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THE 1891–1920 BIRTH COHORT OF QUEBEC CHRYSOTILE MINERS AND MILLERS: DEVELOPMENT FROM 1904 AND MORTALITY TO 1992

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Abstract—This paper draws together the mortality experience for a cohort of some 11 000 male Quebec Chrysotile miners and millers, reported at intervals since 1971 and now again updated. Of the 10 918 men in the complete cohort, 1138 were lost to view, almost all never traced after employment of only a month or two before 1935; the other 9780 men were traced into 1992. Of these, 8009 (82%) are known to have died: 657 from lung cancer, 38 from mesotheliona, 1205 from other malignant disease, 108 from pneumoconiosis and 561 from other non-malignant respiratory diseases (excluding tuberculosis). After early fluctuations, SMRs (all causes) against Quebec rates have been reasonably steady since about 1945. For men first employed in Asbestos, mine or factory, they were very much what might have been expected for a blue collar population without any hazardous exposure. SMRs in the Thetford Mines area were almost 8% higher, but in line with anecdotal evidence concerning socio-economic status. At exposures below 300 (million particles per cubic foot) × years, (mpcf.y), equivalent to roughly 1000 (fibres/ml) × years—or, say, 10 years in the 1940s at 80 (fibres/ml)—findings were as follows. There were no discernible associations of degree of exposure and SMRs, whether for all causes of death or for all the specific cancer sites examined. The average SMRs were 1.07 (all causes), and 1.16, 0.93, 1.03 and 1.21, respectively, for gastric, other abdominal, laryngeal and lung cancer. Men whose exposures were less then 300 mpcf.y suffered almost one-half of the 146 deaths from pneumoconiosis or mesothelioma; the elimination of these two causes would have reduced these men's SMR (all causes) from 1.07 to approximately 1.06. Thus it is concluded from the viewpoint of mortality that exposure in this industry to less than 300 mpcf.y has been essentially innocuous, although there was a small risk or pneumoconiosis or mesothelioma. Higher exposures have, however, led to excesses, increasing with degree of exposure, of mortality from all causes, and from lung cancer and stomach cancer, but such exposures, of at least 300 mpcf.y, are several orders of magnitude more severe than any that have been seen for many years. The effects of cigarette smoking were much more deleterious than those of dust exposure, not only for lung cancer (the SMR for smokers of 20+ cigarettes a day being 4.6 times higher than that for non-smokers), but also for stomach cancer (2.0 times higher), laryngeal cancer (2.9 times higher), and—most importantly—for all causes (1.6 times higher). © 1997 British Occupational Hygiene Society

INTRODUCTION

Attention drawn in 1964 to the apparent serious threats to health posed by airborne asbestos dust led immediately to recommendations that epidemiological enquiries were urgently needed in populations exposed to single fibre types (Working Group

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on Asbestos and Cancer, 1965). Most occupational exposures have been to fibres of more than one type, but mining populations were thought to be exceptions; that in Quebec was not only the earliest but also the largest in the Western world and so appeared particularly suitable for such studies.

The remarkable strength and fire-resistant properties of the asbestos group of fibrous minerals were known in Roman times, but commercial exploitation began only at the end of the nineteenth century. Large deposits of chrysotile in the Eastern Townships region of Quebec had been noted in the Canadian Geological Survey of 1847, and in 1878 about 50 tons of commercially disposable fibre were produced at Thetford, 90 km south of Quebec city. Four years later, mining and milling also started 60 km to the south-west, near Danville. By 1918 the region was producing around 165 000 tons of fibre a year, and production continued to expand into the 1980s, to approximately 1.3 million tons a year. Quebec produced most of the world's supply of asbestos until after World War II; Quebec's share in 1960 was still 45%, but it was only 22% in 1977, and has declined since.

The company operating near Danville opened a small factory making asbestos products in a new, nearby town, called Asbestos. Near the original workings in Thetford, later call Thetford Mines, many small mining operations were opened up.

At the invitation of the Canadian government, and with encouragement from the government of Quebec, our group at McGill University embarked in 1966 on a comprehensive epidemiological survey of the entire Quebec asbestos-producing industry since its inception. Using the considerable body of data available, our primary aim has been to define as accurately as possible the quantitative relationship between exposure to chrysotile asbestos and mortality from lung cancer and malignant mesothelioma. Parallel studies, concerned with the relationship of dust exposure to radiographic appearances, pulmonary function and respiratory symptoms, were also made and reported separately.

Our knowledge of the mortality experienced by chrysotile workers is mainly derived from observations on a birth cohort of almost 11 000 men, followed from first employment (the earliest in 1904) to 1992. The criteria for admission to the cohort were birth between 1891 and 1920 and employment for at least a month in the Quebec chrysotile-producing industry. Those born before 1891 would have been particularly difficult to trace, and those born after 1920 have, even now, not reached an age of 50% mortality. Reports have been published of mortality to several points in time, namely 1966, 1969, 1973, 1975 and 1988; the number of deaths increased from fewer than 2500 to over 7300 (McDonald *et al.*, 1971, 1973, 1980, 1993; Liddell *et al.*, 1977). Tracing up to 1992 has now been completed, and no further tracing is planned.

This report is a comprehensive review of the development of the mortality investigation from its inception. A few figures in earlier publications have had to be very slightly revised.

MATERIALS AND METHODS

Registration

Starting in November 1966, all extant personnel records of the Quebec asbestos-producing industry were transcribed on to over 30 000 cards, one for each record. Each card was given an identity code which incorporated an indicator of the

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company from whose records the cards were prepared, the sex of the worker, and whether he or she was still employed at 1 November 1966. The information recorded included: name, forenames (or initials); date of birth (or age at first employment); date of first employment by this company; date of last recorded employment. On the reverse of each card was entered, in chronological order, the payroll record of each job and mine worked in, with the year and month of start and finish, and dates of periods of leave. Work histories were incomplete for at least 560 men who had worked at one company whose records were not transferred when the ownership of the company changed in 1964, also for a small number of others who had been employed both by this company and others. There was, of course, some movement of labour from one mining company to another. This necessitated bringing together and matching the records of all companies to obtain complete work histories. In the course of tracing ex-employees (see below), this matching process could often be confirmed or amended. In 1971, it was reported that 27 669 persons had been registered, including 6415 currently employed on 1 November 1966; 1039 had been employed by more than one company.

These cards were searched to select into the cohort all those born in the three decades 1891–1920 who had worked for at least one month in the industry. The cohort was found to comprise 11 788 persons, of whom 11 323 were males; the 465 females are not considered further in this report. In tracing to the end of 1971, a further 166 matchings had been made in which a current employee had had an earlier period of employment recorded elsewhere. It was also decided to exclude 50 men who could not be identified with certainty because their first names had not been recorded. The male cohort was thus reduced to 11 107.

