Occupational asthma: Definitions, epidemiology, causes, and risk factors

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INTRODUCTION

Occupational asthma (OA) is a form of work-related asthma characterized by variable airflow obstruction, airway hyperresponsiveness, and airway inflammation attributable to a particular exposure in the workplace and not due to stimuli encountered outside the workplace [1-3]. Two types of OA are distinguished based on their appearance after a latency period: (1) OA caused by workplace sensitizers: allergic or immunological (with a latency period); (2) OA caused by irritants: nonallergic or nonimmunologic, irritant-induced asthma including reactive airways dysfunction syndrome (RADS).

Occupational asthma accounts for approximately 10 to 25 percent of adult onset asthma [4,5]. In the case of allergic OA, a high degree of clinical suspicion is needed as the asymptomatic latency period for sensitization varies from a few months to several years, depending on several factors, including the intensity of exposure, the specific sensitizing agent, and individual susceptibility.

The definition, epidemiology, causes, and risk factors of OA are reviewed here. The pathophysiology, clinical assessment, diagnosis, and management of OA and reactive airways dysfunction are discussed separately. (See "Occupational asthma: Pathogenesis" and "Occupational asthma: Clinical features, evaluation, and diagnosis" and "Reactive airways dysfunction syndrome and irritant-induced asthma".)

DEFINITIONS

Several terms are used to define the various forms of work-related asthma [6]:

- Occupational asthma (OA) begins during adulthood and is induced by exposure to immunologic or nonimmunologic stimuli found in the workplace [4].

- Work-exacerbated asthma (also known as work-aggravated asthma) is defined as preexisting or concurrent asthma that subjectively worsens in the workplace [7,8]. (See "Diagnosis of asthma in adolescents and adults".)

- Irritant-induced asthma results from single or multiple exposures to a nonimmunologic, irritant substance at a high level of intensity. When workplace exposure(s) cause or induce irritant-induced asthma, it is considered a subset of occupational asthma. However, the airway histopathology is different from immunologic OA (table 1). (See "Reactive airways dysfunction syndrome and irritant-induced asthma".)

- Reactive airways dysfunction syndrome (RADS) is the acute form of irritant-induced asthma that is triggered by a single acute high level exposure to a nonimmunologic stimulus [9]. Symptoms begin within minutes of the exposure, and the initial symptoms are followed by on-going asthma-like symptoms and bronchial hyperresponsiveness that last for a prolonged period. (See "Reactive airways dysfunction syndrome and irritant-induced asthma", section on 'Definitions'.)

- Occupational nonasthmatic eosinophilic bronchitis develops in the workplace and causes symptoms that mimic asthma, but is not associated with bronchial hyperresponsiveness. (See "Evaluation of subacute and chronic cough in adults", section on 'Nonasthmatic eosinophilic bronchitis'.)

In immunologic occupational asthma, sensitization refers to the development of specific allergen recognition with specific IgE antibodies or cellular immune mechanisms. (See "The biology of IgE" and "Pathogenesis of asthma" and "Occupational asthma: Pathogenesis", section on 'Immunologic mechanisms'.)

EPIDEMIOLOGY

Occupational asthma (OA) is one of the most common occupational lung diseases in developed countries [10]. A number of factors appear to influence the incidence and prevalence of OA, including sex, geographic location, underlying prevalence of atopy, smoking, and most significantly, the type and intensity of workplace exposure [6].

Several studies have reported on the proportion of asthma attributable to occupational exposure. Most estimates are in the range of 5 to 10 percent of adult onset asthma, although a wide range of rates have been reported [11-14]. Based on data from the 2012 Behavioral Risk Factor Surveillance System and the Adult Asthma Call-Back Survey, the proportion of ever-employed adults with work-related asthma in the United States approaches 16 percent with a variation of 9

to 23 percent among individual states [15]. The European Community Respiratory Health Survey (ECRHS) found a work-attributable risk of asthma of 10 to 25 percent [12,13]. The variability of prevalence rates probably reflects differences in methodology, case definitions, and immunogenicity of specific workplace agents. Furthermore, the fact that asthma is a common disease that often presents de novo or worsens during adulthood due to non-occupational factors, further complicates epidemiologic assessment of OA. Based on the European Community Respiratory Health Survey results, it was estimated that occupational exposures contribute to one in seven cases of severe exacerbation of asthma [16].

