The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec

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The results of studies of respiratory symptoms and function, roentgenographic changes, and mortality in relation to dust exposure in the Quebec chrysotile industry, which has employed some 28,000 workers, are brought together and their implications for control examined.

Breathlessness on exercise, diminished inspiratory capacity, parenchymal and pleural changes, and respiratory disease mortality were related to dust exposure and to each other. Respiratory cancer was also related to dust exposure. The overall excess of deaths from respiratory cancers, including five with malignant mesothelioma, was at most 50% above expectation, based on age-specific rates for Quebec and the mining region.

If safety standards are set, they should be based on epidemiological evidence. From these data for the chrysotile producing industry of Quebec, a resonable figure, based on a 1% risk of acquiring clinically significant disease, would lie between 2 to 4 million particles per cubic foot, calculated for a working life of 50 years. In the absence of epidemiological findings based on fiber counts and lack of a satisfactory means of conversion, particle counts should continue to be used for control in this industry.

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In recent years, exposure to asbestos dust has been widely recognized as an important etiological factor in pulmonary fibrosis, cancer of the lung, and malignant mesothelial tumors. The precise nature of the causal association has usually been obscured by exposure to more than one form of asbestos and to other materials in the manufacture or application of asbestos products. In the mining and milling industry of Quebec, where almost half the world's supply of chrysotile is produced, exposure to other types of asbestos or to materials used to make asbestos products has been very slight. To assess the effects of pure exposure to chrysotile, an epidemiological study in this industry was started in 1966. Our aims were to relate dust exposure assessments to mortality, roentgenographic change, pulmonary function, and respiratory symptoms. Results of these studies have been presented in separate papers on mortality,[1] roentgenographic change,[2] pulmonary function,[3] and respiratory symptoms.[4] The purpose of this report is to bring together these and certain subsidiary findings and to discuss their implications for control.

Outline of Study

Records of the mining companies were used to identify all persons known to have worked in the industry since its inception in 1878. Earlier records tended to be incomplete or missing and certain sets had been destroyed, but altogether some 23,000 employees, mostly men, were identified and information was assembled for this group. Of these, 6,400 were working on Nov 1, 1966, the registration date for the study. The information collected for each person consisted of date of birth and a full industrial history. The latest chest roentgenogram of each employee, if available, was provided by the industrial medical clinics and read by an international group of six readers using the UICC/Cincinnati (now

Table 1.—Number of Men in Dust Exposure Analyses

Analysis	Dust Exposure Level, mpcf-yr						
	<10	10	100	200	400	600+	Total
Mortality study	2,810	3,[illegible]	1,124	1,047	637	[illegible]05	9,692
Roentgenographic study	1,[illegible]37	1,522	1,139	912	713	707	6,529
Ventilation and respiratory symptoms	91	455	159	134	109	67	1,015
Smokers	[illegible]0	331	144	117	93	50	805
Nonsmokers	[illegible]1	73	15	17	13	8	159

Table 2.—Roentgenographic Change Prevalence Percentages

Roentgenographic Change	Dust Exposure Level, mpcf-yr <10	10	100	200	400	800+	Average
Thetford Mines							
Irregular small opacities							
1/0	1.8	3.3	6.3	8.7	12.0	17.2	7.6
2/1	0	0.2	2.2	0.9	2.4	9.2	2.0
Pleural thickening							
Grade 1	2.4	4.5	6.5	5.8	8.3	10.5	6.4
Grade 2	1.3	0.7	0.8	1.2	1.3	2.0	1.1
Asbestos							
Irregular small opacities							
1/0	6.4	3.7	6.3	5.0	6.8	7.0	5.1
2/1	0	0.4	1.5	0.8	0.6	3.9	0.8
Pleural thickening							
Grade 1	2.9	2.6	3.0	3.0	4.7	5.8	3.2
Grade 2	0.0	0.6	1.0	0.5	1.4	0.7	0.7

Rates are standardized for age and years in industry. Males were 36 to 65 years of age at time of roentgenogram.

Table 3.—Age-Corrected Death Rates Per 1,000 Men Born 1891-1920: Deaths to December 1969

Cause	Dust Exposure Level, mpcf-yr <10	10	100	200	400	800+
All causes	365	355	354	313	323	395
Respiratory cancers*	10.3	13.1	13.4	15.5	21.4	32.1
Abdominal cancers	18.0	13.6	18.7	11.6	26.3	28.7
Pneumoconiosis	1.6	1.5	0.8	4.9	4.9	23.6

*Includes malignant pleural mesothelioma.

