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SLATER & GORDON

BARRISTERS & SOLICITORS

14 VENTNOR AVENUE
WEST PERTH 6005
P.O. BOX 1094
WEST PERTH 6005
AUSDOC · D.X. 60724
TELEPHONE · (09) 321 6745
FAX: (09) 321 4840

OUR REF: LF:SF:1707/C122(34)

YOUR REF.

DATE · 14 June 1993

Mr B. Castleman
1722 Linden Avenue
Baltimore, Maryland
USA

Dear Sir

HERMAN ABRAHAM VAN EMDEN v. JAMES HARDIE & BUNNINGS

Since last corresponding with you the solicitors for James Hardie have furnished a further substance of expert evidence being substance of Mr G. Major. A copy of this substance is enclosed for your perusal and comment if appropriate.

On last enquiry our Court was indicating that the earliest possible trial dates in this matter would be October 1993. In our view this is too long a wait and we are currently reviewing what other steps, if any, we can take to secure an earlier hearing date.

Yours faithfully

SLATER & GORDON

enc.

SUBSTANCE OF EXPERT EVIDENCE
OF GERSH MAJOR
ON BEHALF OF THE FIRST DEFENDANT

1. Based on the body of knowledge during 1972-1976 ('the relevant period'), no asbestos related disease was known or foreseeable amongst carpenters who worked with asbestos-cement. I am asked to assume that the relevant products did not contain crocidolite after 1968. The issue of amosite and mesothelioma was unclear and uncertain during the relevant period and, even today, mesothelioma is believed to be a very rare disease amongst persons who worked with chrysotile uncontaminated with the commercial amphibole forms of asbestos. Commercial chrysotile contaminated with traces of tremolite may have caused mesothelioma amongst some miners and millers and some persons who made asbestos products in asbestos textile factories but the exposure necessary to induce it was so much higher than that experienced by carpenters who used asbestos-cement that it can be disregarded as the cause of mesothelioma amongst this working group.

What data?

2. **Latency Period**

We are fortunate that we have Australian data on which to discuss the latent period or time-lag between the first exposure to asbestos and the diagnosis of mesothelioma. Emeritus Professor David Ferguson from the University of Sydney (and later the National Occupational Health and Safety Commission, or Worksafe Australia) and his colleagues described 'the epidemiology of mesothelioma in Australia and the role of occupational and environmental exposure to asbestos' (DA Ferguson et al in Medical J of Aust Aug 17 1986 pp166-172 p.166). The study included the work and environmental history of 858 cases of mesothelioma - I was one of the two occupational hygienists who reviewed

all the histories to determine the nature of exposure to asbestos and assessed the probability that the case was associated with asbestos exposure.

The analysis of the time-lags for the 456 cases which were occupationally exposed is tabulated on page 169 of the Ferguson paper. The time-lag was 30 years or longer in 73% of the subjects who were occupationally exposed to airborne asbestos and in 41% of cases the time-lag was 40 years or longer. Only 5% fall within the tabulated range 10-19 years. The latent period for Mr Van Emden effectively places him in the 5% group - an unusually low time-lag.

Amongst sufferers of mesothelioma - 'periods of 30, 40, or even 50 years are common' (Richard Doll and Julian Peto. ASBESTOS. Effects on health of exposure to asbestos. U.K. HSW, HMSO, 1985 p.3). The Australian study is in accordance with similar investigations in other countries as is shown in a review by JC McDonald (JC McDonald and AD McDonald, Epidemiology of Mesothelioma, a chapter in a book edited by Douglas Liddell and Klara Miller CRC Press 1990 at p.51).

Doll and Peto in ASBESTOS (Supra) write that:

> 'Mesotheliomas of the pleura or peritoneum are normally so rare, other than after occupational or other unusual exposure to asbestos, that any case that occurs after well attested and substantial asbestos exposure is commonly accepted as due to that exposure, subject only to the qualification that the time since the exposure occurred must be long enough to permit the disease to have been produced' (p.3).
>
> 'Contrary to the common belief several studies show that brief exposures produce relatively little risk' (p.51).

