Work-exacerbated asthma

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Abstract

Exposures at work can contribute to both the onset and exacerbation of asthma. This chapter summarizes key information regarding work-exacerbated asthma (WEA), a common condition that has received little attention compared to new occupational asthma. WEA refers to pre-existing or concurrent asthma that is worsened by factors at work. WEA, as with asthma in general, is heterogeneous, with multiple phenotypes and triggers. The prevalence of WEA has ranged from about 15% to over 50% among working adults with asthma in published studies, but is rarely diagnosed by clinicians. WEA occurs in a wide range of industries and occupations, including education, services, manufacturing and construction, and can lead to job changes and unemployment. Multiple factors at work can exacerbate asthma, including various irritants, allergens, molds, cold and exertion. Cleaning products and building renovation in non-industrial workplaces such as schools and offices are commonly implicated. WEA can lead to substantial adverse outcomes, similar to OA. Management of WEA should focus on reducing work exposures and optimizing standard medical management.

Introduction and definitions

Asthma is a common disease, affecting up to approximately 15% of working-age adults [1]. Work-related asthma (WRA) comprises both occupational asthma (OA) in which exposures at work cause new onset asthma, and work-exacerbated asthma (WEA) in which existing asthma is aggravated by conditions at work. An estimated 15% of new adult onset asthma is attributable to exposure to sensitizers or irritants in the work environment [2, 3]. The literature on WRA has focused primarily on sensitizer-induced OA. Asthma exacerbations are common, and can be triggered by a number of factors, including workplace exposures. However, WEA has received less attention than OA. The purpose of this chapter is to summarize relevant

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information about WEA, including the definition and epidemiology of WEA, causative factors, natural history, and a suggested clinical approach for diagnosis and management of WEA.

WEA refers to pre-existing or concurrent asthma that is worsened by work-related factors [3]. A case of 'concurrent asthma' has onset while the individual is employed, but the onset is not attributable to work. As with asthma in general, definitions of WEA have varied depending on the clinical, research, or public health setting, but all depend on defining "asthma" and "asthma exacerbation". This can be challenging, given that asthma is a heterogeneous syndrome that involves multiple phenotypes, multiple factors can exacerbate asthma, and that exacerbations can vary from brief worsening of symptoms to severe episodes requiring hospitalization or resulting in death. Key features of asthma include airway inflammation, airway hyperresponsiveness (reversible airflow obstruction), and recurrent symptoms of wheezing, chest tightness or cough [4, 5]. Clinical studies are more likely to use objective tests such as reversible airflow obstruction on spirometry and medication usage to define asthma. Epidemiological studies are more likely to use self-reports of doctor-diagnosed asthma and asthma symptoms. Work-relatedness is primarily assessed by self-reports of symptoms or medication use relative to work, or occasionally by physiologic indicators such as work-related changes in peak flow rates.

Prevalence of WEA

The prevalence of WEA has been investigated using several approaches. A relatively small but growing number of studies have evaluated the frequency of WEA in general populations of asthmatics and provide estimates of the prevalence of WEA among adult asthmatics (Tab. 1) [6–14]. The prevalence of WEA estimated from these studies was quite variable, as shown in Table 1, ranging from 14% to 38% among all adults with asthma, and from 14% to over 50% among working adults with asthma, a more appropriate at risk group (denominator). The populations studied, age ranges, asthma definitions, geographic locales, and criteria for work exacerbation vary in these studies, as noted in Table 1. Asthma was defined as doctor-diagnosed asthma in most studies, with access to medical records to identify cases, except for studies that selected participants from the general population. Work exacerbation was generally based on self-reported asthma symptoms that had worsened in relation to work, typically within the past year; however, these reports rarely included the frequency or severity of exacerbation.

