DOSE-RESPONSE RELATIONSHIP BETWEEN ASBESTOS EXPOSURE AND INCIDENCE OF ASBESTOSIS

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INTRODUCTION

The asbestos products of a Beijing asbestos factory include textile products, brakes, rubber, asbestos-cement and thermal insulation materials. More than 80% of the raw materials was chrysotile. Both men and women were employed. The purpose of this research was as following:

1. To study the reliability of currently used hygiene criteria of asbestos in China.
2. To provide the scientific basis for the modification of these criteria in the future.

MATERIALS AND METHODS

Beijing asbestos factory was set up by combining three small factories in the 50's. No anti-dust measures were taken at that time. All procedures were operated openly with very simple equipment; the asbestos concentration reached as high as 300 mg/m^3. Since the 1960's, the working conditions have been improved and the asbestos concentration declined greatly. At the end of 1982, 90% of the dust samples had reached the recommended standard.

The asbestos concentration in the air of workplaces was collected from 1957 to 1982. The data from 1967 to 1972 was missing; it was estimated as the average of those in 1966 and 1972. The asbestos concentration level from 1951 to 1956 was calculated as 1.5 times higher than those in 1957.

There are three types of workshops: Raw material, carding and spinning, wearing. In this study, the subjects who worked for more than 1 year were selected. They should not have any exposure history in other factories and mines. According to this criterion, 532 workers who had regular X-ray photographs were selected, among which there were 46 cases with asbestosis at stage I.

At first, the data of dust concentrations were rearranged according to the type of work, and average of the annual asbestos concentrations were estimated year by year. Secondly, for each worker who was exposed to asbestos the actual exposure duration was recorded. An adjustment was given to those who only worked in the workshops part-time, such as repairmen. At last, for each worker, the cumulative dust exposure (D) was estimated according to equation (1).

$$\text{Cumulative dust exposure (mg/year)} = CiTi \qquad (1)$$

where, Ci is the asbestos concentration (mg) at the working time interval, Ti is the length of the i working interval in year.

Both life table and linear regression models were used to analyze the relationship between the incidence of asbestosis and the asbestos exposure.

RESULTS

Life Table Model[3]

Table I was constructed according to the principles of life table model. Let Lx be the total number of observed workers entering the x cumulative exposure interval, Lx+a=Lx-Wx-dx; dx the number of workers who got asbestosis in this interval; Wx the number of censored; Nx the corrected number (Nx=Lx-Wx/2). Other columns in Table I are calculated by using equations (2) to (5):

$$px = dx/Nx \qquad (2)$$

$$qx = 1-px \qquad (3)$$

$$x+aQo = qoqaq2a\ldots\ldots qx \qquad (4)$$

$$x+aPo = 1-x+aQo \qquad (5)$$

where px is the probability of asbestosis in x interval

qx is the probability of not suffering from asbestosis

x+aPo is the cumulative probability

x+aQo is the cumulative probability of not suffering from asbestosis

It is shown from Table I that the probability of cumulative incidence could be 2.54% when the cumulative exposure from 0 to 199 mg/yr. It is clear that the cumulative incidence rate increases as the cumulative exposure increases. The incidence rate might rise up to 56.3% if the cumulative exposure reaches 800 mg/yr. It means that more than half of the employees will suffer from asbestosis if their cumulative exposure reached 800 mg/yr.

In order to search if there is any linear correlation between the asbestos exposure and the asbestosis incidence rate, logarithmic transformation and logit transformation were made to the columns 1 and 9 in Table I respectively (Table II). It is shown that there exists a linear correlation between the logarithm of asbestos exposures and the logit value of cumulative asbestosis incidence rate (Figure 1).

The regression line is obtained from Table II:

$$\text{Logit} = 5.08\text{LgD}-15.23 \qquad (6)$$
$$(r=0.99\ P<0.05)$$

Table I

Probability of Cumulative Incidence under Different Cumulative

Cumulative exposures (mg.yr) (1)	Lx (2)	Wx (3)	dx (4)	Nx (5)	px (6)	qx (7)	x+aQo (8)	x+aPo (9)
0-	532	356	9	354	0.0254	0.9746	0.9746	0.0254
200-	167	62	15	136	0.1103	0.8897	0.8671	0.1329
400-	90	38	11	71	0.1549	0.8451	0.7328	0.2672
600-	41	12	3	35	0.0857	0.9143	0.6700	0.3300
800-	26	6	8	23	0.3478	0.6522	0.4370	0.5630
1000-	12	9	0	7.5	0	1.0000	0.4370	0.5630

Table II

Conversion Value from Table I

Logarithmic dose (upper limit)	* logit
2.30	-3.65
2.60	-1.87
2.78	-1.01
2.90	-0.71
3.00	0.25
3.08	0.25

*logit =Ln[P/(1-P)], P is the cumulative asbestosis rate

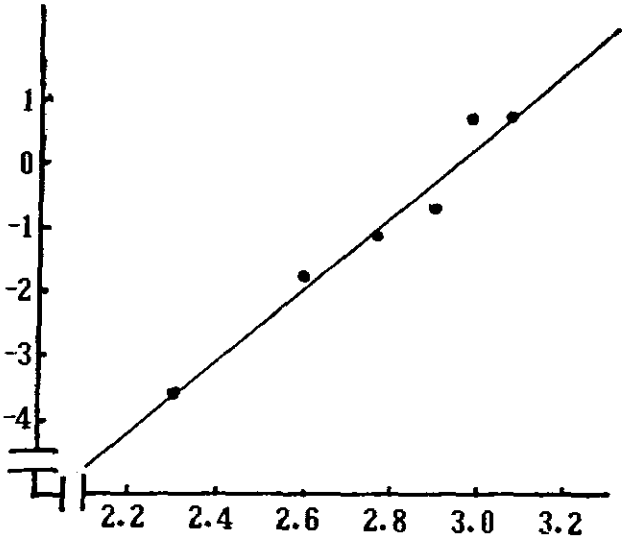

Figure 1. The correlation between logarithmic doses and logit.

Where, D is the cumulative asbestos exposure (mg/yr)

From equation (6), the conclusion could be made that the cumulative asbestosis rate will not be greater than 1% if a person worked for 40 years under the condition that the average asbestos concentration was not higher than 3.09 mg/m^3.

Regression Model[4]

This model was introduced by Dr. Tian in 1980. The principle is to translate the worker's rank into probits then by using regression model to find the relationship between the asbestosis rate and asbestos exposure. The procedure is as follows:

First to build Table III. The purpose is to illustrate the relationship between working duration (in year) and the asbestosis incidence rate among the employees. All employees were divided into two subgroups, one with asbestosis, the other without asbestosis, two equations could be given by using the least square method separately:

$$Y = 3.4806+0.1174X \quad (7)$$

$$Y = 1.9858+0.1867x \quad (8)$$

where Y is the probit
X is the working duration (in year).

