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Lesson 13 — Work-Exacerbated Asthma

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Objectives

1. Define work-exacerbated asthma (WEA) as a type of work-related asthma.
2. Describe the prevalence of WEA.
3. Identify occupational risk factors for exacerbation of asthma.
4. Examine how patients with WEA compare with other patients with asthma.
5. Review options for the management of WEA.

Key words: asthma; exacerbation; occupational exposures; work-related

Abbreviations: OA = occupational asthma; PEFR = peak expiratory flow rate; WEA = work-exacerbated asthma

Asthma is a common disease with a lifetime prevalence of 13.1% and a current prevalence of 8.4% among adults in the United States.[1] Exacerbation of asthma symptoms is also common; about half of those with current asthma experience an asthma attack each year. In the United States in 2004, asthma led to a considerable amount of health-care utilization among adults, including 7.7 million outpatient visits, more than 1.0 million emergency room visits, and 300,000 hospitalizations.[2] Although such utilization is linked to high direct health costs, the indirect costs of adult asthma are also considerable, with a major component being attributable to the work impact of asthma.[3] Statistics from around the world indicate that a high percentage of adults with asthma miss work in a 12-month period because of their asthma: 25% in the United States, 17% in Western Europe, 23% in Central and Eastern Europe, 27% in Asia-Pacific, and 30% in Japan.[4]

Actions that help reduce the adverse workplace impact of this disease are needed and would address a current gap in clinical practice as compared to recommended practice for the recognition and treatment of work-related morbidity. Moreover, exposures in the workplace are a potentially modifiable source of environmental conditions that can trigger asthma symptoms; intervening to reduce such triggers is a cornerstone of management recommended for asthma in general, although its application to the occupational environment also represents a practice gap that should be narrowed.

Defining Work-Exacerbated Asthma

Work-related asthma comprises both occupational asthma (OA), caused etiologically by exposures at work, and work-exacerbated asthma (WEA), that is, worsening of asthma as a result of exposures at work. OA can arise from either sensitizers or irritants. Sensitizers are commonly separated into low-molecular-weight and high-molecular-weight compounds. OA related to irritants includes reactive airways dysfunction syndrome, which is initiated shortly after a single, high-level irritant exposure.

Different definitions have been proposed and used for WEA by researchers and clinicians, depending in part on prevailing standards of care, resources for diagnosis, and medicolegal considerations. There is no single criterion or test that determines the presence or absence of WEA. In the past, many clinicians have not used objective tests to clarify the status of cases with suspected work-related asthma generally, including both OA and WEA. Surveillance data from the early 1990s revealed that only 3.4% of reported work-related asthma cases had evidence in their medical records that the treating physician had used any type of pulmonary function testing to confirm that the case was work related.[5] Certain clinical research centers have used more objective tests and a more specific (meaning more restrictive) definition of WEA.[6-9] Definitions are often more sensitive (meaning more inclusive) in epidemiologic studies and surveillance systems, in which the opportunity to directly examine cases is limited or nonexistent. In these settings, the determination of WEA status often relies on self-report of work-

related symptoms or work-related changes in asthma medication use.

The following case definition of WEA includes four criteria and can be used in both clinical and research settings.