3. **Fibre Type and Mesothelioma**:

(i) crocidolite;

(ii) amosite;

(iii) chysotile;

(iv) chrysotile with tremolite.

The Defendants followed the normal practices of the time for the use of asbestos-cement which did not contain crocidolite. If it is held that, during the relevant period, some experts in occupational health suspected that forms of asbestos other than crocidolite were associated with mesothelioma amongst insulation workers, I reply that asbestos cement had been used in Australia and overseas for more than fifty years without, to my knowledge, any asbestos-related reported illness amongst carpenters so that the likelihood and seriousness of that risk would not have induced a reasonable employer to take those precautions believed necessary by some to prevent mesothelioma in completely different occupations. Nor would it have induced a reasonable manufacturer of asbestos-cement products to cease production of a material highly valued by the community.

See T+N 1945

4. Having regard to the fact that whole volumes have been written on the subject over the past twenty years, it is safe to say that during the relevant period exposure to airborne crocidolite was the only commercial form of asbestos known to be associated with mesothelioma in low doses. This is particularly true of mesothelioma of the pleura.

5. In 1968 the British Occupational Hygiene Society was to write:

> 'A risk of mesothelioma of the pleura and peritoneum exists in connection with the inhalation of crocidolite dust in particular'.

See IARC 1973

(Hygiene Standards for Chrysotile Asbestos Dust; British Occupational Hygiene Society, 1968).

The Society clearly did not view the association of chrysotile and amosite with mesothelioma as a real possibility and had not changed its views by 1973 when it reviewed its 1968 document (Hygiene Standards for Chrysotile Asbestos Dust; British Occupational Hygiene Society, 1973).

6. In 1991, on the 30th anniversary of the publication of his paper 'Diffuse pleural mesotheliomas and asbestos exposure in the north west Cape Province', JC Wagner wrote a paper entitled 'The Discovery of the Association between Blue Asbestos (my emphasis) and Mesotheliomas and the Aftermath' (JC Wagner in Brit J Industr Med 1991; 48:399-403). The first epidemiological publications concerning mesothelioma and asbestos, as distinct from scattered reports of individual cases from individual physicians in medical journals, were directed towards the association with crocidolite.

7. During the relevant period there were conflicts in the evidence on the health effects of mineral fibres. Peter Elmes wrote a chapter called Conflicts in the Evidence on the Health Effects of Mineral Fibres in a book edited by Douglas Liddell, Mineral Fibres and Health (Supra). Concerning the prestigious 1964 New York conference on the Biological Effects of Asbestos, Elmes wrote:

> 'Medical scientists in the field (my emphasis) accepted the relationship between asbestos exposure and mesothelioma, [but] it was some years before this was the generally accepted view.' (p.325, first line).

and also wrote concerning the conference:

> 'Second, the importance of fibre type was not recognised. In their original study, Wagner and colleagues ..... no cases of mesothelioma were reported from the areas of South Africa where chrysotile and amosite were mined and milled, although conditions were similar' (p. 325).

That is to say that, unlike crocidolite, no cases of mesothelioma were reported following exposure to low levels of chrysotile and amosite.

8. Elmes elucidates the erroneous belief that chrysotile is as dangerous as the amphiboles by referring to asbestos toxicity studies on animals which he demonstrates were flawed. It is interesting to note that in 1985 Wagner notes the probability that he used contaminated fibre in his 1969-74 animal experiments and that this has since been confirmed (J.C. Wagner in CANCER May 15 1986 p1905).

