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Silicosis and Lung Cancer in U.S. Metal Miners

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ABSTRACT. The association between silicosis and lung cancer mortality was estimated in 9 912 (369 silicotics and 9 543 nonsilicotics) white male metal miners. These miners were examined by the U.S. Public Health Service during 1959–1961 and were followed through 1975. Miners were excluded from this study if they were employed in a mine during 1959–1961 that used diesel equipment underground. The ores that were mined consisted of copper, lead-zinc, iron, mercury, lead silver, gold and gold-silver, tungsten, and molybenum. The standardized mortality ratio (SMR, U.S. white male rates) for lung cancer was 1.73 (95% CI: .94–2.90) in silicotics and 1.18 (95% CI: .98–1.42) in nonsilicotics. Additionally, SMRs were higher in silicotics than in nonsilicotics, even in most subgroups stratified by cigarette smoking habit, type of ore mined, years of service in an underground job, radon exposure group, or year of hire. When lung cancer mortality between silicotics and nonsilicotics was compared, the age-adjusted rate ratio (95% CI) was 1.56 (.91–2.68), and the age- and smoking-adjusted rate ratio was 1.96 (.98–3.67). Corresponding figures for miners who were employed in mines with low levels of radon exposure were 1.90 (.98–3.67) and 2.59 (1.44–4.68), respectively. These findings indicate that lung cancer mortality risk was increased in silicotics, and this probably did not result from chance or bias. However, confounding from radon exposure could not be ruled out. The findings indicate that further follow-up of this cohort is needed.

IN RECENT YEARS, the relationship between silica exposure, silicosis, and lung cancer has received increased attention. Goldsmith et al.[1] hypothesized silica to be a lung carcinogen or co-carcinogen alone or through the route of silicosis. Heppleston[2] suggested there was no conclusive evidence for silica or silicosis to cause lung cancer in humans. The International Agency for Research on Cancer[3] (IARC, 1987; pp. 108–09) recommended that there was sufficient animal evidence for an association between silica and cancer, and there was "limited" human evidence for an association. McDonald[4] concluded that although evidence is credible for the carcinogenicity of silica, "alternative explanations such as chance, bias, or confounding have not been excluded."

Shortcomings of previous studies of lung cancer mortality in silicotics include (a) noncomparability of reference groups, (b) possible detection bias, and (c) possible confounding from other carcinogenic risk factors such as cigarette smoking and exposure to other known occupational carcinogens.

In this study we estimate lung cancer mortality risks during 1959–1975 in a cohort of silicotic metal miners who were diagnosed for silicosis in 1959–1961. These data offer advantages over those from some previous studies because we employed an internal reference

group of nonsilicotics, radiographs for both silicotics and nonsilicotics were classified for silicosis according to a standardized method, and individual data on cigarette smoking and exposure to radon and diesel were available.

Methods

During 1959–1961, the U.S. Public Health Service administered medical examinations to 12 258 miners who had been employed at 50 underground mines (16 states), that produced 18 different metal ores.[5] Mortality results from 1959 to 1975 were reported by Costello.[6] In this report, we present data for 9 912 white males (369 silicotics and 9 543 nonsilicotics) who had been employed in 38 nonuranium mines in which diesel equipment had never been used prior to 1961. A description of the cohort is given in Table 1.

The 1959–1961 medical examination consisted of a posteroanterior chest radiograph, a spirometry test, and a questionnaire on medical symptoms, cigarette smoking habits, and work history. Radiographs were classified for pneumoconiosis by three radiologists according to the 1959 *ILO classification*,[7] which provided for 5 major categories of profusion of small opacities (i.e., 0, Z, 1, 2, and 3) and size of large opacities. Categories 1–3 were evidence of increasing profusion, and category Z was suspect for pneumoconiosis. For the purpose of this report, silicosis was defined as radiographic evidence of categories 1, 2, or 3 small rounded opacities or large opacities.

Cause of death for those who died during or before 1975 was determined from death certificates. The number of years spent in underground metal mine jobs prior to the time of examination was used as a surrogate estimate of cumulative dust exposure. Individual cigarette smoking status at the time of the 1959–1961 examination was classified as current smoker, ex-smoker, or nonsmoker.