1. Those at risk for WEA have preexisting or concurrent (coincident) asthma. This means the individual at risk for WEA experienced the onset of asthma either before entering the workplace of interest (*ie,* preexisting asthma) or while already in the workplace of interest, but not because of conditions in that workplace (*ie,* concurrent or coincident asthma). The workplace of interest can be either a new job or an existing job in which exposures have changed either permanently (*eg,* processes or materials newly used or produced) or temporarily (*eg,* remodeling of the workplace or inadvertent chemical spills or releases).
2. There is a plausible temporal relationship in which the implicated workplace exposure is closely linked in time to the exacerbation of asthma. This relationship can be established by selfreports of symptom patterns or medication use, or by more objective evidence, such as serial peak expiratory flow rate (PEFR) measurements that follow a work-related pattern. A similar pattern could be seen in OA, but not in conjunction with point 1, above.
3. There are exposures or conditions at work that can exacerbate asthma. The exact causative agent might not always be identified, particularly when the treating clinician has neither the resources nor opportunity or access to investigate the patient's worksite. However, identifying the responsible agent can assist in confirming a suspected case of WEA, as well as contribute to prevention by identifying exposures that existing cases and unaffected individuals should avoid.
4. OA is unlikely. Someone with asthma is at risk for both OA and WEA, and as noted above, work-related symptoms are present in both types of cases. Further evaluation may be needed to determine whether a case of work-related symptoms is best classified as OA or WEA. If a patient with sensitizer-induced OA experiences exacerbation of symptoms because of reexposure to the original causative agent, this should be considered a recurrence of the OA rather than a new case of WEA. It is also possible for a person with nonoccupational asthma to become newly sensitized to a workplace material. The index of suspicion for this scenario should be raised in the context of work with potent sensitizers, such as isocyanates. In contrast, WEA would be a more appropriate diagnostic label if a person with asthma (even previous OA) experiences symptoms because of a workplace exposure to an irritant, in particular an irritant known or suspected to evoke an exaggerated response among persons with nonspecific airway hyperresponsiveness (*eg,* sulfur dioxide or chlorine). An individual with previous asthma in long-term remission whose disease recurs in relation to work is not automatically assumed to have WEA and could very well have OA.

Prevalence of WEA

The best estimates of WEA prevalence can be derived from studies conducted in general population or general health-care settings. These are summarized in Table 1. These studies differ by country, time frame for determining WEA, and case definitions, all of which could influence the estimate of WEA prevalence. The studies expressed WEA prevalence as a percentage of all adults *with asthma* and/or of working adults *with asthma*. The latter approach to the prevalence denominator was used in Table 1 when such data were available, as working adults with asthma are a more appropriate risk set for WEA. Considering together all 12 studies included in Table 1, the estimated prevalence of WEA ranges from a low of 13% to a high of 58%, with a median of 22.5% of adults with asthma. The first three studies in Table 1 relied on more objective criteria for WEA. The study by Bolen and colleagues[10] used a work-related pattern of PEFR to define WEA. Caldeira and colleagues[11] reviewed information collected by interview to decide which individuals with preexisting asthma had symptoms that were worsened by workplace exposures. Most cases of WEA in the third study listed in Table 1[12] fulfilled two criteria: self-reported work-related symptoms or medication use and the determination by an expert panel that the individual was exposed to asthma agents at work. The participants for the study by Bolen[10] came from the cohort for the third study. However, these two surveys used different criteria for WEA and were conducted approximately 2 years apart. These three estimates of WEA prevalence based on more objective criteria were 13%,[11] 14%,[10] and 24%,[12] with a median of 14%. In contrast, the nine studies in the second part of Table 1 defined WEA primarily using self-reports of asthma symptoms made worse by work. This second group had a broader range of prevalence estimates, from 18 to 58%, and a higher median of 25%.

Table 1—*Prevalence of WEA From Studies Conducted With Adults in General Population or General Health-Care Settings**

Reference	Country	Time Frame for WEA	No. of Asthma Cases	Criteria for WEA	Prevalence of WEA	
					In All Adults With Asthma	In Working Adults With Asthma
More Objective Criteria for WEA						
Bolen, Henneberger, Liang, et al, 2007[10]	United States	Current	95 (all employed)	Researchers determined that pattern of serial PEFR was consistent with WEA	NA	14%
Caldeira, Bettiol, Barbieri, et al, 2006[11]	Brazil	Ever	227	Researchers reviewed information from interview to determine if preexisting asthma was worsened by exposure at work	13%	NA
Henneberger, Derk, Sama, et al, 2006[12]	United States	Last 12 mo	598 (557 employed)	Combination of relevant exposure, as judged by researchers, and self-reported work-related symptoms or medication use	23%	24%
More Subjective Criteria for WEA						
Abramson, Kutin, Rosier, et al, 1995[13]	Australia	Ever	159	Self-reported respiratory symptoms at work associated with particular job	20%	NA
Blanc, Ellbjar, Janson, et al, 1999[14]	Sweden	Ever	160	Self-reported being at work ever makes chest tight or wheezy	38%	NA
Goh, Ng, Hong, et al, 1994[15]	Singapore	Current	802	Self-reported work environment is asthma trigger	27%	NA
Henneberger, Hoffman, Magid, et al, 2002[16]	United States	Current job	1,461	Self-reported current work environment makes asthma worse	25%	NA
Henneberger, Deprez, Asdigian, et al, 2003[17]	United States	Last 12 mo	42 (28 employed)	Self-reported coughing or wheezing is worse at work than away from work	14%	21%
Johnson, Dimich-Ward, Manfreda, et al, 2000[18]	Canada	Current job	106	Self-reported wheezing or dyspnea at or after work in current job	34% wheezing, 31% dyspnea	NA
Johnson, Toelle, Yates, et al, 2006[19]	Australia	Current	694 (all employed)	Self-reported asthma better on weekends or holidays	NA	18%
Mancuso, Rincon, Charlson, 2003[20]	United States	Current job	102	Self-reported asthma made worse by at least one of six current job conditions (carpeting, dust, temperature, cigarette smoke, no ventilation, chemical smells)	NA	58%
Saarinen,	Finland	Past	969 (all	Self-reported asthma	NA	20%

