ORIGINAL ARTICLE

The frequency of workplace exacerbation among health maintenance organisation members with asthma

P K Henneberger, S J Derk, S R Sama, R J Boylstein, C D Hoffman, P A Preusse, R A Rosiello, D K Milton

Occup Environ Med 2006;**63**:551–557. doi: 10.1136/oem.2005.024786

See end of article for authors' affiliations

Correspondence to: Dr P Henneberger, NIOSH/CDC, M/S H2800, 1095 Willowdale Road, Morgantown, WV 26501, USA; pkh0@cdc.gov

Accepted 3 April 2006
Published Online First 6 April 2006

Objectives: Workplace conditions can potentially contribute to the worsening of asthma, yet it is unclear what percentage of adults with asthma experience workplace exacerbation of symptoms. The objective of this investigation was to determine the prevalence of workplace exacerbation of asthma (WEA).
Methods: Adults with asthma aged 18–44 were enrolled into the baseline survey of a longitudinal study. Members of a health maintenance organisation were considered candidates for participation if they fulfilled membership, diagnostic, and treatment criteria based on automated review of electronic billing, claims, and pharmacy records. Diagnosis and treatment were confirmed by manual review of medical records. A telephone questionnaire was administered. A work related symptom score was assigned to each participant based on responses to questions about work related asthma symptoms, medication use, and symptom triggers. Blinded to participants' answers to these questions, two researchers independently reviewed the self-reported work histories and assigned exposure ratings. A final exposure score was then calculated. Participants with sufficient evidence for work related symptoms and exposure were classified as having WEA.
Results: Of the 598 participants with complete data, 557 (93%) were working, and 136 (23%) fulfilled the criteria for WEA. Those with WEA were more likely to be male and to report that they had been bothered by asthma symptoms during the past seven days.
Conclusions: Workplace exacerbation of asthma was common in this study population, occurring in over a fifth of these adults with asthma. Physicians should consider that work can contribute to the exacerbation of symptoms when treating adults with asthma.

Work related asthma is the most common non-asbestos lung disease seen in occupational health clinics in the United States,[1 2] as well as the most common occupational lung disease in many industrialised countries.[3 4] Researchers in the US have estimated that from 3% to 21% of asthma among adults is attributable to workplace exposures.[5–9] A recent review of the relevant literature by a committee of the American Thoracic Society concluded that 15% of asthma among adults can be attributed to occupation.[9] Work related asthma includes individuals with new onset asthma caused by workplace exposure to sensitisers or irritants, as well as those with pre-existing asthma that is exacerbated by workplace exposures.[10]

The prevalence of workplace exacerbation of asthma (WEA) has been investigated using a variety of data sources. In the province of Ontario in Canada, the Worker Compensation Board reported that half of all asthma claims received between 1984 and 1988 involved exacerbation.[11 12] Cohorts of patients with occupational asthma treated in occupational and environmental medicine clinics in Massachusetts and Washington included 18% and 27%, respectively, with WEA.[13 14] The Sentinel Event Notification System for Occupational Risks (SENSOR) is a case based surveillance programme that has been applied to different diseases in the US. From SENSOR activities in four states during 1993–95, 19% of work related asthma cases were considered to be work aggravated asthma, with the rest classified as new onset cases.[10]

The prevalence of WEA can also be expressed as a percentage of all cases of adult asthma, an estimate that can be derived by conducting population based (or quasi-population based) studies. From a community based sample of adults in Australia, 20% of respondents with asthma stated that their symptoms were worse as the result of work.[15] From a study in Norway, 33% of the men and 18% of the women with asthma reported that they had ever had respiratory symptoms associated with work that abated on weekends or holidays.[16] From an interview survey of adults with asthma enrolled in a health maintenance organisation (HMO) in the state of Colorado in the US, approximately 25% of the 1461 participants reported that their asthma was made worse by their current work environment.[17] From a population based survey conducted in the state of Maine, 25% of 64 adults with asthma reported that their coughing or wheezing was worse at work.[18]

Although the existing population based surveys were informative, an in-depth study that used a more rigorous case definition was needed to provide a more accurate estimate of the prevalence of WEA and a better understanding of the circumstances that contributed to this problem. This additional information could then be used to plan preventive actions. Therefore, in 2000, the National Institute for Occupational Safety and Health (NIOSH) initiated the Workplace Exacerbation of Asthma Project to

Abbreviations: FCHP, Fallon Community Health Plan; HMO, health maintenance organisation; HSRB, Human Subjects Review Board; ICD-9, International Classification of Diseases, Ninth Revision; NIOSH, National Institute for Occupational Safety and Health; SENSOR, Sentinel Event Notification System for Occupational Risks; WEA, workplace exacerbation of asthma

investigate the frequency, causes, and consequences of WEA. This project was divided into three studies conducted over a five year period. Adults with asthma were enlisted and interviewed in the baseline study, and were re-interviewed approximately two years later in the follow up study. The validation study was intended to validate self-reported work related asthma symptoms in a subset of baseline study participants. Consistent with the intention of conducting a population based study, NIOSH contracted with a research department in an HMO to implement data collection.

