Scand J Work Environ Health 1981;7(4):310-311

<https://doi.org/10.5271/sjweh.2543>

Issue date: Dec 1981

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Key terms: [car paint](#); [letter to the editor](#); [letter to the editor](#); [life-threatening pulmonary reaction](#); [life-threatening reaction](#); [prepolymerized isocyanate](#); [pulmonary reaction](#)

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/7347916

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LETTERS TO THE EDITOR

Life-threatening pulmonary reaction to car paint containing a prepolymerized isocyanate

We want to describe a case who had slight symptoms of increased bronchial irritability for many years and developed a life-threatening pulmonary reaction when exposed for the third time in a year to aerosolized polyurethane car paint containing a prepolymerized isocyanate. He also had specific immunoglobulin E (IgE) antibodies to the isocyanate.

This patient was a 35-year-old non-smoking male who had been completely healthy except for a slight tendency to cough and to feel chest tightness when inhaling gasoline and diesel combustion fumes. As a gasoline station manager for 8 a, he had frequently been exposed to these air pollutants.

His big interest through the years had been motor vehicles, initially motorcycles and later on rally-cross cars. For this reason he had often trimmed and repaired cars in small, badly ventilated workshops. When he sprayed his sports car the first time with a polyurethane paint containing prepolymerized hexamethylenediisocyanate (HDI) and also 1.6% or less of a monomer of HDI (Bayer Desmodur 75 N), he developed a cough and chest tightness in the evening of that day only.

A year later he again sprayed his car with the same paint. In the evening he got a cough and considerable chest tightness which persisted for two weeks.

A month later he worked on his car in the room next to one where a friend was spraying with the same polyurethane paint. Within 15 min he started to cough, his chest became tight, and he developed a chill. The symptoms progressed into a serious asthmatic reaction, for which he was admitted to an intensive care unit at the local hospital on the following day.

His clinical status included dyspnea, prolonged expirium and crepitating rales. No wheezing was heard. Blood gases showed hypoxia with aB-pO₂ 7.0 kPa. No fever, leucocytosis, or eosinophilia was found. He was initially treated with intravenous theophylline and subcutaneous terbutaline. In spite of a good therapeutic response his forced expiratory volume in 1 s (FEV_{1.0}) was only 1.4 l the day after admittance. He was discharged from the hospital in good condition after another 6 d.

The patient's serum was analyzed three months later with the radioallergosorbent test (RAST) method, and IgE antibodies, particularly to HDI-HSA [HDI covalently combined with human serum albumin (HSA)] and to MDI (diphenylmethanediisocyanate)-HSA and to a less extent to TDI (toluenediisocyanate)-HSA, were demonstrated (1). The positive result with MDI and TDI was probably due to cross-reactivity, since he had not been exposed to these isocyanates. Table 1 shows the results of the RAST. Total IgE in serum, determined by the paper radioimmunosorbent test (PRIST) method (Pharmacia), was 110 kU/l.

Six months later the patient was skin-tested with the prick test method. Common allergens gave negative results, while

Table 1. Radioallergosorbent test (RAST) for immunoglobulin E antibodies in serum. A pool of nine sera from nonsensitized persons gave an uptake lower than 250 counts/min on all discs. (HSA = human serum albumin, HDI = hexamethylenediisocyanate, MDI = diphenylmethanediisocyanate, TDI = toluenediisocyanate)

Disc	Counts/min	RAST class
HSA	260	0
HDI-HSA	6,600	3
MDI-HSA	5,900	3
TDI-HSA	1,800	1

conjugates of HDI-HSA, as well as MDI-HSA, (5 mg/ml) elicited significant wheal and flare reactions. Twenty controls were all found to be negative when tested with the isocyanate conjugates. Electrophoresis and ultraviolet spectrometry showed that the conjugates had an average substitution of 8 mol of HDI and 6 mol of MDI per mole of HSA.

Bronchial challenge on the same day gave an obstructive reaction with a 25 % drop in FEV_{1.0} with 1 mg of metacholine. Upon a final challenge with 5 mg of metacholine, the FEV_{1.0} dropped to a level 55 % below the base line within 5 min. Because of the severe reaction he displayed in the occupational setting, we restrained from carrying out a bronchial provocation test with HDI.

After his last severe reaction he has suffered problems with tightness in his chest when exposed to all kinds of dusts and odors, and he particularly has to avoid entering motorshops when spray painting is going on. He uses a salbutamol inhalation spray once or twice most days. Interestingly he has never noticed wheezing.

The findings of IgE antibodies and skin reactivity to HDI-conjugated HSA by the RAST and the prick test, which to our knowledge have not earlier been described, as well as bronchial hyperreactivity with a strongly positive metacholine test, may indicate alternative mechanisms behind his hypersensitivity. We are puzzled by the signs of both specific and nonspecific mechanisms in this case and have not been able to determine which is the most important in causing his asthma.

References

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