ORIGINAL ARTICLE

A descriptive study of work aggravated asthma

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Background and Aims: Work related asthma (WRA) is one of the most frequently reported occupational lung diseases in a number of industrialised countries. A better understanding of work aggravated asthma (WAA), as well as work related new onset asthma (NOA), is needed to aid in prevention efforts.
Methods: WAA and NOA in the United States were compared using cases reported to the National Institute for Occupational Safety and Health (NIOSH) from four state Sentinel Event Notification Systems for Occupational Risks (SENSOR) surveillance programmes for 1993–95.
Results: A total of 210 WAA cases and 891 NOA cases were reported. WAA cases reported mineral and inorganic dusts as the most common exposure agent, as opposed to NOA cases, in which diisocyanates were reported most frequently. A similar percentage of WAA and NOA cases still experienced breathing problems at the time of the interview or had visited a hospital or emergency room for work related breathing problems. NOA cases were twice as likely to have applied for workers' compensation compared with WAA cases. However, among those who had applied for worker compensation, approximately three-fourths of both WAA and NOA cases had received awards. The services and manufacturing industrial categories together accounted for the majority of both WAA (62%) and NOA (75%) cases. The risk of WAA, measured by average annual rate, was clearly the highest in the public administration (14.2 cases/10^5) industrial category, while the risk of NOA was increased in both the manufacturing (3.2 cases/10^5) and public administration (2.9 cases/10^5) categories.
Conclusions: WAA cases reported many of the same adverse consequences as NOA cases. Certain industries were identified as potential targets for prevention efforts based on either the number of cases or the risk of WAA and NOA.

Work related asthma (WRA) is one of the most frequently reported occupational lung diseases in a number of industrialised countries.[1–3] Several studies in the United States have estimated the proportion of adult onset asthma attributable to workplace exposures, with figures ranging from 2% to 21%.[4] WRA can be classified into two distinct conditions: work aggravated asthma (WAA), which is pre-existing asthma that is exacerbated by workplace exposures; and work related new onset asthma (NOA), which is caused by workplace exposure to sensitisers or irritants.[5] WAA is a serious but often preventable occupational lung condition that may be completely reversed when the offending exposure is recognised early and removed, but may lead to chronic impairment when recognition is delayed and the offending exposure is prolonged.[6 7]

The Sentinel Event Notification Systems for Occupational Risks (SENSOR) is a case based surveillance programme in the United States that is applied to different occupational health outcomes. SENSOR is currently active in eight states and collects information on a variety of occupational conditions, including: acute pesticide poisoning, burns, dermatitis, noise induced hearing loss, silicosis, and asthma. The National Institute for Occupational Safety and Health (NIOSH) funds surveillance activities for WRA under the SENSOR programme in California, Massachusetts, Michigan, and New Jersey.

From 1993 to 1995, 19.1% of all SENSOR asthma cases were considered to be WAA.[5] However, in an earlier study conducted on a subset of SENSOR data between 1988 and 1992 in Michigan and New Jersey, WAA accounted for only 6.9% of all WRA cases.[8] An additional analysis of SENSOR data between 1988 and 1994 in Michigan reported only 7.3% of all WRA as WAA.[9] Other studies have reported percentages considerably higher than those described in Michigan and New Jersey. In one analysis of 945 SENSOR cases identified in California between 1993 and 1996, researchers found that 35% of WRA cases were classified as WAA.[10] In a Canadian study, the Ontario Worker Compensation Board reported that half of all asthma claims received between 1984 and 1988 were for WAA.[11 12] Other surveillance programmes in the United Kingdom,[13 14] the province of Quebec in Canada,[15] and South Africa[16] did not count cases of WAA as part of their activities.