Karjalainen, Martikainen, et al, 2003[21]	month	employed)	symptoms caused or worsened by work at least weekly in past month
* NA = not applicable			

Occupational Risk Factors for Exacerbation of Asthma

As with exacerbation of asthma that is not work-related, WEA can result from any one of several exposures in workplaces. WEA is often associated with exposure to irritants, such as dusts, environmental tobacco smoke, paints, solvents, ammonia and other cleaning agents, and chemical fumes.[14,21-26] WEA and OA cases tend to be attributed to different types of exposures, with WEA less often caused by specific sensitizing agents.[23,26] The irritant exposures that exacerbate asthma are generally at much lower levels than when the same chemicals cause the onset of asthma, that is irritant-induced OA. Unfortunately, there is little quantitative evidence to determine what exposure levels are generally harmful vs safe regarding exacerbation of asthma symptoms. In addition, many nonchemical conditions that can be work-related are potential factors that could exacerbate asthma. These include viral infections (as with a day-care worker exposed to sick children at work), physical exercise, high or low temperatures, and stress.[9,12,21,22,25,26]

WEA can occur in workers with various types of jobs. Based on surveillance for work-related asthma in the United States, more than half of the reported WEA cases were from two occupational categories: technical, sales, and administrative support on the one hand and managerial and professional specialties as a second category.[23] The two occupations with the highest average annual incidence rates (*ie,* number of cases per workers with asthma per year) were (1) operators, fabricators, and laborers as a group and (2) service workers.[23] Studies have also been conducted in selected occupational groups with an elevated frequency of WEA, including those working at swimming pools,[27] with cosmetics,[28] in construction,[29] and in cleaning jobs,[30] as well as those serving in the military.[31] Even those employed in relatively "clean" environments can have problems with exacerbation of asthma where there has been water incursion and subsequent mold growth, as demonstrated in office buildings,[32] academic institutions,[33] and health-care centers.[34] In this scenario, WEA could be considered a subset of building-related illness, but should not be confused with "sick building syndrome."

Patients With WEA Compared With Other Asthma Patients

Several studies have compared the clinical characteristics of WEA cases with either OA cases or with other non-occupationally-related asthma patients. These published reports vary considerably in their methods, but generally have found that WEA and OA cases are often more similar than distinct. For example, researchers have observed these that these two types of work-related asthma cases to have similar levels of nonspecific airway hyperresponsiveness,[8,35] medication requirements,[35] asthma severity scores,[7,35] and need for medical care.[8] Both types of work-related asthma cases seem to benefit symptomatically when removed from exposure, although the follow-up improvement in airway hyperresponsiveness and medication requirements may be less for patients with WEA than for patients with OA that is immunologically mediated.[35]

When comparing WEA and non-work-related asthma, investigators have usually observed that cases of WEA are more severe. Specifically, those with WEA are more likely to experience exacerbations that lead to a specific treatment,[16,36] doctor visits,[8,36] emergency room treatment,[8,36] or hospital admission[8] than others with asthma unrelated to work. Two studies reported that patients with WEA had more symptomatic days than did those whose symptoms did not worsen at work, although their use of maintenance asthma medications was not different.[12,16] Other research suggests that individuals with WEA need ongoing asthma medication treatment more often than those with non-work-related asthma.[21]

In terms of socioeconomic consequences, WEA and OA cases have been observed to be associated with similar, extended periods of unemployment and loss of income related to asthma.[7,37] Studies that examined asthma-related job changes reported them to be either about the same in frequency for WEA and OA[35,37] or less common for WEA,[7,23,26] but not more common for WEA.

