

30, and 120 min). After the 120-min exposure, the subject developed an atypical reaction of the progressive type (maximum fall in FEV₁ of 22% at 390 min after exposure); 8 h after exposure, FEV₁ was still 19.6% lower than baseline and returned to baseline after salbutamol. The next morning, PC₂₀M was assessed at 24 mg/ml. Three weeks later, we re-exposed him with simultaneous measure of lung volumes: an early-late reaction (24% fall in FEV₁ at 210 min after exposure) occurred (Fig. 1) with a rise in residual volume of 32%, in functional residual capacity of 23%, and in total lung capacity of 8% of baseline. PC₂₀M at the end of the day was 128 mg/ml. Handling methacrylate- and cyanoacrylate-based adhesives in a challenge room for 120 min each did not provoke any significant change in FEV₁ or in PC₂₀ (Fig. 1). Such an exposure had induced asthmatic reactions in sensitized subjects in our laboratory.

The possibility of bronchial cross-reactivity between acrylates was examined in two other subjects. A 57-year-old male, nonatopic ex-smoker developed rhinitis and asthma 1 year after starting exposure to CA- and MA-based adhesives. Exposure to CA for 30 min in the laboratory was followed by an early-late fall in FEV₁ (maximum fall of 25% at 300 min after exposure) and a simultaneous rise in lung volumes (FRC +36% of baseline); there was a change in PC₂₀M from 18 mg/ml to 3.6 mg/ml. Exposure to MA in the same way for 120 min did not cause any change in FEV₁ or in PC₂₀M in the following 8 h. The second subject was a 23-year-old dental technician, a nonatopic smoker, who developed OA 3 years after starting work. Exposure to MA for 120 min in the laboratory did not provoke a significant change in FEV₁ or PC₂₀M. However, SIC with CA caused an early-late reaction with a maximum fall in FEV₁ of 47.8% at 180 min after a progressive exposure of 30 min; hydrocortisone was administered intravenously, leading to functional recovery.

We report a case of work-related rhinitis and asthma due to diacrylate, confirmed by SIC, without bronchial cross-reactivity to other

acrylates (CA, MA). To our knowledge, diacrylates do not cause OA. Estlander et al. described a patient with allergic conjunctivitis, but no chest or nasal symptoms, who was sensitized to several methacrylates as well as to diacrylates, as confirmed by patch testing (5). We knew that unknown additives were in the product to which our patient was exposed, but pure diacrylate was not available for testing. As forced expiratory maneuvers are effort-dependent, we confirmed that the fall in FEV₁ was associated with airway obstruction by measuring lung volumes; this showed air trapping and hyperinflation. Acrylates are known irritants; however, the latency period before development of symptoms and the nonimmediate response after SIC in our subject are more suggestive of a specific and sensitizing mechanism, despite the absence of nonspecific bronchial hyperresponsiveness. However, as for most low-molecular-weight agents, the immunologic mechanism cannot be elicited. Changes in bronchial responsiveness were not observed after the late asthmatic reaction, but such changes are not constant in these circumstances (6). OA without evidence of bronchial hyperresponsiveness has been described by others (7, 8).

We found no clinical cross-reactivity between CA and MA in two other subjects with OA to CA, nor between diacrylate and monoacrylates in our subject. As is the case for formaldehyde, the physical state of the agent (vapor for monoacrylates, aerosol for diacrylate) may explain this finding (9).

In conclusion, we report two new interesting findings:

- 1) a new occupational sensitizing agent, diacrylate, causing rhinitis and asthma
- 2) the absence of clinical bronchial cross-reactivity between different acrylates.

However, further studies on this last finding are warranted.

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Asthma to latex in a seamstress

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Key words: asthma; occupational diseases; latex; textile industry.

● LATEX is recognized as a cause of occupational asthma (OA) in various working environments. Latex allergy in the textile industry has been recently reported (1). We herein describe the first case of a seamstress who developed OA to latex.

