

Occupational asthma caused by Ipe (*Tabebuia spp*) dust

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Abstract. *Background:* Ipe is a resistant hardwood that contains naphthoquinones. It is easily found and frequently used in South and Central America. Naphthoquinones are skin sensitizers.

Objective: To describe a case of occupational asthma related to Ipe wood dust.

Methods: The patient was submitted to a clinical evaluation consisting of a respiratory symptom questionnaire, occupational history, serial measurements of lung function by spirometry, skin prick tests, patch tests, specific IgE and specific bronchial provocation tests to Ipe dust.

Results: Serial lung function measurements showed sustained regression of obstruction following removal from exposure. Skin prick tests, but not patch tests, were positive to Ipe, and a specific bronchial challenge showed a late asthmatic reaction. Specific IgE search was negative.

Conclusions: Exposure to Ipe wood dust can lead to occupational asthma. The underlying mechanism should be investigated.

Key words: occupational asthma, wood dusts, Ipe wood, *Tabebuia spp*, specific bronchial provocation tests

Introduction

Allergic reactions related to exposure to wood dusts are common. The skin is the most affected organ, followed by the respiratory system. A full spectrum of respiratory diseases associated with occupational and environmental exposures to wood dusts has been previously described [1,2]. Wood dusts contain lignin and cellulose but allergic/inflammatory reactions are probably elicited by chemical constituents of the dust, parasitic fungi and by other chemicals of common use in the wood/furniture industry, as wood preservatives, varnishes and paints.

Until 1996, occupational asthma (OA) due to exposure to wood dusts have been described for 22 different botanic species and 3 other non-classified

woods [3]. A recent Medline search disclosed 3 further reports of wood species causing OA [4-6]. Epidemiological evidences also suggest that pine wood dust exposure is associated with an increased risk of asthma [7] and bronchial hyperresponsiveness [8,9].

Ipe, the common name for the lapacho group of the *Tabebuia* genus, consists of about 20 species of trees and is found in Central and South America [10]. They all contain naphthoquinones. In Brazil, the main species are *Tabebuia ochraceae*, *Tabebuia impetiginosa*, *Tabebuia longifolia* and *Tabebuia serratifolia*. All are known in international trade as Ipe. They are commonly used in construction works for external structures, stairs and parquets. We are describing the case of a wood worker who developed asthma after exposure to Ipe dust.

Case Report

A 52-year old male was referred to our outpatient clinic in September 2000. He was a wood sawyer and trimmer since 1994. In March 2000 he developed symptoms of hoarseness and shortness of breath. Shortly afterwards, he noted wheezing with shortness of breath during afternoons and evenings, of increasing severity needing treatment in emergency rooms with inhaled medications. He felt better on weekends. Four months after the symptoms began he was put on sick benefit, away from the industry. He rapidly improved, needing sporadic inhaled bronchodilators. In August 2000 he was submitted to a direct laryngoscopy that showed supraglottic redness and a vocal cord nodule. He had neither a previous history of respiratory diseases nor atopic symptoms. A former smoker of 18 pack-year, he stopped in 1997.

The symptoms began a few months after he started to saw and trim Ipe wood. Formerly he used to work with pine wood only.

Methods and Results

Results of lung function are shown in Table 1 (Pulmonaire, Jones, Oakbrook, IL, USA).

Occupational-type specific inhalation provocation tests [11] with Ipe dust (45 days after removal from exposure) and Garapa (*Apuleia leiocarpa*) dust were performed one month apart. Garapa is a hardwood from the Leguminosae family that does not contain naphthoquinones [10]. The patient handled freshly sawed dusts in a closed room for 20', tipping dust with two recipients, and disclosed a late asthmatic reaction to Ipe dust only. Results are shown in Figure 1.