Table 4.—Relative Risk of Death From Respiratory Cancer in Men by Dust Group, Estimated From Retrospective Analysis

Dust Exposure Level, mpcf-yr	No. of Cases	No. of Controls	Relative Risk
< 10	32	186	1.0
10	41	188	1.3
100	13	39	1.9
200	14	61	1.3
400	15	40	2.2
800+	19	22	5.0
Total	134	536	

Table 5.—Age-Corrected Mortality Per 1,000 Men From Respiratory Cancer and Pneumoconiosis at Thetford Mines and Asbestos

	Dust Exposure Level, mpcf-yr <10	10	100	200	400	800+
Respiratory cancer						
Thetford Mines	9	12	18	15	24	31
Asbestos	11	13	8	18	13	43
Pneumoconiosis						
Thetford Mines	4	1	1	7	6	24
Asbestos	0	0	0	5	3	24

UC/ILO classification.[7] A follow-up of ex-employees was set up and death certificates and autopsy reports were examined for those who had died. One section of this article deals with results in women; the rest is concerned with men only.

Dust Exposure.—Dust exposure levels were determined for each recorded job within the industry, and for each year of employment. These levels were used to calculate a total dust exposure index for each worker. Gibbs and Lachance[8] described the techniques used and Gibbs[9] dealt further with certain qualitative aspects. The main index was based on particle counts from midget impinger samples, expressed as millions of particles per cubic foot (mpcf). A total dust exposure index was calculated for each worker by multiplying the measurement of dust exposure by the number of years of exposure at that level, and was expressed as mpcf-years.

The results which follow are presented in terms of range of total dust exposure based on particle counts (Tabe 1). Table 1 also lists the number of men included in the main analyses considered in this report.

Since disease is probably caused by asbestos fibers rather than dust particles, it has been recommended that standards for occupational exposure be based on fiber counts obtained by the membrane filter technique.[10] If there were a reasonably consistent relationship between particle counts by midget impinger and fiber counts by membrane filter, the results of our surveys could also be presented and interpreted in terms of fiber counts. A study was carried out by Gibbs and Lachance[11] in which 87 side-by-side midget impinger and membrane filter measurements were made at nine locations in each of five mines and mills of the Quebec chrysotile industry. The correlation between the two types of measurement was too poor to justify the use of any single conversion factor.

Roentgenographic Changes.—The two main roentgenographic indices of response to asbestos exposure are irregular small opacities and pleural thickening. The prevalences of these and of all other roentgenograp

changes were higher in the Thetford Mines area than in the Asbestos area of Quebec, although the two areas are only 50 km apart and the asbestos mined is geologically similar. The association of the changes with dust exposure was also stronger at Thetford Mines than at Asbestos.

Table 2 shows that at Thetford Mines the prevalence of irregular small opacities of category 1 or more rose steadily with increasing dust exposure whereas for category 2 or more the relation to dust exposure was less marked, except for the considerable rise in the most exposed workers. Even with allowance for differences in dust exposure, the prevalence of category 1 or more was markedly age-related, rising to 14.8% for those 61 to 65 years old. This age effect was much lower for category 2 or more, and amounted to only about one third of the change associated with dust exposure.

At Asbestos, the prevalence of irregular small opacities of category 1 or more rose to 9.2% for those aged 61 to 65, and there was virtually no relation to dust exposure. With respect to only category 2 or more, the relation to age was much lower and prevalence increased among those with the highest dust exposure.

For pleural thickening, the effect of age was slightly greater than the effect of exposure on the prevalences of grade 1 or more, and of grade 2 or more, for both mining areas.

The biggest difference among all the roentgenographic features was in pleural calcification, for which the prevalence of grade 1 or more was 0.4% at Asbestos and 5.2% at Thetford Mines. In spite of the relationship to dust exposure, no correlation exceeded 0.3. This is largely a reflection of the very high proportion of men who showed no roentgenographic change. Some of the differences between the two areas were not surprising, as overall dust exposure levels at Asbestos were considerably lower than at Thetford Mines, but others cannot yet be explained in terms of dust exposure or geology. If variation in fiber content of dust were an important factor, the results in mine workers, for whom the fiber proportion would be low, should differ from those for mill workers, in whom the fiber proportion would be higher. Altogether, there were 5[illegible] workers who had worked for ten or more years entirely in mining or entirely in milling. Analysis of roentgenographic changes in these men showed that mill workers had a slightly higher prevalence of irregular small opacities, but the differences between Thetford Mines and Asbestos remained. No other consistent differences could be detected between the mine and mill workers.

