

Letter

Occupational asthma caused by sensitization to a cleaning product containing triclosan

A recent consensus statement has highlighted the increased risk of developing asthma after exposure to workplace and domestic cleaning products.¹ Specific cleaning agents, such as benzylalkonium chloride, have been recognized as causes of occupational asthma by sensitization and now comprise a significant burden of work-related asthma. European Respiratory Society guidelines recommend that sensitizer-induced occupational asthma should be diagnosed by identifying the workplace as the cause and confirming sensitization to an asthmagen by specific inhalation challenge (SIC) in the absence of any available specific IgE tests.² We present the first case of occupational asthma caused by the biocide triclosan, commonly used in cleaning and personal care products.

A 26-year-old woman presented with a 7-month history of cough, wheeze, and chest tightness that became progressively worse throughout the working week and better on days away from work. She required treatment with a budesonide-formoterol inhaler, 400/12 μ g twice a day. She had childhood eczema and asthma and previous anaphylaxis caused by peanuts at the age of 16 years. She had been employed as a nursery nurse for 12 months, caring for 30 children aged 2 to 3 years. Her work tasks involved mopping using 5% trisodium nitrilotriacetate (Versatile) cleaner daily and cleaning tables using liquid cleaner containing 0.05% triclosan (Antibac; pH 7.0). She used latex gloves and a gel containing 70% ethanol for hand cleaning (Proform, Gompels Healthcare Ltd, Melksham, United Kingdom). Clinical examination and chest radiographic findings were unremarkable, and spirometry results were normal (forced expiratory volume in 1 second [FEV₁], 3.55 L [103% predicted]; forced vital capacity [FVC], 4.05 L [103% predicted]). Total IgE level was less than 2 kU/L, the results of testing for serum specific IgE to latex were negative, and the eosinophil count was 0.02×10^9 /L. Two-hourly peak flow measurements made at home and work for 4 weeks were analyzed using the OASYS computer program.³ The OASYS score was 3.5 and the area between the curves score was 27 L/min per hour, both confirming a significant work effect. The patient underwent SIC testing, with negative control challenges to both the hand gel and 5% trisodium nitrilotriacetate cleaner (diluted to 1:100). She was subsequently exposed to triclosan surface cleaner diluted to 1:60 with water by painting on cardboard for a total of 30 minutes and experienced a sustained, immediate decrease in FEV₁ by 24% from a baseline of 3.42 L (Fig 1), consistent with sensitization.⁴ There was no clinically significant change in fractional exhaled nitric oxide (FeNO) (pre-SIC, 9 ppb; 24 hours

after SIC, 9 ppb); all measurements were performed according to European Respiratory Society/American Thoracic Society guidelines.⁵ Nonspecific bronchial reactivity (NSBR) by methacholine challenge, using the Jaeger dosimeter, was normal (provocation dosage that caused a decrease in FEV₁ of 20%, >2.1 mg) before and 24 hours after SIC. After the diagnosis of occupational asthma, the patient remained employed in the same role, the liquid cleaner containing 0.05% triclosan (Antibac) cleaner having been substituted in the workplace. She remained relatively symptom free with 400/12 μ g of inhaled budesonide-formoterol twice a day, and spirometry performed 24 months after diagnosis revealed an FEV₁ of 3.24 L (96% predicted) and an FVC of 3.87 L (100% predicted). A further serial peak expiratory flow analysis at this time revealed an OASYS score of 2.5 and an ABC score of -2 L/min per hour.

