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RELATIONSHIP OF MORTALITY TO MEASURES OF ENVIRONMENTAL ASBESTOS POLLUTION IN AN ASBESTOS TEXTILE FACTORY

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Abstract—Three groups of men and women employed at a Rochdale asbestos textile factory have been followed to 30 June 1983 and their mortality compared with that expected at national and Rochdale rates, i.e. (i) 145 men first employed before 1933 who had served 20 yr or more in scheduled areas, (ii) 283 women first employed in 1933 or later who had served ten or more years in scheduled areas, and (iii) a principal cohort of 3211 men first employed between 1933 and 1974. Systematic measurements of the ambient pollution in the workplace have been made since 1951 either in terms of particles ml^{-1} or of fibres (of specified size) ml^{-1} and estimates have been made of the average concentration of particles (or fibres) to which individual members of the principal cohort were exposed. From these data estimates have been made of the quantitative relationship between ambient exposure and mortality from lung cancer and mesothelioma. These results, together with other published data, suggest that the lung cancer rate among chrysotile textile workers is approximately doubled following a cumulative exposure of 100 fibre ml^{-1} yr, irrespective of age at first exposure. Chrysotile asbestos was used in the factory throughout, but some crocidolite (approx. 5.0%) was used between 1932 and 1968 and this may have contributed, perhaps substantially, to the observed mesothelioma risk. For the purpose of risk prediction we have rather arbitrarily assumed that the mesothelioma risk caused by a similar concentration of pure chrysotile would have been roughly half that observed in this factory. This implies that 35 yr chrysotile exposure at an average concentration of 1 fibre ml^{-1} might eventually cause mesothelioma in about one worker in 200 if exposure began at age 20, although the risk would be substantially lower if exposure began later in life. If enforcement of the current U.K. control limit for chrysotile of 0.5 fibre ml^{-1} results in an average level of 0.25 fibre ml^{-1} in the workplace, these estimates suggest that the risk of eventually developing asbestos-related lung cancer or mesothelioma to a man first employed at age 20 who works in the asbestos textile industry for 35 yr may be about 0.8%. This prediction is of doubtful accuracy, however, as the exposure data were derived largely from static particle counts and the correlation between particle and fibre counts is poor.

INTRODUCTION

QUALITATIVE evidence of the risk of cancer from exposure to asbestos has been reported on numerous occasions and has frequently been reviewed (see, e.g. ACHESON and GARDNER, 1979; ROYAL COMMISSION, 1984; NATIONAL RESEARCH COUNCIL, 1984).

There is, however, very little precise evidence of the size of the risk related to specific measurements of pollution that is of any value for determining the relationship between dose and effect. One of the few factories which might provide such evidence is a textile factory in Rochdale which has used predominantly chrysotile, where two of us work (TG and RC) and where measurements of pollution have been made systematically for more than 30 yr. The factory has been the subject of several reports (Doll, 1955; Knox *et al.*, 1965; Knox *et al.*, 1968; Peto *et al.*, 1977; and Peto, 1978 and 1980). These, however, have been restricted to observations on small and selected groups and only average dust levels have been included in them, except for one small case-control study of recent employees who had developed lung cancer (Peto, 1980). The same factory featured (unnamed) in two reports by the British Occupational Hygiene Society (1968, 1983) which considered the relationship between exposure and the incidence of symptoms characteristic of asbestosis, but not mortality from cancer.

When, therefore, two of us (RD and JP) were asked by the Health and Safety Commission to review the effects of exposure to asbestos on health, we thought it worthwhile to extend the observations to include a larger and more representative cohort observed over a longer period and to compile detailed individual job histories in the hope that any excess mortality could be related to dust measurements specific for individuals. We report now the results of our mortality study in detail. The many difficulties in using these results to predict quantitatively the medical effects of exposure to chrysotile asbestos are discussed elsewhere (Doll and Peto, 1985).

MATERIAL AND METHODS OF INVESTIGATION

The factory

The factory was established in 1855 to weave cotton and it was not until 1879 that experiments were started with asbestos. Over the next 30 yr techniques were developed to produce a wide range of textile and non-textile asbestos products through adapting the crafts of cotton carding, spinning, and weaving, and adding new processes. The volume of trade increased steadily at first and then rapidly between 1900 and 1910 when processes were developed for making compressed asbestos fibre (asbestos and rubber gasket material) rubberized conveyor belting, woven asbestos fabrics for brake-lining manufacture and a range of plaited strings and ropes for sealing applications.

In 1920, the company amalgamated with three others. During the subsequent depression it retooled and rebuilt, dispensing finally with cotton processing. The range and quantity of products continued to expand in the 1930s and came to include yarns, tapes, webbings, cloths, ropes, impregnated felts, packings and other jointing materials for a great variety of applications. Non-asbestos industrial and transmission belting was also developed at Rochdale, but these lines of production eventually grew so large that they had to be transferred to a new factory at Hindley Green in 1949. The Hindley Green factory also made railway locomotive asbestos insulation mattresses between 1958 and 1962, produced asbestos-cement spray between 1959 and 1969, and shared in the production of a range of asbestos textile and resinated asbestos felts from 1958 to 1972, when all asbestos production here ceased.

The developments in improved dust control that occurred during the period 1950–1965 at the Rochdale factory have been described by Bamblin (1959) and Hills

(1965). The main features of a large-scale programme of work covering both plant and process changes included:

(a) The replacement of hand-mixing by enclosed drum mixing. An oil-emulsion was also developed to be added to the fibre at the fiberizing stage to reduce dust levels at downstream operations.

(b) The development of enclosed mechanical bag-filling units to pack asbestos fibre into 'impermeable' bags and replace 'hand-pomming' methods of packing and the use of hessian sacks.

(c) A complete re-design of the carding ventilation systems, to improve hood-capture efficiency at the various dust-emission points and the conveyance of dust through the ducting to sleeve filters, and the development of a ventilated system to capture the dust emitted during 'dusty' card-stripping operations.

(d) Improved ventilation at the weaving operations.

(e) The provision of damping systems to damp yarn prior to certain doubling, winding and weaving operations.

Additional measures have included the enclosure, by 1972, of all the carding units in 'respirator' zones to isolate them from the main (spinning) working area. The direction of air flow was reversed so as to prevent dust from the more dusty processes being drawn towards cleaner locations and the incoming replacement air from heated plenum systems balanced to provide slight negative pressure in working areas.

In 1968 a range of asbestos textiles made by the 'Fortex' wet-dispersion process was introduced. In this method, the raw asbestos is opened by a chemical process, formed into a colloidal water-based dispersion and then extruded into a coagulating agent to form a wet strand. The strand is then twisted and spun into a yarn. The process therefore eliminates the need for the traditional mechanical opening and carding operations and the textile products resulting from the Fortex process are inherently less dusty than their conventionally made counterparts.

Asbestos textile production has continued at Rochdale to the present day, but the last 10 yr have seen a gradual change-over to new substitute materials, such as glass fibre and Kevlar, a synthetic aromatic polyamide fibre. This change has accelerated in the past 3 yr, until now approximately one-third of the factory's output is non-asbestos. Accompanying these changes the total number of employees, shop-floor plus staff, has dropped from a peak of around 3000 in the mid-1960s to approximately 1250 in the autumn of 1984.

Details of the types of asbestos used throughout this period are given in Appendix A.

The populations studied

The population studied consists of three groups. The first is composed of 145 men who had worked in scheduled areas in the factory for at least 20 yr, including some time before 1933. These men had been identified and followed previously to the end of 1978 (PETO, 1980) and we have now extended observation of the survivors to the end of June 1983. The results are unfortunately of little value for the purpose of estimating the quantitative relationship between exposure and risk, as no measurements of early dust levels were made. All that can be said is that the conditions some time before 1933, when the first Asbestos Industry Regulations (1931) came into force, were certainly very

much dustier than they were subsequently. The second group, which has also been followed up to the end of June 1983, consists of 283 women first employed in 1933 or later who had been employed for 10 yr or more in scheduled areas and had been followed previously to the end of 1974 (PETO *et al.*, 1977).*

Our previous studies had also included men first employed in 1933 or later who had worked in scheduled areas for ten or more years. For these, we have now substituted two sub-groups: (A) men who were first employed in 1933 or later, who had ever worked in scheduled areas or on maintenance (which is not a scheduled occupation but involves periodic exposure) and had completed 5 yr total service by the end of 1974, and (B) a 1 in 10 sample of all male employees first employed between 1 January 1933 and 31 December 1974, irrespective of where or how long they had worked. These two sub-groups partially overlap, but duplication of the results is avoided by including in the follow-up of the latter the experience of men who eventually completed 5 yr service only for the period until that service was achieved. This combined group, totalling 3650 men (reduced to 3211 after exclusions. *cf.* pp. 314–317), now constitutes the principal cohort in our present study.

Sources of information for identification and follow-up

Information needed for identifying and following up the men and women in the first and second groups was obtained from our previous records. Information required for identifying men in the third group (principal cohort) was obtained by compiling a register of all men and women who had ever worked in the factory since the end of 1932 from the personnel records held by the company in 1975. This included the names of approximately 21 000 men and 10 000 women and constituted the frame from which both our samples were drawn.

The names of all the men and women on the original register provided by the company were matched against the national mesothelioma register maintained by the Health and Safety Executive; the few persons who were found in this way to have developed a mesothelioma were noted, irrespective of their length and period of service. Lists of all deaths and of all men who had developed symptoms of asbestosis that were known to the company were also searched and all those men who had completed 5 yr service between 1 January 1933 and 31 December 1974, including some scheduled or maintenance work, but who had been missed when the original register was compiled, were added to the cohort, as well as any other eligible individuals who were found during further searches of factory records. The inclusion of these additional cases does not greatly alter the overall results. Analyses without them may, we suspect, slightly underestimate the overall risk, while their inclusion probably overestimates it. Records dating back 40–50 yr are inevitably lost occasionally for a variety of reasons, but the records of men who have developed asbestos-related diseases may be more likely to be taken to other departments for medical or legal reasons and subsequently mislaid.

To check the completeness and accuracy of the original register a 1 in 20 sample of all male personnel records, a total of approximately 1100 individuals, was drawn and compared with the register. The original register was compiled by several clerical

* Re-examination of employment records led to the addition of two men and the exclusion of one woman. In previous reports there were 143 men in the first cohort and 284 women in the second cohort.

workers in the personnel department and records were held in various forms and in several different files. A block of 79 consecutive names was missing and these were added to the register. A further 24 Asian and 54 other names were, however, also missing and the register therefore seems likely to have been seriously incomplete for Asians, but approximately 95% complete for other workers. The reasons for this incompleteness include errors in the original compilation, mis-filing, temporary removal, and inaccuracy in recording names and dates of birth.

Follow-up information about all men and women in the groups studied, which was not already known to us from factory records and our previous studies, was obtained from the National Health Service Register held by the Office of Population Censuses and Surveys at Southport.

Identification and classification of mesotheliomas

Most suspected mesotheliomas among past employees were, of course, known to the Company, but any among men who worked in the factory briefly, particularly in the 1930s and 1940s, might not have been. It was not surprising, therefore, that a few additional cases were discovered from the national mesothelioma register, as described previously, as well as from death certificates. The list of mesotheliomas among previous employees, both male and female, that we were thus able to compile is, we believe, complete within the limits set by the accuracy of medical diagnosis for cases occurring since 1967, when the national mesothelioma register was established. In view of the long induction period for this disease, it is unlikely that any asbestos-related cases diagnosed in workers first exposed in 1950 or later have been missed, although one or two in earlier workers may have been. All available diagnostic information on these cases, including specialist reports on the microscopic appearance of the tumours, was reviewed by one of us (RD) and the diagnosis of mesothelioma classified as 'established' on the basis of post-mortem evidence with or without histological confirmation (43 cases), 'presumptive' on the basis of death certificate information alone (3 cases), 'uncertain', because of conflicting evidence (1 case), or 'incorrect' (12 cases).* Details of all suspect cases are included in Appendix B. Most of the 'incorrect' cases appeared on company lists of possible mesotheliomas, but mesothelioma was mentioned on the death certificate for only four (Appendix B, cases 101, 106, 109 and 112). The remaining eight, all of whom died of lung cancer or asbestosis, appear to have been transferred in error from other company lists of suspect deaths, as we could find no evidence that they had mesothelioma.

Job histories

For each individual in the principal cohort, a detailed work history was compiled which included the section of the factory in which he had worked, the type of work (carding, weaving, etc.), the detailed occupation (the actual machine used, if specified, and categories of occupation such as autolathe setter, carding assistant, etc.), the employment code number (which sometimes identified the job more specifically) and

* Dr Christopher Wagner of the MRC's Pneumoconiosis Unit and a member of UICC's mesothelioma panel specially reviewed a number of doubtful cases and Dr Anthony Newman Taylor, lecturer in occupational medicine at the Brompton Hospital, advised on the diagnosis of choice in the rare case for which the evidence was inconsistent.

whether the job was scheduled. Previous work with asbestos before joining the Company was also recorded, as well as work at the other local asbestos factory owned by the Company at Hindley Green. If a man transferred to work at Hindley Green after inclusion in the cohort, a note was made of the date of transfer, but no further job details were recorded.

Presentation of results

In presenting our results we have adopted the following conventions. First, we have attributed deaths to mesothelioma whenever this seemed to have been the most likely cause after review of the available information, irrespective of the diagnosis on the death certificate. Individuals who died of some other independent cause, but with a mesothelioma present, have been classified as dying of the other cause (Appendix B, cases 4, 17 and 33), but, as it happens, no such deaths occurred in the groups under special study. When death was certified as due to mesothelioma, but review of the data indicated that the diagnosis was incorrect, the revised diagnosis has been used instead (Appendix B, cases 101, 106 and 112). In all other instances, death has been attributed to the cause to which it was attributed in the national statistics.

Secondly, we have excluded all observations on men and women over 85 yr of age. This has been our standard practice in epidemiological studies for the following reasons: (i) the cause of death in very old people is often not investigated in great detail and the diagnosis in this age group is more likely to be incorrect than at younger ages; (ii) the Registrar General does not give mortality rates for 5-yr age groups over 85 yr of age and calculation of an expected number of deaths in this age group is liable to be biased if the age distribution of the group is different from that nationally; and (iii) if any men have died or emigrated without this fact being discovered, the numbers will accumulate and could become a significant proportion of the total thought to be alive with the rapid diminution in the number of true survivors in this age group.

Calculation of expected deaths

The mortality expected in the various groups studied has been calculated in the usual way by multiplying the person-years at risk in each 5 yr age group in each quinquennium by the corresponding sex, age and calendar specific mortality rates for England and Wales as a whole. Mortality in Rochdale was not, however, identical with that in the whole country and the extent of the difference differed for different diseases and at different times. The effect of this on the validity of the comparison between the observed and 'expected' rates is discussed in the next section in relation to Table 5.

RESULTS

Mesothelioma

A total of 34 men and 13 women who had worked at the Rochdale factory at some time since it opened in 1902 were accepted as having died of mesothelioma (see Table B1, Appendix B). All the mesotheliomas were pleural except one, which was peritoneal in origin and occurred in a man who worked in a high exposure area from 1926 until 1939 and had been exposed to asbestos for one year previously in another factory (case 12) and may have been exposed to crocidolite. Eighteen were in men first exposed before 1933, of whom 7 had worked for 20 yr or more in scheduled areas and

are therefore included in our first (pre-1933) cohort. Fourteen of the 16 men who started work in 1933 or later are included in our principal cohort. The remaining two cases (cases 11 and 19) occurred in men who had worked at Rochdale for a year or less after substantial exposure to asbestos elsewhere (one at Hindley Green) and would therefore have been excluded from our main analysis had they chanced to be included in our 1 in 10 sample of short-term workers (see below).

Five of the 13 women who developed mesothelioma were first employed in 1933 or later. These five women had worked for 8, 4, 3, 0 and 0 yr in scheduled areas. One of the two women who had not worked in the scheduled areas had worked for 10 yr on maintenance; the husband of the other had been employed for 2 yr as a weaver in the factory 20 yr before her death. None of these cases qualified for inclusion in our female cohort, however, as none had started work in 1933 or later and worked in scheduled areas for 10 yr or more.

