Silicosis among Gold Miners: Exposure–Response Analyses and Risk Assessment

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ABSTRACT

Objectives. This study sought to estimate the risk of silicosis by cumulative exposure-years in a cohort of miners exposed to silica, as well as the lifetime risk of silicosis under the current Occupational Safety and Health Administration (OSHA) standard (0.09 mg/m^3).

Methods. In a cohort study of 3330 gold miners who worked at least 1 year underground from 1940 to 1965 (average 9 years) and were exposed to a median silica level of 0.05 mg/m^3 (0.15 mg/m^3 for those hired before 1930), 170 cases of silicosis were determined from either death certificates or two cross-sectional radiographic surveys.

Results. The risk of silicosis was less than 1% with a cumulative exposure under 0.5 mg/m^3-years, increasing to 68% to 84% for the highest cumulative exposure category of more than 4 mg/m^3-years. Cumulative exposure was the best predictor of disease, followed by duration of exposure and average exposure. After adjustment for competing risks of death, a 45-year exposure under the current OSHA standard would lead to a lifetime risk of silicosis of 35% to 47%.

Conclusions. Almost 2 million US workers are currently exposed to silica. Our results add to a small but increasing body of literature that suggests that the current OSHA silica exposure level is unacceptably high. (*Am J Public Health.* 1995;85: 1372–1377)

Introduction

Silicosis in the United States has become a relatively rare disease since exposure levels were lowered in the 1930s and 1940s. Nevertheless, mortality and morbidity as a result of silicosis continue to occur here. There were approximately 1000 deaths per year from silicosis in the late 1960s, falling to 500 per year in the late 1980s.[1] Data are generally unavailable on silicosis incidence. However, four states that initiated reporting systems in the late 1980s found 400 cases between 1987 and 1990.[2]

An estimated 1.7 million US workers outside of the mining industry are exposed to crystalline silica.[1] Among miners, silica exposure is common but is highly variable, depending on the silica content of the ore. Approximately 200 000 miners have potential silica exposure.

Despite the abundance of historical literature on silica and silicosis, surprisingly little work has been done on the quantitative relationship between exposure and disease. In 1993, Hnizdo and Sluis-Cremer[3] published data on 2235 South African gold miners, 313 (14%) of whom developed silicosis. Silicosis was ascertained longitudinally with annual chest x-rays, which were taken even after workers left the workforce. Silicosis was defined as the presence of rounded opacities with profusion of at least 1/1 according to International Labor Organization (ILO) categories. Converting dust measurements to silica measurements (respirable dust was approximately 30% silica), these authors found that the average cumulative exposure for the whole cohort was about 2 mg/m^3-years over an average 25 years of exposure. No silicosis occurred when cumulative exposures were below 0.9 mg/m^3-years. On the other hand, the cumulative risk of silicosis was approximately 25% at 2.7 mg/m^3-years and 77% at the highest observed levels of 4.5 mg/m^3-years.

Muir et al.[4] found a much lower risk of disease per unit of cumulative exposure. These authors studied 2109 gold and uranium miners in Canada hired between 1940 and 1959, with an average cumulative silica exposure of 2.5 mg/m^3-years over an average 16 years of exposure.[5] Thirty-two (1.5%) of these men developed silicosis, as determined by multiple chest x-rays and defined by a finding of rounded opacities with a reading of at least 1/1 according to ILO categories. The cumulative risk based on a majority (three or more positive) for five readers was about 1% for 3 mg/m^3-years, 2% for 6 mg/m^3-years, and 6% for 12 mg/m^3-years. One explanation for the smaller effect of exposure in these data is that x-rays were taken only while the miners were working, so cases that developed in miners who had left work would have been missed.

Ng and Chan[6] conducted a cross-sectional study of 338 granite workers employed for at least 1 year (and an average of 17) between 1967 and 1985; these workers represented 91% of those currently employed and 61% of past workers who were still alive at the time of the study. The authors estimated that respirable granite dust was 27% quartz. The prevalence of small rounded opaci-

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ties of at least 1/1, confirmed by at least two of three readers, was 13% for 1 mg/m^3-year, 25% for 5 mg/m^3-years, and 22% for 10 mg/m^3-years. No cumulative risks were calculated. The authors acknowledged that their prevalences were underestimates because decedents were excluded from the study. (The underlying cause of death was silicosis for 17 of 53 known decedents.)