Mortality.—This study was limited to those who had worked for one month or more, and who were born 1891 to 1920. This age group was selected because the records of older persons were frequently incomplete and few of the younger ones had died. An initial analysis[1] was limited to deaths before Nov 1, 1966, but the follow-up will continue until at least 1974. By the end of December 1969, 87.5% of the 11,572 persons in the cohort and 99% of those who had worked ten years or more had been traced.[2] Of these, 3,270 had died, comprising 65.4% of those born 1891 to 1895 but only 9.8% of those born 1916 to 1920.

The age-standardized mortality per 1,000 men for certain causes of death are given in Table 3. Cancer of the lung showed a rising rate with increasing dust exposure, particularly in the two highest dust exposure groups. Cancers of the gastrointestinal tract also showed a rise in the two highest, and pneumoconiosis in the highest categories of dust exposure. Of 134 deaths in men from respiratory cancer, five were from pleural mesothelioma. These cases showed no clear relationship with dust exposure. There were no peritoneal mesotheliomas. Mortality from all causes fell with increasing dust exposure up to the highest dust group, probably because those who died young could not attain a high dust exposure. On the basis of Quebec death rates, the expected number of respiratory cancer deaths in the cohort was 139, or about 9% on the basis of estimated rates in the mining region. This suggests that mortality from respiratory cancer in the Quebec chrysotile industry as a whole has been at most 50% above expectation and probably about 27%.

The primary method of analysis used in our report on deaths up to 1966[1] had two main weaknesses. The first was that length of exposure might well have been related to length of survival and thus might tend to obscure differences in mortality between exposure groups. We looked for evidence of such an interaction using the parametric approach of Berry,[3] and though we failed to find a significant effect, the possibility remained. The second weakness lay in the fact that deaths accumulated over many years were used in a single calculation of mortality. It is reassuring that our subsequent analysis,[2] which dealt with deaths over a three-year period, 1967 through 1969, gave essentially the same results. To some extent these problems are somewhat academic, since the total excess mortality in the industry from respiratory cancer, compared with mortality of the general population, was so small. The data are of practical importance nevertheless in defining the form of the dose-response relationship, essential for the setting of safety standards.

For this reasoson, when the follow-up is eventually completed, an analysis based on man-years of exposure[4] is planned to minimize these possible errors. Meantime, another method of analysis (G. Eyssen, MSc, and F. D. K. Liddell, MA, unpublished data) has been employed that, we believe, eliminates certain of these problems, though it does not make full use of the data available and provides only estimates of relative rather than absolute risk. For this analysis, the dust exposures of the 134 men included in the 1969 analysis of respiratory cancer mortality were compared with a sample of men, four for each case, selected at random among persons living at the time of the death of the respiratory cancer case and born in the same year. The distribution of cases and controls by dust exposure category is presented in Table 4. It can be seen that the pattern of relative risk obtained by this approach is quite similar to that for respiratory

Table 6.—Mortality From All Causes and Roentgenographic Changes at Thetford Mines

Roentgenographic Changes	No. of Observed and Expected Deaths by Year of Birth Cohort* 1891-1895 Obs	Exp	1896-1900 Obs	Exp	1901-1905 Obs	Exp	1906-1910 Obs	Exp	1911-1915 Obs	Exp	1916-1920 Obs	Exp	Total Obs	Exp
Parenchymal changes only	17	10.6	7	7.8	4	3.9	11	4.5	7	1.6	2	1.6	48	30.0
Pleural changes only	11	10.3	15	10.8	10	9.2	5	5.2	2	3.4	2	2.4	45	47.3
Both parenchymal and pleural changes	14	11.4	17	12.7	8	6.6	6	2.2	3	0.6	1	0.2	49	33.7

* Expected number was calculated from death rates in men without roentgenographic change.

cancer mortality shown in Table 3.