9. Similar views were expressed by another major epidemiologist, JC McDonald in 1980 (JC McDonald in International Agency for Research on Cancer, Scientific Publications No. 30, 1980, pp587-591) who wrote concerning 'all cases of malignant mesothelioma reported to the end of 1975':

> 'Exposure to airborne crocidolite in man had clearly proved far more hazardous than that to chrysotile, although, in animal experiments (my emphasis), the carcinogenic potential of all types of asbestos and other mineral fibres seemed similar'.

and further:

> "The status of amosite was uncertain; the substantial incidence of mesothelioma in amosite factory workers conflicted with the apparent

> infrequency among amosite miners. From 1930 on, amosite had been used for insulation materials in the USA; and from about 1950 it had become the major constituent. Possibly (my emphasis) this explained the high incidence among American insulators.'

The opening sentence in the last quotation is vitally important to an argument on foreseeability of mesothelioma amongst carpenters who used asbestos cement which contained amosite; the 'industry effect' or Amosite Anomaly discussed by Elmes (p332 of Peter Elmes, Conflicts in the Evidence on the health Effects of Mineral Fibres, a chapter in a book edited by Douglas Liddell). Even today there is no evidence that amosite causes mesothelioma in those carpenters other than the alleged disease of Van Emden.

10. At the 1964 New York conference Dr Irving J Selikoff reported on mesothelioma amongst insulation workers (IJ Selikoff et al in Annals of NY Acad of Sci Vol 132 Art 1 Dec 31 1965 p139). These persons applied (and removed) asbestos-containing thermal insulation to hot pipes, boilers, turbines etc and much of the insulation contained amosite. However the epidemiological methodology was criticised by some, principally because the information gained did not point specifically to amosite as the agent causing the disease. It was not certain from the report if amosite actually caused mesothelioma. Selikoff noted that (p.142 first line):

> '[chrysotile] has been confirmed by analyses of magnesia block obtained during repair work. In later specimens so obtained, crocidolite (my emphasis) has also been found.'

11. It should be noted that the New York insulators were highly exposed to airborne asbestos; the title of the paper concerns asbestosis and the (one) mesothelioma

case also suffered from asbestosis. Selikoff wrote (IJ Selikoff et al (Supra) at p151):

> '... it appears evident that asbestosis is an important risk among insulation workers exposed to asbestos. .... This risk includes lung cancer and mesothelioma associated with (my emphasis) the pulmonary asbestosis.'
>
> and further (p.152)
>
> 'We conclude that asbestosis and its complications (my emphasis) are significant hazards among insulation workers in the United States at this time.'

12. There is little doubt that there were strong suspicions amongst persons working in the research field of asbestos and health that exposure to airborne amosite under some conditions was associated with mesothelioma during the relevant period but no suggestions that, like crocidolite, low levels of fibres caused the disease.

13. In 1972, the International Agency for Research on Cancer organised a meeting of experts on the biological effects of asbestos (Proceedings published in 1973 - I.A.R.C. Scientific Publications 8, pp10-17, 1973). Strong words concerning amosite and causation of mesothelioma are not used:

> 'There is evidence that all types of commercial asbestos except anthophyllite may (my emphasis) be responsible. Evidence for an important difference in risk in different occupations (my emphasis) and with the type of asbestos has increased. The risk is greatest with crocidolite, less with amosite, and apparently less with chrysotile.'

14. The last sentence in the previous quotation was used by others in later years but, during the relevant period, there was no evidence on which to judge, quantitatively, the meaning of 'less with amosite'. The evidence followed, to some extent, in later years culminating [late] in 1992 with the publication of Sluis-Cremer et al in Brit J Indstr Med 1992 which, on p.574 concludes:

> 'Of particular note is the comparatively low risk of mesothelioma in amosite workers. There can now be no question that crocidolite is far more dangerous than amosite at least insofar as mesothelioma is concerned.'

and (on p.573)

> 'Although no precision can be claimed for them, the ratios above show clearly that crocidolite has a toxicity for mesothelioma about an order of magnitude [i.e. ten times, my insertion] higher than that for amosite. This is much more convincing evidence than any available previously, which has had to be culled from comparisons across a very few studies of exposures to a single fibre type. Even the evidence of proportional mortality is sufficient, however, for confidence that chrysotile is much less toxic for mesothelioma even than amosite.'

