# New causes of occupational asthma

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**Purpose of review**

This review focuses on new causative agents of occupational asthma published in 2009 and 2010. The recent developments in the diagnostic tools employed in occupational asthma caused by new agents are summarized.

**Recent findings**

Work exposures are a significant contributor to the burden of asthma and, therefore, there is great scientific interest in work-related asthma. Although the majority of cases probably represent work-aggravated asthma, in a relevant proportion of cases asthma is actually caused by one or more agents present in the workplace. The list of causative agents of immunologically mediated occupational asthma is continuously growing, and new agents and professions are described each year. New insights gained in occupational asthma include routine evaluation of airway inflammation and identification of allergenic triggers using molecular diagnosis.

**Summary**

A variety of novel high and low-molecular-weight agents have been shown to induce occupational asthma. Apart from the identification of the allergenic sources implicated, molecular diagnosis and detailed characterization of the culprit allergens contributes to increase our knowledge into the pathogenic mechanisms of this disorder. Assessment of airway inflammation helps to confirm the diagnosis and to better understand the physiopathology of the different types of occupational asthma.

**Keywords**

allergens, causative agents, occupational asthma, specific inhalation challenge

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## Introduction

Work exposures are a significant contributor to the burden of asthma. The median proportion of adult cases of asthma attributable to occupational exposure is 10–25% [1]. In a population-based study [1], a significant excess asthma risk was seen after exposure to substances known to cause occupational asthma. It has also been reported that occupation contributes to approximately one in seven cases of severe exacerbation of asthma in a working population [2•]. The high-risk occupations and industries associated with the development of occupational asthma vary depending on the predominant industrial sectors in a particular country [1,3].

Work-related asthma can be broadly defined as occupational asthma or asthma caused by specific agents in the workplace, and work-aggravated asthma [4]. There are two major forms of occupational asthma: allergic asthma characterized by a latency period; and irritant-induced asthma characterized by rapid onset of asthma following single or multiple exposures to high concentrations of irritant compounds. Although the majority of cases probably represent work-aggravated asthma, in a relevant proportion of cases asthma is actually caused by one or more agents present in the workplace [5]. Agents that cause occupational asthma can be divided into two groups according to their molecular weight: agents of high and low molecular weights.

Recent developments in the field of sensitizer-induced occupational asthma include routine incorporation of inflammation assessment into the diagnostic work-up [6•,7], and implementation of molecular diagnosis with native or recombinant allergens, which makes it possible to identify potential disease-eliciting molecules [8•,9].

## High-molecular-weight agents

High-molecular-weight agents are protein-derived antigens that cause sensitization through an immunoglobulin E (IgE)-mediated mechanism.

### Food and fishing industry

Cartier [10] has reviewed the various foods, food additives, and contaminants that have been associated with occupational asthma. Lucas *et al.* [11] have reviewed occupational asthma in the commercial fishing industry,

indicating that it more commonly occurs due to crustaceans, but molluscs and fin fish are implicated as well [11].

Rosado *et al.* [12] described the first case of occupational asthma from aerosolized octopus allergens in a seafood processing worker. Immunoblotting revealed IgE-binding bands of 43 and 32 kDa that could correspond to tropomyosin (38–40 kDa) as the responsible allergen for the sensitization.

Pérez Carral *et al.* [13] reported three workers at the same fish farm who experienced rhinoconjunctivitis and asthma while classifying fish by size. All three patients were sensitized to turbot and could eat this fish. The allergens were parvalbumin in one case and a different allergen in the remaining two patients.

Occupational asthma caused by exposure to the fish and cephalopod parasite *Anisakis simplex* through an IgE-mediated mechanism has also been reported among fish processors [14,15].

### Cereal and vegetal-derived allergens

In the past years, purified wheat proteins either in natural or recombinant forms have been implicated in the pathogenesis of baker's asthma.

Palacin *et al.* [16•] reported that recombinant wheat lipid transfer protein (Tri a 14) is a potential tool for baker's asthma diagnosis. Tordesillas *et al.* [17] found that Tri a 14 and its homologous allergen Pru p 3 (peach non-specific lipid transfer protein) have a similar conformational region involved in IgE-binding, although their electrostatic features are different. Common and specific sequential IgE-binding regions were mapped in both allergens, leading to a variable degree of cross-reactivity [17].

Lehto *et al.* [18•] carried out a study on 20 patients with baker's rhinitis, asthma, or both who were sensitized to wheat flour. They found that 12 patients reacted to α-amylase inhibitor, 9 to peroxidase, 9 to thaumatin-like protein, and 6 to lipid transfer protein 2G.