It seemed possible that the roentgenographic differences between Thetford Mines and Asbestos might also be reflected in mortality. There was little evidence of this. The age-standardized mortality per 1,000 population, for all causes, all malignant neoplasms, all respiratory diseases, and all circulatory diseases were 342, 54, 20, and 120, respectively, at Asbestos and 362, 61, 22, and 121, respectively, at Thetford Mines. All detailed comparisons showed the same similarity. For example, in Table 5, the age-standardized mortality by dust exposure is presented for respiratory cancer and pneumoconiosis. The only apparent difference is that the respiratory cancer rate rose in relation to dust slightly earlier but to a less extent at Thetford Mines.

Mortality and Roentgenographic Changes.—Of 10,120 persons traced in the mortality study, 9,692 were men and of these 5,082 had chest roentgenograms and 785 had died. At Thetford Mines, there were 354 deaths in 2,448 men traced. Death rates were calculated by date of birth, dust exposure, and the presence or absence of parenchymal or pleural roentgenographic changes. The rate from all causes combined was increased in the highest dust exposure group but, with allowance for exposure and date of birth, those whose roentgenograms showed parenchymal changes had a higher mortality (220/1,000) than those without roentgenographic changes (131/1,000) or with pleural changes only (126/1,000).

In Table 6, the observed number of deaths by year of birth and roentgenographic change is compared with the expected number based on those without roentgenographic change. Of 97 deaths in those with parenchymal changes, 33 were in excess of the expected figure. Considering only deaths from respiratory disease, including tuberculosis and cancer, we calculated in a similar way that there were 32 deaths in those with parenchymal change compared with eight expected, an excess of 24. Thus of the 33 excess deaths in this group at Thetford Mines, 24 were attributed to respiratory causes.

At Asbestos, the difference between the mortality of those with and without roentgenographic changes was less. Calculations similar to those used for Table 6 showed that only seven of the 431 total deaths were associated with parenchymal change, and of these 2.4 were attributable to respiratory causes.

Dust exposure levels at Thetford Mines were much higher than at Asbestos; thus, in the highest exposure group there were 356 and 69 men, respectively, on whom roentgenogram had been performed and who had been subsequently traced. Though the excess death rate in this highest exposure group was about 9% at both places, the numbers of excess deaths were therefore 35 and 6 respectively.

Calcified pleural plaques were not related to mortality as there were 60 deaths in persons with pleural plaques compared with an expected 59 deaths.[17]

Pulmonary Function.—A total of 1,015 current employees underwent pulmonary function studies during the summers of 1967 and 1968.[15] The sample chosen for study was stratified to include a higher proportion of older than younger workers, as the former were more likely to show changes in function. These tested comprised 81% of the sample selected.

The variation in some of the lung function indices by dust level in smokers and nonsmokers is shown in Fig 1, 2, and 3. The results of each test were standardized to age 50 years, height 170 cm, and weight 70 kg. Total lung volume fell slightly with increasing dust exposure but exposure had little effect on other

Table 7.—Age-Corrected Prevalence Percentages by Dust Exposure and Cumulative Smoking Habits

		Dust Exposure Level, mpcf-yr <10	10	100	200	400	800+
Bronchitis	Never smoked	10	19	19	46	21	45
	Up to 100 cigarette-years	18	0	12	0	45	0
	100-499	41	28	21	33	42	43
	500-999	32	38	44	28	47	58
	1,000	100	47	45	55	54	35
Breathlessness	Never smoked	0	14	24	31	13	44
	Up to 100 cigarette-years	0	12	39	21	41	43
	100-499	16	14	8	22	42	18
	500-999	9	19	21	13	14	29
	1,000	0	23	27	31	34	40