In 1974, following a review of methods of analysis (Liddell, 1975; Liddell *et. al.*, 1977), it became clear that it would be necessary to devise a new data system (Rodrigues, 1976). As a preliminary to setting up this system, an even more careful search of the 30 000 record cards was carried out; some of them had been wrongly excluded eight years earlier, but rather more had been wrongly included mainly because they had worked for less than a month. A substantial number of further matchings was discovered in tracing to the end of 1973 and, as a result of these changes, the number of men correctly in the cohort was found to be 10 951; for nearly 1200 there had been more than one record card. In later tracing, to 1975, 1988 and 1992, further matches (12, 14 and 5) were discovered, as well as the fact that two women had been included as men. The final size of the cohort was thus 10 918.

Tracing

The initial search for ex-employees was by telephone or postal inquries in and around the mining towns, to establish whether the person in question was alive or dead on 1 November 1966; if reported dead, the exact date and place were sought from relatives, friends, priests, church registers and town clerks. For those not traced by these means, a systematic search was made in the provincial death records and certificates. In addition, a search on our behalf was made in the index of the Canadian Unemployment Insurance Commission (UIC) 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 1 November 1966, and 30 more had re-registered after that date; initially, all 628 were presumed to be alive. In the tracing to

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the end of 1969, more effort could be put into checking and obtaining correct addresses. This enabled stricter criteria of survival to be applied; in particular, no assumption that they were alive was made about the 598 men registered with the UIC.

Subsequent tracing through 1973 was undertaken by several research assistants and, since then, to 1975, 1988 and now to 1992 by the same two research assistants, one in each of the two mining regions, who have helped with this work almost from its inception. Additional inquiries had nevertheless to be made through vehicle licensing bureaux and a wide range of social security death indexes in the provinces of Canada, the states of the U.S.A., and many other countries.

The task became progressively more difficult because of problems of access to persons in old people's homes and widespread, but highly variable, legislation on the disclosure of information. Nevertheless, of those known to be alive in 1976, only 48 were subsequently lost to view. When follow-up was terminated, there had been over 8000 deaths while 1771 men were still alive at the last report, that is, after May 1992.

Cause of death

By 1 January 1970, there had been 3270 deaths. Death certificates were found for 2950 (90%) and reliable information, mainly from hospitals, on the cause of death in a further 104 (3%). Of the remaining 216 deaths, 51 had occurred before death registration in Quebec became compulsory in 1926, and most of the other 165 were outside Quebec, mostly outside Canada. When tracing to the end of 1975 was reported, there were only 125 deaths, among 4463, for which the cause was unknown. In later tracing, the proportion of deaths for which a copy of the certificate or equivalent could be found was over 98%, and adequate information was collected on most of the rest. The total of deaths from ill-defined and unknown causes by the end of follow-up in 1993 was 198: for roughly half of these we had not been able to find any information about cause; the other half had been given the relevant ICD code (799 in the Ninth Revision) officially.

Before 1976, the cause of death was coded according to the Seventh Revision of ICD. In the few cases where this revision had not been used on the death certificate, coding was done by a senior coder of the department of Demography of Quebec who had recently retired, and later usually by a person who regularly performed this task for the Quebec Department of Vital Statistics. For deaths after 1975, we took the code on the death certificate for which either the Eighth or Ninth Revision of the ICD had been used. In those few cases where a death certificate could not be obtained, the best information available was used to code the cause; if there was no information, the code 799 was allocated. In this report, all causes of death are identified by the ICD code of the Ninth Revision.

An exception to the use of ICD codes was made for mesothelioma, where a 'best diagnosis' was made after careful scrutiny of all available related clinical, biopsy and necropsy records; the code in our file was amended from what had been entered as routine, so that a death from mesothelioma should not be counted as one from any other cause. In reaching the total number of deaths from pneumoconiosis (108), eight that had been coded wrongly in the Eighth or Ninth Revision were re-coded correctly.

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Exposure calculations

From the 30 000 record cards, a list was compiled by Dr Graham Gibbs of all named jobs in each company from the beginning of its operations. There were 13 346 job names, but amalgamation of those considered identical reduced the number to 5783. A description of each of these 5783 jobs was obtained from existing evaluation systems and by interviewing employees with long service.

Since 1948, dust surveys in the mining and milling industry had been conducted almost annually at all operating companies by one observer (Mr Maurice Lachance) using the midget impinger, following the technique adopted by the Asbestos Textile Institute (Air Hygiene and Manufacturing Committee, 1963). Estimates of past and present dust conditions were made after interviews with employees of long service in collaboration with superintendents or others with special knowledge of past conditions. Account was taken of process changes, installation of dust control systems and other factors which affected dust levels. A total of 3096 dust measurements, made periodically since 1944, was used as a guide to exposure in the factory. All estimates were listed and mean annual concentrations were calculated. From these, the dust level for each of the 5783 classes, year by year, was placed on a 13-point scale. In each of the 13 categories, a representative value, approximating to the mean, was allocated as follows: 0.5, 2, 7, 12, 17, 22, 27, 32, 37, 42, 47, 70 and 140 million particles per cubic foot (mpcf). A full description is given by Gibbs and Lachance (1972).

The same authors describe several indices of exposure, including the one that has been adopted throughout these studies, namely accumulated dust exposure. A subject's exposure for a particular year was calculated as the product: (the fraction of the year worked in a specific job) × (the dust level for the year for that job) × (an adjustment for the length of the working week). If a subject worked in more than one job in any year, the relevant products were added to obtain the year's exposure. As the work-week was 60–72 h before 1938, 48 h 1938–1949 and 40 h thereafter, the adjustments adopted by Gibbs and Lachance were factors of $72/40 = 1.8$, $48/40 = 1.2$, and 1, respectively; however, in our work from 1974 we have used $66/40 = 1.65$ for the adjustment before 1938. From these yearly estimates, we compiled, for every man, a record for each of the 63 years from 1904 (the first recorded date of employment) to 1966, of the fraction of the year worked and his dust exposure (mpcf × years) in the year.

