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Respiratory Cancer and Other Chronic Disease Mortality Among Silicotics in California

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Silicotics have increased mortality from tuberculosis (TB) and from nonmalignant respiratory diseases (NMRD), including silicosis and silicotuberculosis. Since the publication of the International Agency for Research on Cancer monograph in 1987 indicating that silica was a probable human carcinogen, there has been an extensive debate about the cancer risks among silicotics. The authors identified 590 claims for silicosis among a registry of lung diseases compiled from California Workers' Compensation cases from 1945 to 1975. Using state vital records, we determined the mortality risks from 1946 to 1991. Our findings confirmed that these claimants had a significantly elevated risk for all causes of death with a standardized mortality ratio (SMR) of 1.30 (95% confidence interval [CI] = 1.18, 1.43); TB had a SMR of 56.35 (95% CI = 41.10, 75.40) and NMRD a SMR of 3.80 (95% CI = 3.11, 4.60). Cancers of the trachea, bronchus, and lung had a SMR of 1.90 (95% CI = 1.35, 2.60). For malignancies of the large intestine, there was a previously unreported SMR of 2.08 (95% CI = 1.14, 3.50). Mortality from all diseases of the heart was significantly less than expected with a SMR of 0.68 (95% CI = 0.55, 0.83); cancers of the prostate and lymphatic system were also significantly low with SMRs of 0.26 (95% CI = 0.03, 0.94) and 0.17 (95% CI = 0.04, 0.97), respectively. Workers with silicosis should be warned about these chronic disease risks, and prevention efforts to control occupational silica dust exposure should become a higher priority.

Key words: silicotics, tuberculosis, nonmalignant respiratory disease, pulmonary cancer, cancer of the large intestine, Workers' Compensation, prevention

INTRODUCTION

Since Biblical times there has been awareness that workers who mine and smelt metal ores or who work in the "dusty trades" suffer from fatal and debilitating pulmonary diseases including silicosis, silicotuberculosis, and other respiratory ailments [Raffle et al., 1987; Goldsmith, 1994a]. Initial concern about silica's carcinogencity arose in the 1930s when a Liverpool, England pathologist published a post-

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mortem review of 14 cases of silicosis in which he found 4 patients with cancer [Dible, 1934]. This finding produced extensive debate in the occupational medicine community about whether silica exposure was carcinogenic or whether patients with silicosis had increased risks for cancers of the respiratory system and other tumors [Goldsmith et al., 1982; 1986]. The world's cumulative literature up to 1986 was summarized by the International Agency for Research on Cancer [IARC], and updated recently in reviews by Simonato et al. [1990], Pairon et al. [1991], and Goldsmith [1994a,b]. IARC concluded that the evidence for pulmonary carcinogenicity in laboratory animals was sufficient and for humans, it was limited; and thus crystalline silica was judged to be a probable human carcinogen. The IARC review panel noted that studies of silicotics suffered from several possible research flaws including diagnostic bias, a lack of adjustment for smoking, and concerns about medico-legal definitions of silicosis [IARC, 1987]. This research seeks to address some of these issues directly.

In this current study, we selected patients from a registry of occupational pulmonary diseases (developed by the authors) who filed a claim for silicosis with the California Workers' Compensation Appeals Board. Our purpose was to test the hypothesis that silicosis claimants have elevated mortality risks for silica-related pulmonary diseases, including silicosis, tuberculosis (TB), and other nonmalignant lung diseases, and cancers of the respiratory tract, digestive system, and lymphopoietic system.

MATERIALS AND METHODS

Detailed procedures have been described for the methodology used to assess the mortality risks from California Workers' Compensation claims [Beaumont et al., 1995]. Briefly, from a registry of 1,918 Workers' Compensation Appeals Board claimants for occupational respiratory diseases [Goldsmith et al., 1994], we identified all 590 claims for silicosis from January 1, 1946 to December 31, 1975, and followed their mortality to January 31, 1991 using vital records linkage. Claims listing TB, emphysema, pneumonia, or cancer were excluded from the registry. Each subject who filed for silicosis had a physician's report which we extracted from the registry. When the insurance carrier included a second medical exam of the subject in the file, we also entered the diagnosis from that physician. Chest films were not stored in the Workers' Compensation files, and thus were not available for the claimants in this study. Motor vehicle records were used to trace study subjects and determine vital status. When subjects were found to have moved from California, we queried those states to determine whether subjects died, and if deceased, we obtained copies of death certificates. From the claim files, we recorded the following information: date of birth, date of claim for silicosis, Social Security number, industry, amount of the disability award, and smoking status when it was included in the physicians' reports. From death certificates, we abstracted race, date of death, and underlying cause of death code.