Men employed before 1933

Table 1 shows the updated results for the group of 145 men who have been studied previously: that is, men who worked in scheduled areas in the factory for at least 20 yr, including some time before 1933 (DOLL, 1955; PETO, 1980). The numbers of deaths observed and expected are shown separately for men with more or less than 10 yr service before 1933. As in previous publications, a gross increase is seen in mortality from lung cancer, mesothelioma, and asbestosis, which is greater (for lung cancer and asbestosis) for men who had worked more than 10 yr before 1933 than for those who had worked for less. The first ASBESTOS INDUSTRY REGULATIONS (1931) had come into force by the end of 1932 and the very high relative risk of lung cancer in men who had worked for ten or more years before that date (13 observed against 1.58 expected) was originally reported thirty years ago (DOLL, 1955). This cohort of 69 men has now been followed to extinction, save for two men, one born in 1873 and one in 1874, who ceased to be followed up at 85 yr of age (Table 1).

The mortality from circulatory disease was also raised in these pre-1933 men, particularly in the earlier sub-group, owing presumably to the effect of pulmonary fibrosis on the heart, and there was a small and statistically non-significant increase in mortality from gastro-intestinal cancer (7 deaths against 4.80 expected).

Women

Table 2 shows the updated results for the group of 283 women who had been studied previously: that is, women who were first employed between 1 January 1933 and 31 December 1962 and who worked in scheduled areas for at least 10 yr. The numbers of deaths observed and expected are shown separately for women who were first employed before or after the end of 1950. The numbers of deaths are still too few for any material conclusions to be drawn from them. It may be noted, however, that there was an excess of deaths from lung cancer in women first employed before 1950 (4 against 1.44 expected) and that one death from asbestosis also occurred in this group. No deaths from lung cancer or asbestosis have as yet occurred in the small group who were first employed after 1950.

The occurrence of one death from asbestosis in a woman first employed between 1933 and 1950 and the increased mortality from lung cancer in men first employed at the same period (see below) suggests that the small increased mortality from lung

TABLE 1. NUMBERS OF DEATHS OBSERVED AND EXPECTED IN 145 MEN EMPLOYED FOR 20 YR OR MORE IN SCHEDULED AREAS WITH SOME SERVICE BEFORE 1933, BY TIME EMPLOYED BEFORE 1933 AND CAUSE

Cause of death	Deaths in 69 men employed 10 yr or more before 1933			Deaths in 76 men employed less than 10 yr before 1933			Total deaths in 145 men		
	Observed	Expected	SMR*	Observed	Expected	SMR	Observed	Expected	SMR
Lung cancer	13	1.58	8.23	7	3.97	1.76	20	5.55	3.61
Mesothelioma	2			5			7		
Cancer of oesophagus	0	0.20	0.00	0	0.25	0.00	0	0.45	0.00
Cancer of stomach	2	1.02	1.96	2	1.26	1.59	4	2.28	1.76
Cancer of colon/rectum	2	0.98	2.04	1	1.08	0.92	3	2.07	1.45
Cancer of larynx	0	0.11	0.00	0	0.11	0.00	0	0.22	0.00
Cancer of kidney	0	0.08	0.00	0	0.15	0.00	0	0.22	0.00
Other cancers	4	1.86	2.16	2	2.83	0.71	6	4.69	1.28
Asbestosis	9			6			15		
Other respiratory	5	3.97	1.26	4	5.89	0.68	9	9.86	0.91
Circulatory	26	13.43	1.94	23	18.86	1.22	49	32.30	1.52
Other diseases	4	4.47	0.89	6	3.57	1.68	10	8.04	1.24
Violence	0	1.08	0.00	0	1.15	0.00	0	2.23	0.00
All causes	67	28.79	2.33	56	39.11	1.43	123	67.90	1.81

* Standardized mortality ratio: i.e. observed deaths as proportion of expected.

TABLE 2. NUMBERS OF DEATHS OBSERVED AND EXPECTED IN 283 WOMEN EMPLOYED FOR 10 YR OR MORE IN SCHEDULED AREAS SINCE 1932, BY DATE OF FIRST EMPLOYMENT AND CAUSE

Cause of death	Deaths in 191 women first employed 1933–50			Deaths in 92 women first employed 1950–74			Total deaths in 283 women		
	Observed	Expected	SMR*	Observed	Expected	SMR	Observed	Expected	SMR
Lung cancer	4	1.44	2.77	0	0.46	0.00	4	1.90	2.11
Mesothelioma	0			0			0		
Cancer of oesophagus	0	0.25	0.00	0	0.07	0.00	0	0.32	0.00
Cancer of stomach	2	0.86	2.34	0	0.22	0.00	2	1.08	1.85
Cancer of colon/rectum	3	1.58	1.90	1	0.44	2.26	4	2.02	1.98
Cancer of larynx	0	0.04	0.00	0	0.01	0.00	0	0.06	0.00
Cancer of kidney	0	0.14	0.00	0	0.04	0.00	0	0.18	0.00
Other cancers	6	8.30	0.72	3	2.47	1.22	9	10.77	0.84
Asbestosis	1			0			1		
Other respiratory	5	3.48	1.44	2	0.89	2.25	7	4.37	1.60
Circulatory	10	12.29	0.81	0	3.29	0.00	10	15.58	0.64
Other disease	8	8.98	0.89	2	2.19	0.91	10	11.16	0.90
Violence	2	1.52	1.32	0	0.43	0.00	2	1.95	1.03
All causes	41	38.87	1.06	8	10.51	0.76	49	49.38	0.99

* See footnote to Table 1

cancer in women may well be occupational in origin. It is doubtful, however, whether national rates are appropriate for calculating the number of deaths expected for lung cancer in working women, a higher proportion of whom are likely to have been regular cigarette smokers than in the population at large.

No mesotheliomas are shown in Table 2, despite the fact that a case was recorded in an eligible woman in a previous publication (Peto *et al.*, 1977). Deaths attributed to mesothelioma had not been reviewed individually in previous reports and the additional evidence that we now have shows that the certified diagnosis was probably incorrect and that the diagnosis should have been carcinoma of the lung (Appendix B, case 112).

Principal cohort

The number of men in our principal cohort and the numbers excluded for the purpose of detailed study are shown in Table 3, together with their status at the time follow-up ceased (30 June 1983). Men with Asian surnames (who were identified independently without knowledge of the results of follow-up) were excluded because a high proportion had emigrated or had not been traced and because of the evidence that many were likely to have been missed when the original register was compiled (see p. 309). Men who had previously been exposed to asbestos in the course of their work were excluded because of the lack of information about the extent to which they had been exposed. The 27 men in this group included six who had transferred from Hindley Green, another local factory owned by the same company. The 86 men who were transferred to Hindley Green after qualifying for inclusion in the study group contributed to the man-years at risk and hence to the expected numbers of deaths up to the date of transfer, but were excluded subsequently. Finally, the 17 men whose names were discovered in additional searches after the compilation of the register from the 1975 personnel records were also excluded.

The effect of these exclusions can be assessed from Table 4, which shows the numbers of deaths in these groups that were observed from asbestosis, mesothelioma, lung cancer and all causes and the numbers expected from the two latter categories. The few deaths that were observed in Asians were appreciably less than the number expected at national rates and it seems likely that exclusion of these men has avoided the introduction of bias due to the incompleteness of information about them. One man who transferred to Hindley Green developed mesothelioma. Substantial amounts of crocidolite had been used in this factory and quantitative measurements of the amount of exposure, comparable to those made in Rochdale, have, therefore, not been included. The inclusion of their experience would clearly have exaggerated the effect of employment in Rochdale. The men with previous asbestos exposure must also be excluded for the same reason. None of them developed mesothelioma, but three died of asbestosis and the group experienced a small excess of lung cancer.

Whether the men whose names were discovered by special searches after the register was compiled should be excluded is more debatable. One had died of asbestosis and this group, as was to be expected, also suffered an excess of lung cancer. The records of men who die of asbestos-related disease may be removed from the personnel department for medical or legal reasons and subsequently inadvertently mislaid, while any history of previous asbestos work may be more likely to be recorded in them. Excluding them may result in an underestimate of risk, but including them probably leads to an

TABLE 3. STATUS AT 30 JUNE 1983 OF MEN IDENTIFIED AS MEMBERS OF PRINCIPAL COHORT

Status	10% sample (B)			5 or more years' employment with some in scheduled areas or on maintenance (A)				Men after transfer to Hindley Green	Total cohort	
	Previous asbestos exposure*	Asians	Remainder	Previous asbestos exposure*	Asians	Remainder	Added		Before exclusions	After exclusions
Transferred to group A after qualification†	3	22	180							
Transferred to Hindley Green	0	0	25	0	0	61	0			
Emigrated	0	10	58	0	11	47	0	2	128	107
Died at age 85 or over and therefore censored	0	0	5	0	0	10	0	1	16	16
Lost to sight	0	44	104	1	20	31	0	5	205	140
Died before age 85	2	2	443	11	11	670	12	38	1189	1151
Alive	7	165	1004	6	132	753	5	40	2112	1797
Total	12	243	1819	18	174	1572	17	86	3650	3211

* Including previous employment at Hindley Green.

† These men appear in both groups A and B, so that the total numbers of men studied are obtained by adding the totals in A and B and subtracting the number transferred from B to A (e.g. total men with previous exposure = 12 + 18 − 3 = 27).

TABLE 4. NUMBER OF DEATHS FROM ALL CAUSES AND FROM DISEASES DUE (OR SOMETIMES DUE) TO ASBESTOS IN EXCLUDED GROUPS

Cause of death	Previous asbestos exposure		Asians		Transferred to Hindley Green		Missed from register	
	Observed	Expected	Observed	Expected	Observed	Expected	Observed	Expected
Lung cancer	2	0.65	2	1.93	3	2.99	3	0.43
Mesothelioma	0	—	0	—	1		0	
Asbestosis	3		0	—	1		1	
All causes	13	7.28	13	20.42	38	25.48	12	4.38

overestimate. They are excluded from all subsequent tables except Table 16, where the effect of their omission is shown in relation to the estimate of risk per unit dose.

All but 135 (4.2%) of the 3211 men in our principal cohort (including the 86 who were under observation in it until their transfer to Hindley Green) were traced (Table 3). These 135 were regarded as lost to follow-up and hence did not contribute to the man-years at risk or expected numbers of deaths from the date of leaving the factory. The majority had been employed for less than 5 yr and, among those with 5 yr service or more, only 31 (2.0%) were untraced.

MORTALITY BY CAUSE

The mortality of these 3211 men from different causes of death is shown in Table 5 in comparison with the mortality expected at national rates and that observed in Rochdale County Borough in 1969–73, the period round the last census for which detailed local mortality rates have been published. Separate figures are given in Table 6 for men with less than or more than 10 yr duration of employment in the factory and for periods of observation less than or more than 20 yr after first employment.

The total mortality is higher than that expected from national mortality rates (SMR 1.14)* but slightly less than the rate in Rochdale towards the middle of the period of observation (SMR 1.16). The mortality from circulatory disease was also slightly less than that in Rochdale and so was the mortality from non-malignant respiratory disease, despite the fact that it included seven deaths from asbestosis. Only for mesothelioma and lung cancer was the mortality significantly raised in comparison with both national and local Rochdale rates (SMR for lung cancer 1.31, $P<0.01$†). The

TABLE 5. NUMBERS OF DEATHS OBSERVED AND EXPECTED IN 3211 MEN IN THE PRINCIPAL COHORT BY CAUSE, COMPARED WITH THE MORTALITY IN ROCHDALE IN 1969–73

Cause of death	Number of deaths Observed	Expected	SMR	SMR in Rochdale in 1969–73
Mesothelioma	11	—		—
Cancer of lung	132	100.45	1.31*	0.99
Cancer of oesophagus	11	6.59		
Cancer of stomach	29	28.99		
Cancer of colon/rectum	20	26.72	0.81	0.98
Cancer of larynx	4	2.58		
Cancer of kidney	1	4.07		
Other cancers	52	75.65		
Asbestosis	7	--		1.45
Other respiratory disease	169	129.72	1.36	
Circulatory disease	536	467.29	1.15	1.18
Other diseases	85	89.72	—	—
Violence	56	41.17	—	—
All causes	1113	972.94	1.14	1.16

* We have expressed SMRs throughout this report as proportions of the expected mortality rather than as percentages of it: that is 1.14 rather than 114. They are, therefore, essentially the same as relative risks.

† All significance levels are 2-sided.

TABLE 6. NUMBERS OF DEATHS OBSERVED AND EXPECTED IN MEN IN THE PRINCIPAL COHORT BY CAUSE, DURATION OF EMPLOYMENT IN SCHEDULED AREAS, AND TIME SINCE FIRST EMPLOYMENT

	Less than 10 yr in scheduled areas				10 yr or more in scheduled areas			
	Time since first employment				Time since first employment			
	Less than 20 yr		20 yr or more		Less than 20 yr		20 yr or more	
Cause of death	Observed	Expected	Observed	Expected	Observed	Expected	Observed	Expected
Mesothelioma	1	—	1	—	0	—	9	—
Cancer of lung	30	29.23	53	45.44	9	6.66	40	19.13
Cancer of oesophagus	2	1.80	6	3.13	0	0.38	3	1.27
Cancer of stomach	9	10.11	9	11.70	2	2.18	9	5.00
Cancer of colon/rectum	5	8.35	8	11.81	2	1.70	5	4.86
Cancer of larynx	0	0.87	4	1.08	0	0.19	0	0.45
Cancer of kidney	0	1.30	1	1.78	0	0.27	0	0.72
Other cancers	15	23.56	23	33.83	1	4.53	13	13.73
Asbestosis	1	—	0	—	2	—	4	—
Other respiratory disease	47	36.98	87	59.83	6	7.90	29	25.01
Circulatory disease	157	132.14	250	217.51	27	28.16	102	89.48
Other diseases	39	42.22	28	29.10	6	6.46	12	11.95
Violence	23	22.56	25	11.40	2	2.93	6	4.27
All causes	329	309.12	495	426.60	57	61.34	232	175.87

mortality from lung cancer in Rochdale was so close to the national rate—even closer than it had been previously when the workers in the factory were first studied and the SMR (in 1946–49) was 0.96—that the difference between the Rochdale and national rates is ignored in the rest of this report and the numbers of deaths from lung cancer in various sub-groups are compared directly with the numbers expected from the national rates without any correction for locality.

The results recorded in Table 6 show that the excess mortality from mesothelioma and from lung cancer was concentrated in men employed in scheduled areas for ten or more years and in the period 20 yr or more after first employment (mesothelioma 3.9% of all deaths against 0.2% in all other men and at all other times; SMR for lung cancer 2.09 against 1.13 for all other men and at all other times), while the mortality from asbestosis was more evenly spread over different periods after first employment (1.7% of all deaths 20 yr or more after first employment in men employed for ten or more years in scheduled areas against 3.5% less than 20 yr after first employment in the same group of men and 0.1% in all other men). In contrast, the mortality from other respiratory disease and from circulatory disease was, if anything, slightly diminished in long-term employees (SMRs, respectively, 1.06 and 1.10 in men employed for ten or more years in scheduled areas against 1.38 and 1.16 for men employed for less) which conforms with the 'healthy worker' effect that is characteristic of stable employment (Fox and Collier, 1976). All other cancers showed a mortality rate that was somewhat less than that expected for Rochdale (SMR 0.81 against 0.98), but the rate was slightly raised for men employed in scheduled areas for ten or more years who were observed 20 yr or more after first employment (SMR 1.15 against 0.73 for all other men and all other periods of observation). This increase, in long-term employees, is limited to gastro-intestinal cancer, but is not quite statistically significant (SMR 1.53, $P=0.1$).

Lung cancer

The results obtained in Tables 5 and 6 show that the men employed in the Rochdale factory continued to suffer an excess mortality from lung cancer, even when they were first employed after 1933. Moreover, the concentration of this excess in long-term workers more than 20 yr after first exposure strongly suggests that it was occupational in origin. Other factors may, however, have contributed to it as well. Local factors have been excluded by the comparison with the SMR for Rochdale, but socio-economic factors in general—and cigarette smoking in particular—could have played some part. The information available about the smoking habits of the employees is too incomplete for us to use, but the results of the survey carried out by the Health and Safety Executive at the start of its national asbestos mortality study show that cigarette smoking was somewhat more prevalent among asbestos textile workers than among men in the country as a whole and that this could account for an excess mortality from lung cancer of about 5% (Doll and Peto, 1985; Carter, personal communication). It might, therefore, be responsible for much of the excess observed in men employed for less than 10 yr (SMR 1.11) and in men of all durations of employment less than 20 yr after first exposure (SMR 1.09). It can, however, have contributed only a very small proportion of the excess in men employed for more than 10 yr observed more than 20 yr after first exposure (SMR 2.09). We shall, therefore, ignore the effects of smoking habits in estimating the dose–response relationship except in so far as they may help to explain anomalous findings in short-term employees.

