

Occupational asthma caused by acrylic compounds from SHIELD surveillance (1989–2014)

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Background	Acrylic monomers (acrylates), methacrylates and cyanoacrylates all cause asthma by respiratory sensitization. Occupational inhalation exposures occur across a variety of industries including health care and dental work, beauty, laboratory science, assembly and plastic moulding.
Aims	To examine notifications of occupational asthma caused by acrylic compounds from a UK-based regional surveillance scheme, in order to highlight prevalent exposures and trends in presentation.
Methods	Retrospective review of all cases reported to the SHIELD surveillance scheme for occupational asthma, West Midlands, UK between 1989 and 2014. Patient data were gathered on demographics, employment, asthma symptoms and diagnostic investigations including serum immunological testing, serial peak flow analysis and specific inhalation challenge tests. Descriptive statistics were used to illustrate worker characteristics and evidence for sensitization to acrylic compounds.
Results	There were 20 affected patients out of 1790 total cases of occupational asthma (1%); all cases were confirmed by OASYS (Occupational Asthma SYStem) analysis of serial peak flow measurements, with three additional positive specific inhalation challenge tests. Three out of 20 (15%) patients were current smokers and 11/20 (55%) were atopic. A variety of exposures and industries were implicated including: manufacturing, health care, beauty and printing and a novel presentation seen in teachers exposed to floor adhesives.
Conclusions	This is the largest reported series of occupational asthma caused by acrylic compounds, which remain an important aetiological factor in this disease. Exposure occurs in a variety of industries, particularly in manufacturing and is seen with other, perhaps better recognized sensitizing agents such as isocyanates and epoxy resins.
Key words	Acrylate; acrylic; asthma; cyanoacrylate; hypersensitivity; methacrylate; occupational asthma.

Introduction

Acrylates (acrylic monomers) are esters of acrylic acid, containing a vinyl group (two double-bonded carbon atoms) directly bonded to a carbonyl group (Figure 1); these readily polymerize to form plastics known as ‘acrylics’ or ‘poly-acrylates’ that have been used variously in industry since the 1930s as adhesive resins, surface coatings, synthetic textiles, printing ink and hard plastic. Methacrylates are esters of methacrylic acid and the extra methyl- group reduces polymer chain mobility, increasing structural rigidity. The most commonly produced is methyl methacrylate, used in the manufacture of poly methyl methacrylate (PMMA) hard plastic, which has traditionally been used for hard contact lenses, transparent glass (Perspex, Plexiglass) and plastic balls for extra-pleural plombage (Lucite). Along with newer

derivatives [for example, ethylene glycol di-methacrylate (EGDMA), 2-hydroxyethyl methacrylate (2-HEMA)], methyl methacrylate has also found application commercially as dental filler and adhesive, orthopaedic bone cement, histological fixation medium and UV-curable gel for nail extensions. Cyanoacrylates are chemically distinct from other acrylates and methacrylates and contain a cyanide group. They readily polymerize in the presence of small quantities of water or humid air and form a strong bond between various materials. Cyanoacrylates have been produced commercially since the 1950s as instant glues including the simplest homologue methyl cyanoacrylate (Loctite Superglue), and as surgical tissue adhesives.

Skin sensitization to cyanoacrylate and methacrylate was reported as early as the 1940s [1] and is described in assembly workers [2], dental personnel [3] and nail

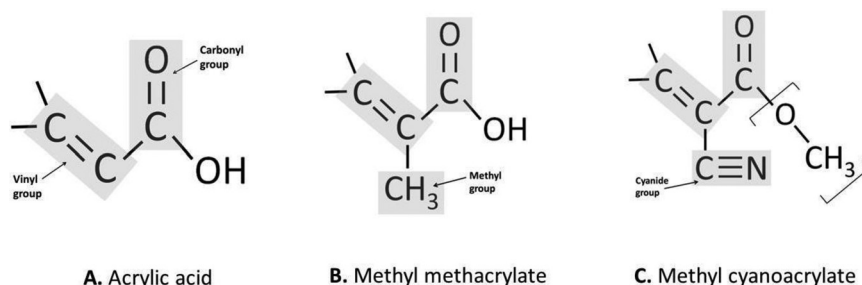


Figure 1. Structure of acrylic acid and related compounds. (A) Acrylic acid comprises a vinyl group attached to a carbonyl group. (B) Methyl methacrylate is the simplest isomer of methacrylic acid in which a methyl group replaces the vinyl hydrogen ion. (C) Methyl cyanoacrylate contains a cyanide molecule, which replaces the vinyl hydrogen ion, and is produced commercially as Loctite Superglue.