Constantin *et al.* [8•] analysed the IgE reactivity profiles of patients suffering from baker's asthma, wheat-induced food allergy and grass pollen allergy to micro-arrayed recombinant wheat flour allergens and grass pollen allergens. They identified recombinant wheat flour allergens which are specifically recognized by patients suffering from baker's asthma. Profilin was identified as a cross-reactive allergen recognized by patients suffering from baker's asthma, food and pollen allergy.

Miedinger *et al.* [19] reported for the first time a case of IgE-mediated occupational asthma to malt in a machine operator for a malt manufacturing company, and a case of hypersensitivity pneumonitis in a longshoreman who was initially referred for possible occupational asthma. Specific inhalation challenge (SIC) helped to differentiate occupational asthma from hypersensitivity pneumonitis caused by malt.

Kim *et al.* [20] reported three cases of occupational asthma caused by exposure to rice powder in the work environment. All three patients showed positive skin prick tests (SPTs) and IgE determinations to rice extract, and SIC induced immediate and late asthmatic responses.

Pirson *et al.* [21] described the case of a patient working in a factory producing inulin from chicory who developed rhinoconjunctivitis and asthma to the dust of dry chicory roots. A SIC with dry chicory elicited acute rhinoconjunctivitis and an early asthmatic response. SPT results were positive to birch pollen and fresh/dry chicory, and negative for inulin. Specific IgE to rBet v 1 was strongly positive. IgE immunoblotting with chicory extract showed that the main immunoreactive band corresponded to a protein of approximately 17 kDa, and this immunoreactivity was inhibited with purified Bet v 1. This case documents occupational rhinoconjunctivitis and asthma due to IgE sensitization to inhaled chicory allergens, including one identified for the first time as a 17-kD Bet v 1 homologous protein.

Marigold flour is prepared from the flowers of *Tagetes erecta* or the flowers of *Calendula officinalis*, plants belonging to the Compositae family. Marigold flour has been extensively used by the food additive industry as poultry feed colorant. Lluch-Pérez *et al.* [22] reported the first case of IgE-mediated occupational allergy (rhinitis and asthma) to marigold flour. It was demonstrated by SPT, nasal challenge test and specific IgE determination. A 60 kDa IgE-binding band was observed by immunoblotting, and cross-reactivity between extracts from marigold flour and *H. annuus* pollen was demonstrated.

### Laboratory animals

Jeal and Jones [23] reviewed the burden of rodent allergy and the complex exposure–response relationships between allergen exposure and sensitization and asthma, as well as risk factors for rodent allergy and mechanisms of tolerance.

De las Heras *et al.* [24•] have described a biologist who developed rhinitis and occupational asthma when she worked with gerbils (*Meriones unguiculatus*). A new gerbil allergen of 23 kDa was identified in the gerbil urine, epithelium, hair and airborne samples. Partial characterization of this allergen suggested that it possibly was a lipocalin.

Bos d 6, bovine serum albumin (BSA) is a major allergen in beef and a minor allergen in milk. It is also commonly used in research laboratories. Choi *et al.* [25] reported a case of occupational asthma and rhinitis in a laboratory worker caused by the inhalation of BSA powder, in which an IgE-mediated response was demonstrated.

#### Pests and arthropods

Occupational asthma caused by the mold *Chrysonilia sitophila* (asexual state of *Neurospora sitophila*), which was previously reported in the lodging industry, has been recently shown to affect also workers in the coffee industry. Occupational asthma has been demonstrated by SPT, serial peak expiratory flow (PEF) measurements, and IgE analyses [26–28].

A case of chronic cough related to occupational asthma with sensitivity to dry sausage mould (*Penicillium nalgiovensis*) has been reported in a worker in a semi-industrial pork butchers' [29]. The diagnosis was based on positive SPT, spirometry, and a favourable outcome after avoidance of the allergen.

Miedinger *et al.* [30] have described the case of an engineer who worked for an electric power company who developed occupational asthma due to caddis flies (Phryganeiae) confirmed by a SIC using an extract of these insects.

*Amblyseius californicus* has been recently added to the list of predatory mites that induced IgE sensitization and occupational asthma among greenhouse workers [31].

#### Wood

Campo *et al.* [32] evaluated the frequency of work-related specific sensitization and respiratory symptoms in carpentry apprentices with exposure to wood dust and diisocyanates. SPT to a panel of 14 different woods were performed in 101 apprentices. Sensitization to wood was detected in 9% of participants, all of whom were atopic with a history of rhinitis, and two of them were asthmatics. Seven apprentices showed a positive SPT reaction to olive tree wood, one to obeche wood, and one to pine tree wood.

