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A SURVEY OF DUST EXPOSURE AND LUNG DISEASE IN
THE ASBESTOS-CEMENT INDUSTRY IN
NEW SOUTH WALES

Cecil G. Roberts and Harry A. Whitte

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HISTORICAL

Probably the earliest recorded case of asbestosis was that reported by Montague Murray in London, in 1900, concerning the death of a workman, aged 33, who had been exposed to asbestos dust for some fourteen years. In 1910, after the Registrar-General had drawn attention to the death of an asbestos worker from "acute pulmonary fibrosis", medical examinations were made of all employees in the factory which had employed him, and the various aspects of the industry (e.g. dust, ventilation, sickness) were investigated. This investigation indicated, but did not prove, that asbestos possessed injurious properties. A later case concerned a woman, aged 33, who died in 1924 after eighteen years' exposure to asbestos. The post-mortem examination revealed extensive fibrosis of the lungs.

In 1930 Merewether and Price reported to the Home Office on the results of their investigation during 1928-9 into the asbestos industry in England. It was carried out on that group of persons, mainly engaged in textile manufacture, who were exposed to pure asbestos, or asbestos mixed with a very small proportion of cotton or other vegetable fibre. In all 363 persons out of about 2500 were examined, those with the longest employment in each factory being selected. Of those examined 95 were found to have diffuse fibrosis of the lungs. The authors considered that with exposure to high dust concentrations the fibrosis would be fully developed in seven to nine years, with possible death after about thirteen years; with lesser dust concentrations, maturation of the fibrosis might take as long as twenty-five years. Unfortunately, only relative figures of dust concentration are shown, and most of these figures apply to processes connected only with the asbestos textile industry. Opening and handling fibre, without local exhaust ventilation, would be regarded as a high dust concentration operation, and this is the only one listed that would be common to both the asbestos textile and asbestos-cement sections of the industry. The authors found that neither age nor sex appeared to have any effect on the incidence of the fibrosis.

In 1936 a report was published by S.B. McPherson of a survey of a group of 210 persons exposed to asbestos dust in the asbestos-cotton industries. Twenty doubtful, twenty-five moderate and eight advanced cases of asbestosis were found. The average exposure of the whole group of thirty-three persons with moderate or advanced asbestosis was 10.3 years. Paradoxically, those with moderate asbestosis (10.3 years) had a greater exposure than those with advanced asbestosis (10.0 years). There was some evidence that asbestosis develops more rapidly in younger persons. Those under forty years of age developed moderate asbestosis in 9.5 years and advanced asbestosis in 8.0 years; the corresponding figures for workers over forty years were 12.4 and 11.0 years, respectively. No activating or aggravating effect of the dust on tuberculosis was observed.

Two years later the United States Public Health Service published its study by Dreessen et al., of asbestosis in the asbestos textile industry. Medical examinations were carried out on 492 men and 118 women in three factories. The dust samples were taken by Greenburg-Smith impinger. In addition, chemical analyses were made of settled dust taken from ledges at breathing level, and of airborne dust collected by bag-type filter. Owens' dust counts were used for sizing. The total dust exposure of each workman in millions of particle-years per cubic foot was found by multiplying the dust count for each operation by the number of years spent on it, and summing the products so obtained. Division by the total number of years the worker was exposed gave the weighted dust concentration experienced by the workman over that period. This method of assessing exposure has been adopted in this report.

Since their tests were made by impinger, a direct comparison is possible with the tests of the present survey which were also taken by impinger, and this is done later.

Only three persons out of 108 exposed to a weighted dust concentration below five million particles per cubic foot, but 70 out of 339 persons exposed to a greater concentration were affected by asbestosis. From these figures, the authors concluded "Because clean-cut cases of asbestosis were found only in dust concentrations exceeding five million particles per cubic foot, and because they were not found at lower dust concentrations, five million particles per cubic foot may be regarded as the threshold value for asbestos-dust exposure until better data are available.

The most significant work on asbestosis experimentally produced in animals was recently carried out in the United States by Vorwald, Durkan and Pratt. The animals used were guinea pigs, rats, mice, rabbits, cats and dogs. From the results of their experiments, the authors made these observations, among others.

