

PLAINTIFF'S EXHIBIT

CAP-1677



ASBESTOS INFORMATION ASSOCIATION

1745 Jefferson Davis Highway, Crystal Square 4, Suite 509
Arlington, Virginia 22202 • (703) 979-1150

Info:
to Ted
J. J. T.
J. J. T.
J. J. T.
J. J. T.

August 12, 1982

Memorandum For:

Messrs. Aldridge, Turners Bldg. Products
Beasley, CAPCO
Betts, Atlas Bldg. Materials
Dougherty, CertainTeed
Reis, J-M
Schwarz, Supradur
vonDohlen, IBP

Subject:

Forwarding of paper on Health Risks
in Chrysotile Asbestos Manufacturing
Processes

The enclosed paper has been received from the Asbestos International Association and is sent along for information.

Please note that a brief on the paper, prepared by Dr. Kevin Browne, is also attached.

Bob

B. J. Pigg
Executive Director

BJP/dh

Enclosures

cc: Hans Weill, M.D.

CAPCO JEN 0012752

ASBESTOS INTERNATIONAL ASSOCIATION

68 GLOUCESTER PLACE, LONDON W1H 3HL

MEMORANDUM

TO: Executive Committee Members

AIA/7/1/1/PROD

FROM: Director General

AIA/20/1/16/HAS

2 August 1982

Subject: Health Risks in Chrysotile Asbestos Manufacturing Processes

In view of the interest shown in a recent television film about Turner and Newall's U.K. asbestos factories, the attached which concerns a Turner and Newall factory is forwarded for information. Also enclosed are comments (paper H P Thomas et. al.) by Dr Kevin Browne, Medical Adviser to Cape Industries.


DIRECTOR GENERAL

cc BCAP Members
MAP Members

bcc Mr M R Austin
Mr R Dorner
Brig. D B Kapoor
Mr D Kelleher
Mr B Pigg
Mr J C Rodrigues
Mr R Schwarz
Mr A Sharon
Mr M Tanabe
Mr B B Taylor

CAPCO JEN 0012753

Further follow-up study of workers from an asbestos cement factory

H F THOMAS, I T BENJAMIN, P C ELWOOD, AND P M SWEETNAM

From the Medical Research Council Epidemiology Unit, Cardiff CF2 3AS, UK

ABSTRACT A further follow-up traced 1970 workers employed at an asbestos cement factory for at least six months between 1936 and 1977. At the beginning of this period some crocidolite was used in the factory but by the end of 1936 chrysotile had become the only type of asbestos in use. Only 378 women were employed during the period concerned, and of the 30 who had died, none had a cause of death that is generally associated with exposure to asbestos. The mortality experience of the men was examined separately for all workers, all workers alive after 15 or more years after first exposure, and a smaller group of workers who were employed in 1935-6 and may have been exposed to crocidolite. In none of the three groups was there an appreciably raised standardised mortality ratio (SMR) for the causes of death investigated. These were: all causes, all neoplasms; cancers of the lung and pleura, and cancers of the gastrointestinal tract. An excess of lung cancers noted in the first follow-up study in 1964 was not found in this study. Two pleural mesotheliomas were identified but in both cases the men had worked at the factory before 1936 and therefore had been exposed to crocidolite. No cancers of the larynx were found.

In 1964 members of this unit reported on the mortality experience of 1261 workers (1024 men, 237 women) who had worked in an asbestos cement factory near Cardiff for six months or longer between 1936 and 1962.¹ Their findings suggested that white asbestos (chrysotile) may not be a serious hazard so far as mesothelioma or abdominal tumours are concerned, though there was some evidence of an excess of deaths from carcinoma of the lung and bronchus. Since that study several other surveys have been reported (reviewed by Acheson and Gardner²), and at present the main human evidence is that deaths due to mesothelioma after exposure to chrysotile alone are rare, while for lung cancer there is a raised relative risk. In studies of Canadian chrysotile miners³ this was of the order of 1.2. It has also been suggested that cancers of the gastrointestinal tract⁴ and larynx⁵ are related to exposure to asbestos, but the evidence is inconclusive and particularly so for subjects who have worked only with chrysotile asbestos.