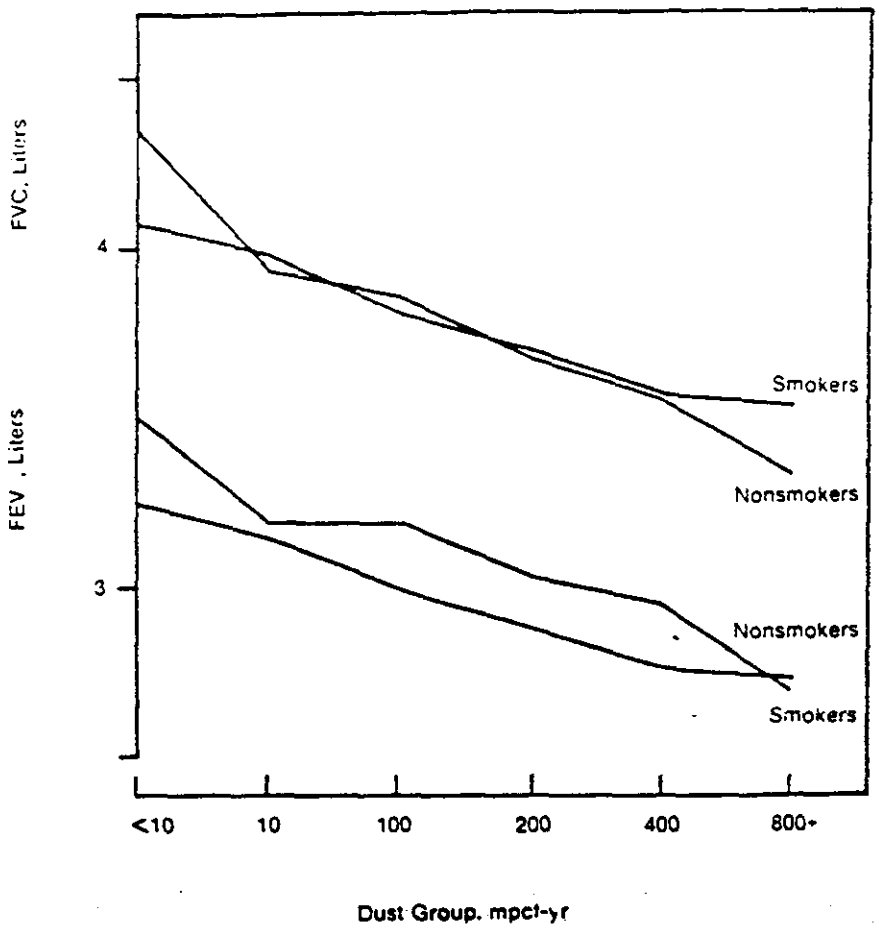

Fig 2.—Variation in forced expiratory volumes with dust group, standardized for age, height, and weight.

the same age and year of death but who had never been employed in the industry. There was a higher proportion of nonsmokers in patients than controls, which suggests that pulmonary cancer may be caused by asbestos exposure in the absence of smoking. The controls were on average heavier smokers, which reinforces the point but does not rule out the possibility of synergism between asbestos exposure and cigarette smoking. Clear evidence was obtained from a survey of all known cases of malignant mesothelioma in Canada that this disease was not related to smoking.[54]

Women.—The number of women ever employed in the Quebec chrysotile industry is small. In the mortality study, 428 of 465 women in the cohort were traced and of these 54 had died. Only 79 women had worked for more than ten years, and few had been exposed heavily to asbestos dust. One death was ascribed to lung cancer and none to pneumoconiosis.

Chest roentgenograms were available for 294 women, most of whom were 30 years old or less. The only roentgenographic changes recorded were one subcategory 0/1 rounded small opacities, one subcategory 0/1 irregular small opacities, and one grade 1 ill-defined cardiac outline.

Comment

Although death is the most definite and serious manifestation of exposure to asbestos, it is not the most sensitive. Excess deaths related to exposure altogether were probably in total no more than 2% of the 3,270 deaths in the cohort study. Most of these were attributed to respiratory cancer or pneumoconiosis and almost all were in the two highest dust exposure categories. Thus, excess mortality was virtually confined to men with exposure equivalent to at least 400 mpcf-yr. Detailed examination of mortality showed no particular cause, except pneumoconiosis, with a rate above that of the general population of Quebec.

An investigation was made of the 236 known cases of malignant mesothelioma in Canada, 1960 through 1970. The manner of death certifications of these patients were examined,[55] and the pathological findings reviewed in detail by the Mesothelioma Panel of the Canadian Tumour Reference Centre.[55,56] Epidemiological inquiries showed that 23% of men and 1% of women had definite or probable occupational exposure to asbestos, and a further 1% of men and 6% of women had lived in the home of an asbestos worker. In the remaining cases, none had ever lived within 32 km of an asbestos mine or mill. Of all known cases of mesothelioma in Canada, only nine have been associated in any way with the Quebec chrysotile mining and milling industry. Seven (five in the cohort) had been employed and two were women whose fathers had worked in the industry.