1. Long chrysotile asbestos fibres produced a peribronchiolar fibrosis of the lung, similar to human asbestosis, in all animals except the mouse and the dog. The reason for the failure of the mouse and the dog to respond is not known. Long asbestos fibres were necessary to produce the reaction.
2. The mode of action of the long asbestos fibre is primarily mechanical, not chemical, and appears to depend on flexing action, since inflexible glass wool fibres do not produce fibrosis, nor do asbestos fibres which have had their flexibility destroyed by heat.
3. Evidence was produced showing that dust concentrations of 138 million particles per cubic foot, containing 0.6% fibres greater than ten microns in length (i.e. under 1 million asbestos fibres greater than ten microns per cubic foot) will produce experimental asbestosis in guinea pigs. The actual lower limit of concentration of long fibres necessary to produce experimental asbestosis was not established.
4. The duration of exposure required to develop the pulmonary reaction to inhaled asbestosis dust is inversely proportional to the concentration of long

particles in the air. From the authors' figures, about 25 million particle-years per cubic foot would produce a very early asbestosis, whilst with an exposure of 50 million particle-years per cubic foot the fibrosis would be quite marked.

5. Further conclusions reached were that experimental asbestosis ceases to progress on discontinuance of dust exposure and that inhalation of asbestos dust did not alter significantly the final outcome of experimental tuberculosis in two series of guinea pigs exposed to that dust.

DESCRIPTION OF THE INDUSTRY

The major activity of the asbestos industry of New South Wales is the manufacture of asbestos cement and its fabrication into flat and corrugated sheeting and into a large range of moulded products. In this report only this side of the industry will be dealt with, but it is intended to deal with other sections of the industry in a separate report.

The manufacture of asbestos-cement products in New South Wales is confined to three firms operating in Sydney who in this section of the industry employ approximately one thousand persons.

The raw materials used are asbestos, cement and water. The asbestos is received in bags from West Australia, South Africa, Canada and a small quantity from Baryulgil in New South Wales. From these sources the following types of asbestos are obtained: amosite (an amphibole mineral), chrysotile (fibrous serpentine) and crocidolite (blue asbestos).

The cement is mainly received in bulk, either by rail wagons or trucks, which are emptied direct on to screw conveyors. The conveyors feed bucket elevators and belts which carry the cement to storage bins. A certain amount of cement is received and stored in bags against any hold-up in bulk supplies. On arrival the bags of asbestos are manhandled and stored in stacks. As required the bags are again manhandled and conveyed from the store to the pulverisers or disintegrators. This manhandling of bags is carried out by groups of men generally known as "asbestos gangs". The next step is blending of the asbestos by emptying and mixing with shovels the contents of appropriate bags. The mix is then shovelled or hand-fed into the pulverisers. This work is carried out by a further group, sometimes known as "blower men". The pulverisers are either pug or hammer mills. The pulverised product is then blown through ducts to storage hoppers, usually known as fibre-rooms. As required, the blown asbestos is generally raked through small doors into barrows, weighed and then wheeled to the tide mills or beaters where the asbestos-cement mix is prepared. Into these machines water is run and the weighed asbestos fibre added. Towards the end of the beating period, ranging from 15 to 40 minutes, a weighed quantity of cement is added from a conveyor belt. Only those operatives taking part in these earlier stages of the manufacturing process are likely to be exposed to excessive dust, but there is a minor process of reclaiming dry scrap which gives rise to a lesser exposure, but one which is, nevertheless, a good deal greater than that found in the subsequent stages of manufacture where the processes are mainly carried out wet. It might be mentioned here that there

is little exposure to cement as opposed to asbestos dust and in any case cement dust does not constitute any serious hazard to the lungs.

From the tide mill or beater the liquid mix of asbestos-cement is run into an agitator whence it is fed as required on to felt belts of the sheet making machines. Here most of the water is removed by filtration and suction through the belt. The sheet now in course of preparation is transferred from the belt on to rollers of circumference appropriate to the length of the sheet required, and is built up to a desired thickness as the rollers rotate. It may be cut and removed from the rollers either manually or mechanically. For a flat sheet, it is then stacked between steel plates, but when a corrugated sheet is required, it is first hand moulded on a corrugated steel sheet and then stacked between corrugated sheets. In some cases the handling of these wet sheets is entirely manual and in others highly mechanised. The sheets are then allowed to stand for curing and drying. The reaction which takes place is exothermic and hastens the process. Some sheets are taken away from the machines for the manufacture of a wide range of moulded articles. After moulding, these articles are placed in dryers to expedite the curing and drying process and so increase the turnover of moulds.

After the sheets have been cured and dried, they are in most instances trimmed to size by guillotine, band-saws and emery wheels. A recent development has been wet trimming of the sheets to the final size prior to the curing and drying process. In this way the amount of dry scrap is being reduced and with it the exposure to dust already mentioned in connection with dry scrap.