technicians [4]. Most allergic contact dermatitis manifests as acute or chronic finger pulpitis and is associated with positive patch tests and negative skin prick tests (SPTs) to acrylic- or albumin-conjugated-acrylic compounds [5,6]. Cyanoacrylate monomers have lower tendency to vaporize in conditions of high atmospheric humidity because they react with water vapour to form less volatile polymers [7], so it has been suggested that the less humid the working environment the greater the risk of sensitization to cyanoacrylates [8]. Higher homologues (ethyl- and butyl-) are less volatile than methyl cyanoacrylate, but this reduces their bond strength, making them less desirable as adhesives [9].

Case reports and a number of series of respiratory sensitization to acrylic compounds have been published (Table 1), implying exposure for a number of occupations: operating theatre staff [10], optical laboratory technicians [11], assembly and manufacturing operatives [8,12], beauticians [5,13], automotive repairers [7], printers [14], a graphic arts worker [15], a secretary [16], a plumber [17], dental professionals [6] and model makers [18,19]. We aimed to describe all SHIELD notifications of occupational asthma caused by sensitization to acrylic compounds (1989–2014), in order to illustrate common presentations and highlight new occupational exposures.

Methods

Newly diagnosed workers in the West Midlands with occupational asthma have been prospectively notified to the Midland Thoracic Society's voluntary surveillance scheme (SHIELD), by regional respiratory and occupational physicians, since 1989 [20]. Cases are notified when a clinical diagnosis of occupational asthma is more likely than not (i.e. on balance of probability), provided there is evidence of asthma with deterioration related to occupational exposure to an asthmagen, and a symptomless latent interval; work-exacerbated asthma is excluded. No specific tests are required although 70% of all SHIELD notifications are based on serial peak expiratory flow (PEF) measurements, 46% on allergen-specific

immunoglobulin E and 11% on specific inhalational challenge (SIC) testing [21].

All cases of occupational asthma notified to SHIELD since 1989 were retrieved from a Microsoft Excel database at the Heart of England NHS Foundation Trust, containing all diagnoses of occupational lung disease made by the Birmingham Occupational Lung Disease Service. These were hand-searched to identify all notifications where the causative agents were acrylates, methacrylates, cyanoacrylates or acrylic co-polymers. All cases were cross-checked with Heart of England NHS Foundation Trust medical records to eliminate transcription error.

For each case identified, demographic data (age, gender, smoking history, atopic status) and exposure data (occupation, causative agent, latency of exposure) were gathered, as well as information about diagnostic tests: (i) OASYS (Occupational Asthma SYStem) software analysis of two-hourly PEF measurements utilizing both OASYS score [22] and 'Area between the Curves' (ABC) score [23] and (ii) SIC tests. For each illustrative case, further diagnostic information was gathered, comprising chest radiograph, non-specific bronchial reactivity, spirometry and SPT; medical records were searched for any missing data. Descriptive analysis was undertaken: parametric data were displayed using means and SD, non-parametric data using medians and interquartile range (IQR) and categorical data using percentages.

Ethical approval for the study was sought and obtained from Yorkshire and The Humber – South Yorkshire Research Ethics Committee (reference: 16/YH/0308). Written consent for publication was obtained from the illustrative cases (patients 1, 6 and 18).