The Occupational Cohort Mortality Analysis Program developed by Marsh and co-workers [1986] was used to calculate standardized mortality ratios (SMRs) and 95% confidence intervals (95% CIs). Mortality rates were standardized for age, year, and race and compared to U.S. male rates assuming a Poisson distribution. We examined causes of death of interest by industry, by amount of compensation

TABLE I. Characteristics of Silicotics Filing Workers' Compensation Appeals Claims From 1946 to 1975 in California

No. of study subjects	590
No. and percent male	585 (99%)
Year of birth (median)	1906 (±13)
Age when claim filed (median)	57 (±10) years
Age at death (median)	68 (±9) years
No. and percent deceased	421 (71%)
Proportion deceased nonwhite	4%
Compensation awarded (median)	$2,024 (±4,936)

awarded by the claims hearing officer, by time since claim filed, and by smoking status. Because smoking information was not available for all study subjects, a common adjustment process developed by Dr. Olaf Axelson was used to assess the lung cancer risk explained by smoking [Axelson, 1978].

RESULTS

Table I provides descriptive information on the 590 silicosis claimants, of whom 585 were males. On average, these men were born in 1906 (±13), were 57 (±10) years old when they filed their claim, and died at age 68 (±9 years). There were 4% of the deaths among nonwhites, and 71% of the claimants are confirmed deceased by California or other states' death records. The amount of compensation awarded ranged from zero to $33,564, and the median level was $2,024 (unadjusted for inflation).

Tables II and III list SMRs and 95% CIs for selected noncancer causes of death and for malignant neoplasms. Mortality from TB had a SMR of 56.34 (95% CI = 41.10, 75.40) and mortality from all nonmalignant respiratory disease (NMRD) produced a SMR of 3.80 (95% CI = 3.11, 4.60), confirming in these claimants the well-known association between silicosis and these two causes of death. Deaths from emphysema were also elevated with a SMR of 3.41 (95% CI = 2.02, 5.39), and there was a SMR of 6.81 (95% CI = 5.28, 8.63) for deaths from silicosis and other nonmalignant pulmonary diseases. In this cohort there were significantly low SMRs of 0.58 (95% CI = 0.33, 0.95) for cerebrovascular diseases, 0.68 (95% CI = 0.55, 0.83) for all heart diseases, and 0.66 (95% CI = 0.53, 0.83) for ischemic heart disease. There was significant excess mortality for cirrhosis of the liver, SMR = 2.58 (95% CI = 1.29, 4.61). There was a doubling of the risks for motor vehicle accidents and for suicides, SMRs = 2.03 and 2.01, respectively, though the lower 95% CI was <1.00 for both causes. All cause mortality showed a significantly elevated risk with a SMR of 1.30 (421 observed deaths, 324.9 expected), and this was attributable to the elevated rates of death from TB and NMRD.

Table III displays selected findings for malignant neoplasms. There were elevated SMRs of 1.22 and 1.24 for all malignant neoplasms and for cancer of the digestive organs and peritoneum. However, there was a statistically significant SMR of 2.08 for cancer of the large intestine (95% CI = 1.14, 3.50). Cancers of the respiratory system and trachea, bronchus, and lung both showed a statistically significant doubling of risk with SMRs of 1.99 and 1.90, respectively. There was an

TABLE II. Standardized Mortality Ratios (SMR) and 95% Confidence Intervals (95% CIs) for Selected Noncancer Causes of Death Among 590 Silicotics Filing Workers' Compensation Claims in California, 1946–1975