In the current analyses, we used data from the baseline study to determine what percentage of adults with asthma fulfilled criteria for WEA.

METHODS

Human subjects review and approval

The NIOSH Human Subjects Review Board (HSRB) reviewed and approved the research protocol, as did the HSRB of the contracted HMO, Fallon Clinic, Inc, Research Department in Eastern and Central Massachusetts.

Identification of participants

Potential participants were selected from members of the Fallon Community Health Plan (FCHP). Almost all (99%) FCHP members were enrolled into the plan without health screening. Most (83%) entered as part of an employer based programme, but an additional 16% were Medicare or Medicaid patients. Study participants were enrolled if they met criteria applied using both automated review of electronic billing, claims, and pharmacy records, and manual chart review. These methods and their validity are discussed in a previous publication.[19]

1. Electronic records were scanned each month for 16 consecutive ''index'' months (that is, September 2000 to December 2001) to identify potential participants who met age and FCHP membership criteria:

(a) adult aged 18–44 years in index month

(b) enrolled with the FCHP for at least six months prior to the index month.

2. Next, exclusion criteria were applied to electronic records from the previous 12 months to identify the at-risk population. People were excluded if there was a recorded diagnosis of congestive heart failure (International Classification of Diseases, Ninth Revision (ICD-9) codes 428.0–428.9), bronchiectasis (ICD-9 494), emphysema (ICD-9 492.0–492.8), pulmonary embolism (ICD-9 415.0–415.9), or pulmonary hypertension (ICD-9 416.0–416.9). People were also excluded if they did not have one of the following in the previous 12 months: emergency room visit with a primary diagnosis of asthma (ICD-9 493.0–493.91), hospital admission with a primary diagnosis of asthma, diagnosis of dust pneumonopathy (ICD-9 504–507), diagnosis of red cedar asthma (ICD-9 495.88), diagnosis of detergent asthma (ICD-9 507.8), asthma diagnosis and outpatient nebulisation treatment, dispensing of any one of several types of long acting asthma controller medications (for example, beta-agonist, leukotriene inhibitor, theophylline, corticosteroid), diagnosis of asthma and dispensing of oral steroids, asthma diagnosis and dispensing of four or more beta-agonist metered dose inhalers.

3. The electronic records from the index month for the at-risk population were then scanned to identify which potential participants were receiving current treatment for asthma, according to whether they met one of the following criteria:

(a) emergency room visit for asthma (ICD-9 493.0–493.91), or

(b) hospital admission with a primary diagnosis of asthma, or

(c) outpatient diagnosis of asthma accompanied by one of the following:

i. dispensing of a minimum of one beta-agonist inhaler, or

ii. dispensing of a beta-agonist inhaler with theophylline, or

iii. dispensing of a steroid or cromolyn inhaler, or

iv. dispensing of an oral steroid taper, or

v. an outpatient treatment with intravenous theophylline, or

vi. an outpatient treatment with nebulizer, or

vii. dispensing of a nebulizer to relieve airway obstruction, or

viii. dispensing of a leukotriene inhibitor.

(d) diagnosis of dust pneumonopathy (ICD-9 504–507), or

(e) diagnosis of red cedar asthma (ICD-9 495.88), or

(f) diagnosis of detergent asthma (ICD-9 507.8), or

(g) dispensing of any one of several types of long acting asthma controller medications (for example, beta-agonist, leukotriene inhibitor, theophylline, corticosteroid), with or without a diagnosis of asthma.

4. A research nurse manually reviewed the medical records of all potential study participants to determine if there was a confirmed diagnosis of asthma with onset prior to the past year. During this review of records, the research nurse abstracted demographic and medical data, and assigned a level of severity.[20] The four levels of severity were mild intermittent, mild persistent, moderate persistent, and severe persistent.

The contractor contacted potential participants meeting criteria 1–4 by mailing each one a letter which explained the study. The letter was followed by a telephone call approximately two weeks later. Interviewing was conducted March 2001 through August 2002. Interviewers attempted to reach each potential respondent seven times at various times during the day and week before giving up. The next selection criterion was determined early in the telephone interview.

5. When reached by telephone, the potential participant reported ''yes'' to both of the following questions:

(a) Have you ever had asthma?

(b) Have you taken any medication for asthma or other breathing problems within the past 12 months?