Columns (2) and (3) are estimated from equations (7) and (8) respectively. Columns (4) and (5) are the reverse transformations from columns (2) and (3). The values in column (6) are calculated according to equation (9).

$$P=B/A.K.1000\% \quad (9)$$

where K is called prevalence rate of asbestosis. In the last column are the expected cumulative prevalence rates estimated from equation (10).

$$P = -11.8064+4.47X \quad (10)$$

where X is the mid-value of working duration.

Table III

Estimation of Asbestosis Prevalence Rate with Different Length of Working Time

Length of Working time X	probability units: exposed workers	probability units: workers with asbestosis stage I	cumulative frequency: exposed workers A	cumulative frequency: workers with asbestosis stage I B	P (B/A.K. 1000‰)	$\hat{P}$ (‰)
5	4.0677	2.9191	0.176	0.019	9.34	10.54
7	4.3025	3.2924	0.243	0.044	15.66	19.48
9	4.5374	3.6657	0.322	0.092	24.71	28.42
11	4.7722	4.0391	0.410	0.169	35.65	37.36
13	5.0071	4.4124	0.503	0.279	47.98	46.30
15	5.2419	4.7857	0.596	0.416	60.38	55.24
17	5.4767	5.1590	0.684	0.564	71.32	64.18
19	5.7116	5.5323	0.762	0.703	79.80	73.12
21	5.9464	5.9057	0.829	0.818	85.35	82.06
23	6.1813	6.2790	0.882	0.900	88.27	91.00
25	6.4161	6.6523	0.922	0.951	89.22	99.94

Table IV

Recommended Concentrations Under Different Models (in mg/m^3

Cumulative Prevalence	Life Table Model	Regression Model
0.005	2.26	1.83
0.010	3.09	2.19

For the 532 employees, another equation was given where the variable is working duration, and the dependent variable here is the cumulative asbestos exposure.

$$D = 23.7015 + 13.1195X \quad (11)$$

where D is the expected asbestos exposure, X is working duration.

By combination of equations (10) and (11), the temporal variable working duration will be eliminated, we get equation (12) which reveals the relationship between the cumulative prevalence rate and the cumulative asbestos exposure.

$$D = 58.3668 + 2.9350P \quad (12)$$

Equation (12) could be used to estimate the recommended criteria for asbestos concentration (Table IV).

The recommended concentration is defined in such a way that the probability of asbestosis for the employees who have worked for 40 years under this concentration will not precede 0.5% or 1%.

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The Sanitary and Antiepidemic Station of Chaoyang district of Beijing participated in this work.

THE CORRELATION BETWEEN SILICOSIS AND LUNG CANCER —PATHOLOGICAL EVIDENCES FROM 5 AUTOPSIED CASES

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Recent epidemiologic studies suggested a high risk of lung cancer among workers exposed to silica dust. The Occupational Hospital, an organization to cure workers with silicosis caused by railway-tunnel-building, also found that among these patients the risk of dying from lung cancer was greater than compairson population. This paper reported certain pathological correlation between silicosis and lung cancer based on pathological materials.

ILLUSTRATIVE CASES

Case 1: 53 year-old man, tunnel-building for 9 years, with massive silicotic fibrotic lesions in both upper lobes of lungs. In the subpleural of both base parts of lungs, was found numerous greyish-white nodules, about the size of peas (Figure 1). Microscopically, the bronchiolar epithelium showed obvious hyperplasis, the dialated bronchiolar lumens were full of hyperplastic epithelium. A few of these bronchioles showed anaplastic change and began to invade into surrounding tissues, presenting an early appearance of adenocarcinoma (Figures 2, 3).

Case 2: 58 year-old man, tunnel-building for 34 years, with history of smoking. There is a massive silicotic fibrotic lesion associated with tuberculosis in right upper lobe. The bronchogenic carcinoma also developed in the same lobe (Figure 4). Why did the cancer of bronchus develop in the same lobe? The bronchus near the massive silicotic lesion showed deformation and their epithelium often being destroyed, andd proliferation metaplasia anaplasia ensued, finally squamous cancer developed (Figures 5, 6).

Case 3: 58 year-old man, tunnel-building for 8 years, with history of smoking. In addition to the generally distributed silicotic nodules throughout the whole lungs, the nodules in the left upper lobe showed tendency to coalesce. In the site where the superior and inferior bronchi bifurcated, there was a large silicotic enlarged lymph node 2.3 cm in diameter which oppressed on the superior and inferior bronchi, the cancer developed right there (Figure 7). Microscopically, showed low-differentiated large cell cancer (Figures 8, 9).

Case 4: 60 year-old man, railway-building for 38 years, with history of smoking. In both upper, middle and lower lobes scattered with silicotic nodules. In the middle lobe of the right lung (near the hilar), there was a massive silicotic fibrotic lesion (5×2 cm). In the right hilar region, a bronchogenic large tumor surrounded the bronchuss and obstructed its lumen (Figure 10). Microscopically, that was a low-differentiated small cell cancer (Figures 11, 12).

Case 5: 58 year-old man, tunnel-building for 6 years, with history of smoking. All lobes of both lungs scattered with silicotic nodules. In the upper lobe of left lung there was a (5×4.5×4.5 cm in size) black and white interlacing region.

Figure 1. (Case 1): In the subpleural silicotic collagenous region of base part of right lung was found numerous greyish-white nodules. (forked tail arrowed).