The findings for WEA vs non-work-related asthma are mixed for socioeconomic outcomes. Some studies have found that patients with WEA have more job changes[14,37] and more lost workdays[16] and are more likely to have a loss of income.[37] Other studies, however, have reported similarities between WEA and non-work-related asthma with respect to job changes,[9] lost workdays,[9,12,36] and rates of unemployment.[36,37] With respect to patient-centered implications of WEA, a recent study found that persons with WEA had a lower quality of life compared with patients whose asthma was not related to work.[38]

Clinical Management of Patients With WEA

A consensus document on work-related asthma that was published recently by the ACCP includes guidance on how to diagnose and manage WEA.[25] Several key points from that document are summarized in the following paragraphs. According to those guidelines, with any suspected case of WEA it is important to confirm that the person truly has asthma, because the symptoms might be related to other conditions, such as vocal cord dysfunction or irritation of the upper airways. WEA can occur in those with a prior diagnosis of OA, so long as the work-related worsening is not attributed to the original agent that caused OA. That document also emphasizes the role of objective criteria, including determining work-related patterns in serial PEFR measurements.

Because both OA and WEA involve worsening of asthma in relationship to work, clinical management can also involve investigation to further differentiate one from the other. With established active cases of asthma that experience an abrupt exacerbation of symptoms related to work, it can be relatively easy to reach a determination of WEA. With patients who have experienced work-related symptoms for a prolonged period of time, distinguishing between OA and WEA can be more difficult and may require referral to a specialist (*eg,* an occupational medicine or pulmonary physician with expertise in occupational respiratory diseases).

Determining the causative agent helps to confirm the suspected case, better informs patient management regarding what exposures should be avoided, and guides efforts to limit that exposure in the workplace to prevent similar problems in coworkers. Atypical episodic exposures relating to (1) chemical releases or building renovations or (2) the introduction of new processes or materials can make it easier to identify the likely cause of WEA. As already noted, a wide range of exposures can exacerbate asthma and these can often be assessed by taking a careful occupational history. The patient can assist with this process by obtaining a material safety data sheet, or MSDS, for each chemical or product at work. Other sources of information to consider are published reports about problems in similar workplaces, symptoms among the patient's coworkers, and exposure-monitoring reports from industrial hygiene investigations conducted in the workplace. A visit to the workplace can help in gathering this information, bearing in mind that the permission must first be obtained from both the patient and the employer. Unfortunately, often such a visit is not a realistic option for clinicians given considerations of cost, time, and access, as noted previously. The possible contribution of exposures outside work, such as environmental allergies, should also be considered. Immunologic tests for responsiveness to specific occupational and nonoccupational allergens can help in identifying the causative exposure, but used alone are unlikely to confirm or exclude suspected WEA. Unfortunately, the clinician cannot always isolate a single cause of WEA. Jobs can involve exposures that are unknown to the worker and possibly the employer as well. Worksite investigations of exposures are rare and do not always yield clear results. Mixed exposures also can frustrate plans to identify a single causative agent.

The treatment of WEA involves both optimizing the standard medical management of asthma and enlisting the help of the patient and employer (with the patient's permission) to limit aggravating exposures. The reduction or elimination of occupational exposures can prove difficult, as the patient often does not have ultimate control over the determinants of such exposures (for example, the chemicals or ventilation equipment used). Although difficult to attain in practice, limits on exposures can often allow a patient with WEA to remain working in the same job (whereas in OA, complete cessation of exposure may be central to the optimal management of the condition). Nonetheless, in more severe cases of asthma with continuing exacerbating exposures at work, job change may ultimately be necessary.

The management of a case of work-related asthma should include support of the patient's claim for compensation when appropriate. Compensation systems differ among nations (as well as among states

and provinces within nations) in their case criteria and benefits for work-related asthma. Also, while several systems compensate for OA, fewer provide benefits for WEA. Another aspect of case management is submitting reports to surveillance programs. Many countries conduct surveillance for work-related asthma that relies on voluntary or mandatory reporting by treating clinicians.[39] Health officials use these reports of WEA and OA to track the number and characteristics of cases over time, guide interventions to prevent further cases, and document the impact of prevention efforts.

