Silicosis and Workers' Compensation in New Jersey

Martha Stanbury, MSPH
Patrick Joyce, JD, MD, MPH
Howard Kipen, MD, MPH

The employer is expected to maintain responsibility for health care expenses and lost income that result from occupational injury and illness through the workers' compensation insurance system. However, financial support for individuals with occupational illnesses, especially those with long latency, is often from sources other than workers' compensation. Silicosis, a well defined, chronic, occupational lung disease, can be viewed as a sentinel for the inadequacy of the public policy to compensate workers for chronic occupational lung disease. Three hundred twenty-nine patients with confirmed silicosis were identified by the silicosis surveillance program in the New Jersey Department of Health using source data from 1979 through 1992. One hundred seventy-seven of these individuals provided information on the status of any compensation claims against their employer. Only 31% of these patients stated that a claim had been filed; 84% of those whose claims were settled were awarded payments. Severity of radiologic findings was not associated with the likelihood of filing a claim or with being awarded a payment; whereas, smoking was associated with these outcomes. The implications of these findings for the health care system are discussed.

It is accepted public policy that health care expenses and lost income that result from occupational injury and illness should be the financial responsibility of the employer through workers' compensation insurance. For occupational illness, however, the financial responsibility has often been borne by general health insurers or the public through Social Security disability, Medicare, or other forms of public assistance rather than workers' compensation.[1,2] Such an externalization of costs arising from occupational disease does not promote safety and health in the workplace and is contrary to the public policy of employer responsibility for the care of patients with occupational disease.

Since World War I, workers injured on the job have usually received compensation through the state workers' compensation system rather than through a personal injury lawsuit against the employer. For the most part, workers' compensation has been the only recourse for compensation available to workers. Generally, where the injury arises out of and in the course of employment, the employer's liability is established subject to a few transactional hurdles, such as timely filing of a claim. The workers' compensation system has been much slower to recognize and accommodate the work-relatedness of certain diseases than injuries. Silicosis, a well defined lung disease that is known to occur only as a result of continued occupational exposure to airborne silica, was the first chronic disease to be compensable under any state workers' compensa-

From the Division of Epidemiology, Environmental, and Occupational Health Services, New Jersey Department of Health, Trenton, New Jersey (Ms Stanbury); Washington County Hospital Association, Maryland (Dr Joyce); and Department of Environmental and Community Medicine, Robert Wood Johnson Medical School, Piscataway, New Jersey (Dr Kipen).

This work was completed while Dr Joyce was a resident in the Department of Environmental and Community Medicine, Robert Wood Johnson Medical School, Piscataway, NJ.

Address correspondence to: Martha Stanbury, MSPH, Occupational Health Service, New Jersey Department of Health, CN 360, Trenton, NJ 08625-0360.

1076-2752/95/3712-1342$3.00/0

tion system. It was incorporated into most state workers' compensation programs by the early 1940s, subsequent to a large number of silicosis deaths associated with construction of a tunnel at the Gauley Bridge in West Virginia in the 1930s.[3]

The provision of health care and income replacement for patients with silicosis can be viewed as a sentinel for the inadequacy of the public policy to compensate workers for chronic, long latent, occupational diseases. The New Jersey Department of Health maintains a register of silicosis patients.[4] In this study, data from confirmed cases in the register are used to assess the adequacy of the workers' compensation system to compensate patients with silicosis.

Methods

The New Jersey Department of Health (NJDOH) is one of several state health departments with silicosis surveillance systems that participate in the Sentinel Event Notification System for Occupational Risk (SENSOR) program.[5] SENSOR is a state-based, occupational disease surveillance project that is funded by the National Institute for Occupational Safety and Health (NIOSH). Physicians and hospitals are required by regulation to notify the NJDOH of silicosis patients. Case reports are compiled from physician reports, hospital discharge data, computerized mortality data where the underlying or contributing cause of death was silicosis (ICD 502), and a limited review of medical screening records from companies.