Radon exposure index. Radon exposure data, which were available for 28 of the 38 study mines from 1 527 area samples, had been collected by the Bureau of Mines (BOM) in 1959 and by the Mine Safety and Health Administration (MSHA) during 1976–1988. Ra-

Table 1.—Number of Workers (No.) and Person-Years (PY) for Silicotics and Nonsilicotics, by Risk Factor

Risk factor	Nonsilicotics		Silicotics	
	No.	PY	No.	PY
Cigarette smoking habit				
Current smoker	6 777	94 181	213	2 527
< 25 y smoked	4 797	68 086	64	823
⩾ 25 y smoked	1 933	25 411	147	1 685
Years smoked unknown	47	684	2	19
Ex-smoker	981	13 594	88	1 093
Nonsmoker	1 734	24 462	68	888
Smoking habit unknown	51	665	0	0
Ore Type*				
Copper	2 976	41 720	133	1 635
Lead-zinc	1 943	27 419	76	926
Iron	2 047	27 446	50	620
Mercury	263	3 527	11	103
Others	2 314	32 791	99	1 223
Service underground (y)				
< 20	8 171	115 289	150	1 809
⩾ 20	1 372	17 613	219	2 698
Radon exposure group†				
A–D	5 874	80 016	186	2 200
E–H	3 669	52 886	183	2 307
Year of hire				
< 1934	1 577	20 130	261	3 131
1935–45	2 461	34 025	85	1 083
⩾ 1945	5 505	78 747	23	293
Total	9 543	132 902	369	4 507

*Number of mines: 9 copper, 8 lead-zinc, 10 iron, 4 mercury, and 7 others (1 lead-silver, 2 lead silver and lead zinc, 2 gold and gold-silver, 1 tungsten, and 1 molybdenum).
†See Table 2 for group definitions.

don exposure data had not been collected for 10 of the 38 study mines, but exposures had been extrapolated by the BOM from samples taken at nearby facilities (MSHA, personal communication). For this report, each of the 38 mines was categorized into 1 of 7 groups (A–H [Table 2]) based on the highest exposure concentration in working levels (WL) from all samples taken (< .1, .1–.5, ≥ .5 WL), the percentage of samples with a radon exposure level above .1 WL (< 1%, 1–20%, ≥ 20%), and the number of samples collected (< 10, ≥ 10). ("Working levels" is defined as any combination of short-lived decay products in 1 l of air that will result in the emission of 1.3×10^5 MeV of alpha energy.) The 10 mines without any sample data were categorized based on data from nearby mines that posibly had similar exposures.

Groups A–D represented mines that possibly had "low" levels of radon, and groups E–H represented mines that possibly had "high" levels of radon in some areas. These data were used to determine mines that had low levels of radon (A–D) so that the relationship between silicosis and lung cancer could be estimated with minimal radon confounding.

Comparison groups. Lung cancer mortality rates were compared with those from external and internal reference groups. Rates in silicotics were compared with those for white males in the United States by employing a person-years (PY) analysis. Person-years were accumulated from date of examination to December 31, 1975, during 5-y age intervals and 5-y time periods. Standardized mortality ratios (SMRs) were estimated according to the method described by Monson.[8] The method of Bailar et al.[9] was employed to test whether SMRs were greater than 1.0 and for computing 95% confidence intervals.

Lung cancer mortality rates (deaths/PY) in silicotics were also compared with those of nonsilicotics in the study sample by employing an analog of the Mantel-Haenzel (MH) estimator (with continuity correction) for incidence density data.[10] The MH indicator density ratio is similar to the MH relative risk, but it uses PY as the number at risk for the denominator of death rates. Person-years and observed numbers of lung cancer deaths were tallied by 10-y age intervals of follow-up in the silicotic and nonsilicotic reference groups. The MH age-and-smoking-adjusted rate ratios, chi-squares, and 95% test-based confidence intervals (CI) were calculated.

Internal comparisons between silicotic and nonsilicotic metal miners were also made with an iterative weighted least squares regression model.[11] Lung cancer mortality risk was assumed to be Poisson distributed and exponentially related to age, cigarette smoking, and silicosis. The model was fitted to observed lung cancer death rates (deaths/PY) stratified by 9 5-y age intervals (< 35, 35–39, 40–44, ... , 65–69, > 69), 3 cigarette smoking groups (current, ex-, and nonsmokers), and two silicosis categories (silicotic and nonsilicotic).