Crocidolite asbestos was used between 1932 and 1935 at the factory on which this study is based. The factory changed ownership in May 1935, and

from around that time until the closure of the factory in 1980 only chrysotile from South Africa and Canada was used, mainly for the manufacture of sheeting and associated fittings for the building industry.

Materials and methods

The factory roll used in the 1964 study was updated by the personnel officer at the factory. In all, 1592 men and 378 women were recorded as having worked at the plant for six months or more from 1936 to 1977 inclusive. Information on the vital status of subjects (as at 31 December 1977) was obtained through contacts at the factory, by home visiting, and to a smaller extent through official agencies.

Causes of deaths were coded according to the *International Classification of Diseases (ICD) Eighth Revision (1965)*. The statistical analysis used a person years at risk (MYCL)⁶ programme with England and Wales age-specific death rates for quinquennial periods from 1911 to 1970.⁷ As the rates in the latter publication extended only up to the end of 1970 the 1966-70 rates were also used for the period 1971-7. Analysis was undertaken of mortality due to all causes, all neoplasms (ICD 140-239), cancers of the lung and pleura (ICD 162-163), and gastrointestinal cancers (ICD 151-154). Separate analyses were under-

Received 2 October 1981

Accepted 29 October 1981

Table 1 Total mortality (all causes) by time since entry into study

Time since entry (years)	Expected deaths	Observed deaths	Standardized mortality ratio (SMR)
< 4	29.3	16	55
5-9	33.6	30	89
10-14	37.2	44	118
≥ 15	243.2	261	107
Total	343.3	331	102

taken for all workers and for those who were alive 15 years after first exposure. The latter analysis was used in the 1964 report¹ on the grounds that such a period was thought necessary before it was justifiable to attribute any death, wholly or in part, to exposure to asbestos. As the transition from the use of crocidolite to chrysotile may have taken several months a separate analysis was performed on workers who had worked in the factory in 1935 and 1936.

Results

The follow-up was successful in tracing 1540 (97%) men and 358 (95%) women. Only 30 female deaths were identified. None of these related to the specific cancers with which the analyses were concerned. In view of the small numbers of women workers and recorded deaths detailed analysis was confined to men.

Table 1 shows the total (all cause) mortality by time (in years) since entry into the study—that is, first employment in the factory. Table 2 shows the mortality due to all causes, all neoplasms, and cancers of the lung, pleura, and gastrointestinal tract for men in the three groups previously described—all men (1540), all men alive 15 or more years after first exposure (1147), and men who were employed in 1935 (249). In this study 18 war deaths (ICD 990-

999) were found and all but one occurred overseas. As such deaths would not have been included in the calculation of the expected numbers of deaths, their exclusion in the calculation of the workers' SMR gives a more realistic figure. The deaths due to cancers of the lung and pleura comprised 27 cancers of the bronchus and lung (ICD 162.1), one adenocarcinoma (ICD 163.9), and two mesotheliomas of the pleura (ICD 163.0). No deaths due to cancer of the larynx were recorded. Asbestosis was not mentioned on any of the male or female death certificates examined.

Discussion

The overall SMR of the men is 102 (excluding war deaths). Table 1 gives some evidence of "the healthy workers" effect² with a rise in SMR from 55 to 118 as time since first exposure increases, although after 15 or more years the effect seems to disappear. South-east Wales, where the factory was located, has a high all-cause mortality (SMR 111),³ and the overall SMR of the workers exposed to asbestos compares favourably with this. The all-cause mortality (table 2) is not raised for men who were alive 15 or more years after first exposure (SMR 107) nor for the smaller group who were employed in 1935-6 (SMR 94).

The picture for the cancers is broadly similar (table 2). For all neoplasms the SMRs are similar for the total population (92), men alive 15 or more years after first exposure (95), and men employed in 1935-6 (103). It is unlikely that the slight gradient in SMR is of any significance.