Work aggravated asthma also has been described in several clinical case series. Among 71 patients referred for occupational asthma to a hospital based occupational and environmental medicine clinic in Washington state, 27% (19/71) were classified as WAA.[17] At an occupational and environmental medicine clinic in Massachusetts, researchers found that 18% (10/55) of patients with definite or probable occupational asthma were determined to have WAA.[18] At an asthma referral clinic in Ontario, a review of physician administered questionnaires between 1972 and 1990 identified that of 51 patients who reported a worsening of symptoms at work, 49% were likely to have WAA.[19]

Studies in the United States and Canada have examined the association between workplace exposures and WAA. Tarlo and colleagues reported that among 25 WAA cases referred to an asthma clinic in Ontario, dusts and second-hand smoke were the most frequently described exposures.[19] Jajosky and colleagues listed several agents associated with WAA, with mineral and inorganic dust and indoor air

Abbreviations: NOA, new onset asthma; RADS, reactive airways dysfunction syndrome; SENSOR, Sentinel Event Notification Systems for Occupational Risks; WAA, work aggravated asthma; WRA, work related asthma

Main messages

- Among adults with WRA, those with WAA differ from those with work related NOA.
- WAA cases were significantly more likely to be employed in the service industry (40% *v* 29%) and in technical, sales, and administrative support occupations (34% *v* 18%) compared with NOA cases.
- The most common exposure agent was mineral and inorganic dusts for WAA cases and diisocyanates for NOA cases.
- A similar percentage of WAA and NOA cases had visited a hospital or emergency room for work related breathing problems.

Policy implications

- An understanding of the differences between WAA and NOA cases will contribute to planning and implementing effective preventive interventions.

pollutants comprising 16.2% and 9.0% of all agents reported between 1993 and 1995, respectively.[5] However, there is very little information on occupations or industries most commonly associated with WAA.

We analysed SENSOR data from 1993 to 1995 to describe WAA and compared it with NOA. We identified agents, occupations, and industries associated with WAA, and compared these percentages with NOA cases identified during the same time period. We also compared WAA and NOA cases on several demographic, personal characteristics, and the consequences of WRA. An understanding of the similarities and differences between WAA and NOA will contribute to planning effective prevention activities.

METHODS

Case identification and follow up in the SENSOR asthma programme

Details on how WAA and NOA cases are identified in the SENSOR asthma programme have been published elsewhere.[5 8 20 21] Cases are identified from several different sources, including physician reports and hospital records. Programme staff administered questionnaires to identified cases. Core data elements about the case were collected by all states, including personal characteristics, pattern of asthma symptoms in relation to work, reported exposures associated with asthma symptoms, industry and occupation, history of allergies, and adverse impacts of having WRA.[5]

Case definition and classification

According to the standard SENSOR classification scheme, a person was defined as having WRA if he or she was diagnosed with asthma, and there was a temporal association between asthmatic symptoms and work. Cases were then further classified as either WAA or NOA. Individuals with WRA were classified as having WAA if the following conditions were met: (1) the affected individual experienced asthma symptoms or had treatment for asthma in the two years prior to entering a new work setting; and (2) they experienced an increase in asthma symptoms or increased use of their asthma medications after entering that new exposure setting.[5] The new exposure setting could include starting a new job, a change of work processes, and/or the introduction of new agents in the workplace. WRA cases with no asthmatic symptoms or treatment for asthma in the two years before entering a new work setting were classified as having NOA, which included reactive airways dysfunction syndrome (RADS).

Average annual incidence rates

We calculated average annual incidence rates for major industrial and occupational categories for both WAA and NOA, as follows:

For each major industrial or occupational category:

$$\text{WAA average annual incidence rate} = \frac{\text{No. of WAA cases in the four states during the 1993–95 period}}{\text{No. of employed persons with current asthma in the four states}} \div 3$$

$$\text{NOA average annual incidence rate} = \frac{\text{No. of NOA cases in the four states during the 1993–95 period}}{\text{No. of employed persons without current asthma in the four states}} \div 3$$

We arrived at the number of employed persons with and without current asthma through a series of calculations. First, we obtained the current percentages of males and females in each major industrial or occupational category in the United States, which were available from the Bureau of Labor Statistics.[22] We multiplied these percentages by the total number of persons working in each major industrial or occupational category for each of the four states.[23] The resulting estimated numbers of male and female workers in each industry or occupation for each state were then multiplied by the respective state specific percentages of males and females who had reported ever being told by a doctor that they had asthma, as indicated by the 2000 Behavioral Risk Factor Surveillance System (BRFSS) survey.[24] This yielded the estimated expected numbers of males and females who had ever had asthma in each major industrial or occupational category. Finally, we multiplied these numbers by the state specific percentages of males and females with current asthma who reported ever having asthma in the 2000 BRFSS survey. The resulting figures were the total estimated numbers of males and females in that state with current asthma for each major industrial and occupational category. These values were summed across the four states to obtain denominators for the overall WAA rates. We divided the number of WAA cases for all four states by this denominator to arrive at an estimated rate over the three year period. We then divided this rate by 3 to arrive at an estimated annual incidence rate. We arrived at the denominator for the NOA average annual incidence rate through the same series of calculations, except that the numbers of male and female workers in each major industrial and occupational category for each state were multiplied by the respective percentages of males and females who did not have current asthma, as indicated by the 2000 BRFSS survey.