Results

Lung cancer mortality (Table 3) was increased in silicotics (SMR = 1.73, 95% CI: .94–2.90) but did not reach statistical significance. Lung cancer SMRs were higher in silicotics than in nonsilicotics, even in most subgroups, after tabulating by cigarette smoking habit, years service in an underground metal mine, type of ore mined, radon exposure group, or year of hire.

SMRs were significantly ($p < .05$) increased in silicotics and nonsilicotics who were current smokers in 1959–1961—particularly in those who had smoked cigarettes for more than 25 y (2.69 in silicotics, 1.76 in nonsilicotics). However, when general population mortality rates are used, figures may be overestimated for smokers and underestimated for nonsmokers.

The SMRs were also significantly increased in (a) nonsilicotics (1.52) who had worked in excess of 20 y in an

Table 2.—Distribution of Mines, by Radon Exposure Group

Group	Radon exposure*			Mines		Number of samples	Number of samples/mine
	Maximum	Percentage	Number	With samples	Without samples		
A	<.1	<1%	≥10	2	0	38	19
B	<.1	<1%	<10	10	5	32	2
C	.1–.5	1–20%	≥10	5	0	184	37
D	.1–.5	1–20%	<10	0	4	0	0
E	.1–.5	≥20%	≥10	1	0	94	94
F	.1–.5	≥20%	<10	2	1	10	3
G	≥.5	≥20%	≥10	6	0	899	149
H	≥.5	≥20%	<10	2	0	6	3

*Each mine was categorized into one of seven groups (A–H) based on the following criteria: maximum = highest radon exposure in working levels (WL) from all samples taken at a mine during 1959–1988; percentage = percentage of all samples taken during 1959–1988 that exceeded .1 WL; and number = number of samples collected during 1959–1988.

Table 3.—Observed (Obs.) and Expected (Exp.) Number of Lung Cancer Deaths and SMRs for Silicotics and Nonsilicotics, by Risk Factor

Risk factor	Nonsilicotics				Silicotics			
	Obs.	Exp.	SMR	95% CI	Obs.	Exp.	SMR	95% CI
Cigarette smoking habit								
Current smoker	106	63.5	1.67*	1.36–2.02	9	4.1	2.17	.99– 4.12
< 25 y smoked	41	26.7	1.53*	1.10–2.08	1	1.1	.89	.02– 4.96
⩾ 25 y smoked	64	36.2	1.76*	1.36–2.26	8	3.0	2.69*	1.16– 5.30
Ex-smoker	7	15.0	.47*	.19– .96	4	2.1	1.93	.53– 4.93
Nonsmoker	5	20.7	.24*	.08– .57	1	1.9	.53	.01– 2.95
Ore type†								
Copper	44	36.6	1.20	.87–1.61	3	3.1	.96	.20– 2.81
Lead-zinc	31	24.5	1.26	.86–1.29	4	1.7	2.42	.66– 6.21
Iron	17	18.0	.94	.55–1.51	2	1.1	1.77	.21– 6.38
Mercury	8	3.0	2.66*	1.15–5.24	3	.2	14.03*	2.89–40.99
Others	18	17.7	1.02	.60–1.61	2	1.2	1.01	.12– 3.68
Years worked underground								
< 20	74	70.8	1.05	.82–1.31	5	2.8	1.78	.58– 4.16
⩾ 20	44	29.1	1.52*	1.10–2.03	9	5.3	1.70	.78– 3.23
Radon exposure group‡								
A–D	79	62.5	1.26	.99–1.57	9	4.2	2.14	.98– 4.06
E–H	39	37.4	1.04	.74–1.42	5	3.9	1.28	.41– 2.99
Year of hire								
< 1934	46	38.8	1.19	.87–1.58	10	6.4	1.57	.75– 2.89
1935–1944	37	33.2	1.11	.78–1.53	2	1.4	1.39	.17– 5.01
⩾ 1945	35	27.8	1.26	.88–1.75	2	.3	7.24	.87–26.14
Total	118	99.9	1.18	.98–1.42	14	8.1	1.73	.94– 2.90
Total, excluding mercury miners	110	1.0	1.14	.93–1.37	11	7.9	1.39	.70– 2.49

*$p < .05$.
†See footnote (*) in Table 1.
‡See Table 2 for group definitions.

underground metal mine and in (b) silicotics (14.03) and nonsilicotics (2.66) who had been employed at a mercury mine. After excluding mercury miners, SMRs were similar in silicotics (1.39) and nonsilicotics (1.14).