As over 2400 men in the cohort were still employed in 1967, attempts were made to estimate exposures yearly up to 1985, when the last man had retired. It did not prove feasible to use the same methods as previously. Instead, each man was allocated dust levels as follows: for 1967, the same level as in 1966; for each subsequent year, a proportion of that level in accordance with the average trend of fibre concentration for his specific mine or mill. From these levels, we estimated yearly exposures from 1967 to 1985 (McDonald *et. al.*, 1993), and extended each man's exposure record by a further 19 years. To give much-needed greater flexibility for the calculation of exposures—to the age of 55, for instance, or, for case-referent analyses, in relation to the age at death of the case—the exposure file was re-organized: first, the annual record of exposure, incorporating the adjustment for length of the working week, was changed to the dust level, with an indicator of the work-week adjustment; second, each man's work history was recorded annually

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from the year in which he started to the year in which he finished, thus reducing the maximum number of years from 82 to 59; and thirdly, the format was changed slightly. With these changes, the complete file was reduced in size by over one-quarter, but remained enormous (5.9 Megabytes).

Place of employment and place of registration

There were eight companies in business in 1966 from whose records work histories were compiled: one (Company 1), in Asbestos, was very large, and there were seven others, mainly in or near Thetford Mines. The earliest, and by far the largest, operations were at what we call Company 3 (comprising 13 mines) at Thetford Mines and at the mine and mill in Asbestos. Also early in production was Company 4 in Thetford Mines and a small asbestos products factory in Asbestos (owned by Company 1). The other companies (5–9) were all quite small and started operations much later, one (Company 5) as late as 1965. Many employees in the more recently established companies had worked previously elsewhere in the industry and often this was not indicated in the extant records. There were also frequent unrecorded movements of personnel between the mine and mill and the factory at Asbestos.

In what follows, a man's 'place of registration' has been taken from the identity code indicator of the company from whose personnel files his record was preparded.

Smoking history

Smoking habit was obtained by questionnaire in 1970. For 99.6% of those 6583 men alive then, the questionnaire was completed by the subject himself, and proxies responded for over 90% of those who had died since 1950. No attempt was made, however, to obtain histories for those who had died earlier. Each man was classified according to cigarette smoking at the time of his report, but it was not feasible to allocate a smoking category in a proportion of cases: this proportion was 20% of those for whom proxies had responded, 12% of those who died during 1970–1974, and 5% for those 5566 who had survived into 1975. These survivors were classified as follows: non-smoker of cigarettes (1050 men); ex-smoker (1174); current smoker of less than 20 cigarettes a day (1174); and current smoker of 20 or more a day (1876); with 292 men not allocated to any of these categories.

Statistical analyses

Analyses of mortality were made by the subject-years method (Berry, 1983), with the PYRS program (Coleman *et al.*, 1989). As reference, we used death rates (all causes) for males in 16 age groups by quinquennia from 1990 as follows: 1950–1992, Quebec; 1930–1949, Canada; 1925–1929, an amalgam of the 1926–1929 rates for Canada and the 1925 rates for Canada excluding Quebec; 1900–1924, the 1921–1924 rates for Canada, excluding Quebec, reproduced five times. Cause-specific rates were available only from 1950.

Some of the men traced alive after May 1992 will have died before the end of the year, but their deaths will not have been recorded. Therefore, for analytical purposes, the effective end of the study was taken as 31 May 1992. The numbers of deaths observed (O) were compared with the numbers expected (E) from the experience of the reference population described above, taking account of age and

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calendar period, by means of O/E ratios, and we follow the usual practice of terming them standardized mortality ratios (SMRs). Some authors recommend censoring at the age of 85; we saw no compelling reason to follow this practice.

To study relations with exposure, we calculated each man's exposure accumulated to the age of 55. There were, however, 236 men, mainly caretakers recruited from among ex-employees, who had not started recorded employment by the age of 45; these were excluded, along with five others whose work histories were inconsistent. For these analyses, the study interval—that period over which each subject is studied in a particular aspect of the analysis—could not start before the subject's 55th birthday (Liddell *et al.*, 1977) and earlier deaths or losses were excluded.

There can be no sensible reference rates for pneumoconiosis or mesothelioma, so for deaths from these causes we used a second option in the PYRS program to obtain rates per 100 000 subject-years.

Bases of tables

The bases for the various tables were chosen to give the greatest numbers for meaningful analysis. The Appendix tabulates the numbers of men, of subject-years, and of deaths in various classifications. For several purposes, the experience of men first registered at Companies 5–9 was excluded, so the three columns refer to: (i) all places of registration; (ii) the mine and mill in Asbestos and the factory there, and Companies 3 and 4 in Thetford; and (iii) Companies 5–9. Lines (a) to (f) relate to the complete cohort of 10 918 men: subject-years to the end of May 1992 are given for all men in line (b), and for those still alive in 1950 in line (c); deaths to the end of tracing are given in line (d), to the end of May 1992 in line (e), and for 1950–1992 in line (f). Lines (g) to (l) refer to the cohort after the exlusion of 241 men not employed before the age of 45 or with inconsistent work histories. Lines (g), (h) and (k) correspond to lines (a), (b) and (e), respectively; in line (i), subject-years before age 55 have been excluded from the numbers in the previous line. Lines (j) and (l) relate to the period 1950–1992, at ages of 55 or greater.

Bold figures in square brackets in the Appendix indicate text tables based on the relevant entries. More numbers are cited in the Appendix than are directly referred to; this is to allow the various tables to be related one with another.

FINDINGS

The growth of the cohort from 1904 and its decline until 1992 are summarized in Table 1, where numbers of entries, losses and deaths are given by decade. The first entries were of two men who started work in 1904, the last towards the end of 1966 shortly before registration took place, by which time the total recruitment was 10 918. A total of 1138 men were lost to view, 923 of them before 1935, the great majority never having been traced after employment of only a very few months. Many of the 215 later losses were also untraced after short employment. Also given in Table 1 are net entry (entries *less* losses within the period) and survivors (net entry *less* deaths) into the following period. Of the total net entry of 9780 men, 8009 (almost 82%) had died; the first death was in 1910, the last at the end of 1992. This left 1771 (18%) who were still alive when last traced, that is, during the second half of 1992. Figure 1 presents this material cumulatively by quinquennium.

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Table 1. The growth and decline of the cohort

Period	Entries (a)	Losses (b)	Net entry (a)−(b)	Deaths (c)	Survivors (a)−(b)−(c)
1904–1914*	437	63	374	8	366
1915–1924	3430	492	2938	86	3218
1925–1934	2878	368	2510	176	5552
1935–1944	2482	65	2417	353	7616
1945–1954	1406	33	1373	550	8439
1955–1964	218	26	192	1184	7447
1965–1974	67	43	24	1905	5566
1975–1984	—	15	−15	2208	3343
1985–1992†	—	33	−33	1539	1771
1904–1992	10918	1138	9780	8009	1771

*11-year period.
†8-year period.

