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Mortality in the Chrysotile Asbestos Mines and Mills of Quebec

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Of 11,788 persons born between 1891 and 1920 employed in the Quebec asbestos mining industry, 88.4% were traced. Of these 2,457 (23.6%) had died. Exposure indexes for each worker were calculated from job dust levels and duration of employment. The overall mortality was lower than expected for the population of Quebec but in the highest dust category, comprising 5% of the cohort, the age-standardized rate was 20% higher than in the other groups. Respiratory, cardiovascular, and malignant disease in equal proportions accounted for the excess. There were 101 deaths from respiratory cancer including three from malignant mesothelioma, an estimated excess of about 15 deaths. The difference in rates for respiratory cancer between those maximally and minimally exposed was fivefold and, though perhaps exaggerated, was apparently determined by accumulated dust exposure and duration of employment.

THE REMARKABLE qualities of the asbestos group of fibrous minerals have been recognized since antiquity, but mining and milling on an industrial scale began only at the end of the 19th century. In the Eastern Townships region of Quebec, deposits of chrysotile asbestos in serpentine rock were noted in the 1847 Canadian Geological Survey. The first mine was opened at Thetford in 1878, and within 30 years the region was producing most of the world's asbestos. The proportion fell as Russian, South African, and Italian mines came into operation, but Quebec still produces about 40% of the world's supply, now estimated at about 4 million tons a year.[1]

There are two main mining areas, one at Thetford Mines and neighboring towns of Black Lake and Broughton, and the other at Asbestos. The Thetford area was developed by many different companies, but with amalgamation the number has now been reduced to six. At Asbestos, the mining has been carried out since 1882 by one large company which also operates a small factory in the town for the manufacture of mixed asbestos products. There is a small mine owned by another company a few miles away.

Concern for the health effects of asbestos has paralleled growth in its production, and the main available evidence was reviewed fully by Wright[2] in 1969. The first cases of diffuse pulmonary fibrosis after prolonged exposure were noted by Murray in 1907, and by 1930 asbestosis was recognized as an important occupational hazard. Controls introduced since then have reduced considerably the dust concentration to which workers in the mines, mills, and primary manufac-

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turing industries are exposed. Though the prevention of asbestosis is far from complete in these industries, it is still generally believed that this can be achieved by more strict environmental control. Another aspect of the problem appeared in 1947 when Merewether[2] showed a link between asbestos exposure and lung cancer. The potential extent of this hazard was increased by reports of Wagner and others[1] during the last ten years that malignant tumors of the pleura and peritoneum are related to certain types of industrial work and perhaps also to neighborhood exposure.

The association between lung cancer and asbestos was first found in the British textile industry. Merewether's inquiry showed an unduly high proportion of lung cancers at autopsy in cases of asbestosis, and other observations among textile workers have confirmed the association.[3-5] Two studies in particular which take some account of the degree of exposure suggest a dose-response relationship. Knox et al[6] found a considerable excess of lung cancer in persons exposed before 1933 when asbestos control regulations were enforced, but none in persons exposed for ten years or more in the most dusty areas since then. These findings must be reviewed when a longer period of observation has elapsed, since Newhouse[3] (also in a textile factory) found an excess of lung cancer in persons heavily exposed 20 years earlier for periods of less than two years. Workers exposed to light or moderate dust concentrations showed no excess of bronchogenic cancer even after 20 years of exposure.

Selikoff and his colleagues[7] found an eightfold increase in lung cancer compared with national figures in a cohort study among the members of an American insulation workers union who had been exposed for 20 years or more. He also found a gross excess of pleural and peritoneal mesotheliomas. On the other hand, in a national survey of all known mesothelial tumors in Canada, 1960 to 1968, McDonald and her colleagues[10] found a history of occupational contact with asbestos in a relatively small proportion of cases. This exposure was mainly in insulation and allied trades, rather than in the asbestos-producing industry.