Cancers of the lung and pleura show the same SMR (93) for all men and for those alive 15 or more years after first exposure. These SMRs do not suggest an increased risk of lung cancer in workers in the asbestos cement industry. No information is available on the smoking habits of the men, but workers

Table 2 Mortality by all causes, all neoplasms, and selected cancers for the total male population, men alive 15 years or longer after first exposure, and for men employed in 1935-6

Cause of death (ICD code)	Study group	Expected deaths	Observed deaths	Standardized mortality ratio (SMR)
All causes	Total population	343.3	331	102
	Alive ≥ 15 years after first exposure	243.2	261	107
	Employed in 1935-6	88.3	83	94
All neoplasms (140-239)	Total population	80.7	74	92
	Alive ≥ 15 years after first exposure	61.0	58	95
	Employed in 1935-6	21.5	22	103
Cancers of lung and pleura (162, 163)	Total population	33.0	30	93
	Alive ≥ 15 years after first exposure	23.8	24	93
	Employed in 1935-6	9.2	7	76
Cancers of gastrointestinal tract (151-154)	Total population	19.6	18	92
	Alive ≥ 15 years after first exposure	14.1	14	99
	Employed in 1935-6	5.0	6	120

in dusty industry generally smoke more than the general population¹⁸ and therefore a slightly raised SMR could have been expected. In the 1964 study¹ a raised SMR (23.5) for cancers of the lung, bronchus, and pleura was reported in men alive 15 years or more after first exposure, but the numbers were small (seven deaths observed v 3.0 expected). The cut-off date for that study was the last day of 1962. Our recent follow-up identified two other deaths from lung cancer (in 1953 and 1959) that were untraced in the earlier study. In the follow-up period since 1962, however, these earlier apparent excesses are balanced by later deficits. Only one such death was recorded during 1963-5. Thus by the end of 1965 the SMR was 152 (10 observed; 6.6 expected). In the 12 years after 1965 14 deaths were identified, five fewer than the 19.2 expected. Thus by the end of 1977 the SMR was 93 (24 observed; 25.8 expected).

As a group the 27 men who died of lung cancer (ICD 162) had a mean age (at first exposure) of 39 years (SD 11); 15 worked at the factory for under two years and only two worked there for more than 10 years. The mean period from first exposure to death was 22 years (SD 9.5).

Both workers who died of mesothelioma of the pleura were employed at the factory during the period 1932-5 when crocidolite was used. One worked as a beater attendant and process worker from 1932 until early 1936 but was then transferred to the drawing office and eventually left the company in 1953. He died in 1962. The other man had four short periods of employment at the factory between 1932 and 1948 but rejoined in 1949 and stayed until his death in 1974. He was a process worker. Postmortem analysis of his lungs showed a high chrysotile content but also a crocidolite value that was higher than would be expected if there was no occupational exposure (G Berry, personal communication). In view of the general evidence that exposure to crocidolite poses a greater risk of contracting lung cancer than exposure to chrysotile² it is perhaps surprising that the SMR for workers employed in 1935-6 is only 76. This figure, however, is based only on seven deaths (9.2 expected), and possibly only a small proportion of those first employed in 1936 used crocidolite.

Deaths due to cancer of the gastrointestinal tract were not increased in the total male population (SMR 92) or in those men alive 15 or more years after first exposure (SMR 99). The raised SMR (120) for men employed in 1935 and 1936 is due to one extra death (6 observed, 5.0 expected). These figures do not suggest that the chrysotile asbestos cement workers are at an excess risk of gastrointestinal cancer, a finding consistent with the results of studies of chrysotile miners and millers³ and chrysotile asbestos textile workers.¹¹

Thus the general results of this mortality survey suggest that the population of the chrysotile asbestos cement factory studied are not at any excess risk in terms of total mortality, all cancer mortality, cancers of the lung and bronchus, or gastrointestinal cancers. The two deaths due to pleural mesothelioma could not be attributed to exposure solely to chrysotile asbestos. They were probably, however, related to crocidolite exposure at the factory during 1932-5.

There are several possible reasons why no increase in general or specific mortality is apparent in this population of chrysotile asbestos cement workers. It is generally recognised that the asbestiform chrysotile does not penetrate the lungs to the same extent as amphiboles—for example, crocidolite and amosite¹² and chrysotile has only rarely been incriminated as the only type of exposure to asbestos in cases of mesothelioma.² Indeed, postmortem studies have found that the number of chrysotile fibres in cases of mesothelioma has not differed from controls.¹³ Asbestos cement dust is also, in many ways, more akin to cement than asbestos,¹⁴ and therefore the airborne dust in the factory (apart from in the early fibre discharging and mixing stages) may not pose such a hazard. Dust levels in asbestos cement factories are generally lower than in other asbestos handling industries,¹⁵ mainly because the manufacture of sheeting and accessories is largely a wet process, involving only 10-15% asbestos (by weight).