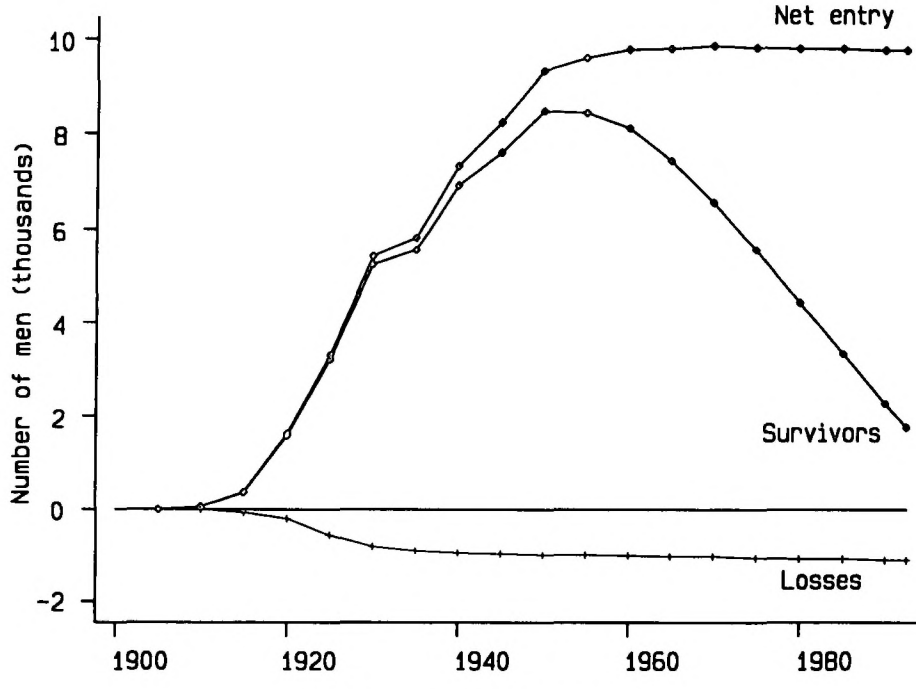

Fig. 1. Growth and survival, 1904–1992.

In Table 2 information is presented by quinquennium of birth. The first three lines give total entry, losses, and net entry. The 923 men untraced before 1935 came almost exclusively from among those born before 1906, so that losses were 16% of those born in 1891–1905, but only 5% of those born 1906–1920. Survival is presented in terms of age, and expressed as the percentage of net entry (or total traced). In this table, 'na' means that during the relevant five years of age there were not only further deaths but also some men who survived to the end of tracing before reaching the upper age; these numbers are shown in the last two lines of the table.

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Table 2. Survival by quinquennium of birth

	Quinquennium of birth					
	1891–1995	1896–1900	1901–1905	1906–1910	1911–1915	1916–1920
Total entry	1505	1938	1971	1816	1754	1934
Losses	299	329	244	144	72	50
Net entry	1206	1609	1727	1672	1682	1884
Number known to be alive at:						
age 55	996	1327	1423	1418	1443	1619
	(82.6)*	(82.5)	(82.4)	(84.8)	(85.8)	(85.9)
age 70	665	801	905	931	924	1083
	(55.1)	(49.8)	(52.4)	(55.7)	(54.9)	(57.5)
age 75	464	565	658	699	692	na
	(38.5)	(35.1)	(38.1)	(41.8)	(41.1)	
age 80	303	344	420	450	na	
	(25.1)	(21.4)	(24.3)	(26.9)		
age 85	167	183	208	na		
	(13.8)	(11.4)	(12.0)			
age 90	66	65	na			
	(5.5)	(4.0)				
age 95	21	na				
	(1.7)					
Further deaths by the end of tracing	13	45	97	188	201	204
Survivors at the end of tracing	8	20	111	262	491	879
	(0.7)	(1.2)	(6.4)	(15.7)	(29.2)	(46.7)

Table 3 shows the distribution of the 8009 deaths according to year and age of death, and by selected specific causes. A dash (—) indicates a void cell: before 1950, the oldest men in the cohort were still under 60 years of age; and no-one was over 84 before 1975. The codes in this, and all subsequent tables, are as in the Ninth Revision of the International Classification of Diseases after any necessary re-coding. However, for the 38 deaths found on investigation to be due to mesothelioma, no code is cited.

As shown in Table 4, almost 85% of the total entry to the cohort were first registered either in the mine and mill in Asbestos or at Company 3 in Thetford Mines, and another nearly 11% in the factory in Asbestos or at Company 4 in Thetford Mines; the remaining 523 men (less than 5%) were first registered at Companies 5–9. The 7628 deaths to the end of May 1992, excluding those of men first registered at Companies 5–9, are shown in Table 5 by decade and place of registration, together with Standardized Mortality Ratios (SMRs). In the period 1904–1909, a very small number of deaths was expected at all four places (total 0.75), but the first death occurred only in 1910. Among men from Companies 5–9, there were 324 deaths which, related to the expected number of 310.6, yielded SMR = 1.04. More detail would not be meaningful.

There were no deaths before 1918 among men from Companies 3 or 4, but 18 at Asbestos (mine and mill), half of them due to injury and poisoning (ICD cdes 800–999); these were also the causes of death of 11 men at the Asbestos mine and mill and

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Table 3. Deaths by year, age and cause of death

Cause of death (ICD code*)	Age at death	Year of death: Before 1950	1950–1974	1975–1984	1985–1992	Total	
All causes (001–999)	<65	857	1976	399	3	3235	
	65–74	—	1149	885	532	2566	
	75–84	—	280	773	724	1777	
	≥85	—	—	151	280	431	8009
Malignant neoplasms							
Oesophagus (150)	<65	1	10	2	0	13	
	65–74	—	5	5	0	10	
	75–84	—	5	3	0	8	
	≥85	—	—	1	0	1	32
Stomach (151)	<65	14	62	5	0	81	
	65–74	—	40	27	10	77	
	75–84	—	7	15	14	36	
	≥85	—	—	1	2	3	197
Small intestine, colon and rectum (152–154)	<65	7	47	8	0	62	
	65–74	—	28	22	12	62	
	75–84	—	5	17	29	51	
	≥85	—	—	7	5	12	187
Pancreas (157)	<65	5	20	5	0	30	
	65–74	—	13	12	6	31	
	75–84	—	3	11	6	20	
	≥85	—	—	1	0	1	82
Other abdominal (155–156; 158–159)	<65	3	17	2	0	22	
	65–74	—	13	10	4	27	
	75–84	—	1	8	10	19	
	≥85	—	—	0	1	1	69

