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Occupational asthma due to bethabara wood dust

M.-R. Yacoub, C. Lemièrre, M. Labrecque, J.-L. Malo*

Key words: asthma; occupational disease; wood dust.

Exposure to wood dust can elicit skin and respiratory symptoms in sensitized subjects. Wood of a variety of tree species has been found to cause occupational asthma (OA) in exposed workers (1, 2).

Bethabara is a tree that grows in Brazil as well as in Central and South America and some of the Lesser Antilles. Bethabara is one of the commercial names used to identify the large group of trees known as 'Lapacho' of the various species of *Tabebuia*, belonging to the Bignoniaceae family. Its resilient properties and its resistance to all insect and fungal attacks make it an excellent material for the wood industry, as well as for decorative and specialty items such as billiard cues, archery bows and fishing rods. We describe a case of occupational asthma caused by bethabara wood dust supported by documented specific inhalation challenges (SIC).

A 39-year-old man, an ex-smoker, was employed as a railway platform fitter. His work consisted of joining wood pieces, specifically, bethabara wood. He began to have respiratory symptoms – dyspnoea, dry cough and wheezing – 3 months after starting to work with bethabara wood. He noticed that these symptoms were exacerbated by dust accumulation in the workplace. These

symptoms clearly improved during weekends and vacations. His personal history was indicative of asthma during the first 2 years of childhood.

Skin-prick tests to common aeroallergens were positive for house dust mites. Respiratory function tests did not show airflow limitation ($FEV_1 = 3.24$ L, 82% predicted, $FVC = 4.22$ L, 91% predicted, $FEV_1/FVC = 77\%$), but showed mild airway hyper-responsiveness (methacholine $PC_{20} = 8$ mg/ml). The subject underwent specific inhalation challenges in our laboratory as described by Cloutier et al. (3) to simulate fine particle exposure. On a control day, the subject inhaled white wood dust particles (a mixture of pine, spruce and fir wood dust particles from Eastern Canadian species) by means of a particle generator for 30 min. Spirometry values remained constant throughout the day. A methacholine inhalation challenge performed at the end of the control day showed mild airway hyper-responsiveness (PC_{20} : 7.4 mg/ml). The differential sputum cell count was within the normal range (sputum eosinophilic count: 1.4%) (4). On three subsequent days, aerosolization of bethabara wood dust was carried out for progressively longer periods of time (totals of 5, 30 and 120 min of exposure respectively). On the last day of exposure, FEV_1 started to decrease by 100 min following exposure and reached a maximum drop of 21.7%, 390 min after exposure. Results of the SIC are illustrated in Fig. 1. A methacholine inhalation challenge and sputum induction performed the last day of exposure showed

no significant change in airway responsiveness (PC_{20} : 5.4 mg/ml) but rather a significant increase in the sputum eosinophil percentage (eosinophil count: 9.3%). These findings confirmed the diagnosis of OA due to bethabara wood dust.

Hypersensitivity to bethabara has been described only once in Germany in 1971 (5). In this study, epicutaneous tests to lapachol and desoxylapachol, the two chemical components isolated from bethabara wood, were performed on a carpenter who experienced exacerbated skin and respiratory symptoms in his workplace. A mild erythema was observed upon skin testing with the highest concentration of desoxylapachol and was interpreted as an irritant effect caused by the agent. The fact that erythematous reaction was weaker in a healthy control than in the carpenter led the authors to formulate a hypothesis of a type IV hypersensitivity reaction.

In 1962, Schulz found that desoxylapachol was probably the principal sensitizer in *Tabebuia* species (6). This low-molecular-weight agent is recognized as a strong sensitizer. The presence of other low-molecular-weight sensitizers in bethabara wood cannot be ruled out.

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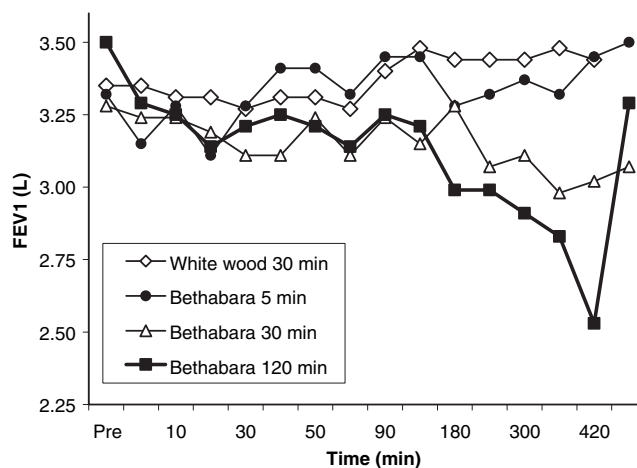


Figure 1. Results of specific inhalation challenges.