Results

Twenty patients with occupational asthma caused by sensitization to acrylic compounds were diagnosed between 1 January 1989 and 31 December 2014, out of 1790 (1%) total cases of occupational asthma (summarized in Tables 2 and 3). Occupational asthma was confirmed by OASYS analysis of serial PEF measurements in all 20 patients, with positive SIC tests in

Table 1. All published case series of occupational asthma caused by acrylic compounds, showing implicated acrylic monomers and polymers, degree of atopy, latency between exposure and symptom onset and type of asthmatic reaction on SIC testing

Year	Author	Asthma cases	Atopy	Use of acrylic compound	Latency of exposure	Type of specific asthmatic reaction
1985	Lozewicz <i>et al.</i> [8] London, UK	6 cases due to: CA (5) MMA (1)	3/5 ^a	Adhesive (CA) Dental filler (MMA)	0.5–120 months	1 immediate (MMA) 3 late (CA) 2 dual (CA)
1993	Savonius <i>et al.</i> [14] Helsinki, Finland	15 cases due to: CA (10) MMA (3) AA (1) PA (1)	3/15	Adhesive (CA, MMA) Dental filler (MMA) Soft earplugs (MMA) Printing ink (AA) UV-hardening paste (PA)	0.25–166 months	3 immediate [CA (1), AA (1), PA (1)] 8 late [CA (6), MMA (2)] 4 dual [CA (3), MMA (1)]
1996	Kopferschmit-Kubler <i>et al.</i> [12] Strasbourg, France	2 cases due to CA	0/2	Adhesive	0.75–84 months	1 late 1 immediate
1998	Piirilä <i>et al.</i> [6] Helsinki, Finland	9 cases due to MMA	3/9	Dental filler	Mean 13.5 years (±8.9 SD)	2 immediate 7 late
2001	Quirce <i>et al.</i> [7] Madrid, Spain	2 cases due to CA	0/2	Adhesive	3–18 months	2 late
2008	Sauni <i>et al.</i> [13] Helsinki, Finland	2 cases due to MMA	1/2	UV-curable gel for sculptured nails	60–72 months	2 dual
2008	Roche <i>et al.</i> [5] Valencia, Spain	3 cases due to MAs	1/3	UV-curable gel for sculptured nails	12–36 months	No SIC tests. Positive dermatological patch tests to a variety of MA monomers
2011	Quirce <i>et al.</i> [11] Madrid, Spain	2 cases due to PMMA	0/1 ^a	Hard eyeglass lens	23–24 years	1 late 1 dual

AA, acrylic acid; CA, cyanoacrylate; MA, methacrylate; MMA, methyl methacrylate; PA, poly-acrylate.

^aMissing data for one patient per series.**Table 2.** Characteristics of eight patients with occupational asthma caused by predominantly methyl methacrylate

Patient	Patient demographics				Disease aspects			
	Age	Gender	Occupation	Smoker	Atopy	Latency exposure to symptom onset	Chemical	Use
1 ^a	58	Male	Plastic moulder (prosthetic limbs)	Never	Yes	15 years	Methyl methacrylate	Laminating carbon- and glass-fibre
2	47	Male	Orthopaedic theatre practitioner	Never	Yes	20 years	Methyl methacrylate	Open bowl mixing of bone cement
3	29	Male	Fabricator of aluminium tanks and radiators	Ex	No	7 years	Methyl methacrylate	Adhesives
4	51	Male	Injection moulder acrylic polymers	Never	No	1 year	Methyl methacrylate	Plastic granules injection moulding
5	36	Female	Nail technician	Never	Yes	8 years	Methyl methacrylate, cyanoacrylate	Light-curing gel nails
6 ^a	47	Female	Orthopaedic theatre nurse	Never	No	14 years	Methyl methacrylate	Orthopaedic bone cement
7	42	Female	Dentist	Never	Yes	10 years	Methyl methacrylate, cyanoacrylate	Dental filler
8	55	Female	Midwife	Never	Yes	9 years	Methyl methacrylate	Tissue adhesive spray

^aDiagnosis confirmed with SIC as well as serial PEF measurements.

