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Mortality among persons with silicosis reported to disease surveillance systems in Michigan and New Jersey in the United States

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Michigan and New Jersey in the United States maintain silicosis disease registers. In 1988—1992, 372 cases of silicosis were confirmed in Michigan, and, in 1979—1992, 288 were confirmed in New Jersey. A proportionate mortality ratio (PMR) analysis was performed on data from 292 deceased silicotics. Increases in PMR values were found for nonmalignant respiratory disease (NMRD) and lung cancer. The PMR values for NMRD were statistically elevated in all the analyses. The overall proportionate cancer mortality ratio (PCMR) for lung cancer was 1.78 [95% confidence interval (95% CI) 1.22—2.61]. For patients having ever smoked cigarettes, the PCMR for lung cancer was 1.82 (95% CI 1.18—2.81). Never smoking silicotics had a lung cancer PCMR of 1.48 (95% CI 0.43—2.86). For those who had never applied for workers' compensation the corresponding PCMR was higher, 2.10 (95% CI 1.21—3.69), than for those who had applied, 1.45 (95% CI 0.70—2.99).

Key terms cigarettes, lung cancer, obstructive lung disease, silica, workers' compensation.

Repeated epidemiologic studies among patients with silicosis have found an increased risk of death from lung cancer (1). The hypotheses for this association are (i) silica is a carcinogen; (ii) there is an effect from concomitant exposures to other carcinogens (eg, polycyclic aromatic hydrocarbons in foundries); (iii) there is an increased prevalence of cigarette smoking among workers with silicosis; (iv) there is selection bias from studying workers who applied for workers' compensation; (v) there is an effect secondary to fibrosis not specific to silica. The data used in this analysis are from two state-wide silicosis registries. Diagnoses were confirmed and sufficient information was collected on the reported cases to examine the effect of cigarette smoking and concomitant occupational exposures. Because reports are mainly received from hospitals and private practitioners, we have also been able to examine whether patients who apply for workers' compensation have the same risk of lung cancer as workers who do not apply.

Subjects and methods

Michigan and New Jersey maintain silicosis disease registers. Michigan's silicosis register requires hospitals, clinics, employers, and physicians to report all known or suspected occupational diseases. New Jersey's silicosis register became mandatory for hospitals in 1985, and for physicians in 1990.

In both states patients were interviewed and their medical records were reviewed. "B readers" certified by the National Institute for Occupational Safety and Health (NIOSH) evaluated all the chest films to confirm the diagnoses. Confirmed patients were those with a history of silica exposure and either a positive lung biopsy or 1/0 or greater nodular opacities in radiographs and absence of medical conditions which can produce similar X-ray findings. The patients were interviewed by telephone using a standardized questionnaire including a complete work history, smoking history, and whether patients ever applied for workers' compensation. If subjects were deceased, then the next-of-kin was interviewed. From 1988 to 1992, 372 cases of silicosis were confirmed in Michigan, and, from 1979 to 1992, 288 cases of silicosis were confirmed in New Jersey.

The data from both states were combined. Silica exposure industries were coded according to the Standardized Industrial Classification System (2). Vital status was determined through a review of hospital and physician records, including death certificates. Furthermore, to confirm the vital status of all cases in Michigan, all confirmed silicosis cases were matched against the National Death Index data base. In New Jersey, all confirmed cases were matched against listings of all New Jersey deaths.

Proportionate mortality and proportionate cancer mortality ratios (PMR and PCMR, respectively) were calculated with the O/E program of the National Cancer Institute, a modified version of a published program (3). The analysis was limited to white and black men, and age- and time-adjusted mortality ratios with 95% confidence intervals (95% CI) were calculated. Expected ratios of cause of death were derived from male population rates of the United States (3). In addition, smoking-specific expected ratios of cause of death for lung cancer and obstructive lung disease were derived from the CPSII survey of the American Cancer Society (Thun, personal communication).

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Because some patients had missing data, the number of deaths varied in each analysis.

Results

Of the 660 confirmed silicosis patients 292 had died and the PMR was statistically increased for all nonmalignant respiratory disease (NMRD) and statistically decreased for all cancer and all circulatory disease (table 1). Not surprisingly, silicosis was the most common cause of respiratory mortality with 55 deaths. There were 42 deaths from emphysema. Table 1 shows that the PCMR for lung cancer was significantly increased, 1.78 (95% CI 1.22—2.61), while digestive cancer was significantly decreased, 0.25 (95% CI 0.09—0.74).

Tables 2—4 show the PCMR values for lung cancer and PMR values for all respiratory disease by duration of employment, smoking status, and profusion category from chest X rays.

The PMR values for all respiratory disease and lung cancer were greater for ≥ 30 years than for < 10 years of exposure. However, there was no trend of increasing duration of exposure for either lung cancer or respiratory disease (table 2).