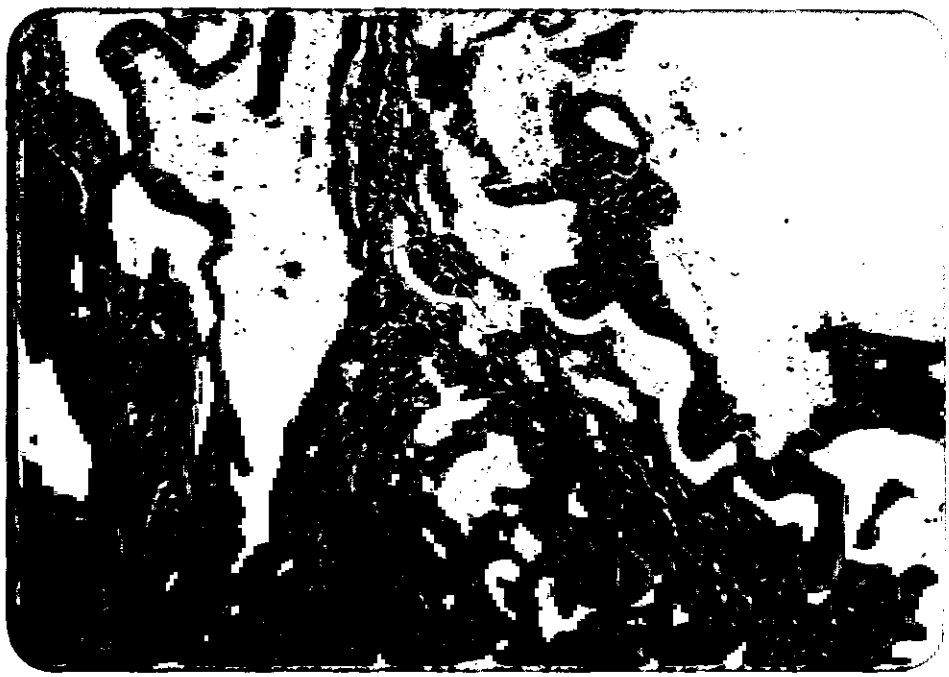

Figure 2. (Case 1): A few of greyish-white nodules present an early appearance of adenocarcinoma (H&E, ×50).

Figure 3. (Case 1): Higher power view of cancer shown in Figure 2 showing poorly differentiated tumour cells (H&E, ×200).

Figure 4. (Case 2): Bronchogenic cancer developed in the same lobe that the massive silicotic lesion existed. (Forked tail arrowed the silicotic lesion; the others arrowed the the cancer and tuberculosis area.)

In that region there was a silicotic fibrotic coalescing lesion in the size of 2×1.5 cm (Figure 13). Microscopically, the white region was cancer. Histologically, it was bronchiole-alveolar cancer (Figure 14). This case associated with tuberculosis too (Figure 15).

DISCUSSION

From the pathological viewpoint, in those 5 cases of silicosis associated with lung cancer, the development of cancer all have some correlation with the preexisting silicosis. In two of those five cases (Cases 2, 5), the cancer developed on the same lobe the massive silicotic lesion existed. In one case (Case 4) the cancer developed in the right hilar bronchus close to the massive silicotic fibrotic lesion. In one case (Case 3) the cancer developed on the site where the left bronchus bifurcated into superior and inferior bronchi oppressed by the silicotic enlarged lymph node. In another case (Case 1) the cancer developed from multiple deformed bronchioles which remained in the subpleural silicotic collagenous region.

Emmanuel Farber[1] put forward that "Dependence on cell proliferation for initiation" of carcinogenesis and "In fact, we think that the rate- limiting step in some types of cancer development, such as in the liver, the urinary bladder, and the pancreas, to name but three, may not be the exposure to a carcinogen but rather the presence or absence of concomitant cell or tissue damege." At silicosis it can be seen that varied silicotic lesions of the lung often caused deformation stenosis of the bronchus and the mucomembrane of bronchus often continuously damaged. It created an important favourable factor for the development of lung cancer. It is well known to us that Stenbeck et al.[1] intratracheally instilled SiO_2 or Hap or SiO_2 together with Hap to the syrian golden hamsters, the lung cancer incidence of the latter group was 4 folds more than the Hap group. Holland et al.[1] made an experiment that Fischer-344 ratsss were exposed to silica (Mun-U-Sil) 6 hours per day, 4 days a week for 24 months at an airborne concentration of 12 mg/m^3. It produced a respiratory epithelium tumor incidence of 27%. Hesterberg et al.[3] cultivated hamster's embryo cells with Min-U-Sil (a high SiO_2 content) showed tumor transmutation.

Holland[1] suggested that in his animal experiment the carcinogenesis of the silica were due to lung scaring produced by silicotic lesion in rats, but in our st u dy only one case (Case 1) belongs to the scar cancer. Saffiotti[3] suggested that the target cell of the silica carcinogenesis is bronchiole-alveolar cell but in our study only one case (Case 5) is bronchiole-alveolar cancer. All others (Cases 2, 3, 4) the cancer developed on the basis of repeat bronchus and their mucomembrane damage caused by the silicotic lesions. The development of those bronchogenic lung cancers corresponded more or less with the pattern of Farber and animal experiment of Stenback.

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Figure 5. (Case 2): The squamous carcinoma developed on the bronchus near the massive fibrotic lesion (H&E, ×50).

Figure 6. (Case 2): The massive silicotic lesion associated with tuberculosis in the same lobe. The alveolar filled with macrophages and Langhan's giant cells and lymphocytes (H&E, ×100).

Figure 7. (Case 3: In the site where the left bronchus bifurcated, there was a large silicotic enlarged lymph node (forked tail arrowed, the white spot is a metastatic focus), which oppressed on the bronchi, the cancer developed right there (single tail arrowed).

Figure 8. (Case 3): Microscopically, this cancer shown in Figure 7 shows an appearance of low-differentiated large cell cancer (H&E, ×100).

Figure 9. (Case 3): The malignant cancer cells spread via perivenous channels (H&E, ×200).