References

1. Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System Survey Data. Atlanta, GA: Department of Health and Human Services, 2007
2. National Center for Health Statistics. Asthma prevalence, health care use, and mortality: United States, 2003-05: Department of Health and Human Services, 2007
3. Cisternas MG, Blanc PD, Yen IH, et al. A comprehensive study of the direct and indirect costs of adult asthma. J Allergy Clin Immunol 2003; 111:1212-1218
4. Rabe KF, Adachi M, Lai CKW, et al. Worldwide severity and control of asthma in children and adults: the global asthma insights and reality surveys. J Allergy Clin Immunol 2004; 114:40-47
5. Jajosky RA, Harrison R, Reinisch F, et al. Surveillance of work-related asthma in selected U.S. states using surveillance guidelines for state health departments--California, Massachusetts, Michigan, and New Jersey, 1993-1995. MMWR CDC Surveill Summ 1999; 48:1-20
6. Buyantseva LV, Liss GM, Ribeiro M, et al. Lost workdays due to work-aggravated asthma in Ontario [abstract]. Proc Am Thoracic Soc 2008; 5:A527
7. Larbanois A, Jamart J, Delwiche JP, et al. Socioeconomic outcome of subjects experiencing asthma symptoms at work. Eur Respir J 2002; 19:1107-1113
8. Lemiere C, Forget A, Dufour MH, et al. Characteristics and medical resource use of asthmatic subjects with and without work-related asthma. J Allergy Clin Immunol 2007; 120:1354-1359
9. Tarlo SM, Leung K, Broder I, et al. Asthmatic subjects symptomatically worse at work: prevalence and characterization among a general asthma clinic population. Chest 2000; 118:1309-1314
10. Bolen AR, Henneberger PK, Liang X, et al. The validation of work-related self-reported asthma exacerbation. Occup Environ Med 2007; 64:343-348
11. Caldeira RD, Bettiol H, Barbieri MA, et al. Prevalence and risk factors for work related asthma in young adults. Occup Environ Med 2006; 63:694-699
12. Henneberger PK, Derk SJ, Sama SR, et al. The frequency of workplace exacerbation among health maintenance organisation members with asthma. Occup Environ Med 2006; 63:551-557
13. Abramson MJ, Kutin JJ, Rosier MJ, et al. Morbidity, medication and trigger factors in a community sample of adults with asthma. Med J Aust 1995; 162:78-81
14. Blanc PD, Ellbjar S, Janson C, et al. Asthma-related work disability in Sweden: the impact of workplace exposures. Am J Respir Crit Care Med 1999; 160:2028-2033
15. Goh LG, Ng TP, Hong CY, et al. Outpatient adult bronchial asthma in Singapore. Singapore Med J 1994; 35:190-194
16. Henneberger PK, Hoffman CD, Magid DJ, et al. Work-related exacerbation of asthma. Int J Occup Environ Health 2002; 8:291-296
17. Henneberger PK, Deprez RD, Asdigian N, et al. Workplace exacerbation of asthma symptoms: findings from a population-based study in Maine. Arch Environ Health 2003; 58:781-788
18. Johnson AR, Dimich-Ward HD, Manfreda J, et al. Occupational asthma in adults in six Canadian communities. Am J Respir Crit Care Med 2000; 162:2058-2062
19. Johnson A, Toelle BG, Yates D, et al. Occupational asthma in New South Wales (NSW): a population-based study. Occup Med (Lond) 2006; 56:258-262
20. Mancuso CA, Rincon M, Charlson ME. Adverse work outcomes and events attributed to asthma. Am J Ind Med 2003; 44:236-245
21. Saarinen K, Karjalainen A, Martikainen R, et al. Prevalence of work-aggravated symptoms in clinically established asthma. Eur Respir J 2003; 22:305-309
22. Berger Z, Rom WN, Reibman J, et al. Prevalence of workplace exacerbation of asthma symptoms in an urban working population of asthmatics. J Occup Environ Med 2006; 48:833-839
23. Goe SK, Henneberger PK, Reilly MJ, et al. A descriptive study of work aggravated asthma. Occup Environ Med 2004; 61:512-517
24. McClellan VE, Garrett JE. Asthma and the employment experience. NZ Med J 1990; 103:399-