Chengal is a resistant rainforest hardwood that is commonly used in South-East Asia. Exposure to chengal wood dust can lead to occupational asthma and rhinitis of uncertain mechanism [33].

Cabreuva (*Myrocarpus frondosus*) is a hardwood-flooring Amazonian tree that belongs to the Fagaceae family. Pala *et al.* [34•] reported the case of an atopic man employed as a parquet floor layer who developed occupational rhinitis and asthma caused by this wood. SPT with cabreuva wood dust was negative. SIC elicited rhinitis and a dual asthmatic response. IgE reactivity toward a 75 kDa protein and a positive basophil activation test strongly suggested a role of IgE in cabreuva wood-induced respiratory allergy.

Kespohl *et al.* [35] determined the prevalence of specific IgE sensitization to extracts from beech and pine wood among woodworkers, and tried to discriminate whether IgE binding was targeted to peptidic or carbohydrate pine and beech wood epitopes. They measured serum IgE reactivity to three cross-reactive carbohydrate determinants (CCDs) markers: horseradish peroxidase, bromelain and MUXF3. In many patients with positive IgE to beech wood or pine wood, IgE binding was significantly inhibited by horseradish peroxidase, suggesting that IgE binding was mostly directed to carbohydrate structures. In some sera, inhibition was partial, indicating that the sera recognized peptidic as well as carbohydrate IgE epitopes. In only a few sera, no inhibition by horseradish peroxidase was observed, indicating that IgE-mediated binding was exclusively based on peptidic epitopes.

Cross-reactive carbohydrate determinants are complex glycans present in glycoproteins from plant and some invertebrate animals (i.e. insects). Specific IgE antibodies to CCDs can be highly cross-reactive and frequently occur in patients polisensitized to common aeroallergens, thus complicating the diagnosis of occupational allergy [36•].

## Low-molecular-weight agents

LMW chemicals are incomplete antigens (i.e. haptens) that must bind to autologous or heterologous proteins to become immunogenic. New LMW agents are continuously recognized as inducing OA, in most cases through an IgE-independent mechanism.

#### Vancomycin

A 33-year-old man, employed in a pharmaceutical industry, developed rhinorrhoea, cough, dyspnoea and chest discomfort at work, which consisted of purifying vancomycin to manufacture into its powder form [37]. The diagnosis of vancomycin-induced occupational asthma was based on clinical history, work-related symptoms, and increased PEF variability at the workplace. The authors suggest that a direct histamine releasing effect by vancomycin, and not an IgE-mediated mechanism, is the pathogenic mechanism of occupational asthma due to vancomycin.

#### 5 aminosalycilic acid

A 56-year-old man complained of cough, dyspnoea and wheezing 1 month after beginning to work in manufacturing a drug containing 5 aminosalycilic acid (5-ASA). SPT with 5-ASA (10 mg/ml) was negative. Methacholine

test was negative, fractional exhaled nitric oxide (FeNO) was 32 ppb and induced sputum showed no eosinophils at baseline. A SIC to 5-ASA (5% in lactose) at a mean concentration of 2.65 mg/m$^3$ for 30 min elicited a late asthmatic reaction. Twenty-four hours after the challenge, methacholine $PC_{20}$ was 10 mg/ml, FeNO 53 ppb and induced sputum showed 65% eosinophils [38].

#### Colistin

An atopic 24-year-old man working in a pharmaceutical company transporting and storing raw material developed occupational rhinitis and occupational asthma to colistin [39]. Three months after starting his current job, he developed rhinitis, which improved over the weekends. Nine months after the onset of rhinitis, exposure to colistin caused him to suffer sudden cough, wheeze, and dyspnoea. The SIC confirmed the diagnosis of occupational asthma and rhinitis to colistin. Specific IgE was not detected.

#### Cephalosporin intermediate product

Pala *et al.* [40] reported the first case of occupational rhinitis and occupational asthma due to 7-Amino-3-thiomethyl-3-cephalosporanic acid (7-TACA), an intermediate product in cephalosporins synthesis. A 44-year-old pharmaceutical company worker, assigned to cephalosporins product line, developed sneezing, dry cough and shortness of breath. SPT with 7-TACA was not performed because the compound was not soluble in saline. A SIC with exposure to 0.5% 7-TACA mixed with 99.5 mg of lactose in a 7.46 m$^3$ challenge room elicited an early asthmatic reaction and rhinitis. Nasal eosinophils increased 4 h after challenge but no change in induced sputum eosinophils was observed before and after SIC. Methacholine challenge test was negative before and after SIC.

