

Occupational asthma caused by tali and jatoba wood dusts

To the Editor:

Tali (*Erythrophileum suaveolens*), also called elondo, missanda, or muave, is an African tree belonging to the Leguminosae family. Its hardwood is widely used for flooring, heavy construction, railway cross-ties, and dock work. Exposure to tali wood has been shown to cause allergic contact dermatitis.¹

Jatoba (*Hymenaea courbaril*) is a huge canopy tree, belonging also to the Leguminosae family, indigenous to the Amazon rainforest and parts of tropical Central America. The *Hymenaea* genus comprises two dozen species of tall trees distributed in tropical parts of South

America, Mexico, and Cuba. Because of its hardness, jatoba wood can be used for many things including furniture, tool handles, and flooring.

To the best of our knowledge, these are the first reported cases of asthma caused by tali and jatoba wood dusts.

Case 1 was a 34-year-old man, a nonsmoker. He had worked as a carpenter for 13 years. In the last 10 years, he performed the woodworking in buildings under construction and the fine cutting of wooden floors. During the last 5 years, he started to have sneezing, nasal discharge, ocular itching, and nasal stuffiness as well as dyspnea, dry cough, and wheezing when working with tali wood. Asthma symptoms were more intense at the end of the work shift and at night, but he remained symptom-free during holidays and days off work. Although he also handled pine, ash, makore, and oak wood, he did not notice respiratory symptoms with these woods. The patient had shown a progressive improvement in asthma symptoms by avoiding, as far as possible, the exposure to tali wood. He had a history of asthma in childhood, which completely subsided at the age of 10 years. Physical examination, blood tests, and basal spirometry results were normal.

Case 2 was a 49-year-old man, an ex-smoker. Since he was 20 years old, he worked as a carpenter. In the last 18 years, his job consisted of the installation and fine cutting of hardwood floors, mostly jatoba, tali, chestnut, pine, and oak. About 8 years before consultation, he noticed nasal symptoms upon exposure to tali and jatoba wood dusts. Shortly thereafter, he started to have asthma attacks in the evening and at night when working with these woods. He markedly improved on holidays and days away from work. Physical examination, blood tests, and basal spirometry results were normal.

Tali and jatoba wood dusts were extracted (10% wt/vol) in ammonium bicarbonate (0.125 mol/L, pH 8.2). After 8 hours of extraction at 4°C, the suspension was centrifuged at 10,000 rpm for 30 minutes, the pellet was discarded, and the remaining solution was filtered through a 0.22- μ m pore size membrane and dialyzed against distilled water by tangential ultrafiltration in 3000 Da cut-off membranes (Millipore, Bedford, Mass). Finally, the extracts were lyophilized.

The serum levels of specific IgE against tali and jatoba wood extracts were determined in duplicate by enzyme-linked immunosorbent assay, using a concentration of 20 μ g/mL and serum dilution series starting from 1:2 as described elsewhere.² A result was considered positive when a serum bound 4 times more specific IgE than the mean titer of 5 atopic unexposed controls.²

Serial monitoring of peak expiratory flow was consistent with work-related asthma in both patients. Specific inhalation challenge (SIC) with wood dust was performed in a 7-m³ challenge chamber equipped with vacuum exhaust and high-efficiency particulate air filtration. The wood dust aerosol was generated by asking the patients to tip the wood dust from one tray to another for increasing time periods (1, 5, 15, 30, and 60 minutes) as previously described.³ The patients wore protective clothing and vinyl gloves during the challenge. The con-

TABLE I. Patient characteristics and results of inhalation tests and sputum assessment

	Age/ sex	Atopy	FEV ₁ (% of predicted)	PC ₂₀ met at baseline (mg/mL)	SIC with tali wood extract at 10% w/v (% fall in FEV ₁)	SIC with tali wood dust at 5 mg/m ³ (% fall in FEV ₁)	PC ₂₀ met post-SIC with tali wood dust	Sputum eosinophils (%) at baseline/24 h after tali SIC	SIC with jatoba wood dust at 5 mg/m ³ (% fall in FEV ₁)
Case 1	34/M	yes	95	4.54	LAR (25)	LAR (20)	0.30	0/30	ND
Case 2	49/M	no	107	0.53	LAR (19)	DAR (21)	0.21	8/42	EAR (20)

SIC, Specific inhalation challenge (maximum percent fall in FEV₁); M, male; ND, not done; PC₂₀ met, PC₂₀ methacholine; EAR, early asthmatic reaction; LAR, late asthmatic reaction; DAR, dual asthmatic reaction.