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Larynx (161)	<65	4	14	0	0	18	
	65–74	—	1	6	2	9	
	75–84	—	1	9	3	13	
	≥85	—	—	0	0	0	40
Trachea, bronchus and lung (162)	<65	2	135	70	2	209	
	65–74	—	80	107	87	274	
	75–84	—	10	64	83	157	
	≥85	—	—	6	11	17	657
Mesothelioma (†)	<65	0	5	6	0	11	
	65–74	—	1	7	12	20	
	75–84	—	0	2	5	7	
	≥85	—	—	0	0	0	38
Other (Rem 140–209)	<65	24	124	21	0	169	
	65–74	—	82	79	64	225	
	75–84	—	28	74	67	169	
	≥85	—	—	7	28	35	598
Heart disease (391–392; 402; 404; 410–429)	<65	97	783	155	0	1035	
	65–74	—	471	307	163	941	
	75–84	—	107	312	228	647	
	≥85	—	—	57	83	140	2763
Cerebrovascular disease (430–438)	<65	11	93	19	0	123	
	65–74	—	104	78	24	206	
	75–84	—	42	71	62	175	
	≥85	—	—	14	24	38	542
Respiratory tuberculosis (010–012)	<65	174	60	0	0	234	
	65–74	—	12	2	0	14	
	75–84	—	1	1	2	4	
	≥85	—	—	0	0	0	252

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Table 3.—*continued*

Cause of death (ICD code*)	Age at death	Year of death: Before 1950	1950–1974	1975–1984	1985–1992	Total	
Pneumoconiosis (501–505)	<65	1	30	6	0	37	
	65–74	—	12	24	13	49	
	75–84	—	1	5	9	15	
	≥85	—	—	2	5	7	108
Other respiratory disease (Rem 460–519)	<65	67	76	23	0	166	
	65–74	—	64	70	42	176	
	75–84	—	19	74	62	155	
	≥85	—	—	21	43	64	561
Injury and poisoning (800–999)	<65	217	196	25	0	438	
	65–74	—	35	28	19	82	
	75–84	—	9	20	9	38	
	≥85	—	—	3	5	8	566
All other known causes	<65	176	256	47	1	480	
	65–74	—	159	94	60	313	
	75–84	—	38	83	114	235	
	≥85	—	—	29	62	91	1119
Ill-defined or unknown causes (799)	<65	54	48	5	0	107	
	65–74	—	29	7	14	50	
	75–84	—	3	4	21	28	
	≥85	—	—	2	11	13	198

*Code in the 9th revision of the International Classification of Diseases.
†See text for definition of mesothelioma in this table.

Table 4. Vital status at the end of follow-up, by place of first registration

	Total entry (a)	Lost (b)	Net entry (a)−(b)	Dead (c)	Survivors at end of tracing (a)−(b)−(c)
Asbestos					
Mine and mill	4503	472	4031	3331	700
Factory	792	84	708	553	155
Thetford Mines					
Company 3	4732	557	4175	3490	685
Company 4	368	10	358	305	53
	10395	1123	9272	7679	1593
Companies 5–9	523	15	508	330	178
Complete cohort	10918	1138	9780	8009	1771

Table 5. Deaths through 1992.05.31, with SMRs, by place of first registration (excluding Companies 5–9)

	Asbestos				Thetford Mines			
	Mine and mill		Factory		Company 3		Company 4	
	0	SMR	0	SMR	0	SMR	0	SMR
1904–1914*	8	2.45	0	0	0	0	0	0
1915–1924	54	2.11	0	0	32	1.14	0	0
1925–1934	88	1.20	9	0.85	74	0.94	5	0.60
1935–1944	145	1.27	20	1.16	169	1.35	17	1.55
1945–1954	224	0.96	26	0.69	257	1.09	27	1.39
1955–1964	484	1.04	99	1.32	534	1.17	43	1.14
1965–1974	807	1.06	129	1.03	824	1.13	83	1.42
1975–1984	909	1.06	156	1.02	950	1.16	72	1.11
1985–1992†	590	1.12	110	1.06	625	1.22	58	1.67
1904–1992	3309	1.08	549	1.05	3465	1.16	305	1.28

*11-year period.
†Period of 7 years 5 months.

13 at Company 3 in the period 1918–1924. In the same period, there were 20 coded 481 (pneumococcal pneumonia), 18 of men in their twenties, 17 of whom died in 1918; only two of these 20 deaths were of men from Company 3. One reason for the high SMRs for the decade 1934–1944, which covered much of World War II, was elevated rates of trauma, particularly of men from Asbestos. At the end of tracing, 59 nonagenarians were still alive, 13 of them aged 95 or older, two still living at 99. The 14 deaths at ages 95 or older were coded: 185, 250, 404, 411, 429, 436, 441, 486 (2), 496 (2), 518, 799 (2).

Deaths from all causes and from cancer at several selected sites are shown by place of registration, together with SMRs, in Table 6; necessarily, this table relates only to the period from 1950, when cause-specific reference rates became available, to May 1992. The SMRs (all causes) were closely similar to those calculated over the complete period 1904–1992; cf. Table 5.

The main part of Table 7 is concerned with the 136.62 thousand subject-years from the age of 55, but excluding Companies 5–9 [see Appendix line (i); they had, of course, been contributed after 1944]. They are shown by place of registration and in

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Table 6. Deaths from selected causes, 1950–1992,* by place of first registration, with SMRs

Place of first registration	Cause of death (ICD code †)									
	All causes (001–999)		Stomach cancer (151)		Other abdominal cancer (150; 152–159)		Larynx cancer (161)		Cancer of trachea, bronchus and lung (162)	
	0	SMR	0	SMR	0	SMR	0	SMR	0	SMR
Asbestos										
Mine and mill	2924	1.06	67	1.05	132	0.80	12	0.87	253	1.29
Factory	508	1.06	10	0.92	27	0.93	5	2.03	49	1.34
Thetford Mines										
Company 3	3080	1.16	90	1.46	165	1.03	14	1.04	280	1.45
Company 4	267	1.29	9	1.85	13	1.04	1	0.95	25	1.65
	6779	1.11	176	1.25	337	0.92	32	1.04	607	1.38
Companies 5–9	316	1.03	7	1.12	16	0.84	4	2.31	39	1.35
Total	7095	1.11	183	1.24	353	0.91	36	1.11	646	1.37

*1 January 1950–31 May 1992.
†Code in the 9th revision of the International Classification of Diseases.