The age-adjusted lung cancer risk in silicotics (95% CI) was 1.56 (.91–2.68) times higher than that in nonsilicotics (Table 4). The age-adjusted lung cancer risk in silicotics who were current smokers was 1.39 (.71–2.69) times higher than that in nonsilicotic current smokers. Corresponding figures for ex-smokers and nonsmokers were 3.94 (1.55–10.05) and 3.77 (1.03–13.78), respectively. The age-adjusted lung cancer risk in silicotics was significantly different from that in nonsilicotics for ex-smokers and nonsmokers but not for current smokers or for those who had smoked cigarettes for at least 25 y.

The age-and-smoking-adjusted lung cancer risk in silicotics was 1.96 (1.19–3.23) times higher than that in nonsilicotics, and it was 1.70 (.97–2.95) times higher after mercury miners were excluded. These figures should be interpreted cautiously because there was only one lung cancer death among nonsmoking silicotics.

The age-adjusted lung cancer risk in silicotics who were employed at mines in which there was low radon exposure (groups A–D) was 1.90 (.98–3.67) times higher than that in nonsilicotics, and the age-and-smoking-adjusted risk was 2.59 (CI: 1.44–4.68) times higher. The age-adjusted lung cancer risk in silicotic current smokers who were employed at mines in the lowest radon exposure groups A and B was 3.82 (1.78–8.21) times higher than that in nonsilicotics. There were no lung cancer cases in silicotic exsmokers or in silicotic nonsmokers at mines in groups A and B.

Results from the Poisson model (Table 5) indicated that the age-and-smoking-adjusted lung cancer risk in silicotics was 1.57 (.94–2.64) times higher than that in nonsilicotics and was 1.29 (.67–2.49) times higher after mercury miners were excluded. In our model, silicosis and the interaction between silicosis and cigarette

smoking habits were not statistically significant factors related to lung cancer mortality.

Discussion

Lung cancer mortality was higher in silicotics than in nonsilicotics, and this trend was observed when using U.S. white males and nonsilicotic metal miners as reference groups. The increase in lung cancer mortality was observed regardless of cigarette smoking habit, years spent in underground jobs at a metal mine, and low radon exposure.

Chance and cigarette smoking are unlikely explanations for the increased lung cancer mortality in silicotic cigarette smokers who were employed at mines with low radon exposure (groups A–D). However, misclassification of radon exposure could not be ruled out.

If misclassification explains the increase in lung cancer mortality in miners exposed to low radon levels, perhaps chance accounts for the increase in the total cohort. Age-adjusted risks were not significantly higher in silicotics than in nonsilicotics, and although age-and-smoking-adjusted rate ratios were significantly increased, these figures were questionable because of the few lung cancer cases in silicotic nonsmokers. The number of silicotics (4 507 person-years) afforded a probability (power) of at least .8 to detect a rate ratio greater than 3.0. Because estimates of the age-adjusted rate ratio were generally less than 3.0 in most subgroups, power was low to detect an association. Results indicate that further follow-up of this cohort is needed.

Although cigarette smoking, metal mine dust exposure, and radon exposure could enhance the risk of lung cancer, confounding from these factors does not explain unconvincingly all of the increased lung cancer mortality in silicotics. Even in the event that all silicotics had smoked cigarettes and all nonsilicotics had never smoked, confounding from smoking would account for less than a 30% increase in lung cancer risk.[27] Thus, cigarette smoking probably does not explain age-adjusted rate ratios of more than 1.3, which we observed in this study.