Cause of death	Observed	Expected	SMR	95%CIs
Tuberculosis (010–018)[a]	45	0.8	56.35	41.10,75.40
Cerebrovascular disease (430–438)	16	27.4	0.58	0.33,0.95
All heart disease (390–398, 402, 404, 410–429)	97	142.9	0.68	0.55,0.83
Ischemic heart disease (410–414)	80	120.3	0.66	0.53,0.83
Nonmalignant respiratory disease (460–519)	105	27.6	3.80	3.11,4.60
Emphysema (492)	18	5.3	3.41	2.02,5.39
Silicosis/other nonmalignant respiratory disease (460–466, 470–478, 494–496, 500–519)	68	10.0	6.81	5.28,8.63
Cirrhosis of liver (571)	11	4.3	2.58	1.29,4.61
Motor vehicle accidents (E810–825)	6	2.9	2.03	0.75,4.43
Suicides (E950–959)	6	3.0	2.01	0.73,4.37
All causes (001–999)	421	324.9	1.30	1.18,1.43

[a]International Classification of Diseases Codes, 9th Revision.

TABLE III. Standardized Mortality Ratios (SMR) and 95% Confidence Intervals (95% CIs) for Selected Malignant Neoplasms Among 590 Silicotics Filing Workers' Compensation Claims in California, 1946–1975

Causes of death	Observed	Expected	SMR	95%CIs
All malignant neoplasms (MN) (140–208)[a]	81	66.3	1.22	0.96,1.52
MN of digestive organs (150–159)	23	18.5	1.24	0.79,1.86
MN of large intestine (153)	14	6.7	2.08	1.14,3.50
MN respiratory system (160–165)[b]	43	21.6	1.99	1.44,2.68
MN of the trachea, bronchus, and lung (162)	39	20.5	1.90	1.35,2.60
MN of prostate (185)	2	7.7	0.26	0.03,0.94
MN of lymphatic system (200–208)	1	5.7	0.17	0.04,0.97
All other MN (171, 173, 195–199)	5	4.7	1.06	0.34,2.46

[a]International Classification of Disease Codes, 9th Revision.
[b]Includes three deaths from laryngeal cancer and one death from pulmonary mesothelioma.

elevated SMR of 3.48 for cancer of the larynx (not shown), but the risk was based on small numbers (three deaths; 0.86 expected; 95% CI = 0.72, 10.18). There were either no deaths or no statistically significant risks for smoking-related cancers of the pancreas, bladder, and kidney (data not shown). There were statistically significant deficits for cancers of the prostate gland with a SMR = 0.26 (95% CI = 0.03, 0.94), and for lymphatic and hematopoietic tissues with a SMR = 0.17 (95% CI = 0.04, 0.97).

The remaining analyses will be limited to TB, all NMRD, and cancers of the large intestine and trachea, bronchus, and lung. Table IV compares the SMRs for four industrial groups from which the silicosis cases arose: construction, mining and quarrying, metallurgy/foundries, and utility/transportation/miscellaneous industries. For silicosis claimants, the SMRs for TB and NMRD were elevated for all industry groups, with utility/transportation industries showing a SMR of 99.07 (based on four deaths). Silicotic claimants from construction had the greatest risks for neoplasms of the large intestine (SMR = 3.28) and lung (SMR = 4.04). Subjects from mining and quarrying industries had SMRs of 2.01 and 1.66 for cancers of the large intestine and

TABLE IV. Standardized Mortality Ratios and Observed Numbers of Deaths for Tuberculosis (TB), Nonmalignant Respiratory Disease (NMRD), and Cancers of the Large Intestine and Lung by Industry Among 590 Silicotics Filing Workers' Compensation Claims in California, 1946–1975

Industry	TB	NMRD	Large intestine	Lung
Construction	8.65 (1)[a]	2.36 (11)[b]	3.28 (4)	4.04 (17)[b]
Mining and quarrying	67.00 (34)[c]	3.82 (64)[c]	2.01 (8)	1.65 (19)
Metallurgy and foundries	44.41 (6)[c]	4.20 (21)[c]	0.86 (1)	0.57 (2)
Utilities/transportation/ miscellaneous industries	99.07 (4)[c]	7.31 (9)[c]	2.83 (1)	0.76 (1)

[a]Observed number of deaths.
[b]Significant at $p < 0.05$.
[c]Significant at $p < 0.01$.

lung. Silicotics from the metallurgy and foundry industry had a reduced mortality risk for cancers of the large intestine and lung. Claimants for silicosis arising from the utility/transportation/miscellaneous industries had a SMR of 2.83 for cancer of the large intestine and a risk of 0.76 for pulmonary cancer, although the last set of risks was based on small numbers of deaths. The only meothelioma death was in this industrial group.