The frequency of WEA has also been expressed as a percentage of all WRA cases, with a denominator that includes both OA and WEA cases (Tab. 2) [15–26]. Most of these studies were conducted in clinical referral settings or relied on surveillance or worker compensation systems for identifying cases of WRA. Estimates

Table 1. Prevalence of WEA among adults with asthma (selected studies)

Reference	Setting*	No. of asthma cases	Age (years)	Criteria for WEA (self-reported on questionnaire unless indicated otherwise)	Prevalence of WEA among	
					All adults with asthma	Working adults with asthma
Abramson 1995 [6]	G Pop	159	43 mean	Respiratory symptoms at work	20%	NA§
Blanc 1999 [7]	G Pop	160	20–44	Report chest tightness at work	38%	NA
Bolen 2007 [8]	HMO	95 all employed	18–44, 34 mean	Pattern of serial peak expiratory flow rate consistent with WEA	NA	14%
Goh 1994 [9]	Clinics	802	20–54	Work environment is asthma trigger	27%	NA
Henneberger 2002 [12]	HMO	1,461	18–44	Current work environment makes asthma worse	25%	NA
Henneberger 2003 [10]	G Pop	42 28 employed	18–65, 42 mean	Coughing or wheezing is worse at work than when not at work	14%	21%
Henneberger 2006 [11]	HMO	598 OA excluded	18–44	Relevant exposure and work-related pattern of symptoms or medication use	23%	24%
Mancuso 2003 [13]	Primary care	102 all employed	39 mean	At least one of several job conditions makes asthma worse	NA	58%
Saarinen 2003 [14]	NHI system	969 OA excluded	20–65, 43 mean	Asthma symptoms caused or worsened by work at least weekly in past month	NA	20%

* Abbreviations for setting: G Pop, general population; HMO, health maintenance organization; NHI, national health insurance

§ NA, not applicable

Table 2. Prevalence of WEA among all work-related asthma (WRA) cases (selected studies)

Reference	Clinical setting*	Age (years)	Criteria for WEA†	No. with WRA	% with WEA
Caldeira 2006 [15]	1922 subjects selected from birth cohort	23–25	Pre-existing asthma worsened by exposure at work	81	36%
Curwick 2006 [16]	Workers compensation (WA)	43 median	SENSOR Criteria	301	45%
de Fatima 2007 [17]	Cleaners	37 mean F, 34 mean M	Pre-existing asthma, and cleaning-related sx	26	58%
Fletcher 2006 [18]	OHC (NY)	20–60, 43 mean	SENSOR Criteria	454	14%
Goe 2004§ [19]	SENSOR surveillance	18–70+	SENSOR Criteria	1101	19%,
Larbanois 2002 [20]	Referral clinic	32–54	Asthma sx temporally related to work exposure and negative SIC	157	45%
Lemiere 2007 [21]	Referral clinic	Adults	Aggravation of asthma sx at work, negative SIC	351	41%
Pechter 2005§ [22]	SENSOR cases in health care	41 median	SENSOR Criteria	305	23%
Reinisch 2001§ [23]	Survey of physician first reports (CA)	18–65+	SENSOR Criteria	444	35%
Rosenman 2003§ [24]	SENSOR cases cleaning products	18–70+	SENSOR Criteria	236	20%
Tarlo 1995 [26]	Worker compensation	Mean ~40	Asthma worse at work, with or without prior asthma, no sensitizer exposure. Objective testing common	469	50%
Tarlo 2000 [25]	Asthma clinic	Mean 46	Work-related sx, irritants or other aggravating factors, no sensitizer exposure. Objective testing common	51	49%

* Abbreviations Clinical Setting: OHC system, occupational health clinic; Referral clinic, clinic for suspected work-related asthma; Worker compensation, data from worker compensation system.

† Abbreviations for Criteria WEA: rx, medications; sx, symptoms; SIC, specific inhalation challenge

§ A subset of subjects reported in [9]. SENSOR, Sentinel Event Notification Systems for Occupational Risks. Data from CA, MA, MI, NJ, unless otherwise specified.

SENSOR criteria WEA, asthma in 2 years before new occupational setting, asthma symptoms work-related, more asthma symptoms or medications in new setting

of prevalence of WEA as a percentage of all WRA cases ranged from 14% to over 50%, as shown in Table 2. These estimates were highly dependent on factors such as physician recognition, referral patterns, reporting system, and local workers compensation laws. WEA typically is not diagnosed by clinicians in settings where it is not clearly recognized as a condition under worker's compensation rules, such as in New York State and many other states in the United States [18]. Where WEA is recognized, as in the Canadian province of Ontario, or more recently in the province of Quebec, the number of cases of WEA compared to OA tends to rise [21, 25, 26].

The prevalence of WEA has also been described in selected groups of asthmatic workers, such as office workers, cleaners, or construction workers (Tab. 3), with up to 70% of asthmatics reporting exacerbation of their asthmatic symptoms related to their work [17, 27–32]. As with the other clinical and epidemiological studies, the worker populations and diagnostic criteria were variable.

