

Occupational rhinitis and asthma caused by inhalation of *Balfourodendron riedelianum* (Pau Marfim) wood dust

A. BASOMBA, E. BURCHES, A. ALMODOVAR & D. HERNANDEZ F. DE ROJAS

Servicio de Alergia, Hospital Universitario la Fe, Valencia, Spain

The case of a 30-year-old wood-worker, with rhinitis and asthma induced by exposure to the dust of Pau Marfim wood (*Balfourodendron riedelianum*) is reported. Hypersensitivity to this wood was confirmed by positive skin test, bronchial challenge test and RAST. The bronchial response was inhibited by sodium cromoglycate. Unexposed persons did not exhibit reactivity to this wood in any of the tests. When electrophoretic analysis of proteins in polyacrylamide gel was applied to a crude (phosphate-buffered) extract, an homogeneous distribution of low MW material was detected, with no clearly defined bands, suggesting the presence of filamentous type proteins. To the best of the author's knowledge, this is the first reported case of occupational rhinitis and asthma due to Pau Marfim wood dust. A type I (IgE-mediated) hypersensitivity mechanism was demonstrated but the non-glomerular nature of the proteins precluded further identification of the antigens involved.

Key words: occupational asthma; Pau Marfim; *Balfourodendron riedelianum*; wood dust allergy.

Accepted for publication 25 November 1990

The use of wood in the building industry, the manufacture of furniture and veneer production, etc., continues to increase. The total world timber demand is about 2,500 million m³, and it is forecasted that by the year 2000, the demand will be 4-5,000 million m³. Most of the wood used for industrial purposes comes from the Amazonian region, Central Africa (particularly Zaire), and South-East Asia (8).

Clinical conditions caused by contact with or inhalation of different types of wood dust have been recognized for some time. The manifestations of such conditions are of a toxic, irritant or allergic nature.

The particles of wood dust not only cause irritation of the nasal, ocular and bronchial tissues but can also reach the distal parts of the respiratory tract; they are also a relatively frequent cause of skin lesions, headache, nausea, vomiting, etc.

Observations have been made on the association between clinical conditions and many dif-

ferent types of wood dust, such as oak (9), mahogany (9), cedar of Lebanon (6), African maple (7), and red cedar (2), the wood dust of the latter being the most studied (4). However, there are between 60 and 70 different types of wood dust capable of provoking symptoms by contact or by inhalation (8).

In this study, a case of occupational allergic rhinitis and bronchial asthma caused by hypersensitivity to Pau Marfim wood dust (*Balfourodendron riedelianum*) is described for the first time.

PATIENT AND METHODS

The patient is a 30-year-old man who has been working as a carpenter for the past 5 years. At the time of consultation he presented symptoms of rhinitis and asthma, which had appeared 9 months before. He had no history of previous respiratory symptoms. According to the patient, the symptoms were related to the inhalation of a

wood dust, commonly referred to as Pau Marfim, which he had been working with for 2 years. Such symptoms as sneezing, nasal hydnorrhea and nasal pruritus would appear on Mondays and subside at weekends and on his days off.

He was initially treated with bronchodilators and corticosteroids. As, however there was a progressive deterioration in his clinical condition, treatment with oral theophylline and SCG, salbutamol and inhaled beclomethasone was introduced. In addition, the patient was advised to wear a mask when at work. Despite these measures, the symptoms did not subside, although there was a slight decrease in severity.

The clinical picture of rhinitis and bronchial symptoms only disappeared when the patient changed his job. Subsequently, during a 1 year observation period, the symptoms did not reccur.

At the time of the initial consultation, pulmonary auscultation revealed rhonchi and wheezes in both lung fields. A pattern of obstruction, reversible with salbutamol, was revealed by spirometry. Routine investigation and examination of sputum revealed nothing of relevance. Chest X-rays were normal.

The serum IgE level was 217 IU/ml and skin prick tests with common inhalant extracts were negative. A bronchial provocation test with toluene diisocyanate was negative (this substance was used in the patient's workplace).

Only one other of the five exposed subjects working in the same firm, with poor ventilation conditions, manifested symptoms of rhinitis related to Pau Marfim dust. This subject could not be studied.

Extract for in vivo RAST and immunoprecipitation tests

Pau Marfim dust was crushed in Frugoni's liquid (10% w/v). After filtration (millipore filter) and confirmation of sterility, consecutive 10-fold dilutions were used for intracutaneous test and bronchial challenge. These tests were carried out in the patient and two asthmatic subjects not exposed to Pau Marfim wood dust, who acted as controls.

The bronchial challenge test was carried out when the patient was in optimum baseline condi-

tion, after he had left his job and was without symptomatic treatment. The same test was repeated 2 weeks later, with the previous administration of inhaled cromoglycate (40 mg).

Specific IgE was measured by direct RAST using cyanogen bromide activated discs, coupled with the crude extract of Pau Marfim (1, 3). Immunoprecipitation was carried out according to the standard methodology (Ouchterlony).

Determination of antigenic fractions

A total of 10 g Pau Marfim wood dust was suspended in 100 ml of phosphate buffer (0.018 M, pH 7.8), homogenized, shaken at room temperature, filtered and centrifuged. One ml of the supernatant was treated with acetone (1 ml) and the precipitate thus formed was separated by centrifugation, dissolved in phosphate buffer (0.018 M, pH 7.8), and dialysed.