Data management and analysis

Primary industry for each case had been coded by the states using the four digit 1987 Standard Industrial Classification (SIC) system developed by the US Department of Labor.[25] In addition, primary occupation had been coded following the 1990 Occupational Classification System from the US Bureau of Census.[26] Industrial and occupational categories had also

been created by using major groupings previously developed by the US Department of Labor and the US Bureau of Census.

Agents had been coded by the states using the Association of Occupational and Environmental Clinics (AOEC) classification system.[27] Table 2 in Jajosky *et al* presented agents for both WAA and NOA.[5] For WAA, the five most frequently reported agents in descending order were mineral and inorganic dusts, not otherwise specified (nos); indoor air pollutants; chemicals, nos; paint; and smoke, nos.[5] The top five agents in descending order for NOA were indoor air pollutants; chemicals, nos; lubricants, which included metal working fluids; mineral and inorganic dusts, nos; and cleaning materials, nos.[5] In the Jajosky *et al* article, agents were listed singly according to AOEC codes.[5] For our analysis, we combined cleaning agents, nos (AOEC code 322.00), bleach (050.02), and household cleaners (322.04) into a single cleaning agents category. Similarly, we arrived at an inclusive diisocyanates category by combining diisocyanates, nos (221.00), toluene diisocyanate (221.01), and methylene diisocyanate (221.02). Cleaning agents and diisocyanates are two categories of exposure that have commonly been associated with work related asthma.

We analysed all data using the SAS System for Windows.[28] Tests of statistical significance were conducted using the χ^2, continuity corrected χ^2, and Fisher's exact test for categorical data, and Student's *t* test for continuous data. We considered all p values that were less than or equal to 0.05 to be statistically significant.

RESULTS

Source of case reports, demographic characteristics

Between 1993 and 1995, 1101 cases of WRA were identified by the four participating state SENSOR programmes. Of these, 210 (19.1%) were classified as having WAA, with the remaining 891 cases classified as NOA. In comparison with NOA cases, WAA cases were significantly more likely to be younger (mean age 38 years *v* 42 years), female (69% *v* 53%), and non-white (31% *v* 21%) (table 1). In addition, WAA cases were significantly more likely to have ever been told they had allergies (75% *v* 42%) or had reported a family history of allergies or asthma (57% *v* 35%). However, WAA cases were significantly less likely to have ever smoked cigarettes (39% *v* 53%) than NOA cases. About 90% of both WAA and NOA cases were identified from physician reports, with the remaining cases coming from hospital discharge records and other sources.

Table 1 Personal characteristics of work aggravated and new onset asthma cases

Characteristic	Categories	WAA (n = 210)	NOA (n = 891)
Source of case reports	Physician reports	91%	90%
	Hospital discharge data	8.5%	8%
	Other	0.5%	2%
	Total	100%	100%
Demographic features	Mean age (SEM)*	38.0 (0.68)	42.0 (0.35)
	Female, %†	69%	53%
	Non-white, %†	31%	21%
	Ever smoke cigarettes, %†	39%	53%
History of allergies	Ever told had allergies†	75%	42%
	Family history of allergies or asthma†	57%	35%

*p<0.01, by *t* test.
†p<0.01, by χ^2 test.