Exposures and WEA

As with non-work-related asthma, a number of diverse exposures and factors have been associated with WEA (Tab. 4). Overall, most commonly reported are various irritant mixed exposures, such as dusts, second-hand smoke, solvents, cleaning products, and fumes at work [9, 18, 25–27]. Common allergens and molds, frequently in settings with inadequate indoor air quality such as office buildings and schools, have also been reported as triggers for asthma symptoms [3, 18, 28]. In addition, non-chemical conditions can also exacerbate asthma at work, including extremes of temperature, physical exertion, emotional stress, and viral infections [3, 11, 14, 25–27, 33].

There are few quantitative data regarding the levels of work exposures that trigger asthma, and comparisons between studies and with different definitions or criteria for WRA can be difficult. However, together these studies indicate that exposures associated with WEA, as compared to new onset OA, were less likely to be specific sensitizing agents, and more likely to be irritant exposures [3, 9, 25]. The irritants associated with WEA typically occur at lower levels than those reported to cause new onset irritant-induced OA [3].

WEA has been reported in a wide range of industries and occupations, including public administration, wholesale and retail trade, cleaning, manufacturing, education, and construction [7, 9, 12, 14, 27]. Several studies have suggested a healthy worker effect, where asthmatic workers leave workplaces with more asthma triggers [10, 12, 34]. Although the specific asthma triggers can be difficult to identify, it is clear that WEA can occur in numerous different work settings, including non-industrial workplaces such as office buildings, schools, and laboratories.

Table 3. Prevalence of WEA among selected asthmatics in certain jobs or work settings (selected studies)

Reference	Type of workers/ work setting	Age (years)	Criteria for WEA (self-reported on questionnaire unless indicated otherwise)	Number asthmatics	Prevalence WEA among asthmatics
Berger 2006 [27]	Low income inner city patients	18–55	Asthma worse at current or most recent job (janitors, security guards, clerks, restaurant workers, textile workers)	301	51%
Cox-Ganser 2005 [28]	Office workers	46 mean	Work-related asthma symptoms in water-damaged building	67	34%
De Fatima 2007 [17]	Cleaners	37 mean F 34 mean M	Cleaning-related asthma symptoms	39	38% (46% females/ 18% males)
Gouge 1994 [29]	Soldiers in Iraq	21–44	Exacerbation of asthma symptoms	10	70%
Jacobs 2007 [30]	Swimming pool workers	16–65 40.5 mean	In last 1 year more asthma attacks and asthma medications, compared to Dutch population	624 working at pools (number with asthma unclear)	OR = 2.6 asthma attack $p < 0.05$
Kreiss 2006 [31]	Cosmetic workers	42 mean	Report asthma worse with workplace exposure or activities	175 108 pre/67 post hire	28% pre-hire asthmatics 58% post-hire asthmatics
Sauni 2001 [32]	Construction workers	18–64	Symptoms worse at work or occupational dust cause symptoms	76	68% worse at work, 66% dust causes symptoms

Table 4. Selected industries, jobs and exposures associated with WEA

Selected industries / Jobs associated with WEA
Technical, sales and administrative support
Public administration, teaching
Laboratory and medical technicians
Cleaners, janitors
Manufacturing (textile workers, operators, laborers)
Selected exposures associated with WEA
Second-hand smoke
Dusts
Smoke, welding fumes
Chemicals (cleaning products, paints, solvents, acids, ammonia)
Common allergens / molds
Abnormal temperatures
Physically strenuous work
Viral respiratory infections

WEA should also be considered in the context of non-work-related asthma exacerbations, which are common, and quite variable in severity, time course and etiology. A number of factors and/or triggers can exacerbate asthma, including viral infections, allergens, irritants, non-compliance with medications, exercise, stress, or other medical conditions (e.g., gastroesophageal reflux or sinusitis) [35]. In both settings many of these exposures are preventable, but in the work setting the patient typically has less control over the environment.

Clinical characteristics and natural history of WEA

The clinical characteristics and natural history of WEA have received much less attention than those related to OA. Several studies have now compared clinical characteristics of patients with WEA to those with OA or to other asthmatics. The study populations, selection and diagnostic criteria, and other methodological features have been quite variable, making findings within and between studies difficult to interpret and compare. A limited number of studies comparing WEA and OA have found predominantly similarities between these groups, including similar levels of asthma severity, airway hyperresponsiveness, medication usage, and needs for healthcare [9, 20, 21, 26, 36].