As seen in table 3 there was no monotonic trend of increasing PMR or PCMR values for lung cancer with increasing profusion of small rounded opacities or for the presence of large opacities. Although there was no trend by profusion category, the PMR values for respiratory disease were statistically elevated for all the profusion categories.

With the American Cancer Society's expected risks for never and current smokers as the comparison, the PCMR was 3.94 (95% CI 1.53—10.12) for never smoking silicotics and 1.50 (95% CI 0.82—2.76) for current smokers. The PMR values for chronic lung disease showed similar risk levels (table 4).

The PCMR was 1.45 (95% CI 0.70—2.99) for those who had applied for workers' compensation when compared with 2.11 (95% CI 1.21—3.9) for those who had not sought compensation. The risks for respiratory disease were statistically increased whether or not the patients had applied for workers' compensation. The risks for lung cancer were statistically elevated for foundry work-

Table 1. Race adjusted proportionate mortality ratios (PMR) and proportionate cancer mortality ratios (PCMR) for male silicotics — Michigan 1988—1992 and New Jersey 1979—1992. (95% CI = 95% confidence interval)

Cause of death	Number of deaths	Ratio[b]	95% CI
All causes	292	..	..
Infectious disease	6	1.54	0.71—3.37
Tuberculosis	1	2.28	0.40—12.93
All cancer	46	0.67*	0.50—0.89
Cancer of digestive organs[c]	3	0.25*	0.09—0.74
Cancer of lung[c]	26	1.78	1.22—2.61
Cancer of prostate[c]	7	0.99	0.48—2.04
All circulatory disease	80	0.54*	0.44—0.68
All respiratory disease	134	4.87*	4.12—5.76
Pneumonia	14	1.24	0.74—2.08
Emphysema	6	2.23*	1.02—4.86
All external causes	5	0.51	0.22—1.19

[b] Expected based on mortality ratios for the general male population of the United States.
[c] PCMR.
* P < 0.05.

Table 2. Proportionate mortality ratios (PMR) and proportionate cancer mortality ratios (PCMR) for male silicotics by duration of years worked — Michigan 1988—1992 and New Jersey 1979—1992. (95% CI = 95% confidence interval)

Cause of death	< 10 years			10—19 years			20—29 years			≥ 30 years		
	Number of deaths	Ratio[a]	95% CI	Number of deaths	Ratio[a]	95% CI	Number of deaths	Ratio[a]	95% CI	Number of deaths	Ratio[a]	95% CI
Cancer of lung (PCMR)	2	1.54	0.42—5.61	4	1.63	0.63—4.20	7	1.48	0.71—3.05	10	1.73	0.94—3.19
All respiratory disease (PMR)	8	6.03*	3.05—11.91	20	5.99*	2.89—8.88	35	4.86*	3.50—6.77	56	4.43*	3.41—5.75

[a] Expected based on ratios for the general male population of the United States.
* P < 0.05.

Table 3. Proportionate mortality ratios (PMR) and proportionate cancer mortality ratios (PCMR) for male silicotics by profusion category for pneumoniocosis — Michigan 1988—1992 and New Jersey 1979—1992. (95% CI = 95% confidence interval, PMF = progressive massive fibrosis)

Cause of death	Category 1[b]			Category 2[c]			Category 3[d]			PMF		
	Number of deaths	Ratio[a]	95% CI	Number of deaths	Ratio[a]	95% CI	Number of deaths	Ratio[a]	95% CI	Number of deaths	Ratio[a]	95% CI
Cancer of lung (PCMR)	8	1.70	0.86—3.34	5	1.42	0.60—3.32	2	2.16	0.59—7.88	7	1.85	0.90—3.82
All respiratory disease (PMR)	23	4.43*	2.46—5.54	25	3.92*	2.65—5.78	12	6.23*	3.56—10.89	54	5.76*	4.41—7.51

[a] Expected based on mortality ratios for the general male population of the United States.
[b] Category 1 = 1/0, 1/1, 1/2 International Labour Office 1980.
[c] Category 2 = 2/1, 2/2, 2/3 International Labour Office 1980.
[d] Category 3 = 3/2, 3/3, 3/+ International Labour Office 1980.
* P < 0.05.

Table 4. Proportionate mortality ratios (PMR) and proportionate cancer mortality ratios (PCMR) for male silicotics by smoking status — Michigan 1988—1992 and New Jersey 1979—1992. (95% CI = 95% confidence interval)

Cause of death	Smoking status					
	Never			Current		
	Number of deaths	Ratio[a]	95% CI	Number of deaths	Ratio[a]	95% CI
Cancer of lung (PCMR)	4	3.94*	1.53—10.12	10	1.50	0.82—2.76
Obstructive lung disease, white males only (PMR)	3	5.97*	1.20—17.45	7	3.58*	1.44—7.39

[a] Expected based on the CPS-II of the American Cancer Society.
* $P < 0.05$.

ers (PCMR 1.65) and for workers in all other industries (PCMR 2.15). The PMR values for respiratory disease were statistically increased regardless of type of industry (tables not presented).