Of the four main types of asbestos fiber—chrysotile, crocidolite, amosite, and anthophyllite, the two textile factories referred to above[6,8] used mainly chrysotile fiber, together with some crocidolite, and in one of the two, some amosite. As in most industrial applications different types of fibers are mixed, the carcinogenic effects of a single fiber type in practice can only be studied in mining and milling. Chrysotile fiber is of greatest importance because of its qualities and extensive usage. The only studies of lung cancer in chrysotile miners and millers were reported by Braun and Truan in 1958[11] and by Kogan et al in 1966.[12] In the first of these, nine deaths from lung cancer were observed in the Quebec industry compared with six expected from provincial rates. The second was in the Soviet Union and showed that compared with the general population the mortality from lung cancer was increased by a factor of 1.9 for miners, 3.1 for millers, and 2.3 for factory workers. The present investigation forms part of a comprehensive epidemiological survey of the entire Quebec asbestos-producing industry since its inception. Using the considerable volume of data available, our primary aim has been to define as accurately as possible the quantitative relationship between exposure to chrysotile asbestos and the incidence of lung cancer. The results from parallel studies concerned with the relationship of dust exposure to radiographic appearances, pulmonary function, and respiratory symptoms will be reported separately.

Materials and Methods

Registration.—A register was compiled in the personnel department of each asbestos mining company in the Eastern Townships region of Quebec listing all persons currently or previously employed, as of Nov 1, 1966. A card was made out for each employee on which was recorded his name, date of birth, address, and a detailed work history which included the department and mine, dates of starting and finishing, for every job, and all periods of leave.

In one of the 44 mining companies represented in our survey, the records of at least 560 persons had been destroyed. These were of ex-employees and others not transferred when ownership of the company changed in 1964. The work histories of a small number of older men who had been employed partly in this company and partly in others were also incomplete.

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Table 1.—*Subjects of Study and Results of Tracing by Age and Sex*

Year of Birth	Persons			Traced			% Traced	Dead	% of Persons Traced
	M	F	Total	M	F	Total			
1891-1895	1,553	14	1,567	1,179	13	1,192	76.1	611	51.3
1896-1900	1,982	31	2,013	1,588	28	1,616	80.3	655	40.5
1901-1905	2,027	50	2,077	1,724	45	1,769	85.2	498	28.2
1906-1910	1,897	99	1,996	1,741	92	1,833	91.8	330	18.0
1911-1915	1,837	117	1,954	1,773	113	1,886	96.5	221	11.7
1916-1920	2,027	154	2,181	1,976	149	2,125	97.4	142	6.7
All	11,323	465	11,788	9,981	440	10,421	88.4	2,457	23.6

Table 2.—*Subjects of Study and Results of Tracing by Dust Index and Years of Employment*

Years		Dust Index						
		<10	10-	100-	200-	400-	800-	All
<1	No.	3,043	654	38	3	0	0	3,738
	% traced	76.6	75.1	76.3	66.7	...	...	76.3
1-	No.	1,025	1,975	450	264	122	64	3,900
	% traced	86.3	88.6	89.3	90.5	91.0	90.6	88.3
10-	No.	55	1,117	566	522	432	255	2,947
	% traced	100.0	99.0	98.9	99.0	99.3	99.6	99.1
30-	No.	0	204	181	250	303	265	1,203
	% traced	...	100.0	100.0	100.0	100.0	99.6	99.9
All	No.	4,123	3,950	1,235	1,039	857	584	11,788
	% traced	79.9	89.9	94.9	97.0	98.4	98.6	88.4

There was of course some movement of labor from one mining company to another. This necessitated bringing together and matching the records of all the companies, to obtain complete work histories. In the course of tracing ex-employees, the matching process could often be confirmed or amended. In all, 27,669 men and women were registered, including 6,415 currently employed on Nov 1, 1966. Of this total, 1,039 persons had been employed by more than one company.

Using the occupational histories, a list was compiled of all named jobs in each company from the beginning of its operation. A description of each of the 13,346 jobs listed in this way was obtained from existing evaluation systems and by interviewing employees with long service. Jobs with several names but involving the same work and dust exposure were combined and the codes finally needed for classification thus reduced to 5,783. For each of these and for each year of operation, the average dust exposure was estimated on a 13-point scale. Throughout the industry in the dryers, crushers, and mills, the concentrations of dust at working places had been measured annually since 1949 by Maurice Lachance, Eng, and since 1946 by various investigators in the small factory. All measurements were made with either a midget impinger or Greenburg-Smith, impinger and recorded in millions of dust particles per cubic foot (mpcf). Levels of exposure in mining and maintenance jobs and in other operations where no dust measurements had been made were estimated by investigating present-day dust levels and adjusting these according to changes in operating procedure reported by mining and maintenance personnel. A full description of geological and environmental features and of the methods and results of dust measurement are to be published by G. W. Gibbs, MSc, and Mr. Lachance.