#### Turpentine

A case of a 27-year-old art painter using turpentine as a thinner for oil-based paints who developed asthmatic reactions after 5 years of working with turpentine has been described [41]. Turpentine is a fluid obtained by distillation of wood resins containing a mixture of terpenes. It is a known agent inducer of contact dermatitis. A SIC showed a late phase asthmatic reaction and an increase of eosinophils in sputum 24 h after the challenge.

#### Iron welding fumes

Muñoz *et al.* [42] described three patients with occupational asthma secondary to exposure to welding fumes generated during metal arc welding on iron. The exposure time ranged from 7 to 43 years and the time of the onset of symptoms following the start of exposure was 2–12 years. Patients were diagnosed by SIC. Environmental levels of Fe, Cd, Cu, Cr, Ni, $NO_2$, NO, CO, and O produced during the SIC did not exceed threshold limit values. Samples of induced sputum were obtained before and after the SIC and showed an increase in neutrophils and concentrations of IL-8, TNF-alpha and TNF-beta after the SIC.

#### Dodecanedioic acid

Moore *et al.* [43] reported the first case of occupational asthma due to electronic colophony-free gel flux predominantly containing dodecanedioic acid. The patient worked as an electronics instructor and occupational asthma was demonstrated by serial PEF measurements and by a positive SIC with dodecanedioic acid fluxes, whereas the SIC was negative to the colophony wire and wire containing predominantly palmitic acid. Dodecanedioic acid has an asthma hazard index of 0.94. The asthma hazard index is a quantitative structure–activity relationship for prediction of the likelihood (hazard index between 0 and 1) that a chemical has a asthmagenic potential (a value >0.5 has a high probability of being an asthmagen) (reviewed in [44]).

#### Rhodium salts

A 27-year-old atopic operator of an electroplating plant developed work-related shortness of breath and runny nose with sneezing after exposure to rhodium salts [45]. The patient showed positive SPT reactions and positive early asthmatic reactions with rhodium and platinum salts.

#### Trimethylolpropane triacrylate

Sánchez-Garcia *et al.* [46•] described a nonatopic 62-year-old woman who had been working for 20 years selling lotto inside a 4-m$^3$ kiosk. Over the past 3 years she had been using point of sale terminals (POS) to print lotto coupons. She had a 2.5-year history of rhinoconjunctivitis, facial oedema, cough, shortness of breath and wheezing within 30–60 min after arriving at her workplace. A SIC was performed in a 7-m$^3$ chamber; the patient painted on cardboard with the tint provided by the lottery company, which contained trimethylolpropane triacrylate, and an isolated early asthmatic reaction was observed. Twenty-four hours after the challenge, methacholine $PC_{20}$ was 1.68 mg/ml (baseline $PC_{20}$ 6.18 mg/ml) and FeNO 22 ppb (baseline 14 ppb), and induced sputum showed a 100% increase in eosinophils. An occupational type challenge was performed using the patient's own POS and printing coupons, and again an early asthmatic reaction developed. After being away from her work environment she was asymptomatic without any medication. The patient had developed occupational asthma induced by an acrylate released from thermal activated paper used in POS.

Occupational asthma caused by acrylate compounds in different work settings and new professions is increasingly being reported. For instance, rhinitis, asthma and

other respiratory disorders have been recently described among nail technicians, particularly artificial nail applicators [47–49].

### Triglycidyl isocyanurate

New insights into occupational asthma caused by triglycidyl isocyanurate (TGIC) have been gained. A 28-year-old woman developed work-related asthma symptoms when aluminium frames were treated with an electrostatic powder paint containing 2.5–10% TGIC [50]. Occupational asthma was confirmed by serial PEF measurements and methacholine test. A SIC with TGIC (4% in lactose) at a mean concentration of 3.61 mg/m$^3$ for 15 min induced an isolated early asthmatic response. No IgE to TGIC was detected by ELISA testing and no IgE-binding bands were found by immunoblot analysis.

Anees *et al.* [51] reported six workers exposed as bystanders to heated TGIC who developed occupational asthma confirmed by serial PEF measurements. SIC testing resulted in late or dual asthmatic reactions to heated TGIC in four of four tested and was negative in three control asthmatics. One worker tested only with unheated TGIC had a negative SIC test. Thus, heated TGIC can cause OA from bystander exposure.

## Conclusion

New insights gained in occupational respiratory allergy include routine evaluation of airway inflammation and identification of allergenic triggers using molecular diagnosis. A variety of novel high and low-molecular-weight agents have been shown to induce occupational asthma. Apart from the identification of the allergenic sources implicated, molecular diagnosis and detailed characterization of the culprit allergens, as well as elucidation of the patterns of airway inflammation involved, contribute to increase our knowledge into the pathogenic mechanisms of this disorder. These advances may help to develop better preventive and therapeutic strategies.

## References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
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Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 150–151).


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