Similarly, the few studies that have evaluated removal from exposure or more long-term outcomes in WEA have generally found outcomes similar to those for OA, with persistent asthma but improved symptoms and reduced airway inflammation away from exposure [36, 37], although some differences have also been noted, such as higher doses of inhaled corticosteroids in WEA [36]. These findings may reflect selection criteria for the two groups of patients.

A relatively small number of studies have also compared WEA to other asthma cases. Overall, these studies have tended to find more asthmatic symptoms and/or more frequent or more severe asthma exacerbations in asthmatics with WEA compared to other asthmatics [11, 14, 21, 38]. However, some of these same studies have found less severe asthma in WEA using different criteria for asthma severity, and other studies have found less frequent asthma exacerbations in subjects with WEA [11, 25].

Socioeconomic impact

Recent studies have begun to evaluate the social and financial consequences of WEA, comparing WEA to asthma unrelated to work or to occupational asthma [9, 20, 39]. Again, comparisons within and between studies is difficult, as the studies were based on different patient populations, asthma severities, age ranges, and diagnostic criteria. Despite these limitations, studies have generally found that WEA is associated with similar outcomes to those of OA in terms of prolonged unemployment, loss of income, and frequent job changes [9, 20, 39]. However, other studies have reported less frequent job changes for WEA subjects [9, 20, 26].

Diagnosis, management and prevention of WEA

Clinical studies addressing optimal strategies for diagnosing and managing WEA are limited, with the great majority of prior studies addressing sensitizer OA. An expert panel assembled by the American College of Chest Physicians recently published a consensus document on the diagnosis and management of work-related asthma, including WEA [3]. Relevant conclusions are summarized briefly. WEA should be considered in any patient with worsening asthma and/or work-related asthma symptoms. The diagnosis of asthma should be clarified, based on the clinical history, typical symptoms and exam findings, and documentation of reversible airflow obstruction or airway hyperresponsiveness (e.g., bronchodilator response or methacholine challenge test), although this can be difficult to demonstrate in some asthmatics. A careful occupational and medical history is essential. Information on job exposures, type of industry, ventilation, the onset and timing of symptoms in

relationship to work, medication use, symptoms in co-workers, and use of protective equipment should be obtained. History of asthma, childhood asthma, atopy, rhinitis and sinusitis should also be clarified, with particular focus on the first onset of asthmatic symptoms, clinical course and triggers.

The relationship between work and asthma exacerbations should be assessed, most commonly by careful documentation of changes in symptoms and medication use temporally related to work, including specific tasks or jobs at work. More severe exacerbations may be documented by health care visits or physiological changes (e.g., in peak expiratory flow rate or forced expiratory volume in one second). Additional sources of exposure information include Material Safety Data Sheet (often abbreviated as MSDS), a union, the employer (with patient's permission), and government agencies. WEA frequently is due to a mixture of substances rather than a single substance, such as encountered in work settings with several irritant gases, construction dust from multiple materials, or many types of cleaning products in use.

WEA should be distinguished from OA, especially if a specific sensitizing agent is identified at work. This can be challenging if the asthma is longstanding and the worker is no longer at the suspect job, since chronic asthma tends to respond nonspecifically to multiple triggers. Factors at work and outside work that trigger the asthma should be identified. Thus allergy testing may be useful in atopic asthmatics.

Data on management and prevention of WEA is also very limited. The goal is to improve asthmatic symptoms by reducing work triggers and optimizing asthma treatment. If unsuccessful, a change to a job with fewer triggers may be necessary. Prevention also focuses on reducing work exposures and factors that can trigger asthma, and potentially modifying a worker's job to avoid triggers such as cold weather.

Summary

WEA refers to pre-existing or concurrent asthma that is worsened by work factors. WEA is common in both industrial and non-industrial settings, but has received less attention than OA that is caused by work. A review of the current literature on WEA demonstrates that the prevalence of WEA among working adults is variable, ranging from approximately 15% to 50%. Numerous different exposures or conditions at work can exacerbate asthma. WEA clinically shares many features with OA, including persistent asthma and adverse socioeconomic outcomes (prolonged unemployment, reduction in income). Management of WEA should focus on reducing work exposures and optimizing standard medical management, with a change in jobs only if necessary.

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