Before 1949, few dust measurements were recorded, and estimates of exposure for that period are, therefore, only approximate. Interviews with employees of long service enabled comparisons of dustiness to be made with conditions since 1949. The dates of installation or modification of dust control systems were also taken into account.

Duration of exposure for each person was expressed in years or fractions of years after subtraction of all periods of leave, and was corrected to a working week of 40 hours. A dust index was calculated for each employee by adding together the products of time spent on each job and estimated average dust concentration. For example, suppose the dust index for a man was 600: this might mean that he had worked for five years at 40 mpcf, 15 years at 20 mpcf, and 20 years at 5 mpcf (ie, 200 + 300 + 100). It would be useful if it could be assumed

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that an index of 600 were also equivalent to a working life of 40 years at 15 mpcf, but this would entail assumptions which may or may not be justified.

Selection of Cohort.—The study of mortality was based on employees in an age group thought likely to yield the most valuable information. The cohort selected comprised 11,788 persons born between 1891 and 1920 inclusive who had been employed for one calendar month or more. Those born after 1920 would have had periods of exposure well short of a working lifetime and would still not have reached an age of high mortality. Those born before 1891 would have been very difficult to trace, and, as mentioned above, some of their work histories were incomplete or missing. The selected cohort comprised among others 1,203 persons who had worked for 30 years or more, 3,738 who had worked for less than a year, and 1,080 who had worked for more than a year but whose job had not involved any appreciable exposure to dust. Thus, there was a substantial group with minimal exposure available for comparison with others who had been heavily exposed.

Tracing and Ascertainment.—The search for ex-employees was mainly initiated in the mining towns. Telephone or postal inquiries were made first to establish whether the person in question was alive or dead on Nov 1, 1966. If reported dead, the exact date and place was sought from relatives, friends, and parish registers. For those not traced by these means, a systematic search was made in the provincial death records and certificates, and about 200 persons were found in this way. In addition, a search on our behalf was made in the index of the Canadian Unemployment Insurance Commission for any person not found by means of local enquiries. The names of 598 persons were found who had registered or re-registered between 1964 and Nov 1, 1966, and 30 more had re-registered after that date. All 628 were presumed to be alive for the purpose of our study.

Information was obtained concerning 10,421 (88.4%) of the 11,788 persons in the cohort: 7,965 were still alive (including 2,484 currently employed) and 2,457 (23.6% of those traced) were reported dead. Copies of death certificates were obtained from Canadian Provincial and US State Registrars for 2,211 (90%) persons. For 59 more (2%), an acceptable cause of death was obtained by other means: 30 were accidental deaths in the mines or during active service with the armed forces and 29 were caused by disease. Twenty-two of the 29 had occurred before the introduction of death registration in Quebec in 1926, and the cause of death was stated by relatives. In the other seven, the cause of death was obtained from a reliable source. A death certificate could not be obtained for the remaining 187: 47 had occurred before 1926, and 100 were deaths outside Quebec, mostly outside Canada. The cause of death was coded according to the Seventh Revision of the International Classification of Disease (ICD [World Health Organization, Geneva, 1957]). This was done by senior coder of the Department of Demography of Quebec who had recently retired.

The proportion of subjects traced by age, sex, and category of exposure is shown in Tables 1 and 2. Success in tracing depended mainly on duration of employment: 94% of those employed for one year or more and over 99% of all those employed for ten years or more were traced. Information was least satisfactory in persons born before 1900 and in those employed for less than one year. The principal reasons for failure were (1) no relative or friend could be located in the neighborhood and (2) insufficient identifying information, which would have enabled us to make a request to the Unemployment Insurance Commission or to locate a death certificate in Quebec. There were no other obvious reasons for bias towards discovering the living or the dead, but those traced dead or alive are likely to have been long-term rather than transient residents.