The effect of sex on the reported incidence of OA was examined in a 12 year study in Finland [12]. The risk of OA among men was highest among bakers, laundry workers, shoemakers and shoe repairers, tanners, fellmongers and pelt dressers (removing fur from hides for leather work), and also metal plating and coating workers. For the women, the risk was highest among shoemakers and repairers, railway and station personnel, jewelry engravers, engine room crew, molders, round-timber workers, and bakers.

The geographic location influences the distribution of asthma and bronchial responsiveness in the population and, thus, the prevalence of OA. As an example, there is a low prevalence of OA, atopy, and bronchial hyperresponsiveness in Mediterranean and Eastern European countries, compared with English-speaking countries [13].

Several cross-sectional studies of populations at high risk of developing OA demonstrate that approximately 2 to 8 percent per person-year of those exposed to high-molecular-weight (proteinaceous) agents, such as laboratory animals, latex, and flour, and 5 to 10 percent of subjects exposed to low-molecular-weight (chemical) agents develop OA (table 2) [11,17,18].

The proportion of individuals with asthma who have worsening asthma symptoms at work (work-exacerbated asthma) is approximately 10 percent. This figure was derived from a meta-analysis of 43 risk estimates from 19 different countries that suggested that 10 percent of all adult-onset asthma cases appear to be occupationally related [19].

Prospective studies in apprentices have shown that the incidence of symptoms, sensitization, and probable OA due to high-molecular-weight occupational agents is highest during the two first years after starting exposure and is less when these apprentices are reassessed at a time they have been working for several years in the same type of occupation [20,21].

CAUSATIVE AGENTS

The agents that cause occupational asthma (OA) include a wide variety of airborne agents in the workplace [3,22]. More than 350 agents have been reported to cause OA [5,23]. Common workplace stimuli are listed in the table (table 2) and a more complete list is available online (Agents causing occupational asthma). New agents are added each year.

Stimuli that cause immunologic OA are divided into two categories, high and low-molecular-weight agents. Flour (high-molecular-weight [HMW] agent) and diisocyanates (low-molecular-weight [LMW] compounds used as hardeners in paints, various glues, and insulation) are the most common causative agents in developed countries, accounting for approximately 20 percent of OA cases [10,24]. (See "Occupational asthma: Pathogenesis", section on 'Immunologic mechanisms'.)

Nonimmunologic causes of OA include a large number of irritant gases, fumes, smoke, and aerosols. These are discussed separately (table 3). (See "Reactive airways dysfunction syndrome and irritant-induced asthma", section on 'Definitions'.)

High-molecular-weight — HMW agents include proteins and polysaccharides of plant or animal origin (>5 to 10 kD) that are complete sensitizing antigens, capable of causing IgE-dependent OA [5]. Thus, they require an initial period of exposure, elaboration of specific IgE antibodies, presence of these specific antibodies on the surface of airway mast cells, and activation of the mast cells following re-exposure to the antigen leading to the clinical features of occupational asthma (figure 1). (See "Occupational asthma: Pathogenesis", section on 'IgE-mediated'.)

A list of the most common causes of OA due to HMW compounds is provided in the table (table 2). The main classes of HMW compounds associated with OA and the associated occupations are as follows:

- **Animals** – Laboratory and farm animal workers are exposed to mammalian proteins from fur/hair, saliva, urine, and dander [22,25]. Proteins excreted in animal urine are a common cause of sensitization among laboratory and veterinary workers. A personal history of sensitization to dogs or cats is a risk factor for laboratory animal sensitization, although general atopy is less predictive [22]. The role of concomitant exposure to endotoxin in rates of sensitization and symptomatology is not known.

- **Insects** – Insects can be a source of potent allergens in the workplace. Examples include mealworm larva exoskeleton dust in bait workers and biologic pest control insects (eg, predatory wasps) used by agriculture workers [26,27].