Primary industry and primary occupation

WAA cases were most likely to be employed in the service industry (40%), followed by manufacturing (22%) and public administration (16%) (table 2). The three most common industries for NOA cases were the same as WAA cases; however, the first two were reversed in order relative to the WAA cases: manufacturing (46%), services (29%), and public administration (10%). By primary occupation, WAA cases were most commonly employed in technical, sales, and administrative support professions (34%), followed by managerial and professional specialities (22%), operators, fabricators, and labourers (18%), and service (17%) professions (table 3). In contrast, NOA cases differed significantly ($p < 0.0001$) from WAA cases, with over a third employed as operators, fabricators, and labourers (36%), followed by technical, sales, and administrative support (18%), managerial and professional specialities (17%), and precision production, craft, and repair occupations (16%).

Average annual incidence rates for WAA and NOA

We calculated average annual incidence rates of WAA and NOA among employed persons with and without current asthma, respectively. The rate of WAA among employed persons with current asthma was 3.9 cases/10^5/y, and the rate of NOA among employed persons without current asthma was 1.3 cases/10^5/y. We also calculated average annual incidence rates for WAA and NOA for each major industrial and occupational category. For WAA cases, the highest rate was found among persons working within the public administration industry (14.2 cases/10^5/y) (table 2). Manufacturing and public administration industries had the highest rates among NOA cases (3.2 cases/10^5/y and 2.9 cases/10^5/y, respectively). Incidence rates of WAA were similar among all major occupational categories, although operators, fabricators, and labourers, as well as those in service occupations, had slightly higher incidence rates (table 3). Operators, fabricators, and labourers had the highest average annual incidence rate for NOA cases by occupation (3.4 cases/10^5/y) (table 3).

Agents

In California, Massachusetts, and New Jersey, up to three agents were recorded for each WRA case. In Michigan, up to two agents were recorded for each case. Overall, more than one agent was recorded for 64 (30%) WAA cases and 257 (29%) NOA cases. The most common agents for WAA cases included, in descending order: mineral and inorganic dusts, nos; indoor air pollutants; chemicals, nos; cleaning agents; paint; smoke, nos; and glues, nos (table 4). The most common agents for NOA were: diisocyanates; indoor air pollutants; cleaning agents; chemicals, nos; lubricants, nos; mineral and inorganic dusts, nos; and smoke, nos.

Consequences of work related asthma

A majority of both WAA (75%) and NOA (79%) cases reported still experiencing breathing problems at the time of the interview. Over half of all WAA and NOA cases (59% and 52%, respectively) had visited an emergency department, and about a fourth (23% of WAA and 25% of NOA) had been hospitalised for work related breathing problems.

A significantly higher proportion of NOA cases had applied for workers' compensation at the time of the interview—with 40% of NOA cases and 21% of WAA cases, respectively ($p < 0.0001$). However, among the individuals whose applications had been decided, there was little difference between the two groups in the percentage of claims awarded, with 72% for WAA and 77% for NOA. WAA (73%) and NOA (71%) cases were equally likely to no longer be exposed to suspected agents in the workplace. Of those no longer exposed, NOA

Table 2 Percentage distributions and average annual incidence rates of work aggravated and new onset asthma cases by primary industry

	WAA (n = 210)		NOA (n = 891)		All
Industry	**%**	**Rate†**	**%**	**Rate‡**	**Rate§**
Services**	40%	4.3	29%	1.1	1.4
Manufacturing**	22%	5.0	46%	3.2	3.3
Public administration*	16%	14.2	10%	2.9	3.7
Wholesale/retail trade**	9%	1.7	3%	0.2	0.3
Transportation, communication, and other public utilities	7%	4.2	4%	0.7	1.0
Finance, insurance, and real estate	2%	1.2	1%	0.2	0.2
Construction	2%	1.4¶	4%	0.7	0.8
Agriculture	1%	2.7¶	3%	1.4	1.5
Mining	0.5%	9.6¶	<1%	0.6¶	1.1¶
Unclassified	0.5%	–	<1%	–	–
Overall	100%	3.9	100%	1.3	1.5

†Average annual rate per 100 000 workers with current asthma per year.
‡Average annual rate per 100 000 workers without current asthma per year.
§Average annual rate per 100 000 workers per year, irrespective of asthma status.
¶Rate based on less than five cases.
*p<0.05, **p<0.01, by χ^2 test.

cases were twice as likely as WAA cases to have left the company (47% *v* 23%). Among both types of cases, medical reasons, such as workers' compensation, disability, sick leave, and physician's advice, were the most commonly reported motivations for leaving the company.

