AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 39:72–83 (2001)

Physician Reports of Work-Related Asthma in California, 1993–1996

Florence Reinisch, MPH,[1]* Robert J. Harrison, MD, MPH,[1] Sarah Cussler, MA,[1] Marcos Athanasoulis, MPH,[2] John Balmes, MD,[3] Paul Blanc, MD, MSPH,[3] and James Cone, MD, MPH[1]

Background *Work-related asthma is a leading cause of occupational respiratory illness.*
Methods *Work-related asthma was studied in California over a 36-month period, from March 1, 1993 to February 29, 1996. The surveillance system identified cases from Doctor's First Reports (DFRs), a mandated physician reporting system. Structured follow-up telephone interviews of DFR asthma cases were conducted to collect work history, exposure, and medical information. Statewide employment data was used to calculate disease rates among industry groups.*
Results *Based on 945 cases of work-related asthma, the average annual reporting rate for work-related asthma in California was 25/million workers. We estimate that the actual rate is 78/million, adjusted for likely underreporting. Janitors and cleaners (625/million) and firefighters (300/million) had the highest reporting rates of work-related asthma. Half of all work-related asthma cases were associated with agents not known to be allergens.*
Conclusions *A greater proportion of work-related asthma associated with irritant exposures was identified than has previously been reported. The surveillance data provide a very conservative estimate of the incidence of work-related asthma.* Am. J. Ind. Med. 39:72–83, 2001. *© 2001 Wiley-Liss, Inc.*

KEY WORDS: work-related asthma; surveillance; occupational asthma; irritant inhalation; RADS

INTRODUCTION

In the United States asthma is a common chronic condition, with rising prevalence, morbidity, and mortality. Many studies have attempted to evaluate the proportion of all asthma cases that are related to occupational exposures. Occupational exposures are likely to account for 5–21% of all adult asthma [Milton et al., 1998; Kogevinas et al., 1999]. As the overall prevalence of asthma continues to increase in the US and in Western Europe [CDC, 1995; Weiss, 1993], the number of work-related asthma cases are also likely to rise [Taylor, 1995]. Increased exposure to asthma-inducing chemicals in the workplace may be an important factor in the occupational contribution to the overall asthma increase [Chan-Yeung and Malo, 1994].

Prevention of disease is paramount since the economic and personal consequences of occupational asthma are severe. A majority of work-related asthma cases do not fully recover from asthma, leading to a 25% unemployment rate and frequent reduction of income following diagnosis [Ameille et al., 1997; Chan-Yeung, 1990; Tarlo et al.,

[1]California Department of Health Services, Occupational Health Branch, Oakland, CA
[2]School of Public Health, University of California, Berkeley, CA
[3]School of Medicine, University of California, San Francisco, CA

All of the work was performed by staff affiliated with the California Department of Health Services, Occupational Health Branch, except for Paul Blanc and John Balmes who were affiliated with the University of California, San Francisco and Marcos Athanasoulis who was affiliated with the University of California, Berkeley.

Contract grant sponsor: National Institute for Occupational Safety and Health; Contract grant number:U50/CCU910074

*Correspondence to: Florence Reinisch, MPH, California Department of Health Services, Occupational Health Branch, 1515 Clay Street, Suite 1901, Oakland, CA 94612. E-mail: freinisc@dhs.ca.gov

Accepted 16 May 2000

1995]. Even quantifying the problem is difficult: misdiagnoses, underreporting of diagnosed cases, and the small number of established population-based surveillance systems which collect incident cases limits recognition and intervention efforts for all affected groups.

The plethora of naturally occurring and man-made agents and processes associated with work-related asthma complicate identification of causal agents, appropriate intervention, and prevention. The California Department of Health Services, Occupational Health Branch, with funding from the National Institute for Occupational Safety and Health (NIOSH) established a statewide surveillance program for work-related asthma. Since 1993, the California SENSOR program has identified all cases of work-related asthma from Doctor's First Reports (DFRs), a well-established reporting system in California for all occupational injuries and illness. The program is based on a national model called SENSOR—the Sentinel Event Notification System for Occupational Risks [Matte et al., 1990]. The primary goal of the SENSOR program has been to enhance reporting and surveillance of important work-related health conditions at the state level. The national SENSOR model was adapted to the California setting.

We analyzed the data from follow-up interviews among adults with work-related asthma identified through the SENSOR program in order to study the correlates of disease. In particular, we wanted to compare new onset asthma cases with work-aggravated disease.

MATERIALS AND METHODS

Case Reports

We used the SENSOR model as a basis for developing a surveillance and intervention program for work-related asthma in California. Sentinel cases of work-related asthma were identified from an existing reporting source, Doctor's First Reports (DFRs). The DFR is a useful statewide surveillance tool based on California labor code-mandated reporting of all medical claims for known or suspected occupational illnesses or injuries within 5 days of providing medical services [California Labor Code, 1994]. Moreover, physician reimbursement for medical services from workers' compensation insurers requires submission of a DFR. Federal government, maritime and railroad workers, and a majority of self-employed persons are excluded from this system. The patient provides basic demographic and descriptive information about the nature of injury or illness noted on the DFR and the physician completes medical information including diagnosis. All workers' compensation insurers initially receive the DFR and then are required to send them to the State of California Division of Labor Statistics and Research where California Department of Health researchers review the reports. The mean length of time between the medical examination noted on the DFR and California Department of Health's receipt of the DFR is 4 weeks.