- **Fish and shellfish** – Occupations with exposure to fish and shellfish can lead to occupational asthma, particularly crab and shrimp processing.

- **Flours and cereals** – Bakers, food processors, and dock workers are at risk for OA from flours and cereals due to intrinsic proteins of the flour and also additives/contaminants such as mites and enzymes (see below) [22].

- **Enzymes** – Enzymes are used in a variety of occupations, such as detergent manufacturing (amylases, lipases, proteases), pharmaceutical preparation, and baking (amylase) [10,22,28]

- **Natural rubber latex** – Natural rubber latex, which is derived from the rubber tree Hevea brasiliensis (Hev b), used to be a major cause of OA, but the risk has considerably decreased

due to the reduction in use of powdered latex gloves. A description of the Hevea latex allergens and the evaluation and management of patients with latex-associated respiratory complaints are described separately. (See "Latex allergy: Epidemiology, clinical manifestations, and diagnosis" and "Latex allergy: Management".)

Dermatitis is uncommon among workers with OA due to HMW compounds, although a history of contact dermatitis to latex is reported by some workers with latex OA [22].

Low-molecular-weight — LMW chemicals (eg, diisocyanates, trimellitic anhydride, formaldehyde) are incomplete antigens (ie, haptens) that combine with a human protein to produce a sensitizing neoantigen (table 2). In addition, some of these agents also appear to cause OA through a non-IgE mechanism. (See "Occupational asthma: Pathogenesis", section on 'Immunologic mechanisms'.)

The most common LMW chemicals that cause OA include the following:

- Diisocyanates – Toluene diisocyanate, methylene diphenyl diisocyanate, and hexamethylene diisocyanate are used in polyurethane and plastics production, spray painting, foam coating manufacturing, and adhesive/sealant production use [22].
- Wood dusts – Exposure to dust from many species of wood can cause OA. Among these woods the most well known, because it has been extensively studied in clinical and epidemiological studies, is Western red cedar that contains plicatic acid, the causal substance [22].
- Acrylates – Cyanoacrylates and methacrylates are used in plastics and adhesives and are a cause of OA among dental hygienists, prosthetists, and nail salon workers [29,30].
- Persulfate salts – Persulfate salts are the major causative agents of OA in hairdressers [31,32]. Skin prick and patch tests to persulfate are variably positive [31,32]. Specific bronchial provocation challenge to persulfate salts in the laboratory yields early, late, and dual responses [31-33]. (See "Contact dermatitis in children", section on 'Patch testing' and "Occupational asthma: Clinical features, evaluation, and diagnosis", section on 'Specific inhalation challenge' and "Bronchoprovocation testing", section on 'Antigen challenge'.)
- Metals and metalloids – Many metal salts (eg, platinum, chromium, nickel, cobalt, zinc) can cause OA. Also, welders frequently develop OA probably through exposure to metal-derived products, although the mechanism is poorly elucidated.

Combined exposures — Workers in certain professions (eg, bakers, health care workers, auto body repair workers, hairdressers) may be exposed to several different sensitizers and these may be of both high and low-molecular-weight [22].

Among bakers, OA associated with wheat flour inhalation may be caused by sensitization to omega-5 gliadin (Tri a 19), alpha-amylase inhibitors, thioredoxins (cross react with grass allergens), wheat lipid transfer protein (Tri a 14), wheat serine protease inhibitor, mites, and baking additives such as fungal alpha-amylase (Asp o 21) [34-37]. Furthermore, partial homology between wheat thioredoxins and human thioredoxins may lead to cross-reactivity of the specific IgE and perpetuation of the IgE-mediated mast cell activation after removal from airborne wheat exposure [34].

Health care workers may be exposed to glutaraldehyde (instrument sterilization), antibiotics, psyllium, aerosolized medications (eg, pentamidine, ribavirin), acrylates (orthopedic adhesives), methylene diphenyl diisocyanate (synthetic plaster casts), metals in dental alloys, formaldehyde and also chloramines and quaternary ammonium compounds (cleaning agents) [22,38-40]. Latex is a much rarer cause of occupational asthma since the use of powder-free gloves [41].