Cavariani et al.[7] conducted a longitudinal study based on annual x-rays of 2840 ceramic workers employed from 1974 to 1991. Exposures to respirable silica in the late 1980s averaged 0.2 mg/m^3 for workers in sanitary ware (about two thirds of the workers) and 0.02 mg/m^3 for workers in crockery (about one third of the workers). Exposures were thought to be three to five times higher in earlier years. The cumulative risk of silicosis (based on rounded opacities of at least 1/1), calculated by duration of employment, reached 48% for those employed for 30 or more years.

Other studies in the literature with quantitative data are not as comparable to our own analysis as those listed above. Rice et al.[8] studied 216 silicotics diagnosed by chest x-ray while actively working in dusty trades in North Carolina. These authors conducted a case–control study in which the referent group was exposed to approximately 1 mg/m^3-year of quartz. Odds ratios were calculated and were found to increase by cumulative exposure, but absolute risks were not reported and the referent group itself had 101 cases.

McDonald and Oakes[9] analyzed a subcohort of the one analyzed here (1321 gold miners employed for at least 21 years), with follow-up through 1973. From the underlying cause listed on the death certificate, they ascertained silicosis and found 40 cases of silicosis as well as 49 cases of tuberculosis. An industrial hygienist estimated average exposure and divided it into five categories based on observed dust counts from 1937 to 1973 and observed work histories. Using the entire cohort, McDonald and Oakes estimated the risk of dying from silicosis/tuberculosis (as the underlying cause of death) by category of average exposure. They found an increasing linear trend in risk of about 2.4% for each 0.1 mg/m^3 of silica exposure. No risk by cumulative exposure was calculated.

Finally, Theriault et al.[10] studied 684 Vermont granite shed workers for silicosis prevalence. Based on x-rays taken of active workers, these authors calculated the percentage of men with silicosis based on either rounded or irregular opacities with ILO categories of 1/0 or greater. Individual exposure data were not available, but assuming an average exposure of 0.5 mg/m^3 respirable silica, these authors calculated that 30% of the men had silicosis with 0 to 35 years of exposure, after which time the percentage increased until it reached about 60% with 55 years of exposure. These data are difficult to interpret owing to their cross-sectional nature and to the inclusion of irregular opacities, which, unlike rounded opacities, did not show a relationship to dust exposure.

To further clarify the exposure-response relationship for silicosis, we conducted a cohort study of 3330 gold miners with high levels of historical silica exposure in a South Dakota gold mine.[11]

Methods

Cohort Definition and Exposure Data

The cohort included all White male miners who worked at least 1 year underground between 1940 and 1965. The average year of first exposure was 1945, the average length of follow-up was 37 years, and the average length of employment underground was 9 years. During 106 000 person-years of observation with follow-up through 1990, 1551 of the 3330 miners died. Only 2% of the cohort was lost to follow-up.[11] Death certificates were obtained for virtually all decedents. Mortality based on underlying cause showed excesses for tuberculosis (mostly silicotuberculosis) (standardized mortality ratio [SMR] 3.52, 36 deaths) and for a combined category of pneumoconioses and nonspecific chronic obstructive pulmonary disease (SMR 2.61, 92 deaths). Mortality analyses for silicosis specifically, limited to 1960 onward, showed an SMR of 87.3 based on 24 deaths.

Silica was measured in millions of particles per cubic foot (mppcf) of respirable dust. Conversion to currently used units of milligrams per cubic meter required an estimate of the silica content of the dust. The respirable silica content of the airborne dust in the mine was estimated at 13%.[12] This figure, which is an average based on 82 samples of respirable dust (range: 1% to 48%) obtained in two surveys in the 1970s, is slightly higher than the 9% to 10% estimated for the Vermont granite sheds.[13,14] This prompted the adoption of a slightly higher conversion factor than the 10 mppcf = 0.075 mg/m^3 conversion suggested by Davis et al.[15] Accordingly, based on multiplying 0.075 by 13/9.5, a conversion of 10 mppcf = 0.1 mg/m^3, which coincidentally conforms to the traditional conversion factor used for Vermont granite, was adopted for this study.

A job-exposure matrix was created to estimate dust exposures for each job in the mine over time. All full-time underground jobs were assembled into five major groups (laborers, miners, motormen, supervisors, and skip loaders) based on similarity in job function and dust exposures. A sixth category grouped all jobs not considered full-time underground jobs; these jobs were considered nonexposed. Average dust exposures for the job categories were then calculated using existing measurements for each year from 1937 to 1975. The gold mine operated from the early 1900s; exposures prior to 1937 were estimated at 25 mppcf by industrial hygienists familiar with early mine conditions. Although the mine continued to operate after 1975, exposure levels were low; moreover, only 14% of our cohort were still employed as of 1975. Thus, while no job history data were collected after 1975, this resulted in little underestimation of cumulative exposure in our cohort.