These cancer rates are very low in comparison with the studies of New York insulation workers[44] and the London crocidolite factory workers,[45] in both of which there were much higher rates of malignant neoplasms, particularly of mesothelioma. Other studies in the Soviet Union[46] and Italy[47] support the view that only high levels of exposure to chrysotile during mining and milling have an appreciable effect on mortality.

Although roentgenographic changes had some relationships to dust exposure, the clinical significance of the minor changes which were also related strongly to age is uncertain. Irregular small opacities of category 2 and pleural thickening of grade 2 are usually considered of clinical importance, and these were much less influenced by age. Changes of this order occurred in 1.0% and 0.7% of the entire working population. In men aged 61 to 65, employed an average of 20 years in the industry, the rates were 5.0% and 2.5% at an average exposure level of 13 mpcf. These rates are very

functional residual capacity (FRC) or residual volume (RV). Thus exposure to chrysotile affected only the inspiratory capacity portion of the total lung volume (TLV). Forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) both declined with increasing exposure, the FVC vital capacity falling by about 15% in the highest dust group and the FEV rather less. Neither steady state nor single-breath diffusing capacities showed any effect of exposure at rest; on exercise, the steady state diffusion declined slightly.

Analysis of the relation between lung function and roentgenographic changes showed that in those with small opacities of category 2 or more there was an average reduction of 20% in three indices: FRC, RV, and single-breath diffusing capacity at rest. Most indices showed a progressive reduction with increasing category of small opacities, but only VC and FVC showed significant reductions in association with the earliest roentgenographic changes. Pleural changes were also associated with reductions in pulmonary function, which ranged from about 3% in those without parenchymal change to about 6% in those with advanced parenchymal change. An additional small survey showed that changes in pulmonary mechanics possibly precede roentgenographic and other function changes.[8] Detailed analysis of those aged 61 to 65 showed that those with an obstructive lung function profile, ie, RV and TLC greater than expected and flow rates less than expected, had had heavier dust exposure, more symptoms of bronchitis, and more irregular small opacities than those with normal or restrictive patterns of pulmonary function.[14]