Table 7 shows the mortality from lung cancer in the principal cohort by duration of employment in scheduled areas and time since first exposure, separately for men first exposed between 1933 and 1950 and between 1951 and 1974. The results 20-34 yr after first exposure for men first exposed between 1933 and 1950 are consistent with an increase in relative risk with increasing duration of exposure. The trend is not statistically significant, but the SMR (2.01) is significantly elevated for men with ten or more years' exposure (24/11.95; $P<0.01$). No such trend is apparent beyond 35 yr after first exposure, when the SMR actually falls from 1.23 (12/9.76) for less than 10 yr exposure to 0.93 (4/4.30) for ten or more years' exposure. The results for men first exposed in 1951 or later suggest a rather different picture, the relative risk 20-32 yr* after first exposure rising from 0.82 (7/8.49) for less than 10 yr to 4.17 (12/2.88: $P<0.001$) for ten or more years' exposure.

The apparent difference between the trends observed in different periods is the result of two factors: (i) a significantly elevated overall incidence in men first exposed before 1951 with less than a year's exposure (44 observed, 30.51 expected; $P<0.05$) and (ii) a greater relative risk 20-32 yr after first exposure in men with ten or more years' scheduled exposure, who were recruited after 1950 (12 against 2.88 expected) than in men with similar duration of exposure who were recruited earlier (24 observed against 11.95 expected). The increased risk in men employed before 1951 with less than a year's exposure seems likely to be due largely to chance or to selection bias, as the relative risk was lower in men employed for 1-9 yr and it was not observed at a later period. The reason for the increased relative risk of men with more than 10 yr exposure who started work after 1950 is discussed later (pp. 39 and 40).

Mesothelioma

Fourteen men in the principal (post-1933) cohort died of mesothelioma. Three, however, are not shown in Table 5: two cases (24 and 25) because the affected men died after the last follow-up date (30.6.83) and one (case 27) because the man was transferred to the Hindley Green factory before the disease was diagnosed (16 yr before death) and is shown only in Table 4.

One of the remaining 11 cases in men who were included in the post-1933 cohort seems virtually certain not to have been caused by asbestos exposure at Rochdale. The affected man (case 14) had worked at Rochdale for only 4 months and died 4 yr later in 1951 at the age of 35. He had no known previous exposure and seems likely to be an 'incidental' case caused either by childhood exposure or by other or natural causes. In North America, one third to a half of all mesotheliomas, or about one death in 3000 to 5000 men, cannot be related to asbestos exposure (McDONALD and McDONALD, 1980; PETO *et al.*, 1981) and, as the incidence of non-asbestos induced mesotheliomas is presumably similar in Britain, the occurrence of one such case in any cohort in which over 1000 deaths have occurred would not be remarkable. Our attribution of this case to non-occupational causes is supported by the unusually young age at which death occurred, which is characteristic of non-occupational cases possibly attributable to causes other than asbestos (PETO *et al.*, 1981). Alternatively, the diagnosis may be incorrect. We have only the certified cause of death as 'endothelioma of pleura' and

* Follow-up closed in mid-1983 so that no deaths have been observed in this group of men more than 32 yr after first exposure.

TABLE 7. OBSERVED (O) AND EXPECTED (E) NUMBERS OF LUNG CANCER DEATHS AMONG MEN FIRST EMPLOYED IN 1933 OR LATER IN A CHRYSOTILE TEXTILE FACTORY IN ROCHDALE

	Years since first exposure	Duration of service in scheduled areas: Less than 1 yr O	E	1–4 yr O	E	5–9 yr O	E	10–19 yr O	E	20 yr or more O	E	Total O	E
First exposed 1933–50	0–19	13	8.75	1	1.86	6	4.70	5	4.31			25	19.62
	20–34	22	16.55	4	3.55	8	7.09	15	5.90	9	6.05	58	39.14
	35+	9	5.22	0	1.60	3	2.94	0	1.39	4	2.91	16	14.06
	Total	44	30.51	5	7.01	17	14.73	20	11.60	13	8.95	99	72.81
First exposed 1951–74	0–19	7	8.74	1	1.88	2	3.31	4	2.35			14	16.28
	20–32	6	5.72	1	0.96	0	1.81	8	1.83	4	1.05	19	11.37
	Total	13	14.46	2	2.84	2	5.12	12	4.18	4	1.05	33	27.65
All men	0–19	20	17.49	2	3.74	8	8.01	9	6.66			39	35.90
	20–34	28	22.27	5	4.51	8	8.90	23	7.73	13	7.10	77	50.51
	35+	9	5.22	0	1.60	3	2.94	0	1.39	4	2.91	16	14.06
	Total	57	44.97	7	9.85	19	19.85	32	15.78	17	10.00	132	100.46

have been unable to obtain any more specific information. This case was therefore ignored in subsequent analysis of mesothelioma incidence.

The incidence pattern based on the 10 remaining cases is shown in Table 8 by duration of exposure in scheduled areas and time since first exposure. Apart from the marked effect of time since first exposure, the most striking aspect of these figures is the low incidence among men who worked in scheduled areas for less than 10 yr. If men with less than 10 yr exposure had suffered the incidence rates in each interval observed in men with ten or more years' exposure, they would have been expected to contribute 25 mesothelioma deaths, whereas only one was in fact observed. This man had worked for only 18 months in a dusty area in 1936–38, but was subsequently employed in non-scheduled areas at Rochdale for over 30 yr (case 6). As with lung cancer, there is no evidence of any reduction in risk after 1951, although there are too few cases for useful analysis. If the overall rates are applied to the man-years in each interval since first exposure for men first employed since 1950, the expected number of cases is approximately one. One death from mesothelioma was, in fact, observed in the post-1950 group, but two more have been observed in the 15 months since the follow-up ceased (in men first employed, respectively, in 1952 with 24 yr employment in scheduled areas and in 1959 with 20 yr employment on maintenance).

PERSISTENCE OF HAZARD AFTER 1950

Mortality from the three asbestos-related diseases in men first employed between 1951 and 1974 provides no reason to believe that the hazard was materially reduced in that period compared with the hazard in the preceding 18 yr (1933–50). No deaths were recorded from asbestosis, but only two were expected at the rates previously observed (see DOLL and PETO, 1985). The previous paragraph shows that there was no reduction in the number of cases of mesothelioma, while the relative risk from lung cancer in men with ten or more years' exposure in scheduled areas observed 20–34 yr after first exposure was actually increased (12 observed deaths with 2.88 expected against 24 observed deaths with 11.95 expected; $0.05 < P < 0.10$). Possible reasons for this difference are discussed later (pp. 39 and 40).

Long-term pattern of relative risk for lung cancer

The numbers of deaths from lung cancer in men employed in scheduled areas for 20 yr or more are shown in Table 9 according to date of first employment and time since first exposure. The decrease in relative risk observed between men with more than 10 yr exposure before 1933 (8.23) and men with some but less than 10 yr exposure (1.76) did not extend to men with no such exposure (1.70). A progressive reduction was, however, seen in men observed 20–34 yr after first exposure (SMRs 15.56, 3.92 and 1.83).

The results shown in Table 9 suggest that the widely accepted assumption that the relative risk for lung cancer increases or remains at its maximum level with the passage of time after first exposure may be incorrect, as in each cohort the relative risk more than 35 yr after first exposure is lower than between 20 and 35 yr. A similar reduction was also observed in men who had been employed in scheduled areas for 10–19 yr since 1933, and Table 10 shows that the reduction in relative risk beyond 35 yr after first exposure compared with that 20–34 yr after first exposure is observed at all ages (see DOLL and PETO, 1985, for discussion).

TABLE 8. MESOTHELIOMA MORTALITY IN THE PRINCIPAL COHORT, BY DURATION OF SCHEDULED SERVICE. OBSERVED DEATHS (O) AND MAN YEARS (MY)

Years since first employment	Duration of scheduled service: Less than 1 yr		1-4 yr		5-9 yr		10-19 yr		20-29 yr		30 yr or more		Total	
	O	MY	O	MY	O	MY	O	MY	O	MY	O	MY	O	MY
0-19	0*	28015.0	0	4785.6	0	8520.5	0	4814.1					0*	46135.1
20-24	0	4668.2	0	877.4	0	1416.8	0	1423.3	1	848.3			1	9234.1
25-29	0	3469.8	0	631.7	0	1103.5	0	869.7	1	935.3			1	7010.0
30-34	0	2041.2	0	421.3	0	707.2	3	469.7	2	599.7	0	86.2	5	4325.3
35-39	0	840.4	0	237.7	0	383.3	0	204.0	1	257.1	0	106.9	1	2029.4
40 or more	0	402.3	1	148.4	0	249.1	1	102.3	0	121.7	0	103.4	2	1127.3
All periods	0*	39436.9	1	7102.0	0	12380.4	4	7883.0	5	2762.2	0	296.6	10*	69861.1

* A man exposed for 4 months who died from 'endothelioma of the pleura' 4 yr later has been excluded (see text)

TABLE 9. OBSERVED AND EXPECTED NUMBERS OF LUNG CANCER DEATHS IN MEN EMPLOYED IN SCHEDULED AREAS FOR 20 YR OR MORE BY DURATION OF SERVICE BEFORE 1933 AND TIME SINCE FIRST EXPOSURE

Years since first exposure	Duration of service before 1933: 10 yr or more		Some, but less than 10 yr		None (first employed 1933 or later)	
	Observed	Expected	Observed	Expected	Observed	Expected
20-34	7	0.45	4	1.02	13	7.10
35 or more	6	1.13	3	2.95	4	2.91
All periods	13	1.58	7	3.97	17	10.00

TABLE 10. LUNG CANCER MORTALITY IN MEN WITH PROLONGED EXPOSURE IN SCHEDULED AREAS, DIVIDED BY YEARS SINCE FIRST EXPOSURE AND AGE

Exposure in scheduled areas (yr)	Age (yr)	Period since first exposure: 20-34 yr		35 yr or more	
		Observed	Expected	Observed	Expected
20 or more, some before 1933	Under 60	9	0.99	1	0.57
	60-69	1	0.43	3	1.99
	70 and over	1	0.05	5	1.52
10 or more, none before 1933	Under 60	6	3.63	0	0.28
	60-69	21	7.30	2	1.63
	70 and over	9	3.90	2	2.39
Total	All ages	47	16.30	13	8.37

Case control comparison of occupational risks

For each lung cancer known to have occurred in men 20 or more years after first exposure, 10 controls matched as closely as possible, and always within 5 yr, on date of birth and date of first employment were selected at random from men in the cohort who were still alive when the index case died. Ten eligible controls were found for all but 3 cases (2 with 7 controls and 1 with 9). The 93 cases shown in Table 7 were included, plus a further 4 who are known to have died since the follow-up date,* one who died at 85 yr of age and was therefore censored in the cohort analyses and 6 for whom lung cancer was mentioned on the death certificate but was not the certified cause of death. Duration of employment was calculated in six groups of scheduled areas, in maintenance and in non-scheduled areas up to the date of death of the index case. The overall results (Table 11) show a relative risk of 1.76 for men with ten or more years' exposure in scheduled areas compared with those with less, which is almost identical to the ratio of the corresponding SMRs for deaths occurring 20 or more years after first exposure (Table 7).† The corresponding relative risk for the 24 cases first exposed in 1951 or later is 3.27, which is substantially lower than 5.05, the ratio of SMRs based on the 19 cases beyond 20 yr after first exposure shown in Table 7. This reduction is due to

* Deaths of men in the cohorts under investigation are recorded regularly by the Office of Population Censuses and Surveys and our knowledge of them is unbiased by knowledge of their occupational history.

† The analyses took account of the matching, although with 10 controls per case unmatched analyses would give almost identical results.

TABLE 11. COMPARISON OF DURATION OF EXPOSURE OF LUNG CANCERS AND MATCHED CONTROLS IN SCHEDULED, NON-SCHEDULED AND MAINTENANCE WORK

Areas included in analysis	Duration of employment: Under 5 yr Cases	Under 5 yr Controls	5–9 yr Cases	5–9 yr Controls	10 yr or more Cases	10 yr or more Controls	Relative risk comparing 10 yr or more against under 10 yr
All men							
All scheduled areas	49	541	14	214	41	278	1.70
All non-scheduled areas except maintenance	94	907	8	61	2	65	0.33
Maintenance	97	962	2	23	5	48	1.08
All men							
All scheduled areas	49	541	14	214	41	278	1.76*
Men first employed in 1951 or later							
All scheduled areas	9	129	2	46	13	62	3.27

* In the top half of the table the effects of exposure in scheduled, non-scheduled and maintenance work were estimated simultaneously. The estimated effect of scheduled exposure shown in the bottom half is based on an analysis of this variable alone.

the fact that only one of the further five post-1950 cases included in the case-control analysis had worked for over 10 yr in scheduled areas. An analysis including scheduled, non-scheduled and maintenance work showed no increase in risk for maintenance (relative risk = 1.08, based on 5 cases) or non-scheduled work (relative risk = 0.33, based on 2 cases). The numbers of cases with long exposure in the separate scheduled areas are small (Table 12) and, although the relative risks for several of the larger areas are greater than one, none is individually statistically significant.

A similar comparison against matched controls for the 12 mesothelioma deaths (including the two that occurred since the follow-up date) that occurred more than 20 yr after first exposure is shown in Table 13. This analysis is, however, restricted to exposure occurring during the first 20 yr after first employment, as the induction period for mesothelioma is very long and later exposure is unlikely to be relevant. The only areas for which any cases occurred in men exposed for more than 10 yr were plaiting and weaving (4 cases), Harridge Mill—a fibre warehouse in which dusty opening and mixing operations were carried out and where crocidolite exposure may have occurred, but dust measurements were not made—(3 cases), carding and spinning (2 cases), and maintenance (1 case). The man with over 10 yr maintenance work had never been employed in a scheduled occupation, but both of the men with 5–9 yr non-scheduled exposure had also worked in Harridge Mill (one for 16 months and one for over 12 yr). There are too few cases to justify formal calculation of relative risks for individual areas, but the much higher risk in men exposed for more than 10 yr is again evident (relative risk 14.7; $P < 0.001$).

Lung cancer mortality in relation to dose and duration of exposure in the principal cohort

Exposure data. Our estimates of dose-specific risk are determined largely by the exposure data for the period 1951–60, which we have also used as a basis for assessing exposure levels between 1933 and 1950. These early measurements were in particles per millilitre (p ml^{-1}) and we have therefore presented the results in these units. Many of the particles counted were not asbestos fibres and, hence, it is uncertain how far the observed relationship provides a reliable guide to the effects of modern conditions measured in fibres per millilitre (f ml^{-1}). To present the results in this way, measurements in f ml^{-1} taken since 1961 have had to be converted to p ml^{-1}. The results cannot, however, have been much affected by any error in this conversion, as dust levels were substantially higher between 1933 and 1960 than in later years.

The exposure data available to us are in many respects similar in quality to those relating to chrysotile miners and millers in Quebec (MCDONALD *et al.*, 1980; DAGBERT, 1976) and have the advantage that the dusts to which the employees were exposed are likely to have included a smaller proportion that was not asbestos and they are probably superior to any available for other cohorts in which a substantial increase in risk has been observed. Routine sampling of particle counts using a thermal precipitator at 23 fixed sampling points began in 1951 and between 18 and 25 samples were taken annually at each sampling point in the years 1952–1955. The number of sampling points, sampling frequency and method of measurement varied in later years (see pp. 328 *et seq.* and Appendix C). Detailed studies of the results of parallel methods of measurement of both particles and fibres were conducted in 1977, although we suspect that the comparison of routine measurements obtained by different methods in successive years may provide a more reliable (although still far from satisfactory)

TABLE 12. COMPARISON OF DURATION OF EXPOSURE OF LUNG CANCERS AND MATCHED CONTROLS IN VARIOUS SCHEDULED AREAS

Areas included in analysis	Duration of employment: Under 5 yr Cases	Under 5 yr Controls	5-9 yr Cases	5-9 yr Controls	10 yr or more Cases	10 yr or more Controls	Relative risk,* comparing 10 yr or more against under 10 yr
Plaiting and weaving	94	897	2	67	8	69	1.25
Carding and spinning	78	846	13	101	13	86	1.66
Doubling	94	976	4	24	6	33	1.97
Harridge Mill	102	1010	2	17	0	6	0.00
Fiberizing	96	975	5	20	3	38	0.89
Other scheduled areas	96	970	3	26	5	37	1.31

* Estimated by fitting all six areas simultaneously.