The estimated daily dust exposures (constant over yearly intervals) for each of the five job categories were weighted (multiplied) by a factor estimating how much daily time was spent underground by miners in these jobs; a factor of 1 was assigned to work done in the 1920s, decreasing in later years.[12] For each miner, estimated daily dust levels were summed over time, and the resulting measure was used as the estimate of cumulative exposure.

Mining practices changed over time, resulting in decreased exposure. Wet drilling had replaced dry drilling by 1926. Primary crushing of the ore was transferred above ground in the mid-1930s. In 1950, blasting was restricted to times when miners were not in the mine, which substantially reduced exposure to dust, and ventilation increased continually over time, which lowered dust levels.

Definition of Cases and Analysis

Silicosis cases were identified through death certificates or through x-rays taken during two cross-sectional surveys in 1960 and 1976. In total, 170 cases were identified, 128 by death certificate only, 29 by x-ray only, and 13 by both death certificate and x-ray.

TABLE 1—Descriptive Data for Cohort Members with Silicosis and Rest of Cohort

	Members with Silicosis (n = 170)	Rest of Cohort (n = 3160)
Year first exposed, mean (SD)	1926 (9.7)	1946 (11.2)
Frequency by year first exposed, no. (%)		
<1920	43 (25)	45 (1)
1920–1929	79 (46)	285 (9)
1930–1939	32 (19)	517 (16)
1940–1949	12 (7)	913 (29)
1950–1959	4 (2)	1048 (33)
1960+	0 (0)	352 (11)
Year of birth, mean (SD)	1899 (11)	1920 (13)
Cumulative exposure (mg/m3-years), mean (SD)	2.58 (1.31)	0.54 (0.79)
First exposed <1920	3.93 (1.01)	3.80 (1.56)
First exposed 1920–1929	2.65 (0.89)	2.01 (0.73)
First exposed 1930–1939	1.64 (0.50)	0.94 (0.59)
First exposed 1940–1949	0.60 (0.32)	0.28 (0.24)
First exposed 1950–1959	0.18 (0.16)	0.17 (0.16)
First exposed 1960+	NA	0.08 (0.07)
Years of exposure, mean (SD)	20.8 (8.7)	8.2 (7.9)

TABLE 2—Silicosis and Person-Time at Risk, by Cumulative Exposure

Cumulative Exposure[a] (mg/m^3-Years)	No. Observed Cases	Person-Years at Risk	Crude Rate $\times 10^5$	Adjusted[b] Rate $\times 10^5$
0–0.2	5	51 767	9.6	9.6
0.2–0.5	5	23 447	21.3	18.3
0.5–1.0	15	11 409	131.1	93.9
1.0–2.0	33	10 778	306.2	211.2
2.0–3.0	44	5 308	826.2	522.2
3.0–4.0	42	1 308	3211.0	2254.1
4.0+	26	772	3367.8	2082.2

[a]Assuming a conversion of 10 mppcf = 0.1 mg/m3.
[b]Adjusted for age and calendar time.

From death certificates, a case was defined as any decedent with a listing (underlying or contributing cause) of silicosis, silico-tuberculosis, respiratory tuberculosis, or pneumoconiosis. Few death certificates listed tuberculosis alone (n = 18) while many listed silico-tuberculosis, and it seemed likely that those who died with tuberculosis may well have had silico-tuberculosis. In total, out of 1551 deaths, 141 (9%) were attributed to silicosis according to the death certificates.

Cases were also identified from a 1960 Public Health Service cross-sectional radiographic survey of 837 miners, 636 of whom were in our cohort.[16] Surveyed miners who were not in the cohort generally did not fulfill the cohort definition of White men employed for more than 1 year underground between 1940 and 1965. Among men surveyed in 1960 in our cohort, 32 individuals had either simple (small opacities, n = 10) or complicated (large opacities, n = 22) silicosis, based on the 1959 ILO classification system.

Detailed ILO classifications of x-rays from three readers were also available from a 1976 cross-sectional Public Health Service survey of 526 miners,[17] 229 of whom were in our cohort. (Some had also been in the 1960 survey.) Of these, according to at least one reader, 10 more individuals had small, rounded opacities of either category 1/1 (n = 5) or category 2/2 or greater (n = 5), based on the then-current ILO classification system. Four of the 10 also had large opacities.

