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# MORTALITY FROM LUNG CANCER AND OTHER CAUSES IN THE CHRYSOTILE ASBESTOS MINES AND MILLS OF QUEBEC.

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The remarkable qualities of the asbestos group of fibrous minerals have been recognised since antiquity but mining and milling on an industrial scale began only at the end of the nineteenth 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, S. 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 neighbouring 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 1892 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


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considerably the dust concentration to which workers in the mines, mills and primary manufacturing 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 showed a link between asbestos exposure and lung cancer. The potential extent of this hazard was increased by reports of Wagner and others[3] during the last ten years that malignant tumours of the pleura and peritoneum are related to certain types of industrial work and perhaps also to neighbourhood 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[4] and other observations among textile workers have confirmed the association[5-8]. 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 10 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[8] (also in a textile factory) found an excess of lung cancer in persons heavily exposed for periods of less than 2 years, 20 years earlier. Workers exposed to light or moderate dust concentrations showed no excess of bronchogenic cancer even after 20 years exposure.


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Selikoff and his colleagues[9] in a cohort-study among the members of an American insulation workers union for 20 years of more, found an eight-fold increase in lung cancer compared with national figures. He also found a gross excess of pleural and peritoneal mesotheliomas. On the other hand, in a national survey of all known mesothelial tumours in Canada, 1960-8, 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 fibre - chrysotile, crocidolite, amosite and anthophyllite - the two textile factories referred to above[6,8] used mainly chrysotile fibre, together with some crocidolite, and in one of the two, some amosite. As in most industrial applications different types of fibres are mixed, the carcinogenic effects of a single fibre type can only in practice be studied in mining and milling. Chrysotile fibre 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, 1958[11] and by Kogan et al, 1966[12]. In the first of these, 9 deaths from lung cancer were observed in the Quebec industry compared with 6 expected from Provincial rates. The second was in the Soviet Union and showed that compared with the general population the mortality rate 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


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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 & METHODS

### Registration

A register was compiled in the personnel department of each asbestos mining company in the Eastern Townships region of Quebec of all persons currently or previously employed, as of 1 November 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 for every job, the department and mine, dates of starting and finishing 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.

There was of course some movement of labour from one mining


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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 1 November, 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. The concentrations of dust at working places had been measured annually since 1949 by Mr. M. Lachance throughout the industry in the dryers, crushers and mills, and since 1946 by various investigators in the small factory. All measurements were made with 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


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environmental features and of the methods and results of dust measurement are to be published by G.W. Gibbs and M. 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 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 5 years at 40 MPCF, 15 years at 20 MPCF and 20 years at 5 MPCF (i.e. 200 + 300 + 100). It would be useful if it could be assumed that an index of 600 were also equivalent to a working life of say 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


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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 20 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. There was thus a substantial group with minimal exposure available for comparison with others heavily exposed.

Tracing and ascertainment

The search for ex-employees was mainly initiated in the mining towns. Telephone or postal enquiries were first made to establish whether the person in question was alive or dead on 1st November, 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 for some 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 1st November 1966, and a further 30 had re-registered after that date. All 628 were presumed alive for the purpose of our study.

Information was obtained about 10,421 (88.4%) of the 11,788 persons in the cohort; 7,965 were still alive (including 2,484 currently


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employed) and 2457 (23.6% of those traced) were reported dead. Copies of death certificates were obtained from Canadian Provincial and U.S. State Registrars for 2211 (90%). In a further 59 (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 in 1926 of death registration in Quebec and the cause of death was stated by relatives, and in the other 7, 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 (WHO Geneva, 1957). This was done by a 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 are shown in Tables 1 & 2. Success in tracing depended closely on duration of employment; 94% of those employed for one year or more and over 99% of all those employed for 10 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 neighbourhood and 2) insufficient identifying information which would have enabled a request to be made to the Unemployment Insurance Commission or a death certificate to be found 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 local


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rather than transient.

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 to lung cancer (ICD 162,163) one certified as a hydronephroma and one as pulmonary fibrosis had been wrongly coded. Enquiries 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; and in another five, though the diagnosis had been made in hospital, no clinical or pathological record was found. A further 17 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 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 tumour was thought to be metastatic. Thus, of 95 certified cases, 3 had been included incorrectly, 47 were confirmed pathologically, 28 hospital diagnoses were presumed correct, and in 17 no further information was obtainable.