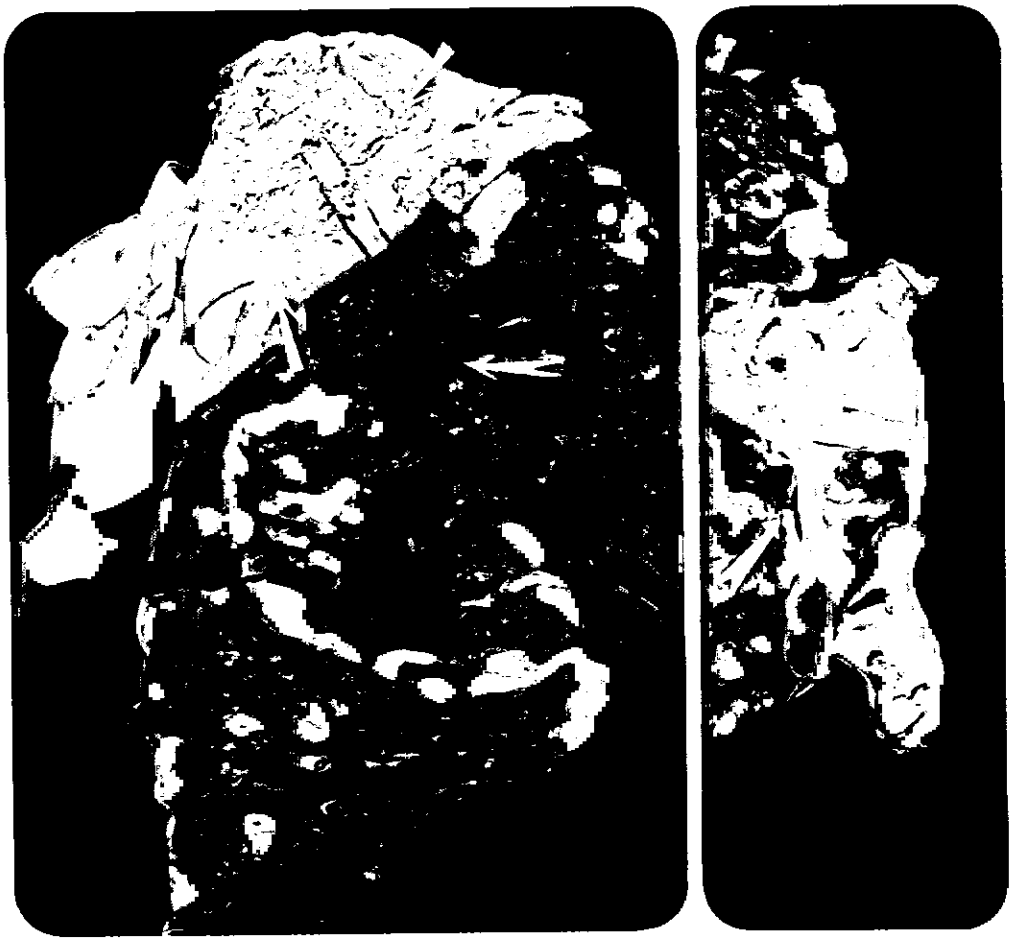

Figure 10. (Case 4): The massive silicotic lesion near the right hilar (forked tail arrowed) and in the hilar region a bronchogenic large tumor developed (single tail arrow). The right photo shows the bronchogenic cancer obstructed the bronchial lumen.

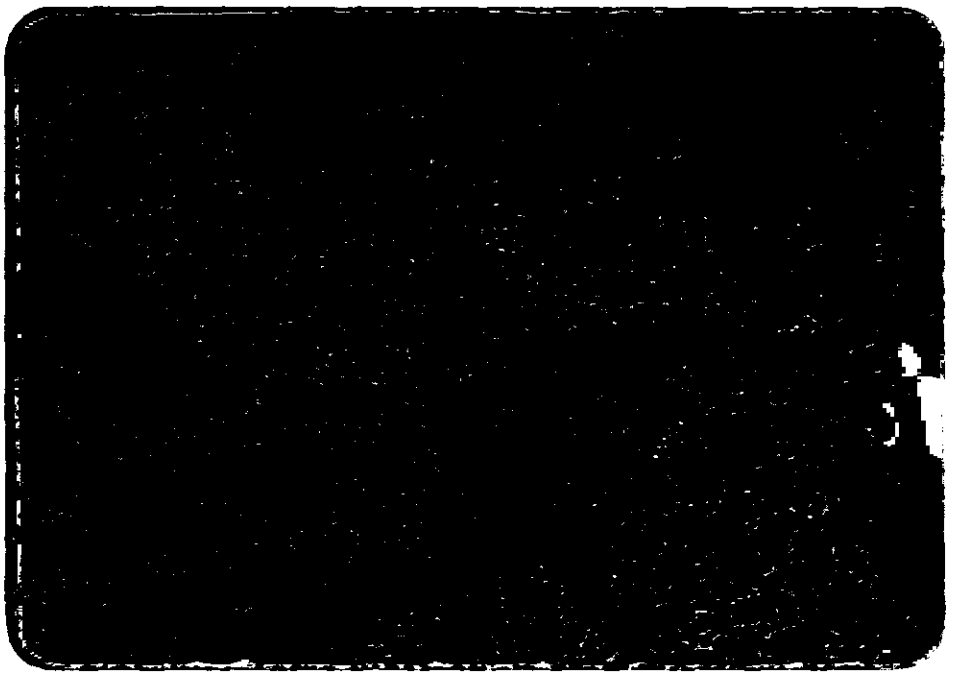

Figure 11. (Case 4): Microscopically, this cancer was a low-differentiated small cell cancer (H&E, ×50).

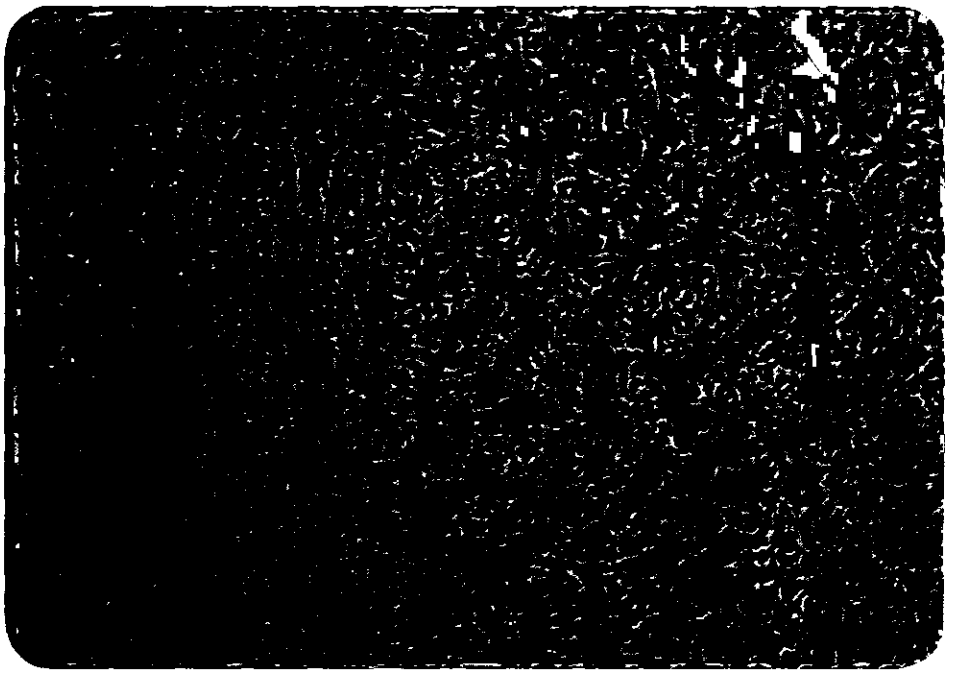

Figure 12. (Case 4): Metastic cancer in the liver shows an appearance of adenocarcinoma (H&E, ×100).