Workers in auto body repair shops are often exposed to diisocyanates in urethane paints (spray paint hardening agents), acrylates (adhesives), amines (glues and epoxies), and anhydrides (epoxy resin, dye) [22].

In addition to persulfate salts, hairdressers are exposed to bleaching agents, henna dye, latex, and also secondary, tertiary, and quaternary amines (in hair dyes) [22,31,32].

OCCUPATIONAL RISK FACTORS

Exposure intensity — The intensity of the exposure is the most important factor in the development of occupational asthma (OA) [11]. A dose-response relationship between exposure and both the prevalence and incidence of OA has been demonstrated for both high- and low-molecular-weight compounds including Western red cedar, diisocyanate, colophony (ie, soft core solder), acid anhydride, flour, alpha-amylase, natural rubber latex, cow dander, and rat urine [22,42-44].

Despite the known dose-response relationship, permissible exposure limits have not been established by the Occupational Safety Administration (OSHA) for many stimuli because it is unknown if it is the intensity, duration, or cumulative dose of exposure that is most important. As an example, intermittent short-term exposure to elevated levels of diisocyanates may represent the greatest risk for the subsequent development of OA [45]. After an individual becomes sensitized to an occupational agent, even a low-level exposure (well below permissible limits) can precipitate life-threatening bronchospasm.

Reducing the exposure intensity in the workplace can reduce the rate of OA. (See "Occupational asthma: Management, prognosis, and prevention", section on 'Prevention'.)

Exposure to very high concentrations of some agents associated with occupational asthma (eg, diisocyanates) has been reported to cause reactive airways dysfunction syndrome (RADS) [46]. In addition, one case report has described development of immunological sensitization to diphenylmethane diisocyanate and onset of immunological OA following an episode of RADS caused by irritant levels of that diisocyanate [47]. (See "Reactive airways dysfunction syndrome and irritant-induced asthma".)

Specific occupational exposures — The specific agent to which a worker is exposed influences the frequency of developing OA [48]. Using skin test positivity as a marker of immunological sensitization, with or without bronchial hyper-responsiveness, the following prevalences of OA due to high-molecular-weight (HMW) compounds were estimated:

- 3 to 7 percent of technicians exposed to laboratory animals [49]
- 5 to 13 percent of bakers exposed to flour-related antigens [50]

Similarly, a prospective study of nearly 800 apprentices with new exposure to HMW agents identified the following incidences of probable OA over the 44 months of the study [51]:

- 8 percent of technicians exposed to laboratory animals
- 4 percent of bakers exposed to flour-related antigens
- 2 percent of dental hygienists exposed to latex

OTHER RISK FACTORS

Most workers exposed to occupational sensitizing agents do not develop occupational asthma (OA) [11,52]. The likelihood of developing the disease is influenced by a variety of host factors, including atopy, cigarette smoking, and genetic predisposition.

Atopy — Atopy is consistently associated with sensitization to high-molecular-weight (HMW) agents [11,22,53]. Thus, atopic individuals who wish to enter high-risk workplaces should be advised of the potential for developing OA or exacerbating preexisting asthma and should receive regular medical follow-up. Despite this increased risk, atopy should not be used as a screening tool in high-risk workplaces because the positive predictive value of atopy for the subsequent development of symptoms is low [54,55]. Among supermarket bakery workers, specific sensitization to work-related proteins was two to four times as likely among atopic compared with nonatopic workers, but overall, less than 30 percent of atopic bakery workers developed OA [50].

Cigarette smoking — Cigarette smoking is a risk factor for sensitization to some HMW occupational allergens, including coffee, castor beans, shrimp, and snow-crab, but not to laboratory animal allergens [56]. Cigarette smoking is also a risk factor for sensitization to a few low-molecular-weight (LMW) agents, such as platinum salts and phthalic anhydride, but appears to be less important in promoting sensitization to other LMW agents [56].