Table 7. Subject-years (thousands) from the age of 55 on, by place of first registration and in relation to exposure accumulated to the age of 55

Exposure (mpcf.y)	Asbestos		Thetford Mines	
	Mine and mill	Factory	Company 3	Company 4
1945–1992*				
<3	12.13	3.31	6.71	0.04
3, <10	8.05	1.48	6.80	0.04
10, <30	8.60	1.48	7.61	0.39
30, <60	6.47	1.43	5.20	0.42
60, <100	6.27	1.14	4.86	0.67
100, <200	6.41	1.11	8.09	0.69
200, <300	3.37	0.40	5.33	0.71
<300	51.30	10.35	44.60	2.96
300, <400	2.43	0.08	3.08	0.56
400, <1000	5.92	0.39	8.07	1.02
≥1000	0.99	0.02	4.52	0.32
Total	60.64	10.84	60.28	4.86
1950–1992*				
Total	59.99	10.77	59.67	4.78

*The periods ended 31 May 1992.

relation to exposure accumulated to the age of 55. Just one-fifth (27.40 or 20.1%) were at exposures of at least 300 mpcf.y, but the proportions differed greatly: 4.5% at the factory in Asbestos; 15.4% at the mine and mill there; 26.0% at Company 3; and 39.1% at Company 4. The last line of this table gives the distribution by place of registration of the 135.21 thousand subject-years accumulated after 1949.

In Table 8, the 6161 deaths after the age of 55, in the period 1950–1992, are presented, with SMRs, by selected causes and according to exposure accumulated to the age of 55. The SMRs in the last line of this table are closely similar to those in the last line of Table 6. The apparently anomalous laryngeal cancer SMR of 3.12 for 300–400 mpcf.y reflects an excess (O–E) of 2.7 deaths; this was balanced by a shortfall of 2.3 deaths for higher exposures, so that, at exposures of 300 mpcf.y or more, the excess was 0.5 deaths and the SMR 1.09.

Pneumoconiosis and mesothelioma form the subject of Table 9, which shows the numbers of deaths from 1945 onwards and after the age of 55 attributed to these causes, by place of registration and in relation to exposure accumulated to the age of 55; also given are rates per 100 000 subject-years. The temporal restrictions excluded 12 deaths due to pneumoconiosis of men in their late 40s or early 50s, eight of them from Company 3, and one mesothelioma death at the age of 40 (see below). Also excluded were eight deaths from Companies 5–9, seven ascribed to pneumoconiosis and one due to mesothelioma. There was also one pneumoconiosis death that the PYRS program did not identify.

Analysis of deaths in relation to smoking habit was confined to the years after 1974, when the proportion of unclassifiable questionnaires became satisfactorily low. Table 10 gives the numbers of deaths from selected causes, with SMRs, among the 5274 men with acceptable smoking histories, in relation to four classes of cigarette smoking habit.

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Table 8. Deaths from selected causes, 1950–1992,* after age 55, in relation to exposure accumulated to the age of 55, with SMRs

	Cause of death (ICD code†)									
	All causes (001–999)		Cancer of stomach (151)		Other abdominal cancer (150; 152–159)		Cancer of larynx (161)		Cancer of trachea, bronchus and lung (162)	
Exposure (mpcf.y)	0	SMR	0	SMR	0	SMR	0	SMR	0	SMR
<3	1029	1.06	32	1.41	51	0.86	7	1.45	75	1.12
3, <10	718	1.03	22	1.38	39	0.91	6	1.71	64	1.27
10, <30	806	1.08	15	0.89	49	1.05	2	0.51	61	1.03
30, <60	590	1.06	13	1.07	31	0.90	1	0.34	60	1.32
60, <100	572	1.13	13	1.16	38	1.20	3	1.11	61	1.45
100, <200	707	1.13	16	1.15	31	0.79	2	0.59	67	1.27
200, <300	397	1.01	7	0.80	21	0.85	3	1.45	35	1.10
<300	4819	1.07	118	1.16	260	0.93	24	1.03	423	1.21
300, <400	263	1.10	7	1.29	11	0.73	4	3.12	29	1.46
400, <1000	731	1.24	16	1.21	33	0.90	2	0.64	88	1.84
≥1000	348	1.62	17	3.21	15	1.12	0	0	47	2.97
Total	6161	1.11	158	1.26	319	0.93	30	1.04	587	1.36

*1 January 1950–31 May 1992.
†Code in the 9th revision of the International Classification of Diseases.

Table 9. Deaths ascribed to pneumoconiosis and mesothelioma, 1945–1992,* after the age of 55, by place of first employment and in relation to exposure accumulated to the age of 55

	Asbestos				Thetford Mines			
	Mine and mill		Factory		Company 3		Company 4	
Exposure (mpcf.y)	0	Rate †	0	Rate	0	Rate	0	Rate
Pneumoconiosis								
<30	2	6.95	0	0	4	18.94	0	0
30, <100	4	31.40	0	0	1	9.93	0	0
100, <300	4	40.91	2	132.15	9	67.07	4	285.21
300, <400	1	41.13	0	0	5	162.28	1	177.56
400, <1000	10	168.87	1	256.45	14	173.43	2	196.41
≥1000	2	201.55	0	0	20	442.29	2	627.69
Total	23	37.93	3	27.67	53	87.92	9	185.20
Mesothelioma								
<30	2	6.95	0	0	0	0	1	212.85
30, <100	0	0	3	116.90	3	29.80	0	0
100, <300	2	20.45	2	132.15	9	67.07	0	0
300, <400	1	41.13	0	0	4	129.82	0	0
400, <1000	3	50.66	0	0	4	49.55	1	98.20
≥1000	0	0	0	0	1	22.11	0	0
Total	8	13.19	5	46.16	21	34.84	2	41.16

*1 January 1945–31 May 1992.
†Per 100,000 subject-years.

DISCUSSION

Methodology

Almost all those lost to view had been employed for such short times, typically one or two months, that virtually no useful information was lost. From 1950, when causes of death were first recorded in Quebec, there were only 122 losses among the 8484 men (less than 1.5%) known to be alive in that year. The fact that causes of death were not recorded until 1950 was of no great moment as lung cancer and mesothelioma, the most important causes for study, were almost unknown in Quebec for another decade. Moreover, censoring at the age of 85 was found to have very little effect—SMRs with and without such censoring only very occasionally differed by more than 0.01. For about 100 deaths, mainly before 1950, no information could be obtained about causes; the effects on patterns of cause-specific mortality after 1950 (perhaps 60 cases among over 7000 deaths, or less than 1%) would be quite small. Finally, although 57 deaths during the last seven months of 1992 were excluded, there was no indication that there was any material effect on the patterns of mortality.