TABLE 13. COMPARISON OF DURATION OF EXPOSURE DURING THE 20 YR FOLLOWING FIRST EMPLOYMENT, OF MESOTHELIOMAS AND MATCHED CONTROLS IN VARIOUS OCCUPATIONS*

Area	Duration of employment: Under 5 yr Cases	Under 5 yr Controls	5-9 yr Cases	5-9 yr Controls	10 yr or more Cases	10 yr or more Controls
Plaiting and weaving	8	110	0	5	4	5
Carding and spinning	8	95	2	14	2	11
Doubling	12	113	0	5	0	2
Harridge Mill	9	117	0	0	3	3
Fiberizing	12	113	0	3	0	4
Other scheduled areas	12	118	0	1	0	1
All scheduled areas	2	64	0	25	10	31
All non-scheduled areas except maintenance	10	108	2	8	0	4
Maintenance	11	109	0	5	1	6

* One man has been excluded—see footnote to Table 8 and text.

conversion factor. We have, therefore, used the conversion obtained from comparison of the measurements in 1960 and 1961 when measurements were made respectively in p ml^{-1} by the Casella thermal precipitator and in f ml^{-1} by the Ottway long-running precipitator: that is 1 f ml^{-1} = 35 p ml^{-1} (see Appendix C and DOLL and PETO, 1985).† Annual averages at selected sampling points between 1952 and 1972 (shown in Figs 1(a) to (d)) suggest that the substantial variation sometimes observed between average levels measured with the same instrument in successive years is due more to real differences in average levels than to random error, as there is a marked correlation between the patterns of fluctuation observed at different sampling points within the same area. Such changes did not usually occur at the same time in different areas, however, and they seem unlikely to have seriously biased the overall averages for 1960 and 1961 on which our particle to fibre conversion is based. The marked change for 'cheese winding' shown in Fig. 1(a), when the membrane filter results for 1965 were compared with those for previous and succeeding years, was due to moving the sampling point and indicates the great importance that the choice of sampling point may have.

Individual exposure estimates. Selected sampling points that provided similar results were grouped together, giving the classification into four categories for each 5-yr period since 1951 that is shown in Table 14. The mean values for 23 sampling points for the periods 1951–1955 and 1956–1960 on which this categorization is based are given in Table 15. For each period, jobs were assigned to the category that was judged to be most representative by a senior staff member in the Health Physics Department at the factory (RC). For most jobs this was unambiguous, as in many areas the majority of sampling points gave results in the same range and most occupations involving exposure exclusively or predominantly in such areas could be assigned with reasonable

† This conversion factor was used by two of the authors in their report to the Health and Safety Commission (DOLL and PETO, 1985). The results of a study conducted by the Company in 1977 and cited by the British Occupational Hygiene Society (1983), suitably modified to correspond to counting without a microscope eyepiece graticule, were similar (1 f ml^{-1} = 30 p ml^{-1}).

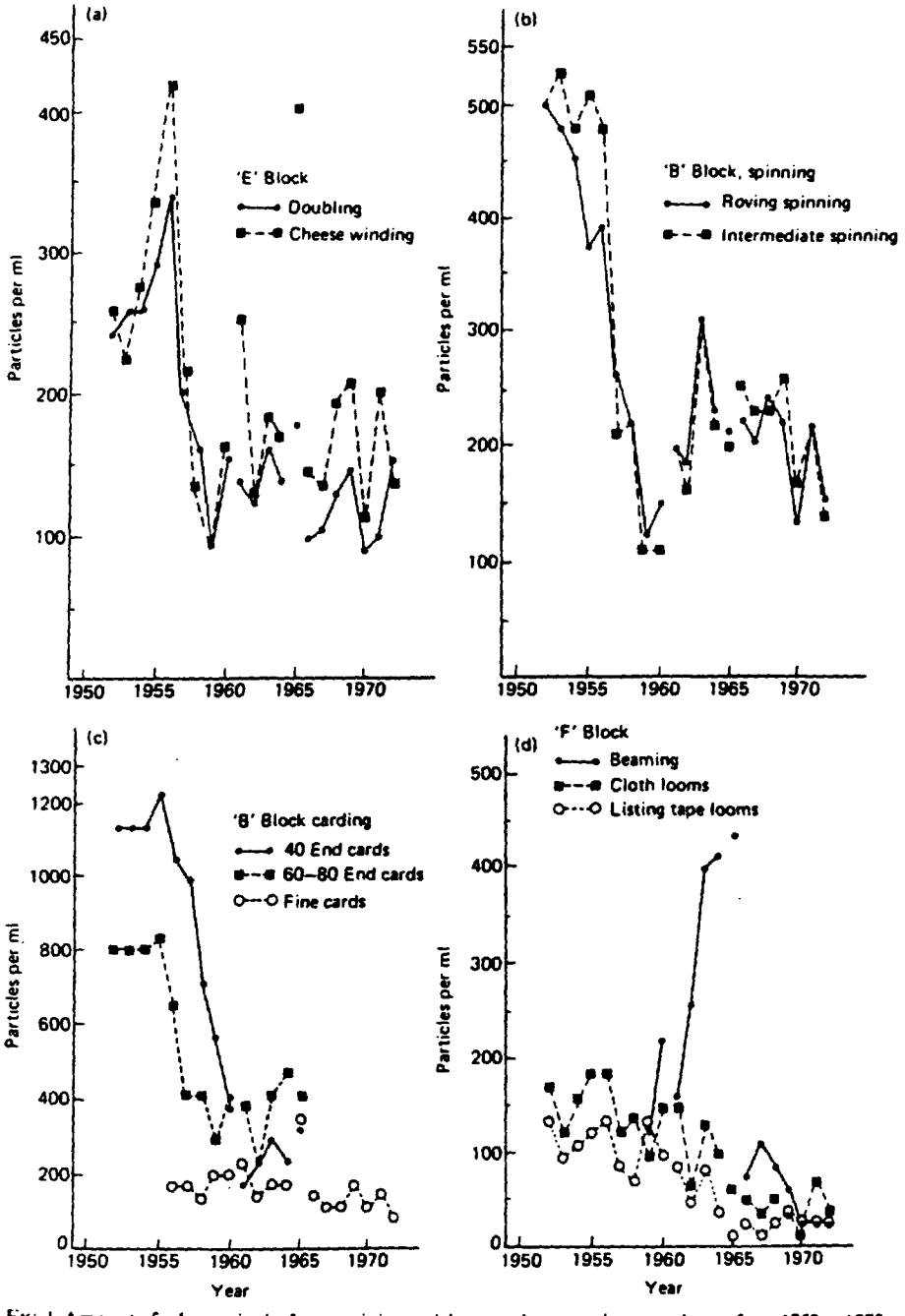

FIG. 1. Amount of asbestos in the factory air in particles per ml averaged over each year from 1952 to 1972 at two sampling points for doubling and cheese winding (Fig. 1(a)), two sampling points in spinning (Fig. 1(b)), three sampling points in the carding area (Fig. 1(c)) and sampling points for beaming, cloth looms and listing tape looms (Fig. 1(d)). Measurements were taken with a Casella thermal precipitator (1951–60), an Ottway long-running thermal precipitator (1961–64), a membrane filter (1965), and either a membrane filter or a Royco automatic particle counter (1966–72). Results taken since 1961 are converted to particles per ml to correspond to Casella TP measurements (see text).

TABLE 14. CLASSIFICATION OF SCHEDULED WORK AREAS BY DUST LEVEL* (letters in parentheses refer to blocks in the factory)

Classification	Period:	1951-55	1956-60	1961-65	1966-70	1971-74
Very high	Level:	978 p ml^{-1} 40 end cards (B) 60-80 end cards (B) Fibre unloading and stacking† Harridge Mill†	978 p ml^{-1} Fibre unloading and stacking† Harridge Mill†	20 f ml^{-1} Unloading (W) Stacking (W) Harridge Mill†	20 f ml^{-1} Unloading (W) Stacking (W) Harridge Mill†	None
High	Level:	490 p ml^{-1} Roving spinning (B) Inter spinning (B) Crightons (D) Mixing floor (D) NRA/felt cards (D) Electrical sliver cards (D)	568 p ml^{-1} 40 end cards (B) 60-80 end cards (B) Nuttall chopper (X)	15 f ml^{-1} Coarse single cards (B) 60-80 end cards (B) Nuttall chopper (X)	15 f ml^{-1} Coarse single cards (B) 60-80 end cards (B) Nuttall chopper (X)	None
Medium	Level:	270 p ml^{-1} Inter doubling (B) Fibre rope (D) Inter spinning (D) Opening machines (K) All other areas except fine spinning (E)	320 p ml^{-1} Crightons (D) Mixing floor (D) NRA/felt cards (D) Inter spinning (D) Electrical sliver cards (D) Roving and inter spinning (B) Beaming (E)	7.5 f ml^{-1} 40 end cards (B) Roving, inter spinning (B) Fine cards (B) Fibre rope cards (BW) Fibre rope braiders (BW) Cheese winding (E) Beaming (F) Rotary waste machines (X)	7.5 f ml^{-1} Carding sections 1-3 and 6-9 (B) Roving and inter spinning (B) Fibre rope cards (BW) Miracle mill (X)	7.5 f ml^{-1} Carding sections 1-3 and 6-9 (B) Roving and inter spinning (B)
Low	Level:	157 p ml^{-1} All other scheduled areas Maintenance	153 p ml^{-1} All other scheduled areas Maintenance	2.5 f ml^{-1} All other scheduled areas Maintenance	2.5 f ml^{-1} All other scheduled areas Maintenance	2.5 f ml^{-1} All other scheduled areas Maintenance

* Non-scheduled areas are regarded as causing no exposure.
† Very dusty areas not monitored, but classified subjectively or from later measurements.

TABLE 15. AVERAGE PARTICLE COUNTS (p ml^{-1}) IN 1951-55 AND 1956-60

Sampling point		Period					
		1951-55			1956-60		
Block	Operation	Mean	Number of samples	Category*	Mean	Number of samples	Category*
B	40 end cards	1145.9	88	VH	701.3	55	H
B	Inter spinning	507.4	88	H	268.1	36	M
B	60-80 end cards	809.6	88	VH	435.5	52	H
B	Roving frames	454.7	89	H	254.3	37	M
B	Inter doubling	382.9	89	M	227.7	30	L
K	Opening machines	211.0	87	M	167.0	24	L
D	Crighlons	450.3	88	H	395.9	30	M
D	NRA/felt cards	610.6	89	H	352.6	50	M
D	Fibre rope braiding	276.8	89	M	173.9	54	L
D	Mixing floor	467.6	89	H	368.7	30	M
D	Electrical sliver cards	449.4	89	H	400.7	54	M
D	Inter spinning	358.1	89	M	266.2	29	M
E	Flying doubler	260.0	89	M	218.8	34	L
E	Beaming	202.8	89	M	254.3	28	M
E	Fine spinning	161.6	87	L	Not sampled		—
E	Cheese winding	269.3	89	M	242.9	35	L
E	Fine cards	189.4	87	M	Not sampled		—
E	Pirn winding	280.8	88	M	195.5	28	L
U	Dry plaiting	148.0	84	L	141.9	30	L
F	Ordinary looms	153.0	86	L	158.3	17	L
F	Cloth weaving	156.5	87	L	137.7	45	L
F	Big looms	201.9	86	L	213.0	17	L
F	Listing looms	119.4	87	L	103.7	29	L

* Mean levels were averaged to obtain the average exposure levels for each category (VH = very high H = high M = Medium L = low) shown in Table 14. A further 11 sampling points (average 125.6 p ml^{-1}) also contributed to the average for the 'low' category for 1956-60.

confidence. Jobs that involved substantial exposure at various levels, or in areas where sampling was not conducted, were assigned on the basis of personal judgment. The primary basis for classification was the 'prefix number', a 3- or 4-digit employment code that specified the area and type of employment. When this was not recorded, the block and department coding was used. Jobs for which the block and/or department were not specified were classified individually.

This procedure suffered two major limitations. First, no routine measurements were taken before 1951, so that the assignment of exposure categories for men employed between 1933 and 1950 was less firmly based. There were few major process changes between these dates and most earlier jobs could be assigned 1951-55 exposure levels with reasonable confidence. In some areas, however, conditions had improved and a higher category (again at 1951-55 levels) was assigned for pre-1951 exposure. The second, and more important, difficulty was the absence of reliable data for the highest exposure levels. The highest measured levels in 1951-55 were recorded at two sampling points in the carding department (Fig. 1(c)), but other jobs in areas where no samples were taken, including loading and stacking operations and work in the Harridge Mill fibre warehouse, were known to involve very high exposure; these were arbitrarily assigned the average observed in the two highest carding levels in 1951-55.

TABLE 20. OBSERVED AND PREDICTED NUMBERS OF MESOTHELIOMAS
The predicted numbers (P_3) are based on a cubic residence time model with linear dose-response. (Alternative predicted numbers (P_4) based on a fourth-power residence time model are also shown.)

Duration of scheduled service (yr)		Years since first employment: Less than 20	20-24	25-29	30-34	35-39	40 or more	Total
Less than 1	Obs.	0*	0	0	0	0	0	0
	P_3	0.10	0.13	0.19	0.19	0.11	0.12	0.84
	P_4	0.05	0.10	0.18	0.20	0.13	0.17	0.84
1-4	Obs.	0	0	0	0	0	1	1
	P_3	0.07	0.09	0.12	0.12	0.09	0.08	0.57
	P_4	0.04	0.07	0.11	0.13	0.12	0.12	0.60
5-9	Obs.	0	0	0	0	0	0	0
	P_3	0.29	0.37	0.55	0.63	0.51	0.51	2.84
	P_4	0.15	0.27	0.50	0.68	0.65	0.76	3.01
10-19	Obs.	0	0	0	3	0	1	4
	P_3	0.40	0.40	0.49	0.47	0.31	0.21	2.27
	P_4	0.21	0.28	0.41	0.47	0.36	0.28	2.00
20-29	Obs.	..	1	1	2	1	0	5
	P_3		0.31	0.70	0.76	0.48	0.34	2.58
	P_4		0.22	0.59	0.74	0.53	0.42	2.49
30 or more	Obs.			–	0	0	0	0
	P_3			–	0.16	0.31	0.43	0.90
	P_4			–	0.16	0.35	0.56	1.07
Total	Obs.	0	1	1	5	1	2	10
	P_3	0.86	1.30	2.04	2.32	1.80	1.69	10.00
	P_4	0.44	0.93	1.79	2.39	2.13	2.32	10.00

* One man excluded—see footnote to Table 8 and text.

TABLE 21. COMPARISON OF OBSERVED AND PREDICTED MESOTHELIOMA INCIDENCE, BASED ON A CUBIC RESIDENCE TIME MODEL WITH LINEAR DOSE-RESPONSE, BY (A) CUMULATIVE DOSE DURING THE FIRST 10 YR OF EMPLOYMENT AND (B) TOTAL CUMULATIVE DOSE

Cumulative dose (p ml^{-1} yr)	A. Men who completed 10 yr total service, by cumulative dose during the first 10 yr service		B. All men, by total cumulative dose	
	Observed	Predicted	Observed	Predicted
< 1000	0	0.28	0*	0.83
1000 –	5	1.93	2	0.90
2000 –	0	0.85	1	1.00
3000 –	0	1.13	1	1.29
4000 –	2	2.55	1	1.12
5000 or more	3	3.27	5	4.87
Total	10	10.00	10	10.00

* One man has been excluded—see footnote to Table 8 and text.

published in reasonable detail are that of chrysotile miners and millers in Quebec (MCDONALD *et al.*, 1980) and that of the chrysotile textile workers in South Carolina studied independently by MCDONALD *et al.* (1983) and DEMENT *et al.* (1982); and in both cases detailed exposure data were described elsewhere (DAGBERT, 1976, and DEMENT, 1980) and only briefly summarized in the main reports. If linear dose–response is assumed, the dose-specific risk estimate, at least for lung cancer, can be estimated quite adequately from the average exposure and overall excess risk of long-service workers (PETO, 1978); but, if detailed exposure data are not presented, it is difficult to distinguish reports based on very sparse (and in some cases virtually non-existent) measurements of exposure taken more than 15 or 20 yr ago from the few for which there are extensive particle counts and some basis for their conversion to fibre counts. Even the best historical exposure data, and certainly our own, are seriously deficient in several important respects and an appreciation of these limitations is central to any assessment of the resulting risk estimates. The heaviest exposures were rarely measured, particle and fibre counts correlate poorly and it is not known whether a reduction in either implies a proportional reduction in the concentration of carcinogenic fibres, many of which are not counted by optical microscopy.