As an example, among platinum refinery workers, smoking is the most important risk factor for sensitization. In an observational cohort study of 91 workers followed for approximately seven years, there was an increased risk of developing skin test reactivity to workplace antigens in smokers when compared to nonsmoking controls (relative risk 5, 95% CI 1.7-15.2) [57]. Similarly, synergistic interaction between smoking and atopy has been noted in workers exposed to tetrachlorophthalic anhydride or laboratory animals, with atopic smokers demonstrating the highest prevalence of sensitization to these antigens [58,59].

However, being sensitized does not mean that the worker will develop clinical symptoms or occupational asthma. The role of smoking as a risk factor for the development of occupational asthma to high molecular weight agents is controversial, particularly with flour, laboratory animals, latex, and enzymes [56]. In workers processing snow-crab and salmon, smoking is thought to increase the risk of occupational asthma. With low molecular weight agents, data on smoking and occupational asthma are either conflicting (as for platinum salts) or not available.

Genetics — Several studies have examined whether genetic characteristics would explain why some exposed workers develop OA, while other exposed workers do not. Genes that have been implicated include human leukocyte antigen (HLA) class II molecules and polymorphisms in non-HLA genes (eg, glutathione S-transferases, N-acetyltransferase, tumor necrosis factor alpha, prostaglandin-endoperoxide synthase). Genetic susceptibility to occupational asthma is discussed in greater detail separately.(See "Occupational asthma: Pathogenesis", section on 'Genetics' and "Genetics of asthma" and "Human leukocyte antigens (HLA): A roadmap".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Occupational asthma".)

SUMMARY AND RECOMMENDATIONS

- Occupational asthma (OA) begins during adulthood and can be caused by immunologic or non-immunologic stimuli. OA due to an immunologic stimulus has a latency period between exposure and symptom onset, while OA due to a nonimmunologic stimulus does not. When a nonimmunologic stimulus triggers OA, it is referred to as reactive airways dysfunction syndrome (RADS) and irritant-induced asthma. (See "Reactive airways dysfunction syndrome and irritant-induced asthma".)

- Stimuli that cause immunologic OA are divided into two categories, high-molecular-weight (eg, flour, laboratory animal proteins) and low-molecular-weight (eg, diisocyanates, trimellitic anhydride, formaldehyde). (See 'Causative agents' above.)

- Low-molecular-weight chemicals are incomplete antigens (ie, haptens) that combine with a protein to produce a sensitizing neoantigen. High-molecular-weight organic agents are complete sensitizing antigens. (See "Occupational asthma: Pathogenesis".)

- OA should be suspected and evaluated in every patient with adult-onset asthma. It is estimated that 5 to 25 percent of all adult-onset asthma cases are occupationally related. Common workplace stimuli are listed in the table (table 2). A more complete list is available online (Causes of occupational asthma). (See 'Causative agents' above.)

- Most workers who are exposed to allergens in the workplace do not develop OA. The likelihood of developing OA is influenced by occupational factors, such as intensity of exposure and the particular sensitizing agent, and host factors, such as atopy, cigarette smoking, and genetic predisposition. (See 'Other risk factors' above.)

- The clinical evaluation, diagnosis, and management of OA are discussed separately. (See "Occupational asthma: Clinical features, evaluation, and diagnosis" and "Occupational asthma: Management, prognosis, and prevention".)

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GRAPHICS

Comparison of occupational asthma with a latency period and reactive airways dysfunction syndrome

	Occupational asthma with a latency period	Reactive airways dysfunction syndrome and irritant-induced asthma
Latency period	Present	Absent
Diagnosis	Various - eg, peak expiratory flow monitoring, specific inhalation challenges; IgE-mediated allergy if suspected agent is of a high-molecular-weight nature	History; pulmonary function testing
Pathology	Similar to asthma	Acute: epithelial shedding and hemorrhage
		Chronic: regeneration of epithelial cells; few lymphocytes and neutrophils; increased collagen deposition and thickness of basement membrane
Pulmonary function testing	Better reversibility to bronchodilator	Less reversibility to bronchodilator

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Major causes of occupational asthma and rhinitis