For the 3-year period, March 1, 1993 through February 29, 1996 all respiratory cases were selected by manual review among the approximately 1.2 million DFRs that are filed annually. Possible respiratory cases were selected based on the identification of any of the following key words within the DFR: *inhalation, chemical exposure, respiratory, lungs, coughing, wheezing, shortness of breath, dyspnea, trouble breathing, sinus/sinusitis, rhinitis, throat irritation, reactive airways, asthma, reactive airways dysfunction syndrome* (RADS), *byssinosis, bronchitis, pneumonitis, tuberculosis, psittacosis, coccidioidomycosis, lung cancer, mesothelioma, pleural disease, or pneumoconiosis*. All of the reports initially identified were then screened and coded for respiratory diagnosis by a single occupational physician. Of the 25,570 reports selected based on the key words listed above, the physician excluded 16,092 DFR reports with other non-respiratory diagnoses. The majority of the non-respiratory diagnoses were ''chemical exposure'' without respiratory symptoms (e.g., central nervous system, eye, or dermatological symptoms). A total of 9,478 unique, respiratory reports remained after these cases were excluded. These reports included a total of 4,563 (48%) cases with chemical, dust or smoke inhalation with lower respiratory symptoms (cough, chest tightness, shortness of breath, wheezing or chest pain); 2,592 (27%) cases with chemical, dust or smoke inhalation with upper respiratory symptoms (sore throat, rhinitis, hoarseness); 886 (9%) cases with a diagnosis of bronchitis; 336 (4%) cases with a diagnosis of bronchospasm; and 112 (1%) cases with other respiratory diagnoses (tuberculosis, byssinosis, coccidioidomycosis, lung cancer, mesothelioma, pleural disease, pneumoconioses, or psittacosis). All DFR reports with a diagnosis of either asthma, reactive airways disease, or RADS were categorized as asthma cases (Fig. 1). All duplicate reports were eliminated as well as secondary DFR reports for an individual that related to a medical examination within 1 week of the original medical examination, yielding 945 reports eligible for interview.

Case Follow-up

We conducted follow-up interviews to gather additional data not provided by the DFR, and additionally, to permit identification of new onset asthma vs. work-aggravated asthma cases. Medical records were not reviewed to independently verify diagnoses. The structured telephone interview collected demographic, occupational, and health history data. All interviewed cases provided informed consent to participate in the study, and all protocols were approved by the State of California Health and Welfare

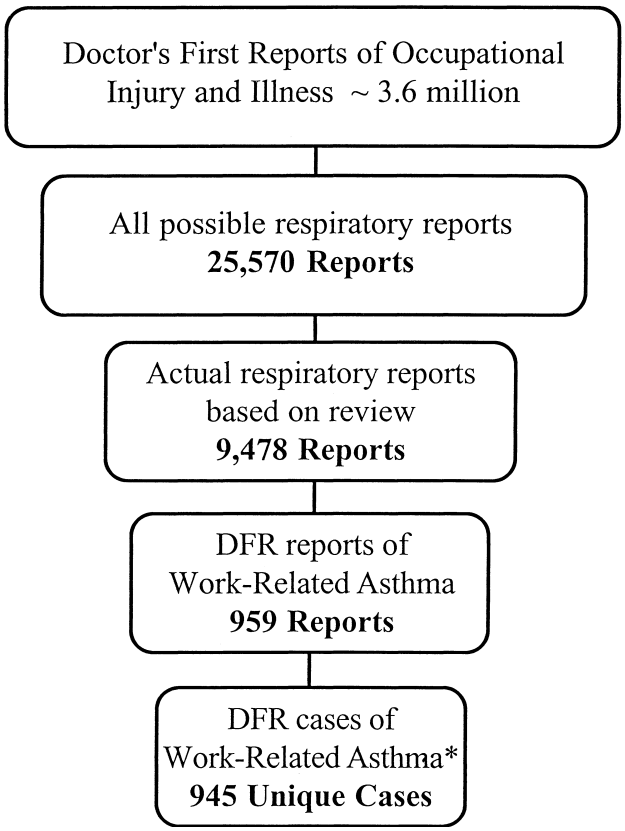

FIGURE 1. Surveillance of work-related Asthma in California March 1, 1993–February 29, 1996.

* Physician diagnosis of Asthma, Reactive Airways Disease, or Reactive Airways Dysfunction Syndrome.

Agency Committee for the Protection of Human Subjects. For those reports of work-related asthma that could not be interviewed, Hispanic ethnicity was designated based on surname [U.S. Census Bureau, 1980].

If no telephone number was provided on the DFR, study interviewers attempted to identify a telephone number by using directory assistance and address-based telephone directories. Each asthma case for which there was an identifiable telephone number was contacted to enlist participation at least six times at varying days and times by an English–Spanish bilingual interviewer. Cases were interviewed at an average of 9 months after initial receipt of the DFR. Of the 945 unique cases eligible for interview, 813 (86%) DFRs contained a contact telephone number or sufficient information to obtain a telephone number, of which 459 (56%) completed an interview, 109 (13%) declined to participate, and 245 (30%) could not be successfully contacted. Based on the interviews, we excluded 15 cases (3.3%) who stated the initial DFR diagnosis was incorrect. A total of 444 interviewed cases remained for final analysis.

Case Coding and Classification

NIOSH certified coders assigned 1987 Standard Industrial Classification (SIC) codes for industry and 1990 Census Occupational Codes (COC) for occupation based on information contained on the DFR. Cases lacking sufficient data to assign a SIC or COC code were categorized as ''nonclassifiable''. We did not attempt any method of assigning SIC codes for reports that lacked information on the type of business. For interviewed cases, a second set of SIC and COC codes was assigned based on the worker's reported occupation at the time of his/her first work-related asthma diagnosis. Exposure agents identified from follow-up interviews were coded according to an exposure database developed by the Association of Occupational and Environmental Clinics [AOEC, 1996]. In this coding scheme, agents known to induce occupational asthma have been designated based on comprehensive literature reviews. Each interviewed case was associated with up to six exposure agents. Exposure agents were not weighted and no additional information was collected to verify exposure.

