

# Previously Undetected Silicosis in New Jersey Decedents

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**Background** *Despite a reported decline in mortality and hospitalizations associated with silicosis [U.S. Department of Health and Human Services, 1999], this decline may be artifactual, stemming in part from underdiagnosis by physicians.*

**Methods** *This study estimates, through radiological confirmation, the prevalence of unrecognized silicosis in a group of silica-exposed New Jersey decedents whose cause of death was chronic obstructive pulmonary disease (COPD), tuberculosis, or cor pulmonale. Two expert readers re-evaluated the chest X-rays of this group to determine the presence or absence of silicosis. The study population was considered to be presumptively exposed to silica dust by virtue of their usual industry of employment as listed on the death certificate.*

**Results** *Radiographic evidence of silicosis was found in 8.5% of this population, and evidence of asbestosis was found in another 10.7%, for a total of 19.2%.*

**Conclusions** *The existence of previously unrecognized silicosis and asbestosis in 19.2% of this study group suggests that occupational lung disease is under-recognized and, hence, undercounted. Am. J. Ind. Med. 44:304–311, 2003. © 2003 Wiley-Liss, Inc.*

**KEY WORDS:** *silicosis; New Jersey; surveillance; occupational disease/diagnosis; asbestosis; pneumoconiosis/radiography*

## INTRODUCTION

Silicosis is an irreversible lung disease caused by work-related exposures to dust containing crystalline silica. Because the body cannot clear or metabolize the respirable portion of the inhaled mineral dust particles, fibrosis develops

in the upper regions of the lungs, which interferes with their normal expansion. The immune system scavenger cells known as alveolar macrophages are destroyed, with fibrotic nodules forming around them [Balaan and Banks, 1998]. Although there is no cure for this disease and treatment is only palliative at best, silicosis is entirely preventable with dust control measures at the workplace. Each case usually signifies the existence of other cases at the same work site, and therefore a missed case implies other missed cases. The under-counting of silicosis—and other occupational disease—is a paramount public health issue in that it undermines the justification of resources for preventive programs to curb if not eliminate such disease.

Silica exposure brings about changes to the immune system [Hnizdo and Murray, 1998; Haustein et al., 1990; Rosenman et al., 1999], and the World Health Organization's International Agency for Research on Cancer [IARC] in 1996 designated crystalline silica as a human carcinogen, group 1 [International Agency for Research on Cancer, 1997], although this finding is still debated [Checkoway and Franzblau, 2000].

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Silicosis is strongly associated with chronic obstructive pulmonary disease (COPD) [Chia et al., 1992; Rosenman and Zhu, 1995], which appears frequently as a cause of death among workers in silica-exposed occupations [U.S. Department of Health and Human Services, 1994]. Cor pulmonale, or right heart failure, is an end-stage complication of silicosis. Studies in Ontario [Kusiak et al., 1993] and South Africa [Murray et al., 1993] have shown silicosis to be a significant risk factor for cor pulmonale.

Although reports to the Centers for Disease Control (CDC) show a sharp decline in silicosis from 1,100 deaths in 1968 to 255 deaths in 1992 [U.S. Department of Health and Human Services, 1999], large number of workers in many industries continue to be exposed to silica dust in the course of their work, frequently at levels exceeding the current OSHA permissible limit. Deaths from severe silicosis continue to be reported in workers under the age of 45 [Roberts and Castellano, 1997]; these are likely to be index cases, signifying the presence of other cases of varying severity in co-workers. This study was initiated to estimate the extent of previously undetected silicosis.

## METHODS

### Uncovering Cases Through Co-Morbidities

A diagnosis of silicosis is confirmed by chest radiography together with documented occupational exposure to crystalline silica dust. To test the hypothesis that silicosis has been overlooked by physicians, we identified potential cases of silicosis by selecting decedents who had worked in silica-exposed industries but whose cause of death was other than silicosis. Expert readers re-evaluated the chest X-rays of these potential silicotics for evidence of the rounded opacities characteristic of silicosis.