	Occupation at risk
Low molecular weight chemicals	
Isocyanates (eg, toluene diisocyanate, diphenylmethane diisocyanate, hexamethylene diisocyanate, naphthalene diisocyanate)	Polyurethane workers, roofers, insulators, painters
Anhydrides (eg, trimellitic anhydride, phthalic anhydride)	Manufacturers of paint, plastics, epoxy resins
Metals (eg, chromic acid, potassium dichromate, nickel sulfate, vanadium, platinum salts)	Platers, welders, metal and chemical workers
Drugs (eg, beta-lactam agents, opiates, other)	Pharmaceutical workers, farm workers, health professionals
Wood dust (eg, Western red cedar, maple, oak, exotic woods)	Carpenters, woodworkers
Dyes and bleaches (eg, anthraquinone, carmine, henna extract, persulfate, reactive dyes)	Fabric and fur dyers, hairdressers
Amines (eg, chloramine, quaternary amines)	Chemists, cleaners, plastic manufacturers
Glues and resins (eg, acrylates, epoxy)	Plastic manufacturers
Miscellaneous (eg, formaldehyde, glutaraldehyde, ethylene oxide, pyrethrin, polyvinyl chloride vapor)	Laboratory workers, textile workers, paint sprayers, health professionals
High molecular weight organic materials	
Animal proteins (eg, domestic and laboratory animals, fish and seafood)	Farmers, veterinarians, poultry processors, fish and seafood processors
Flours and cereals	Bakers, food processors, dock workers
Enzymes (eg, pancreatic extracts, papain, trypsin, *Bacillus subtilis*, bromelain, pectinase, amylase, lipase)	Bakers, food processors, pharmaceutical workers, plastic workers, detergent manufacturers
Plant proteins (eg, wheat, grain dust, coffee beans, tobacco dust, cotton, tea, latex, psyllium, various flours)	Bakers, farmers, food and plant processors, health professionals, textile workers