Information on ability to work (usual or modified work) and the number of lost workdays was abstracted directly from information provided on the DFR. For a subset of interviewed cases ($n = 141$, or 32% of interviewed cases) a supplemental question was asked to collect additional information about work status and filing a workers' compensation case.

Interviewed cases were classified according to the NIOSH surveillance case classification criteria for work-related asthma [Jajosky et al., 1999]. This case classification system categorizes work-related asthma as new onset or work-aggravated asthma. New onset asthma includes both reactive airways dysfunction syndrome (RADS) (i.e., persistent asthma symptoms induced by a one-time, high-level inhalation exposure at work), and occupational asthma. Occupational asthma cases are further classified according to whether the suspected agent is a known asthma inducer (i.e., an agent previously documented in the medical literature to cause occupational asthma). Cases without exposure to a known asthma inducer primarily comprise exposure to respiratory irritants. Cases of work-aggravated asthma are defined as those with a history of symptomatic or treated asthma within 2 years of entering a new occupational exposure setting, and who experience an increase in symptoms or an increase in the use of asthma medication upon entering the new work setting.

Statistical Analysis

Asthma rates were calculated based on DFR reports of work-related asthma that were eligible for interview. The annual reporting rate for work-related asthma was calculated by dividing the mean annual number of reported DFR

cases by the average number of people in the workforce over the 3-year reporting period. Thus, 1993, 1994, and 1995 employment data were used to determine average annual employment in each industry sector [California Employment Development Department (EDD), March 1995 benchmark]. Rates of work-related asthma by Standard Industrial Classification (SIC) division or group were calculated using EDD industry sector employment figures. The 1990 California census data were used as the denominator for calculating gender-and occupation-specific (COC) rates of work-related asthma. Overall rates of work-related asthma were lower when census data were used for the denominator, as the census denominator is larger than the EDD denominator.

Interviewed and non-interviewed cases were compared with respect to the distribution of demographic characteristics, COC, and SIC, using the χ^2 statistic [Epi Info v.6]. For all statistical analyses, data were grouped so that at least five cases were expected in each strata.

We used logistic regression to estimate the odds ratios for factors with potential to influence the classification of a case as work-aggravated vs. new onset [SAS, 1994]. All variables (age, gender, family history of asthma, allergies, current smoker, former smoker, occupation, industry) that were believed *a priori* to influence the outcome were included in the final model, whether or not the individual parameter estimates were statistically significant. Industry and occupational group were evaluated separately in logistic models due to non-independence between industry and occupation categories. No statistically significant effect was observed by industry group or occupational group when evaluated in separate models. Since independent analysis of occupation and industry yielded the same outcome as including both occupation and industry, we used the full model described above.

RESULTS

Rates of Work-Related Asthma

The 945 reports of work-related asthma eligible for interview were reported by 504 different physicians. The crude annual reporting rate for work-related asthma was 25/million employed workers (95% CI 23/million to 27/million) and 251/million (95% CI 246/million to 256/million) for all classified occupational respiratory illness. A higher case rate of work-related asthma was found among women (28/million) than among men (18/million).

Table I shows industry-specific average annual reporting rates of work-related asthma for major industry group divisions (SIC group divisions). Rates of work-related asthma using major SIC group divisions ranged from 11/million among employed persons in retail trade (SIC groups 52–59) to 44/million among non-Federal government workers (SIC groups 91–97). Specific industry groups with elevated rates of work-related asthma included local and suburban transit workers (127/million), electric, gas, and sanitary services workers (82/million), lumber and wood products workers (74/million), and chemical and allied products workers (52/million). The average annual reporting rate for work-related asthma was 25/million. A previous study estimated that DFRs identify only 32% of all occupational exposure cases [Blanc et al., 1989]. Based on this estimated underreporting, our adjusted annual reporting rate of work-related asthma is 78/million.

Table II shows annual rates of work-related asthma case reports by occupational group and by occupational category for those categories with rates greater than the overall reporting rate for work-related asthma. The highest rates of work-related asthma were reported among workers in the

TABLE I. Rates of Work-Related Asthma by Industry March 1, 1993—February 29, 1996

Division (SIC)	No. of cases	Rate/1,000,000[a]
Agriculture, forestry, and fishing (01—09)	40	36
Mining (10—14)	2	21
Construction (15—17)	31	22
Manufacturing (20—39)	125	23
Transportation and utilities (40—49)	70	38
Wholesale trade (50—51)	35	16
Retail trade (52—59)	72	11
Finance, insurance, real estate (60—67)	31	13
Services(70—89)	359	27
Government (91—97)	154	44
Non-classifiable	26	–
Total	945	25

[a]Average annual case rate per 1,000,000 employed workers in California (1993—1995 EDD data).