The New Jersey State Department of Health and Senior Services (NJDOHSS) has been conducting case-based silicosis surveillance since 1987 through SENSOR, a federally funded program [Baker, 1989]. Under SENSOR, New Jersey requires reports of new cases from hospitals and physicians. Each reported case is entered into a statewide silicosis registry for confirmation of the diagnosis through review of the chest X-ray and verification of appropriate work-related exposures. For each confirmed case, NJDOHSS identifies the industry where exposure occurred and assigns the relevant U.S. Department of Labor standard industrial classification (SIC) code.

Industrial codes found in the New Jersey silicosis registry were selected for use in this study. COPD, tuberculosis, and cor pulmonale were selected as diagnoses of interest, which met the criteria of being associated with silicosis and the likelihood of having chest radiographs available in the hospital files.

### Death Certificates

Death certificates provide a convenient cross-match of diagnosis and usual industry. As of 1990, New Jersey was one of 25 states to code occupation and usual industry on death certificates and report to the National Center for Health Statistics (NCHS). When a death occurs in a hospital, the death certificate includes the name of the hospital, which is a potential source of chest X-rays plus admission and discharge diagnoses.

### Study Population

Any individual who died as an inpatient in a New Jersey hospital during the 3-year period 1991–1993 was eligible for inclusion in the study if three additional criteria were met: (a) an underlying cause of death of either tuberculosis, cor pulmonale, chronic bronchitis, emphysema, or chronic airways obstruction; (b) a readable chest X-ray; and (c) usual industry on the death certificate in an SIC found in the NJ silicosis registry: mining, not coal SIC (14) construction (15,16,17) plastics, soaps (28), glass, cement, structural clay, pottery, miscellaneous mineral/stone (32), blast furnaces, foundries, primary metals (33), or shipbuilding and repair (37).

### Preliminary List of Eligible Deaths

New Jersey state regulations (26:8–4) require that hospitals maintain records for a minimum of 10 years, and X-rays for 5 years. We utilized records for the entire 3-year period 1991–1993, during which time there were a total of 732 New Jersey deaths meeting these criteria. The NJDOHSS generated a list of these death certificates by death certificate number, year of death, cause of death, and usual (longest) industry. Because of the need to limit the size of the study to 300 observations, the maximum number of chest X-rays that could be processed by expert readers, it was necessary to use a sample of the data.

### Sampling Strategy

Of the 732 deaths, 455 (62%) occurred in the construction industry, but the proportion of construction workers in the New Jersey registry between 1979 and 1990 was only 7%. Therefore, in order to keep the construction industry more in balance with the other industries, to address the lower potential for exposure, and to keep the number of cases under 300 for X-ray reading purposes, a random sample of 100 construction cases was drawn, bringing the new total to 377 cases. The proportion of construction workers was lowered from 62 to 27%.

## Sending for and Processing Records

After sending for and reviewing the death certificates, 107 were found to be unusable in that death did not occur in a New Jersey hospital: 56 died at home, 11 died out of state, 11 were dead on arrival, and 29 died in nursing homes. This resulted in a total of 270 cases where data was potentially available from New Jersey hospitals. These death certificates were abstracted for age at death, name of hospital, underlying cause of death, usual industry, race, gender, and whether there was any mention of silicosis on the death certificate as a contributing cause of death.

After abstracting the death certificates, letters were sent to each hospital requesting admission sheets, discharge summaries, chest radiographs, and postero-anterior and lateral view radiology reports. A log was maintained of each X-ray received from the hospitals. Upon receipt, X-rays were examined to confirm the identity of the patient and to eliminate micro-films, films, other than chest X-rays, or blank or severely over-exposed or under-exposed films. At the request of the readers, multiple X-rays were sorted and placed in chronological order. Chest X-rays were batched at the NJDOHSS and then forwarded to the Division of Respiratory Disease Studies (DRDS) at NIOSH in Morgantown WV for evaluation by the reading team. For each case, X-rays were affixed with three blank copies of the NIOSH roentgenographic interpretation sheet (B-reader sheet) labeled with the death certificate number.