Table 3. Characteristics of 12 patients with occupational asthma caused by acrylates, acrylic polymers and cyanoacrylates

Patient demographics						Disease aspects		
Patient	Age	Gender	Occupation	Smoker	Atopy	Latency exposure to symptom onset	Chemical	Use
9	43	Female	Assembly of rubber ring seals for traffic lights	Ex	Yes	3 years	Cyanoacrylate	Adhesive
10	38	Female	Assembly of plastic car parts	Current	No	18 months	Cyanoacrylate	Adhesive
11	49	Male	Assembly of wire cables	Never	Yes	18 months	Cyanoacrylates	Adhesive
12	54	Female	Secondary school teacher	Current	No	10 months	Alkyl-cyanoacrylate	Floor tile adhesive
13	49	Male	Office work for manufacturing company	Never	No	6 months	Alkyl-cyanoacrylate	Floor tile adhesive
14	51	Male	Injection moulder acrylic polymers	Ex	No	3 years	Acrylic co-polymer	Plastic extrusion
15	55	Male	Printer	Never	No	6 months	Multifunctional acrylates	UV-curable printing ink
16	25	Female	Teaching assistant	Ex	Yes	5 months	Alkyl-cyanoacrylate	Floor tile adhesive
17	52	Female	Assembly of wire cables	Never	Yes	Unknown ^b	Cyanoacrylate	Adhesive
18 ^a	47	Female	Teacher	Never	No	8 months	Acrylic co-polymer (Styccobond)	Floor tile adhesive
19	40	Female	Assembly of air compressors	Current	Yes	3 years	Cyanoacrylate	Loctite adhesive
20	51	Male	Paper coating operative	Ex	Yes	26 years	Multifunctional acrylates	UV-curable coating

^aDiagnosis confirmed with SIC as well as serial PEF measurements.^bPoor perceiver of symptoms.

the three patients described earlier. Eleven out of 20 patients (55%) were female and mean age at diagnosis was 46 years (SD = 8.8). Three out of 20 (15%) patients were current smokers and 11/20 (55%) were atopic. The median latency from exposure to acrylic compounds to onset of symptoms was 36 months (IQR = 11.5–114). A variety of industries were implicated: manufacturing ($n = 11$), health care ($n = 4$), education ($n = 3$), hair and beauty ($n = 1$) and printing ($n = 1$). Agents containing acrylic compounds were commonly adhesives ($n = 11$), but also emulsions/coatings ($n = 3$), bone cement ($n = 2$), printer ink ($n = 1$), nail extensions ($n = 1$), dental filler ($n = 1$) and injection moulding substrates ($n = 1$).

Patient 1

A 58-year-old male presented to the occupational lung disease clinic with chest tightness and wheeze of 6 years duration. Onset of symptoms was within 1 week of returning to work, following 12-month sickness absence due to injuries sustained in a road traffic collision. His symptoms were worse first thing in the morning and towards the end of a working day, and symptoms were better after a weekend away from work. He had also developed rhinitis and asthma symptoms on exposure to cats and grass pollen. He had been employed as a manufacturer of prosthetic limbs for 14 years prior to prolonged sickness absence, and his work involved two

main processes: (i) making laminated prosthetic legs from glass- or carbon-fibre, mixed with a thermoplastic Orthocryl resin containing methyl methacrylate, and (ii) making and cutting Pedilen rigid-foam body compensations, using a hardener containing methylene diphenyl diisocyanate (MDI). In the outpatient clinic, and while still exposed at work, chest radiograph was normal, exhaled nitric oxide (FE_{NO}) = 43 ppb, forced expiratory volume in 1 s (FEV_1) = 3.74 L (113% predicted), forced vital capacity (FVC) = 4.76 L (115% predicted), $FEV_1/FVC = 79\%$, and he exhibited moderate bronchial reactivity to methacholine challenge ($PD_{20} = 0.22$ mg by the Jaeger method; normal >1.00). He had positive SPT to cat, dog, grass pollen, tree pollen, *Aspergillus*, *Penicillium* and *Cladosporium*. Two-hourly serial PEF measurements were undertaken at home and at work while exposed, and OASYS analysis revealed definite occupational asthma (OASYS score = 3.8; ABC score = 63 L/min/h). He was admitted for SIC testing and experienced a significant late asthmatic reaction and fall in FEV_1 on mixing and curing Orthocryl resin containing methyl methacrylate (Figure 2). He also had a subsequent positive SIC test to MDI. He was removed from exposure to both agents immediately and 18 months after diagnosis remained on sick leave with full pay, while the employer introduced new control measures. His spirometry remained normal and asthma symptoms were well controlled on the stated inhaled medication regime.