Diagnosis of Lung Cancer.—To make our figures for lung cancer as accurate as possible, we investigated and reviewed all certified cases and searched also for cases not described as such on the death certificate. Of 95 deaths coded as due to lung cancer (ICD 162 and 163), one was certified as due to a hydronephroma and another as due to pulmonary fibrosis, both of which had been coded incorrectly. Inquiries about the remaining 93 cases were made from hospitals, physicians, and pathologists, and information was received in 76. In five of these it was reported that there had been an autopsy, but we were unable to obtain pathological details. In another five, though the diagnosis had been made in the hospital, no clinical or pathological record was found. Seventeen more had been certified by family physicians but we did not succeed in finding out where and how the diagnosis had been made.

In the remaining 48 cases, pathological reports were obtained, 25 at autopsy and 23 at biopsy. A diagnosis of carcinoma of the trachea was made in one, of bronchus or lung in 44, and of malignant mesothelioma of pleura in two. In one case the histology was that of a melanosarcoma, and the pulmonary tumor was thought to be metastatic. Thus, of 95 certified cases, three

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had been included incorrectly, 47 were confirmed pathologically, 28 hospital diagnoses were presumed correct, and in 17 no further information was obtained.

The following steps were taken to discover cases of lung cancer that had not been mentioned as such on the death certificate:

1. A letter was sent to the certifying physicians whenever it was stated that an autopsy had been or would be performed. These numbered 306 in all; 33 deaths were certified as due to lung cancer and 273, as due to other causes. In 18 replies, it was stated that there had been no autopsy; in 175, that the certified diagnosis was confirmed at autopsy; and in 18, that it was incorrect. These 18 yielded five additional cases of lung cancer. The diagnoses for these had been certified as renal tumor (ICD 180), infectious hepatitis (ICD 092), pulmonary fibrosis (ICD 519), diabetes (ICD 260), and asbestosis (ICD 523).

2. We asked the physicians in charge of the industrial clinics at Thetford Mines and Asbestos for lists of all deaths known to be due to lung cancer. Their records included only employees who had given up work because of ill health, those who were pensioned, and those who had applied for workmen's compensation. They listed 24 of the 96 lung cancer deaths found by the methods already described plus three additional cases. These three deaths had been certified as due to tuberculosis (ICD 019), asbestosis (ICD 523), and coronary heart disease (ICD 420), and though there had been autopsies for the purpose of compensation, no mention of this was made on the death certificate. In addition, there were three autopsies for compensation purposes in which a small lung cancer was noted as an incidental finding. The pathologist had not considered these cancers to have contributed to death and the causes certified were asbestosis (ICD 523) in two cases and cardiac infarction (ICD 420) in a third.

3. A search was made for any case of primary malignant mesothelial tumor of the pleura that might have been given a code other than 162. One additional case coded as a benign pleural tumor was found.

4. We reviewed cases reported by pathologists in the national survey of primary malignant mesothelial tumors in Canada between 1960 and 1968, and five were in men who had worked in the Quebec asbestos-producing industry. Two were born in 1922 and were, therefore, not in our cohort, and two died in 1967 after the present study had ended. A fifth case, diagnosed by biopsy, was eligible for both studies.

To summarize the results of all these inquiries, three cases were removed from the group of 95 malignant neoplasms of the bronchus, trachea, or lung (ICD 162 and 163) and nine were added, making 101 in all (100 men and one woman). Included in the total were the following: cancer of the trachea, in one case; malignant mesothelial tumor, in three; and cancer of the bronchus or lung, in 97.

Results

Female Mortality.—There were only 465 women in the cohort; almost all had short exposures and all but 33 were in the two lowest dust-index categories. In all, 440 (94.6%) were traced, and of those, 45 had died. Tuberculosis (11), malignant neoplasms (12), circulatory (nine) and respiratory (one) diseases, and trauma (one) were the causes of death in 34. Death was due to other causes in eight cases, and the cause of death was unknown in two; there was one death from lung cancer. Apart, perhaps, from tuberculosis, these figures are not unusual and will not be considered further.