401
25. Tarlo SM, Balmes J, Balkissoon R, et al. Diagnosis and management of work-related asthma: American College Of Chest Physicians Consensus Statement. Chest 2008; 134:1S-41S
26. Tarlo SM, Liss G, Corey P, et al. A workers' compensation claim population for occupational asthma: comparison of subgroups. Chest 1995; 107:634-641
27. Jacobs JH, Spaan S, van Rooy GB, et al. Exposure to trichloramine and respiratory symptoms in indoor swimming pool workers. Eur Respir J 2007; 29:690-698
28. Kreiss K, Esfahani RS, Antao VC, et al. Risk factors for asthma among cosmetology professionals in Colorado. J Occup Environ Med 2006; 48:1062-1069
29. Sauni R, Oksa P, Vattulainen K, et al. The effects of asthma on the quality of life and employment of construction workers. Occup Med (Lond) 2001; 51:163-167
30. de Fatima Macaira E, Algranti E, Medina Coeli Mendonca E, et al. Rhinitis and asthma symptoms in non-domestic cleaners from the Sao Paulo metropolitan area, Brazil. Occup Environ Med 2007; 64:446-453
31. Gouge SF, Daniels DJ, Smith CE. Exacerbation of asthma after pyridostigmine during Operation Desert Storm. Mil Med 1994; 159:108-111
32. Cox-Ganser JM, White SK, Jones R, et al. Respiratory morbidity in office workers in a water-damaged building. Environ Health Perspect 2005; 113:485-490
33. Park JH, Schleiff PL, Attfield MD, et al. Building-related respiratory symptoms can be predicted with semi-quantitative indices of exposure to dampness and mold. Indoor Air 2004; 14:425-433
34. Cox-Ganser J, Rao CY. Benefis Healthcare, Great Falls, Montana. Cincinnati, OH: U.S. Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, National Technical Information Service, 2001
35. Pelissier S, Chaboillez S, Teolis L, et al. Outcome of subjects diagnosed with occupational asthma and work-aggravated asthma after removal from exposure. J Occup Environ Med 2006; 48:656-659
36. Breton CV, Zhang Z, Hunt PR, et al. Characteristics of work related asthma: results from a population based survey. Occup Environ Med 2006; 63:411-415
37. Cannon J, Cullinan P, Newman Taylor A. Consequences of occupational asthma. BMJ 1995; 311:602-603
38. Lowery EP, Henneberger PK, Rosiello R, et al. Quality of life of adults with workplace exacerbation of asthma. Qual Life Res 2007; 16:1605-1613
39. Bakerly ND, Moore VC, Vellore AD, et al. Fifteen-year trends in occupational asthma: data from the Shield surveillance scheme. Occup Med (Lond) 2008; 58:169-174

Lesson 13, Volume 23—Work-Exacerbated Asthma
Poststudy Questions

1. Which of the following statements regarding case definitions for work-exacerbated asthma (WEA) is true?
   A. Surveillance for work-related asthma suggests that most physicians use pulmonary function testing to confirm the relationship between asthma and work
   B. To date, the same case definition for WEA has been used by nearly all clinicians and researchers
   C. Case definitions for WEA are rarely influenced by prevailing standards of care and medicolegal considerations
   D. Definitions of WEA used in epidemiologic studies or surveillance systems are often more sensitive than those used in clinical settings
   E. The definition of WEA is primarily concerned with identifying the exposure, whether at work or elsewhere, that was responsible for the initial onset of asthma

2. The case definition of WEA includes all the following *except*:
   A. The workplace exposure is linked in time to the exacerbation of asthma
   B. Those at risk for WEA have pre-existing or concurrent (coincident) asthma
   C. There are conditions at work that can cause exacerbation of asthma

D. The exact chemical at work that causes WEA is determined conclusively
E. Occupational asthma is unlikely

3. Estimates of the prevalence of WEA from studies conducted in general population or general health-care settings can be characterized by which one of the following?
A. Range from a minimum of 15% to a maximum of 25%
B. Are still unavailable for countries outside North America
C. When considered all together, have a median value in the range of 20 to 25%
D. Are rarely based on case definitions that rely on self reports of work-related respiratory symptoms
E. Are higher when more objective criteria are used to define WEA