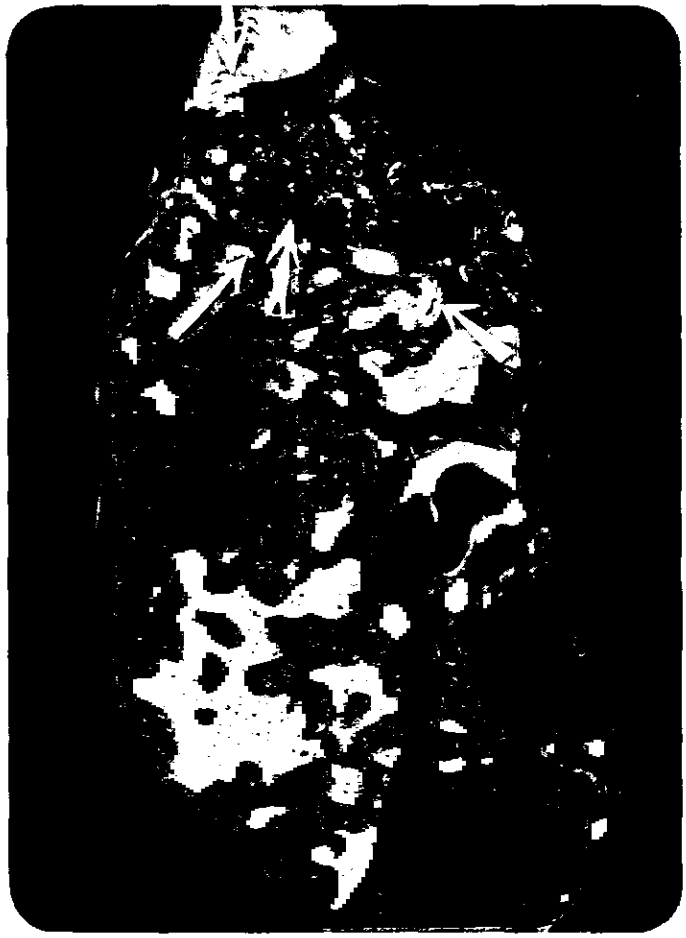

Figure 13. (Case 5): In the upper lobe of left lung there was a silicotic fibrotic lesion in the size 2×1.5 cm (forket tail arrow) surrounded with cancer (single tail arrowed area).

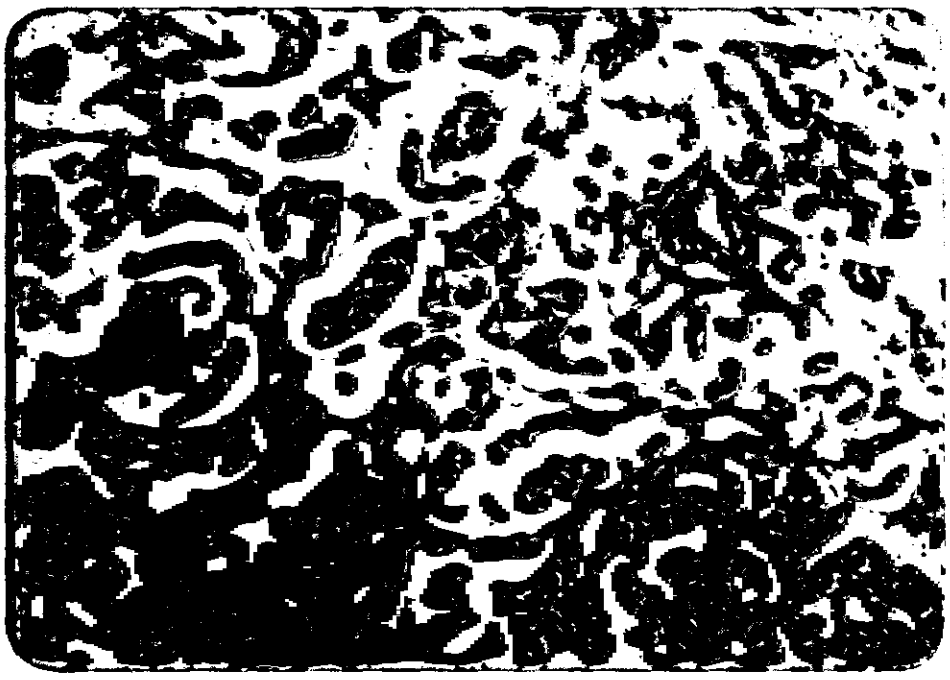

Figure 14. (Case 5): Microscopically, this cancer was a bronchiole-alveolar cell cancer (H&E, ×100).

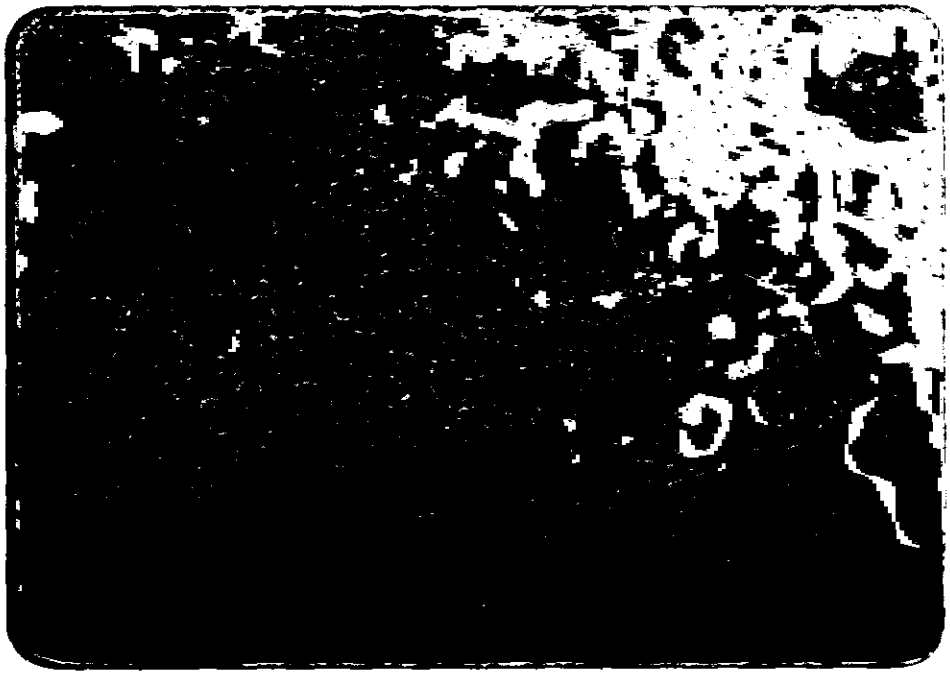

Figure 15. (Case 5): This case associated with tuberculosis too. Note the tubercles with caseation (H&E, ×50).

BEHAVIORAL TOXICITY IN SILICOTIC PATIENTS (Pilot Study)

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Silicosis is the most common and most severe occupational disease in China. Most silicotic patients complain of amnesia, concentration difficulties and psychomotor slowing, etc. Up to now these problems have not drawn sufficient attention in China. In order to explore the neuropsychological impairment in silica dust-exposed workers, the Wechsler Adult Intelligence Scale—Revised in China (WAIS-RC)[1] and some of the tests on the Wechsler Memory Scale—Revised in China (WMS-RC)[2] were used. A summary of the use of this battery in detecting the behavioral effects of silicon on silicotic patients is given.