Male Mortality.—To permit comparisons of mortality by dust index and years of exposure, death rates were calculated in five-year cohorts by date of birth. Equivalent average death rates[13] were then calculated using a standard population with equal numbers in each age group. Since the actual number at risk in each cohort was similar, this method of age standardization was particularly appropriate. The standardized rates are shown by dust index and main disease groupings in Table 3. In the lower five dust categories, equivalent average death rates for "all causes" were approximately the same, but in the top category, containing 5% of the total cohort, mortality was about 20% higher than the rest. Malignant neoplasms, circulatory diseases, and respiratory diseases, in approximately equal proportions, accounted for the excess. Two groups showed the opposite trend: "trauma," probably because rates fell with advancing age, and "unknown causes," probably because death certificates were difficult to find for older men with short periods of employment.

Table 4 shows that the main contribution to the excess in malignant disease was from cancer of the bronchus, trachea, and lung

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Table 3.—*Equivalent Average Death Rates per 1,000 Men by Dust Index*

	Dust Index						
	<10	10-	100-	200-	400-	800-	All
No. of men	3,006	3,408	1,148	1,002	842	575	9,981
Tuberculosis (ICD 001-019)	18.8	19.9	31.6	23.8	28.5	25.8	22.5
Malignant neoplasms (ICD 140-209)	38.8	39.3	32.2	27.5	45.1	61.8	38.6
Circulatory diseases (ICD 400-469)	85.0	80.8	82.5	83.5	94.8	107.3	85.8
Respiratory diseases (ICD 470-529)	9.5	15.3	19.5	16.7	15.3	41.6	15.6
Trauma (ICD 800-999)	34.7	30.8	36.3	35.2	33.3	13.7	31.5
Other causes	46.8	52.9	39.0	47.7	38.3	58.9	48.4
Unknown causes	30.6	21.4	16.5	6.2	7.2	3.4	20.8
All causes	264.1	260.4	257.6	240.6	262.9	312.5	263.1

Table 4.—*Equivalent Average Death Rates per 1,000 Men for Malignant Neoplasms (ICD 140-209) by Dust Index*

	Dust Index						
Location	<10	10-	100-	200-	400-	800-	All
Esophagus & stomach (ICD 150-151)	10.2 (32)	4.5 (13)	1.7 (2)	7.0 (5)	11.3 (8)	13.7 (8)	7.5 (68)
Intestine & rectum (ICD 152-154)	2.5 (8)	4.9 (15)	3.2 (3)	3.9 (3)	5.6 (4)	8.7 (5)	4.0 (38)
Other abdominal areas (ICD 155-159)	4.0 (13)	3.0 (9)	1.7 (1)	1.3 (1)	2.8 (2)	1.7 (1)	2.9 (27)
Bronchus, trachea & lung (ICD 162-163)	7.6 (25)	8.6 (26)	11.2 (10)	8.9 (8)	15.8 (11)	24.2 (14)	9.9 (94)
Other malignant neoplasms	14.4 (47)	18.6 (54)	14.5 (11)	6.4 (5)	9.5 (6)	13.5 (8)	14.4 (131)
All malignant neoplasms (ICD 140-209)	38.8 (125)	39.3 (117)	32.2 (27)	27.5 (22)	45.1 (31)	61.8 (36)	38.6 (358)

(ICD 162 and 163). In this group there was little difference between rates in the four lower dust categories, but the fifth and sixth groups showed an upward trend. A similar trend with years of exposure was found (Table 5), but the figures within the body of the table suggest a closer relationship with dust than with years. Subtraction of the three incorrectly coded cases and addition of the nine lung cancer cases found at autopsy increased slightly the rate among persons with highest dust and longest exposure, but did not materially change the picture (Table 5). Further analyses to distinguish better the relative importance of years of exposure and dust index are described as follows.

Rates for cancer of the intestine were about one third of those for cancers of the bronchus, trachea, and lung, but showed a very similar trend. Rates for cancer of the esophagus and stomach, on the other hand, did not appear related to dust in any consistent manner. Other abdominal neoplasms were less frequent still and also unrelated to dust. This is important since it might be expected that unrecognized peritoneal mesotheliomas would be found within this group.