Graphic 66185 Version 6.0

Agents responsible for reactive airways dysfunction syndrome

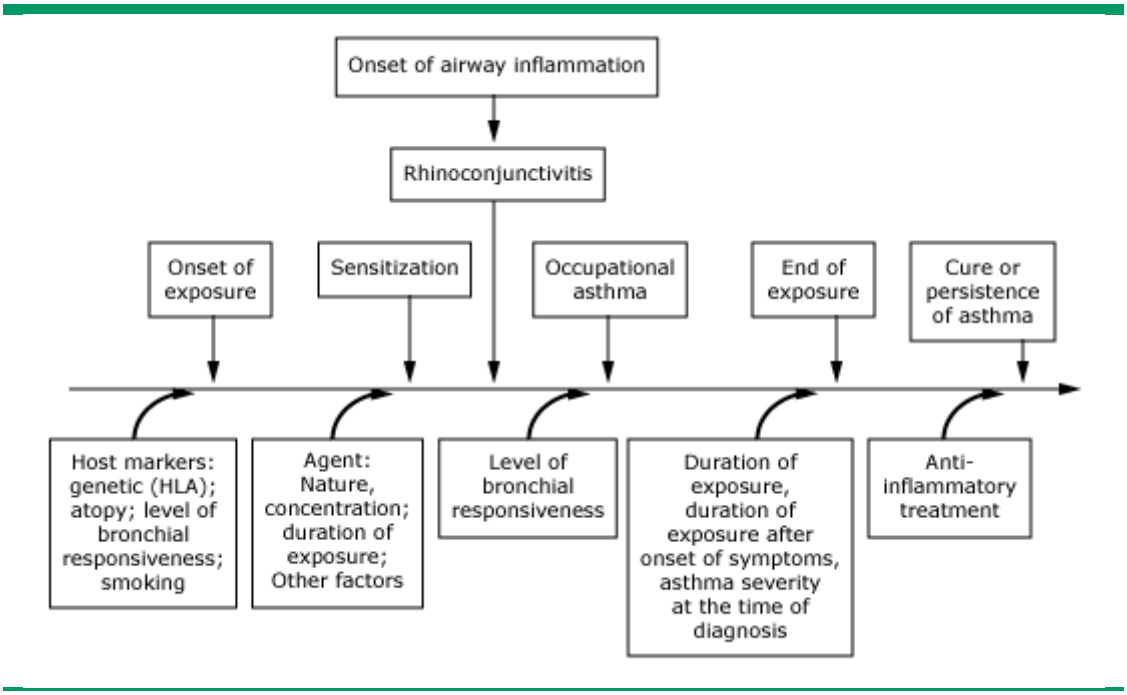
Agent	Type of investigation	Evidence	Reference
Acetic acid	Epidemiological	H, S, BHR	Am Rev Respir Dis 1991; 144:1058
	Case report	H, S, P	Br J Ind Med 1989; 46:67
Chloridic acid	Case report	H, S, BHR	Chest 1988; 94:476 Chest 1990; 98:928
Sulfuric acid	Case report	H, S, BHR	Chest 1988; 94:476
Various acids	Case report	H, S, BHR, P	Chest 1994; 105:1895
	Case report	H, S, BHR	Chest 1989; 96:297
Bleaching agent	Case report	H, S, BHR	Chest 1988; 94:476
Cleaning agents	Case report	H, S	Chest 1976; 69:372
Sealing agents	Case report	H, S, BHR	Chest 1985; 88:376
Ammonia	Case report	H, S	Mayo Clin Proc 1983; 58:389
	Case report	H, S, P	Thorax 1992; 47:755
Phtalic anhydride	Case report	H, S, BHR	Rev Pneumol Clin 1993; 49:247
Bromochlorodifluoromethane (Halon 1211)	Case report	H, S, BHR	Occup Env Med 2004; 61:712
Bromotrifluoromethane (Halon 1301)	Case report	H, S	Eur Respir J 1999; 13:1192
Chlorine	Epidemiological	H, S, BHR	Occup Env Med 1994; 51:225
	Clinical study	H, S, BHR, P	J Allergy Clin Immunol 1994; 93:12 Eur Respir J 1997; 10:241
Chlorofluorocarbons (CFC)	Clinical study	H, S, BHR, P	Scand J Work Environ Health 2003; 29:71
Chloropicrin	Experimental	P	Toxicol Appl Pharm 1984; 74:417
Metal coat remover	Case report	H, S, BHR	Chest 1985; 88:376
Diesel	Case report	H, S, BHR	J Occup Med 1993; 35:149
Diethylaminoethanol	Epidemiological	H, S	J Occup Med 1994; 36:623
Sulfur dioxide	Case report	H, S, BHR, P	Chest 1990; 98:928 Am Rev Respir Dis 1979; 119:555
	Case report	H, S, BHR	Chest 1989; 96:297
Epichlorohydrin	Experimental	P	Toxicol Appl Pharm 1984; 74:417
Fire/smoke	Case report	H, S, BHR	Chest 1988; 94:476
	Case report	H, S	J Occup Med 1991; 33:458
Formalin	Case report	H, S	Lancet 1975; 2:603
Formaldehyde	Case report	H, S, BHR	Allergy 2004; 59:115
Hydrazin	Case report	H, S, BHR	Chest 1985; 88:376

Iodine	Case report	H, S, BHR, P	Ind Health 2009; 47:681
Diisocyanates	Case report	H, S, BHR	Scand J Work Environ Health 1981; 7:310
	Case report	H, S, BHR, P	Allergy 1996; 51:262
	Experimental	S	Toxicol App Pharmacol 1987; 89:332
	Case report	H, S, BHR	Chest 1989; 96:297
Calcium oxide	Case report	H, S, BHR	Chest 1989; 96:297
Ethylene oxide	Case report	H, S, BHR, P	Br J Ind Med 1992; 49:523
Heated paints	Case report	H, S, BHR	Chest 1989; 96:297
Pulverized paints	Case report	H, S, BHR, P	Chest 1989; 96:297 Chest 1985; 88:376
Perchloroethylene	Case report	H, S, BHR	Chest 1988; 94:476
Vapors (chlorine, mustard, phosgene, etc)	Case report	H, P	Br Med J 1915; 165

H: history; S: spirometry; BHR: bronchial hyperresponsiveness; P: pathology.

Graphic 55545 Version 5.0

Natural history of occupational asthma with a latency period



Graphic 80111 Version 3.0

Contributor Disclosures

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Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

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