If the potential participant provided the ''yes'' answers specified in criterion 5, the interviewer continued the telephone call by reviewing the purpose of the study, answering questions, and asking the individual to provide verbal informed consent. To compensate participants for their time, an honorarium of US$20 was provided to each person who completed the questionnaire. Once efforts to contact someone were terminated, a code was assigned to indicate the final call disposition, such as questionnaire completed, refused to participate, or could not contact.

As a study of the exacerbation of existing asthma, it was necessary to ensure that participants did not have recent onset of their asthma. This was addressed during the review of medical records, in which we ascertained that onset was prior to the past year (criterion 4). To be certain that the person's asthma did not start during the past year, we required the following based on information gathered during the interview:

6. Participant reported that the date of their first asthma attack was at least 14 months before the date of interview.

Telephone interviews

The baseline questionnaire incorporated items from several other questionnaires.[21 22] Responses to questions provided information on asthma history (for example, date of onset and suspected cause of asthma), severity of current asthma, and the asthma-work association. A series of validated questions were included to measure the social impact of

asthma.[23] Also, respondents were asked to provide detailed descriptions of jobs they had held during the previous 12 months.

We used a computerised assisted telephone interview system to administer the baseline questionnaire. Before starting the interviews, the NIOSH project manager trained six interviewers in basic interviewing techniques. Topics of instruction included the interviewers' role in the project, asking the questions, clarifying the questions, and probing for appropriate responses. All interviews were recorded on audiotape so that the NIOSH project manager could monitor the quality and accuracy of the data collection. After an interviewer was trained, the project manager reviewed the audiotapes from the first 10 questionnaires administered by the interviewer. Initial problems included not reading the questions as written and insufficient probing for appropriate responses. After the initial review of 10 questionnaires for each interviewer, a 20% sample of the remaining completed questionnaires were reviewed. The findings from these monitoring efforts were communicated back to the contractor's project manager in a timely manner in an effort to improve the interviewers' performance.

Criteria for workplace exacerbation of asthma

The definition of workplace exacerbation of asthma was based on a self-report of work related worsening of symptoms and a judgment by an expert panel that the participant was likely to have had exposure to asthmagenic agents (for example, sensitisers and irritants) at work.

A work related symptom score (0–3) was assigned to each participant based on responses to questionnaire items concerning the previous 12 months. One point was assigned for each of the following that were met: (1) asthma symptoms got better on weekends, vacations, or other times when away from work; (2) use of an inhaler or nebulizer was greater on work days; and (3) in response to an open-ended question about asthma triggers, the participant identified conditions at work that set off asthma symptoms or breathing trouble.

Review of the data revealed problems with how participants responded to the open-ended question about asthma triggers. In particular, some participants listed conditions that made their asthma worse, and then stated that these conditions bothered them everywhere they went, including at work. This broad assignment of location was less precise than desired, so two of the researchers independently re-evaluated the responses and assigned each reported work related trigger to one of five categories: (1) traditional work related asthma agents (for example, chemicals, dust, fumes, molds); (2) physical work related agents (for example, physical exertion, cold air, humidity, temperature change); (3) stress at work; (4) second hand smoke at work; (5) an infection or non-occupational allergy. After independently evaluating each of the responses, the two researchers compared their results and resolved disagreements on a case-by-case basis. Any work related triggers in the first four categories resulted in a ''yes'' response for the new workplace trigger variable. The results of this validation effort were the third element used to construct the final work related symptom score for each employed participant.

An industrial hygienist and an epidemiologist independently reviewed the detailed job descriptions and assigned exposure scores to each job held during the 12 months before interview. The description of each job was based on responses to 29 items in the questionnaire. The information gathered included job title and tasks, what the company did or manufactured, ventilation in the worksite, responses to an open-ended question about chemicals and materials used or contacted at work, and responses to a series of close-ended questions about different types of exposures and conditions at work that could contribute to the exacerbation of asthma. The reviewers were blinded to any other information about the participant, including responses used to determine the work related symptom score. Both reviewers were familiar with asthma agents that occur in occupational settings. They relied on various reference materials to inform their judgments, including a published list of potential agents[24] and a list of occupational asthmagens compiled by the Association of Occupational and Environmental Clinics that is available on the internet.[25]

Table 1 Protocol for assigning subscores for exposure to asthma agents

Score	Probability	Intensity	Frequency
0	<50%	Low	Once per week or less
1	≥50% but <80%	Moderate	Some part of most days
2	≥80%	High	Most of the time

Ratings were assigned separately for sensitiser and irritant exposures and ranged from 0 to 2. Three subscores were assigned, one each for probability, intensity, and frequency of exposure. The subscores followed the criteria presented in table 1. To arrive at a score for either sensitisers or irritants, the three subscores were tallied. The sum of subscores and the corresponding score were: sum = 0 or 1 then score = 0; sum = 2 or 3 then score = 1; sum = 4, 5, or 6 then score = 2. The scores from the two reviewers were added together to achieve separate summary scores for sensitisers and irritants. The sum of scores and corresponding summary score were: sum = 0 then summary score = 0; sum = 1 or 2 then summary score = 1; sum = 3 or 4 then summary score = 2. The final exposure score was the higher of the summarised sensitiser and irritant scores.