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

i) A letter was sent to the certifying physicians whenever it


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was stated that an autopsy had been or would be performed. These numbered 306 in all, 33 certified as lung cancer and 273 as other causes. In 18 the reply 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 5 additional cases of lung cancer. These had been certified as renal tumour (180) infectious hepatitis (092), pulmonary fibrosis (519), diabetes (260) and asbestosis (523).

ii) We asked the physicians in charge of the industrial clinics at Thetford Mines and Asbestos for lists of all deaths known to them 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 had been certified as tuberculosis (019), asbestosis (523) and coronary heart disease (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 3 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 (523) in two cases, and cardiac infarction (420) in the third.

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

iv) We reviewed cases reported by pathologists in the national


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survey of primary malignant mesothelial tumours 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 end of the present study. The fifth case, diagnosed by biopsy, was eligible for both studies.

Thus, to summarize the result of all these enquiries, three cases were removed from the group of 95 malignant neoplasms of the bronchus, trachea or lung (ICD 162, 163) and 9 were added, making 101 in all (100 males and one female). In the total were one cancer of the trachea, 3 malignant mesothelial tumours and 97 cancers of the bronchus or lung.

## 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, 44 had died. The causes of death were tuberculosis (11), malignant neoplasms (12), circulatory (9), respiratory (1), trauma (1) other (8), not known (2); there was one death from lung cancer. Apart perhaps from tuberculosis these figures are not remarkable, 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 5-year cohorts by date of birth.


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Equivalent average death rates [43] 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.

These 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 (ICD 162, 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 cells of the table suggest a closer relationship with dust than with years. Subtraction of the 3 incorrectly coded cases and addition of the 9 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 below:

Rates for cancer of the intestine were about one-third of those for cancers of the bronchus, trachea and lung, but showed a very


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similar trend. Rates for cancer of the oesophagus 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, 4 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 10 and 29 years.

In the circulatory diseases group there was also excess mortality in the highest two dust categories mainly in persons employed between 10 and 29 years. It was present equally in "arteriosclerotic and degenerative heart disease", ICD 420-422 (which included more than three-quarters of all circulatory deaths) and in "other circulatory diseases" ICD 400-419 and 423-469.

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, 1950-1966, to all the men traced who were alive in 1950 with an adjustment for those who started work after 1950. The number of deaths among males thus expected was 1824


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whereas the number observed in the cohort was 1674.

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

In the six countries 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 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 figure of 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 probably between 10 and 20.

Factor evaluation

Although the number of men in each cohort was 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 analysing 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,


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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.

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 ratio test = 88.9; $X^2$ = 90.4; each with 95 degrees of freedom; $P > 0.5$.


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## DISCUSSION

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 similar 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 5% of men in the highest dust-index category whose standardized mortality was about 20% above the rest. Two-thirds of the excess 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 1000 current employees there was no such relationship, after allowing for age, between smoking habits and either dust exposure or duration of employment.

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. In Table 5 it may be seen that there is a five-fold difference between 6.7, the mortality rate for those with the lowest amount and duration of exposure, and 35.3 for those with the highest. This is confirmed by the parametric analysis (Table 7) which further suggests that accumulated dust exposure and duration of employment are about equally important in determining the difference. Unfortunately,


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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 from Quebec rates. It will be recalled (Table 2) that it was in this group that tracing was least satisfactory. Failure to ascertain even a small number of lung cancer deaths would have made an appreciable difference to the rates. Short-term employees who were traced tended to be those who had stayed in the neighborhood where, in any case, lung cancer rates are 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 post mortem examination. In our study 34% of 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 is impossible to say.


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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 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 tumours 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 Newhouse (8,17) in a London asbestos factory. The magnitude of the difference can be inferred from the following figures: -

| | Selikoff et al | Newhouse | Present study |
|---|---|---|---|
| Number of men studied | 632 | 4,806 | 9,981 |
| Number of deaths | | | |
| - all causes | 380 | 436 | 2,413 |
| - lung cancer | 72 | 42 | 97 |
| - mesothelial tumours | 22 | 20 | 3 |


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Though, for reasons of age, methods of ascertainment and probably other factors these figures are not entirely comparable, they suggest that the results of Newhouse are similar to those of Selikoff et al for mesothelial tumours and perhaps for 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 in respect of malignant mesothelioma and it seems unlikely that they are compatible in 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 on insulation and processing are exposed to additional factors which explain the difference.