Following receipt of a report of a case of silicosis, the NJDOH interviews the patient or next of kin and requests supporting medical records and chest roentgenographs. The interviewer obtains information on occupational history, claims status regarding workers' compensation, smoking, and demographic background.

The medical records, work history, case information, and chest roentgenograph are reviewed by a NIOSH certified physician ("B-reader") to confirm the diagnosis. NIOSH has published a case definition that includes a history of occupational exposure to silica and a chest radiograph or other imaging technique interpreted as consistent with silicosis or pathologic findings consistent with silicosis.[6] Radiologically, the findings of silicosis include bilateral, rounded opacities in the upper lung zones. The opacities are classified by the International Labour Organization system[7] into four major categories of profusion or concentration: 0, 1, 2, or 3. In category 0, small opacities are absent or very few; in category 3, small opacities are very numerous and normal lung markings are usually obscured. Opacities larger than 1 cm are indicative of complicated silicosis, also known as progressive massive fibrosis. Patients found to conform to the SENSOR epidemiologic case definition are classified as patients with confirmed silicosis. Confirmed cases reported from 1979–1992 form the data base for this study.

Results

Three hundred twenty-nine (36%) of the 917 reported cases were patients with confirmed silicosis. An additional 148 (16%) of the 917 reported cases had worked primarily in coal mines and had little or no work in a known silica-using industry; they were classified as having coal workers' pneumoconiosis rather than silicosis. The remaining 440 patients included those with insufficient data to make a determination (284; 31%), individuals with asbestosis (29, 3%), and those not meeting radiologic or occupational exposure criteria (127, 14%). Two hundred sixty-eight (81%) of the 329 patients with confirmed silicosis were first identified by the NJDOH silicosis register from hospital discharge data. Physicians reported 19 (6%) patients with confirmed silicosis. Twenty-seven (8%) of the 329 patients with confirmed silicosis were identified from medical screenings at five work sites where patients with confirmed silicosis had been identified previously. The remaining 15 (5%) patients were first identified by death certificates.

The mean age of the 309 patients for whom birth dates were known was 71 years at time of report, with 85% being age 60 and older. Two hundred ninety-seven (90%) patients were men; 254 (85%) of the 300 individuals with known race were white. Onset of exposure was known for 250 patients. One hundred seventy-nine (72%) of the 250 patients were first exposed before 1950; four (2%) were first exposed since 1970. The mean duration of exposure for these 250 patients was 27 years with a range from 1 to 65 years. Exposure to silica occurred predominately in potteries, foundries, and sand or other surface mining (Table 1).

One hundred seventy-seven of the 329 cases, or their next of kin, who could be contacted and who gave consent, were interviewed and asked about the status of any compensation claims they had filed against an employer. The 27 cases identified through screening were not asked this question at the time of the screening and were not followed up to determine whether, subsequent to the screening diagnosis, they filed any claims against their employers. The remaining 125 individuals either declined to be interviewed or could not be contacted.

Table 2 shows the results of the 177 interviews. Fifty-five (31%) patients filed a claim. Forty-four of the claims were settled: 37 (84%) of settled claims were awarded, and 7 (16%) were denied. Eleven (20%) claims were pending or the results were unknown at the time of the interview. Individuals first exposed after 1950 were more likely to file a claim than were individuals exposed before 1950 (50% compared with 28%), and, of the 44 individuals whose claims were settled, those exposed after 1950 were more likely to be awarded a payment than those exposed before 1950 (100% com-

TABLE 1
Industries in Which Silica Exposure Occurred among the Patients with Confirmed Silicosis in the New Jersey Silicosis Surveillance System