TABLE II. Rates of Work-Related Asthma by Occupation March 1, 1993—February 29, 1996

Occupation (COC)	No. of cases	Rate/1,000,000[a]
Managerial and professional	145	12
Registered Nurse (095)	28	49
Technical, sales, and admin. support	241	17
Health Technologists/Technicians (203–208)	28	66
Technicians, N.E.C.(235)	13	60
Eligibility Clerks (377)	9	296
General Office Clerks (379)	36	66
Teacher's Aide (387)	11	76
Services	171	30
Supervisor Firefighters (413)	4	248
Firefighters (417)	23	300
Police (418)	16	100
Correctional Inst. Officer (424)	8	173
Guards (426)	15	53
Food Preparation (444)	12	57
Nursing Aides (447)	18	40
Janitors and Cleaners (453)	32	625
Welfare Services Aides (465)	4	206
Farming, Forestry, and Fishing	42	32
Farm Workers (479)	19	48
Agricultural Graders/Inspectors (488-489)	4	82
Precision production, craft, and repair	72	14
Painters (579)	7	31
Operators, Fabricators, and Laborers	146	27
Mixers/Blender Operators (756)	4	169
Paint Spraying Machine Operators (759)	6	118
Welders (783)	10	67
Bus Drivers (808)	7	64
Material Moving Equipment Operators (859)	5	271
Laborers (889)	21	62
Non-classifiable	128	–
Total	945	21

[a]Average annual case rates per million employed workers (1990 Census Occupational Codes). Detail shown for occupational groups with at least 4 cases and asthma rate $>$ 25/million.

farming, forestry, and fishing trades (32/million), followed by services workers (30/million) and operators, fabricators, and laborers (27/million). Specific occupations with at least four cases and the highest rates of work-related asthma were janitors and cleaners (625/million), firefighters (300/million), and eligibility clerks (296/million).

Comparison of Interviewed and Non-Interviewed Reports

To assess the extent of participation bias, interviewed cases were compared with the non-interviewed reports with respect to gender, age, Hispanic ethnicity based on surname from the DFR, occupation, and industry group (Table III). Interviewed cases and non-interviewed reports differed significantly ($P < 0.05$) for all variables tested: age, gender, ethnicity, industry, and occupation. Interviewed cases were likely to be older, female, and non-Hispanic, and drawn from the technical sales industry and services occupation.

Figure 2 shows the final case classification for interviewed cases. Of the 444 cases in the final analysis, 65% (n = 290) were new onset work-related asthma and 35% (n = 154) were work-aggravated asthma. New onset asthma cases associated with a previously documented allergen

TABLE III. Characteristics of Interviewed and Non-Interviewed Asthma Reports March 1, 1993–February 29, 1996 (n = 945)

	No. interviewed (%) n = 459	No. not interviewed (%) n = 486	Total reports n = 945
Gender[a]			
Male	177 (41)	254 (59)	431
Female	282 (55)	231 (45)	513
Missing	0 (0)	1 (100)	1
Age group[a]			
<18	0 (0)	1 (100)	1
18–20	5 (33)	10 (67)	15
21–30	75 (40)	113 (60)	188
31–40	148 (49)	155 (51)	303
41–50	144 (57)	110 (43)	254
51–60	67 (56)	53 (44)	120
61–65	12 (57)	9 (43)	21
65+	4 (50)	4 (50)	8
Missing	4 (11)	31 (89)	35
Enthnicity[a]			
Hispanic	85 (42)	118 (58)	203
Non-Hispanic	374 (51)	366 (49)	740
Missing	0 (0)	2 (100)	2
Occupation[a]			
Managerial and professional	91 (63)	54 (37)	145
Technical sales	142 (59)	99 (41)	241
Services	77 (45)	94 (55)	172
Farming	17 (40)	25 (60)	42
Precision craft	26 (36)	46 (64)	72
Laborers	62 (42)	84 (57)	147
Non-classifiable	45 (35)	84 (67)	126
Industry[a]			
Agriculture, forestry, fishing	20 (43)	26 (57)	46
Mining	1 (50)	1 (50)	2
Construction	7 (23)	24 (77)	31
Manufacturing	58 (46)	67 (54)	125
Transportation and utilities	34 (49)	36 (51)	70
Wholesale trade	16 (46)	19 (54)	35
Retail trade	27 (38)	45 (63)	72
Finance, Ins., real estate	11 (35)	20 (65)	31
Services	197 (55)	160 (45)	357
Government	79 (52)	74 (48)	153
Non-classifiable	9 (39)	14 (61)	23

[a] χ^2 statistic significant at $P < 0.05$.

accounted for 8.8% (n = 39) of interviewed cases; new onset asthma not associated with a previously documented allergen represented 50.5% (n = 224) cases; and RADS accounted for 6.1% (n = 27) of interviewed cases.

Risk Factors and Exposures

We used a logistic regression model to estimate the risk factors associated with new onset cases using work-