DISCUSSION

Strengths and limitations of study

The strengths and limitations of using the SENSOR database for WRA have been discussed elsewhere.[5] Although the number of states participating in the programme is limited, SENSOR is currently the only available surveillance programme in the United States for detecting and monitoring trends in WRA. SENSOR has encouraged case identification and reporting by providing educational materials to physicians for detecting individuals with WRA, as well as keeping them up to date on the latest SENSOR surveillance and intervention activities.[5] However, SENSOR was never intended to be a comprehensive or representative sample of all WRA cases in the United States; many cases are not reported due to lack of recognition by a healthcare professional or incomplete reporting.[5] Accordingly, the average annual incidence rates reported in tables 2 and 3 are assumed to be underestimates of the ''true'' rates.

Differences between NOA and WAA cases in industry and occupation

It should first be noted that comparisons between WAA and NOA were made only across major industrial and occupational categories, which may fail to identify similarities and differences in more specific industries and occupations. However, there are several possible explanations as to why a major industrial or occupational category could account for a disproportionate number or rate of WAA or NOA cases. One reason might be the varying types and levels of exposures across workplaces. For example, industries with very potent agents and/or high level exposures would be expected to have high incidence rates for both WAA and NOA, which might explain the rates observed for manufacturing. However, those with asthma may self-select out of particular industries or occupations where they feel harmful exposures are likely (for example, the manufacturing industry) and self-select into industries or occupations where they feel they will not be exposed (for example, the service industry). Therefore, people with asthma who comprise the at-risk pool for WAA may not be equally distributed by industry and occupation, and consequently, exposure levels alone may not predict the frequency of WAA. In calculating incidence rates for WAA, we assumed that people with asthma were equally

Table 3 Percentage distributions and average annual incidence rates of work aggravated and new onset asthma cases by primary occupation

	WAA (n = 210)		NOA (n = 891)		All
Occupation	**%**	**Rate†**	**%**	**Rate‡**	**Rate§**
Technical, sales, administrative support**	34%	3.8	18%	0.7	0.9
Managerial and professional specialty	22%	3.0	17%	0.8	0.9
Operators, fabricators, and labourers**	18%	5.8	36%	3.4	3.5
Service*	17%	4.8	11%	1.1	1.4
Precision production, craft, repair**	7%	3.1	16%	1.9	1.9
Farming, forestry, and fishing	2%	4.1	2%	1.3	1.4
Other	–	–	<1%	–	–
Overall	100%	3.9	100%	1.3	1.5

†Average annual rate per 100 000 workers with current asthma per year.
‡Average annual rate per 100 000 workers without current asthma per year.
§Average annual rate per 100 000 workers per year, irrespective of asthma status.
*p<0.05, **p<0.01, by χ^2 test.

Table 4 Seven most frequently reported agents among WAA and NOA cases*†

WAA cases	n	%	NOA cases	n	%
1. Mineral and inorganic dusts, nos‡	34	16%	1. Diisocyanates	99	11%
2. Air pollutants, indoor	19	9%	2. Air pollutants, indoor	67	8%
3. Chemicals, nos	17	8%	3. Cleaning agents	62	7%
4. Cleaning agents	15	7%	4. Chemicals, nos	56	6%
5. Paint	13	6%	5. Lubricants, nos	55	6%
6. Smoke, nos§	10	5%	6. Mineral and inorganic dusts, nos	45	5%
7. Glues, nos	9	4%	7. Smoke, nos§	40	4%

*Based on AOEC exposure coding system, 2/96.
†64 WAA cases and 257 NOA cases indicated more than one agent.
‡nos, not otherwise specified.
§Does not include environmental tobacco smoke.

distributed by industry and occupation. If this assumption is incorrect, perhaps due to the healthy worker selection effect, some rates we presented would be underestimates and other rates would be overestimates.