Hospital records were abstracted at NJDOSS for name of attending physician, smoking status of patient (current, former, never, and unknown), any mention of silicosis or fibrosis on the discharge summary, or the X-ray report. All information, including that from the death certificates, was entered onto a coding sheet and later onto a SAS data set for analysis.

Although the protocol called for each X-ray to be read independently by two "B" readers, the readers opted to read side-by-side concurrently, using a consensus approach. The X-ray readers each filled out a separate (B-reader) sheet with the results of their classification. Although readers were blinded to all descriptors about the subjects, including other diagnoses, usual occupation, age, race, and sex, they were told that 25% of subjects had been in the construction industry, where exposures to silica dust do not always exist. Both readers have extensive experience in interpreting films, and previously passed a NIOSH-administered proficiency examination in radiologic classification of films, which qualified them as "B" (expert) readers [Wagner et al., 1992]. Both the coding sheet for each subject and the B-reader sheets contained the unique death certificate number to allow linkage of the two.

## Statistical Analysis

Any decedent found to have evidence of silicosis with a profusion category of at least 1/0 was considered to be a missed diagnosis. Proportions with 95% confidence intervals (CIs) were calculated for subjects found to have a missed diagnosis.

## RESULTS

### Characteristics of the Study Population

Of the 270 subjects for whom hospital records and chest X-rays were requested, 93 were lacking either chest X-rays and/or hospital records. This left a total of 177 files with records adequate for interpretation. Table I shows the distribution of the study subjects by underlying cause of death and usual industry as shown on the death certificate. Table II shows distribution by cause of death, gender, age, race, and smoking status. This is primarily an older, white, male population. Smoking status was unknown for 122 (68.9%) cases. Of the 55 cases whose smoking status was known, 76.3% were documented as being either current or former smokers.

Construction workers excluded from the sample did not differ from the entire construction group with respect to original cause of death; similarly, those excluded because they died outside the hospital or had unusable records did not differ with respect to industry. The 93 lacking chest X-rays or hospital records did not differ in cause of death or industry from the 270 subjects for whom hospital records and chest X-rays were requested. (Data not shown.)

**TABLE I.** Distribution of Silicosis Cases by Industry and Underlying Cause of Death (n = 177), New Jersey

Industry	Underlying cause of death				Total (%)
	TB/CP <sup>a</sup>	CB <sup>a</sup>	ES <sup>a</sup>	CAO <sup>a</sup>	
Mining, not coal	0	0	2	3	5 (2.8)
Construction	9	2	6	32	49 (27.7)
Plastics/synthetics	2	0	2	7	11 (6.2)
Soaps/cosmetics	2	1	4	14	21 (11.9)
Glass	6	0	4	11	21 (11.9)
Cement, concrete, etc <sup>a</sup>	1	2	2	6	11 (6.2)
Blast furnaces	2	0	3	19	24 (13.6)
Foundries/primary metal	2	0	8	12	16 (9.0)
Shipbuilding	1	0	0	12	13 (7.3)
Total (%)	25 (14.1)	5 (2.8)	31 (17.5)	116 (65.5)	177 (100.0)

CP, cor pulmonale; CB, bronchitis; ES, emphysema; CAO, chronic airways obstruction.

<sup>a</sup>Includes structural clay, pottery, miscellaneous mineral and stone.