ASSESSMENT OF THE DUST EXPOSURE

In New South Wales, dust concentrations are usually determined by the Owens' dust counter, an average of several counts being made over a suitable period of time, usually one half-hour or more. However, in order to be able to compare our figures with corresponding American ones, and with the American standard of five million particles per cubic foot, it has become customary to use the midget impinger when sampling atmospheres containing airborne asbestos.

The midget impinger, a more portable development of the Greenburg-Smith impinger, is a jet-type instrument in which 0.1 cubic feet of air per minute is drawn through a one-millimetre orifice, and impinged on to the flask bottom five millimetres below. The flask contains ten cubic centimetres of a collecting liquid which, in this Division, is usually twenty percent ethyl alcohol in distilled water. The liquid is carefully filtered before use through a double thickness of filter paper in order to remove any suspended dust.

After test, the collecting liquid is diluted to a suitable volume. This volume depends on visual assessment of the amount of dust collected in the liquid. An aliquot part is then run into a counting chamber. The one we use is a Spencer bright-line haemocytometer. From the cell count, the total number of particles in the collecting liquid can be calculated. The volume in cubic feet of air samples is found by dividing the time in minutes of sampling by ten. Division of the former quantity by the latter gives the dust concentration in particles per cubic foot.

There are both advantages and disadvantages in the use of the midget impinger. The microscope set-up, used for counting does not resolve particles below one micron in size. This may not be such a disadvantage if the larger particles of asbestos (e.g. fibres over ten microns in length) have the significance ascribed to them by some investigators. Shattering of the larger particles tends to occur, but this defect is common to all jet-sampling instruments in which the dusty air is drawn through an orifice at high velocity. In any test, only one sample need be obtained, instead of a series of samples as with the Owens' dust counter. This facilitates counting, but does not allow assessment of the relative dustiness of the different phases of an operation, nor may a statistical analysis be made of the result.

Generally, dust samples taken in this survey covered a period of ten minutes, or longer if all phases of the operation being tested had not been covered. The total exposure of each workman in millions of particle-years per cubic foot was determined by multiplying the dust count for each operation by the number of years he had spent on it, and summing the products so obtained.

The weaknesses of this method of assessing a workman's dust exposure are obvious. It assumes that the dust concentration obtained by test is approximately the mean dust concentration to which that man has been exposed over a period of years; it ignores both short-term (e.g. hourly) fluctuations in the dustiness as well as long-term (e.g. over periods of years) trends towards more dusty or less dusty conditions. The former can be minimised by making more dust tests at all working points, but the latter cannot be estimated without having taken tests some years before in the same places. Unfortunately, this had not been done in any of the factories concerned.

Full details of the dust tests taken, are not given in this report but, in Table 1, which follows, the average, the minimum and the maximum dust counts are shown for the processes described above. They are set out in descending order of average dustiness.

Table 1

Position of Tests	No. of Tests	Nature of Work	Midget Impinger Count mill. part./cub. ft.		
			Average	Low-est	High-est
Asbestos pulverisers & disintegrators	4	Asbestos unbagged, blended by shovel & fed to disintegrator	15.7	3.5	24.7
Tide Mills and Beaters	6	Blown asbestos usually raked into barrows & wheeled to beater. Wet-mixed with cement in beater	2.6	0.1	7.8
Scrap bagging points	3	Pulverised & blown scrap filled into bags from hoppers	2.6	0.8	5.3

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Table 1 (continued)

Location of Tests	No. of Tests	Nature of Work	Midget Impinger Count Mill. ft./cub. ft.		
			Average	Low-est	High-est
Fibre rooms	3	Woven asbestos raised into burrows from fibre rooms	1.1	0.3	2.3
Cutters and Bandsaws	5	Corrugated asbestos-cement sheets and moulded articles trimmed to size	1.6	0.3	4.0
Scrap pulverisers and disintegrators	3	Scrap asbestos-cement shovelled & hand-fed to disintegrator	1.6	0.5	3.4
General factory activities, here (handling & moulding wet sheets)	3	Asbestos-cement sheets being manufactured nearby, and other items being hand-moulded	0.3	0.2	0.4

DESCRIPTION OF THE POPULATION SURVEYED AND ITS EXPOSURE TO DUST

The total number of employees surveyed was 175, out of a possible total of 900, and they were all men. It will be seen from Table 2 that a large proportion of the employees