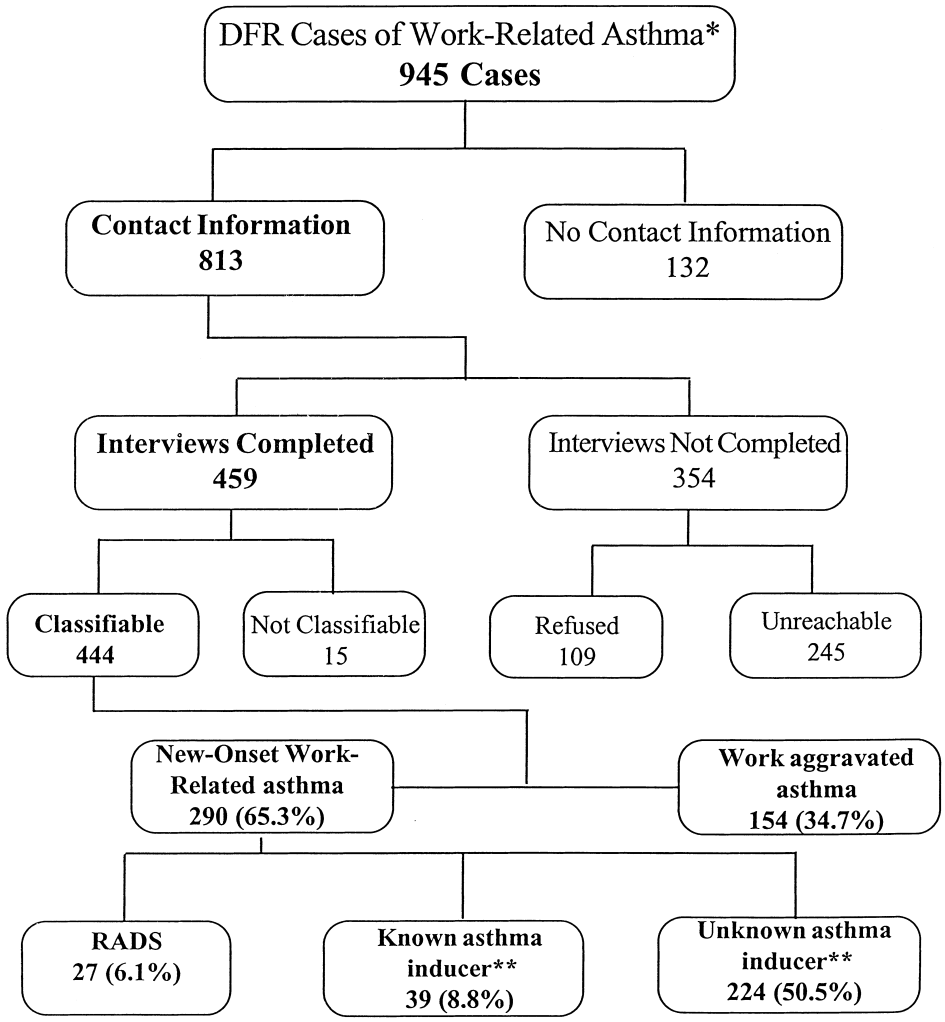

FIGURE 2. Case classification. * Physician diagnosis of asthma, reactive airways disease, or Reactive Airways Dysfunction Syndrome. ** Without objective evidence.

aggravated cases as the reference group. Variables included in the model were gender, age, family history of asthma, personal allergy history, smoking history, occupation and industry (Table IV). New onset work-related asthma cases are less likely to be female (OR = 0.46 (CI 0.26–0.85)); more likely to be older (mean age for new onset cases 41 vs. 38 for work-aggravated cases (OR for a 1 year increase in age = 1.04 (CI 1.01–1.07)); less likely to have a history of allergies (OR = 0.21 (CI 0.11–0.38)); and are less likely to have a family history of asthma (OR = 0.54 (CI 0.33–0.87).

The frequency of exposure agents for new onset and work-aggravated asthma cases is shown in Table V. The "miscellaneous chemicals" group included indoor air pollutants (n = 31), pesticides (n = 29), glues (n = 11), photo developing chemicals (n = 7), and textile dust (n = 5). The exposure agents for cases associated with a known asthma inducer included toluene diisocyanates (n = 8), formaldehyde (n = 4), California Redwood dust (n = 3), grain dust (n = 3), and egg protein (n = 3). The reported exposure agents for RADS cases were diverse and included ammonia gas, acids, dusts, and paint/lacquer.

Work Disability

After receiving a diagnosis of asthma on a DFR, physicians indicated that the majority of cases were unable to return to usual work. Of the 563 (60%) DFR reports that contained information on the ability to work based on physician assessment, 212 (38%) were considered able to perform their usual work, 115 (20%) were not able to perform their usual work, 183 (33%) were only able to perform modified work, and 53 (9%) cases' ability to work was indeterminate. For those cases (n = 298) that could not perform their usual work activities, 27% (n = 56) lost more than 7 workdays. Based on interview data, 139 (31%) reported that they were still exposed to the substances associated with their breathing problems, 283 (64%) were no longer exposed and 22 (5%) did not know if they were

TABLE IV. Risk Factors Associated with New Onset Asthma Cases (n = 444)

Variable	Odds ratio[b]	95% CI
Demographic and medical		
Age[a]	1.04	1.01–1.07
Gender—female[a]	0.46	0.26–0.85
Family history of asthma[a]	0.54	0.33–0.87
Atopic history[a]	0.21	0.11–0.38
Current smoker	0.5	0.24–1.05
Former smoker	1.23	0.68–2.22
Occupation		
Technical, sales, and admin. support	0.68	0.37–1.26
Services	0.90	0.42–1.92
Farming, forestry, and fishing	1.70	0.33–8.73
Precision production, craft, and repair	0.56	0.18–1.69
Operators, fabricators, and laborers	2.19	0.78–6.17
Industry		
Construction	3.87	0.24–61.4
Manufacturing	1.9	0.39–9.14
Transportation and utilities	0.46	0.09–2.37
Wholesale trade	2.94	0.17–51.8
Retail trade	0.89	0.16–4.82
Finance, insurance, real estate	0.71	0.1–4.91
Services	1.32	0.32–5.42
Government	1.31	0.31–5.53

[a]OR significant at $P < 0.05$.
New onset cases n = 290; work-aggravated cases n = 154.
[b]Adjusted ORs based on logistic regression model.

still exposed to substances associated with their breathing problems. At the time of follow-up interview, exposure to the associated agents had ceased for the following reasons: job change (12%), use of offending substance stopped (25%), engineering controls or new personal respiratory protection (5%), fired or laid off (10%), left job due to physician advise (15%), left job due to own health concerns (7%), and miscellaneous reasons (25%). A majority of interviewed cases (61%) reported that co-workers at their workplace also suffered from breathing problems. Among those asked about the status of worker's compensation claims (n = 141), 58 (41%) had filed a claim, 72 (51%) had not filed a claim, and 11 (8%) were unsure whether they had filed a claim. Information on claim status and resolution was not collected.