15. The words 'difference in risk in different occupations' are important. In my opinion the real issue is not 'does amosite cause mesothelioma' but 'does amosite cause mesothelioma in carpenters who use asbestos-cement which contains it?' During the relevant period this was not known and remains unknown today.

16. During the relevant period, the view was:

> 'There is evidence of an association of mesothelial tumours with air pollution in the

> neighbourhood of crocidolite mines and of factories using mixtures of asbestos fibre types. ... There is evidence of no excess risk of mesotheliomas from asbestos air pollution which has existed in the neighbourhood of chrysotile and amosite mines' (I.A.R.C. Scientific Publications 8, 1973, at p12).

17. There is also more recent evidence concerning amosite reported by McDonald (JC McDonald and AD McDonald, Epidemiology of Mesothelioma, a chapter in a book edited by Douglas Liddell and Klara Miller CRC Press 1990 p.162):

> 'Also negative was an analysis of mortality from mesothelioma in persons residing within half mile of an insulation-products factory in Paterson, NJ, which used mainly amosite asbestos. Many cases of mesothelioma had occurred in the employees of this plant, and amosite dust was still found in the attics of houses in the neighbourhood'.

This, of course, is in marked contrast to experience with crocidolite and demonstrates two facts of importance, viz:

> Crocidolite is more harmful than amosite
>
> and
>
> Low levels of amosite have not been shown to cause mesothelioma.

18. The 1972 I.A.R.C. Conference (I.A.R.C. Scientific Publications 8, pp10-17, 1973) recommended as a high priority research project 'Assessment of excess cancer risks following exposure to only one type of fibre' and continued 'Crocidolite: Further studies are required in occupational groups exposed only to crocidolite or amosite or chrysotile in manufacturing and application parts of the industry to establish more clearly

differences in risks due to different fibres.' (p.14). The experts went to work assiduously; it is sufficient to say that extensive scientific investigations into asbestos and health commenced during our relevant period but were not completed until later.

19. In 1973 the British Occupational Hygiene Society published 'Hygiene Standards for Airborne Amosite Asbestos Dust' (Hygiene Standards for Airborne Amosite Asbestos Dust. B.O.H.S. 1973) which included:

> 'The sub-committee believes it has insufficient knowledge of the relationship between airborne amosite dust exposure and the risk of asbestosis (my emphasis) to permit an accurate statement of the degree of protection afforded by a specified hygiene standard. Nevertheless, on the basis of comparisons between the effects of amosite and chrysotile dust on men and animals it is recommended that the standards for amosite should be no less stringent than those for chrysotile' (p.1)

and

> 'The hygiene standards are related to the risk of developing asbestosis (my emphasis). A cancer risk also exists, but the quantitative relationship between the intensity of exposure and the risk of cancer is less well defined. Evidence at present available indicates that if a standard is maintained such that the risk of asbestosis (my emphasis) is small, the risk of cancer of the lung attributable to asbestos will be smaller still (Knox et al, 1968: Elmes and Simpson, 1971). The position in relation to mesotheliomas of the pleura and peritoneum is still uncertain' (p.3).

20. As the Committee notes, information on the health effects of amosite was 'very scanty' during our relevant period and, in my view, mesothelioma amongst carpenters who used asbestos-cement which contained some amosite was unforeseeable.

21. In May 1969 the Australian National Health and Medical Research Council followed the B.O.H.S. and recommended occupational exposure limits of 4 f/cc (or 4 fibres/millilitre - 4 fpmL) for both chrysotile and amosite (Hygiene Standards for Contaminants of the Air of the Workplace; NH&MRC 15-16 May 1969). It made no recommendations for crocidolite which was no longer used in Australian industry and there was no scientific basis on which to recommend any level for this type of fibre. The NH&MRC Recommendation continued unchanged throughout our relevant period.