The protein concentration of the original extract (Micro-Biuret colorimetric method) was found to be 4.88 mg/ml. The concentration of sialic acid (Tiobarbituric colorimetric method) was 195.9 µg/ml.

Polyacrylamide gel (5%) electrophoresis was carried out in a continuous phosphate buffer (0.1 M, 0.1% SDS, pH 7.1) medium. Electrophoresis was performed at a current intensity of 8 mA. Coomassie Blue was used for staining the electrophoretic bands.

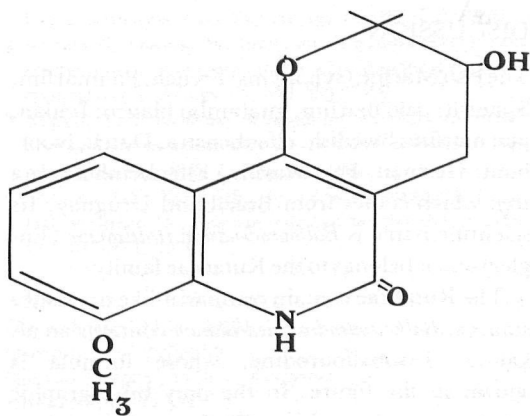


Fig. 1. 4-Isobalfouredine

RESULTS

The *intracutaneous test* with a 1/100 dilution of original extract gave an immediate positive reaction, with a papule of 80 mm². This was not followed by either an Arthus type reaction or a delayed reaction. The test was negative in the two control subjects.

The *bronchial challenge test* was positive in the patient. After 2-min inhalation of a 1/10 dilution of the original extract, there was a > 25 % decrease in FEV₁, with clinical signs of bronchial obstruction. The crisis subsided with inhaled salbutamol and was not followed by a delayed or late reaction. The challenge test was negative when repeated 2 weeks later with the previous administration of SCG. The bronchial provocation test was negative in the two control subjects.

Immunoprecipitation of the patient's and controls' sera was negative.

RAST was positive with the patient's serum (class 3, according to the Pharmacia reference curve: Pharmacia, Sweden), and negative with the sera of the two controls.

Polyacrylamide gel electrophoresis. Proteins with a molecular weight < 9 kilodaltons were detected. Their distribution, and the absence of clearly defined bands (which did not permit determination of antigenic fractions), suggest the presence of filamentous type proteins, such as those found in hair and other specimens, in contrast to the glomerular type of protein with forms clearly defined bands.

DISCUSSION

The Pau Marfim (synonyms: French, Paumarfim; Spanish, palo marfim, guatambu blanco; Italian, pau marfim; Swedish, elfenbensträ; Dutch, ivoorhout; German, Pau Marfim, Elfenbeinholz) is a tree which comes from Brazil and Uruguay. Its scientific name is *Balfourodendrum riedelianum* (Engler) and it belongs to the Rutaceae family.

The Rutaceae contain coumarin-like toxic substances; *Balfourodendron riedelianum* contains an alkaloid, 4-isobalfourodine, whose formula is shown in the figure. In the only bibliographic reference to this subject (5) the toxic reactions

caused by Pau Marfim wood dust have been attributed to this substance.

The patient's symptoms of rhinitis and asthma were related to his work in a poorly ventilated environment. This condition could have contributed to the sensitization. Only one other subject was suffering from rhinitis related to the inhalation of Pau Marfim wood dust, but this case could not be studied. The results of skin and provocation tests and RAST gave confirmation of an IgE dependent hypersensitivity mechanism underlying the pathogenesis of the condition. However, due to the non-glomerular structure of the proteins, the chemical agent responsible for the sensitization and symptomatology, could not be identified.

This is the first confirmed case of occupational allergic rhinitis and asthma related to the inhalation of *Balfourodendron riedelianum* wood dust.

REFERENCES

1. Axen R, Ernback S. Chemical fixation of enzymes to cyanogen activated polysaccharide carriers. *Eur Biochem* 18, 351-360, 1971.
2. Brooks SM. An epidemiologic study of workers exposed to western red cedar and other wood dust. *Chest* 80, 30, 1981.
3. Ceska M, Erikson R, Barga J. Radioimmunosorbent assay of allergens. *J Allergy* 49, 1-9, 1978.
4. Chang-Young M, Vedal, Kus J, Maclean L, Enarson D, Tae K. Symptoms of pulmonary function and bronchial hyperreactivity in western red cedar compared to those in office workers. *Am Rev Respir Dis* 130, 1038, 1984.
5. Freise FW, Smal, Vergiftungsf, 7 C, 1 (1936). In Hausen Bjorn M. (ed): Woods injuries to human health. A manual. Walter de Gruyter, Berlin, 1981.
6. Grechnerg J. Respiratory symptoms following brief exposure to cedar of Lebanon dust. *Clin Allergy* 2, 219, 1982.
7. Hinojosa M, Moneo I, Domínguez J, Delgado E, Losada E, Alcover R. Asthma caused by African Maple (*Triplachiton scleroxylon*) wood dust. *J Allergy Clin Immunol* 74, 782, 1984.
8. Hausen Bjorn M. Woods injuries to human health. A manual. Walter de Gruyter. Berlin, 1981.
9. Sosman AJ, Schlueter DP, Fink JN, Barboriak JJ. Hypersensitivity to wood dust. *N Engl J Med* 281, 977, 1968.

Address:

Dr. A. Basomba
Servicio de Alergia
Hospital La Fe
Avda. Campanar, 21
46009 Valencia
Spain

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.