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Asthma Induced by Dust From Urea-Formaldehyde Foam Insulating Material*

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A patient developed severe asthma following insulation of her house with urea-formaldehyde foam. Bronchial challenge with the buoyant dust of the foam caused an asthmatic attack; inhalation of formaldehyde, 3 ppm, did not.

Urea-formaldehyde foam insulating materials (UF foam) are used extensively in the United States for wall cavity insulation in homes, schools, hotels, and hospitals and for frost protection as an overlay on agricultural crops. The preparation and chemistry of the UF foam have been described elsewhere.¹⁻³ The industry claims that the UF foam is energy-saving and safe.³ Little is known about the prevalence of disease induced by the UF foam. *In vitro* experiments that tested the two major components of the UF foam, the "resin" and the "foaming solution," have raised the possibility that these unpolymers starting materials may have deleterious genetic and even carcinogenic potential.⁴ In addition, inhalation of formaldehyde fumes may cause asthma.⁵ At levels above 5 ppm, formaldehyde gas irri-

tates the mucous membranes,⁶ and in the United States, industrial regulations specify that no employee should be exposed to a concentration of formaldehyde greater than 1 ppm for any 30-minute period of sampling.⁷

CASE REPORT

A 47-year-old white woman was evaluated at the Mayo Clinic for a 2½-year history of perennial, severe steroid-resistant asthma. She had asthma as an infant until two years of age, but then was symptom-free and in good health until her 45th year when she again developed asthma in the fall of 1977. She had lived in the same farm house for the previous 26 years. Her symptoms began shortly after insulation of her home with UF foam in the fall of 1977. In April 1978, she developed, without any apparent cause, a severe asthma attack requiring emergency hospitalization. Since March 1979, despite treatment with glucocorticoids, her asthma worsened, and she was hospitalized eight times prior to her visit to the Mayo Clinic in December 1979. Her asthma would worsen at night, especially upon awakening. She had never smoked nor had anyone else in her immediate family. Physical examination was unremarkable except for some stigmata from chronic glucocorticoid therapy, and the chest was clear to auscultation and percussion. Chest x-ray film findings were within normal limits, and the standard laboratory tests were negative or normal. Skin tests to common inhalants and molds, as well as to weeds, grasses, and trees of the northern midwest were negative.

Bronchial Provocation Tests

All tests were performed on different days starting at 8:00 AM. Baseline flow-volume loops were determined each day prior to bronchoprovocation. All measurements of the baseline FEV₁ were 85 percent or greater of the predicted value. The first 40 minutes of each bronchial challenge were divided into four periods of ten minutes each. The bronchoprovocations were executed at the beginning of each period, and flow-volume loop measurements were done at the end of each. Subsequently, additional flow-volume loops at 7 and 24 hours after bronchoprovocation were recorded to check for late reactions. A drop in FEV₁ of 20 percent or greater from the baseline was considered a positive test. On day 1, we established the baseline flow-volume loop. On day 2, we performed bronchial challenge with the technique described by Pepys and Hutchcroft⁸ using the dust of the UF foam the patient had brought from her home. The patient was exposed to the inhalation of the fine buoyant dust for a total of two minutes. As shown in Figure 1, following four sequential challenges with the UF foam dust, her FEV₁ dropped in 50 minutes to 64 percent and 1½ hours later, to 51 percent of baseline. By 60 minutes, the patient developed severe shortness of breath, wheezing, and a dry cough. These symptoms were reversed promptly by 0.3 ml of epinephrine (1:1,000), subcutaneously. No late reaction occurred. On day 3, we performed a control bronchial challenge using aluminum oxide dust (Fig 1) to exclude the possibility she might be nonspecifically reactive to any kind of dust. No bronchial reaction occurred. On day 4, we tested gaseous formaldehyde at a concentration of 3 ppm in room air. This concentration of gaseous formaldehyde was prepared as suggested in reference 9. The patient inhaled this mixture for a total of eight minutes. No bronchial reaction was noted, and the patient remained asymptomatic. On day 5, we tested dust of urea-formaldehyde resin bought from a local dealer. No reaction was noted. Two subjects with

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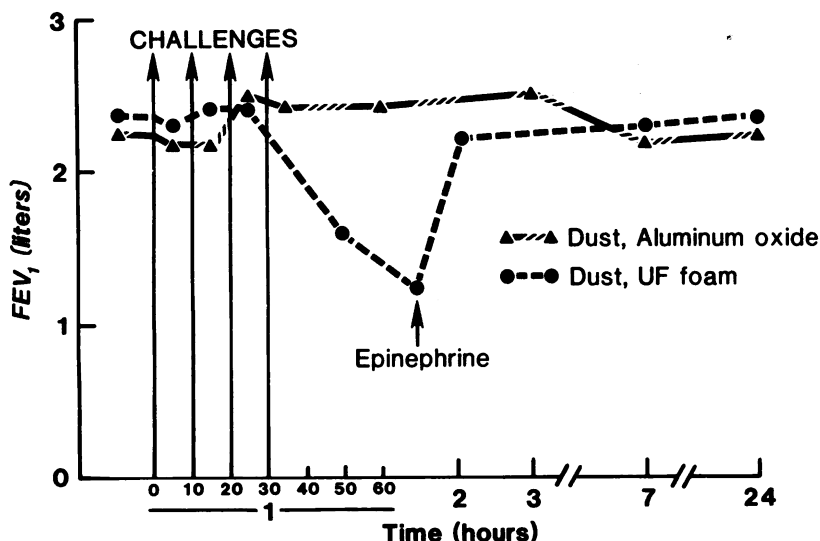


FIGURE 1. Bronchial reactions on inhalation testing with aluminum oxide dust (triangles) and UF foam dust from patient's home (closed circles). FEV₁ = forced expiratory volume at one second.

asthma living in homes insulated with UF foam, one asthmatic and two normal subjects not exposed to UF foam, were similarly tested by bronchial challenge to UF foam dust. No reaction occurred. In all bronchoprovocation tests, the patient and the controls were aware of the nature of the challenge materials. Because of the characteristic appearance of the dust, it was not possible to perform the challenge in a blind fashion.

DISCUSSION

We found no reports suggesting a cause-and-effect relationship between exposure to UF foam and asthma either *ex novo* or aggravation of pre-existing asthma. The case of our patient suggests the latter. We believe the reaction of our patient to bronchoprovocation with UF foam dust was specific for this material because she did not react to aluminum oxide, an inert dust, and other patients with asthma, including two who were exposed to UF insulation, did not react when challenged with this UF foam dust. The severity of the response to the challenge greatly exceeded that obtained by suggestions.¹⁰ It seems appropriate that in taking the history of an asthmatic, a specific question should be asked about the nature of the home insulation. We were informed by NAUFIM (National Association of Ureafoam Insulation Manufacturers-Florence, Ky) that emission of gaseous formaldehyde may last for years following defective installment of an incorrectly formulated UF foam; this has also been reported by Baumann.¹¹ The air level of formaldehyde in the indoor environment of the patient's residence should be measured and appropriate advice given.

Unfortunately, the positive bronchial challenge in our patient offers little information concerning which one of the many components of the UF foam caused the reaction. The final material is a complex mixture of compounds, some of which are unknown.¹⁻³ It does appear, however, that the formaldehyde was not the cause of our patient's asthma since bronchoprovocation with 3 ppm of gaseous formaldehyde caused no reaction. We advised our patient to stay away from her home, and we reevaluated her four months later. Her asthma was

well controlled with theophylline and terbutaline without the need for glucocorticoids.

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