Occupational asthma caused by exposure to ash wood dust (Fraxinus americana)

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ABSTRACT: A 63 year old man reported rhinitis and asthma, which occurred only at work where he was exposed to ash wood dust. Monitoring of peak expiratory flow rates (PEFR) and bronchial responsiveness to histamine when off work and at work showed increased variation of PEFR at work but no significant changes in nonspecific bronchial responsiveness assessed by the provocation concentration producing a 20% fall in FEV₁ (PC₂₀). Specific inhalation challenges were carried out in a special challenge room with ash wood dust. During the exposure for only 3 minutes, the mean concentration of particles was 3 mg·m⁻³ and about 50% of particles had a diameter <10 μ. An immediate bronchospastic reaction was documented. Antibodies to a human serum albumin (HSA) ash wood conjugate were not significantly increased.


Many types of wood dust have been reported as causing occupational asthma [1]. Here we present the case of an individual who developed occupational asthma due to ash wood dust.

Case report

A 63 year old man started to work for a furniture company 20 yrs before consulting. His work consisted of assembling the furniture parts in the same building as various other processes such as woodcutting and sandblasting. The most commonly used wood in this company had always been ash. Seven years before consulting, he started to report coughing, wheezing, and shortness of breath which at the time were not related to work. These symptoms lasted for a year and a half and then, for some unknown reason, disappeared completely. Three years before his visit, he reported rhinitis and asthma which occurred only at work. When he was away from work, he was almost completely asymptomatic after 10 days. Return to work resulted in progressive asthma symptoms, with nocturnal awakenings. His symptomatology steadily increased afterwards and did not improve at Weekends.

He was seen after being away from work for three weeks. Skin tests, performed by the prick method with a battery of 15 common allergens (Bencard Allergy Service, Toronto, Ontario; Hollister-Stier, Mississauga, Ontario) including tree and ash tree pollens, were negative. Combined monitoring of peak expiratory flow rates (PEFR) and bronchial responsiveness to histamine [1, 2] when off work and at work was carried out. This showed increased variations of PEFR at work as compared with periods off work, but no significant changes in the provocative concentration of histamine causing a 20% fall in forced expiratory volume in one second (FEV₁) (PC₂₀) (fig. 1).

Specific inhalation challenges using ash wood dust were carried out after three days away from work. Baseline spirometry showed an FEV₁ forced vital capacity of 2.90 /4.09 l (71.0%) as compared with predicted values [3] of 3.10 /3.73 l (80%) thus showing mild airway obstruction. The device for nebulization of sawdust has been thoroughly described elsewhere [4].

A control day of non-exposure showed a maximum fluctuation of 10% in FEV₁ six hours after beginning the monitoring. Control exposure to wood dusts was carried out on the following day. These wood dusts originated from black spruce (Picea mariana) (90%), balsam fir (Abies balsamea) (5%) and jack pine (Pinus banksiana) (5%). They are commonly found in Eastern Canada and have not been incriminated in causing occupational asthma. Exposure for 30 min did not cause any significant changes in FEV₁ in the minutes (maximum change of 9% 10 min after the end of exposure) and hours (maximum fall of 14% at 7 h) following the challenge (fig. 2). PC₂₀ was 1.25 mg·ml⁻¹ 8 h after the control exposure, thus showing moderate bronchial hyperresponsiveness.

Exposure to wood dust of ash on the next day for a total of 3 min resulted in a 22% fall in FEV₁, which was maximal 10 min after the end of challenge with progressive recovery in the first hour (fig. 2). There was a subsequent fall of 16% in FEV₁ at 6 h after the challenge. However, this fall did not appear significantly...
more pronounced than on the control day. PC_{20} at the end of the day was unchanged at 1.4 mg·m^{-3}. Mean concentrations of wood dusts were 3 mg·m^{-3} on both exposures. 50.3 and 57.5% of particles were <10 μm in the case of control and ash wood dusts, respectively, allowing penetration into the bronchial tree.

Monitoring of PEFR after exposure to the control and active wood dusts, until going to bed on the following evening, did not reveal significant fluctuations (≥10%). Antibodies to a human serum albumin (HSA)-ash wood conjugate [5] were not significantly increased (radioallergosorbent test (RAST) ratio value of 0.9, comparing the uptake of radioactivity of patient’s serum to the uptake of pooled serum from normal unexposed subjects).

**Discussion**

Wood dusts originating from several species have been incriminated as causing occupational asthma [1]. To our knowledge, only one article reports that ash (Fraxinus) wood dust could be incriminated [6].

Confirmation of the diagnosis in our subject was made in two different ways. Firstly, even if no significant changes in bronchial responsiveness were demonstrated after a period at work, there were significant fluctuations in peak expiratory flow rates. The absence of changes in bronchial responsiveness could be attributed to the fact that an isolated immediate bronchospastic reaction was documented. These reactions do not usually cause an enhancement in bronchial responsiveness [7]. Secondly, specific inhalation challenges in the laboratory exposing our subject to ash wood dust caused an immediate bronchoconstriction. In carrying out these tests, we dissociated the possible irritating effect of wood dust by comparing the reaction with an exposure to another wood dust, for an even longer interval. Using a special device for nebulization described elsewhere [4], we also showed that the concentration and diameter of inhaled particles was comparable for the control and active wood dusts and was below the TLV-STEL value for wood dust of 10 mg·m^{-3}. Even if higher concentrations had been used, it is known that exposure of asthmatic subjects...
concentrations of wood dusts close or above the TLV-STE L value do not enhance bronchial hyperrespon siveness [4].

Our subject did not have increased specific IgE antibodies to ash wood dust. Demonstration of specific IgE antibodies has been made in some cases of occupational asthma due to wood dust but, in most cases, the mechanism has not been elicited.

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References


Asthme professionnel par exposition à la poussière de bris de frêne. J.L. Malo, A. Cartier.

RÉSUMÉ: Un homme âgé de 63 ans, présentant une rhinite et un asthme survenant sur les lieux du travail lors d'expositions à la poussière de bois de frêne. L'enregistrement répété des valeurs du débit de pointe et de l'hyperréactivité bronchique à l'histamine, en période de travail et à distance de périodes de travail, a montré des variations des débits de pointe plus marquées au travail sans toutefois qu'il n'apparaisse de changement significatif de l'hyperréactivité bronchique à l'histamine. Nous avons effectué un test de provocation bronchique spécifique dans une chambre de provocation spéciale en utilisant la poussière de bois de frêne rapportée par le sujet. Durant l'exposition, qui a duré seulement 3 minutes on note une concentration moyenne de particules de 3 mg/m³, dont environ 50% des particules ont un diamètre <10 μ. Le sujet a présenté une réaction bronchospastique immédiate. Nous n'avons pas retrouvé d'augmentation significative des anticorps de type IgE à un conjugué (albumine humaine-poussière de bois de frêne). Eur Respir J., 1989, 2, 385-387.