Our decision matrix for a work related pattern is closely modeled after the approach developed by Milton and colleagues.[8] The final exposure score and work related symptom score were cross tabulated to determine strength of evidence for WEA. If someone had worked more than one job in the past 12 months, the exposure score was used for the most recent job or the job worked at most if there were multiple current jobs. Cases were classified as having no, weak, moderate, or strong evidence for workplace exacerbation based on a decision matrix. Participants with moderate or strong evidence were classified as having WEA. This group included all the participants with both exposure and symptom scores greater than 0, and those with a combination symptom/exposure score of 3/0.

Statistics

Data analyses were conducted using SAS statistical software for personal computers.[26] To test for statistical significance, we used χ^2 for comparisons involving categorical data, and in particular the continuity corrected χ^2 for data in 2-by-2 tables.[27] We used Student's t test for comparisons involving continuous data.[27] p values less than or equal to 0.05 were considered statistically significant.

RESULTS

There was a monthly average of approximately 48 000 people aged 18–44 years enrolled in the HMO during the study period. Based on review of the electronic patient records, 1251 individuals met selection criteria 1–3, of whom 978 also met criterion 4 that was based on manual chart review. The interviewers were not able to reach 322 (33%) of the 978 by telephone after seven attempts. Of the 656 contacted by telephone, 640 (65% of 978) agreed to participate and 16

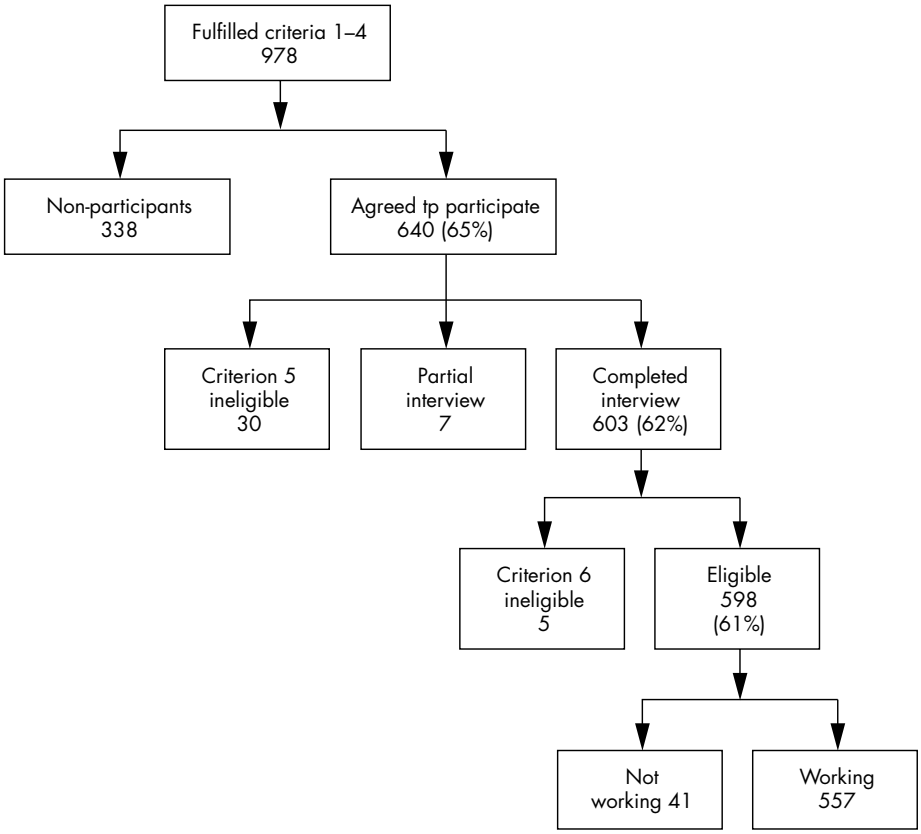

Figure 1 Disposition of candidates for study.

refused (fig 1). Another 30 potential participants reported they did not have asthma or did not use medications for asthma in the past 12 months (that is, did not fulfill selection criterion 5) and were not interviewed. Of the 610 who started the interview, seven completed only part of the baseline questionnaire and five were excluded due to recent onset of asthma (criterion 6). Data from the remaining 598 participants (that is, 61% of the 978 who fulfilled selection criteria 1–4) were included in the following statistical analyses and will be called participants.