Another question is why the average annual incidence rates of WAA and NOA were so high for the industrial category public administration. One potential explanation is the fact that public administration includes those working in fire and police protection (SIC 9224 and 9221, respectively). Henneberger and colleagues found that public administration had the highest rate of non-fatal work related inhalation injuries (16.4 inhalations per 10 000 workers) among all major industries.[28] When only firefighters and police were considered, this rate almost doubled to 31.5 inhalations per 10 000 workers.[29] Of the 1101 cases reported to SENSOR between 1993 and 1995, 122 cases were employed in public administration. Of those, 24% (n = 29) were in police or fire protection. According to private market research statistics, only 11.6% of those in public administration are employed in police or fire protection.[30] The apparent over-representation of police and firefighters in our data could have contributed to the increased rates for public administration.

Race could have also contributed to the high rate of WAA in the public administration industry. According to Mannino and colleagues, asthma proportionately affects more non-whites than whites.[31] Recent labour statistics indicate that the public administration industrial category, on the whole, has proportionately more non-whites than the overall workforce (16.7% *v* 11.3%, respectively).[22] Additionally, SENSOR data indicated that WAA cases in public administration were about 50% more likely to be non-white than WAA cases in all other industries (42% *v* 29%, respectively).

Prevention

At least two different approaches could be taken to target primary prevention efforts. One approach would be to focus on those industries that have the greatest number of persons with WAA or NOA. The industry groups with the most cases were services (40%) for WAA and manufacturing (46%) for NOA. Services and manufacturing combined comprised 62% of WAA cases and 75% of NOA cases. A second approach would be to focus on those industries which had the highest risk as indicated by average annual incidence rates. Public administration and manufacturing had high average annual incidence rates for both WAA and NOA (table 2). By focusing efforts on specific industries within services, manufacturing, and public administration with a high risk of work related asthma, a large proportion of both WAA and NOA could potentially be averted. Secondary prevention efforts could be directed towards increasing healthcare provider awareness of the potential for WAA, including educating healthcare providers about which worker populations are at greater risk.

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ECHO

Gastroenteritis from a non-chlorinated water supply

Please visit the *Occupational and Environmental Medicine* website [www.occenvmed.com] for a link to the full text of this article.

In developed countries the most common bacterial cause of gastroenteritis is campylobacter infection. Most of these infections are sporadic and known risk factors include drinking unpasteurised milk or untreated water, eating chicken, barbecuing, and living or working on a farm. Several community outbreaks of campylobacter gastroenteritis arising from contamination of the water supply have been described but the mechanism of water contamination has usually been unknown. Now an outbreak in a rural community in southern Finland has been traced to a non-chlorinated well water supply.

In August 2000 there were 463 known cases of gastroenteritis among a population of 8600 people. The median age of those affected was 41 years (range 1–96 years) and 62% were female. The outbreak began around 27 July and ended around 24 August, the peak incidence being on 7 August. Seventy four stool samples were submitted from patients and *Campylobacter jejuni* was isolated from 24. No other pathogens were isolated. One sample of tap water contained *C jejuni*. Samples from other parts of the water supply were negative.

A case-control study included 113 patients and 241 controls. Ninety four per cent of patients and 58% of controls had drunk unboiled tap water during the previous two weeks (matched odds ratio (MOR) 21). For 88% of cases and 57% of controls the drinking water in the home was unboiled tap water and the risk of illness increased with the amount of unboiled tap water drunk. Drinking water from private wells, bottled water, or boiled water was protective. Consumption of eggs was associated with a decreased risk. Only drinking unboiled tap water remained significant after adjustment for other factors.

About two thirds of this population received their water from two water stores supplied by two wells. The water was not chlorinated but it was tested monthly for coliforms. No contamination had been detected before the outbreak. Some holiday homes near one of the wells were not connected to the municipal sewage system and there was a dry toilet and a compost heap about 15 metres from this well. The wells were unfenced and people and animals had free access. The rainfall in July 2000 was high.

Sixteen *C jejuni* isolates from patients were serotyped and all 16 had the same serotype (Penner 12). Eight of these isolates were subtyped by pulsed field gel electrophoresis (PFGE) and all but one showed the same pattern. The isolate from tap water had the same serotype and on PFGE was identical to the seven patient strains. Prior to the outbreak 17 strains of *C jejuni* had been identified in the area, one of which was of the same serotype as the outbreak strain.

The evidence points to this outbreak having arisen from a defective water supply. People were advised to boil their drinking water on 11 August and the water supply was chlorinated from 12 August.

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