Table 4.—Observed and Expected Number of Deaths, Age- and Smoking-Adjusted Rate Ratios* for Silicosis, and 95% CI from MH Analysis

	Age-adjusted rate ratios	95% CI	
		Lower	Upper
	Total group		
Total	1.56	.91	2.68
Cigarette smoking habits			
Current smoker	1.39	.71	2.69
⩾ 25 pack-years	1.62	.82	3.22
Ex-smoker	3.94†	1.55	10.05
Nonsmoker	3.77†	1.03	13.78
Age-smoking adjusted rate ratio	1.96†	1.19	3.23
	Total group—excluding mercury miners		
Total	1.35	.74	2.46
Cigarette smoking habits			
Current smoker	1.02	.46	2.28
Ex-smoker	4.25†	1.66	10.85
Nonsmoker	3.54	.97	12.98
Age-smoking-adjusted rate ratio	1.70	.97	2.95
	Groups A–D‡		
Total	1.90	.98	3.67
Cigarette smoking habits			
Current smoker	1.72	.78	3.80
Ex-smoker	9.35†	2.58	33.80
Nonsmoker	5.08†	1.44	17.91
Age-smoking-adjusted rate ratio	2.59†	1.44	4.68
	Groups A and B‡		
Current smokers only	3.82†	1.78	8.21

*All rate ratios (see text for definition of incidence density ratio) are age-adjusted unless otherwise indicated.
†$p < .05$.
‡See Table 2 for group definition.

Table 5.—Results from a Poisson Model with an Exponential Link Function

Model	Total group, including mercury mines (deviance†)	Total group, excluding mercury mines (deviance†)	*df*
1 Age, smoking, silicosis	25.20	29.19	41
2 Age, smoking	27.52	29.79	42
3 Age, silicosis	68.68*	65.78	43
4 Smoking, silicosis	150.85*	179.25	49
5 Age, smoking, silicosis, smoking × silicosis	22.80	25.24	39
2-1 Silicosis	2.32	.60	1
3-1 Smoking	43.48*	36.59*	2
4-1 Age	125.65*	50.06*	8
5-1 Smoking × silicosis	2.40	3.95	2
Relative risk from model 1 (95% CI)			
Current smokers	6.63 (3.06, 14.40)	6.03 (2.55, 14.28)	
Ex-smokers	2.36 (1.92, 6.01)	2.34 (.83, 6.61)	
Silicosis	1.57 (.94, 2.64)	1.29 (.67, 2.49)	

*Contribution of factor reached significance ($p < .05$).
†Deviance: log-likelihood evaluated at maximum likelihood estimates minus log-likelihood evaluated at sample points (for complete model, see Frome et al.[11]).

Confounding from carcinogenic metals or carcinogens that contaminate some metal ores (e.g., arsenic) was an unlikely explanation for the higher lung cancer mortality in silicotics. The carcinogenicity of metal exposures in our study mines (mercury, copper, lead-zinc, iron, lead-silver, gold, gold-silver, tungsten, and molybdenum) has not been substantiated.[12] Additionally, there is no evidence in our data for the carcinogenicity of these metal mine dusts, except possibly in mercury mines. Costello[6] reported increased SMRs in chrome miners as part of his report on the mortality of workers employed in the original 50-mine study. However, all chrome mines were excluded from this analysis because diesel equipment was used underground.

Whether mercury mine exposures are carcinogenic has not been determined. Cragle et al.[13] found increased lung cancer SMRs in both exposed and unexposed mercury manufacturing plant workers and concluded that a factor other than mercury explained their findings. Mercury mines in our study were in the low radon exposure group B; thus, radon may not account for the increase in lung cancer risk observed in our mercury mine cohort. In any event, the age-adjusted lung cancer risk in silicotics who had been employed at non-mercury mines were higher than that in nonsilicotics, and this difference was statistically significant for ex-smokers (Table 4). Therefore, mercury mine exposure does not account for all of the increase in silicotics.

One reason to suspect that we may have misclassified mines into radon exposure groups is a result of the paucity of historical radon samples (there were no radon exposure data available prior to 1959 or during 1960–1977). Also, lung cancer mortality was expected to be lower in radon exposure groups A–D than in E–H, and this was not found.

Conversely, there are several reasons why mines were not misclassified with respect to radon exposure. First, it is possible that only a few individuals at mines in groups E–H had high radon exposure, and their increased lung cancer risk may have been masked by the low risk of many individuals who had low exposure. This may explain the difference between groups A–D and E–H.

Second, 19, 2, and 37 samples per mine were available for consideration in mines A, B, and C, respectively. Although this is not a large number for estimating exposures over a 30-y period (1959–1989), less than 1% of the samples in groups A and B exceeded .1 WL, and less than 20% in group C exceeded .5 WL. Thus, there is little evidence of high radon exposures in these mines.