Mortality risks were compared by dividing the cohort into three groups according to amount of compensation awarded: 150 workers received no award; 81 received <$1,000; and 358 received ≥$1,000 (one subject's amount was missing). As shown in Figure 1, the SMRs for TB, NMRD, and lung cancer rise as the amount of the award for silicosis increases. There is no gradient for cancer of the large intestine, but only at the ≥$1,000 stratum is the SMR = 2.31 statistically significant. Although we lacked the complete clinical and job exposure information available to California Workers' Compensation examiners, the amount of award is being treated in this analysis as a surrogate for degree of disability from silicosis. Any claimant who was awarded zero compensation must not have succeeded in winning his claim or it was withdrawn; similarly, a worker receiving ≥$1,000 must have demonstrated to those reviewing his case a convincing diagnosis of clinical silicosis. Between these two extremes lies a modest degree of compensation and fibrotic lung disease. Thus, increasing risk gradients by amount of award suggest a positive correlation between silicosis and the chronic disease mortality of interest.

Table V contains SMRs by interval between date of filing for compensation for silicosis and death. There was a mean interval of 4.2 years for deaths from TB with the SMR declining from 243.8 up to 1 year after filing to 9.4 for >20 years interval. For NMRD, there was a mean of 10 years from filing until death, and the SMR declined from 11.6 within 1 year of filing to 0.9 for >20 years after submitting a claim. The risks for cancer of the large intestine were elevated for all periods after filing, with a mean interval of 15.2 years between filing and death. Cancers of trachea, bronchus, and lung showed two peaks at 1–3 years and 5–10 years since filing with all interval periods (except >20 years) demonstrating elevated risks. The mean interval from filing to death for lung cancer was 9.9 years.

The physician reports in the Workers' Compensation files contained smoking information on only 70% of the 590 silicosis files. Of that group, 55% reported being current smokers, 33% were exsmokers, and 12% were nonsmokers. Because of the missing data and because of the lack of standardized information on smoking, we

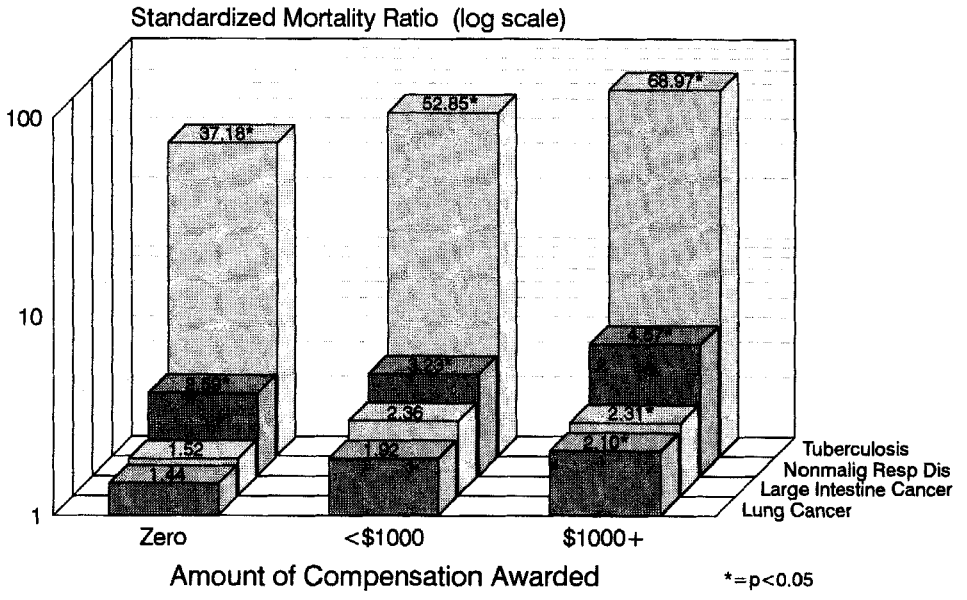

Fig. 1. Standardized mortality ratios (on a log scale) for tuberculosis, nonmalignant respiratory disease, and malignant neoplasms of the large intestine and lung according to the amount of compensation awarded. Workers' Compensation claims for silicosis in California, 1946–1975.