The lung cancer rate that we observed in men first employed in 1951 or later remained high in spite of the large reductions in measured particle counts that occurred in many areas between 1951 and 1960. The risk did not become substantial until at least 20 yr after first exposure, however, and our observations are therefore in effect limited to men first employed before about 1960. Further follow-up will be needed to show whether workers first employed in 1960 or later suffered a detectable increase in risk. The reasons for this difference between our results for men first exposed before and after the end of 1950 are not clear. One possibility is that changes in processing or in the raw fibre used increased the proportion of long but very fine fibres released that were invisible with the optical microscope. Exposure to crocidolite may also have varied. The annual amount used did not change much (see Appendix A), but the way in which it was processed may have done. Alternatively, the apparent difference may be, at least in part, an artefact due to changes in the methods of measurement of pollution. The

* One man excluded—see footnote to Table 8 and text.

conversion factors that have seemed appropriate may not have reflected adequately the carcinogenicity of the particles and fibres that were measured. The difference in the dose-specific estimates of risk (which is just statistically significant, whereas the difference between the relative risks actually observed 20–34 yr after first exposure is not quite significant) is due in part to the lower particle counts observed after about 1955 and our assumption that certain processes entailed higher exposure between 1933 and 1950 than between 1951 and 1955. It may be, too, that the ratio of fibres to particles in the ambient pollution (which was measured in terms of particles until 1961) increased with the reduction in general atmospheric pollution, thus exaggerating the amount of asbestos to which the pre-1951 employees appear to have been exposed.

The majority of long-service employees who began work in or before 1950 were still employed in 1951 and would also have been exposed for some years to the post-1950 conditions and the most plausible explanation of the difference in the relative risks is that it is largely, or even entirely, due to chance and that the difference in the trends in risk with estimated dose is further inflated because exposure to the most carcinogenic fibres was not reduced as much as the changes in particle counts between 1951 and 1960 would suggest. Our belief that this is the case is strengthened by the fact that the 11 additional cases of lung cancer that we were able to use for the case-control analysis in Table 11 included only one man with more than 10 yr scheduled service, who was first employed after 1950, against 4 with less than 10 yr, thus producing a lower relative risk in comparison with men with short exposure than that shown in Table 7. If this interpretation is correct, the estimate of relative risk based on the pooled data is more accurate than that based on men first employed since 1951, but our estimated fibre counts for the period before 1961, when the change from particle to fibre counting occurred, may be too high.

Our dose-specific risk estimates, which were given on pp. 332 and 334, were given in terms of particles per ml. If we assume a conversion factor of 35.3 particles per fibre, as is suggested in Appendix C, these are equivalent to an increase in SMR of 0.0054 per f ml^{-1} yr for the entire cohort and of 0.0150 per f ml^{-1} yr when analysis is restricted to men first employed in 1951 or later.

The only other study of textile workers that provides clear evidence of quantitative effects of exposure to chrysotile asbestos is that reported by MCDONALD *et al.* (1983). If we accept their suggested conversion factor of one million particles per cubic foot (measured with midget jet impingers) to six regulated f ml^{-1} (DOLL and PETO, 1985), the quantitative relationship that they obtain for lung cancer (an increase in SMR of 0.0125 per f ml^{-1} yr) is intermediate between those we estimated for our post-1932 and post-1950 cohorts. In view of the many assumptions that have had to be made in deriving these estimates, the consistency of the results is striking and suggests that for the purposes of prediction it would be sensible to use a rounded-off intermediate estimate of the effect given by

$$\text{SMR} = 1 + 0.01 \times \text{f ml}^{-1}\text{ yr}.$$

This estimate, it will be noted, is derived from observation of two cohorts of textile workers, one of which was exposed only to chrysotile while the other (reported here) was also exposed to small amounts of crocidolite. As, however, the amount was so small (5.0% of the amount used for the manufacture of textiles between 1932 and 1968: see Appendix A) and there is no clear evidence that crocidolite and chrysotile produce

different risks of cancer of the lung, two of us used this estimate in their report to the Health and Safety Commission (DOLL and PETO, 1985) to indicate the hazard from chrysotile. The dose-specific risk estimate for mesothelioma based on our data may, however, be too high for pure chrysotile, as there is consistent evidence that crocidolite causes a greater hazard of mesothelioma than chrysotile. MCDONALD *et al.* (1983), indeed, did not observe any pleural mesotheliomas in their study of chrysotile textile workers in South Carolina and the one peritoneal case that they did observe may have been caused by previous exposure to amphiboles. DOLL and PETO (1985) therefore suggested that, for the purposes of predicting the incidence of mesothelioma following exposure to a given amount of chrysotile, the risk estimate calculated from the Rochdale data should be halved. This rather arbitrary assumption constitutes an uneasy compromise between the South Carolina results, the observation of several cases among Quebec chrysotile miners and millers (MCDONALD *et al.*, 1980) and the data reported here; further data on the mesothelioma risk in chrysotile workers who suffered a substantial excess of lung cancer would be valuable.

The current control limit for chrysotile in the U.K. is 0.5 f ml^{-1} and, where this is properly enforced, average levels are unlikely to exceed about 0.25 f ml^{-1}. The corresponding predicted life-long risks of dying of lung cancer or mesothelioma, based on the models that we have adopted and assuming a conversion factor of 35 particles per fibre, are shown in Table 22, which is reproduced from DOLL and PETO (1985). These predictions, which are calculated from current male death-rates in England and Wales for lung cancer and other causes, suggest that the risk to men first employed at age 20 who are exposed for 35 yr will be approximately 0.8%. The mesothelioma risk is likely to be similar in smokers and non-smokers, but for lung cancer the risks for smokers will be about 50% higher than those shown in Table 22 and, for non-smokers, less than 10% of those shown.

The observation in this study that there were very few mesotheliomas in men with less than 10 yr exposure compared with those with more (1 against 25 expected at the rates for the latter) is of particular interest, as it is still widely believed that short exposure can cause a substantial risk of mesothelioma. A similar effect of duration of exposure was observed among Australian crocidolite miners (HOBBS *et al.*, 1980). The mesothelioma risk caused by short exposure thus seems to be extremely low, except

TABLE 22. PREDICTED NUMBERS OF ASBESTOS-INDUCED DEATHS DUE TO LUNG CANCER AND MESOTHELIOMA OCCURRING BEFORE AGE 80 YR AMONG 1000 MEN IN CHRYSOTILE TEXTILE MANUFACTURE EXPOSED TO A LEVEL OF 0.25 f ml^{-1}.
(Based on England and Wales male death rates in 1981–82 for lung cancer and all causes. DOLL and PETO, 1985.)

Age at first exposure (yr)		Duration of exposure (yr) 5	15	25	35
20	Lung cancer	0.93	2.79	4.62	6.29
	Mesothelioma	0.53	1.12	1.34	1.40
30	Lung cancer	0.94	2.79	4.48	5.66
	Mesothelioma	0.23	0.46	0.53	0.54
40	Lung cancer	0.93	2.64	3.85	4.24
	Mesothelioma	0.08	0.15	0.16	0.17

perhaps for very heavy exposure to amosite (Seidman *et al.*, 1979) or to crocidolite in manufacture (Jones *et al.*, 1980).

The limitations of the models that we have fitted for lung cancer and mesothelioma and of the corresponding predicted risks for different durations of exposure and ages at first exposure are discussed in Doll and Peto (1985). Two important qualitative predictions of these models are that the relative risk for lung cancer will be almost independent of age at first exposure and that the ratio of the excess lung cancer risk to the mesothelioma risk will increase sharply with increasing age at first exposure. Both are confirmed by our results for men first exposed at different ages who worked for ten or more years in scheduled areas (Table 23) and by comparable observations on North American insulation workers (Peto *et al.*, 1982). This qualititative consistency is, however, rather weak evidence for other more specific assumptions implicit in these models. There are too few mesotheliomas in this cohort for the cubic residence time model to be tested critically, but our data on lung cancer do not conform precisely to the predicted pattern in relation either to the passage of time or to duration of exposure.

Table 23. Lung cancer and mesothelioma mortality 20 or more years after first exposure among men exposed for 10 or more years in scheduled areas

Age at first exposure (yr)	Lung cancer Obs.	Exp.	SMR	Mesothelioma Obs.	Ratio of lung cancer excess to mesothelioma
Under 25	3	1.45	2.06	2	0.8
25-34	14	6.88	2.03	3	2.4
35 or over	23	10.79	2.13	4	3.1
Total	40	19.13	2.09	9	2.3

The reduction in relative risk that we observed beyond 35 yr after first exposure, which has also been observed in other studies (Walker, 1984) seems likely to be real, but the effect of different durations of exposure and, in particular, the risk to short-service workers, is less clear. The elevated risk in our study in men employed for less than a year before 1951 (44 observed, 30.51 expected; Table 7) seems unlikely to be occupational in origin, as it was unrelated to time since first exposure and did not occur in men employed for between 1 and 9 yr. We, therefore, suspect that this excess was caused by a combination of chance and selective bias, perhaps because some of these transient employees left the factory because of respiratory symptoms or smoked more heavily than the rest of the workforce or the local population. A disproportionate excess of lung cancer following short-term exposure that is too large to be readily attributable to chance or bias has, however, been observed in several other cohorts exposed to a variety of substances (Doll and Peto, 1985). This is an anomaly that merits further study, but is beyond the scope of this report.

Whatever the assumed model, the SMR for lung cancer can be estimated either as we have done, by comparing the observed mortality with the expected number calculated from local or national rates, or by using the data on workers with low cumulative doses to estimate the true underlying rate in the workforce studied in the absence of its occupational exposure. Such an internal control group may be more

Conditions in carding had greatly improved by 1956, but no material change occurred in these other dusty areas and they were allocated the same (1951–55) level from 1933 to 1960.

The dust levels (in p ml^{-1}) for each category shown in Table 14 for the period 1951–60 were calculated by averaging the results for the corresponding sampling points, with the exception mentioned above of 'very high' for the period 1956–60. The measurements (taken in f ml^{-1}) since 1961 were in certain areas very much more variable than the earlier particle counts, however, and the sampling points were changed several times between 1961 and 1972. It was, therefore, difficult to select an appropriate set of sampling points that could be regarded as representative, particularly for the higher exposure levels. For example, the annual means for 1966 at different sampling points in one area ranged from 1.5 to 35.6 f ml^{-1} and the annual means for the unloading platform in 1964, 1965, 1966 and 1967 were 3.0, 11.5, 30.0, and 16.4 f ml^{-1}, respectively. The levels assumed for different exposure categories for this period (from 20 f ml^{-1} for 'very high' to 2.5 f ml^{-1} for 'low'—see Table 14) were chosen as an approximation to the averages for the jobs assigned to them, but they were not formally calculated. They seem likely to be approximately correct, however, as they correspond quite closely in each category apart from the lowest to the observed average particle counts for 1951–60 (assuming a conversion factor of 35 particles to one regulated fibre*: see Appendix C).

We observed little or no excess risk before 20 yr after first exposure and ignored exposure in the 5 yr preceding death in all dose–response calculations. Our results are therefore dominated by the pre-1961 exposure estimates, so that any error in more recent estimates or in their conversion to particle counts cannot have greatly influenced our dose–response analysis.

Dose–response relationship

To allow for the delay between exposure to asbestos and any resulting increase in lung cancer mortality, man-years and deaths are allocated in the following analyses to categories of duration of exposure, average dose and cumulative exposure with a lag of 5 yr. Thus, for example, if a man achieved a cumulative exposure of 1000 p ml^{-1} yr on 1 July 1958, the man-years that he contributed up to 1 July 1963 (and his death, if he died before that date) are allocated to the first cumulative dose category (under 1000 p ml^{-1} yr). These analyses also differ from those in previous tables in the treatment of duration of exposure. Men were classified according to duration of service in scheduled areas in earlier analyses, whereas the dose–response analyses are based on total duration of employment in the factory.

Lung cancer mortality occurring 20 or more years after first employment is analysed by cumulative exposure in Table 16. Results are shown both overall and separately for men recruited in 1951 or later. The data are consistent with a linear increase in relative risk with increasing cumulative dose, the model that we have used as a basis for risk prediction (DOLL and PETO, 1985) and the combined results suggest that the SMR is increased by 1.53×10^{-4} per p ml^{-1} yr. The corresponding estimate based

* For the purpose of controlling exposure to asbestos, fibres are counted under the optical microscope that are more than 5 μm long and have aspect ratios (that is ratios of length to diameter) of more than 3 to 1. These are referred to here as 'regulated fibres'.

TABLE 16. OBSERVED AND EXPECTED LUNG CANCER MORTALITY 20 OR MORE YEARS AFTER FIRST EMPLOYMENT, BY CUMULATIVE DOSE ALLOWING FOR A LAG OF 5 YR.
(Addition of eligible cases not included in the principal cohort gives the observed and expected numbers shown in parentheses.)

Cumulative dose (p ml^{-1} yr)	Period first exposed: 1951 or later					Period first exposed: 1933 or later				
	Observed	Expected		Predicted*	Mean† dose	Observed	Expected		Predicted*	Mean† dose
< 1000	5 (5)	5.41	(5.41)	5.87	199	34 (34)	29.53	(29.59)	30.47	209
1000 –	3 (3)	1.99	(1.99)	3.24	1487	8 (8)	7.66	(7.68)	9.31	1409
2000 –	2 (2)	1.29	(1.29)	2.63	2448	11 (12)	6.60	(6.68)	9.14	2511
3000 –	0 (0)	0.59	(0.60)	1.46	3496	6 (6)	5.66	(5.68)	8.67	3474
4000 –	4 (5)	0.80	(0.81)	2.30	4420	10 (12)	4.29	(4.30)	7.28	4551
5000 or more	5 (5)	1.30	(1.31)	5.89	8331	24 (24)	10.83	(10.84)	25.84	9057
Total	19 (20)	11.37	(11.40)	21.39		93 (96)	64.56	(64.78)	90.71	

* Predicted numbers are calculated from the formula: Relative risk = $1 + k$ (mean dose), where $k = 4.24 \times 10^{-4}$ (post-1950 data) or 1.53×10^{-4} (all data).
The formula was fitted so as to give a minimum value of chi-square. An alternative method using maximum likelihood estimates of k gives values of 3.61×10^{-4} (post-1950 data) and 1.47×10^{-4} (all data). The minimum chi-square estimates when the additional cases are included become 4.99×10^{-4} (post-1950 data) or 1.73×10^{-4} (all data).

† Mean doses in p ml^{-1} yr were calculated from the cumulative exposures up to 5 yr before the death of the index case of the matched controls shown in Table 11.

only on men first employed in 1951 or later is, however, substantially higher (4.24×10^{-4}) and the difference between the estimates is statistically significant ($P < 0.05$). The effect of including the 1[illegible] eligible men who were missed when the original register was compiled (Table 3) is also shown in Table 16. This would probably lead to a slight overestimation of the risk, but the effect would in any case not be large.

Relationship with duration of exposure

The effect of duration of exposure, separate from level of exposure, is examined in Table 17. Each man's average exposure level throughout his period of employment was calculated by dividing cumulative dose by duration of employment up to that date, and a man exposed at different levels may therefore transfer several times to a higher or lower exposure level during employment, although not afterwards. As in Table 16 each transfer between exposure or duration categories is actually effected 5 yr after it occurred. The overall results suggest little or no excess risk when men are employed for under 10 yr and no increase with increasing duration of employment beyond 20 yr at any exposure level. At the highest level (over 400 p ml^{-1}) the risk appears to be virtually unrelated to duration of exposure owing to the excess in men exposed for less than 1 yr (12 observed, 7.60 expected, $P = 0.2$). The pattern of the results in Table 17 is not materially altered by restricting analysis to deaths occurring 20–35 yr after first exposure and cannot therefore be attributed to the correlation between duration of exposure and time since first exposure. The corresponding results for non-malignant respiratory disease (Table 18) show a similar pattern to those for lung cancer, except that death from asbestosis occurred only in men who had been employed for ten or more years.