Most men in the cohort had left the industry before reaching the age of 55 and exposure after that age of the minority who remained would be slight in comparison to that accumulated up to 55, the measure we adopted. This measure was not calculated for 241 men but, as these were only 2.2% of the cohort, their exclusion can have had little effect. However, entry into analyses in relation to exposure had to be on each man's 55th birthday (Liddell *et al.*, 1977), which meant that deaths before the age of 55 had to be excluded. Nevertheless, almost 80% of all deaths, and 90% of

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Table 10. Deaths from selected causes, 1975–1992,* in relation to cigarette smoking

Cigarette smoking habit (with number of men)	Cause of death (ICD code†)									
	All causes (001–999)		Stomach cancer (151)		Other abdominal cancer (150; 152–159)		Larynx cancer (161)		Cancer of trachea, bronchus and lung (162)	
	0	SMR	0	SMR	0	SMR	0	SMR	0	SMR
Non-smokers of cigarettes (1050)	722	0.88	13	0.93	42	0.88	2	0.54	33	0.55
Ex-smokers (1174)	707	0.96	6	0.46	42	0.92	6	1.56	49	0.72
Smokers of <20 cigarettes a day (1174)	793	1.24	20	1.74	36	0.90	4	1.16	87	1.43
Smokers of ≥20 cigarettes a day (1876)	1279	1.42	31	1.89	51	0.87	8	1.54	240	2.55
All classifiable (5274)	3501	1.13	70	1.27	171	0.89	20	1.23	409	1.45

*1 January 1975–31 May 1992.
†Code in the 9th revision of the International Classification of Diseases.

deaths due to lung cancer, were included, and the patterns in Table 8 are not likely to be greatly distorted. In interpreting Table 9, the exclusions of the early deaths, 12 ascribed to pneumoconiosis and one to mesothelioma, should be borne in mind. This last exclusion was a pathologically uncertain case, diagnosed without autopsy, and his death at the age of 40 was most unusual as the ages at death of the other 37 mesothelioma cases ranged from 57 to 79.

In attempts to simplify consideration of Tables 8 and 9, the relevant mortality or rate ratios were adjusted for differences in exposure. However, these adjusted ratios were found to be quite misleading, because they could not be further adjusted to take into account differences in age and calendar period, and so were abandoned.

For several purposes, the men first registered at Companies 5–9 were ignored. The mines were too disparate to be treated as one group, and too small to be considered separately, accounting in all for a tiny proportion of the cohort's experience. As the SMR (all causes) over their whole existence was close to unity, their inclusion could have had only very minor effects.

Epidemiology

Apart from one break, at the time of the recession, the cohort grew steadily from 1915 until 1949, at which stage the industry had ceased to recruit heavily from men born before the 1920s. Net entry had reached an effective plateau by 1960, and from the same year, the survival curve had the familiar shape (Fig. 1), and survival curves for men in each birth quinquennium were not dissimilar. Almost all men born from 1891 to 1900 had died by the end of tracing: survivors numbered only 28 (1.0% of 2815). For later birth quinquennia the percentages of survivors were 6.4, 15.7, 29.2 and 46.7%.

The most common causes of death were heart disease and stroke, which accounted for 3305 deaths (41.3% of 8009), followed by lung cancer (657, 8.2%), malignant neoplasms, except of lung and abdomen, and excluding mesothelioma (638, 8.0%), abdominal cancers (150–159: 567, 7.1%), injury and poisoning (566, 7.1%), and respiratory diseases other than tuberculosis and pneumoconiosis (561, 7.0%). There were 252 deaths (3.1%) from respiratory tuberculosis, 108 (1.3%) ascribed to pneumoconiosis and 38 (0.5%) due to mesothelioma. Lung cancer and mesothelioma were virtually unknown before 1960. On the other hand, respiratory tuberculosis, although not uncommon before 1950, was increasingly rare thereafter, with only five cases after 1974.

The proportion of the net entry who survived to the end of tracing, 18% for the complete cohort (1771 out of 9780), varied according to place of registration from 15% for Company 4 to 22% for the factory in Asbestos, and to 35% for Companies 5–9. This degree of variation is not fully reflected in the SMRs (1904–1992) for at least two reasons. First, as is well-known, SMRs are quite insensitive even to large fluctuations in death rates when the actual numbers of deaths are comparatively small. Thus many of the high decadal SMRs of Table 5 are 'lost' in the 1904–1992 ratios which are dominated by the SMRs for the last few decades. Secondly, as Companies 5–9 did not start operations until quite recently, all their recruits into the cohort were already survivors to the time of registration. High decadal SMRs before 1925 cannot be explained on the grounds that the reference mortality was not based on the population of Quebec. Rather it would appear that

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part of the explanation lies in casualties in World War I and in the 1918 influenza epidemic.

SMRs (all causes) were only slightly raised in Asbestos, but rather more so at Thetford Mines, particularly at Company 4. For stomach cancer, SMRs were quite low at Asbestos, but high in Company 3 and especially Company 4; however, all SMRs for other abdominal cancer were below, or close to, unity. Laryngeal cancer SMRs were generally close to unity, the only apparent exceptions being based on very few deaths. Lung cancer SMRs were elevated everywhere. There was little sign of any tendency for SMRs, whether for all causes or for the specified malignancies, to be higher the higher the level of exposure at least up to 300 mpcf.y: for all causes, the Spearman rank correlation coefficient, r_s (Armitage and Berry, 1994), over seven levels of exposure, was 0.14; for lung cancer it was 0.12; and it was negative for each of the other three cancers. A coefficient would have had to be greater than 0.57 to be 'significant' even at the 20% level.

Regardless of the degree of exposure up to 300 mpcf.y, *total mortality* was approximately 7% worse than in the general male population of Quebec, possibly reflecting socio-economic differences. At higher exposures, SMRs (all-causes) were elevated, particularly in the most severe exposure group. The two rather high *stomach cancer* SMRs for exposure less than 10 mpcf.y meant that, even up to 1000 mpcf.y, the trend was negative ($r_s = -0.27$); the excess mortality from this cause, averaging 17%, was probably due to socio-economic factors. In the group with extreme exposure, namely at least 1000 mpcf.y, the SMR of 3.21 was caused by an excess of less than 12 deaths. For *other abdominal cancer* and *laryngeal cancer* the correlation coefficients were negative over all 10 levels of exposure, with the cohort's mortality close to that of the reference population. The lack of trend of *lung cancer* SMR with exposure up to 300 mpcf.y suggests that the 21% excess was due to some other factor, probably smoking. At exposures over 300 mpcf.y, the excess of lung cancer was 80.4 deaths, but around one-fifth of this would have been due to smoking.

