

Correspondence

Evidence for allergic reactions in isocyanate asthma

To the Editor:

The publication by Jones et al¹ dealing with the question whether isocyanate-induced asthma has an allergic (IgE-mediated) background or not is diagnostically important. Most essential is the use of immunohistochemistry and in situ hybridization to determine mRNA for Cε, IL-4, and IL-5 in bronchial biopsy specimens. The authors investigated 12 patients with suspected isocyanate-induced asthma. Five of them showed asthmatic reactions to hexamethylene diisocyanate or methylene diphenyl diisocyanate inhalational challenge tests (duration of exposure obviously 15 minutes; administered diisocyanate concentrations are not provided).

The authors' conclusion in the summary, that the striking absence of both bronchial Cε and IL-4 RNA message after inhalation challenge with diisocyanates indicates that diisocyanate-induced asthma is a non-IgE-mediated disease, requires some critical remarks.

First, as opposed to the findings of Jones et al,¹ Maestrelli et al² reported significantly increased IL-4 expression in isocyanate responders with asthma. Second, challenges longer than 15 minutes obviously do not represent workplace conditions with regard to exposure durations and their peaks in most workplaces. Further, nowadays mainly polyisocyanates or isocyanurates are used as hardeners or for injection molding. Did the authors consider the composition of the products used in individual workplaces?

According to several publications, IgE-mediated sensitization in isocyanate workers occurs, and nearly all of these sensitized subjects are symptomatic.^{3,4} Our studies revealed only 1 asymptomatic case within more than 200 IgE-positive subjects. It remains unclear why 2 studies did not find evidence for IgE-mediated sensitization in isocyanate asthma. It should be mentioned that bronchial asthma is not the only isocyanate-induced disorder.

Nonobstructive bronchitis, rhinitis, conjunctivitis, cutaneous hypersensitivity, and hypersensitivity pneumonitis represent further diagnoses that may be associated with antibody responses. The production of specific antibodies in a dose-dependent manner (with regard to both antibody titers and prevalence) could be demonstrated in animal studies.

The limited number of patients with asthma studied by Jones et al¹ does not permit drawing final conclusions. We have to wait and see whether further investigations will support these initially interesting findings. The current literature indicates that pathomechanisms involved in isocyanate-induced disorders are complex and heterogeneous. There is overwhelming evidence for acute and chronic inflammation in the airways caused by isocyanates that is associated with cellular immune responses, and in a subgroup of symptomatic workers also with IgE-mediated sensitization. There is no reason to assume that specific IgE antibodies whose levels are rather high in some affected isocyanate workers do not have clinical relevance.

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