An excess of lung cancer in short-service workers occurred only in men first employed before 1951 (see Table 7). We therefore analysed the overall mortality experience of men first employed before 1951 who worked for less than 10 yr, divided by duration of scheduled and total service (Table 19). The results for lung cancer tend to suggest that the excess is not asbestos-related, but there are too few men with no scheduled service to provide an adequate comparison group. For non-malignant respiratory disease the risk was rather greater among men with scheduled service, but again the difference does not approach statistical significance. The interpretation of these and other similar data is discussed more fully elsewhere (DOLL and PETO, 1985).

Quantitative relationship for mesothelioma

We have adopted a 'cubic residence time' model for mesothelioma which predicts that subsequent incidence is increased by each brief period of further exposure by an amount proportional to the intensity and duration of that exposure and to the cube of the time since it occurred (DOLL and PETO, 1985). This implies that the death rate at age t caused by prolonged exposure that began at age t_1 and ended at age t_2 at a constant level L will be given by the equation

$$k \cdot L \cdot [(t-t_1)^4 - (t-t_2)^4],$$

where k is a constant. Denoting ages at starting and ending the jth period of employment by t_{1j} and t_{2j}, respectively and the corresponding exposure level in p ml^{-1} by L_j, the overall death rate at age t will therefore be

TABLE 17. OBSERVED AND EXPECTED LUNG CANCER DEATHS 20 OR MORE YEARS AFTER FIRST EXPOSURE, BY DURATION OF EMPLOYMENT AND AVERAGE LEVEL OF EXPOSURE, ALLOWING A LAG OF 5 YR

Average exposure during employment (p ml^{-1})	Duration of employment: <1 yr Obs.	<1 yr Exp.	1–5 yr Obs.	1–5 yr Exp.	5–10 yr Obs.	5–10 yr Exp.	10–20 yr Obs.	10–20 yr Exp.	20+ yr Obs.	20+ yr Exp.	Total Obs.	Total Exp.
Zero (non-scheduled only)	3	2.79	2	2.26	1	0.78	2	0.61	0	0.65	8	7.09
1–199	5	3.56	3	2.17	3	5.61	8	7.87	8	5.20	27	24.41
200–399	2	1.66	1	1.17	1	1.75	12	3.66	6	3.15	22	11.39
400 or over	12	7.45	2	1.89	10	6.19	7	3.23	5	2.91	36	21.66
Total	22	15.45	8	7.49	15	14.33	29	15.38	19	11.91	93	64.56

TABLE 18. OBSERVED AND EXPECTED DEATHS FROM NON-MALIGNANT RESPIRATORY DISEASE 20 OR MORE YEARS AFTER FIRST EXPOSURE, BY DURATION OF EMPLOYMENT AND AVERAGE LEVEL OF EXPOSURE, ALLOWING A LAG OF 5 YR.
(Asbestosis deaths included, and also shown separately in parentheses.)

Average exposure during employment (p ml^{-1})	Duration of employment: <1 yr Obs.	<1 yr Exp.	1–5 yr Obs.	1–5 yr Exp.	5–10 yr Obs.	5–10 yr Exp.	10–20 yr Obs.	10–20 yr Exp.	20+ yr Obs.	20+ yr Exp.	Total Obs.	Total Exp.
Zero (non-scheduled only)	5	3.66	0	2.57	2	0.82	2	0.64	1	0.85	10	8.53
1–199	7	4.91	4	2.56	11	7.97	17	11.51	5	6.58	44	33.54
200–399	2	1.92	1	1.51	4	1.97	4	5.85	5 (1)	3.52	16 (1)	14.77
400 or over	17	8.32	3	2.80	14	9.15	8 (1)	4.11	8 (2)	3.64	50 (3)	28.01
Total	31	18.80	8	9.44	31	19.91	31 (1)	22.10	19 (3)	14.59	120 (4)	84.84

TABLE 19. MORTALITY OF MEN EMPLOYED FOR LESS THAN 10 YR WHO STARTED WORK BEFORE 1951, BY DURATION OF TOTAL SERVICE AND SCHEDULED SERVICE

Cause of death	Employed less than 1 yr						Employed between 1 and 9 yr					
	Some service in scheduled areas			No service in scheduled areas			Some service in scheduled areas			No service in scheduled areas		
	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR
Lung cancer	21	13.70	1.53	5	3.41	1.47	28	23.42	1.20	4	3.56	1.12
Mesothelioma	1*						0					
Other cancers	13	19.68	0.66	7	5.34	1.31	31	35.43	0.87	2	5.12	0.39
Asbestosis	0			0			1			0		
Other respiratory disease	35	17.37	2.02	5	5.06	0.99	48	34.48	1.39	4	4.20	0.95
Circulatory disease	75	62.14	1.21	24	17.26	1.39	129	115.47	1.12	11	15.67	0.70
Other diseases	9	14.15	0.64	5	4.15	1.20	26	25.92	1.00	3	3.86	0.78
Violence	8	5.85	1.37	1	1.65	0.61	15	9.29	1.62	1	1.69	0.59
All causes	162	132.88	1.22	47	36.87	1.28	278	244.01	1.14	25	34.10	0.73

* This is the man referred to in the footnote to Table 8.

$$I(t) = k \cdot \sum_j L_j[(t-t_{1j})^4 - (t-t_{2j})^4].$$

The model was fitted by computing the formula for $I(t)$ for each year of observation and period of employment for each man and choosing k so that the total predicted number equalled 10, the observed number. The predicted numbers of cases calculated in this way are shown in Table 20. The corresponding estimate of k is 3.52×10^{-12}. Thus, for example, the predicted death rate 40 yr after first exposure for a man who worked continuously for 15 yr at a level of 350 p ml^{-1} (which corresponds to approximately 10 f ml^{-1}; see Appendix C) and then left is $k \cdot 350 \cdot (40^4 - 25^4)$ or 2.7 per 1000 p.a. There are too few cases to test the model stringently, but the observed and predicted numbers in Table 20 are in reasonable agreement for different times since first employment. For duration of employment, however, the results suggest that men exposed for less than 10 yr suffered a rather lower risk than that predicted (1 observed, 4.25 predicted), in which case the risk for men who worked in scheduled areas for ten or more years may have been underestimated. Restricting analysis to men with ten or more years' scheduled service increases the estimate of k to 5.50×10^{-12}. The predicted number of cases in men first exposed in 1951 or later up to the end of June 1983 is 1.31, compared with 1 observed and a further 2 since follow-up closed.

There are too few cases for the effects of exposure level, duration of exposure and time since exposure to be examined in detail. In an attempt to assess the extent to which estimated exposure levels correlate with risk, at least in long-service workers, we have compared the numbers observed and those predicted by our model, restricting the analysis to men who completed 10 yr total service, and tabulated by the total dose (and hence average exposure level) up to the date of completing 10 yr service. If our model is even approximately correct, this early exposure is very much more important than subsequent exposure, and this comparison should therefore indicate whether low exposure is more or less dangerous than a linear dose–response would imply. The results are consistent with linearity (Table 21), but again there are too few cases for it to be tested critically. The corresponding comparison for all periods of service, tabulated by total cumulative dose (right-hand part, Table 21) shows an excellent correspondence between observed and predicted numbers at each level, but this comparison does not provide a very useful test of the model, as total cumulative dose gives equal weighting to any exposure, irrespective of when it occurred, and is also dependent on duration of exposure.

Expected numbers based on a fourth rather than third power residence time model are also shown in Table 20. The fit is virtually identical to that for the third-power model in relation to duration of exposure and slightly better in relation to time since first exposure. The difference in relation to overall predicted risk is, however, marginal, and much larger numbers would be needed to estimate the exponent precisely.

DISCUSSION

The detailed examination of the mortality patterns and exposure data in this report are, perhaps, as important as the actual risk predictions, which are similar to those based on an earlier analysis of a smaller study in this factory (PETO, 1978). The only other cohorts in which a substantial excess risk has been observed and for which the original particle counts and the basis for their conversion to fibre counts have been

TABLE 20. OBSERVED AND PREDICTED NUMBERS OF MESOTHELIOMAS

The predicted numbers (P_3) are based on a cubic residence time model with linear dose-response. (Alternative predicted numbers (P_4) based on a fourth-power residence time model are also shown.)

Duration of scheduled service (yr)		Years since first employment: Less than 20	20–24	25–29	30–34	35–39	40 or more	Total
Less than 1	Obs.	0*	0	0	0	0	0	0
	P_3	0.10	0.13	0.19	0.19	0.11	0.12	0.84
	P_4	0.05	0.10	0.18	0.20	0.13	0.17	0.84
1–4	Obs.	0	0	0	0	0	1	1
	P_3	0.07	0.09	0.12	0.12	0.09	0.08	0.57
	P_4	0.04	0.07	0.11	0.13	0.12	0.12	0.60
5–9	Obs.	0	0	0	0	0	0	0
	P_3	0.29	0.37	0.55	0.63	0.51	0.51	2.84
	P_4	0.15	0.27	0.50	0.68	0.65	0.76	3.01
10–19	Obs.	0	0	0	3	0	1	4
	P_3	0.40	0.40	0.49	0.47	0.31	0.21	2.27
	P_4	0.21	0.28	0.41	0.47	0.36	0.28	2.00
20–29	Obs.		1	1	2	1	0	5
	P_3		0.31	0.70	0.76	0.48	0.34	2.58
	P_4		0.22	0.59	0.74	0.53	0.42	2.49
30 or more	Obs.			—	0	0	0	0
	P_3			—	0.16	0.31	0.43	0.90
	P_4			—	0.16	0.35	0.56	1.07
Total	Obs.	0	1	1	5	1	2	10
	P_3	0.86	1.30	2.04	2.32	1.80	1.69	10.00
	P_4	0.44	0.93	1.79	2.39	2.13	2.32	10.00

* One man excluded—see footnote to Table 8 and text.

TABLE 21. COMPARISON OF OBSERVED AND PREDICTED MESOTHELIOMA INCIDENCE, BASED ON A CUBIC RESIDENCE TIME MODEL WITH LINEAR DOSE-RESPONSE, BY (A) CUMULATIVE DOSE DURING THE FIRST 10 YR OF EMPLOYMENT AND (B) TOTAL CUMULATIVE DOSE

Cumulative dose (p ml^{-1} yr)	A. Men who completed 10 yr total service, by cumulative dose during the first 10 yr service		B. All men, by total cumulative dose	
	Observed	Predicted	Observed	Predicted
< 1000	0	0.28	0*	0.83
1000 –	5	1.93	2	0.90
2000 –	0	0.85	1	1.00
3000 –	0	1.13	1	1.29
4000 –	2	2.55	1	1.12
5000 or more	3	3.27	5	4.87
Total	10	10.00	10	10.00

* One man has been excluded—see footnote to Table 8 and text.

published in reasonable detail are that of chrysotile miners and millers in Quebec (McDONALD *et al.*, 1980) and that of the chrysotile textile workers in South Carolina studied independently by McDONALD *et al.* (1983) and DEMENT *et al.* (1982); and in both cases detailed exposure data were described elsewhere (DAGBERT, 1976, and DEMENT, 1980) and only briefly summarized in the main reports. If linear dose-response is assumed, the dose-specific risk estimate, at least for lung cancer, can be estimated quite adequately from the average exposure and overall excess risk of long-service workers (PETO, 1978); but, if detailed exposure data are not presented, it is difficult to distinguish reports based on very sparse (and in some cases virtually non-existent) measurements of exposure taken more than 15 or 20 yr ago from the few for which there are extensive particle counts and some basis for their conversion to fibre counts. Even the best historical exposure data, and certainly our own, are seriously deficient in several important respects and an appreciation of these limitations is central to any assessment of the resulting risk estimates. The heaviest exposures were rarely measured, particle and fibre counts correlate poorly and it is not known whether a reduction in either implies a proportional reduction in the concentration of carcinogenic fibres, many of which are not counted by optical microscopy.

The lung cancer rate that we observed in men first employed in 1951 or later remained high in spite of the large reductions in measured particle counts that occurred in many areas between 1951 and 1960. The risk did not become substantial until at least 20 yr after first exposure, however, and our observations are therefore in effect limited to men first employed before about 1960. Further follow-up will be needed to show whether workers first employed in 1960 or later suffered a detectable increase in risk. The reasons for this difference between our results for men first exposed before and after the end of 1950 are not clear. One possibility is that changes in processing or in the raw fibre used increased the proportion of long but very fine fibres released that were invisible with the optical microscope. Exposure to crocidolite may also have varied. The annual amount used did not change much (see Appendix A), but the way in which it was processed may have done. Alternatively, the apparent difference may be, at least in part, an artefact due to changes in the methods of measurement of pollution. The

* One man excluded—see footnote to Table 8 and text.

conversion factors that have seemed appropriate may not have reflected adequately the carcinogenicity of the particles and fibres that were measured. The difference in the dose-specific estimates of risk (which is just statistically significant, whereas the difference between the relative risks actually observed 20–34 yr after first exposure is not quite significant) is due in part to the lower particle counts observed after about 1955 and our assumption that certain processes entailed higher exposure between 1933 and 1950 than between 1951 and 1955. It may be, too, that the ratio of fibres to particles in the ambient pollution (which was measured in terms of particles until 1961) increased with the reduction in general atmospheric pollution, thus exaggerating the amount of asbestos to which the pre-1951 employees appear to have been exposed.

The majority of long-service employees who began work in or before 1950 were still employed in 1951 and would also have been exposed for some years to the post-1950 conditions and the most plausible explanation of the difference in the relative risks is that it is largely, or even entirely, due to chance and that the difference in the trends in risk with estimated dose is further inflated because exposure to the most carcinogenic fibres was not reduced as much as the changes in particle counts between 1951 and 1960 would suggest. Our belief that this is the case is strengthened by the fact that the 11 additional cases of lung cancer that we were able to use for the case-control analysis in Table 11 included only one man with more than 10 yr scheduled service, who was first employed after 1950, against 4 with less than 10 yr, thus producing a lower relative risk in comparison with men with short exposure than that shown in Table 7. If this interpretation is correct, the estimate of relative risk based on the pooled data is more accurate than that based on men first employed since 1951, but our estimated fibre counts for the period before 1961, when the change from particle to fibre counting occurred, may be too high.

Our dose-specific risk estimates, which were given on pp. 332 and 334, were given in terms of particles per ml. If we assume a conversion factor of 35.3 particles per fibre, as is suggested in Appendix C, these are equivalent to an increase in SMR of 0.0054 per f ml^{-1} yr for the entire cohort and of 0.0150 per f ml^{-1} yr when analysis is restricted to men first employed in 1951 or later.

The only other study of textile workers that provides clear evidence of quantitative effects of exposure to chrysotile asbestos is that reported by McDONALD *et al.* (1983). If we accept their suggested conversion factor of one million particles per cubic foot (measured with midget jet impingers) to six regulated f ml^{-1} (DOLL and PETO, 1985), the quantitative relationship that they obtain for lung cancer (an increase in SMR of 0.0125 per f ml^{-1} yr) is intermediate between those we estimated for our post-1932 and post-1950 cohorts. In view of the many assumptions that have had to be made in deriving these estimates, the consistency of the results is striking and suggests that for the purposes of prediction it would be sensible to use a rounded-off intermediate estimate of the effect given by

$$\text{SMR} = 1 + 0.01 \times \text{f ml}^{-1}\text{ yr}.$$

This estimate, it will be noted, is derived from observation of two cohorts of textile workers, one of which was exposed only to chrysotile while the other (reported here) was also exposed to small amounts of crocidolite. As, however, the amount was so small (5.0% of the amount used for the manufacture of textiles between 1932 and 1968: see Appendix A) and there is no clear evidence that crocidolite and chrysotile produce

different risks of cancer of the lung, two of us used this estimate in their report to the Health and Safety Commission (DOLL and PETO, 1985) to indicate the hazard from chrysotile. The dose-specific risk estimate for mesothelioma based on our data may, however, be too high for pure chrysotile, as there is consistent evidence that crocidolite causes a greater hazard of mesothelioma than chrysotile. MCDONALD *et al.* (1983), indeed, did not observe any pleural mesotheliomas in their study of chrysotile textile workers in South Carolina and the one peritoneal case that they did observe may have been caused by previous exposure to amphiboles. DOLL and PETO (1985) therefore suggested that, for the purposes of predicting the incidence of mesothelioma following exposure to a given amount of chrysotile, the risk estimate calculated from the Rochdale data should be halved. This rather arbitrary assumption constitutes an uneasy compromise between the South Carolina results, the observation of several cases among Quebec chrysotile miners and millers (MCDONALD *et al.*, 1980) and the data reported here; further data on the mesothelioma risk in chrysotile workers who suffered a substantial excess of lung cancer would be valuable.