**TABLE II.** Distribution of Silicosis Cases (n = 177) by Cause of Death, Gender, Age, Race, and Smoking Status; New Jersey

	Frequency	Percent
Cause of death		
Tuberculosis	4	2.3
Cor pulmonale	21	11.9
Chronic bronchitis	4	2.3
Emphysema	32	18.1
Chronic airways obstruction	116	65.5
Gender		
Male	154	87.0
Female	23	13.0
Age		
50–64	21	11.9
65–74	57	32.2
75–84	67	37.8
85+	32	18.1
Race		
Black	10	5.6
White	167	94.4
Smoking status		
Current	22	12.4
Never	13	7.3
Former	20	11.3
Unknown	122	68.9

### Previously Unrecognized Silicosis and Asbestosis

As shown in Table III, 15 previously unrecognized cases of silicosis and 19 previously unrecognized cases of asbestosis were identified by the readers. Table IV shows the distribution of these cases by cause of death, gender, age, race, and cause of death, and Table V shows the distribution by industry. Among the ten African-American workers, no missed cases of silicosis were observed, although three (30%) showed evidence of asbestosis.

The severity of undetected silicosis is shown in Table VI, which classifies the stage of disease by profusion category, industry, and age group. Profusion category 2 denotes denser concentrations of opacities than in category 1, signifying a more advanced stage of silicosis. Six of the fifteen silicosis

**TABLE III.** Prevalence of Previously Unrecognized Cases of Silicosis and Asbestosis With 95% Confidence Intervals (CIs)

Missed cases	Frequency	Percent	95% CI
Silicosis	15	8.5	4.8–13.6
Asbestosis	19	10.7	6.6–16.3
Total	34	19.2	13.7–25.8

**TABLE IV.** Distribution of Cases With Evidence of Silicosis and Asbestosis by Gender, Age, Race, and Cause of Death

Category	Re-evaluation results		
	N	Silicosis (%)	Asbestosis (%)
Gender			
Male	154	14 (9.1)	18 (11.7)
Female	23	1 (4.3)	1 (4.3)
Age			
50–64	21	2 (9.5)	1 (4.8)
65–74	57	4 (7.0)	8 (14.0)
75–84	67	5 (7.5)	9 (13.4)
85+	32	4 (12.5)	1 (3.1)
Race			
White	165	15	16
Black	10	0	3
Cause of death			
TB	4	1 (25.0)	2 (50.0)
Cor pulmonale	21	1 (4.8)	1 (4.8)
Chronic bronchitis	4	0	1 (25.0)
Emphysema	32	1 (3.1)	3 (9.4)
Chronic air obstruction	116	12 (10.3)	12 (10.3)

cases fell into category 2. Progressive massive fibrosis (PMF), the most severe form of chronic silicosis, was detected in two construction workers and one shipbuilder: all three were white workers over the age of 65.

Of the fifteen cases of silicosis that were detected, the original radiology reports were available for thirteen. A comparison of the findings in the radiology reports with the B-reader findings are presented in Table VII. The B-readers

**TABLE V.** Distribution of Cases With Evidence of Silicosis and Asbestosis by Industry

Category	N	Silicosis n (%)	Asbestosis n (%)
Industry <sup>a</sup>			
Mines, not coal	5	2 (40.0)	0
Construction	49	3 (6.1)	6 (12.2)
Plastics/synthetics	11	1 (9.1)	1 (9.1)
Soaps/cosmetics	21	1 (4.8)	4 (19.0)
Glass	21	2 (9.5)	1 (4.8)
Cement	2	1 (50.0)	0
Non-metallic mineral	4	0	1 (25.0)
Blast furnaces	24	3 (12.5)	2 (8.3)
Iron & steel foundry	6	0	2 (33.3)
Primary metals	16	0	2 (12.5)
Shipbuilding	13	2 (15.4)	0

<sup>a</sup>No evidence of silicosis or asbestosis was found among the five workers in the structural clay and pottery industries.