4. Which of the following statements about exposures that can exacerbate asthma at work is *false*?
A. They can include nonchemical conditions, such as extreme temperatures or physical exercise
B. Common workplace asthma triggers have been studied enough to set cutoff levels below which workers with asthma are not bothered
C. Exposures are often to irritant chemicals
D. They can be some of the same exposures that trigger asthma at home or in other settings, including secondhand smoke and cleaning agents
E. The exposures can be temporary, such as dusts and gases from building renovations

5. Regarding the industries and occupations that pose a risk for WEA, which one of the following is true?
A. They can include relatively "clean" workplaces, such as schools, health-care centers, and office buildings
B. They are characterized by having exposures only to high-molecular-weight sensitizing agents
C. They do not include the cleaning profession, which really is "clean" and does not pose a risk for WEA
D. They have not been identified in studies yet
E. They are limited to "dirty" trades, such as manufacturing

6. In general, OA and WEA cases have been observed to be similar, if not always exactly alike, with respect to which of the following?
A. Asthma severity
B. Requirement for asthma medication
C. Nonspecific airway hyperresponsiveness
D. Improvement in symptoms when removed from exposure
E. All of the above

7. Which of the following statements is true about the socioeconomic and psychosocial consequences of WEA?
A. Loss of income is about the same for WEA and non-work-related asthma
B. The frequency of asthma-related job changes is consistently the same for WEA and OA in studies that measured this outcome
C. Loss of income is usually worse for OA than WEA
D. A recent study reported that quality of life is lower for WEA compared with asthma unrelated to work
E. All of the above

8. Which of the following statements about evaluating a suspected case of WEA is *false*?
A. It is important to ascertain that the patient truly has asthma, and that his/her symptoms are not related to some other condition, such as upper airway irritation
B. Ultimately, a single test reveals whether the person has WEA
C. WEA and OA cases can both present with work-related asthma symptoms, and further investigation is needed to differentiate one from the other

D. With more difficult cases, it might be necessary to refer a patient and have a specialist determine whether the patient has WEA or OA
E. All of the above

9. Which of the following statements is true about an exposure that causes a patient's WEA?
A. Knowledge of this exposure can be used in managing the case by identifying which exposures should be avoided
B. Knowledge of this exposure can be used to alert the employer (with the patient's permission) to an agent that might cause problems for other workers who are similarly exposed
C. The treating physician might never identify the causative exposure
D. This responsible exposure might actually be a mix of different chemicals, making it difficult to identify a single causative agent
E. All of the above

10. Management of a WEA case should *always* include which of the following?
A. Advising the patient to leave his or her current job
B. Referring the patient to a specialist
C. Optimizing the standard medical management of asthma
D. Ensuring that the employer does not learn which workplace exposure is responsible for the WEA
E. Advising the patient to minimize contact with coworkers when not at work

Answers to Poststudy Questions

1. **D**
2. **D**
3. **C**
4. **B**
5. **A**
6. **E**
7. **D**
8. **B**
9. **E**
10. **C**

DEPARTMENT OF HEALTH & HUMAN SERVICES

National Institute for
Occupational Safety and Health (NIOSH)

Memorandum

Date: July 8, 2009

From: Epidemiologist, FSB, DRDS

Subject: Reprint of Publication

To: Deputy Director, DRDS
Thru: Chief, FSB
Team Leader, FSB

Attached is a reprint of an article I co-authored that recently appeared in *Pulmonary and Critical Care Updates*. The DRDS clearance number is 2009-066M. **Work Exacerbated Asthma.** It is provided for your retention.

REFERENCE:

Henneberger, P.K., Work Exacerbated Asthma. *Pulmonary and Critical Care Updates.* Lesson 13,Vol.23.(2009).

Thank you,

Paul K. Henneberger, MPH, ScD

Attachment:

cc:
Director, DRDS (3)
Director, EID (3)
Librarian, ALOSH (2)
Chief, FSB,DRDS (1)
Chief, LRB,DRDS (1)
Chief, SB,DRDS (1)