SUBJECTS

Eight men and one woman exposed to silica dust were included in our study. All of these cases were exposed to silica dust for at least 4 years and had been diagnosed as silicosis by the panel of experts authored by national health ministry and Sichuan provincial government. The ages of the cases were from 41 to 68 (55.33 ± 10.56 years). All the cases had grown up in the countryside and had received 1 to 6 years (4 ± 2 years) education. Details of the study subjects are summarized in Table I.

Table I
Details of the 9 Patients with Silicosis

Number of patient	Sex	Age (year)	Education received (yrar)	Duration of exposure to silica dust (year)
1	male	61	6	24
2	male	64	1	20
3	male	47	4	4
4	male	41	5	4
5	male	68	3	22
6	female	62	1	4
7	male	52	6	6
8	male	42	6	15
9	male	43	4	10
		53.33±10.56	4 ± 2	11.89±8.49

RESULTS

The detailed results of neuropsychological tests are shown in Table II. Almost all the cases got full scores on experience and orientation subtests of WAIS-RC. Seven of nine cases (77.8%) got only zero scaled score on the test of counting from 1 to 100. And none of the cases got more than 7 scaled scores on the test of reversing from 100 to 1. No one could completely recall the short story and no one could get more than 8 scaled scores. There were more than 5 cases (55.6%) failed to perform some subtests on WAIS-RC, with scores lower than $\bar{X} - D$ according to age-appropriate norms.

DISCUSSION

Unfortunately, we haven't finished the whole controlled trial, so we cannot statistically process the data. The results suggested that patients with silicosis suffered from some impairment of short-term memory, but remote memory was spared. The poor performance on subtests of reversed counting (from

Table II
Results of Behavior Tests on 9 Patients

Variable	Subjects								
	1	2	3	4	5	6	7	8	9
experience	5	4.5	4	5	4.5	4	5	5	5
orientation	5	5	5	5	5	5	5	5	5
1--100 (scaled	0	0	8	0	0	0	0	0	3
100--1 score)	4	6	6	0	3	2	4	1	7
short story	6	4	6	1.5	2	2	11	5	2
digit span									
forward	6	6	7	9	8	8	7	7	6
backward	3	3	4	3	3	3	5	6	4
information	14*	11*	16	11*	8*	9*	11*	19	16
comprehension	13*	16	14	15	10*	14*	16	15	14*
arithmetic	8*	11	8*	11*	11	7*	9*	14	8*
similarity	6*	4*	9	3*	2*	2*	5*	18	8*
vocabulary	38	34	14*	36	21	21*	28*	71	38
digit symbol	32	23	19*	19*	25	10*	14*	36	27*
picture completion	9	7*	8*	9*	5*	5*	5*	14	6*
block design	14*	28	20*	26	26	22*	24	26	18*
picture arrangement	20	12	8*	18	8*	4*	16	24	4*
object assembly	10*	22	21	21*	9*	13*	25	24	29

* less than $\bar{X} - D$

100 to 1) and digits backward gave us a deep impression that the mental tracking ability was markedly impaired. Especially when we compare the scores of digits backward with those of digits forward, the difficulties of attention and concentration will be more obvious. All the cases achieved a forward span of 7 or more but recalled much fewer digits reversed, which meant the patient's mental tracking deficits were much more severe than verbal memory difficulties. When doing arithmetic, all the cases asked us to repeat questions for 2 to 9 times (4.78 ± 2.64). We think the lower scores on arithmetic are partly due to attention difficulties and short-term memory disorders.

We found the patients did serial counting from 7 to 100 very slowly and most of them got only zero scaled score. We believe this kind of operation measures speed of response rather than long-term memory. Because digit symbol test is the one most likely to be sensitive to psychomotor slowing,[3,4] the poor performance of this test indicates some impairment in this aspect. Although the results of other tests showed some impairment, we can't confirm their significance because of lack of controlled group.

According to this pilot study, we could conclude that exposure to silica dust can produce chronic toxic effects on human behavior, which are quite similar to those resulting from organic solvents.[5,6] We hope the investigation being done by us will give a more clear and definite conclusion.

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SIMILARITIES IN LUNG CANCER AND RESPIRATORY DISEASE MORTALITY OF VERMONT AND NEW YORK STATE TALC WORKERS

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ABSTRACT

The risks from malignant and non-malignant respiratory deaths of New York State and Vermont State talc workers with at least one year of employment have been compared for both miners and millers. The mortality patterns are similar. In both areas, the talc miners have a 4.5 fold risk of lung cancer, and the talc miners have no increased risk of lung cancer. In both areas, all workers appear to have an increased risk of non-infectious, non-neoplastic respiratory disease (NNRD) mortality, although only the Vermont millers show a statistically significantly elevated risk (7.9 fold). Thus, although the New York talc has been described as asbestiform talc and the Vermont talc as non-asbestiform talc, the mortality patterns of the workers appear to be inconsistent with that classification in that their lung cancer mortality rates are no different and only the Vermont talc millers show a significantly increased NNRD mortality.

INTRODUCTION

Studies of talc miners and millers in the New York and Vermont talc industry include analyses of mortality, morbidity, industrial hygiene, and mineralogy. Mineralogical differences between the two talcs have been highlighted. The upstate New York talc contains an elongated particulate not found in the Vermont talc that is considered by scientists at the National Institute for Occupational Safety and Health (NIOSH) as tremolitic asbestos and by scientists at the Bureau of Mines and at the company that owns the plant as true talc particulates and as prismatic non-asbestiform tremolite. NIOSH has called the New York State talc asbestiform talc and the Vermont talc non-asbestiform talc. Leaving the question of the mineralogical label of these particulates to the mineralogists, we have elected to examine the respiratory health outcomes of the employees at these two talc industries.

MATERIALS

The initial shaft of the New York State talc plant was sunk in 1947. Mining and milling operations started in 1948. The mortality experience (1947 through 1978) of all persons hired at the plant between 1947 and 1977 has been reported.[1] Mortality analysis was restricted to the 705 male employees (all caucasian). None of the 36 women employees had died of a respiratory condition. Sixty percent of the men worked at the plant for at least one year; twenty percent for two months to one year; and twenty percent for less than two months. Mortality analysis was reported separately for the 280 white male employees employed at the talc plant for less than one year and for the 425 white male employees employed for at least one year. That report[1] suggested that prior employment jobs accounted for the lung cancer rate.

In-plant job records and prior employment histories on the job applications were analyzed. Employees were classified from the inplant job records as miners (187 worked exclusively in the mine), millers (152 worked exclusively in the mill), and others (34 worked in both the mine and the mill, 11 worked neither in the mine or the mill, and 41 had uninformative records).