The 380 non-participants were similar in age to the participants, but more likely to be male (44% *v* 31%, $p<0.0001$). Members of both groups were about equally likely to have moderate or severe asthma (that is, 39% of participants, 38% of non-participants). However, the participants had proportionately more in the mild persistent category (30% *v* 23%) and fewer in the mild intermittent category (30% *v* 39%) than the non-participants ($p = 0.02$).

The study participants were overwhelmingly white (95%) and only 6% reported Hispanic ethnicity. Women (69%) outnumbered men (31%) by more than 2 to 1. By design, their ages ranged from 18 to 44 years in the index month when they were first identified as candidates for study, but one of the participants had attained the age of 45 by the time he was interviewed. So, the 598 study participants ranged in age from 18 to 45 years when interviewed, with a mean of 32.8 years (SD 7.8). The participants included 19% current and 23% former cigarette smokers, and 37% had completed college. Of the 446 who answered the question about income, 52% earned a gross weekly salary of at least $550.

The age at onset of asthma ranged from 1 to 42 years, the mean was 15.5 (SD 10.8), and 366 (61%) participants had onset before the age of 18. A family history of asthma was common, with 183 (31%) reporting that either their mother or father had asthma. When asked what they thought caused their asthma, many participants replied heredity, infections,

Table 2 Percentage of working participants judged to have had exposure to sensitisers and/or irritants at work, by age and gender*

Age (years)	Male	Female
18–25	62% (26/42)	40% (36/91)
26–35	61% (39/64)	37% (42/113)
36–45	61% (45/74)	43% (75/173)
Total	61% (110/180)	41% (153/377)

*$p \leqslant 0.05$ in each age category comparing males to females.

Table 3 Distribution of 598 baseline study participants by strength of evidence for workplace exacerbation of asthma

Exposure score†	work related symptom score 0	1	2	3	Total
0	215‡ None	92 Weak	15 Weak	13 Moderate*	335
1	121 Weak	66 Moderate*	22 Moderate*	11 Strong*	220
2	19 Weak	19 Moderate*	3 Strong*	2 Strong*	43
Total	355	177	40	26	598

*23% (n = 136) of the 598 study participants had moderate or strong evidence, and were classified as having workplace exacerbation of asthma.
†Based on the higher of two scores for sensitiser and irritant agents, as independently assigned by two researchers after review of each participant's work history for the 12 months before interview.
‡Includes 41 participants who were unemployed during the 12 months before interview.

Table 4 Comparison of study participants with and without workplace exacerbation of asthma

Descriptive variables*	Workplace exacerbation of asthma		
	Yes (n = 136)	No (n = 462)	p Value
Race, % white	96%	94%	0.44
Ethnicity, % Hispanic	7%	6%	0.91
Gender, % male	45%	27%	<0.0001
Age in years, mean (SEM)	33.3 (0.7)	32.6 (0.4)	0.35
Education, % college degree or more	31%	38%	0.15
Salary, % gross weekly income ⩾$550	47%	53%	0.32
Cigarette smoking, %			0.27
Never	53%	61%	
Former	26%	21%	
Current	21%	18%	
Asthma onset before age 18 years, %	58%	62%	0.45
Asthma severity based on medical records, %			0.32
Mild intermittent	28%	31%	
Mild persistent	27%	31%	
Moderate or severe	45%	38%	
No of days bothered by asthma in past 7 days, mean (SEM)	4.0 (0.2)	3.1 (0.1)	0.002
No of treatments for acute asthma attacks in past 12 months, mean (SEM)	1.4 (0.2)	1.1 (0.1)	0.15
No of days missed work due to asthma in past 12 months, mean (SEM)†	2.8 (0.7)	1.9 (0.3)	0.24

*There were missing values for several variables. Some participants refused to answer questions about their race (n = 2), Hispanic ethnicity (n = 2), and education (n = 1). For gross weekly salary, 446 provided answers, while the other 152 had no salary (for example, unemployed or student) or refused to answer.
†Limited to the 557 participants who were employed in the past 12 months.

or cigarette smoke. Among the 203 who were employed at the time of asthma onset, 20 (10%) reported they thought it was due, at least in part, to workplace exposures.

A total of 263 participants (44% of 598) had an exposure score >0. More participants were judged to have had exposure to sensitisers alone (n = 91, 15% of 598) than to irritants alone (n = 69, 12%), and 103 (17%) had exposure to both kinds of agents. The likelihood of having an exposure score >0 varied little with age (table 2). However, men were more likely than women to have worked in jobs that were judged to have had sensitiser and/or irritant exposures, regardless of age ($p \leqslant 0.05$).

