

was seen at 30 min, with a late response at 4 h, and the patient was sent to the hospital emergency ward. A fall of 40.8% in PEFR was observed, and this was reversed by specific treatment (Fig. 1).

Positive RAST was found to *D. pteronyssinus* (class 2, 1.17 IU/ml) and to *Tyrophagus* (class 4, >44 IU/ml). Specific IgE to the remaining mites, pollens, fungi, and garlic extracts was negative. The three samples studied by microscopy presented infestation by *T. putrescentiae*. The degree of infestation was estimated at 1800 mites/g of dust.

Garlic is considered to be one of the most common causes of dermatitis of the fingertips in suppliers of foodstuffs. However, its association with asthma or rhinitis rarely has been reported in the literature. In 1940, Henson reported a case of occupational asthma, with immediate and late asthmatic reactions, induced by garlic dust in a sausage maker. Since then, we have found six cases that, as in our patient, suggest an etiopathogenic mechanism of IgE-mediate immediate hypersensitivity, determined by skin tests, *in vitro* tests, and immediate response in the bronchial challenge test (3–5).

We think that the asthma suffered by our patient could be considered occupational because, after a symptom-free sensitization period, symptoms and spirometric changes occurred during each exposure to garlic dust but improved rapidly after the patient ceased this activity. The true problem was this: which allergen of garlic dust causes this asthma? Our *in vivo* and immunologic tests showed a relevant association with sensitization to *T. putrescentiae* and a superinfestation by this mite in different samples of garlic dust. Arlian (J Allergy Clin Immunol 1984;74:166) described the allergenic properties of this storage mite, which can infest various dusts. Moreover, Ownby et al. (J Allergy Clin Immunol 1993;92:846) have described a case of anaphylaxis due to ingestion of another member of the Liliaceae family, the onion, showing that the cause was the superinfestation of the onions by *D. farinae*, and not the ingestion of pure onion.

In conclusion, we think that occupational asthma in our patient was not caused by garlic itself, but was due to the superinfestation of the garlic by *T. putrescentiae*. This finding suggests that in asthma induced by inhalation of garlic dust, other, more common causes of allergy which might have contaminated this dust should be checked for, especially storage mites.

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Atopy and season of birth

EXPOSURE during the first months of life is important for atopic disease several years later (2, 3, 7). Especially children at high genetic risk of atopic disease, born during or immediately before the peak pollen season, seem to become sensitized and develop symptoms of allergy more readily than those without such risk factors (3). One large study has failed to demonstrate a seasonal effect (5). The cause of the discrepancy is unclear but can hardly be a selection bias in earlier studies, as at least some of them used objective assessments in unselected groups of subjects (3, 7). In a large study of serum samples, Aalberse et al. (1) showed that the risk of food sensitization peaked among subjects born during the winter and the lowest frequency was found among those born in May.

■ To determine whether the findings by Aalberse et al. (1) were reproducible, we performed skin prick tests in a nonselected series of 710 children, 7 months of age, taking part in a vaccination study. All the families of the children born in Linköping, Sweden, during 1992 were invited to participate at Child Health Centres. Exclusion criteria were circumstances that interfered with communication and follow-up. Contraindications were chronic illness. Furthermore, the first vaccination had to be given before 3 months of age. The study included 52% boys and 48% girls. Skin prick tests were done in duplicate on the volar aspect of the forearm with undiluted cow's milk and hen's egg (large batches frozen in aliquots; each aliquot thawed, refrigerated and used within 72 h) and were regarded as positive only if wheals had a mean diameter of 3 mm or more after 15 min. Tests with histamine 10 mg/ml were included to control for unresponsiveness due to treatment with, e.g., antihistamines (not found), and blank lancets were used to control

for potential dermatographism (not found in this study).

The chi-square-test was used for statistical assessment. The parents gave informed consent and the study was approved by the regional Ethics committee.

■ A positive skin prick test to food was found in 95 (13%) of the 710 babies. Egg sensitization was found in 12% and milk sensitization in 2% of them. As expected, egg sensitization was significantly affected by family history, the highest frequency being observed in children with atopic disease in both parents. The prevalence of egg sensitization was similar in babies of atopic mothers (13%), atopic fathers (15%), and siblings (13%).

However, there was a seasonal pattern in egg sensitization (Fig. 1). The highest prevalence of sensitization to hen's egg (26/150, 17.3%) was found for babies born during October–December ($P < 0.025$ compared with those born during the rest of the year) and the lowest in those born during April–June (19/194, 9.8%). Smoking habits of the family, indoor pets or dampness problems, sex, and age at introduction of egg into the diet had no significant effect on the seasonal distribution of egg sensitization (data not shown). The seasonal effect on egg sensitization is thus in agreement with the results of Aalberse et al. (1). We can only speculate on the explanation. Food allergen exposure by other routes than ingestion, e.g., through food allergens in the air, may be important. Improved insulation and poor ventilation of houses may increase indoor levels of allergens and adjuvants. Babies spend

almost all their time indoors, a fact which might explain this seasonal effect. Other possible explanations include seasonality of respiratory infections acting as adjuvants, respiratory syncytial virus perhaps being particularly interesting (6).

We also found another seasonal effect. The birth of children with a family history of atopic disease was not evenly distributed over the year. Relatively fewer children with atopic parents were born during January–March (for double family history 25/181 vs 122/529, $P < 0.01$; single 61/181 vs 238/529, $P < 0.01$) than during the rest of the year. For parental diseases, the discrepancy in birth distribution over the year was most pronounced for allergic rhinoconjunctivitis (51/180 vs 228/529, $P < 0.001$). Children with no family history of atopy were overrepresented among those born during January–March (95/181 vs 169/529, $P < 0.001$). This discrepancy could be chance, but it is tempting to attribute it to self-selection or effect of parental disease, as the conception time for children born during January–March coincides with the pollen season in Sweden.

The children will be followed to 10 years of age, thus allowing us to see whether the seasonal effect on egg sensitization for those born during the autumn is also followed by an increased risk for allergic manifestations. An increased risk of later sensitization to inhalants and to atopic disease among children sensitized to food allergens in infancy has been documented (4).

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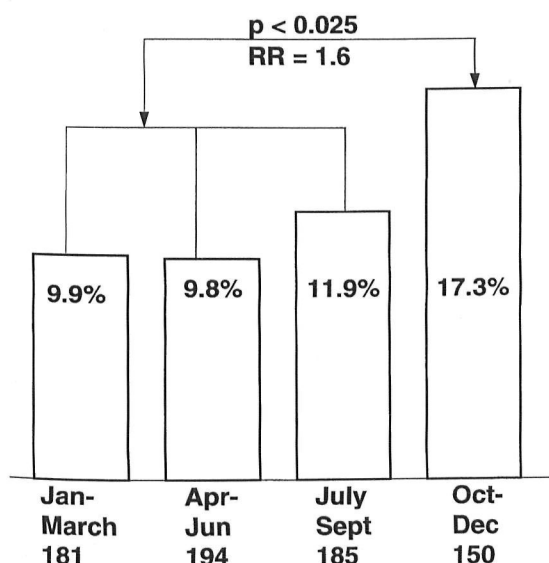


FIGURE 1 Sensitization rate (%) to hen's egg according to skin prick test at 7 months of age in 710 children in relation to when they were born.

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