Industry			No. of Patients (%)
Mining			41 (12)
Construction			24 (7)
Manufacturing			250 (76)
Chemicals and allied products		6	
Rubber and miscellaneous plastics		8	
Stone, clay, and glass		144	
Structural clay	23		
Pottery and related products	76		
Other stone clay	45		
Primary metals		76	
Foundries	57		
Others	19		
Fabricated metal products		8	
Other manufacturing		8	
Other			7 (2)
Unknown			7 (2)
Total			329 (100)

TABLE 2
Claims Filing Status for Patients with Confirmed Silicosis in the New Jersey Silicosis Surveillance System

Filing Status			No. of Patients (%)
Never filed			122 (69)
Filed			55 (31)
Settled		44 (80)	
Awarded	37 (84)		
Denied	7 (16)		
Pending		11 (20)	
Total			177 (100)

pared with 70%). Forty-one (32%) of the 127 individuals who had ever smoked had filed a claim; 12 (25%) of the 48 who had never smoked had filed a claim. (Smoking status was unknown for two individuals.) Smoking and the claim status data were available on 42 of the 44 patients whose claims were settled. Of the 32 individuals who had ever smoked, 29 (91%) were awarded payment; compared with six (60%) of the 10 who had never smoked.

The severity of disease can be estimated by radiologic findings, including the presence of large opacities and the profusion of small opacities. One hundred forty-seven patients had both radiologic findings and interview data available. Eleven (37%) of the 30 patients with large opacities reported having filed claims, whereas 37 (32%) of the 117 patients without large opacities reported having filed claims (Table 3). Of the 41 patients with known radiologic findings and whose claims were settled, those with large opacities were somewhat less likely than patients without large opacities to be awarded a payment (71%/82%). Patients with the greatest profusion of small opacities (category 3 in the ILO system) were somewhat less likely to file a claim than those patients with lesser profusions (Table 3) and were less likely to be awarded a payment (67% awarded: profusion = 3; 83% awarded: profusion < 3). There were no statistically significant associations between radiologic findings and filing of claims or being awarded payments.

Discussion

Only 31% of interviewed patients with confirmed silicosis filed a claim, and 84% of those with settled claims were awarded payments. Severity of radiologic findings was not associated with the likelihood of filing a claim or with being awarded a claim. These findings are similar to findings from Michigan SENSOR silicosis surveillance data.[8] Those who had ever smoked were more likely to file a claim and were more likely to be awarded a payment than those who had never smoked. These findings may be due to an interaction between silicosis and health effects from smoking—an interaction that increases impairment[9] or reduces the latency of impairment, causing the disease to manifest during employment.

Individuals, or their survivors, were not asked why they did not file a claim. Possible explanations could include satisfaction with medical care paid for by regular health insurance, inability to locate the responsible employer, failure to associate the disease with previous employment where there was exposure to silica, loyalty to their employers, the administrative problems of filing a claim, lack of physician support, or ignorance of the compensability of the disease. Many of these individuals had multiple medical problems, and silicosis often was not the primary reason for hospitalization. The age of the patients, the complexity of their medical presentations, and the long latency of silicosis contribute to the plausibility of these explanations. Nevertheless, all patients had received a diagnosis of silicosis from the treating physician.

These 177 interviewed individuals represent only 54% of the 329 patients with confirmed silicosis. Additionally, 284 reported patients were neither confirmed nor excluded because there was insufficient information to make a determination (ie, no work history and/or no radiologic data). Data are presented in Table 4 that address the representativeness of the group of 177 confirmed/interviewed patients compared with the confirmed/not interviewed and the unconfirmed patients. There were no differences in the three groups regarding gender or mean age at the time of the report. There was a sta-

TABLE 3
Radiologic Findings and Claims Status for Patients with Confirmed Silicosis in the New Jersey Silicosis Surveillance System

	Presence of large opacities		Total
	Present	Absent	
Claims Status			
Filed	11 (37)	37 (32)	48 (33)
Not filed	19 (63)	80 (68)	99 (67)
Total	30 (100)	117 (100)	147 (100)