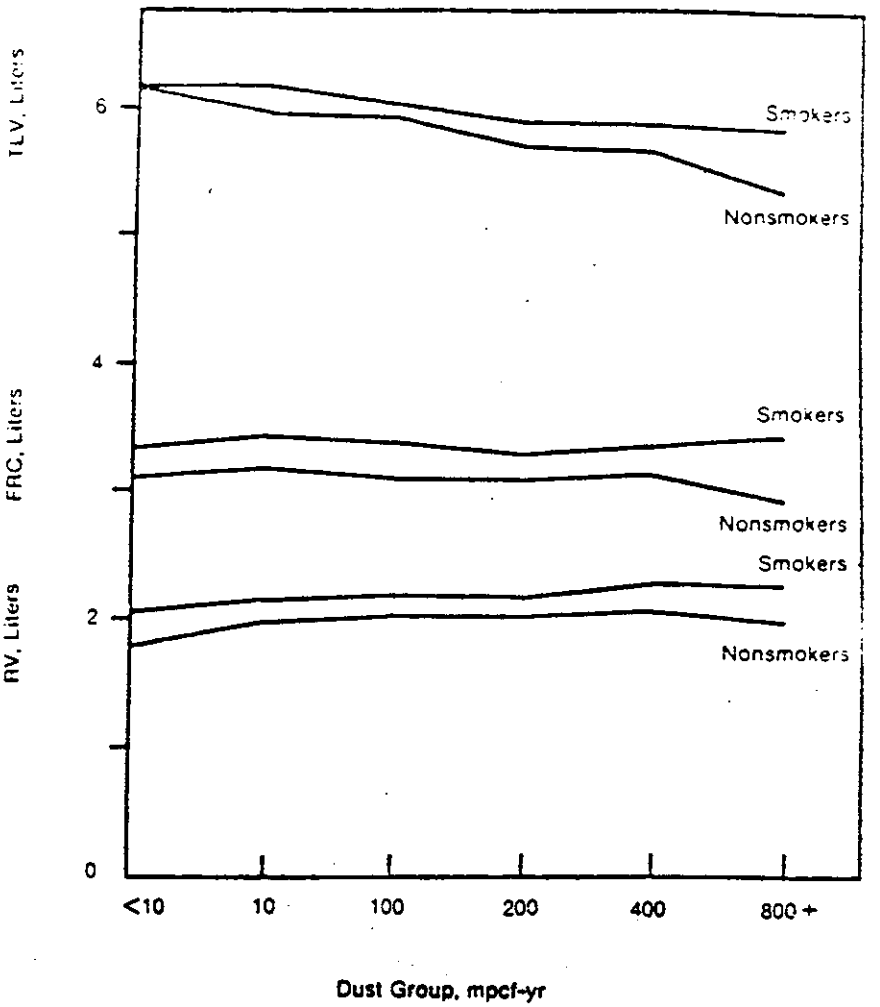

Fig 1.—Variation in lung volumes with dust group, standardized for age, height, and weight.

Respiratory Symptoms.—Each of the 1,015 workers in the function survey answered a slightly modified version of the Medical Research Council (MRC) respiratory questionnaire given in French or English by a bilingual interviewer. Both persistent cough and phlegm (bronchitis) and breathlessness on exercise were related to exposure.[7] Table 7 shows, however, that after standardizing for age, the prevalence of bronchitis rose to about 50% in both the highest dust and highest smoking categories. Thus, the effect of heavy exposure and heavy smoking together were the same as that of either separately. The same relationships have been observed in other dusty trades.[15] In contrast, the prevalence of breathlessness on exercise was unaffected by smoking, but increased steadily with dust exposure.

Effect of Smoking.—Smoking habits were originally determined only for employees in the pulmonary function survey. Figures 1, 2, and 3 show that, with many of the physiological indices, the effect of smoking was greater than the effect of exposure to asbestos dust. This was most obvious in FRC, RV, and perhaps in the steady-state diffusing capacity at rest. Forced vital capacity and FEV_1 were both decreased to a similar extent with increasing dust exposure, but only FEV was lower in smokers. As mentioned above, smoking and dust exposure were both related to the prevalence of bronchitis, but smoking was not associated with breathlessness.

Attempts are now being made to collect smoking histories in the cohort study of mortality, both for those who are still alive and for those who are dead. This may eventually provide prospective and retrospective evidence on the interrelationship of smoking and asbestos exposure. A controlled retrospective study[16] has been made based on deaths from lung cancer in the cohort study. Each patient was matched with a death from lung cancer from the same hospital records in a man of approximately

similar to those found in the chrysotile mining and milling industry in Cyprus, but are much lower than those in the New York insulation workers,[7] or in the British Royal Naval Dockyards.[8]

For pulmonary function and respiratory symptoms, the effect of smoking was generally greater than the effect of exposure to asbestos. However, for inspiratory capacity, forced flow rates, and breathlessness on exercise, relationships to dust exposure were found. With respect to these indices, a 10% reduction in nonsmokers occurred after a total exposure in excess of 100 mpcf-yr. This is close to the lower limit of the dust category at which 1% of subjects had grade 2 roentgenographic changes.

In the Quebec studies, information on dust exposure was obtained, whereas in most other studies quantitative information was not available. Murphy et al [9] found 11 cases of asbestosis in 101 New England shipyard workers with an average exposure of 120 mpcf-years, compared with one in a nonexposed control series. These workers appear to show a larger effect of asbestos exposure than those engaged in chrysotile production. However, the main constituent of the insulating materials used was amosite, with some chrysotile and no crocidolite.

Dust exposure assessments were also made for the Cyprus mining and milling industry,[5] and the rates of roentgenographic change for the same exposure levels were quite similar to those in Quebec. Exposure indices have also been assessed in a study of two asbestos-cement plants in the United States by Enterline and Weill,[7] where the main exposure has been to chrysotile and silica, but the results are not yet available.

The limit standards for occupational exposure to asbestos are based solely on the airborne concentration of dust or fiber, averaged over a lifetime's work, and the results in this report have been presented from this point of view. There was some evidence in our studies that longer exposures for the same total dust levels are associated with slightly higher prevalence rates of roentgenographic