Third, radon did not appear to be a confounder for the relationship between dust exposure and lung cancer mortality in miners who were employed at a South Dakota gold mine.[14,15] Workers who were employed at this mine during 1959–1961 were included in our cohort, and we categorized this mine into radon exposure group C. Radon exposures at this mine ranged from 0–.25 WL in 12 samples that had been collected in 1959 and during 1981–1988. These results are consistent with low radon exposure at mines in groups A–D.

Misclassification of silicosis also was not ruled out, but it is an unlikely explanation for the increased lung cancer mortality observed in our silicotic metal miners. Misclassification may have occurred if some nonsilicotics diagnosed in 1961 subsequently developed silicosis by 1975. However, misclassification of nonsilicotics during the study period would have underestimated lung cancer rate ratios for silicosis and lung cancer, and this would not explain our findings.

Employing the method described by Kleinbaum et al. (p. 234)[10] the number of nonsilicotics in each 10-y age group of current smokers (Table 4) was corrected for possible misclassification of silicosis. For these calculations, we assumed that the sensitivity for classification of silicosis was .3, based on the tenure distribution and prevalance rates in the 1959 survey, and the specificity of silicosis and the specificity and sensitivity of lung cancer were assumed to be 1.0. The age-adjusted rate ratio for silicosis in current smokers was 1.39 (95% CI: .71–3.69) uncorrected and 1.56 (95% CI: .91–2.67) corrected. Thus, in this exercise misclassification of silicosis only slightly reduces the relative risk.

Criteria for a causal association include biological plausibility, strength of association, an exposure-response association ruling out bias and confounding, and consistency among studies. The hypothesis for the carcinogenicity of silica appears plausible as a result of positive findings in animals.[3] However, a consistent association between silica or silicosis and cancer in humans, ruling out confounding and bias, has not been substantiated.

Although lung cancer risks have not been increased in silicotics who have been diagnosed at autopsy,[2,21,29] lung cancer risks in silicotics enumerated from registry and compensation records[16-20,22-28,30-31] have been 1.4–6.5 times higher than those in nonsilicotic reference groups (except in South African gold miners [rate ratio = 1.1]).[21] Selection bias may explain the low risks in silicotics diagnosed from autopsy data. Amandus et al.[30] and Infante-Rivard[31] reported that detection bias probably accounts for some but not all of the increased lung cancer risk in silicotics enumerated from registry data.

Detection bias in silicotics may arise when silicosis is diagnosed as part of an examination of lung cancer. If detection bias were operating in our study, an increase in mortality from all cancers that require a chest radiograph would be expected. Additionally, detection bias may cause an increase in lung cancer rate ratios within 1–5 y after the diagnosis of silicosis, but would probably not explain an increase 5 y or more after diagnosis.

Lung cancer SMRs for 1960–1964—the 5-y period following the 1959–1961 examinations—were 1.9 for silicotics and 1.3 for nonsilicotics. Figures for 1964–1975 were 1.7 for silicotics and 1.3 for nonsilicotics. The SMRs for all cancers, excluding cancer of the lung, were 1.0 for silicotics and .9 for nonsilicotics. Therefore, these data do not indicate that detection bias accounted for all of the increased lung cancer mortality in our silicotic cohort.

The age-and-smoking-adjusted rate ratio (RR) in our study was similar to that reported in other studies, which ruled out exposures to other known occupational carcinogens, adjusted for cigarette smoking, and compared lung cancer rates in silicotics with those in an internal nonsilicotic reference group (our metal miners: RR = 2.0; Hessel et al.[21]: RR = 1.1 from radiographic evidence and RR = 1.9 from autopsy evidence; Mastrangelo et al.[28]: RR = 1.9; Forastiere et al.[24-25]: RR = 2.1; Amandus et al.[30]: RR = 3.9).

In conclusion, our data indicate a relationship between silicosis and lung cancer in a low-radon-exposed group of metal miners, a relationship that does not appear to be explained by chance or bias. However, the effect of confounding from radon exposure could not be determined fully. Lung cancer rate ratios for our silicotic metal miners appear to be similar to those from most other silicotic studies in which internal reference groups were used and in which cigarette smoking habits were adjusted for. Evidence from our study and from other studies suggests that detection bias may not explain all of the increase in lung cancer mortality in silicotics.

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