*Department of Chest Medicine
 Sacré-Cœur Hospital
 5400 West Gouin Blvd
 Montreal
 Canada H4J 1C5
 Tel: 514-338-2796
 Fax: 514-338-3123
 E-mail: malojl@meddir.umontreal.ca

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Selective immediate hypersensitivity to cefodizime

A. Romano*, M. Viola, R.-M. Guéant-Rodriguez,
 R. L. Valluzzi, J.-L. Guéant

Key words: cefodizime; immediate hypersensitivity; skin tests.

Cefodizime is a third-generation cephalosporin used for a wide range of bacterial infections. This report describes a case of anaphylactic shock pro-

voked by cefodizime, in which a selective hypersensitivity was demonstrated.

We studied a 51-year-old woman who had experienced anaphylactic shock (urticaria, conjunctivitis, dyspnoea, hypo tension) within 10 min after intravenous administration of 1 g of cefodizime for preoperative prophylaxis. Her symptoms subsided 3 h after intramuscular adrenaline, as well as intravenous betamethasone (4 mg) and chlorphenamine (10 mg). Eleven months earlier, she had tolerated intramuscular cefodizime, administered for an acute bronchitis.

Her personal and family histories were negative for allergic diseases; in particular, she had not experienced adverse reactions to other drugs.

One year later, the patient underwent skin tests with penicilloypolylysine (Allergopen, Reinbeck, Germany), minor determinant mixture (Allergopen),

Cefodizime-induced anaphylaxis which in vivo tests suggest was a selective IgE-mediated hypersensitivity reaction.

benzylpenicillin, ampicillin and amoxicillin, as well as cefamandole, cefuroxime, ceftazidime, cefotaxime, and ceftriaxone, as previously described (1).

Cephalosporins were used at a concentration of 2 mg/ml in 0.9% NaCl (2). The patient responded positively to intradermal testing with cefodizime (maximum wheal diameter of 10 mm, surrounded by erythema) after 20 min. Prick and intradermal histamine tests produced wheals with maximum diameters of 6 and 10 mm, respectively. No other reactions were observed. Intradermal tests with the above cephalosporins were negative in 40 control subjects, five of who had previously tolerated cefodizime.

Assays (UniCAP[®]; Pharmacia & Upjohn, Uppsala, Sweden) for specific IgE to penicilloyl G, penicilloyl V, ampicilloyl, amoxicilloyl, and cefaclor were negative (<0.35 kU/l). A drug-solid phase radioimmunoassay for detecting specific IgE, previously described by us (3), was negative with regard to cefodizime.

We performed challenges with penicillin V (therapeutic dose: 1 000 000 IU), amoxicillin (1 g), cefaclor (500 mg), cefuroxime axetil (500 mg), orally, and

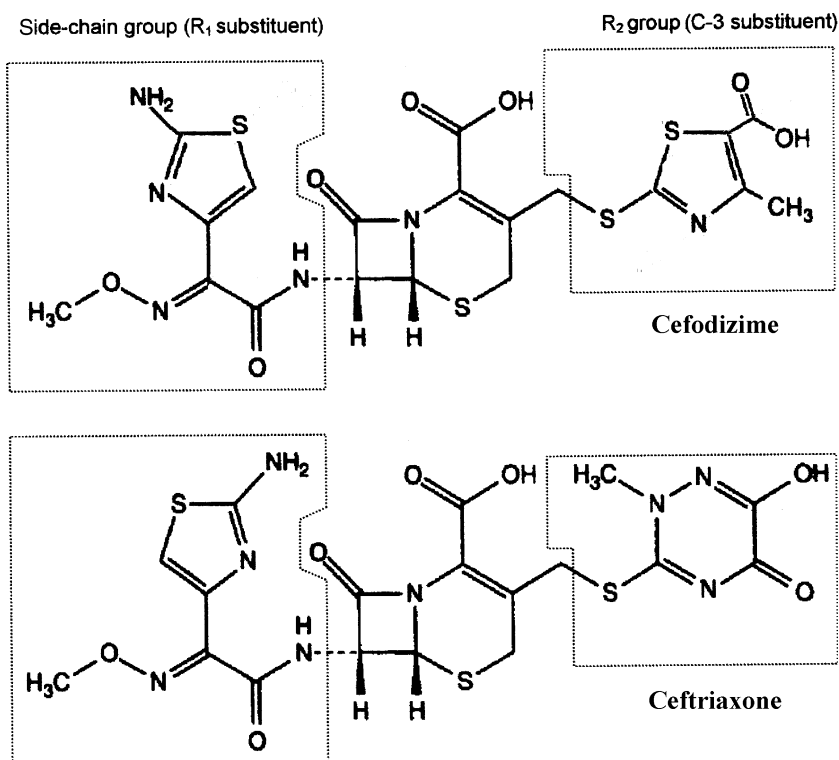


Figure 1. Cefodizime and ceftriaxone chemical structures.