Pneumoconiosis death rates per 100 000 subject-years were clearly associated with exposure at the two main places of employment, but the exclusions from this table may have distorted these associations, and certainly make comparison between the Asbestos mine and mill and Company 3 particularly difficult. There is little sign of corresponding associations with mesothelioma.

Non-malignant respiratory disease, other than pneumoconiosis and tuberculosis, was not studied in the current follow-up, but information is available from earlier reports (McDonald *et al.*, 1980, 1993). From 1910 to 1975 there were 234 deaths from this cause; after 1950 and 20 years or more after first employment, there were 156 such deaths, SMR = 0.99; for exposures to the age of 45 of less than 300 mpcf.y, the SMR was 0.90; for higher exposures, the SMR was 1.62. In the years 1976–1988, there were 256 deaths from this cause, SMR = 1.05; for exposures to the age of 55 of less than 300 mpcf.y and 300 mpcf.y or more, the SMRs were 1.00 and 1.25, respectively. In these years, the effects of cigarette smoking were reflected by SMRs of 0.76 for non-smokers and ex-smokers and 1.37 for definite smokers.

There were strong relationships between smoking habit and SMRs for all causes and for three of the four selected cancers.

The findings from this cohort study, which are clear and consistent, having

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changed little since being first published 25 years ago, must again be put into a wider context. For lung cancer and mesothelioma, the diseases of primary concern, the risks observed are entirely in line with those reported for chrysotile miners in northern Italy (Balangero) and probably Zimbabwe (Rubino *et al.*, 1979; Cullen and Baloyi, 1991) and for chrysotile workers engaged in the manufacture of cement (Thomas *et al.*, 1982; Gardner *et al.*, 1986) and friction products (McDonald *et al.*, 1984; Newhouse and Sullivan, 1989). The risks for both diseases are very much lower than those experienced by South African and Australian crocidolite miners (Sluis-Cremer *et al.*, 1992; De Klerk *et al.*, 1989), South Dakota miners exposed to fibrous tremolite (McDonald *et al.*, 1986) and by workers in the manufacture or use of asbestos products containing varying concentrations of crocidolite or amosite (McDonald, 1990). The much higher risks associated with amphibole exposure in the circumstances mentioned thus seem beyond reasonable doubt. Less clear, however, are the reasons for apparently anomalous findings consistently reported for three asbestos textile factories, two in the U.S.A. and one in the U.K. (McDonald *et al.*, 1983a,b; Peto *et al.*, 1985). In all three, exposure–response findings for lung cancer approximate to those for amphibole workers although in one (Charleston, South Carolina) only chrysotile was used. In contrast, few mesotheliomas were found in the latter plant but many in the other two, where small, but significant, amounts of crocidolite were added. Two hypotheses—fibre length and the use of mineral oils—have been suggested to explain the high lung cancer risk in asbestos textile manufacture but neither has yet been convincingly proved or disproved.

In 1964 when the McGill research program on asbestos was initiated, no serious consideration was given to the possiblity that health in the Quebec production industry might be affected importantly by fibres other than chrysotile. Thus employees in the small manufacturing plant were included in our studies, although it was known that some crocidolite and amosite were used there. Only later was it appreciated that some cases of mesothelioma in the cohort had resulted from exposure to Australian crocidolite in the manufacture of filters for military gas masks (McDonald and McDonald, 1978). It had been realized that many employees at Company 1 moved between mine, mill and factory without this being recorded, but the probable effects of this were only recently evident from lung burden analyses which show crocidolite fibres in a high proportion of autopsies from Asbestos, but not from Thetford Mines (Case and Sébastien, 1987).

The observation in the early 1980s, confirmed subsequently, that most workers in the Quebec industry have, at death, more tremolite fibres in their lungs than chrysotile, especially at Thetford Mines (Rowlands *et al.*, 1982), is no longer of purely academic interest. The most reasonable explanation for the higher carcinogenic potential of amphibole fibres than chrysotile in man lies in their greater durability and biopersistence. There is now evidence that this may account for the higher rate of mesothelioma in cohort members from Thetford than Asbestos, and for workers in certain mines more than in others (McDonald and McDonald, 1995).

The as yet unknown explanation for the high lung cancer risks in textile manufacture and the role of fibre durability in carcinogenesis are directly relevant to the asbestos industry but perhaps even more so for those concerned with man-made mineral fibres. This should not prevent the straightforward interpretation of the

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results described in this paper on chrysotile as produced industrially in Quebec, contaminated as may be with tremolite and other amphibole fibres. These findings indicate that, except at very high levels of exposure, several orders of magnitude higher than any presently permitted, adverse effects on health will not occur. In view of the long latent period, averaging about 40 years, any current excess of asbestos-related mortality will have been due to exposure several decades ago, when exposure levels were often considerably higher; for example, despite a steady fall from an average of approximately 75 mpcf in 1948, the average in this industry in 1968 was still around 10 mpcf (Gibs and Lachance, 1972), roughly equivalent to 30 fibres/ml.

Exhaustive studies of mortality from lung cancer and mesothelioma are nearing completion and will be reported shortly.

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APPENDIX

Numbers of men, of subject-years and of deaths, in various classifications

	Place of first registration		
	All places	Asbestos + Companies 3 and 4	Companies 5–9
Complete cohort			
(a) Number of men	10 918 **[1,2]**	10 395 **[4]**	523 **[4]**
Subject-years (thousands):			
(b) to 1992.05.31	425.16	408.92 **[5]**	16.24
(c) 1950.01.01–1992.05.31	251.82	237.01 **[6]**	14.81 **[6]**
Number of deaths:			
(d) to end of tracing	8009 **[1,2,3]**	7679 **[4]**	330 **[4]**
(e) to 1992.05.31	7952	7628 **[5]**	324
(f) 1950.01.01–1992.05.31	7095	6779 **[6]**	316 **[6]**
After exclusion of 236 men not employed in the industry before age 45 and five men with inconsistent work histories			
(g) Number of men	10.677	10.265	412
Subject-years (thousands):			
(h) to 1992.05.31	419.87	405.86	14.02
(i) to 1992.05.31, after the age of 55	142.63	136.62 **[7,9]**	6.01
(j) 1950.01.01–1992.05.31, after the age of 55	141.22 **[8]**	135.22 **[7]**	6.00
Number of deaths:			
(k) to 1992.05.31	7756		
(l) 1950.01.01–1992.05.31, after the age of 55	6161 **[8]**	5961	200

Bold figures in square brackets indicate text tables based on the relevant articles.

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