Most of the excess mortality from respiratory disease was ascribed to pneumoconiosis (Table 6). There was little evidence that dust-associated deaths were included in other respiratory categories. Of the 28 deaths coded under the pneumoconioses, one was described as anthracosis, four as silicosis, and the remaining 23 as asbestosis. The greatest excess mortality from pneumoconiosis was among persons in the highest dust group who had been employed between ten and 29 years.

In the circulatory diseases group, there was also excess mortality in the highest two dust categories, mainly in persons employed

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between ten and 29 years. It was present equally in the "arteriosclerotic and degenerative heart disease" group (ICD 420 to 422 [which included more than three quarters of all circulatory deaths]) and in the group of "other circulatory diseases" (ICD 400 to 419 and 423 to 469).

Comparison With Quebec Mortality.—The number of deaths from all causes which would have been expected if Quebec death rates had applied was calculated in the following way. Age-specific death rates for the province were applied for each year, 1950 to 1966, to all the men traced who were alive in 1950, with an adjustment for those who started work after 1950. The expected number of deaths among men, thus, was 1,824, whereas the observed number in the cohort was 1,674.

A similar calculation was made for lung cancer deaths. The expected number was 91, whereas 94 male deaths certified as due to this cause were observed. Correction for coding errors and additional autopsy information was not appropriate, since certificates for the general population were uncorrected.

In the six counties of the province which include the mining region (Arthabaska, Beauce, Drummond, Megantic, Richmond, and Wolfe), the lung cancer death rate was about two-thirds the provincial rate and the expected number of deaths was correspondingly lower. However, many ex-employees were no longer living in this area when they died, and most of those who had moved had gone to cities where lung cancer death rates were higher. The best estimate of expected deaths probably lies between the numbers 61, derived from the mining region, and 91, from the province. The excess of observed over expected lung cancer deaths, therefore, lies somewhere between zero and 30 and is most probably between ten and 20.

Factor Evaluation.—Although the numbers of men in each cohort were about the same, their distribution by years of employment and dust index was uneven. In these circumstances, equivalent average death rates could be misleading. Berry[14] recently reviewed some of the methods used for analyzing the importance of factors in multiway tables and described a parametric approach which has the advantage that the adequacy of the model may be checked and statistical significance evaluated. From the number of deaths from any particular cause in groups subdivided by date of birth and year of employment or dust index, expected rates may be calculated assuming no interaction between age and the other two factors. For this analysis, the complementary log log transformation was used.[15] This transformation is appropriate when increasing exposure is associated with proportional increases in the age-specific death rates.

Table 5.—*Equivalent Average Death Rates per 1,000 Men for Cancer of Bronchus, Trachea, and Lung**

	Dust Index						
Years	<10	10-	100-	200-	400-	800-	All
As certified							
<1	6.4	1.7	27.8	0.0	...	...	5.9
	(16)	(1)	(1)	(0)			(18)
1-	11.7	9.5	11.3	5.8	7.6	0.0	9.6
	(9)	(15)	(4)	(1)	(1)	(0)	(30)
10-	0.0	12.4	6.7	5.3	19.9	23.6	13.1
	(0)	(7)	(3)	(3)	(6)	(6)	(25)
30-	...	12.5	14.7	13.9	16.1	27.9	17.2
		(3)	(2)	(4)	(4)	(8)	(21)
All	7.6	8.6	11.2	8.9	15.8	24.2	9.9
	(25)	(26)	(10)	(8)	(11)	(14)	(94)
Using all available information							
<1	6.7	1.7	0.0	0.0	...	...	5.9
	(17)	(1)	(0)	(0)			(18)
1-	12.0	10.1	9.4	11.5	7.6	0.0	10.1
	(9)	(16)	(3)	(2)	(1)	(0)	(31)
10-	0.0	12.8	7.6	9.2	19.9	23.6	14.3
	(0)	(8)	(4)	(4)	(6)	(6)	(28)
30-	...	12.5	14.7	13.9	16.1	35.3	19.1
		(3)	(2)	(4)	(4)	(10)	(23)
All	7.8	9.1	10.1	10.6	15.8	27.7	10.6
	(26)	(28)	(9)	(10)	(11)	(16)	(100)

* By dust index and years of employment. Diagnosis was ICD 162 and 163.