A 36-year-old woman, a nonsmoker, started work as a seamstress in 1981. She worked with cotton and gabardine. From 1991, she sewed elasticized ribbons, using a different sewing machine, for a few days at a time at irregular intervals, but almost weekly. In 1993, she developed nasal

Occupational, skin test-positive asthma in a woman sewing elastic ribbons.

congestion, rhinorrhea and sneezing, and runny and itchy eyes, as well as itchy skin, followed a few weeks later by chest tightness, wheezing, and cough. Symptoms occurred progressively when she worked with the elasticized ribbons, and a few days of exposure also caused nocturnal awakenings. These symptoms improved during weekends and when she was reassigned to other jobs in the same plant. When we first evaluated her in December 1997, after a week of not working with the elasticized ribbons, spirometry was normal ($FEV_1/FVC=2.57/3.19$ l, pred $2.67/3.05$ l) (2). Methacholine PC_{20} was <1 mg/ml (48% fall in FEV_1 at 1 mg/ml, the first concentration given) (3). Skin prick tests (SPT) with 22 common inhalants were negative; SPT with latex (Stallergènes, Paris, France, and Omega, Montreal, Canada) were positive (wheals of 5×4 and 5×3 mm, respectively) with a negative control and a histamine wheal of 5×5 mm. In January 1998, after having worked several days with the elasticized ribbons, FEV_1/FVC fell to $1.74/2.45$, returning to normal after salbutamol ($2.78/3.38$). She was treated with topical nasal and inhaled steroids (fluticasone 1000 µg/day) and salbutamol as needed (higher need when at work). After withdrawal from work in February 1998, there was a marked improvement. Serial peak-flow monitoring at and off work suggested work-related asthma.

In May 1998, we performed specific inhalation challenge (SIC) in the laboratory by the method proposed by Cockcroft et al. (4).

On a control day, exposure to nebulized phosphate-buffered saline (Wright nebulizer, output=0.14 ml/min) for 8 min was not followed by any significant change in FEV_1 ; methacholine PC_{20} at the end of the day was 0.7 mg/ml. The next day, the subject was exposed to nebulized latex extract (Stallergènes, Paris, France) in increasing doses, starting with a dilution of 1/16 for 60 s up to the undiluted extract for 30 s (the undiluted extract caused a 4×4 mm wheal on SPT and contained 100 IR/ml of latex allergen). This exposure induced a dual asthmatic reaction with an immediate fall in FEV_1 of 20.5% at 20 min after exposure with complete recovery at 50 min, followed by a 16.6% fall at 5.5 h with complete recovery after inhaled salbutamol. The next morning, when her FEV_1 was 22% lower than the previous baseline, methacholine PC_{20} was significantly reduced (0.07 mg/ml). Respiratory symptoms were exacerbated for several days with nocturnal awakenings in the 3 days following SIC; she was given fluticasone 2000 µg/day. By 10 days after exposure, FEV_1 had returned to normal ($FEV_1/FVC=2.86/3.32$), and methacholine PC_{20} was 0.25 mg/ml.

OA to latex is reported increasingly in health-care workers (5) and in other industrial environments (6, 7). We describe the case of latex allergy in a seamstress who developed rhinoconjunctivitis and asthma after sewing elasticized ribbons. Recently, latex allergy was demonstrated in 2/10 workers in an elasticized ribbon-manufacturing plant, one of them experiencing work-related asthma confirmed by SIC (1). These workers were sensitized by handling powdered natural rubber, like health-care workers who wore powdered natural rubber gloves, where the latex allergen is adsorbed to the air-suspended powder. Our subject was exposed to rubber in a form that is not usually sensitizing by inhalation; however, sewing the elasticized ribbons induced a fine white dust of cloth and latex that could be inhaled, explaining the route of sensitization.

This case illustrates that one should be mindful of the possibility of latex allergy in different occupational settings.

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Specific immunotherapy for occupational latex allergy

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