

after the injection, there is a wheal at least double the size of the injection bleb, and which shows signs of erythema surrounded by flare. The IDT is compared to a positive control (codeine phosphate: 50 µg/ml) and a negative control (saline solution).

In our subjects, the rate of the mediators had increased. The prick tests to ICMs were 50% negative, whereas the IDTs were positive every time (Table 1).

The severity of and time lapse in the onset of clinical signs, prior exposure, increase in tryptase level which seemed to be correlated to the severity of the reaction (3), positivity of IDTs, and reintroduction of a nonreactive ICM in cutaneous tests without incident all support the hypothesis of IgE mediation. Thus, cutaneous tests appear to be of prime importance in identifying the culprit ICM. ICMs are viscous and spread with difficulty into the epidermis. This may explain the negativity of prick tests. On the other hand, IDTs seem to be more appropriate for a reaction that provokes every time a wheal at least double the injection wheal.

However, this test is positive according to the degree of sensitivity of each patient: the more the patient is sensitized, the weaker the reactive concentration is. The test starts at 10^{-3} and goes to 10^{-1} , a concentration that does not produce histamine release, even with hyperosmolar ICMs, as proved by negative IDTs at 10^{-1} for ioxitalamate (Telebrix®) in nonallergic patients who presented an incident of mild severity to this ICM (1).

Therefore, we suggest that patients who have had an anaphylactoid reaction to an ICM injection should have their tryptase level measured (considered as the marker of mast-cell activation) (5) and should undergo intradermal tests so as to identify and eliminate the culprit ICM in order to avoid any subsequent allergic accidents. Searching for cross-sensitization seems to be necessary. In two cases, this has enabled us to

determine to which ICMs the patients were not sensitized and to inject them with these safely.

*Service d'Anesthésie-Réanimation
Hôpital Central
CO n° 34
54035 Nancy cedex
France
Tel. 0033/3.83.85.15.31
Fax: 0033/3.83.85.10.39
E-mail: mcLaxenaire.nancy@invivo.edu

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Exhaled NO reduced on allergen avoidance

G. L. Piacentini, M. J. M. Del Giudice, A. Bodini, S. Costella, L. Vicentini, D. Peroni, A. L. Boner*

Key words: allergen avoidance; mite; nitric oxide.

● In a previous study (1), we have shown that the level of exhaled nitric oxide (NO) (ENO) represents a sensitive and promptly modifiable marker to monitor antigen exposure in mite-sensitized asthmatic children. This study aimed to determine whether the actual level of exposure to house-dust-mite (HDM) antigen can influence the magnitude of ENO reduction after antigen avoidance in a group of allergic asthmatic children.

We evaluated the level of ENO before (T0) and after a 3-month period of mite avoidance (T1) in an Alpine environment (Misurina, Italian Dolomites; altitude: 1756 m) in a group of 14 schoolchildren sensitized to HDM. The level of HDM group I antigen from the beds of the patients in their family homes was measured by a two-site immunometric ELISA (2). Exhaled NO measurements were made with a chemiluminescence analyzer (Logan LR 2149, Rochester, UK) (3). Values of ENO considered in the data analysis were always measured in the last part of exhalation (plateau of exhaled NO), taking the plateau of the end-exhaled CO₂ reading as representative of an alveolar sample (3).

None of the children in this group had presented airway tract infection for at least 1 month before each of the NO measurements. None of them were allergic to furred pets.

The children had received a regular course of inhaled steroids for at least 3 months at

NO reduction is related to starting exposure level.

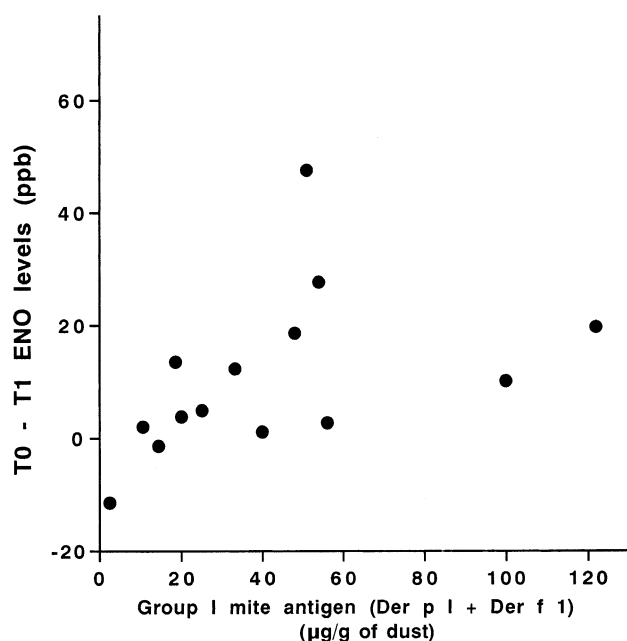


Figure 1. Relationship between difference in ENO level at T0–T1 and HDM group I antigens in study group.

the beginning of the study (T0). The regular treatment with inhaled steroids was gradually withdrawn in all the subjects. None of the subjects had received inhaled steroid treatment for at least 1 month before T1.

The study was approved by the Istituto Pio XII ethics committee, and both children and their parents gave informed consent.

The correlations between the differences of ENO levels at T0 and T1 (T0–T1) and the levels of HDM antigens in the beds of the patients before T0 were tested by Spearman's rank correlation test. The correlation between these values showed a value of $r=0.618$ ($P=0.026$) (Fig. 1).

In this study, we confirm that ENO measurement is a sensitive means to monitor avoidance of antigen exposure in asthmatic children allergic to HDM. The relevance of the effect of the original level of exposure to HDM on ENO reduction after HDM avoidance is even more evident if it considered that this statistically significant level of correlation was achieved in a small group of patients.

The results of this study reinforce the idea that the environmental control measures need to be made as effective as possible to achieve the most complete reduction of HDM in the environment of allergic asthmatic patients, particularly their beds.

*Clinica Pediatrica
Policlinico Borgo Roma
37134 Verona
Italy
Tel. +39 45 8074615
Fax: +39 45 8200993
E-mail: boner@borgoroma.univr.it

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Clinical features of cashew allergy

J. O'B Hourihane*, H. Harris, S. Langton-Hewer, S. A. Kilburn, J. O. Warner

Key words: allergy; cashew nut; fatal reactions.

● CASHEW has recently become widely available in the form of butter spreads. Cashew causes symptoms in formal food challenges (1) and has caused death (2). A report found 0.08% of British 4-year-olds to be allergic to

cashew (3), and 40% of 142 French peanut-allergic subjects

A potentially life-threatening disorder.

were found to be sensitized to cashew (4). We report here the clinical features of cashew allergy in 29 paediatric and adult subjects whose history of reaction was supported by a positive skin prick test to cashew or a raised serum level of cashew-specific IgE. Two subjects had positive open challenges to cashew. This study was approved by the Southampton Joint Ethics Subcommittee.

The age range of the 29 subjects was 1–30 years (median 7.5 years). Nineteen (65%) of the subjects were female. Twenty-six (89%) subjects were children (under 16 years). The median age of onset was 49 months (range 2 months–27 years), and 96% of subjects had