The results of this parametric analysis for respiratory cancer (Table 7) agree remarkably closely with the equivalent average death rates shown in the lower half of Table 5. The rates for the first five dust index levels do not differ significantly but those for the first four are significantly different from the highest exposure group. There are, however, also differences between the rates by years of employment, those for the lower two categories being significantly less than that for men with the longest exposure.

The general fit of the complementary log log model is very good. In no cell of the 24 does the observed number of deaths differ significantly from expectation. Tests for interaction between the 24 cells and the six cohorts gave the following results: likelihood

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Table 6.—*Equivalent Average Death Rates per 1,000 Men for Respiratory Diseases (ICD 470-529) by Dust Index and Years of Employment*

	Dust Index						
Years	<10	10-	100-	200-	400-	800-	All
Pneumonia and bronchitis (ICD 490-509)							
<1	2.6(7)	8.7(4)	0 (0)	166.7(1)	...	...	4.0(12)
1-	7.1(5)	3.7(6)	2.5(1)	4.4(1)	15.5(2)	16.7(1)	5.2(16)
10-	0	6.3(4)	5.9(2)	5.3(3)	5.6(3)	9.5(2)	6.3(14)
30-	...	0	4.9(1)	5.8(1)	0 (0)	3.4(1)	2.9(3)
All	3.8(12)	4.7(14)	4.2(4)	6.6(6)	5.6(5)	6.9(4)	4.9(45)
Pneumoconioses (ICD 523 and 524)							
<1	1.4(3)	4.5(2)	0	0	...	...	1.9(5)
1-	1.4(1)	1.5(2)	0	5.8(1)	0	0	1.5(4)
10-	0	2.3(1)	0	1.2(1)	1.6(1)	34.3(8)	6.3(11)
30-	...	0	0	3.6(1)	7.1(2)	17.9(5)	6.9(8)
All	1.3(4)	2.0(5)	0(0)	3.7(3)	3.9(3)	22.6(13)	3.2(28)
Other respiratory diseases (ICD 470-489, 510-522, 525-529)							
<1	4.5(10)	5.7(3)	0	0	0	0	4.6(13)
1-	3.8(3)	13.2(20)	29.0(9)	9.8(2)	7.9(1)	13.9(1)	12.5(36)
10-	0	2.7(2)	9.2(2)	7.9(2)	11.0(3)	7.8(2)	7.3(11)
30-	...	0	0	0	0	13.5(4)	3.6(4)
All	4.2(13)	8.6(25)	15.3(11)	6.3(4)	5.9(4)	12.1(7)	7.5(64)
All respiratory diseases (ICD 470-529)							
<1	8.6(20)	18.8(9)	0	166.7(1)	...	...	10.5(30)
1-	12.2(9)	18.4(28)	31.5(10)	20.0(4)	23.5(3)	30.6(2)	19.2(56)
10-	0	11.2(7)	15.1(4)	14.4(6)	18.2(7)	51.6(12)	19.9(36)
30-	...	0	4.9(1)	9.3(2)	7.1(2)	34.7(10)	13.4(15)
All	9.5(29)	15.3(44)	19.5(15)	16.7(13)	15.3(12)	41.6(24)	15.6(137)

ratio test, 88.9 and χ^2, 90.4, each with 95 degrees of freedom; and $P > 0.5$.

Comment

At face value, the findings suggest that our cohort of workers in the chrysotile mining industry had a lower mortality than the population of Quebec of the same age. This is generally true of employed persons, provided they are not subjected to an occupational hazard sufficient to offset the considerable selective advantage of being and remaining fit for work. This advantage was clearly lost by the men in the highest dust-index category whose standardized mortality was about 20% above that of the rest. Two thirds of the excess mortality in this group was probably due to pulmonary fibrosis, shown on the death certificate as either asbestosis or in the guise of various respiratory or cardiovascular diagnoses, and the remaining third to cancer, mainly of the respiratory tract.

The high rate of lung cancer in men heavily exposed to asbestos might be explained if such men also tended to smoke more heavily than others. We have no direct evidence on this point for ex-employees, since information on smoking was not routinely recorded by the companies or the medical clinics. However, in a survey of a selected random sample of over 1,000 current employees there was little or no relationship, after allowing for age, between smoking habits and either dust exposure or duration of employment.