The distribution of study participants by the decision matrix is presented in table 3, which includes 557 (93% of 598) participants who were employed in the year before interview, and 41 who were not employed and assigned to the combined exposure/symptom category of 0/0. There were 243 participants (41% of 598) with a work related symptom score >0, and they included 73 (12% of 598) who reported their asthma symptoms got better away from work, 57 (10%) who reported they used an inhaler or nebulizer more at work, and 205 (34%) who reported an asthma trigger at work. About two thirds of the 243 (n = 158) reported an asthma trigger but neither work related symptoms nor medication use. The percentage of working participants with a symptom score >0 increased with the exposure score: 41% (120/294) for exposure = 0, 45% (99/220) for exposure = 1, and 56% (24/43) for exposure = 2, with p = 0.07 by χ^2 test for trend.

A total of 136 (23% of 598) participants had either moderate or strong evidence for workplace exacerbation and, consequently, were classified as having WEA. The WEA participants included 123 (90% of 136) with both exposure and symptom scores >0, and 13 (10%) with a combined symptom/exposure score of 3/0. Approximately half (n = 66, 49%) of the 136 with WEA had a combined score of 1/1, and only 12% (n = 16) had strong evidence for WEA.

Of the 20 participants who reported that work contributed to the onset of their asthma, eight (40%) fulfilled the criteria for WEA. This percentage was greater than the 22% with WEA among all other study participants (p = 0.10). At interview, seven of the eight were still working at the same job they had at asthma onset. One of these eight individuals had onset of asthma as recently as 1 year and 8 months before the survey interview. However, the other seven had had asthma onset earlier, ranging from 3 years and 7 months to 9 years and 7 months before interview.

Those with and without WEA were similar in terms of several descriptive features (table 4). However, the WEA participants were more likely to be male (45% *v* 27%, p<0.0001). Among working participants, men were more likely than women to have an exposure score (see table 2) and a symptom score (48% *v* 41%, p = 0.15) greater than 0. The WEA and non-WEA participants did not differ substantially by severity of asthma as determined by medical records. Yet, those with WEA tended to self-report more severe asthma, as indicated by the last three rows in table 4. In particular, WEA participants reported being bothered by asthma on more days during the past seven days than their non-WEA counterparts (p = 0.002).

DISCUSSION

Potential selection bias

By conducting the study in an HMO, we did not include adults with asthma who were without medical care coverage. Based on a recent analysis of data from the 2003 National Health Interview Survey, 17.8% of employed people in the US did not have health insurance at the time of interview.[28] This segment of the population is likely working in some of the dirtiest jobs in the country. With this in mind, our findings may underestimate the percentage of asthmatic adults with WEA.

Among adults who were enrolled in the HMO, we used a relatively strict definition of asthma. For example, we required participants to have evidence of asthma based on a review of both their electronic (criteria 2 and 3) and paper (criterion 4) medical records. We also excluded potential participants who had not used medications for asthma in the past 12 months (second part of criterion 5), which meant we may have rejected some people with very mild asthma. While we may have excluded some people with asthma, we believe

that the study group was not contaminated with people who did not have asthma.

Work related exposure and symptom scores

We avoided using self-reported occupational exposure because of the potential for bias. Bias was demonstrated in a previous study of respiratory symptoms, in which researchers observed an inflation of the effect estimate when self-reported occupational exposure was used rather than a characterisation of exposure based on a structured occupational history.[29] Our use of a questionnaire combined with expert evaluation is a preferred approach to retrospective evaluation of occupational exposures.[30] The exposure score might still reflect errors due to incomplete or misleading information provided by the participant, or misjudgment by the researcher.

It is likely that some participants in the current study inaccurately perceived the work-relationship of their symptoms, medication use, or asthma triggers, resulting in either overreporting or underreporting. A common concern with occupational diseases is that workers will overreport work related symptoms in order to receive compensation.[31] This type of overreporting was unlikely in the current study. First, participants were selected on the basis of their having asthma and not on whether they sought compensation. Second, the current study was not presented to the participants as a way to gain benefits by claiming work related worsening of asthma. Even in the absence of possible monetary gains, we cannot entirely rule out that reporting by some participants was influenced by a desire to blame or protect their employers.

Our criteria for WEA were not based solely on participants self-reporting that their asthma was made worse by work. They also had to have evidence of relevant work related exposures, as determined by two researchers who independently reviewed and scored the work histories, to fulfill our criteria for WEA. The one exception to the exposure requirement was for those participants with a combined exposure/symptom score of 0/3, who represented just 10% (n = 13) of all 136 WEA cases. At final count, a little over half (n = 136, 56%) of the 243 participants with a symptom score >0 also met the exposure criteria and were considered to have WEA.