Person-time analyses were done to calculate rates by category of cumulative dust. For noncases, cumulative dust exposure was calculated through the end of employment or through the date of record collection (1975), whichever came first. For cases identified by x-ray, cumulative exposure was calculated until the time of x-ray, which was considered the time of silicosis incidence. For cases identified through the death certificate alone, cumulative dose was calculated through the end of employment or through 1975, whichever came first, and date of death was considered the time of silicosis incidence.

The National Institute for Occupational Safety and Health (NIOSH) life table program[18] was used to calculate the silicosis rates (cases per person-time at risk) by seven cumulative exposure categories, stratified by 5-year age and calendar-time intervals. Crude rates were then adjusted for age and calendar time using Poisson regression.[19] Both age and calendar time are highly associated with exposure in that older workers with earlier exposure had higher cumulative exposure. However, there is no a priori reason to believe that age and calendar time should confound an exposure-response analysis because they should not be associated with silicosis; unlike most chronic diseases, silicosis has no background rate for nonexposed populations that changes with age or calendar time. Nevertheless, our data revealed an empirical increase in silicosis rates with age, and rates also varied by calendar time even though cumulative exposure was also included in the Poisson regression model. These associations may occur because our historical information on exposure is deficient, and because age and calendar time are serving to some degree as surrogates for dose. An alternate explanation would be the increasing occurrence of chronic obstructive pulmonary disease, misdiagnosed as silicosis, with increasing age. A disadvantage of controlling for age and calendar time may be an artificial decrease in the exposure-response trend owing to the collinearity of cumulative exposure with age and calendar time, while an advantage may be that these variables are legitimate confounders. The data were analyzed with and without control for these variables, and both results are presented.

Also calculated for our cohort, using standard survival analysis techniques, was their observed cumulative risk by level of cumulative exposure.[20,21] The hazard for each category of cumulative dose was multiplied by the width of the category, and the negative of the sum of these products was exponentiated and sub-

tracted from 1 (see Table 3, footnote b). To adjust these cumulative risks for age and calendar time, expected cases were substituted for observed cases after adjustment for age and calendar time. Expected cases were calculated by multiplying the adjusted rate from Poisson regression by the person-years at risk for each category of cumulative dose.

Silicosis rates (adjusted and unadjusted) by level of cumulative exposure were also used to calculate the lifetime risk (through age 75) of silicosis for a hypothetical subject exposed at the current Occupational Safety and Health Administration (OSHA) standard for 45 years, taking into account withdrawals owing to possible death from any cause.[22,23] Death rates (from all causes) for White US men were used in this analysis, and silicosis rates were considered constant after exposure ceased (e.g., from ages 66 to 75).

Finally, nested case–control analyses were also done to model the effects of duration and intensity separately. Each case was matched to five controls. Controls were selected by incidence density sampling from among those who survived without silicosis to the same age at which the case subject failed. Analyses were conducted via conditional logistic regression, retaining the matching.[21] The only variable in the model was exposure. Results were similar when calendar time was also included.

Results

Table 1 presents descriptive statistics for cohort members with silicosis compared with other cohort members. In general, those with silicosis were employed earlier and had higher cumulative doses than the rest of the cohort. For the cohort as a whole, the median intensity of exposure of silica was 0.15 mg/m^3 for men hired before 1930, 0.07 for men hired between 1930 and 1950, and 0.02 for men hired after 1950. The OSHA permissible exposure limit for respirable dust is (10 mg/m^3)/{(% crystalline silica) + 2}, which in the gold mine under study here is equivalent to about 0.09 mg/m^3 respirable silica.[24] The Mine Safety and Health Administration standard for metal mines is the same as the OSHA standard. The NIOSH recommended level is 0.05 mg/m^3.[25]

Table 2 gives the number of silicosis cases by cumulative exposure category, and the respective rates with and without adjustment for age and calendar time. In no cumulative exposure category is there no silicosis. The cases with lowest cumulative exposure were exposed in the most recent years (lower levels) and had short durations of exposures. These data suggest that some men get silicosis after only brief exposure to low levels or, alternatively, that some of these men either were exposed to silica before or after working at the gold mine studied here or received short-term high exposures ignored by our job-exposure matrix.