The current control limit for chrysotile in the U.K. is 0.5 f ml^{-1} and, where this is properly enforced, average levels are unlikely to exceed about 0.25 f ml^{-1}. The corresponding predicted life-long risks of dying of lung cancer or mesothelioma, based on the models that we have adopted and assuming a conversion factor of 35 particles per fibre, are shown in Table 22, which is reproduced from DOLL and PETO (1985). These predictions, which are calculated from current male death-rates in England and Wales for lung cancer and other causes, suggest that the risk to men first employed at age 20 who are exposed for 35 yr will be approximately 0.8%. The mesothelioma risk is likely to be similar in smokers and non-smokers, but for lung cancer the risks for smokers will be about 50% higher than those shown in Table 22 and, for non-smokers, less than 10% of those shown.

The observation in this study that there were very few mesotheliomas in men with less than 10 yr exposure compared with those with more (1 against 25 expected at the rates for the latter) is of particular interest, as it is still widely believed that short exposure can cause a substantial risk of mesothelioma. A similar effect of duration of exposure was observed among Australian crocidolite miners (HOBBS *et al.*, 1980). The mesothelioma risk caused by short exposure thus seems to be extremely low, except

TABLE 22. PREDICTED NUMBERS OF ASBESTOS-INDUCED DEATHS DUE TO LUNG CANCER AND MESOTHELIOMA OCCURRING BEFORE AGE 80 YR AMONG 1000 MEN IN CHRYSOTILE TEXTILE MANUFACTURE EXPOSED TO A LEVEL OF 0.25 f ml^{-1}.
(Based on England and Wales male death rates in 1981–82 for lung cancer and all causes. DOLL and PETO, 1985.)

Age at first exposure (yr)		Duration of exposure (yr) 5	15	25	35
20	Lung cancer	0.93	2.79	4.62	6.29
	Mesothelioma	0.53	1.12	1.34	1.40
30	Lung cancer	0.94	2.79	4.48	5.66
	Mesothelioma	0.23	0.46	0.53	0.54
40	Lung cancer	0.93	2.64	3.85	4.24
	Mesothelioma	0.08	0.15	0.16	0.17

perhaps for very heavy exposure to amosite (SEIDMAN *et al.*, 1979) or to crocidolite in manufacture (JONES *et al.*, 1980).

The limitations of the models that we have fitted for lung cancer and mesothelioma and of the corresponding predicted risks for different durations of exposure and ages at first exposure are discussed in DOLL and PETO (1985). Two important qualitative predictions of these models are that the relative risk for lung cancer will be almost independent of age at first exposure and that the ratio of the excess lung cancer risk to the mesothelioma risk will increase sharply with increasing age at first exposure. Both are confirmed by our results for men first exposed at different ages who worked for ten or more years in scheduled areas (Table 23) and by comparable observations on North American insulation workers (PETO *et al.*, 1982). This qualititative consistency is, however, rather weak evidence for other more specific assumptions implicit in these models. There are too few mesotheliomas in this cohort for the cubic residence time model to be tested critically, but our data on lung cancer do not conform precisely to the predicted pattern in relation either to the passage of time or to duration of exposure.

TABLE 23. LUNG CANCER AND MESOTHELIOMA MORTALITY 20 OR MORE YEARS AFTER FIRST EXPOSURE AMONG MEN EXPOSED FOR 10 OR MORE YEARS IN SCHEDULED AREAS

Age at first exposure (yr)	Lung cancer Obs.	Exp.	SMR	Mesothelioma Obs.	Ratio of lung cancer excess to mesothelioma
Under 25	3	1.45	2.06	2	0.8
25-34	14	6.88	2.03	3	2.4
35 or over	23	10.79	2.13	4	3.1
Total	40	19.13	2.09	9	2.3

The reduction in relative risk that we observed beyond 35 yr after first exposure, which has also been observed in other studies (WALKER, 1984) seems likely to be real, but the effect of different durations of exposure and, in particular, the risk to short-service workers, is less clear. The elevated risk in our study in men employed for less than a year before 1951 (44 observed, 30.51 expected: Table 7) seems unlikely to be occupational in origin, as it was unrelated to time since first exposure and did not occur in men employed for between 1 and 9 yr. We, therefore, suspect that this excess was caused by a combination of chance and selective bias, perhaps because some of these transient employees left the factory because of respiratory symptoms or smoked more heavily than the rest of the workforce or the local population. A disproportionate excess of lung cancer following short-term exposure that is too large to be readily attributable to chance or bias has, however, been observed in several other cohorts exposed to a variety of substances (DOLL and PETO, 1985). This is an anomaly that merits further study, but is beyond the scope of this report.

Whatever the assumed model, the SMR for lung cancer can be estimated either as we have done, by comparing the observed mortality with the expected number calculated from local or national rates, or by using the data on workers with low cumulative doses to estimate the true underlying rate in the workforce studied in the absence of its occupational exposure. Such an internal control group may be more

appropriate in some circumstances, but we have preferred to use population-based SMRs in the analysis of our data, for four reasons. First, short-term workers may suffer consistently higher mortality than long-term workers, as they constitute a self-selected sub-group. As noted above, we suspect that this occurred in the present study. Second, the assumption that the observed dose-response will be linear may be wrong, either because the model is incorrect or because short-term workers suffer high exposure due to differences in work practices or in the jobs that they are given compared with experienced workers in the same area. Our third reason for relying on general population rates is that the majority of the lung cancers in our study occurred in men born between about 1890 and 1930, and the corresponding British lung cancer rates are among the highest in the world, owing to the high proportion of life-long smokers in Britain. It would therefore be surprising to find an occupational group whose underlying SMR for lung cancer in men exceeds the national average by more than 50%, although in other countries and among British women, this might not be true. The fourth reason is that our study gave a dose-specific risk estimate based on men first exposed since 1951 that was higher than that among men first exposed between 1933 and 1950. An excess risk in short-term workers occurred only in men first exposed before 1951, however, and an internal analysis of relative risk would thus increase (and, in our opinion, exaggerate) this discrepancy.

The small excess of gastro-intestinal cancer observed in this study was not statistically significant. A review of these and other data suggests, however, that such excesses have been consistently observed among asbestos workers, together with a similar small excess of cancers of many other sites; DOLL and PETO (1985) suggest that much, and perhaps all, of these excesses may be due to mis-diagnosis of lung cancers or mesotheliomas that presented clinically as cancers of other types.

In conclusion, we should perhaps emphasize again that our exposure estimates are determined largely by static particle counts taken between 1951 and 1960 and estimates of particle counts for the period 1933–1950 when no routine measurements of any kind were taken. We have converted these to fibre counts in an attempt to predict the effects of current regulations, but the resulting risk estimates are necessarily of doubtful accuracy.

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The computing involved in preparing the original register of names of employees in the factory and in matching them against the national mesothelioma register was carried out by Mrs Irene Stratton. We were also assisted throughout by the factory staff at all levels and in particular by members of the Personnel Department, who gave the fullest co-operation despite the heavy demands made on them. Mr N. Rhodes, Director and General Manager, Mr S. Marks, Personnel Director, and Mr R. Sykes, Senior Manager (Safety and Environmental Control) provided a mass of detailed information, without which we could not have carried out the study.

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APPENDIX A

USE OF AMPHIBOLE ASBESTOS IN THE ROCHDALE WORKS

The asbestos textile works in Rochdale has used chrysotile as its principal raw asbestos from the time it started until today. Before 1965 most was imported from Rhodesia; from 1965 to 1980 all came from Canada; since 1980 Canadian chrysotile has predominated, but a small amount has come from Zimbabwe.

No amosite was used at any time for production purposes,* but some crocidolite was used between 1932 and 1969. Direct purchase of crocidolite began in 1932 and the amount then bought lasted until 1944, when purchases again began to be made and were continued until 1951. The fibre was used to manufacture crocidolite fibre rope and a small amount of carded felts for subsequent resinating with phenolic resins; the former involved fiberizing the asbestos as received from the mines, carding it, and braiding round the fibre core. Between 1933 and 1958 crocidolite was also purchased by another firm and made into crocidolite yarn which was sold to the Rochdale works, where it was doubled (i.e. the strands were twisted together), wound into packages and, mostly, converted into plaited packings for use as an acid-resistant sealing material. Beginning in 1956 more crocidolite was bought and mostly transferred to the factory at Hindley Green where crocidolite yarn and fabric were made, which replaced that previously purchased from elsewhere. From then on until the use of crocidolite ceased, some 25 tonnes of the amount purchased each year were used for the continued production of fibre rope. A small amount of crocidolite cloth was also rubber-coated at Rochdale for the manufacture of acid-resistant joints. The amounts purchased by the two firms and used at the Rochdale works each year between 1932 and 1968 are shown in Table A.1. The total amount purchased for use over the 37 yr amounted to 10 322 tonnes (or 279 tonnes/yr), which was approximately 2.6% of the total amount of asbestos purchased over the same period and approximately 5% of the amount used for the manufacture of textiles.

* A few hundred pounds were used for research in the early 1950s.

Table A.1. Amount of crocidolite asbestos purchased for use at the Rochdale works

Year	Short tons	
	Purchased directly	Purchased elsewhere for manufacture of yarn for sale to Rochdale works
1932	1106	0
1933	0	53
1934	0	340
1935	0	269
1936	1	107
1937	0	0
1938	0	291
1939	0	430
1940	0	245
1941	0	306
1942	0	223
1943	0	0
1944	2	100
1945	20	687
1946	10	224
1947	10	336
1948	10	158
1949	0	173
1950	10	402
1951	10	172
1952	0	820
1953	0	390
1954	0	183
1955	0	289
1956	80	3
1957	120	391
1958	140	28
1959	100	0
1960	70	0
1961	225	0
1962	225	0
1963	190	0
1964	270	0
1965	279	0
1966	328	0
1967	242	0
1968	254	0
Total	3702	6620

APPENDIX B

LIST OF MESOTHELIOMA CASES

Details of 59 men and women who were reported to have developed (or possibly to have developed) a mesothelioma are listed in Tables B.1 and B.2, according to whether the diagnosis of mesothelioma was accepted or rejected.

Certified cause

Only the underlying cause of death is cited, unless mesothelioma appeared in some other position on the certificate.

Diagnostic category

(1) Established:
  (a) Diagnosed macroscopically at operation or *post mortem* examination and diagnosis confirmed histologically.
  (b) Diagnosed macroscopically at *post mortem* examination, no histological evidence available.
  (c) Diagnosis confirmed histologically by Dr Christopher Wagner, despite an element of doubt in some other reports.

(2) Presumptive: Diagnosis based solely on death certificate attribution.

(3) Uncertain: Diagnosis suggestive of mesothelioma, but evidence conflicting.

TABLE B.I. DETAILS OF EMPLOYEES WHO DEVELOPED MESOTHELIOMAS (all pleural unless specified as peritoneal)

	Case	Birth	Year of first employment	Year of last employment	Death	Duration of employment (yr) Total	Duration of employment (yr) In scheduled areas	Certified cause of death	Diagnostic category
Men	1	1907	1925	1961	1974	36	32	Malignant mesothelioma	1(a)
	2*	1908	1923	1949	1978	26	0	Pleural mesothelioma	1(a)
	3	1915	1951	1980	1982	29	29	Pleural mesothelioma	1(b)
	4	1895	1914	1920	1967	9/12	9/12	Myeloid leukaemia (with associated cancer of lung)	1(c)
	5	1907	1929	1950	1975	12	12	*Pleural mesothelioma*	1(b)
	6	1907	1922	1964	1964	37	1	Malignant pleural tumour	1(a)
	7	1905	1941	1968	1968	26	26	Carcinoma of pleura	1(b)
	8	1905	1927	1967	1972	40	29	Mesothelioma	1(a)
	9	1904	1929	1964	1975	35	35	Mesothelioma	1(a)
	10	1913	1936	1972	1972	27	27	Malignant mesothelioma	1(a)
	11*	1914	1937	1938	1979	1	1	Malignant mesothelioma	1(a)
	12*	1907	1926	1939	1968	13	13	Malignant mesothelioma (peritoneum)	1(a) (peritoneal)
	13	1919	1937	1970	1970	27	27	Malignant mesothelioma	1(a)
	14*	1914	1946	1947	1950	4/12	4/12	Endothelioma of pleura	2
	15*	1903	1919	1938	1958	17	8	Carcinoma of lung	3
	16	1910	1929	1946	1970	15	9	Malignant pleural mesothelioma	1(a)
	17*	1909	1929	1949	1975	20	0	Duodenal ulcer (with associated mesothelioma)	1(a)
	18	1907	1940	1970	1970	29	19	Pleural mesothelioma	1(a)
	19*	1917	1951	1951	1974	2/12	0	Mesothelioma	1(b)
	20	1904	1927	1965	1976	38	38	Mesothelioma	1(b)
	21	1906	1937	1950	1979	12	12	Pleural mesothelioma	1(b)
	22	1871	1913	1936	1936	23	23	Endothelioma of pleura	1(b)

	[illegible]	[illegible]	1931	1951	1974	2/12	0	Mesothelioma	1(b)
	20	1904	1927	1965	1976	38	38	Mesothelioma	1(b)
	21	1906	1937	1950	1979	12	12	Pleural mesothelioma	1(b)
	22	1871	1913	1936	1936	23	23	Endothelioma of pleura	1(b)
	23	1900	1918	1961	1973	42	42	Malignant mesothelioma	1(a)
	24	1942	1959	1983	1984	24	24	(Diagnosis confirmed, but death certificate not obtained)	1(a)
	25*	1921	1952	1972	1984	20	0	(Diagnosis confirmed, but death certificate not obtained)	1(a)
	26	1909	1947	1971	1971	24	24	Adenocarcinoma of lungs due to asbestosis	1(a)
	27*	1912	1936	1972	1975	24	24	Malignant lung tumour (mesothelioma)	1(a)
	28	1906	1929	1941	1968	12	10	Pleural mesothelioma	1(a)
	29	1909	1923	1968	1968	45	45	Mesothelioma	1(a)
	30	1907	1923	1971	1974	46	16	Mesothelioma of lung	1(b)
	31	1915	1936	1979	1979	24	1	Mesothelioma of lung	1(b)
	32	1907	1927	1966	1966	19	18	Carcinoma of lung due to asbestosis	1(a)
	33	1907	1947	1972	1979	25	14	Coronary occlusion	1(a)
	34	1908	1939	1951	1974	10	10	Malignant pleural mesothelioma	1(a)
Women	35*	1922	1937	1968	1976	8	0	Malignant pleural mesothelioma	1(b)
	36	1907	1929	1971	1980	42	38	Mesothelioma of lung	1(a)
	37	1911	1928	1937	1973	9	0	Malignant pleural mesothelioma	1(a)
	38	1898	1915	1916	1959	4/12	4/12	Carcinoma of lung	1(a)
	39	1909	1926	1936	1977	10	5	Pleural mesothelioma	2
	40	1910	1924	1969	1969	23	21	Malignant mesothelioma	1(a)
	41	1912	1926	1972	1979	38	38	Pleural mesothelioma	1(a)
	42	1911	1954	1960	1968	4	4	Malignant mesothelioma	1(a)
	43	1930	1950	1968	1979	12	8	Pleural mesothelioma	2
	44*	1907	1962	1972	1972	10	0	Malignant mesothelioma	1(a)
	45	1917	1931	1977	1981	43	37	Mesothelioma	1(b)
	46	1911	1926	1935	1966	9	9	Carcinoma of lung	1(a)
	47	1923	1943	1946	1970	3	3	Malignant mesothelioma of lung	1(a)

* See notes.