No regular measurements of dust were made until 1968. Estimates of levels before then range from 0.1 fibre/ml at the cement machine to 20+ fibre/ml on the beater floor and at hard waste grinding. The past 15 years has seen a pronounced improvement in the dust levels. Regular dust counts at the factory from 1972 until its closure in 1980 were consistently below 2 fibres/ml and the vast majority were below 1 fibre/ml (J Mattison: personal communication). On the recommendation of the Advisory Committee on Asbestos¹⁶ the control limit for chrysotile asbestos has been reduced to 1 fibre/ml and the asbestos cement industry, which is the main user of chrysotile asbestos in the United Kingdom has accepted this limit to protect its 6000 employees. The control limit is intended to protect workers who have a long exposure (50 years). Over half of the population that we have studied was employed for under two years and three-quarters worked in the factory for under four years. Only a small number are likely to have had an exposure in "fibre years" that would be considered high; 46 men worked at the factory for more than 30 years and only eight for more than 40.

Thus while this study gives reassuring findings on the long-term mortality experience of an asbestos cement factory population, the high labour turnover

does not enable conclusions to be drawn on the hazards of sustained long-term exposure to chrysotile asbestos.

We thank the many people who assisted us in this inquiry including the workers and management of TAC Rhose, and especially Mr Graham Fraser, the former personnel manager. A grant of £1000 was received from TAC towards the cost of this study.

References

- ¹ Elwood PC, Cochrane AL, Benjamin IT, Seys-Prosser D. A follow-up study of workers from an asbestos factory. *Br J Ind Med* 1964;21:304-7.
- ² Acheson ED, Gardner MJ. The ill-effects of asbestos on health. In: *Asbestos*. Vol 2. London: HMSO, 1979. Final report of the advisory committee.
- ³ McDonald JC, Liddell FDK, Gibbs GW, Eysen GE, McDonald AD. Dust exposure and mortality in chrysotile mining, 1910-75. *Br J Ind Med* 1980;37:11-24.
- ⁴ Enterline P, De Couffe P, Henderson V. Mortality in relation to occupational exposure in the asbestos industry. *JOM* 1972;14:897-903.
- ⁵ Stell PM, McGill T. Asbestos and laryngeal cancer. *Lancet* 1973;iii:416-7.
- ⁶ Hill ID. Computing man years at risk. *Br J Prev Soc Med* 1972;26:132-4.
- ⁷ Institute of Cancer Research. *Serial mortality tables. Neoplastic diseases. Vol 1. England and Wales, 1911-70*. London: Division of Epidemiology, ICR, 1976.
- ⁸ Fox AJ, Collier PF. Low mortality rates in industrial cohort studies due to selection for work and survival in the industry. *Br J Prev Soc Med* 1976;30:225-30.
- ⁹ Chilvers C. Regional mortality 1969-73. *Population Trends* 1978;11:16-20.
- ¹⁰ Office of Population Censuses and Surveys. *Occupational mortality 1970-2. Series DS No 1*. London: HMSO, 1978: 148.
- ¹¹ Peto J, Doll R, Howard S, Kilen LJ, Lewinsohn HC. A mortality study among workers in an English asbestos factory. *Br J Ind Med* 1977;34:169-73.
- ¹² Wagner JC, Berry G, Skidmore JW, Timbrell V. The effect of the inhalation of asbestos in rats. *Brit J Cancer* 1974; 29:252-69.
- ¹³ Jones JSP, Pooley FD, Clark NJ, et al. The pathology and mineral content of lungs in cases of mesothelioma in the United Kingdom in 1976. In: *Biological effects of mineral fibres*. Lyon: WHO IARC/INSERM MRC, 1979. (Abstract.)
- ¹⁴ Deruyttere A, Baeten J, Helsen J. Characterisation and properties of asbestos cement. In: *Biological effects of mineral fibres*. Lyon: WHO IARC/INSERM MRC, 1979. (Abstract.)
- ¹⁵ Advisory Committee on Asbestos. Health and Safety Commission. *Final report*. Vol 1. London: HMSO, 1979.