Workplace exacerbation and severity of asthma

A recent publication presented characteristics of the 210 cases of work aggravated asthma that were identified during 1993–95 as part of the SENSOR surveillance programme conducted in four states in the US.[32] The authors noted that work aggravated asthma cases were as likely as new onset work related asthma cases to report several adverse outcomes. We anticipated that participants in the current study who fulfilled the criteria for WEA would have more severe asthma than their non-WEA counterparts. Indeed, some of the findings from the current study were consistent with this expectation. For example, the WEA participants reported they were bothered by asthma symptoms on more days in the past seven days than the non-WEA participants ($p = 0.002$). The WEA group also reported more days missed from work because of asthma ($p = 0.24$), which would be expected if work were a source of troublesome exposures.

While the exacerbation of asthma due to workplace exposures could contribute to severity, it is also possible workplace exacerbation is more likely among individuals whose asthma is already severe. For example, in a longitudinal study of adults with asthma that included both new onset and work exacerbated cases of asthma, both baseline severity and workplace exposures were observed to be associated with partial or complete work disability.[33] Longitudinal follow up is needed to accurately determine whether a severe asthma status preceded or followed work related exacerbation. We are following participants from the current study to determine whether WEA status predicts progression of disease over time.

Main messages

- Cases of workplace exacerbation of asthma (WEA) were successfully identified using self-reported data on work related exposures and symptoms/medication use.
- WEA was common, occurring in 23% of adults with asthma in a health maintenance organisation.
- Among adults with asthma, those with WEA were more likely to be male and reported being bothered by asthma on more days during the past seven days.

Policy implications

- WEA is relatively common and should be considered by physicians when treating adults with asthma.

WEA and gender

Women were twice as numerous as men in our study group of adults with asthma, but a disproportionate number of WEA cases were men. This reflects the fact that men were more likely to be employed in workplaces with exposures to agents of relevance to asthma, and to report work related problems with asthma. In a telephone survey of adults in Maine, men accounted for 47% of all respondents and for 75% of the respondents in jobs at higher risk for exacerbation of asthma.[18] However, in a survey of adults with asthma enrolled in a HMO in Colorado, men and women were equally likely to report that their current work environment made their asthma worse.[17]

Conclusion

Physicians should consider that work can contribute to the exacerbation of symptoms when treating adults with asthma. Based on the current study, we conclude that workplace exacerbation of asthma is a relatively common occurrence, identified in 23% of adult asthma cases between the ages of 18 and 44 in this HMO population. The participants with WEA were more likely to be men than women. The WEA participants also reported being bothered by asthma on more days during the past seven days, but it is unclear whether this finding and other indicators of more severe asthma preceded or followed the start of WEA. In an ongoing follow up study, we will analyse data from the participants' medical records to determine whether those with WEA develop more severe asthma over time than those without WEA.

ACKNOWLEDGEMENTS

The authors want to thank Xiaoming Liang for her assistance with computer programming, and all the participants who made this study possible.

Authors' affiliations

P K Henneberger, S J Derk, R J Boylstein, C D Hoffman, Division of Respiratory Disease Studies, National Institute for Occupational Safety and Health, Morgantown, WV, USA
S R Sama, Fallon Clinic, Worcester, MA, USA
P A Preusse, R A Rosiello, Fallon Clinic Research Department, Worcester, MA, USA

D K Milton, Department of Work Environment, University of Massachusetts, Lowell, MA, USA

Ethics approval: the NIOSH Human Subjects Review Board (HSRB) reviewed and approved the research protocol, as did the HSRB of the contracted HMO, Fallon Clinic, Inc, Research Department in Eastern and Central Massachusetts.

Disclaimers: The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health (NIOSH). Mention of any company or product does not constitute endorsement by NIOSH.