TABLE V. Standardized Mortality Ratios and Numbers of Deaths for Tuberculosis (TB), Nonmalignant Respiratory Disease (NMRD), and Cancers of the Large Intestine and Lung Among 590 Silicotics Filing Workers' Compensation Claims in California, 1946–1975, According to Follow-Up Intervals From Claim to Death

Interval (in years)	TB	NMRD	Large intestine	Lung
0–1	243.82 (15)[a,b]	11.59 (8)[b]	4.90 (1)	2.59 (2)
1–3	53.68 (6)[b]	6.18 (9)[b]	2.33 (1)	3.73 (6)[c]
3–5	68.03 (7)[b]	6.88 (11)[b]	2.14 (1)	1.77 (3)
5–10	68.61 (13)[b]	7.86 (33)[b]	2.43 (3)	2.78 (12)[b]
10–20	13.25 (3)[b]	3.66 (35)[b]	1.20 (3)	1.67 (13)
>20	9.38 (1)	0.89 (9)	2.65 (5)	0.69 (3)

[a]Numbers of deaths.
[b]Significant at $p < 0.01$.
[c]Significant at $p < 0.05$.

applied Axelson's method for determining the extent that smoking explains an elevated lung cancer risk [Axelson, 1978]. We assumed that ever smokers are 15 times more likely to have lung cancer than lifetime nonsmokers. Thus, we compared lung cancer risks between California silicotics having 12% nonsmokers and the general male population of the United States where 35% are nonsmokers. In the general population, the confounded risk from Axelson's equation for lung cancer is 10.1, while among silicotics the confounded risk is 13.32. Therefore, the relative risk in the group of silicotics due to a greater prevalence of smoking is (13.32/10.1) = 1.3. Since we observed a significant risk of 1.9 and only 30% of the risk is explained by

smoking, some residual risk is likely related to silicosis or to high exposure to silica dust.

DISCUSSION

This follow-up study of 590 claimants for silicosis demonstrated a significant excess risk for mortality from TB, NMRD, and for lung cancer. A SMR of 56.35 for TB confirms the severity of risk to silicotics in the absence of effective anti-*Mycobacterial* therapies. Since 1980, there have been 26 published epidemiologic investigations indicating significantly elevated risks for lung cancer among silicotics [Goldsmith, 1994a,b]. Consistent with that body of evidence, we found a statistically significant SMR of 1.90 (39 observed; 20.5 expected; 95% CI = 1.35, 2.60) for cancer of the trachea, bronchus, and lung, a risk that rises with level of compensation/degree of silicosis. A nonsignificant SMR of 3.48 was also found for cancer of the larynx. Although other investigators have observed excesses of gastric cancer among silica-exposed cohorts [Kusiak et al., 1993; Greenberg, 1986], we found a previously undocumented doubling of the risk for malignant neoplasms of the large intestine in this cohort. We did not detect significantly elevated risks for neoplasms of the lymphatic and hematopoietic system or for gastric cancers as a whole, sites which have been previously linked with silica exposure by other investigators [IARC, 1987]. Mortality due to cardiovascular diseases and to prostate cancer was significantly reduced. Moreover, significantly elevated SMRs were found for cirrhosis of the liver and for emphysema, suggesting that these silicotic workers may have been habitual consumers of alcohol and tobacco in addition to their high exposures to siliceous dusts.