	Profusion: Small opacities			Total
	0, 1	2	3	
Claims Status				
Filed	19 (32)	22 (36)	7 (26)	48 (33)
Not Filed	40 (68)	39 (64)	20 (74)	99 (67)
Total	59 (100)	61 (100)	27 (100)	147 (100)

tistically significant difference between the interviewed and not interviewed groups regarding race, with whites being more likely to be interviewed. The greater proportion of nonwhites in the not interviewed groups may have been because nonwhites were less likely to maintain a permanent residence because of lower socioeconomic states, and thus were less likely to be located for an interview. Based on a recent study of workers' compensation for nonmalignant, asbestos-related lung disease which found that workers' compensation claims of nonwhites were rejected more often than those of whites,[10] it is reasonable to speculate that nonwhites with silicosis have had even less success with compensation than whites have had in New Jersey. Individuals with more severe radiologic findings (ILO $\geq$ 2/1) were also more likely to be interviewed than not interviewed (odds ratio, 1.79; CL, 1.08–2.99). Thus, the low rate of filing a claim cannot be attributed to a respondent bias toward less radiographically severe disease.

There was a significantly greater proportion of individuals identified from sources other than hospitals in the confirmed/not interviewed group than the other groups. This excess can be mostly explained by reports of workers diagnosed as part of several company screenings in the confirmed/not interviewed group. The B-reader results were provided by the reporting physicians, but addresses and telephone numbers of the employees often could not be obtained from the employers or reporting physicians. It is unknown whether individuals diagnosed from company sponsored screenings would be more likely to seek workers' compensation than individuals whose diagnoses were not associated with their employers. It is reasonable to assume, however, that filed claims in this group would most likely be awarded.

Similar to our results with silicosis patients, studies of patients with asbestos-related diseases have found that relatively few patients or their next of kin filed for workers' compensation benefits.[11,12] However, unlike our results, where 84% of filed claims were awarded payment, less than 65% of filed asbestos disease claims were awarded payment.[10,11] We have no explanation for this difference.

It has been estimated that nearly 60% of all occupational disease workers' compensation claims are denied initially; in comparison, only 10% of the more traditional injury cases are denied.[13] Whereas injury cases are often settled without formal adjudication or the need for counsel to appear at a hearing, disease claims are usually contested because of the increasing monetary value of the claims for occupational illness and disputes over the occupational nexus. For occupational disease claims, the workers' compensation system is more like traditional tort litigation than it was designed to be. In this study, those few individuals who filed claims seemed to be relatively successful in being awarded payment, particularly when associated with recent exposure. It should be noted that the outcome of the claims on 20% of this study population was still pending at the time of the interviews, suggesting that the claims process is lengthy.

The population of silicotics in this study may not be representative of all patients with silicosis in the state because the primary source of case reports was hospital discharge data, which capture sicker and older individuals. It has been estimated that there is over 50% underreporting of the incidence of silicosis when diagnostic information only from hospitalized patients is used.[14] Younger individuals may be more likely to file a claim against their employer than are elderly, hospitalized patients. In this study, a greater proportion of individuals exposed after 1950 filed claims than those exposed before 1950.

The diagnosis and etiology of silicosis are understood, but silicosis is underreported, inadequately compensated, and underwritten by the public at large or by health insurance plans rather than by the responsible employers. As a disease, it is a sentinel for the inadequacy of occupational disease compensation. Studies of the magnitude of occupational disease have documented the disparity between the number of individuals with occupational disease identified by, and presumably compensated by, workers' compensation data and the much larger number identified in

TABLE 4
Comparison of Confirmed/Interviewed with Confirmed/Not Interviewed and Unconfirmed in the New Jersey Silicosis Surveillance System