We used the rates in Table 2 to calculate a lifetime risk of silicosis (through age 75) for an individual exposed to the current standard (0.09 mg/m^3) from age 20 to age 65, after adjusting for competing causes. The lifetime risk is 47%, reduced to 35% if rates are adjusted for age and calendar time. Note that this calculation assumes that the effect of a cumulative exposure over a 45-year period can be estimated from our data, although the average duration of exposure among our cases is only 20 years.

Table 3 gives the survival analysis results for cumulative risk, presenting such risk with and without adjustment for age and calendar time. Figure 1 presents

TABLE 3—Risk of Silicosis for Entire Cohort, by Cumulative Exposure

Cumulative Exposure (mg/m^3-Years)	Silicotics[a]	No. Entering Category	Cumulative Risk[b]	Adjusted Cumulative Risk[c]	Mean Duration of Exposure (Years)	Mean Year of First Exposure
0–0.2	5	3330	.002	.002	2.9	1953
0.2–0.5	5	1800	.005	.005	9.7	1948
0.5–1.0	15	1060	.022	.017	15.4	1942
1.0–2.0	33	684	.084	.060	13.2	1931
2.0–3.0	44	331	.245	.167	18.8	1926
3.0–4.0	42	125	.534	.403	25.5	1921
>4.0	26	52	.844	.678	30.6	1914

[a]Decedents with silicosis on death certificate or men with silicosis diagnosed by x-ray during two cross sectional surveys.
[b]Cumulative risk = 1 − exp[−sum of (hazards · interval width)], where the hazards for each category of cumulative exposure are no. cases/(width · (no. entering category–0.5 · no. cases–0.5 · no. withdrawals).
[c]Adjusted for age and calendar time by substituting expected number of cases (equaling the adjusted rate · person-years) for the observed number of cases for each cumulative exposure category in the above formula for cumulative risk.

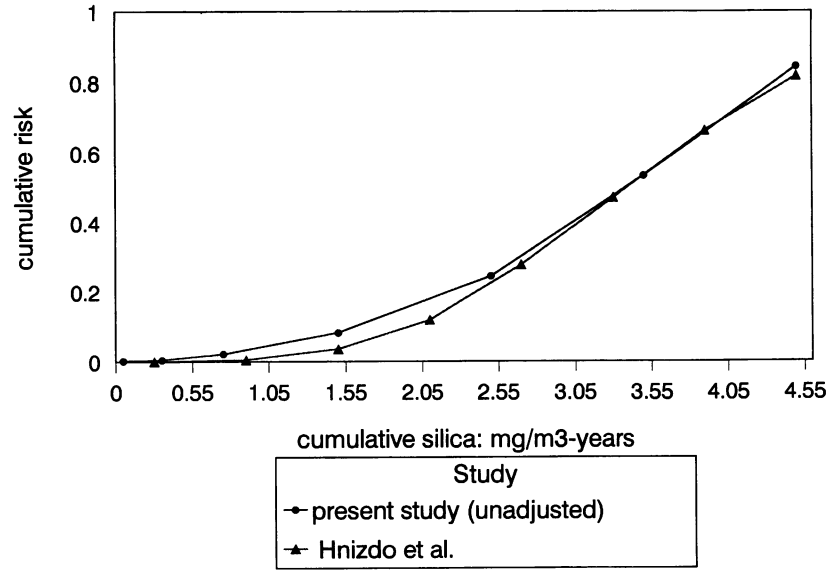

FIGURE 1—Cumulative risk of silicosis, by cumulative silica exposure.

the (unadjusted) cumulative risks from Table 3 as well as the strikingly similar (and also unadjusted) cumulative risks from the analyses by Hnizdo and Sluis-Cremer.[3]

By way of sensitivity analysis, we also conducted the above analyses for tuberculosis only (no mention of silicosis or silico-tuberculosis) without the 18 cases identified through death certificate. Results were quite similar and are not presented. To explore the problem of a lack of work history after 1975 for the 471 cohort members still employed at that date, further sensitivity analyses were conducted by assigning continued exposure to the men until they reached an estimated average length of employment. Again, results were virtually unchanged and are not presented here. Given the low levels of exposure after 1975, it was anticipated that the lack of exposure data for this period would have little effect.

Nested case–control analyses were conducted to determine which exposure measure—cumulative exposure, average exposure, or duration of exposure—was a better predictor of disease. These analyses indicated that a model with cumulative exposure provided a better fit to the data than one with simple duration of exposure (model chi-square based on a difference in -2 log likelihoods; $\chi^2 = 231$ vs 182) and both provided considerably better fit than one with average exposure (model $\chi^2 = 117$). The coefficient for cumulative exposure was 0.913, indicating an odds ratio of 2.49 for each 1-mg/m^3-year increase in cumulative exposure.