Partial smoking adjustment using the Axelson method showed that a 1.3 risk for lung cancer could be explained by differences in smoking habits comparing a sample of silicotics and the general male population. This finding suggests that the nearly two-fold lung cancer risk can only be partially explained by confounding by smoking. Furthermore, with the exception of emphysema, mortality risks for cardiovascular diseases and tobacco-related cancers such as kidney, pancreas, and bladder were not elevated. There also emerges a question of whether smoking and silicosis act synergistically to increase the lung cancer risk, as suggested by the work of Hnizdo and Sluis-Cremer [1991] on silica-exposed gold miners.

With a SMR of 243.82 for TB during the first year after filing for compensation, it is clear that TB was a severe complication among these patients, and the force of mortality for TB remained high compared to the general male population of the United States. Because of resistance of some types of TB to standard therapies, *Mycobacterial* sequelae from silicosis may emerge as a serious health problem for both acute and chronic silicosis patients [Fleming et al., 1990].

Increased SMRs for NMRD and cancers of lung and large intestine have been consistently elevated for claimants from this cohort, although not as striking as those for TB. Silicotics from construction and mining and quarrying industries had consistently elevated SMRs for TB, NMRD, cancers of the large intestine and lung, while metallurgy/foundries showed elevated SMRs for TB and NMRD only. Silicosis claimants from transportation, utility, and miscellaneous industries shared the high risks for TB and NMRD; had a nearly a three-fold excess risk for cancer of large intestine, but only a SMR of 0.8 for lung cancer. The high SMR for lung cancer

among silicotics from construction industries may be related to exposure to other cancer hazards such as asbestos insulation. It is of interest that the sole mesothelioma was not observed in construction, but in the transportation/utility group.

We examined the SMRs according to the level of compensation, and discovered positive gradients for TB, NMRD, and lung cancer, but not for cancer of the large intestine. Many scientists have criticized the use of Workers' Compensation data because they lack specific information on whether the worker actually has fibrosis of the lung [McDonald, 1989]. Assuming that the level of compensation reflects degree of severity, these current findings demonstrate that the greater the degree of silicosis, the greater the mortality risk. For lung cancer, our findings are consistent with positive gradients reported by Miller et al. [1987]; Ng et al. [1990]; Chia et al. [1991]; and Hnizdo and Sluis-Cremer [1991] in their studies of silicotics. Furthermore, it appears that links with TB, NMRD, and cancers of the lung and large intestine are stronger among those receiving awards for silicosis than they are among those not being compensated. There were SMRs of 37.18 and 2.59 for TB and NMRD, respectively, among those who received zero compensation. These findings suggest that the classification of silicosis for purposes of compensation was handled conservatively. Alternatively, uncompensated claimants may have either developed silicosis later, or had mild cases that were diagnosed after leaving the workforce, or were misdiagnosed completely. Because X-rays were found to be only 39% sensitive compared to autopsied cases of silicosis in South Africa [Hnizdo et al., 1993], this seems to be a realistic explanation for the findings among those not receiving compensation. Furthermore, because an unknown number of workers did not seek compensation for silicosis, the risks reported in this study are likely to be an *underestimate* of the true association between silicosis and cancer and chronic disease mortality.

CONCLUSIONS

IARC judged that silica dust exposure was a probable carcinogen [IARC 1987] and our study confirms that silicotics selected from a registry of pulmonary disease compensation claims have an increased risk of mortality from lung cancer. This finding is consistent with the world's occupational epidemiology literature [reviewed in Goldsmith, 1994a,b]. It seems that unadjusted confounding from smoking cannot fully explain the lung cancer excess nor does the risk appear to vary by length of follow-up interval, or amount of compensation. Mortality from TB and NMRD is strongly linked with silicosis. Silicotics from mining and quarrying and from construction industries have the greatest risks when compared to metallurgy and miscellaneous industries, and this may be indicative of differential toxicity of quartz polymorphs found in different industries [Guthrie and Heaney, 1995]. Risk of cancer of the large intestine may also be linked to silicosis disability, but there is a need for confirmation of this finding.

Future research should be focused in three areas: primary prevention of silicosis, studies of silica-exposed workers (without silicosis), and follow-up of more recently diagnosed silicotics. In the meantime, all silica-exposed workers and especially those with extant silicosis should be warned by their physicians of the chronic risks from silica exposure, particularly the cancer and TB hazards associated with high exposure to this ubiquitious fibrogenic material.

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