centrations of total dust particles during the challenges were measured with an aerosol monitor (DustTrack model 8520, TSI, St Paul, Minn). A maximum concentration of 5 mg/m³ was not exceeded. On a control day, the subjects were challenged with pine wood dust to ensure that fluctuations of FEV₁ were <10%. SIC was also performed after the tidal breathing method⁴ with progressive 2-fold dilutions of the aqueous tali wood extract up a maximum concentration of 10% wt/vol. Methacholine inhalation testing was carried out at baseline and 24 hours after SIC with tali wood dust. Two unexposed asthmatic patients were also challenged with tali and jatoba wood dusts as control subjects.

Skin prick tests with common inhalant allergens were positive to grass and Parietaria pollen in patient 1 and negative in patient 2. Skin prick tests with tali and jatoba wood extracts as well as with a panel of common wood extracts (Bencard, Worthing, United Kingdom), including obeche wood (Leti CBF, Tres Cantos, Madrid, Spain), were all negative. Determinations of specific IgE to tali and jatoba extracts also yielded negative results. SIC performed both with the dust and an aqueous extract from tali wood elicited asthmatic reactions in each patient. Patient 1 had a late asthmatic reaction on SIC with tali wood dust on two different occasions at a concentration of 5 mg/m³ for 30 minutes and 1 mg/m³ for 120 minutes, respectively. Patient 2 had a dual asthmatic reaction on challenge with tali wood dust at 5 mg/m³ for 10 minutes and an early asthmatic reaction with jatoba wood dust at 5 mg/m³ for 5 minutes (Table I). Both patients had rhinitis symptoms during the challenge with tali wood dust.

Induced sputum differential cell count was assessed before and 24 hours after SIC with tali wood dust (5 mg/m³), showing a marked increase in the percentage of eosinophils after the challenge (Table I). In patient 1, the differential cell count before/24 hours after the SIC was bronchial epithelial cells 5%/10%, macrophages 6%/30%, neutrophils 30%/30%, and eosinophils 0%/30%; in patient 2: bronchial epithelial cells 1%/0%, macrophages 59%/30%, neutrophils 32%/28%, and eosinophils 8%/42%.

Asthma, nonasthmatic airflow obstruction, and both upper and lower respiratory symptoms have been associated with exposure to several types of soft and hard wood dusts. In the patients herein reported, the diagnosis of wood-induced asthma was based on a combination of the clinical history and objective evidence that exposure to wood dust from tali, jatoba, or both elicited acute respiratory symp-

toms and lung function changes. In addition, the SIC with tali wood induced an eosinophilic inflammatory reaction in the airways. The capability of these wood dusts to provoke variable airflow limitation, to heighten airway hyperresponsiveness, and to induce airway inflammation strongly suggest that tali and jatoba wood dusts are true "inducers" of occupational asthma and not mere "inciters."⁵

Both woodworkers, however, had negative skin prick test responses to tali and jatoba wood, and no specific IgE antibodies against these wood extracts were detected. The pathophysiologic mechanism in asthma caused by some wood dusts appears to be IgE-dependent,^{6,7} whereas other types of wood dust, such as Western red cedar, induce asthma through IgE-independent mechanisms. Examining specific IgE antibodies in this condition is most often unproductive.⁸

Inhalation challenge with plicatic acid, the main non-volatile compound of Western red cedar, and an aqueous extract of this wood induce asthmatic reactions in patients with cedar asthma.⁸ Tali and jatoba wood have a high content of chemical extractives (eg, volatile monoterpene and sesquiterpene phytochemicals and non-volatile diterpene chemicals), which might be involved in the pathogenesis of asthma induced by these woods. As for cedar wood dust, the cause is probably a low-molecular-weight chemical.

The immunologic studies performed strongly suggest that the pathogenic mechanism responsible for the development of respiratory symptoms due to tali and jatoba wood in these woodworkers is IgE-independent. The negative results obtained in two unexposed subjects support the specificity of these findings. The reason why patient 2 had asthma as the result of exposure to both jatoba and tali wood remains to be elucidated. It could be due to the presence of some common chemical compound in both types of hardwood,⁹ to immunologic cross-reactivity,¹⁰ or to cosensitization to both wood dusts. Since specific IgE antibodies were not detected in patient sera, immunologic tests could not be used to evaluate this hypothesis.

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