DISCUSSION

The diagnostic information contained on DFRs has been validated to be an accurate report of physician diagnoses of work-related illnesses. Although the diagnosis on the DFR is often based on a single medical visit, a follow-up study confirmed that the initial diagnosis was the same as the final diagnosis in 95% of cases [CDHS, 1989]. However, the 945 work-related asthma cases identified by the California SENSOR program probably represent an underestimate of the true number of work-related asthma cases in California. While DFRs are a statewide, mandatory reporting source tied to physician reimbursement and are intended for all occupational injuries and illnesses, work-related asthma is very likely under-diagnosed by physicians. Even after the diagnosis of work-related asthma, some physicians may not report cases on the DFR. Even if a DFR is completed, it may not be transmitted by the workers' compensation insurance carrier to State offices. All of these factors may lead to an underestimation of work-related asthma.

In the 1980s, California researchers estimated that of all occupational cases reported to California Poison Control Centers, only 32% were captured by the DFR [Blanc et al., 1989]. Although many appropriate cases are not reported through DFRs, no other reporting source was evaluated to be more comprehensive in reporting occupational illnesses than DFRs. If work-related asthma was underreported through DFRs at a rate similar to the other occupational illness diagnoses evaluated by California researchers using Poison

TABLE V. Exposure Agents Associated with Cases of Work-Related Asthma (n = 444)

Exposure agent[a]	New onset (%)	Work aggravated (%)	Total (%)
Miscellaneous chemicals[b]	97 (15.1)	47 (22.2)	144 (22.4)
Solvents petroleum derivatives, hydrocarbons	53 (8.3)	37 (17.5)	90 (4.0)
Mineral and inorganic dusts	42 (6.5)	35 (16.5)	77 (12.0)
Pyrolysis products	23 (3.6)	11 (5.2)	34 (5.3)
Acids, bases, oxidizers	19 (3.0)	11 (5.2)	30 (4.7)
Cleaning materials	22 (3.4)	8 (3.8)	30 (4.7)
Plant material	17 (2.6)	13 (6.1)	30 (4.7)
Animal materials	17 (2.6)	8 (3.8)	25 (3.9)
Miscellaneous inorganic compounds	13 (2.0)	5 (2.4)	18 (2.8)
Microorganisms	13 (2.0)	4 (1.9)	17 (2.6)
Exhaust	7 (1.1)	8 (3.8)	15 (2.3)
Diisocyanates	12 (1.9)	0 (0.0)	12 (1.9)
Welding	9 (1.4)	1 (0.5)	10 (1.6)
Halogens	4 (0.6)	6 (2.8)	10 (1.6)
Polymers	8 (1.2)	2 (0.9)	10 (1.6)
Physical factors	8 (1.2)	2 (0.9)	10 (1.6)
Aldehydes and acetals	8 (1.2)	1 (0.5)	9 (1.4)
Ethanolamines	8 (1.2)	1 (0.5)	9 (1.4)
Ammonia compounds	7 (1.1)	1 (0.5)	8 (1.2)
Wood dusts	6 (0.9)	0 (0.0)	6 (0.9)
Metal dust, NOS	4 (0.6)	0 (0.0)	4 (0.6)
Ketones	4 (0.6)	0 (0.0)	4 (0.6)
Waste	3 (0.5)	1 (0.5)	4 (0.6)
Alcohols	2 (0.3)	1 (0.5)	3 (0.5)
Halogenated aliphatic hydrocarbons	3 (0.5)	0 (0.0)	3 (0.5)
Pharmaceutical compounds	3 (0.5)	0 (0.0)	3 (0.5)
Insect materials	2 (0.3)	1 (0.5)	3 (0.5)
Metals	1 (0.2)	1 (0.5)	2 (0.3)
Fluorocarbons	0 (0.0)	2 (0.9)	2 (0.3)
Aliphatic nitrogen compounds	2 (0.3)	0 (0.0)	2 (0.3)
Rubber	2 (0.3)	0 (0.0)	2 (0.3)
Flours	2 (0.3)	0 (0.0)	2 (0.3)
Hexavalent chromium compound	1 (0.2)	0 (0.0)	1 (0.2)
Ethers	1 (0.2)	0 (0.0)	1 (0.2)
Epoxy compounds	1 (0.2)	0 (0.0)	1 (0.2)
Acetates	1 (0.2)	0 (0.0)	1 (0.2)
Cyanides and nitriles	1 (0.2)	0 (0.0)	1 (0.2)
Aliphatic and alicyclic amines	1 (0.2)	0 (0.0)	1 (0.2)
Organophosphate pesticides	1 (0.2)	0 (0.0)	1 (0.2)
Aquatic animal materials	1 (0.2)	0 (0.0)	1 (0.2)
Metal dust, NOS	4 (0.6)	0 (0.0)	4 (0.6)
Ketones	4 (0.6)	0 (0.0)	4 (0.6)
Waste/sewage	3 (0.5)	1 (0.5)	4 (0.6)
All other	26 (4.0)	10 (4.7)	36 (5.6)
Total	430	212	642

[a]Multiple exposure reported by cases.
[b]Includes indoor air.

Control Center cases, then the adjusted estimate of work-related asthma in California would be 78/million. This adjusted rate is close to the self-reported annual incidence rate of 80/million workers in Sweden [Toren, 1996] and the physician reported rate of 79/million for male workers in Quebec, Canada [Provencher et al., 1997]. The Surveillance of Work-Related and Occupational Respiratory Disease (SWORD) program reported average annual incidence rates of work-related asthma of 37/million for 1992–1993 and estimated that with consideration for underreporting, the actual rate of diagnosed work-related asthma is closer to 50/million workers [Meredith and Nordman, 1996]. In Finland, where reported rates of work-related asthma have been increasing and where reporting is likely to be more complete, the reported rate of work-related asthma was 150/million in 1991 [Kanerva et al., 1994]. The Michigan SENSOR program reported an annual incidence rate similar to California (29/million), and researchers suggested that the reported rate underestimated the true incidence of work-related asthma due to gross underreporting and misdiagnosis [Rosenman et al., 1997]. Analysis of the Michigan data using capture–recapture methods yielded adjusted annual incidence estimates of 58–204 cases/million/year of work-related asthma [Henneberger et al., 1999]. Comparisons with other surveillance data are useful but consideration must be given to the significant variation in disease definition, differences in reporting systems and compensation, and population differences such as industry and occupation frequencies.