REFERENCES

1 **Hunting K**, Nessel-Stephens L, Schiffman A. *Summary of AOEC database case reports—the first three years: 1991–1993*. Washington, DC: Association of Occupational and Environmental Clinics, 1995.
2 **Hunting K**, Anderson J. *Summary of AOEC database case reports: 1994–1996*. Washington, DC: Association of Occupational and Environmental Clinics, 1998.
3 **Chan-Yeung M**. Occupational asthma. *Chest* 1990;**98**(Suppl 1):148S–161S.
4 **Chan-Yeung M**. Assessment of asthma in the workplace. *Chest* 1995;**108**:1084–117.
5 **Blanc P**. Occupational asthma in a national disability survey. *Chest* 1987;**92**:613–17.
6 **Timmer S**, Rosenman K. Occurrence of occupational asthma. *Chest* 1993;**104**:816–20.
7 **Blanc PD**, Cisternas M, Smith S, *et al*. Occupational asthma in a community-based survey of adult asthma. *Chest* 1996;**109**(Suppl):56S–57S.
8 **Milton DK**, Solomon GM, Rosiello RA, *et al*. Risk and incidence of asthma attributable to occupational exposure among HMO members. *Am J Ind Med* 1998;**33**:1–10.
9 **Balmes J**, Becklake M, Blanc P, *et al*. Environmental and Occupational Health Assembly, American Thoracic Society. American Thoracic Society Statement: Occupational contribution to the burden of airway disease, *Am J Resp Crit Care Med* 2003;**167**:787–97.
10 **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(SS03): 1–20, [Erratum in: *MMWR CDC Surveill Summ* 1999;**48**:833.].
11 **Tarlo SM**, Liss G, Corey P, *et al*. Workers' compensation claim population for occupational asthma. Comparison of subgroups. *Chest* 1995;**107**:634–41.
12 **Chatkin JM**, Tarlo SM, Liss G, *et al*. The outcome of asthma related to workplace irritant exposures: a comparison of irritant-induced asthma and irritant aggravation of asthma. *Chest* 1999;**116**:1780–5.
13 **Gassert TH**, Hu H, Kelsey KT, *et al*. Long-term health and employment outcomes of occupational asthma and their determinants. *J Occup Environ Med* 1998;**40**:481–91.
14 **Wheeler S**, Rosenstock L, Barnhart S. A case series of 71 patients referred to a hospital-based occupational and environmental medicine clinic for occupational asthma. *West J Med* 1998;**168**:98–104.
15 **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.
16 **Bakke PS**, Gulsvik A. Work-related asthma—prevalence estimates in a community sample. *Am J Resp Crit Care Med* 1996;**153**:A432.
17 **Henneberger PK**, Hoffman CD, Magid DJ, *et al*. Work-related exacerbation of asthma. *Int J Occup Environ Health* 2002;**8**:291–6.
18 **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**:781–8.
19 **Sama SR**, Hunt PR, Cirillo CP, *et al*. A longitudinal study of adult-onset asthma incidence among HMO members. Environmental Health: A Global Access Science Source, 2:10,7 August 2003.doi:10.1186/1476-069X-2-10.
20 **Global Initiative for Asthma**. Global strategy for asthma management and prevention. NHLBI/WHO workshop report. Bethesda, MD: National Heart, Lung and Blood Institute, 1995.
21 **Ferris BG**. Epidemiology Standardization Project (American Thoracic Society). *Am Rev Resp Dis* 1978;**118**:1–120.
22 **Burney PG**, Luczynska C, Chinn S, *et al*. The European Community Respiratory Health Survey. *Eur Respir J* 1994;**7**:954–60.
23 **Marks GB**, Dunn SM, Woolcock AJ. A scale for the measurement of quality of life in adults with asthma. *J Clin Epidemiol* 1992;**45**:461–72.
24 **Chan-Yeung M**, Malo JL. Tables of major inducers of occupational asthma. In:Bernstein IL, Chan-Yeung M, Malo JL, Bernstein DI (eds). *Asthma in the workplace, 2nd edn*. New York: Marcel Dekker, Inc, 1999:683–720.
25 **Association of Occupational and Environmental Clinics Exposure Code System [database on the Internet]**. Washington, DC: The Association of Occupational and Environmental Clinics. c2002 – [cited 2005 June 24]. Available at http://www.aoec.org/tools.htm (accessed May 2006).
26 **SAS Institute, Inc**. SAS/STAT® 9.1 User's Guide. Cary, NC: SAS Institute, Inc, 2004.
27 **Colton T**. *Statistics in medicine*. Boston: Little, Brown, and Company, 1974.
28 **Cohen RA**, Coriaty-Nelson Z. Health insurance coverage: Estimates from the National Health Interview Survey, 2003. Available at http://www.cdc.gov/nchs/nhis.htm (accessed June 2004).
29 **Bakke PS**, Honoa R, Gulsvik A. Relation of occupational exposure to respiratory symptoms and asthma in a general population sample: self-reported versus interview-based exposure data. *Am J Epidemiol* 2001;**154**:477–83.
30 **Bouyer J**, Hemon D. Retrospective evaluation of occupational exposures in population-based case-control studies: general overview with special attention to job exposure matrices. *Int J Epidemiol* 1993;**22**(Suppl 2):S57–64.
31 **Baur X**, Huber H, Degens PO, *et al*. Relation between occupational asthma case history, bronchial methacholine challenge, and specific challenge test in patients with suspected occupational asthma. *Am J Ind Med* 1998;**33**:114–22.
32 **Goe SK**, Henneberger PK, Reilly MJ, *et al*. A descriptive study of work aggravated asthma. *Occup Environ Med* 2004;**61**:512–17.
33 **Blanc PD**, Cisternas M, Smith S, *et al*. Asthma, employment status, and disability among adults treated by pulmonary and allergy specialists. *Chest* 1996;**109**:688–96.