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## SYNOPSIS

Of 11,788 persons born 1891-1920 and employed in the Quebec asbestos mining industry, 88.4% were traced and of these 2,457 (23.6%) had died. Exposure indices 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 diseases in equal proportions accounted for the excess. There were 101 deaths from respiratory cancer including 3 pre-malignant mesotheliom, 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.


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Table 1 - Subjects of study and results of tracing by age and sex.

| Year of Birth | Persons M | Persons F | Persons Total | Traced M | Traced F | Traced Total | % traced | Dead | % of persons traced |
|---|---|---|---|---|---|---|---|---|---|
| 1891-95 | 1,553 | 14 | 1,567 | 1,179 | 13 | 1,192 | 76.1 | 611 | 51.3 |
| 1896-00 | 1,982 | 31 | 2,013 | 1,588 | 28 | 1,616 | 80.3 | 655 | 40.5 |
| 1901-05 | 2,027 | 50 | 2,077 | 1,724 | 45 | 1,769 | 85.2 | 498 | 28.2 |
| 1906-10 | 1,897 | 99 | 1,996 | 1,741 | 92 | 1,833 | 91.8 | 330 | 18.0 |
| 1911-15 | 1,837 | 117 | 1,954 | 1,773 | 113 | 1,886 | 96.5 | 221 | 11.7 |
| 1916-20 | 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 |

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*Mortality from Lung Cancer and Other Causes in the Chrysotile Asbestos Mines and Mills of Quebec*
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Table 1 - Subjects of study and results of tracing by age and sex.

| Year of Birth | Persons M | Persons F | Persons Total | Traced M | Traced F | Traced Total | % traced | Dead | % of persons traced |
|---|---|---|---|---|---|---|---|---|---|
| 1891-95 | 1,553 | 14 | 1,567 | 1,179 | 13 | 1,192 | 76.1 | 611 | 51.3 |
| 1896-00 | 1,982 | 31 | 2,013 | 1,588 | 28 | 1,616 | 80.3 | 655 | 40.5 |
| 1901-05 | 2,027 | 50 | 2,077 | 1,724 | 45 | 1,769 | 85.2 | 498 | 28.2 |
| 1906-10 | 1,897 | 99 | 1,996 | 1,741 | 92 | 1,833 | 91.8 | 330 | 18.0 |
| 1911-15 | 1,837 | 117 | 1,954 | 1,773 | 113 | 1,886 | 96.5 | 221 | 11.7 |
| 1916-20 | 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 |


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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 | 3,733 |
| | % 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 | 88.2 |
| 10- | No. | 55 | 1,117 | 546 | 322 | 432 | 255 | 2,947 |
| | % traced | 100.0 | 99.0 | 98.9 | 92.0 | 93.3 | 93.6 | 99.1 |
| 30- | No. | 0 | 204 | 181 | 250 | 303 | 260 | 1,203 |
| | % traced | - | 100.0 | 100.0 | 100.0 | 100.0 | 99.6 | 99.8 |
| All | No. | 4,123 | 3,950 | 1,235 | 1,039 | 857 | 584 | 11,783 |
| | % traced | 79.9 | 89.9 | 94.9 | 97.0 | 98.4 | 98.6 | 88.4 |


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Table 3 - Equivalent average death rates per 1000 men by dust index.

| | Dust Index | | | | | | |
|---|---|---|---|---|---|---|---|
| | <10 | 10- | 100- | 200- | 400- | 800- | All |
| Number of men | 3,006 | 3,408 | 1,148 | 1,002 | 842 | 575 | 9,981 |
| Tuberculosis (I.C.D. 001-019) | 18.8 | 19.9 | 31.6 | 23.8 | 28.5 | 25.8 | 22.5 |
| Malignant neoplasms (I.C.D. 140-209) | 38.8 | 39.3 | 32.2 | 27.5 | 45.1 | 61.8 | 38.6 |
| Circulatory Diseases (I.C.D. 400-469) | 85.0 | 80.8 | 82.6 | 85.5 | 94.8 | 107.3 | 85.8 |
| Respiratory diseases (I.C.D. 470-529) | 9.5 | 15.3 | 19.5 | 16.7 | 15.3 | 41.6 | 15.6 |
| Trauma (I.C.D. 800-999) | 34.7 | 30.8 | 36.3 | 35.2 | 33.8 | 13.7 | 31.5 |
| Other causes | 46.8 | 52.9 | 39.0 | 47.7 | 38.3 | 58.9 | 46.4 |
| 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 |