The California industry groups with the highest rates of work-related asthma were local urban transit (127/million employed), electric, gas and sanitation (82/million employed), lumber and wood (74/million employed), chemical and allied products (52/million employed), and transportation equipment (51/million employed). High rates of work-related asthma are commonly reported for the chemicals and allied products sector, automobile sector, and lumber and wood industry sector [Rosenman et al., 1997]. Our findings of high rates of work-related asthma in these industry sectors suggest that current exposure standards for allergens and respiratory irritants are not fully implemented or that the standards are not sufficiently protective to prevent illness. In contrast, high rates of work-related asthma reported among public administration and government workers (44/million) are not typically reported. This finding largely reflects excess risk among the subgroup of justice, public order, and safety workers (SIC Group 92) who account for half of the cases in this occupational division. Forty percent ($n = 62$) of the public administration and government SIC major group were police officers, firefighters, or correctional officers, many of whom report exposure to mace, capsicum (pepper spray), smoke, and chemical irritants.

The California SENSOR data lack precision with regard to exposure agents. In the absence of routine work site evaluations and specific inhalation challenge testing results, exposure agents could not be confirmed. An average of 1.4 exposure agents were reported by each case. Forty-seven percent of cases reported two or more exposure agents, and 13% of cases reported three or more exposure agents. However, many workers were unable to specify their exposures and thus the broad category of miscellaneous chemicals was the most frequently reported exposure group. In some cases, the exposure process and agents were not known because the case was a bystander or had not received training on the content of the materials that they were required to utilize in their work.

Only 13.4% of new onset asthma cases (8.8% of all cases) were associated with exposure to a previously documented allergen. In contrast, over 70% of all new onset asthma cases (50.4% of all cases) were associated with exposure to agents that were not known to be allergens [AOEC, 1996]. The proportion of cases associated with exposure to agents not known to be allergens is higher than previous reports. The Michigan SENSOR program reported 53% of all cases associated with exposure to a previously documented allergen, and only 30% of all cases associated with exposure to agents not previously documented to be allergens. In the West Midlands region of the UK, physicians reported that an irritant mechanism was responsible for 18% of all reported occupational asthma [Gannon and Burge, 1993]. Many of the agents not known to be allergens are irritants to the respiratory tract. Asthma related to respiratory irritant exposures has been previously described [Kennedy, 1992]. The varying proportion of asthma cases associated with these types of exposures from different surveillance schemes may represent differences in case reporting sources. The reporting systems in the UK and Michigan rely on sentinel occupational and chest clinic reports, while the DFR system used to identify cases in California obtains reports from all types of physicians in all types of care settings. Indeed, the 945 cases in California were reported by 504 different physicians. Exposure to agents not known to be allergens may be reported more frequently by non-occupational or non-pulmonary physicians who evaluate cases from a wide variety of occupational settings. Alternatively, these physicians may not obtain as complete an occupational history or may fail to identify exposure to the offending allergen. Furthermore, reported exposures are not confirmed by workplace investigation, perhaps leading to an overestimate of the proportion of cases due to agents not known to be allergens. For these reasons, the proportion of cases in California that are not associated with an agent known to be an allergen may accurately represent the spectrum of cases identified from a general medical practice.

More than one third (35%) of all our reported cases were identified as work-aggravated asthma. This proportion of work-aggravated cases is higher than previously reported

from the other three SENSOR programs (9.9% of all Michigan cases, 12.8% of all Massachusetts cases, and 12.9% of all New Jersey cases) or reported from a Seattle occupational medicine clinic (27% of all cases) [Jajosky et al., 1999; Wheeler et al., 1998]. In contrast to these reporting systems, the majority of asthma reports in California derive from non-occupational physicians. In contrast to occupational medicine specialists, non-occupational physicians may be more likely to recognize a case of work-aggravated vs. new onset asthma. New onset asthma cases may require the physician to identify workplace risk factors through a more comprehensive occupational history. Therefore, a greater proportion of work-aggravated cases may be reported in California if non-occupational physicians seek reimbursement for treatment of non-occupational asthma that has been aggravated by workplace exposure.

The logistic regression model that compared new onset work-related asthma with work-aggravated asthma highlighted differences between these two groups. Cases with a history of allergies or family history of asthma were approximately half as likely to be a new onset case. Individuals with predisposing risk factors for asthma, atopy and family history of asthma, are likely to first develop asthma due to non-work exposures, but then experience work-related exacerbation of pre-existing asthma. Similar findings were reported from British Columbia where atopy, family history of asthma, but not smoking, were important risk factors for asthma prior to first employment [Siracusa et al., 1995].

CONCLUSIONS

The California SENSOR program has been successful in establishing and maintaining a surveillance system for the analysis of work-related asthma incidence in California. The surveillance program has offered technical assistance to more than 400 workers with work-related asthma. Work-related asthma reporting rates are highest among transit, utility, automobile manufacture, chemical products manufacture, and health care workers and lowest among financial institution workers. The California surveillance system, similar to other state and national surveillance systems, likely provides a very conservative estimate of the incidence of work-related asthma. The actual rate in California is estimated to be 78/million employed workers.