**TABLE VI.** Distribution of Missed Silicosis Diagnoses Among Industry Groups and Age Groups by Disease Profusion Categories (n = 15)

	Profusion category	
	Category 1	Category 2
Industry group		
Mining, not coal	2	0
Construction	1	2 (both are PMF <sup>a</sup> )
Manufacturing		
Plastics/soaps	2	0
Glass/cement/stone	2	1
Metal industries	3	0
Shipbuilding	1	1 (PMF <sup>a</sup> )
Total	11	4
Age group		
50–64	2	0
65–74	1	3 (includes 2 PMF <sup>a</sup> )
75 and over	8	1 (PMF <sup>a</sup> )
Total	11	4

<sup>a</sup>PMF is the most advanced stage of chronic silicosis.

read these reports after completing the classifications, and found that in six cases the radiology report should have alerted the attending physician of the possibility of occupational exposures.

## DISCUSSION

The principal finding of this study was that undetected silicosis and asbestosis exist despite a case-based surveillance system. This study has been a first attempt to

systematically uncover previously unrecognized cases of silicosis in the U.S. Only one previous study was specifically designed to detect unrecognized silicosis. Seeking to identify silicosis in a population whose cause of death was other than silicosis [Murray et al., 1996] utilized autopsy data and work histories in their investigation of black South African gold miners whose cause of death was trauma; they found that the prevalence of silicosis among these decedents increased from 9.3% in 1975 to 12.8% in 1991.

Previous studies with other primary objectives have noted that silicosis has been overlooked by clinicians who sign death certificates. For example, in a mortality study of Vermont granite workers, investigators suggested that misdiagnosis may have occurred in 10 of 25 decedents who had evidence of silicosis in their chest X-ray records but had no mention of silicosis on the death certificate [Davis et al., 1983]. In a study of sandblasters in New Orleans, the death certificates were reviewed for eight of the eleven confirmed silicotics who died after entering the study; only three of eight decedents had any mention of silicosis on the death certificate [Hughes, 1982].

Since there is considerable overlap in the industries where exposures to asbestos and crystalline silica occur, it is reasonable that undetected asbestosis would be observed as well as undetected silicosis.

It has been estimated that almost 500,000 workers in New Jersey were exposed to asbestos in the 45 years since the start of World War II [Stanbury and Rosenman, 1987]. New Jersey ranked third in the nation in number of deaths from asbestosis between 1987 and 1996, and is one of the five leading states in crude mortality rates from asbestosis in this same time period [U.S. Department of Health and Human Services, 1999]. Surveillance of work-related

**TABLE VII.** Comparison of B-Reader X-Ray Reports With the Original Radiology Reports for 13 of 15 Cases With Evidence of Silicosis

Case <sup>a</sup>	Original radiology report comments	B-reader report <sup>c</sup>
1	No infiltrate; single frontal projection; evidence of COPD	1/1; QT; 123456
2	Tiny persistent left pleural effusion; minimal hazy density in right mid-lung; may be residual pulmonary edema	1/0; QQ; 1245
3	Left lower lobe pneumonitis	1/0; QT; 123456
4 <sup>b</sup>	Pulmonary interstitial lung disease	1/0; QT; 123456
5 <sup>b</sup>	Left lower lobe infiltrate; faint increase in upper lobes density may be early infiltrates	1/2; RT; 123456 "B"
6 <sup>b</sup>	Interstitial fibrosis and infiltrative change in both upper lobes	2/1; QT; 123456 "A"
7	Small rounded calcific density at right base; a calcified granuloma; probable fibrosis in left lower lobes	1/1; QT; 123456
8 <sup>b</sup>	Minimal infiltrate in right perihilar region and left lingula	1/0; QQ; 1245
9	Interstitial infiltrate in central lung zones; may be inflammatory or vascular etiology	1/1; QT; 123456
10	Infiltrates at right lung base	1/1; PS; 123456
11	Moderate cardiomegaly	1/0; QQ; 123456
12 <sup>b</sup>	Underlying interstitial thickening or fibrosis	1/0; QT; 123456
13 <sup>b</sup>	Diffuse bilateral infiltrates; atelectasis & consolidation in right upper lobe	2/2; RQ; 123456 "B"

<sup>a</sup>Radiology reports for two cases were missing.