Skin prick tests were performed with a set of 7 common inhaled allergens plus negative and histamine

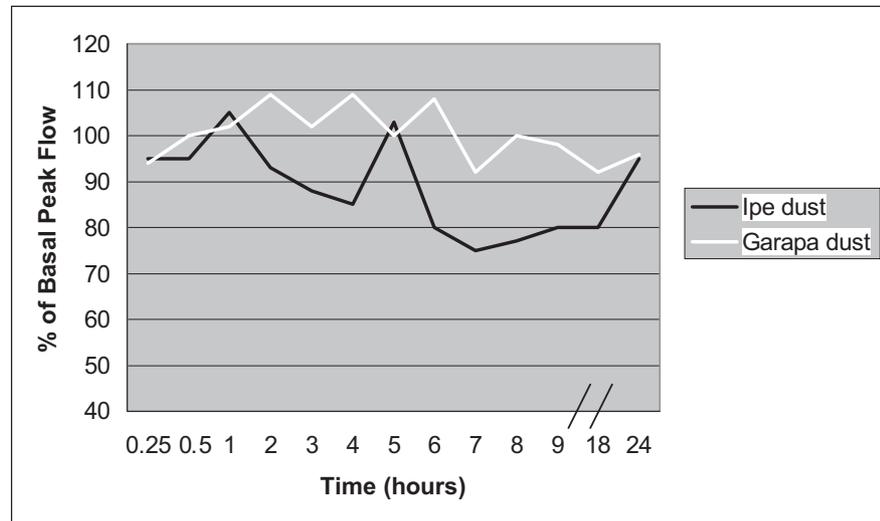


Figure 1. Bronchial challenge test with Ipe and Garapa dust (control)

controls (Alk Abelló, Madrid, Spain) with negative findings. Skin prick tests with weight per volume saline dilutions of freshly sawed Ipe dust revealed wheals of 5mm, similar to histamine, after 60 minutes for 1, 3 and 5% dilutions. Skin patch tests with both saline and alcoholic dilutions were negative after 48 and 96 hours.

For *in vitro* specific IgE measurements, an extract from *Tabebuia spp* dust was prepared in phosphate buffered 0.1 molar saline dialysed against distilled water and lyophilized. A 2mg/ml protein solution was coupled to activated ImmunoCAP \AA at 10,100 and 1,000ul volumes according to standard procedures. Serum from the patient and a control spiked with IgE to 10,000 kU/L were tested and proved negative.

Discussion

Ipe is a hardwood found in Central and South America, resistant to fungi and other biological aggressions. It is suitable for flooring (parquets), stairs, external areas, and sports goods and also for maritime constructions. *Tabebuia spp* belongs to the Bignoniaceae family. They contain naphthoquinones, namely lapachol,

Table 1. Serial spirometries after removal from exposure.

Days away from exposure	15*	45	70
FVC (l)	2.80	3.40	3.44
FEV ₁ (l)	1.84	2.57	2.64
FEV ₁ /FVC (%)	66	76	76

* Positive response to bronchodilator.

deoxylapachol and lapachenole, all capable of render guinea pigs skin sensitized [12]. Descriptions of human deaths and worm elimination from swallowing Ipe dusts are found in the past (Freise FW, 1936, cited in the Botanical Dermatology Database) [10]. Naphtoquinones also present a strong activity against trypanostigote forms of *Trypanossoma cruzi*, the agent of Chagas disease [13].

The reported case is an example of OA. The clinical and occupational history are plausible, there was a clear time relation with the introduction of Ipe wood in the sawmill, symptom regression when away from exposure, functional diagnosis of asthma and an objective measure of lung function decline after specific challenge. Skin prick tests to common inhaled antigens were negative but weight per volume dilutions of freshly sawed Ipe dusts elicited positive wheals after 1 hour. Although delayed prick test reactions may be associated with Type IV hypersensitivity [14] skin patch tests were negative in the present case. Search for specific IgE in the patient's serum was negative. A recent case report and revision of asthma cases from wood dusts showed that in 5/12 cases specific IgE tests were negative [15]. It should be reminded that blood was collected after 2 years of exposure removal.

This is the first report of OA caused by exposure to Ipe wood dusts. As it is a widely used timber in South and Central America, more studies are needed to identify the precise mechanism of this asthma.

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