The cohort of white male employees of the Vermont talc industry was developed from the records of the Vermont State Health Department's annual radiographic survey of employees of the dusty trades, begun in 1937. Selevan et al. of the National Institute for Occupational Safety and Health (NIOSH) defined the Vermont talc study cohort[2] as all white males in the Vermont talc industry on or after January 1, 1940 with at least one year of talc employment prior to January 1, 1970. Individuals who had at least two radiographs in the file and who had worked for any of five talc companies in three geographic areas of Vermont were eligible for the study. Mortality follow-up was continued through 1975 of the 392 men determined to belong to the cohort.

Health Department and company records were scrutinized to determine their job assignments, and each cohort member was classified as a miner after having had one year of exposure in the mine and/or as a miller after having had one year of exposure in the mill. 225 workers were classified as miners; 163 workers were classified as millers (of whom 47 had also been classified as miners); and 51 were not classifiable.

METHODS

This report compares standardized mortality ratios (SMRs)

for malignant and non-malignant respiratory causes of death for miners and millers with at least one year of experience in the Upstate New York talc (said to be asbestiform) industry with those in the Vermont State talc (said to be non-asbestiform) industry. Comparison is reasonable, despite the differences in classification variables between the two studies.

RESULTS

The risks of lung cancer and of non-infectious, non-neoplastic respiratory disease (NNRD) for employees with at least one year in the mines or mills of New York State or Vermont State talc industries are presented, analyzed, and discussed below.

Respiratory Mortality of New York and Vermont Talc Workers

	Observed/Expected Ratios		Standardized Mortality Ratios	
	New York	Vermont	New York	Vermont
Lung Cancer				
Millers	1/1.41	2/1.96	071	102
Miners	5/1.15	5/1.09	460*	435*
Others	0/0.55	0/0.61	---	---
Total	6/3.11	7/3.66	1.92	1.91
NNRD				
Millers	2/0.74	7/0.89	270	787*
Miners	2/0.49	2/0.56	408	357
Others	2/0.38	2/0.34	526	588
Total	6/1.61	11/1.79	373*	615*

* = p<0.05, two-tailed Poisson test

The risk of malignant disease of the lung (lung/respiratory cancer) is not increased for millers but is significantly increased (4.5 fold) in talc miners both in New York (4.60) and in Vermont (4.35). No difference in risk is seen between miners and millers of New York and of Vermont (Figure 1). These data are sufficiently strong to rule out with eighty percent confidence an underlying relative risk for New York miners vs. Vermont miners of 1.7 and with about ninety five percent certainty an underlying risk of greater than 2.0.

The risk of non-malignant respiratory disease (excluding pneumonia and influenza), i.e., NNRD has a significantly increased risk (almost eight-fold) for Vermont talc millers but not for New York talc millers (risk of 2.7, not significant). The risks for NNRD for miners are calculated to be 4.1 and 3.6 (both non-significant) for those from New York and Vermont, respectively (Figure 2).

As for other respiratory system deaths, influenza or pneumonia caused the death of one New York State talc worker (0.9 expected) but no Vermont talc miner (0.7 expected) or miller (0.8 expected). Mesothelioma caused the death of one New York State talc man (15 years after hire which followed 28 years in mining and construction) and of one Vermont talc man.

DISCUSSION

We have attempted to assemble similarly defined cohorts of New York State and Vermont State talc workers in order to compare the respiratory mortality risks of their miners and millers. The exposures of millers generally exceed that of miners by a factor of two to six. Nonetheless, both groups demonstrate a similar excess lung cancer risk only for their millers and not for their miners. The similar lung cancer risks of the two groups of talc workers exposed to the differently described talcs suggest that the elongated particulates seen in the New York State talc have not introduced an increased lung cancer risk. We further observe that the risk of non-infectious, non-neoplastic respiratory death, while apparently increased in all groups, is significantly elevated only among the Vermont millers.

Standardized mortality ratios (SMRs) were calculated for each group based on age-specific, calendar time-specific, cause-specific mortality rates for white males. The New York State study SMRs had been calculated using U.S. rates with death certificates coded according to the eighth revision of the International Classification of Diseases (ICD). The Vermont State study SMRs were first calculated using U.S. rates and then recalculated by its authors using Vermont State rates for non-malignant respiratory disease and respiratory cancer

Figure 1. Respiratory or lung cancer mortality risk for miners and millers of New York State and Vermont State talc.

Figure 2. Non-infectious, non-malignant respiratory disease (NNRD) mortality risks for miners and millers of New York State and Vermont State talc.

COMPARATIVE LUNG MORTALITY RISKS of VERMONT and NEW YORK STATE TALC WORKERS with at least one year experience at Talc Plant

	Vermont		New York		New York		New York	
	Ever employed						< One Year	
All Emp >1 yr.	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR
All Causes	44/37.15	118	64/49.83	128	118/83.58	141*	54/33.75	160*
All Cancers			15/9.55	157	26/15.7	165*	9/6.15	146
Lung Cancer	6/3.61	163	6/3.11	193	12/5.01	240*	6/1.90	316*
NNRD	11/1.79	615*	6/1.61	372*	6/2.64	227	0/1.03	---
Millers								
All Causes			20/21.74	92	35/30.97	113	15/9.23	163
All Cancers			3/4.23	71	6/5.94	101	3/1.71	175
Lung Cancer	2/1.96	102	1/1.41	71	1/1.92	52	0/.51	---
NNRD	7/.89	787*	2/0.74	270	2/1.02	196	0/.28	---
Miners								
All Causes			31/16.76	185*	50/26.32	190*	19/9.56	199
All Cancers			10/3.23	310*	15/5.00	300*	5/1.77	282
Lung Cancer	5/1.15	435*	5/1.09	460*	9/1.66	543*	4/0.57	701*
NNRD	2/0.56	357	2/0.49	408	2/0.77	260	0/0.28	---
Others								
All Causes			13/11.33	115	33/26.29	126	20/14.96	134
All Cancers			2/2.09	96	5/4.76	105	3/2.67	112
Lung Cancer	0/0.55	---	0/0.61	---	2/1.43	140	2/0.82	244
NNRD	2/0.34	588.0	2/0.38	526	2/0.85	235	0/0.47	---

LUNG CANCER

COHORT DEFINITION

Cohort Variable	NEW YORK	VERMONT
Gender	Male	Male
Race	White	White
Employment Dates	1947-1977	1940-1969
Employment Duration	One Year +	One Year +
Mortality Dates	1947-1978	1940-1975