Table 2

No. of Years	No. of Employees
0 - 4.9	99
5 - 9.9	43
10 - 14.9	18
15 - 19.9	10
20 & over	5
TOTAL	175

surveyed, 99 out of a total of 175 had been employed for less than 5 years. This is a point of considerable importance for, as already mentioned, asbestosis has only been found to occur after prolonged exposure and, even with work in very high dust concentrations, the disease is rare in workers with less than 5 years' exposure. Furthermore, many of those surveyed had worked on several different jobs during their employment in the industry and there was a tendency for employees with long years of service to have been moved from processes accompanied by heavy concentrations of dust to processes where there was little dust. Consequently both the considerable staff turnover and turnover of job within the industry tended to reduce individual exposure to

dust. It will also be seen from comparing tables 1 and 3 that a large proportion of the men, 105 out of 175, were employed on jobs where the dust concentration was on the average 0.3

Table 3

Distribution of Employees on Various Types of Work Tested at Time of Survey

Type of Work	No. of Employees
Asbestos Pulverisers & Disintegrators	18
Tide Mills & Beaters	42
Scrap bagging points ^x	0
Fibre Rooms	2
Cutters & Bandsaws	3
Scrap Reclamation Plant	5
General factory atmosphere (handling, moulding wet sheets)	105
TOTAL	175

^x No employees occupied entirely on this work.

million particles per cubic foot and such exposure can be regarded as negligible. Having regard to the two factors of turnover of job and widely different concentrations of dust experienced on different processes, it was considered that a very suitable method of assessing the individual's exposure to dust was to measure it in terms of particle-years per cubic foot. For this purpose dust concentrations for each process were taken as those found at the time of the survey. In the factories where the survey was carried out it seemed probable that dust conditions had been fairly stable for a considerable period. Nevertheless, the possibility of dust concentrations varying from time to time cannot be excluded and the estimate of exposure can be no more than an approximation. The results obtained by this method are classified in Table 4.

Table 4

Classification of Individual Exposure to Dust

Million particles per cubic foot x years	No. of Employees
0 - 4	127
5 - 9	18
10 - 24	16
25 - 49	11
50 - 99	0
100 and over	3
TOTAL	175

Previous surveys carried out elsewhere, and already referred to, have shown that asbestosis is not likely to occur unless exposure has exceeded 5 million particles for more than 5 years, that is to say 25 million particle-years. It will be seen from the table that in only 14 cases had this level of exposure been reached and in view of this a low incidence of asbestosis was to be expected and was, in fact, found.

CLINICAL AND X-RAY FINDINGS

The clinical examination of all the subjects was carried out by medical officers of the Division of Industrial Hygiene and chest x-rays, either by the Tuberculosis Division of the Health Department or by the consultant radiologists to the Division. For the x-ray examinations 14" x 11" films were used in all cases. Where exposure had reached a level at which asbestosis might be expected, the films were reviewed by two or more persons skilled in the assessment of such films and the final judgement on the film was based by one of us (C.G.R.) on the general consensus of opinions given by the viewers. In reaching the diagnosis the clinical findings were taken into consideration, but the clinical evidence of disease was negligible and in only two cases did the symptomatology contribute to a diagnosis of asbestosis. The following table related the cases of asbestosis to dust exposure.

Table 5
Incidence of Asbestosis related to Dust Exposure

Exposure in Million particle-years per cubic foot	Persons Exposed	Persons Affected
Less than 10	145	0
10 - 24	16	2
25 - 49	11	4
50 - 99	6	0
100 and over	3	3
TOTAL	175	9

It will be seen that only two cases of asbestosis occurred among men with less than 25 million particle-years per cubic foot exposure. In both these cases the estimated exposure was 15 million particle-years per cubic foot. Dreessen et al, whose survey has already been referred to,

found 5 cases in 196 persons with exposure of less than 25 million particle-years per cubic foot. It has been pointed out that such estimations of exposure are no more than approximations and it is quite possible that these stray cases where exposure was apparently within the limits of safety were at some period exposed to higher concentrations of dust than were found to exist during the survey on the processes where the men had worked. The incidence of cases, four out of eleven persons exposed at the 25-49 million particle-years per cubic foot level of exposure, is considerably higher than that found by Dreesen, namely three cases out of eighty-four persons exposed. It is interesting that the small number of three persons who had an exposure of over 100 million particle-years per cubic foot all showed signs of asbestosis compared with roughly half in Dreesen's survey. However, the numbers with high levels of exposure found in our survey are so small in comparison with Dreesen's that the differences mentioned are of no statistical significance.

Advanced stages of asbestosis are seldom found with exposure of less than 100 million particle-years per cubic foot and in this survey only one case was found where the disease could be regarded as having passed the early stage and the x-ray revealed the characteristic ground glass appearance of the lung fields. In this case the estimated exposure was 167 million particle-years per cubic foot.

Table 6 shows the relationship between the incidence of asbestosis and the length of exposure to asbestos dust. The latter is not necessarily identical with length of employment in the industry (Table 2), for certain occupations (e.g. watchman, delivery man, clerk) have no exposure to the dust.