<sup>b</sup>Starred cases denote radiology report comments that should have alerted the attending physician to consider occupational exposures.

<sup>c</sup>n/n is profusion; letters refer to size and shape of opacities, final numbers refer to lung zones where opacities appeared; "A," "B" refer to large opacities.



asbestos disease in New Jersey began in 1985 with mandatory reporting by hospitals; physicians have been required to report asbestosis since 1990. Smoking is considered to be the prime risk factor for both COPD and lung cancer, and smoking prevalence is high among silica-exposed populations. Smoking is rarely mentioned in exposure-response studies where the outcome is fibrosis; one investigator observed that smoking is not a contributory cause of silicosis but often aggravates symptoms [Landrigan, 1986]. Rice et al. [1986] found that smoking did not affect the results of their study of North Carolina dusty trades workers, but Finkelstein [1994] found that cigarette smoking was a risk factor for the diagnosis of silicosis in Ontario [Finkelstein, 1994]. A study of Michigan foundry workers found that among highly exposed workers the rate of silicosis was 12.2% in smokers, as compared with 4.4% in never smokers [Rosenman et al., 1996].

It is unfortunate that smoking status was unavailable in the hospital records of nearly 70% of decedents whose cause of death was a respiratory disease. Narratives frequently lacked smoking data, and admission sheet smoking boxes were seldom filled in. This is not surprising because hospital data is primarily recorded for billing purposes and not for research.

There were methodological limitations in this study. The purpose of the "B" reader program is to classify X-rays under the ILO scheme for the presence or absence of parenchymal or pleural lesions characteristic of pneumoconiosis. Classification of X-rays is an epidemiological exercise rather than a clinical one, and diagnosis can be made only in combination of the classification with confirmed exposure to airborne silica dust.

Although the X-rays in this study were classified according to the [International Labour Office, 1980] classification scheme, some requirements were relaxed. For instance, the protocol for the ILO scheme specifies the exact exposure view required. The required view is the postero-anterior (PA-lateral) meaning the X-ray is taken from the back and the side while the subject is standing with full inspiration. Because these patients were end-stage, in many cases the X-rays were administered from the front (anter-postero) with the subject either standing or sitting. The X-rays of this population are subject to the "noise" of end-stage disease. To assure that a sufficient number of cases were available for analysis, the film quality standard was also relaxed, in that only unreadable (category 4) were excluded from the survey. Film quality can be a factor in the classification of radiographs for asbestosis, but this is less of a problem with experienced readers [Parker, 1997].

The consensus approach in reading was adopted because the majority of case files included multiple X-rays, which required identification of the one highest quality film. Readers sat side by side and evaluated each X-ray using the standard reference set of X-rays called for in the ILO

guidelines. A limitation of the consensus approach is that the stronger personality may dominate the discussion and the decision process. Both readers in this study, however, have extensive experience and expertise in interpreting films and it is not likely that any bias occurred from the consensus approach.

Many factors contribute to the interpretation of X-rays, including the size of the patient, the film quality, the experience of the reader, and observer bias of the ILO reader knowing that dust exposure is involved [Epler et al., 1978]. An early study found that physicians differ from each other one third of the time in reading X-ray films [Yerushalmy et al., 1951].

It is possible that these readers have underestimated the extent of silicosis because X-ray classification is limited in sensitivity. Examples appear in the literature of pulmonary impairment and pathological conformation of silicosis in the absence of radiological evidence. In an autopsy-based study comparing pathology to radiological findings for silicosis, the sensitivity values of the three X-ray readers were 0.393, 0.371, and 0.236, respectively; each reader found less than 40% of the cases confirmed by pathology [Hnizdo et al., 1993]. Similarly, an autopsy study of Vermont granite workers found pathological evidence of silicosis in the absence of radiological evidence [Craighead and Vallyathan, 1980]. A review of the literature on asbestos diagnoses found that autopsy series indicated a 40% sensitivity and 80% specificity rate for chest X-rays [Geffer and Conant, 1988].