Cohort Numbers

Miners	152	163
Millers	187	225

NEW YORK VERMONT

Observed/Expected

Millers	1/1.41	2/1.96
Miners	5/1.15	5/1.09
Others	0/0.55	0/0.61

SMR

Millers	71	102
Miners	**460**	**435**
Others	---	---

4/7/88

COMPARATIVE LUNG MORTALITY RISKS of VERMONT and NEW YORK STATE TALC WORKERS with at least one year experience at Talc Plant

Ever employed

< one Year

All Emp >1 yr. LATENCY (Years)	Vermont O/E	Vermont SMR	New York O/E	New York SMR	New York O/E	New York SMR	New York O/E	New York SMR
0-4			0/0.27	---	0/0.42	---	0/0.15	---
5-9			0/0.31	---	0/0.49	---	0/0.18	---
10-14			1/0.45	224	1/0.69	145.0	0/0.24	---
15-19			2/0.60	331	2/0.98	205.0	0/0.38	---
20-24			3/0.79	378	8/1.29	623*	5/0.50	1000
25-29			0/0.65	---	1/1.09	92.0	1/0.44	227
30+			0/0.04	---	0/0.05	---	0/0.01	---
Total			6/3.11	193	12/5.01	240*	6/1.90	316*
0-9			0/0.58	---	0/0.91	---	0/0.33	---
10-19			3/1.05	285	3/1.67	180	0/0.62	---
20-29			3/1.44	208	9/2.38	378*	6/0.94	638*
30+			0/0.04	---	0/0.05	---	0/0.01	---
Total			6/3.11	193	12/5.01	240*	6/1.90	316*
0-4			0/0.27	---	0/0.42	---	0/0.15	---
5-14			1/0.76	132	1/1.18	85	0/0.42	---
15-24			5/1.39	360*	10/2.27	441*	5/0.88	568*
25+			0/0.69	---	1/1.14	87	1/0.45	222
Total			6/3.11	193	12/5.01	240*	6/1.90	316*

4/7/88

COMPARATIVE LUNG MORTALITY RISKS of VERMONT and NEW YORK STATE TALC WORKERS with at least one year experience at Talc Plant

		Vermont		New York	
		O/E	SMR	O/E	SMR
All Causes	Emp >1	44/37.15	118.0	64/49.83	128
All Cancers	Emp >1			15/9.55	157
Lung Cancer	Emp >1	6/3.61	163	6/3.11	193
NNRD	Emp >1	11/1.79	615	6/1.61	372
Pneumonia/Influ	Emp >1	0/1.89	000	1/0.9	109
All Causes	Millers			20/21.74	92
All Causes	Miners			31/16.76	185
All Causes	Others			13/11.33	115
All Cancers	Millers			3/4.23	71
All Cancers	Miners			10/3.23	310
All Cancers	Others			2/2.09	96
Lung Cancer	Millers	2/1.96	102	1/1.41	71
Lung Cancer	Miners	5/1.15	435	5/1.09	460
Lung Cancer	Others	0/0.55	---	0/0.61	---
NNRD	Millers	7/.89	787	2/0.74	270
NNRD	Miners	2/0.56	357	2/0.49	408
NNRD	Others	2/0.34	588	2/0.38	526
4/7/88	Bold =	p <0.05			
Pneumonia/Influ	Millers	0/.83	000		
Pneumonia/Influ	Miners	0/.67	000		
Pneumonia/Influ	Others	0/.39	000		

Standardized Mortality Ratios

	Vermont	New York
Lung Cancer		
Millers	102	71
Miners	435	460
Others	---	---
NNRD		
Millers	787	270
Miners	357	408
Others	588	526

NON-INFECTIOUS, NON-MALIGNANT RESPIRATORY DISEASE

	NEW YORK	VERMONT
Observed/Expected		
Miller	2/0.74	7/0.89
Miners	2/0.49	2/0.56
Others	2/0.38	2/0.34
SMR		
Millers	270	787
Miners	408	357
Others	526	588

with death certificates coded according to the seventh revision of the ICD. This report bases the SMRs on the U.S. rates.

The New York State study reports lung cancer as their measure of malignant respiratory disease and NNRD (non-infectious, non-neoplastic respiratory disease) as their measure of non-malignant respiratory disease. The Vermont State study reports respiratory cancer as their measure of malignant respiratory disease and ONMRD (other non-malignant respiratory disease) as their measure of non-malignant respiratory disease. Both NNRD and ONMRD are terms for total non-malignant respiratory disease, excluding influenza and pneumonia. We have used the labels of lung cancer and NNRD to represent the malignant and non-malignant respiratory disease measures.

Twelve of the thirteen respiratory cancers among the New York State talc workers were lung cancers. The thirteenth case was a man whose five years at the plant included three months as a laborer/oiler in the talc mill and ended with death from mediastinal cancer. Re-analysis of the New York State data as respiratory cancer rather than lung cancer would have reduced the SMR estimates by about 5% but not have altered the comparison between the miners and millers. Both the New York and the Vermont data are compared against U.S. mortality rates.

The Vermont data included persons with experience in both the mine and the mill in each category; the New York data separated them out. There were only 34 such New York workers with experience in both the mine and the mill. Less than 0.1 lung cancer and less than 0.1 NNRD deaths were expected among them, and none were observed. Including this group among the miners and the millers of New York State would not have affected the results.

Studies of both cohorts lack full information on smoking history. Each indicates that most of the lung cancer cases were known to be cigarette smokers, but data on smoking appears to be inadequate for both cohorts. There is no evidence that miners and millers differ in their smoking habits. Thus, it is unlikely that the differences observed in these comparisons could be due to differences in smoking between groups.

The mortality of the experienced employees of the New York and Vermont cohort who worked other than in the mine or the mill for a year were also examined. There were no lung cancer deaths. Each group had two NNRD deaths, yielding non-significant risks of 5.9 for those from Vermont and 5.3 for those from New York.

While the NNRD mortality may be due to dust exposures at the talc plants, the etiology of the lung cancer is less clear. The NIOSH authors[2] concluded that talc dust was unlikely to be the cause of the respiratory cancer, since the risk was seen only in the miners and not seen among the millers, a group with probable higher dust exposure. Radon daughter measurements in the New York mine do not explain the finding. The presence of a particulate in New York dust and not in Vermont talc dust cannot explain the difference.

The CEOH study[1] had supported the hypothesis of risk from prior employments as the explanation for the lung cancer risk of the New York State talc workers, however, that hypothesis has not been examined for the Vermont talc workers. Further study of both cohorts should be undertaken to explain the mortality patterns seen. The small number of cases in either group will probably be a hindrance to a full and clear explanation. Both cohorts should probably be extended to include later employees and the period of follow-up should be brought more current by at least a decade. A four-fold risk of lung cancer seen in two different studies of talc miners (but not millers) cries for an explanation.

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