Triclosan (2,4,4'-trichloro-2'-hydroxydiphenyl ether) is a stable, lipophilic, chlorinated diphenyl ether, which has broad-spectrum antimicrobial properties against bacteria, particularly gram-positive ones, including methicillin-resistant *Staphylococcus aureus*, and against yeasts and dermatophytes. In the United States, United Kingdom, and elsewhere, it is used at concentrations of up to 0.5% as a biocide in consumer products, such as household cleaners, shampoos, deodorants, and toothpaste, and in higher concentrations (<3%) as an antimicrobial cream (Logamel). A US marketplace study found that 46% of all commercial soaps contained triclosan.⁶ Contact allergy to triclosan has been reported sporadically since the 1970s, particularly with concentrations greater than 1%,⁷ as well as a case of immediate hypersensitivity to toothpaste (containing <1% triclosan) causing urticaria and angioedema.⁸ In addition, there is epidemiologic evidence of an association between triclosan exposure in children and the presence of clinical allergy or hay fever.⁹

It is necessary to account for aspects of the SIC. Baseline NSBR was normal, perhaps because of the patient being away from exposure to the causative agent for 3 days before SIC testing and using inhaled corticosteroids; moreover, the evidence of changes in NSBR after immediate reactions to any agent is not established. Baseline FeNO measurement was normal and remained unchanged after SIC to the liquid cleaner containing 0.05% triclosan. The value of FeNO as a marker of eosinophilic airway inflammation in occupational asthma remains unclear.² It is likely to be of less diagnostic value after SIC to low-molecular-weight chemical molecules, such as triclosan, where non-IgE-mediated immunologic mechanisms are implicated and the presence of eosinophilic airway inflammation is less prevalent.¹⁰ Clinicians should be vigilant for inhalational exposures to such biocide-containing products in the home and at work.

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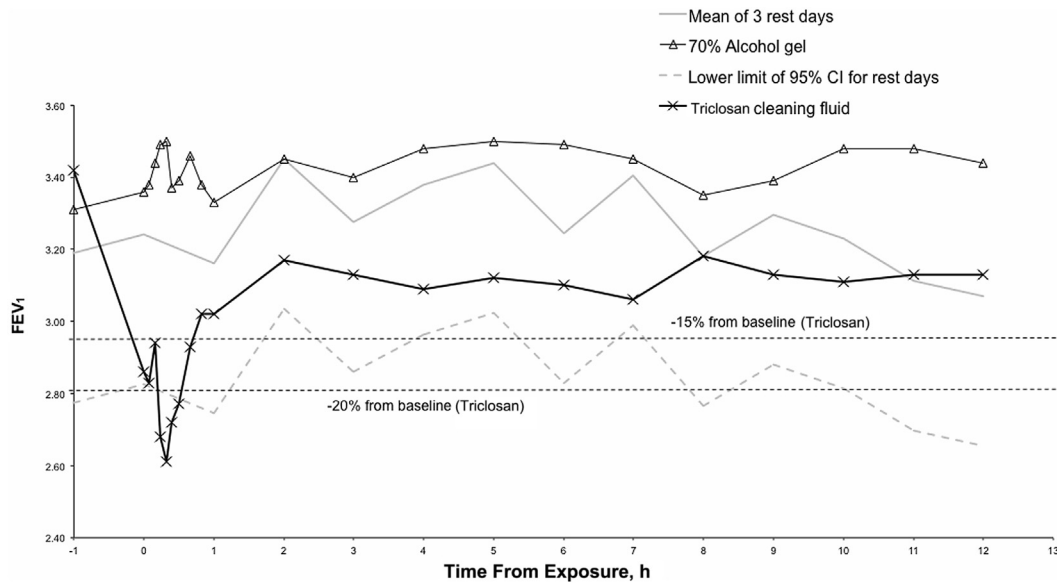


Figure 1. Specific inhalational challenge to cleaning products. A control challenge to 70% alcohol gel by painting onto cardboard resulted in no decrease in forced expiratory volume in 1 second (FEV₁) from baseline. Challenge to liquid cleaner containing 0.05% triclosan (Antibac) resulted in an immediate and sustained asthmatic reaction with a 24% decrease in FEV₁ from a baseline of 3.48 L. The lower limit of the 95% confidence interval (CI) for hourly FEV₁ on 3 rest days without exposure revealed no significant late asthmatic reactions.

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