	Confirmed/ Interviewed (%)*	Confirmed/ Not Interviewed (%)†	Unconfirmed (%)‡
Gender			
male	158 (89)	139 (91)	242 (85)
female	19 (11)	13 (9)	42 (15)
Total	177	152	284
Race*			
white	156 (91)	98 (75)	†
non-white	14 (9)	32 (25)	†
Total known	170	130	
Mean age at time of report	71 (n = 175)	72 (n = 134)	69 (n = 258)
Chest roentgenograph interstitial opacification‡			
<2/1	59 (40)	71 (55)	N/A
≥2/1	88 (60)	59 (45)	N/A
Total	147	130	
First source of report			
Hospital	156 (88)	112 (74)	259 (91)
Other	21 (12)	40 (26)	25 (9)

* Statistically significant: odds ratio, 2.02; 95% CL 1.29–3.16.
† Race was known for only 25 cases and thus is omitted.
‡ Statistically significant: odds ratio, 1.79; 95% CL 1.08–2.99.

other data sources such as hospital discharge data, physician reports, and other sources.[15–17] If the proportion of patients compensated for silicosis, a clearly compensable disease, is so small, one may assume that compensation for occupational diseases that are multifactorial in causation is even less adequate. Many of the work sites where these patients were exposed are still in operation and still exposing employees to hazardous levels of silica dust.[4] Failure to compensate silicosis patients for medical care undercuts the need for economic incentives for employers and employees to follow good health and safety practices in areas at risk for airborne silica exposure.

Workers' compensation was designed "to provide a dependable minimum of compensation to insure security from want during a period of disability."[18] It does not seem to provide security for the majority of this population of silicosis patients. A redesigned health care system must craft a system to compensate victims of occupational disease that maintains the concept of employer responsibility for the occupational diseases of employees, including those employees suffering from diseases like silicosis that have long latency. Physicians have an integral role in the diagnosis, treatment, and reporting of occupational diseases such as silicosis within any system.

Acknowledgments

Funding for this study was provided by the SENSOR cooperative agreement from the National Institute for Occupational Safety and Health to the New Jersey Department of Health and by the New Jersey Department of Health. Dr Joyce was supported by NIOSH training grant TH20H07125. The authors thank Fran Rizzo, Shafiq Rab, Gwendoline Solice-Sample, and David Valiante for their efforts to identify and follow up with silicosis patients.

References

1. *An Interim Report to Congress on Occupational Diseases.* Washington, DC: U.S. Department of Labor; 1980:54–97.
2. *Occupational Illness Data Collection: Fragmented, Unreliable and Seventy Years behind Communicable Disease Surveillance.* Washington, DC: U.S. Congress, Committee on Government Operations; 1984:4.
3. Rosner D, Markowitz G. *Deadly Dust: Silicosis and the Politics of Occupational Disease in the Twentieth Century.* Princeton, NJ: Princeton University Press; 1991:188.
4. Valiante D, Rosenman KD. Does silicosis still occur? *JAMA.* 1989;21:3003–3007.
5. Reilly NJ, Rosenman KD, Watt F, et al. Silicosis surveillance—Michigan, New Jersey, Ohio, Wisconsin. *MMWR.* 1993; 42/No. SS-5:23–28.
6. Centers for Disease Control and Prevention. Silicosis: cluster in sandblasters—Texas, and occupational surveillance for silicosis. *MMWR. Morb Mortal Wkly Rep.* 1990;39:433–437.
7. *ILO International Classification of Radiographs of Pneumoconiosis.* Geneva: Occupational Safety and Health Service, International Labour Office; 1979.
8. Rosenman KD, Reilly MJ, Watt FC. *1993 Annual Report on Silicosis in Michigan.* Lansing, MI: Michigan Department of Health; 1993.
9. Hnizdo E. Loss of lung function associated with exposure to silica dust and with smoking and its relation to disability and mortality in South African gold miners. *Br J Ind Med.* 1992;49:472–479.
10. Nevitt C, Daniell W, Rosenstock L. Workers compensation for nonmalignant asbestos-related lung disease. *Am J Ind Med.* 1994;26:821–830.
11. Barth P. Compensation for asbestos-associated disease: a survey of asbestos insulation workers in the United States and Canada. In Selikof IJ, ed. *Disability Compensation for Asbestos-associated Disease in the United States.* New York: Mount Sinai School of Medicine; 1982; 213–291.

