechanisms of occupational asthma caused by wood dusts, including oak, warrant further investigation.

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