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Table 4 - Equivalent average death rates per 1000 men for malignant neoplasms (I.C.D. 140-209) by dust index.

| | Dust Index | | | | | | |
|---|---|---|---|---|---|---|---|
| | <10 | 10- | 100- | 200- | 400- | 800- | All |
| Oesophagus & stomach (I.C.D. 150-151) | 10.2 (32) | 4.5 (13) | 1.7 (2) | 7.0 (5) | 11.3 (8) | 13.7 (9) | 7.5 (68) |
| Intestine & rectum (I.C.D. 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 (I.C.D. 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 (I.C.D. 162-163) | 7.6 (25) | 8.6 (26) | 11.2 (10) | 3.9 (3) | 15.8 (11) | 24.7 (14) | 9.9 (94) |
| Other malignant neoplasms | 14.4 (47) | 18.6 (54) | 14.5 (11) | 6.4 (5) | 9.3 (6) | 13.5 (8) | 14.4 (131) |
| All malignant neoplasms (I.C.D. 140-209) | 38.8 (125) | 39.3 (117) | 32.2 (27) | 27.5 (22) | 45.1 (31) | 61.8 (36) | 38.6 (358) |


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Table 5 - Equivalent average death rates per 1000 men for cancer of the bronchus, trachea and lung (I.C.D. 162,163) by dust index and years of employment.

| | | Dust Index | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Years | <10 | 10- | 100- | 200- | 400- | 800- | All |
| As certified. | <1 | 6.4 (16) | 1.7 (1) | 27.8 (1) | 0.0 (0) | - | - | 5.9 (18) |
| | 1- | 11.7 (9) | 9.5 (15) | 11.3 (4) | 5.8 (1) | 7.6 (1) | 0.0 (0) | 9.6 (30) |
| | 10- | 0.0 (0) | 12.4 (7) | 6.7 (3) | 5.3 (3) | 19.9 (6) | 23.6 (6) | 13.1 (25) |
| | 30- | - | 12.5 (5) | 14.7 (2) | 13.9 (4) | 16.1 (4) | 27.9 (8) | 17.2 (21) |
| | All | 7.6 (25) | 8.6 (26) | 11.2 (10) | 8.9 (8) | 15.8 (11) | 24.2 (14) | 9.9 (94) |
| Using all available information | <1 | 6.7 (17) | 1.7 (1) | 0.0 (0) | 0.0 (0) | - | - | 5.9 (18) |
| | 1- | 12.0 (9) | 10.1 (16) | 9.4 (3) | 11.5 (2) | 7.6 (1) | 0.0 (0) | 10.1 (31) |
| | 10- | 0.0 (0) | 12.8 (8) | 7.6 (4) | 9.2 (4) | 19.9 (6) | 23.6 (6) | 14.3 (28) |
| | 30- | - | 12.5 (3) | 14.7 (2) | 13.9 (4) | 16.1 (4) | 35.3 (10) | 19.1 (23) |
| | All | 7.8 (26) | 9.1 (28) | 10.1 (9) | 10.6 (10) | 15.8 (11) | 27.7 (16) | 10.6 (100) |


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Table 6 - Equivalent average death rates per 1000 men for respiratory diseases (I.C.D. 470-529) by dust index and years of employment.

| | | Dust Index | | | | | |
|---|---|---|---|---|---|---|---|
| | Years | <10 | 10- | 100- | 200- | 400- | 800- | All |
| Pneumonia & 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) | 5.6(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 523,524. | <1 | 1.4(5) | 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 | 1.5(4) | 2.0(5) | 0 (0) | 3.7(3) | 3.9(3) | 22.6(13) | 3.2(8) |
| 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) |


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Table 7

Age corrected death rates per 1000 men for cancer of the bronchus, trachea and lung* by dust index and years of employment.

(Complementary log log model)

| Years | Dust Index <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 |

*diagnosis based on all available information


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