

and spices, to which, however, he showed positive allergy tests. This may be an example of the well-known occasional lack of correlation between immediate hypersensitivity tests and symptoms, a factor which can make accurate food allergy diagnosis a clinical challenge (8).

Three clinical features of food allergy are illustrated by the case reported here. First, manifestations may appear as late as 12 h rather than the usual 1 h after ingestion. Second, skin test and *in vitro* reactivity to related foods may be present in the absence of any adverse effects resulting from their ingestion. And third, a minor ingredient in a prepared dish may be the offending allergen (1, 2). In the present case, the main course of salmon came under immediate suspicion as the allergenic culprit. However, investigation proved that the dill, not the fish, was responsible.

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Allergic alveolitis from pine sawdust

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Key words: biotinylated lectins; carbohydrate antigen epitopes; extrinsic allergic alveolitis; pine sawdust.

● EXTRINSIC allergic alveolitis (EAA) results from immune reactions to various inhaled occupational organic dusts with a particle size of less than 5 µm. Wood dust has been described as a noxious inhalative agent, and cases of EAA

caused by exposure to wood dust have been reported (1-4).

A 57-year-old, nonsmoking butcher, whose hobby was woodworking, had spent much leisure time in his workshop during the last 10 years. He had experienced fever attacks, increasing dyspnea, and fatigue, especially provoked by exposure to sawdust, over the last 4 years. Basal crepitant rales, radiologic signs of pulmonary fibrosis, an alveolar picture typical of acute alveolitis, and slight restrictive ventilation

defects of the lungs were observed: diffusing capacity, 5.46 mmol/min/kPa (61%); vital capacity, 3820 ml (83%); and FEV₁, 2880 ml (78%). The total cell amount was increased in the bronchoalveolar fluid (660×10⁶/l), and alveolar lymphocytosis was seen (lymph. 89%, macroph. 9%, neutroph. 1%). Blood eosinophil count and serum total IgE value were normal. Neither serum IgE antibodies (Abs) against *Dermatophagoides pteronyssinus*, *D. farinae*, *Alternaria alternata*, *Candida albicans*, *Cladosporium* spp., and *Penicillium* spp. nor serum IgG Abs against *Aspergillus fumigatus*, *A. glaucus*, *Thermoactinomyces vulgaris*, and *Saccharopolyspora rectivirgula* were found.

Sawdust from pine, spruce, birch, beech, and apple was collected from the workshop of the patient, and fungal cultures were done. The antigen extracts were prepared by two methods:

- 1) sawdust was incubated in Coca's solution (5) at room temperature (RT) overnight, sonicated at RT for 10 min, and incubated at RT overnight (extract A)
- 2) sawdust was incubated in ether at RT overnight, evaporated, and incubated in Coca's solution at 4°C for 1 week (extract B).

Both preparations were centrifuged, and the supernatants ultrafiltered and lyophilized. Enzyme-linked immunosorbent assay (ELISA) and gel electrophoresis followed by transference to nitrocellulose and

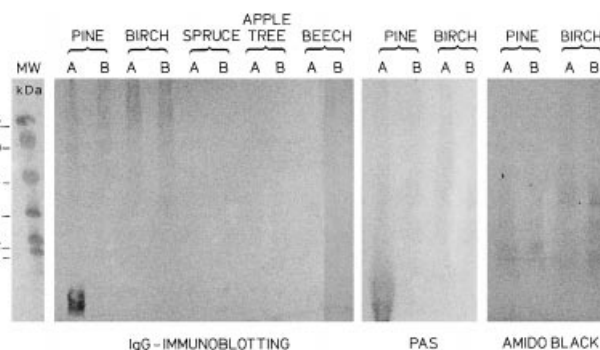


Figure 1. Staining of SDS-PAGE-separated sawdust extracts A and B. Patient serum IgG antibodies detected low-molecular-weight carbohydrate epitopes only of pine extract A. Protein stain (amido black); carbohydrate stain (PAS: periodic acid-Schiff). MW: molecular weight.

immunoblotting were used to detect the specific IgG Abs of the sera from the patient and from five healthy laboratory workers against the antigen extracts (5). Fourfold dilutions of the 4-mg/ml antigen extracts were used in ELISA, while the concentration of antigen extracts used in the electrophoresis was 20 mg/ml. The serum samples were used in 1:100 dilutions. For detection of different carbohydrates of the sawdust extracts, they were dot-blotted against biotinylated lectins concanavalin A, peanut agglutinin, *Lycopersicon esculentum*, wheat germ agglutinin, *Bandeira simplicifolia*, Jacalin, and *Ulex europaeus* agglutinin (Vectastain, CA, USA) (5).

IgG Abs from the patient's serum were detected only against pine sawdust extract A by ELISA and immunoblotting, whereas there were no reactions with the control sera. The amount of IgG Abs in the patient serum against 0.06 mg/ml of pine extract A by ELISA was significantly higher than that in sera from healthy individuals (absorbance 1.1 vs 0.2 [range 0.0–0.3]; $P < 0.001$). Immunoblotting revealed IgG Abs against low-molecular-weight pine-extract A epitopes, which were also stainable with carbohydrate (Fig. 1). Only mannose/glucose-specific biotinylated lectin concanavalin A had significant affinity to pine extract A. In fungal cultures, *Aspergillus*, *Mucor*, *Paecilomyces*, *Penicillium*, and *Trichoderma* spp. were found in very small amounts from all sawdust samples. No other

antigen in the patient's environment other than wood dust was found to cause the disease. Avoidance of wood dust and oral steroid treatment improved the patient's condition.

The antigen extraction in Coca's solution followed by sonication revealed immunologically active epitopes. Possibly, the immunogenic epitopes were not revealed during the ether incubation, or the 1-week incubation in Coca's solution or the ether incubation may have degraded the epitopes. Evidently, the carbohydrate epitopes had degraded during the extraction of B, since none of the lectins bound with the pine extract B.

In reports on EAA after exposure to wood dusts, fungi or thermophilic actinomycetes have been cultivated from the wood dusts, and Abs against the microbes have been found in the patient sera (1–4). In the present case, only trace amounts of fungi, present in normal indoor air, were cultivated from the sawdusts, and these fungi were not considered the etiologic agent. Since this patient fulfilled the criteria of EAA (6), an inhalation challenge test with pine sawdust was not performed.

In this report, we have shown the IgG Ab response to pine sawdust carbohydrates after prolonged exposure to wood dust in a patient with EAA.

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