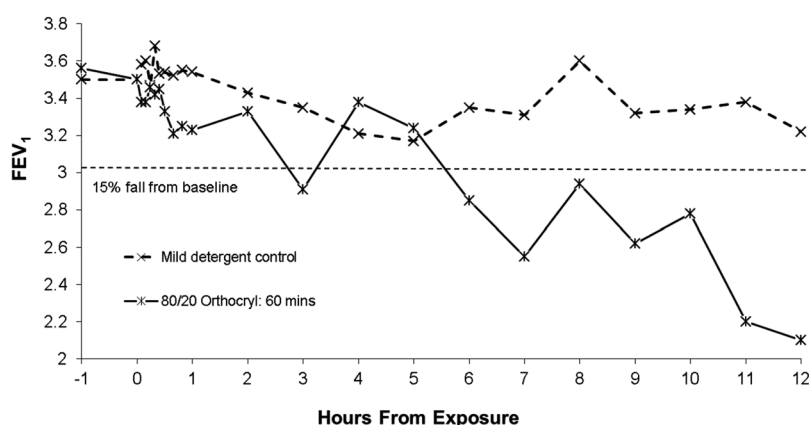


Figure 2. SIC to Orthocryl (Otto Bock 617-H19), a thermoplastic resin containing methyl methacrylate. Patient 1 underwent challenge to Orthocryl by mixing with di-benzyl peroxide hardener in 80/20 combination. Initial FEV₁ was 3.56 L and there was a sustained late falls in FEV₁ of up to 41%, to a minimum of 2.1 L at 12 h post-challenge. Initial control challenge to mixing and cleaning with benzyl-ammonium detergent was normal.

Patient 6

A 47-year-old female presented via her occupational health department with throat irritation, cough, chest tightness and breathlessness of 7 years duration. She had been employed as an orthopaedic theatre nurse for 14 years prior to onset of symptoms, which regularly occurred immediately after handling and mixing bone cement in an open bowl. Symptoms were better on days off duty, and on days where no cementing was undertaken; they were unrelated to use of latex gloves. Chest radiograph was normal, FEV₁ = 2.70 L (100% predicted), FVC = 3.30 L (105% predicted), FEV₁/FVC = 82%, and she exhibited severe bronchial reactivity to methacholine challenge (PD₂₀ = 48 mcg by the Yan method; normal >4800 mcg). She had positive SPTs to grass pollen, *Dermatophagoides pteronyssinus* and *Cladosporium*. Two-hourly serial PEF measurements were undertaken at home and at work while exposed, and OASYS analysis revealed definite occupational asthma (OASYS score = 3.1; ABC score = 25 L/min/h). On a 2-min inhalational challenge to pre-mixed bone cement containing methyl methacrylate, she experienced an immediate asthmatic reaction, with a fall in FEV₁ of 35% from 2.67 L, recovering over 2 h. She was removed from exposure and relocated to pre-operative assessment clinic. Seven years after diagnosis, her spirometry remained normal, she was maintained on inhaled treatment with flixotide/serevent 250/25 mcg two puffs twice a day, and she experienced intermittent asthma symptoms on exposure to airway irritants including chlorine-based cleaning agents.