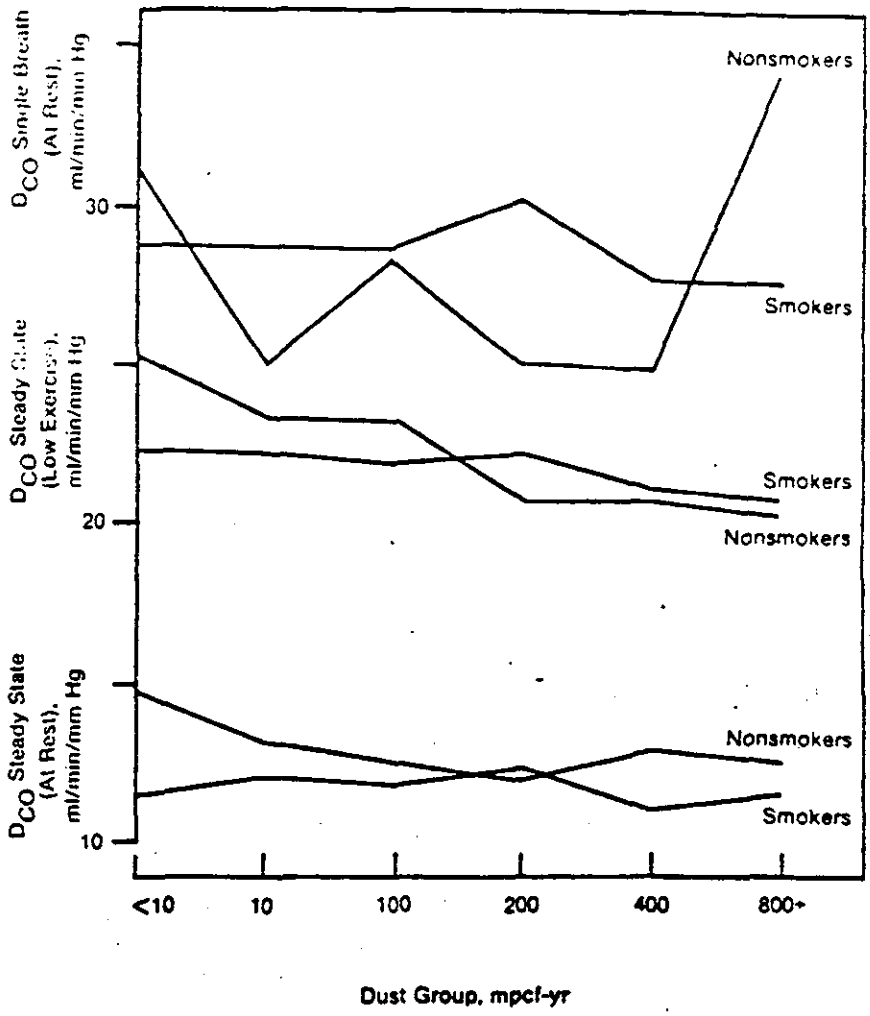

Fig 3.—Variation in diffusing capacity with dust group, standardized for age, height, and weight.

change. However, the roentgenograms were all taken during working life, so the correlation between total dust and years of exposure or years since first exposure is high, and the separation of the contributions from these two factors, duration and concentration, would be very difficult. The cohort study also showed that respiratory cancer mortality was greater in those who took more years to reach a given dust level. Therefore, in assessing the overall effect of asbestos exposure, duration of exposure should not really be ignored. For this reason, it would be unwise to apply the results of this or other descriptive epidemiological studies to widely different exposure patterns.

The British dust standard for chrysotile of 2 fibers per milliliter, averaged over three months, is based on the concept that a 1% risk of acquiring clinically significant disease in a 50-year working lifetime of exposure is acceptable. Considering all facets of disease—death, roentgenographic changes, pulmonary function changes, and respiratory symptoms—the 1% risk is reached by men in our third dust exposure category (100 to 200 mpcf-yr).

The relationships that exist between measurements of total dust and fiber exposure are thus of critical importance if standards are to be set for asbestos production and other asbestos industries. Such standards should be based on dose-response relationships established by sound epidemiological inquiries. To date, very few of those have included any quan-

titative assessments of dust, let alone of fiber exposure. Though safety standards expressed in fiber concentrations have theoretical and conceptual attractions, there is as yet little or no direct epidemiological evidence on which to base them. Our studies appear to provide a reasonable basis for establishing safety standards for chrysotile mining and milling in terms of dust concentration. Without more evidence on the conversion factor that should be applied in different parts of the industry, we cannot express our results confidently in fiber counts. Further efforts to find a satisfactory means of converting midget impinger to fiber counts are merited but, until new epidemiological evidence based on fiber counts has been assembled, it appears unwise to discontinue use of particle counts for control in this industry.

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