Discussion

The risk of silicosis in this cohort increased with cumulative exposure, reaching 68% to 84% for the highest category of cumulative exposure (>4.0 mg/m^3-years). Our results are consistent with the findings of Hnizdo and Sluis-Cremer[3] and are reasonably consistent with those of Ng and Chan[6] and Cavariani et al.,[7] although these last two studies differ in either design (cross-sectional vs longitudinal) or exposure measure (duration of exposure vs cumulative exposure) from our own. On the other hand, we show much higher risks than Muir et al.,[4] which may be partly explained by the fact that Muir et al. detected cases only among active workers, and the average age at end of follow-up was only 44. In contrast, for example, Hnizdo and Sluis-Cremer[3] used a design that was similar to that used by Muir et al.[4] but that followed workers after leaving employment, and they found that, on average, silicosis onset came 3 years after leaving work. Another alternative explanation suggested by Muir[26] is that a lower percentage of silica in respirable dust is less toxic even if cumulative silica exposures are comparable. However, our own data had a relatively low percentage of silica (13%), which was not much higher than that in Muir et al.'s cohort (6% to 8%) and was far lower than that in the cohort studied by Hnizdo and Sluis-Cremer (30%).

Our data are limited by being restricted to silicosis cases detected in two cross-sectional radiographic surveys and identified by death certificates. It would have been preferable to have repeated x-rays over time for each miner, including those miners who had left work. Lacking such data, we were forced to rely principally on death certificates. This may have caused a bias. It is possible that we underestimated the number of cases. About 75% of living cohort members were not x-rayed in the 1960 and 1976 surveys, and these individuals may have had silicosis without our knowledge. However, most of these individuals were exposed in recent years to lower levels and would have been less likely to get silicosis. On the other hand, physicians of decedents may have overdiagnosed silicosis, given a prior knowledge of a high incidence of silicosis in these miners.

There are also several limitations to our estimation of exposure. One problem is the conversion of dust counts to gravimetric measurements (in this case, 10 mpccf = 0.1 mg/m^3), which may not be accurate. This conversion relies on an estimate of 13% silica content of respirable dust. While this estimate was based on a relatively large number of samples (n = 82) collected in two different surveys, there was broad range of content in these samples (1% to 48%, SD = 9), and the percentage of respirable quartz may have differed in earlier years. Without data, there is no way to determine this, although a priori it would seem reasonable that the similar ore would produce similar percentages of respirable quartz over time. Using a sensitivity analysis, we calculated the lifetime risk of silicosis if the percentage of respirable quartz was as high as 18% and as low as 8% (the range from first to third quartiles of the 82 samples); these risks, adjusted for death from any cause, are 19% and 59%, respectively, compared with our original lifetime risk estimate of 47% (unadjusted for age and calendar time).

Another limitation to our data is the lack of actual dust measurements prior to 1937. Instead, an estimate of silica exposure (0.25 mg/m^3) by industrial hygienists familiar with the mine was used for the years prior to 1920, and this estimate was then decreased gradually from 1920 to 1937 as a function of decreased time spent underground. Ninety-two percent of our 170 cases involved men exposed prior to 1937 with, on average, about 50% of their work history also occurring prior to 1937. If we restrict our analysis to men hired in 1937 or later, no miner had a cumulative dose in excess of 2 mg/m^3-years. However, results considering only miners hired after 1937 were about the same as those in the original analysis. Miners hired after 1937 with cumulative exposure in excess of 2 mg/m^3-years had a cumulative risk (unadjusted) of 0.26, compared with the original analysis in which those with a cumulative exposure of 2 to 3 mg/m^3-years had a cumulative risk of 0.25.

In summary, our data indicate that the cumulative risk of silicosis in our cohort is 68% to 84% for those with more than 4 mg/m^3-years of exposure. The estimated lifetime risk of silicosis for someone exposed for 45 years at the OSHA standard (0.09 mg/m^3) is 35% to 47%, after adjusting for competing causes of death. If our data are correct, the OSHA standard would appear unacceptably high. OSHA sets standards to ensure that lifetime excess risks of death or serious disease are less than 1 in 1000 for a lifetime exposure, while our data suggest a lifetime risk of 3 to 4 in 10. □

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