Patient 18

A 48-year-old school teacher presented to the occupational lung disease clinic with cough, chest tightness, wheeze and breathlessness, which began 8 months

after intermittently entering newly built school premises. An increase in severity of asthma symptoms occurred on daily exposure to newly installed carpets and classroom or office furniture. At presentation, she was taking flixotide/salmeterol 125/25 mcg two puffs twice daily, salbutamol 200 mcg four-hourly and ipratropium 20 mcg two puffs twice daily. Chest radiograph was normal, FEV₁ = 1.49 L (60% predicted), FVC = 2.65 L (92% predicted), FEV₁/FVC = 56% and she exhibited severe bronchial reactivity to methacholine challenge (PD₂₀ = 45 mcg, Yan method) while exposed. Two-hourly serial PEF measurements showed definite occupational asthma (OASYS score = 4.0; ABC score = 97 L/min/h). Workplace bronchial challenge was undertaken: following a normal rest day with stable FEV₁ [baseline FEV₁ = 2.26 L (92% predicted)], she underwent a workplace challenge by sitting in a newly finished and furnished meeting room for 40 min: she experienced nasal stuffiness and wheeze within 10 min of exposure, accompanied by a 41% fall in FEV₁ which recovered over 90 min. There was a further 30% late fall in FEV₁ at 8–10 h post-challenge. A similar dual asthmatic reaction was observed after SIC to Styccobond F41 tackifier (acrylic co-polymer adhesive emulsion), by painting onto cardboard for a total of 30 min. Initial FEV₁ = 1.87 L (76% predicted) and this fell by 31% immediately post-challenge, accompanied by wheeze and chest tightness, recovering over 20 min. There was a late fall in FEV₁ by a maximum of 20%, at 8–9 h post-challenge, accompanied by nasal stuffiness, cough and wheeze. She was relocated to additional accommodation without exposure to new carpet tiles and was granted ill-health retirement 2 years later, with on-going symptoms of secondary bronchiectasis, normal spirometry [FEV₁ = 2.22 (97% predicted), FVC = 2.77 (103% predicted), FEV₁/FVC = 80%] and moderate bronchial reactivity to methacholine (PD₂₀ = 101 mcg).

Discussion

We have described 20 cases of occupational asthma caused by inhalational exposure to acrylic compounds, which comprised 1% of the total notifications to SHIELD between 1989 and 2014. All cases were confirmed by OASYS analysis of serial peak flow measurements, with positive SIC tests to either methyl methacrylate or acrylic co-polymer in three cases. Commonly implicated industries were manufacturing, health care and education, and the most frequently encountered causative agents were adhesives, surface coatings and bone cement. A novel presentation was seen in teachers indirectly exposed to floor adhesives.

The strengths of this study are that it is the largest reported series of occupational asthma due to acrylic compounds, utilizing 25 years' worth of data from a well-established UK-based reporting scheme together with workers' National Health Service medical records. Its main weakness is that respiratory sensitization has not been confirmed by SIC testing in the majority of cases, unlike other European series of this type, where perhaps SIC tests are required for compensation purposes. In the UK, objective demonstration of a work-related pattern for asthma by serial PEF analysis and a history of exposure to a sensitizing agent is usually sufficient for award of compensation under the government's Industrial Injuries Disablement Benefit scheme. All of our patients had definite work-related patterns on serial PEF analysis with 19/20 exhibiting pre-symptomatic latent periods of exposure to recognized sensitizing agents (range = 5 months to 26 years; 1/20 = unknown period), suggestive of hypersensitivity occupational asthma. SPT to acrylic compounds has not proved useful in demonstrating specific sensitization [5,6] and is therefore not routinely undertaken in our institution. The majority of previously reported occupations are represented in our series, though we have revealed novel presentations in a small outbreak comprising three patients working in education (two teachers and one teaching assistant), all of whom developed asthma after latent exposure to acrylic flooring adhesive or carpet tile tackifier, in a new-build and refurbished school premises. All three patients displayed latent asymptomatic periods <1 year (5–10 months) and one case (patient 18) exhibited a positive SIC test to acrylic co-polymer.

Despite good evidence for skin and respiratory sensitization, the underlying mechanisms remain a little unclear. Mainly dual or late asthmatic reactions have been reported to occur on SIC testing (Table 1), in predominantly non-atopic individuals, with lack of skin prick positivity to acrylic compounds or conjugates (in our series, no pattern predominated: one late, one dual, one immediate). These modes of asthmatic reaction are normally associated with immunological reactions other

than IgE mediation, and previous authors have highlighted this [6,7,13]. A small number of immediate asthmatic reactions have, however, been reported with methyl methacrylate, acrylic acid and cyanoacrylate [6,12,14], as well as immediate skin hypersensitivity to acrylate [5], thus type 1 hypersensitivity cannot be entirely excluded in susceptible individuals.