The California Occupational Health Branch will continue efforts to educate physicians about the diagnosis and reporting of work-related asthma to enhance the quality of surveillance data and improve the treatment and health of affected workers. Increased opportunities for prevention arise from work site investigations and interventions. Seven work site investigations were conducted that resulted in recommendations to improve workplace exposures. Little recognized exposures, including graffiti removers and capsicum were found to be associated with new onset work-related asthma. This surveillance system will continue to contribute to an increased understanding of the impact of work-related asthma among affected workers, improve recognition of possible asthma inducing agents, and increase physician recognition of disease and early exposure cessation.

ACKNOWLEDGMENT

The California SENSOR program is funded through a cooperative agreement (#U60-CCU902990-06) with the National Institute for Occupational Safety and Health. The authors wish to thank Lilia Vargas of the Division of Labor Statistics and Research for assistance with data collection. In addition, many thanks to the careful work of study interviewer Margo Marcellini, M.P.H.

REFERENCES

Ameille J, Pairon JC, Bayeux MC, Brochard P, Choudat F, Conso A, Devienne A, Garnier R, Iwatsubo Y. 1997. Consequences of occupational asthma on employment and financial status: a follow-up study. Eur Respir J 10:55–58.

Association of Occupational and Environmental Clinics (AOEC). 1996. AOEC exposure database coding system. 1010 Vermont Ave., Suite 513, Washington, DC 20005. Current exposure database on the internet at: www.aoec.org/aoecode.htm

Blanc PD, Rempel D, Maizlish N, Hiatt P, Olson KR. 1989. Occupational illness: case detection by poison control surveillance. Ann Intern Med 111:238–244.

California Department of Health Services. 1989. Provider-based surveillance of work-related illness: the California Doctor's First Report of occupational illness or injury. 1515 Clay St., Oakland, CA 94612. #SR-87-001:1–26.

California Labor Code. 1994. Occupational injury or illness reports; section 6409:64.

Centers for Disease Control and Prevention. 1995. Asthma–United States 1982–1992. JAMA 273:451–452.

Chan-Yeung M. 1990. State of the art—occupational asthma. Chest 98:148s–161s.

Chan-Yeung M, Malo J-L. 1994. Aetological agents in occupational asthma. Eur Respir J 7:346–371.

Epi Info, Version 6: a statistics program. 1990. USD, Inc., Stone Mountain, GA.

Gannon PF, Burge PS 1993. The SHIELD scheme in the West Midlands Region, United Kingdom. Br J Ind Med 50:791–796.

Henneberger PK, Kreiss K, Rosenman KD, Reilly MJ, Chaǹg YF, Geidenberger CA. 1999. An evaluation of the incidence of work-related asthma in the United States. Int J Occup Environ Health 5(1):1–8.

Jajosky RA, Harrison R, Reinisch F, Flattery J, Chan J, Tumpowsky C, Davis L, Reilly MJ, Rosenman KD, Kalinowski D, Stanbury M, Schill DP, Wood J. 1999. 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. 1999. In: CDC Surveillance Summaries, June 25, 1999. MMWR 48:SS-3:1–20.

Kanerva, L, Jolanki, R, Toikkanen J. 1994. Frequencies of occupational allergic diseases and gender differences in Finland. Int Arch Occup Environ Health 66:111–116.

Kennedy SM. 1992. Acquired airway hyperresponsiveness from non-immunogenic irritant exposure. Occup Med 7:287–300.

Kogevinas M, Anto JM, Sunyer J, Toblas A, Kromhout H, Burney P. 1999. Occupational asthma in Europe and other industrialized areas. Lancet 353:1750–1754.

Matte TD, Hoffman RE, Rosenman KD, Stanbury M. 1990. Surveillance of occupational asthma under the SENSOR model. Chest 98:173S–178S.

Meredith S, Nordman H. 1996. Occupational asthma: measures of frequency from four countries. Thorax. 51:435–440.

Milton DK, Solomon GM, Rosiello RA, Herrick RF. 1998. Risk and incidence of asthma attributable to occupational exposure among HMO members. Am J Ind Med 33:1–10.

Provencher S, Labreche FP, De Guire L. 1997. Physician based surveillance system for occupational respiratory diseases: the experience of PROPULSE, Quebec, Canada. Occup Environ Med 54:272–276.

Rosenman KD, Reilly MJ, Kalinowski DJ. 1997. A state-based surveillance system for work-related asthma. J Occup Environ Med 39(5):415–425.

SAS Institute Inc. 1994. SAS PC Windows version 6.10. Ćary, NC.

Siracusa A, Kennedy SM, DyBuncio A, Lin FJ, Marabini A, Chan-Yeung M. 1995. Prevalence and predictors of asthma in working groups in British Columbia. Am J Ind Med 28:411–423.

Tarlo SM, Liss G, Corey P, Broder I. 1995. A workers' compensation claim population for occupational asthma. Chest 107:634–641.

Taylor AN. 1995. Environmental determinants of asthma. Lancet 345:296–299.

Toren K. 1996. Self reported rate of occupational asthma in Sweden 1990–92. Occup Environ Med 53:757–761.

United States Census Bureau. 1980. Passel—Word Spanish surname list. Washington, DC.

Weiss KB, Gergen PJ, Wagener DK. 1993. Breathing better or worse? The changing epidemiology of asthma morbidity and mortality. Ann Rev Public Health 14:491–513.

Wheeler S, Rosenstock L, Barnhart S. 1998. A case series of 71 patients referred to a hospital-based occupational and environmental medicine clinic for occupational asthma. West J Med 168:2:98–104.