Discussion

Similar to other epidemiologic studies of patients from silicosis registers [reviewed by Goldsmith (1)], this analysis showed increased mortality risk from lung cancer and respiratory disease. The PCMR for lung cancer was 1.78 (95% CI 1.22—2.61) and the PMR for respiratory disease was 4.87 (95% CI 4.12—5.76) (table 1). These respiratory deaths were predominantly due to silicosis and chronic obstructive lung disease.

In contrast to the subjects of other studies based on silicosis registers (4—10), most of the patients in this analysis had been reported to the registers by health care providers and not because they had applied for workers' compensation. Only 24% of this cohort was known to have applied for workers' compensation. In addition, information on cigarette smoking habits, years worked, and severity of disease (as determined by X-ray findings) was available for a large percentage of the cohort. Although the number of individuals who never smoked was small, the lung cancer PCMR for persons who never smoked was 3.94 (table 4). Four never smoking silicotics died of lung cancer. Using smoking-specific reference data of the American Cancer Society to generate expected numbers of deaths produced a PCMR of 1.50 for ever smokers and a PCMR of 3.94 (95% CI 1.53—10.12) for never smokers. Our data do not support the argument that an increased prevalence of smokers among workers with silicosis explains the increased risk of lung cancer.

The respiratory disease PMR values were statistically increased for all the smoking categories. These results are consistent with those of studies that show an association between obstructive lung disease and silicosis (11, 12).

Comparison of the PCMR values of persons who did not apply for workers' compensation with those who did showed an increased PCMR for those who did not apply. Thus our study does not support the position that bias towards lung cancer exists among compensation applicants.

We found no trend of increasing lung cancer risk among persons with greater scarring on their X ray (table 3). Our data therefore do not support the hypothesis that the mechanism of increased lung cancer arises from pulmonary scar tissue.

We found no monotonic trend of increasing risk for lung cancer and all respiratory disease by duration silica exposure (table 2). The lack of an association with duration of exposure is inconsistent with the hypothesis that silica is a direct carcinogen. The statistical increase among men who began work before 1949 suggested that high historical exposures and sufficient latency from first exposure are associated with lung cancer among silicotics. This finding would support an effect of silica or some other workplace exposure.

The lung cancer risks for foundry work and "other" industries were statistically increased. Foundry exposures to potential carcinogens such as polycyclic aromatic hydrocarbons may account for this finding (13). There was a wide variety of industries in the "other" category, including surface and underground mining, sandblasting and sandpaper manufacture. Our data do not include actual dust exposure information that would help to determine whether silica dust was the specific factor causing the lung cancer excess.

There are numerous limitations in our study, for example, the small number of deaths and a lack of complete data on each patient.

Information on smoking, workers' compensation, and duration and decade of exposure was obtained from the patient or next-of-kin. To verify smoking status, we reviewed the medical records of the 11 individuals classified as never smokers who died of lung cancer or chronic obstructive lung disease. Five of the medical records confirmed that the patient was a nonsmoker, five did not mention the patient's smoking status, and one had no available clinical record. Three subjects who died of NMRD had smoked one to two cigars a day, and one who chewed tobacco died of lung cancer. Smoking information was collected after the patient left work, and neither the patient's nor the next-of-kin's response was likely to be influenced by a pending workers' compensation proceeding. We are unaware of any reason why there should be any systematic bias in any of the data collected.

Limitations of proportionate mortality analyses are well known. Unlike typical worker cohorts, this group probably had an increased standardized mortality ratio. Most patients' silicosis was diagnosed in the hospital. Thus increased lung cancer findings suggest that the PMR values are underestimates of risk. Most of the patients in this cohort had advanced silicosis, including 39.5% with progressive massive fibrosis. Our results may not be generalizable to the total population of patients with silicosis because some patients hospitalized for lung cancer or NMRD were then diagnosed as having silicosis. Among the 26 patients who died from lung cancer, medical records indicated that 17 had silicosis at least one year. For four patients, the diagnosis was made at the same time, and for the remaining five there was insufficient information to determine when the silicosis diagnosis was made. Another study limitation is that there is a marked deficit in heart disease and all cancer; this finding is reflected in the PMR analysis by the increases in other causes of death. However, the two causes of increased mortality, lung cancer and respiratory disease, are the conditions of interest.

In summary, this study supports the association between lung cancer and silicosis. Our data suggest this finding cannot be explained by smoking, by profusion scores, or by bias from studying patients who had sought workers' compensation. We cannot rule out the importance of concomitant carcinogens in workplaces that use silica. Five other states in the United States now have silicosis registers similar to those in Michigan and New Jersey. In the future, we hope to readdress this issue using silicosis patients from these states.

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