

nonallergic drug hypersensitivity occurred (3). Since both classes of drugs have been associated with CSS (2,4,5), we were unable to identify a single pharmacological cause (possibly linked to the induction of the vasculitic reaction).

Asthmatic patients who are reducing oral corticosteroids and/or introducing new anti-asthmatic (notably LTRA) or antibiotic (notably macrolide) treatments, should be monitored carefully for the appearance of a multisystem disease, by measurement of erythrocyte sedimentation rate, C reactive protein and peripheral blood eosinophil count.

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Allergen in soy oils

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Key words: food allergy; immunoblot; protein; soy oil.

• SOY ALLERGY is of particular importance because of the ubiquity of soy proteins in contemporary foods. This allergy appears to affect 6% of atopic children (1) and 14% of children with cow's milk allergy (2). Soy, which has been responsible for several fatal and severe reactions, is probably an underestimated cause of food anaphylaxis (3). Several proteins have been identified in soy oil (4), but to date, the allergenicity of proteins from deodorized soy oil has never been observed. In this paper we show that allergenic proteins are present in both cold-pressed soy oil and deodorized soy oil. The allergenicity of at least one of these proteins has been demonstrated by Western blot.

The serum was taken from a soy-sensitized woman with a high level of soy specific IgE (18.9 kU/l) (RAST, CapSystem, Pharmacia, Uppsala, Sweden). Two kinds of soy oil were tested: deodorized (type A) and cold-pressed (type

B) soy oils were supplied by the same company. The proteins were extracted from 500 ml of each batch using PBS buffer at 40°C and stirred for 3 h. The aqueous phase was dialyzed for 2 days.

The total protein contents of oils A and B were 0.32 µg/ml and 1.8 µg/ml, respectively. The SDS-PAGE of deodorized and cold-pressed soy oils showed the same pattern (Fig. 1a). The immunoblots of batch A and batch B, conducted with the serum of a soy-sensitized patient, showed two bands at 28 and 56 kDa, respectively, the latter being the main band, whereas the immunoblot using serum of a nonsensitized patient did not exhibit any binding (Fig. 1b).

In conclusion, we showed that allergenic proteins were present not only in cold-pressed but also in deodorized soy oil. We are currently trying to find out whether this allergen is clinically relevant.

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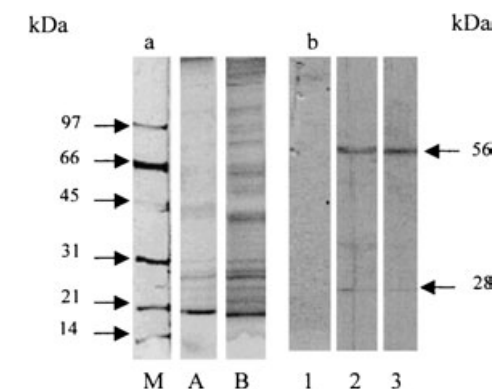


Figure 1. (a) Silver stained SDS-PAGE patterns of proteins from deodorized (A) and cold-pressed (B) soy oils. M: molecular weight marker. (b) IgE immunoblots of proteins from each batch of soy oil. (1) negative control; (2) cold-pressed soy oil (B); (3) deodorized soy oil (A).

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Pigeon Fancier's Lung after low exposure

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Key words: antibodies; child; lymphocyte; Pigeon Fancier's Lung.

● PIGEON FANCIER'S Lung (PFL) is an allergic alveolitis caused by repeated inhalation of pigeon antigens. The immunopathogenesis is not clear, but immune complexes and lymphocyte-mediated responses to IgA and mucin contained in feather bloom and pigeon droppings may be involved (1). One difficulty in understanding the disease is that antibodies and lymphocyte proliferation appear in both symptomatic and asymptomatic fanciers (2,3). PFL should be considered for patients presenting intermittent pulmonary and systemic symptoms and interstitial lung disease. Diagnosis is especially difficult in children.

We present the case of a 3-year-old child, misdiagnosed with tuberculosis, who developed PFL after brief contact with pigeons. He was admitted with dry cough, cyanosis, respiratory difficulty,

A child misdiagnosed as tuberculosis.

myalgia, anorexia, weight loss and fever. He had lymphocytosis, and chest radiography showed a bilateral micronodular infiltrate with hilar adenopathies. His sister had had a positive Mantoux test, so empiric treatment with tuberculostatic drugs was initiated. His condition improved and he was discharged, but returned one week later with the same symptoms. The father kept pigeons and PFL was suspected. Specific IgG antibodies to pigeon serum (ELISA) were 3.0, 2.8, 2.1, 0.5 and 0.4 for the child, his father, his mother, his sister and ten nonexposed controls (mean value), respectively. Specificity was confirmed by ELISA inhibition. A lymphocyte transformation test was performed (Fig. 1). The molecular weight of the pigeon serum proteins and their capacity to fix IgG antibodies was determined by SDS-PAGE and immunoblotting. The patient and his father recognized a 205 kD protein, corresponding to IgA. No staining was observed in the mother, sister or ten nonexposed controls. PFL was diagnosed, tuberculostatics were withdrawn, corticoids were administered (2 mg/kg/day), and avoidance of pigeons advised. The patient improved markedly.

We conclude that PFL may occur in children after brief exposure, with symptoms that mimic other diseases such as tuberculosis. Correct laboratory analyses and a careful clinical study lead to the diagnosis. Interestingly, while the child and his father had similar

immunological responses, the mother had only an antibody response, and the sister none at all, it is not clear why only the child was affected when they all had the same degree of exposure.

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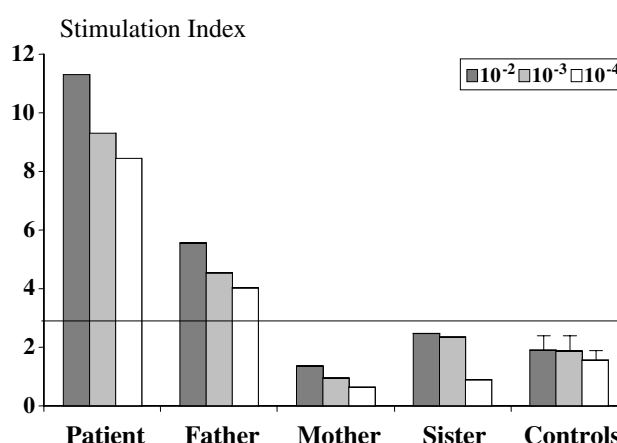


Figure 1. Lymphocyte transformation test to pigeon serum in the patient, his father, mother, sister, and ten nonexposed controls. A 'stimulation index' higher than 3 was considered positive. Three dilutions of pigeon serum were used.