On the other hand, there is the potential for "over-reading" of X-rays. Geffer and Conant [1988] reviewed chest X-rays on hospitalized patients without known industrial exposure; evidence of small opacities at a profusion level of 1/0, or greater in 18% of subjects [Geffer et al., 1984]. Of the positive cases, 60% had no previous dust exposure or medical etiology to explain the opacities. [Epstein et al., 1984] evaluated the chest radiographs of 200 patients entering a large urban hospital according to the 1980 ILO scheme and found that 22 (11%) without known industrial exposure had small opacities consistent with pneumoconiosis.

Although death certificates have been shown to be useful in occupational health surveillance, they can be potential sources of error. Errors in the identification of cause of death [Carter, 1985; Mancuso, 1993] as well as errors in the coding of occupation and industry [Steenland and Beaumont, 1984; McLaughlin and Mehl, 1991] are potential limitations. There was, however, 100% agreement in this study between underlying cause of death and discharge diagnosis on the hospital record. The lack of quantitative exposure data for the subjects in this study is another limitation.

This is a small study conducted in one state during the early 1990s and it would benefit from updating. It bears repeating that silica exposure occurs in a myriad of occupational settings, and that there is great variability among states in their industrial bases. New Jersey's range of industry

is not necessarily representative of that of the U.S. as a whole. Mining, for instance, with the exception of sand mining, is non-existent in New Jersey but is a great source of potential exposure for silicosis. Other diagnoses at death such as trauma might have captured a younger population but the probability of a chest X-ray in the hospital record would have been considerably lower. It is possible that rates of missed diagnoses found in New Jersey may understate the national rates because of the fact that all New Jersey physicians received a fact sheet in 1985 from the Health Department about the recognition and diagnosis of silicosis [Valiante and Rosenman, 1989]. Physicians from other states without the benefit of this notice may have missed even a higher proportion of cases.

Physicians play the pre-eminent role in case-based disease surveillance by virtue of their unique role in recognizing and diagnosing cases but most primary care physicians do not take occupational histories [Goldman and Peters, 1981]. Although 85% of primary care physicians treat patients in the workforce [Campbell and Nicolle, 1981], most receive minimal formal training in the recognition of occupational disease in medical school [Institute of Medicine, 1988].

The under-recognition of occupational disease by physicians may be attributable in part to factors beyond the scope of this study. It must be remembered that many of these cases of pneumoconiosis developed years or even decades ago, prior to the development of co-morbidities. The presence of silicosis in younger patients without complications would have been easier to recognize by the physicians who previously cared for them but are unknown to this study. It would be inappropriate; therefore, to attribute all responsibility for these missed diagnoses in patients with end-stage respiratory disease to the current physicians. It is not possible to tell what proportion of the cases retained the same provider for decades, but is it likely that providers changed for many subjects.

While the prevalence of silicosis has lowered, especially in the granite industry [Graham et al., 1991], this disease is by no means disappearing or becoming a historical curiosity [Markowitz and Rosner, 1998]. Large number of workers continue to be exposed to silica, at levels frequently exceeding the permissible exposure limit. Among active and retired Colorado hard-rock miners, investigators found a 50% risk for silicosis in miners working 45 years at an average exposure level of half the current OSHA standard [Kreiss and Zhen, 1996]. In a study of South Dakota gold miners, the adjusted lifetime risk for silicosis was estimated to be 35% [Steenland and Brown, 1995].

In conclusion, this study suggests that occupational lung disease may be undercounted in high-risk occupations. The promulgation by OSHA of a comprehensive standard for crystalline silica would include ongoing exposure monitoring and medical surveillance, which would significantly improve the recognition of cases and justify more stringent

preventive measures to reduce exposure. Current practitioners as well as medical students need skills in taking an occupational exposure history.

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