The role of atopy in occupational asthma due to low-molecular-weight agents remains controversial, with some studies reporting that atopy confers an increased risk of occupational asthma and/or specific sensitization to acrylic compounds [24], and others not demonstrating an association [14]; this certainly remains unclear for other low-molecular-weight compounds, for example isocyanates, platinum salts and acid anhydrides, where there are clear methodological inconsistencies between studies [25]. In our series, 55% of patients had pre-existing atopic disease or skin prick positivity to common aeroallergens, which is a little higher than expected from other studies.

Confirmed cases of asthma due to acrylic compounds comprised 1% of all occupational asthma notifications to SHIELD made 1989–2014, though it should be noted that 26% of all SHIELD notifications are workers employed in occupations associated with reported cases of skin and respiratory sensitization to acrylic compounds (health care and dental workers, assembly, plastic moulding, beauty, laboratory work) [21]. Exposure to acrylic compounds occurs in a variety of industrial settings where other co-existent sensitizers such as isocyanates are perhaps better recognized by workers, managers and health care professionals, and therefore sensitization may be under-appreciated. There are few data regarding the occurrence of occupational asthma in populations exposed to acrylic compounds; an early study of adhesive production workers in Puerto Rico showed that those exposed to methyl- and ethyl-cyanoacrylate at usual occupational levels were no more likely to develop obstructive lung disease than unexposed workers [26]. However, in a case-control study in an urban Swedish region, Torén *et al.* [27] showed an increased risk of asthma due to exposure to Loctite and Omnifit cyanoacrylate glues [odds ratio (OR) = 1.8]. A subsequent population-based cohort study of adults in Northern Europe using a job exposure matrix showed an increased asthma risk in non-atopic men exposed to acrylates (most commonly as dental staff, plumbers, pipe fitters, electrical and electronic equipment assemblers, mechanics); non-atopics had higher risk [hazard ratio (HR) = 3.3] than atopics (HR = 1.8) [28], and the population attributable risk for asthma caused by acrylates was 5%. Regarding methacrylates, general population-based studies in Northern Europe have shown increased risk of asthma among dental staff [29,30] and subsequently Jaakkola *et al.* [24] observed an increased risk of adult-onset asthma (OR = 2.65) and

respiratory symptoms (OR = 1.69) among dental assistants in Helsinki, Finland.

Acrylic compounds remain an important cause of asthma at work, and exposure can occur in a wide variety of industries. As their use often occurs in assembly and manufacturing settings alongside other sensitizers such as isocyanates and epoxy glues, vigilance and a detailed enquiry about the work process are required.

Key points

- The use of acrylates, methacrylates and cyanoacrylates remains commonplace in manufacturing, health care, beauty and printing industries, and inhalational exposure occurs in a variety of job roles.
- We describe cases of occupational asthma from the West Midlands SHIELD surveillance scheme caused by acrylic compounds and identify novel presentations via indirect exposure to acrylates used in floor surface coatings.
- Occupational health professionals should recognize that acrylic compounds are used widely alongside other respiratory sensitizers such as isocyanates and epoxy resins.

Conflicts of interest

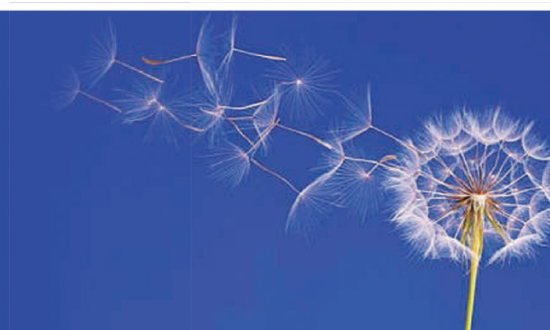
None declared.

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