22. In summary, mesothelioma in a carpenter who used asbestos cement which contained some amosite was not foreseeable -'the position in relation to amosite was uncertain'. Your client ought not to have known that low levels of amosite were associated with the induction of mesothelioma during the relevant period.

23. A reasonable inference following from the B.O.H.S. 1969 Hygiene Standard (Hygiene Standards for Chrysotile Asbestos Dust; British Occupational Hygiene Society, 1968 & 1973) is that it was the Society's view that chrysotile was not implicated in the occurrence of mesothelioma in asbestos workers. Further, the British Government, in the 1969 New Asbestos Regulations, placed severe restrictions on work with crocidolite (Reg 6), the only form of asbestos known at the time to be associated with the induction of mesothelioma at low levels of exposure. Further, in 1970, the UK government in a guidance note on how HM Inspectors of Factories would interpret the expression 'dust consisting of or containing asbestos to such an extent as is liable to cause danger to the health of employed persons' wrote

(UK Dept of Employment and Productivity, Technical Data Note 13. HMSO 1970):

> '..... crocidolite because the concentration of this mineral, that is believed to be liable to be dangerous to health, is very small indeed.'

Clearly the UK authorities did not believe in 1970 that 'the concentration of chrysotile (or amosite) liable to be dangerous to health, is very small indeed' [and hence low levels were not a risk for mesothelioma].

24. In my opinion, during our relevant period, a prudent manufacturer of asbestos cement would not have foreseen dangers of mesothelioma arising from white asbestos or white asbestos with tremolite asbestos, principally because of the words B.O.H.S. 'chrysotile standard' (Hygiene Standards for Chrysotile Asbestos Dust; British Occupational Hygiene Society, 1968 & 1973). In the paragraph beneath the headline CANCER on p.53:

> 'The primary danger of inhaling asbestos dust is asbestosis. It is generally recognised that there is also significant risk of lung cancer associated with asbestosis. A risk of mesothelioma of the pleura and peritoneum exists in connection with the inhalation of crocidolite dust in particular.'

25. Any suggestion that Mr Van Emden's mesothelioma was caused by white asbestos contaminated with tremolite can be countered by reference to JC McDonald and AD McDonald, Epidemiology of Mesothelioma, (Supra) at p.161. Quebec chrysotile is often contaminated with 1.5-2% tremolite, yet:

> 'Quebec chrysotile miners had no cause under 8 years (of exposure) and British textile workers had only one case under 10 years.'

There were tens of thousands of Quebec chrysotile miners and British textile workers many of whom were exposed to high levels of airborne asbestos. Mr Van Emden worked for Bunnings for only 4 years so that the probability that his mesothelioma arose out of his exposure to (low levels of) white asbestos contaminated with tremolite must be vanishingly small. It is worth emphasising that, unlike textile workers, carpenters work with asbestos-cement principally in the open air.

26. Chrysotile is unlikely to have been the cause of Mr Van Emden's mesothelioma because it rarely, if ever, causes mesothelioma and experience with groups of workmen in two different industries suggests that his period of exposure was too short to induce the disease.
Mesothelioma was not foreseeable in a carpenter working with asbestos-cement which did not contain crocidolite during our relevant period.

In my opinion had a prudent Australian asbestos-cement manufacturer wanted to know about the risks for asbestosis amongst carpenters using its products during our relevant period it would have inquired of departments of health, State and Commonwealth. In my opinion, they would have been told that no such risks existed. Further, in my view the opinions of these regulating authorities would have been:

> Asbestos cement ('fibro') is a hard surface material, in which asbestos fibres are reasonably bonded by cement. Work with this material can be carried out safely with hand saws and the other hand tools commonly used by carpenters and also with power drills (NH&MRC Code for the handling of Asbestos by Small Users, Canberra June 1978).

In relation to power saws, the response probably would have been:

7/100 f/cc!

'It is not expected that carpenters would suffer asbestosis following the use of power saws when working with asbestos cement products'.