TABLE B.2. DETAILS OF EMPLOYEES IN WHOM THE DIAGNOSIS OF MESOTHELIOMA WAS REJECTED

	Case	Birth	Year of First employment	Year of Last employment	Death	Duration of employment (yr) Total	Duration of employment (yr) In scheduled areas	Certified cause of death	Reason for suspicion	Diagnosis after review
Men	101*	1902	1944	1967	1969	23	0	Mesothelioma	Death certificate	Poorly differentiated carcinoma of lung
	102	1908	1952	1967	1967	15	15	Carcinoma of lung	Company report	Carcinoma of lung
	103	1906	1955	1972	1972	17	17	Carcinoma of lung	Company report	Adenocarcinoma of lung
	104	1910	1935	1971	1973	30	30	Asbestosis	Company report	Asbestosis
	105	1910	1954	1971	1971	17	12	Carcinoma of lung	Company report	Carcinoma of lung
	106*	1912	1939	1957	1983	18	18	Pleural mesothelioma	Death certificate	Oat cell carcinoma of lung
	107	1911	1929	1969	1969	30	30	Neoplasm with multiple secondaries	Pneumoconiosis	Squamous carcinoma of lung
	108	1915	1947	1973	1975	23	20	Carcinoma of lung	Company report	Squamous carcinoma of lung
	109*	1904	1950	1957	1973	7	7	Myocardial failure with associated mesothelioma	Death certificate	Carcinoma of lung
Women	110	1904	1949	1949	1975	2/52	2/52	Carcinoma of lung	Company report	Carcinoma of lung
	111	1909	1929	1969	1977	33	31	Bronchopneumonia with associated fibrosis of lungs	Company report	Asbestosis
	112*	1911	1940	1970	1974	30	30	Carcinoma of lung (mesothelioma)	Mesothelioma register	Carcinomatosis (adenocystic carcinoma)

* See notes

APPENDIX B. NOTES

Case 2 Subsequently employed at Hindley Green, 1949 to 1967, non-scheduled work throughout.

11 Previously employed by another asbestos company, 1930 to 1937.

12 Previously employed by another asbestos company, 1922 to 1923.

14 Only known previous employment as a mill labourer (not asbestos) 1928 to 1939, Army 1940–1946.

15 Clinical history: April 1956 breathlessness 4/12
November 1956 X-ray of chest, some opacity L. lung base, elevated diaphragm and paradoxical movement. Bronchoscopy normal apart from some displacement. Bronchogram normal.
May 1957 L. laryngeal palsy. X-ray of chest bilateral pleural shadow Bronchoscopy L. bronchus rigid and slightly reddened.
July 1957 Chest opened: extensive tumour over L. pleura invading chest wall, diaphragm, and pericardium, unresectable. No clear indication of primary site.
Histology: small spheroidal cells with large nuclei and some mitoses, both layers of pleura. Vigorous fibroblastic activity. Tumour in bronchial fat consistent with bronchial primary Hospital letter June 1971 'diagnosis of endothelioma has been made'.
No material available for review.

17 Subsequently employed at Hindley Green, 1949 to 1971.

19 Previously employed at Hindley Green, 1945 to 1951.

25 Maintenance work throughout period of employment.

27 Also employed at Hindley Green, 1957 to 1969.

35 Employed as a telephonist or clerk throughout. No known exposure to asbestos at work elsewhere, but possible exposure at home, as her husband was employed as an asbestos weaver for 2 yr in the mid-1950s.

44 Maintenance work throughout period of employment.

101 *Post mortem* examination. Neoplasm involving apex of right pleural cavity, spreading into neck tissue and upper part of right upper lobe including the bronchus. Numerous small neoplastic deposits in the right visceral and parietal pleurae, small deposits in anterior mediastinal, paratracheal, and subcarinal lymph nodes. Small pearly plaques in left parietal pleura and upper diaphragmatic surface. Small neoplastic deposits in right adrenal and a large neoplastic mass in the left adrenal. Histology: perivascular nodular fibrosis of lungs with heavy deposits of carbon and occasional asbestos bodies; pleural neoplasm showed papillary adenocarcinoma consistent with diagnosis of mesothelioma. Diagnosed mesothelioma due to asbestosis. Reviewed by Dr Christopher Wagner 'I am quite certain that this is not a mesothelioma, but is a poorly differentiated carcinoma.'

106 Clinical history: 1980, diagnosed cor pulmonale and perforated duodenal ulcer; May 1981, persistent shadowing right middle and lower lobes of lung, no tumour on bronchoscopy; February 1982, shadowing left upper lobe of lung suggestive of tuberculosis, but tuberculosis excluded; December 1982, left apical lesion spread to pleura and possibility of mesothelioma considered. Radiotherapy for carcinoma of bladder.
Post mortem examination October 1983. Left pleura is grossly thickened and replaced by fleshy pink tumour lining whole of left side of thoracic cage, left diaphragm and large part of pericardium. Left lung largely destroyed by cavitating tumour. Right pleura slightly thickened. Liver contains multiple secondary deposits. Bladder thick and shaggy, but no remnant of tumour. Diagnosed disseminated primary mesothelioma of left pleura. Subsequent histological examination: oat cell carcinoma of lung with well developed asbestosis. Pneumoconiosis Medical Panel diagnosed oat cell carcinoma of lung and asbestosis.

109 Subsequently employed at Hindley Green, 1957 to 1969.
Clinical history: March 1973 pain R. shoulder 4.12, malaise 6·12, recent difficulty in swallowing. Too ill for special examinations and died within a week of admission.
Post mortem examination: tumour infiltrating upper lobe R. lung, secondary deposits in R. adrenal. Severe coronary atheroma with scarring in anterior wall of R. ventricle.
Histology (Dr Christopher Wagner): features compatible with mesothelioma of epithelial type, evidence of asbestosis in lungs. Pneumoconiosis Medical Panel diagnosed carcinoma of lung on macroscopic appearance.

111 *Post mortem* examination. Scar below left ear, site of previous radiotherapy. Large tumour in upper lobe right lung smaller tumour in lower lobe with deposits throughout left lung. Histology: two opinions, (i) appearances consistent with mesothelioma, (ii) tumours all look alike and do not have the structure of a mesothelioma. In places cylindromatous appearances, in others a carcinoid picture: probably secondary deposits from previous tumour below left ear, would fit with malignant salivary tumour. Reviewed by Dr Christopher Wagner: adenocystic carcinoma.

* See notes.

APPENDIX C

R. Doll and J. Peto

CONVERSION OF PARTICLE TO FIBRE COUNTS

Measurements of the amount of 'asbestos' in the factory air have been made routinely since 1951 as follows:

Period	Instrument	Method of evaluation	Parameter	Unit
1951–60	Casella thermal precipitator (CTP)	Incinerated, × 1000 dark field	Particles (including fibres)	p ml^{-1}
1961–64	Ottway long running thermal precipitator (LRTP)	Not incinerated, × 500 light field	Fibres > 5 μm long, ratio length to diameter > 3:1	f ml^{-1}
1965–74	Membrane filter sampler	× 500 phase contrast, full field	Fibres > 5 μm long, ratio length to diameter > 3:1	f ml^{-1}
	or			
	Royco automatic particle counter (RPC)	Automatic	Fibres > 5 μm long, ratio length to diameter > 3:1	f ml^{-1}
1975 to date	Membrane filter sampler	× 600 phase contrast, graticule grid count	Fibres > 5 μm long, ratio length to diameter > 3:1	f ml^{-1}

The particles obtained by the CTP were counted down to a diameter of 0.5 μm. They are not the same as the particles counted in North America, where the instrument used was normally a midget jet impinger. Counts in North America were usually expressed as so many million particles per cubic foot (mppcf). Arithmetically 1 mppcf ≈ 35.3 p ml^{-1}, but the counts cannot be equated in this way, because the instruments used operated on different principles and give different readings for the same degree of pollution.

Both the LRTP and the RPC can provide counts equivalent to those obtained with a membrane filter sampler. The RPC, however, counts particles as well as fibres and is calibrated against the membrane filter sampler under fixed conditions. In certain areas parallel RPC and membrane filter results correlated so poorly that the RPC was not routinely used.

Data collected before 1961 in the form of p ml^{-1} have somehow to be converted to f ml^{-1}, where f stands for the fibres defined by regulations: that is, more than 5 μm long with ratio of length to diameter more than 3:1. The conversion from one to another is, however, of uncertain validity and the use of a single conversion factor under different conditions may give rise to serious error. Analysis of the results of comparative measurements made simultaneously at the Rochdale factory in 1977, which were reported by the Committee on Asbestos of the British Occupational Hygiene Society (1983), led to the conclusion that, using the graticule grid method referred to below, membrane filter counts were related to particle counts by the CTP by the formula

$$f\ ml^{-1} = 0.071\ p\ ml^{-1} + 0.6.$$

For reasons which are discussed in detail elsewhere, we did not think it appropriate to use this formula in our report to the Health and Safety Commission (Doll and Peto, 1985) but preferred to use the relationship derived from comparing the average results obtained by the CTP and the LRTP in routine practice at the same sampling points in the 2 yr 1960 and 1961, which included higher readings more typical of earlier periods than the parallel measurements made in 1977. These average measurements are illustrated in Fig. C.1. We have preferred, too, not to derive the relationship from the regression equation, as random error in the original measurements will tend to flatten the slope below its true value and increase the constant in the equation, giving an overestimate of the fibre count at very low levels and an underestimate at high levels, even if the underlying relationship is in fact linear, and it is preferable to constrain the fitted line to pass through the origin to avoid this bias. The simplest way to achieve this, and the one that we have adopted, is to use the ratio of the averages of the results obtained by the two methods. This is a robust procedure, particularly when random variation on both measurements is large and is not dominated by one or two extreme values. Thus, for example, the correlation between the 1960 particle and 1961 fibre counts is halved when the highest reading is omitted (see Fig. C.1) but the ratio of the averages is hardly altered, falling from 34.0 to 33.0 particles per fibre. As it happens (perhaps coincidentally), regression analyses of the 1977 parallel measurements gave a similar conversion factor, although the formula cited above is different, because an

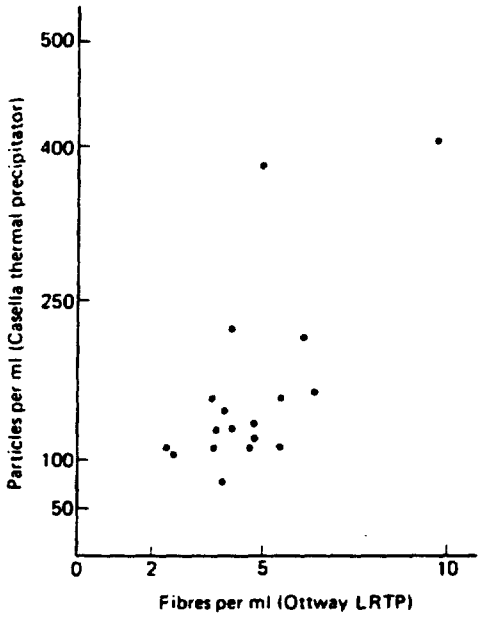

FIG. C.1. Relationship between amount of atmospheric pollution in 18 different parts of the factory measured in particles per ml by the Casella thermal precipitator in 1960 and in fibres per ml measured by the Ottway long running thermal precipitator in 1961.

allowance was made for converting the counts made by the older 'full field' method to those made with the graticule grid, as is discussed below. In our report to the Health and Safety Commission (DOLL and PETO, 1985) we took as the conversion factor, 1 f = 35.3 particles, as this had the corollary of equating 1 f ml^{-1} with 1 mppcf and much of our discussion was concerned with comparing the results obtained in Britain and North America. For the present purpose a factor of 34 would have been more logical, but the effect of the change would have been trivial and not justified by the precision of the estimates.

Two further changes that have complicated comparison of current levels with those measured in the past are the introduction of the graticule grid method of counting fibres and the substitution of personal sampling for static background sampling of selected areas. The former has certainly helped to reduce intra- and inter-observer errors in counting and resulted in approximately doubling the number of fibres counted in Rochdale (BECKETT *et al.*, 1976); but it did not always appear to have the same effect elsewhere and, in our report (DOLL and PETO, 1985), we preferred to ignore it. It is possible, too, that it was counteracted by the change from static to personal counters.

The personal sampler requires measurements to be made by an instrument attached to the coat lapel of an individual worker and has sometimes been regarded as again approximately doubling the counts, but it is far from clear that this is a proper generalization. STEEL (1979), in the report of the Advisory Committee on Asbestos, accepted a factor of 2 as representative, but pointed out that the factor could vary from about 1 to 10. In areas where static measurements (and hence, ambient levels in the building) are less than about 1 f ml^{-1}, personal measurements have usually been found to be considerably higher than the static figures, perhaps because occasional work practices or proximity to emission sources make a substantial contribution, in such circumstances, to the total inhaled dose. The few observations for which the static measurements exceeded about 1.5 f ml^{-1} have, however, shown no clear tendency for the corresponding personal measurements to be higher, as is shown in Figs 3 to 6 of Appendix 2 to the BRITISH OCCUPATIONAL HYGIENE SOCIETY's (1983) report.

We have, however, used the detailed observations that were made in the Rochdale works, on which one of the BRITISH OCCUPATIONAL HYGIENE SOCIETY's (1983) figures was based, to test the hypothesis that the ratio between the results obtained by personal and static sampling (and hence the conversion factor that relates measures of pollution obtained by the two methods) tends to diminish as the amount of pollution increases. We have, therefore, plotted the logarithm of the ratio against the logarithm of the geometric mean of each

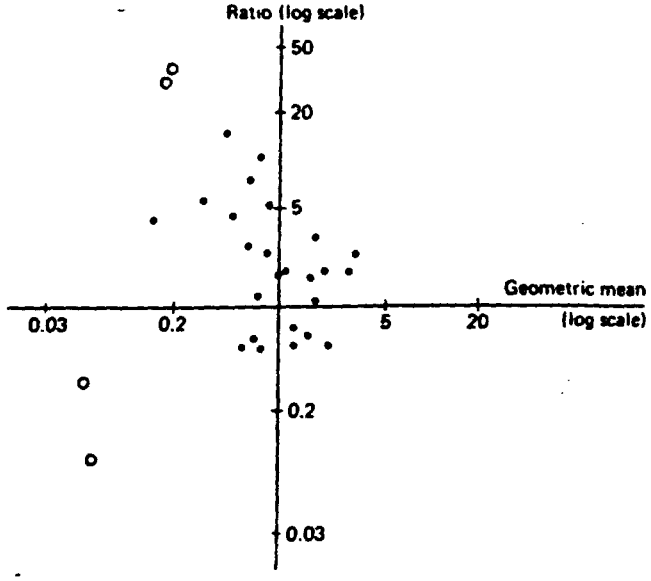

FIG. C.2. Relationship between measurements of ambient pollution by asbestos fibres obtained simultaneously for static samples and nearest operative's personal sample in the factory in 1977: logarithm of ratio of measurements (personal to static) plotted against logarithm of geometric mean of each pair of counts. Unreliable observations based on very low values for either static or personal samples shown as ○.

pair of observations* and show the results in Fig. C.2. The correlation is extremely poor ($r = -0.051$) owing, however, to two outlying points that are based on exceptionally low and unreliable static sample readings. The four points with unreliably low readings (two static and two personal samples) are indicated separately in the figure and, if they are disregarded, the correlation strongly suggests that the ratio diminishes as the mean increases and becomes less than one (i.e. the personal samples fall below the static) when the geometric mean of the readings approaches 2 f ml^{-1}.

We have noted, too, that measurements at the Rochdale factory in 1971, when average dust levels in many areas exceeded 2 f ml^{-1}, were reported as being consistently lower for personal than for static samples in most areas when yearly mean levels were compared (SMITHER and LEWINSOHN, 1973) and, for the purpose of converting measurements taken at the static sampling points in this factory to those that would have been obtained by personal sampling in 1971 or earlier, these figures suggest that the past measurements would be more appropriately halved than doubled.

It is impossible to determine precisely what effect these two changes have had on the comparison between regulated fibre counts as now carried out and previous pollution measured as particles. They may cancel each other out and we have preferred to ignore them.

* To test the hypothesis that the difference between two measurements, x_1 and x_2, of the same quantity, both with normally distributed random error, is independent of q, the true value, it is usual to calculate the correlation between the arithmetic mean of the measurements $\frac{1}{2}(x_1 + x_2)$ and the difference $(x_1 - x_2)$. This avoids the spurious positive correlation between the initial reading x_1 and the observed change $(x_1 - x_2)$ that is produced by regression to the mean. Fibre counts, however, are distributed approximately log normally (DAGBERT, 1976). If, therefore, we designate the logarithm of a personal count p and a parallel static measurements s by x_1 and x_2, respectively, a useful test of the independence of the personal to static ratio and the true dust level is provided by the correlation of the logarithm of their ratio, $x_1 - x_2$, against the logarithm of their geometric mean, $(x_1 + x_2)/2$.

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