Table 6
Incidence of Asbestosis related to
Exposure Time

Exposure Time in years	Persons Exposed	Persons Affected
Less than 5	100	1
5 - 9.9	42	5
10 - 19.9	26	2
20 and over	5	1
TOTAL	175	9

It will be seen that only about one percent of those workmen with less than five years' exposure were affected whilst about ten per cent of those with more than five years' exposure showed signs of asbestosis. These figures are somewhat lower than those found by Dreesen et al, who reported 10 cases (3%) in 288 persons exposed for

less than five years, and 63 cases (40%) in 159 persons exposed for five or more years. The difference between the two surveys is statistically significant and, since we have considered here only periods of exposure, would probably be caused by the higher dust concentrations found in the asbestos textile industry.

A similar table showing the relationship between the incidence of asbestosis and the weighted dust concentration, is given below.

Table 7
Incidence of Asbestosis related to
Weighted Dust Concentration

Weighted Dust Concentration in million particles per cubic foot	Persons Exposed	Persons Affected
Less than 5	157	4
5 - 9.9	13	1
10 - 19.9	5	4
20 and over	0	0
TOTAL	175	9

In this table nearly three per cent of the workmen exposed to a weighted dust concentration of less than 5 million particles per cubic foot were affected. So were 22% of those exposed to a weighted dust concentration of five million or more particles per cubic foot. Corresponding figures from the report of Draessen et al were 3 out of 108 employees (or 3%) and 70 out of 339 employees (or 21%). It is obvious that there is no significant difference between our figures, as given in this table, and the corresponding American ones.

The number of cases with physical signs, or X-ray evidence of respiratory disease other than asbestosis, 22 out of 175 (Table 8) is higher than might be expected.

Table 8
Cases of Respiratory Disease other than
Asbestosis

Disease	Number of Cases
Tuberculosis - Active	2
" Healed	4
Bronchitis	11
Other	5
TOTAL	22

In the two cases of active pulmonary tuberculosis discovered by the survey, the disease was early and exposures to dust were only 1 million and 6 million particle-years per cubic foot respectively. There was nothing to suggest activation of the disease due to dust exposure. One case of healed tuberculosis had undergone sanatorium treatment fourteen years previously and since been employed in the industry for nine years with an exposure of 25 million particle-years per cubic foot without there being any evidence of re-activation of the disease.

In eight of the eleven cases of bronchitis exposure to dust had amounted to only 5 million particle-years per cubic foot or less. In the remaining three cases, exposures were 26, 17 and 14 million particle-years per cubic foot respectively, and in these cases it is possible that dust exposure may have been an aggravating factor in their disease. The undesirability of persons with evidence of respiratory disease being employed in the dusty sections of this industry are obvious and the finding of evidence of past or present respiratory disease in 22 cases out of the 175 cases surveyed emphasises the desirability in this industry of pre-employment medical examination, including chest x-ray.

CONCLUSIONS

The results of this survey confirm the experience of earlier ones in regard to the quantitative relationship between dust exposure and the onset and development of asbestosis. However, in our survey, the percentage of persons affected was less because the dust concentrations of the working environment were in general lower. Nevertheless, processes were found where the dust concentrations were high enough to lead inevitably to the development of asbestosis, provided the employees remained long enough in those particular jobs. Fortunately, these processes employ relatively few workmen, but it is recommended that steps be taken to give these men greater protection against dust.

There are three main methods of dust suppression. Firstly, certain dusty hand work could be mechanised, as the following shows. Prior to this survey, one firm had installed a screw conveyor to transport the blown asbestos from the fibre rooms to the tide mills; since the survey, the same firm has installed a belt conveyor to feed the dry mixed asbestos to the pulveriser. It is considered that both these measures have lessened the exposure to asbestos dust. Such improvements do not entirely eliminate the exposure to dust since there are occasions, such as during repairs or during the complete emptying of fibre rooms, when men may have to enter confined spaces and shovel the fibre for short periods. In these short periods, an approved type of dust respirator would afford adequate protection.

Secondly, the asbestos could be wetted, either with water sprays or with oil sprays. However, this renders both mechanical and pneumatic transportation of the fibres difficult, because the wetted mineral tends to adhere to the sides of ducts, conveyors, etc.

Thirdly, exhaust ventilation could be provided at certain dusty working points, with partial enclosure of these points in order to increase the effect of the exhaust. A minimum air movement of 50 feet per minute across the access opening in the enclosure is suggested. Such an air movement would tend to carry away the fibre, but this could, of course,