12. Stanbury MJ, Tepper A, Ramaprasad R, Solice-Sample G. Asbestosis in New Jersey: a survey of hospitalized patients. *New Jersey Med.* 1991;88:195–199.
13. Barth P, Hunt H. *Workers' Compensation and Work-related Illnesses and Disease.* Cambridge, MA: MIT Press; 1980:256.
14. Rosenman KD, Trimbath L, Stanbury MJ. Surveillance of occupational lung disease: comparison of hospital discharge data to physician reporting. *Am J Public Health.* 1990;80:1257–1258.
15. Markowitz S, Fischer E, Fahs M, Shapiro J, Landrigan P. Occupational disease in New York State: a comprehensive examination. *Am J Ind Med.* 1989;16:417–435.
16. Pollack ES, Keimig DG. *Counting Injuries and Illnesses in the Workplace: Proposals for a Better System.* Prepared by the Panel on Occupational Safety and Health Statistics, Committee on National Statistics, National Research Council. Washington, DC: National Academy Press; 1987.
17. Windau J, Anderson H, Rosenman KD, Rudolph L, Stanbury MJ, Stark A. The identification of occupational lung disease from hospital discharge data. *J Occup Med.* 1991;33:1060–1066.
18. Naseef v. Cord, Inc; 48 N.J. 317, 325, 225 A. 2d 343, 348 (1966).

The Mayan Dust Bowl

By their own accounts, which they carved in hieroglyphics on spectacular monuments, the Maya who lived in the lowlands of the southern Yucatan peninsula between 250 and 900 A.D. were an unusually gifted and cultured people. Well-versed in art and architecture, sophisticated in agriculture, they seemed destined for a long and rosy future. But between 800 and 900 A.D., just when classic Mayan civilization reached its apex, it suddenly collapsed. Archaeologists have long argued about the reason; causes that have been suggested include overpopulation, disease, insect plagues, peasant revolts against the ruling classes, civil wars, and climate change.

The climate-change theory now has some objective evidence to back it up. Beginning around 800 A.D., says paleoclimatologist David Hodell of the University of Florida, the Yucatan suffered a severe 200-year drought. This left the region drier than it has ever been in the past 7000 years—and according to Hodell, it left the Maya unable to sustain their monumental cities.

Hodell and his colleagues examined sediments laid down over many millenia at the bottom of Lake Chichancanab in central Yucatan. They found two pieces of evidence for a drought between 800 and 1000 A.D. First, the sediments deposited in that period were especially rich in gypsum or calcium sulfate. Normally, that mineral is dissolved in the lake water; it precipitates out as a solid and settles to the bottom only when there is too little water in the lake to dissolve it all—that is, during a drought. Second, the researchers examined the ratio of oxygen 18—a heavy oxygen isotope—to ordinary oxygen 16 in crustacean and snail shells in the Lake Chichancanab sediments. Because oxygen 16 is lighter, water molecules that contain it evaporate more readily. As the climate becomes drier and evaporation increases, a proportionately greater amount of oxygen 18 remains behind in the lake. Hodell's group found that the ratio of oxygen 18 to oxygen 16 in the Lake Chichancanab sediments was greatest, indicating that the climate was driest, between 800 and 1000 A.D.

Hodell believes that repeated failure of the summer rains during this period may have set off a chain of disasters including some of the ones suggested by other researchers that ruined the Maya. "The onset of this drought resulted in crop failure, which may have resulted in famine, which may have made the people more susceptible to disease," he says. "You would also have more competition for food. That might explain the increased warfare between the various Maya city-states."

From Breakthroughs. *Discover,* 1995;16:22.