Table 7.—*Age-Corrected Death Rates per 1,000 Men for Cancer of the Bronchus, Trachea, and Lung by Dust Index and Years of Employment**

	Dust Index						
Years	<10	10-	100-	200-	400-	800-	All
<1	6.3	2.0	0.0	0.0	...	...	5.5
1-	11.4	9.4	8.1	8.3	9.6	0.0	9.5
10-	0.0	10.4	9.8	10.3	16.8	23.4	12.8
30-	...	13.3	9.9	14.6	13.7	34.4	17.9
All	7.5	8.7	8.9	11.0	14.6	26.3	10.0

* A complementary log log model was used, and diagnosis was based on all available information.

Our attempts to assess separately the importance of cumulative dust exposure and duration of exposure in relation to lung cancer are capable of more than one interpretation. As shown in Table 5, there is a fivefold difference between the mortality for those with the lowest amount and duration of exposure, 6.7, and that for those with the highest, 35.3. This is confirmed by the parametric analysis (Table 7) which further suggests that accumulated dust exposure and

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Table 8.—*Comparison of Mesothelial Tumor Findings in Two Previous Studies and the Present Study*

	Selikoff et al[16]	Newhouse[8,17]	Present Study
No. of men studied	632	4,806	9,991
No. of deaths			
All causes	380	436	2,413
Lung cancer	72	42	97
Mesothelial tumors	22	20	3

duration of employment are about equally important in determining the difference. Unfortunately, there are possible sources of bias and error which may have contributed to both these effects.

In Table 5, the rates for men with less than one year of employment seem remarkably low; 18 deaths were observed, whereas 29 would have been expected on the basis of Quebec rates. It was in this group that tracing was least satisfactory (Table 2). Failure to ascertain even a small number of lung cancer deaths would have made an appreciable difference in the rates. Short-term employees who were traced tended to be those who had stayed in the neighborhoods where, in any case, lung cancer rates were much lower than in the urban areas of the province and elsewhere, to which those untraced may well have gone. Whatever the explanation, it is difficult to accept, without reserve, the low rate of lung cancer in this group.

A second source of error is related to the ascertainment of lung cancer as a cause of death. It is generally believed that this diagnosis is greatly aided by postmortem examination. In our study, 34% of the cases before correction and 40% after correction had had an autopsy, compared with 12% of all deaths. This might not matter if autopsies were evenly distributed in relation to exposure, but this was not so. The autopsy rates in ascending order of dust-index group were 11%, 11%, 9%, 10%, 16%, and 22%, and by years of employment, 12%, 9%, 17%, and 21%. This trend is also likely to have exaggerated the difference in lung cancer rates in relation to exposure, but by how much it is impossible to say.

Taking all these considerations into account we are inclined to conclude that the true difference between those maximally and minimally exposed may well be closer to threefold than fivefold and that this is partly dust-related and partly time-related. We propose in future analyses, when a longer period of observation will have yielded more data, to use an exposure index based on the concept of amount of dust inhaled and the time that it remains in the lung. Our findings so far appear compatible with such a model.

In the Canadian survey mentioned earlier,[10] primary malignant mesothelial tumors were rarely associated with chrysotile asbestos production in Quebec and the present survey bears this out. Three deaths from this cause were found among nearly 2,500 deaths from all causes in the cohort. This is probably more than would be expected in a comparable number of deaths in the general population, but quite out of line with the findings of Selikoff et al[16] in insulation workers and those of Newhouse[8,17] in a London asbestos factory. The magnitude of the difference can be inferred from the figures in Table 8. Though these figures are not entirely comparable, because of age, methods of ascertainment, or other factors, they suggest that the results of Newhouse[8,17] are similar to those of Selikoff et al[16] for mesothelial tumors and, perhaps, lung cancer. It is clear that the Quebec chrysotile workers have had nothing like the experiences of the American insulation workers or the London factory workers with respect to malignant mesothelioma, and it seems unlikely that they are compatible with respect to lung cancer. These findings strongly suggest either that chrysotile is less likely to cause malignant disease of the lung and pleura than other forms of asbestos, such as crocidolite, or that workers engaged in insulation and processing are exposed to additional factors which explain the difference.

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