27. The experience of the Victorian Division of Industrial Hygiene is outlined by DLG Thomas in the Medical Journal of Australia in January 1957 (DL Gordon Thomas in M.J.A. 19 January, 1956 p75):

> '[Asbestosis] - the following occupations are involved: handling the substance in its raw state; grinding the substance prior to its use in some process; mixing with diatomaceous earth or kaolin to from lagging materials; sawing, cutting and finishing [in factories making them] any product containing asbestos for example, brake linings, asbestos sheeting and various insulating materials; tearing down old lagging; .... spraying asbestos on walls and ceilings as an insulator.'

! factories not in original

in factories making lagging?!!

There was no mention of carpenters in this paper nor is there any mention of the use of asbestos-cement in the Proclamation (attached to Thomas paper in DL Gordon Thomas in M.J.A. 19 January 1956) which arose out of it.

28. To the best of my knowledge and belief there was no publication in the English language before and during our relevant period which included measurements of the airborne fibre level to which carpenters using asbestos cement were exposed. It had been possible to measure it after about 1969 (although not in some states) but my inquiries convince me that no Australian regulating authority had done so before the end of our relevant period nor had any measurements been made on building sites by government instrumentalities before about 1985.

29. **Asbestosis and Lung Cancer**

In my opinion, your client ought not to have known of any risks for asbestosis arising from white asbestos or white asbestos with tremolite asbestos or white asbestos with a small amount of brown asbestos in asbestos-cement products during the relevant period.

30. During the relevant period 'it has been shown that there is an increased incidence of [cancer of bronchus or lung] in people who already have asbestosis. It is not yet certain if asbestos can contribute to cancer of the lung when asbestosis is not already present' (U.K. H.S.W. An interim statement by the Advisory Committee on Asbestos, HMSO 1977). Further 'Evidence at present available indicates that if a standard is maintained such that the risk of asbestosis is small, the risk of cancer of the lung attributable to asbestos will be smaller still' (NH&MRC Report on the Health Hazards of Asbestos, Canberra. A.G.P.S. 1982 p.5).

Contradicted by IARC '77

31. **Conclusion**

In summary, a reasonable manufacturer of asbestos-cement ought not to have known during the relevant period of any dangers of chrysotile, amosite or chrysotile contaminated with tremolite amongst carpenters who worked with its products.

32. Except for the Victoria 1945 Regulations and the 1956 Proclamation and the rather anachronistic 1970 Queensland Asbestos Rule under the Factories and Shops Act, there was no mention of asbestos in Australian factories or health legislation. The National Health & Medical Research Council drafted Model Regulations on Asbestos in 1976 which were adopted in the various states in 1977 (or even later in W.A.). In my view knowledge concerning asbestos and health was not widespread generally during the relevant period.

33. In my opinion a reasonable asbestos-cement manufacturer ought not to have known that carpenters working with its

products or in the vicinity of others working with its products would be exposed to harmful concentrations of asbestos fibres. ['Asbestos dust' contained the motes to which I have made reference; during the relevant period only 'asbestos fibres' had any relevance in discussions of asbestos and health. The statutory occupational exposure limits for chrysotile and amosite were expressed in fibres per millilitre.]

34. In my opinion, during the relevant period your client ought not to have known that exposure to its products which did not contain crocidolite, particularly the exposures pleaded by the Plaintiff, would cause mesothelioma. The body of knowledge at the time was such that 'the position in relation to mesotheliomas of the pleura and peritoneum was uncertain'. (Hygiene Standards for Airborne Amosite Asbestos Dust. B.O.H.S. 1973).

35. **Plaintiff's Expert Evidence**

I will now comment on and put into context some of the publications and comments made in the substances of evidence of the Plaintiff's experts.

As to knowledge of asbestos-related disease 1924-64:

> '..... With a few important exceptions, the evidence [in the early 1960's] rested on scattered reports of small numbers of cases, and the cases themselves were sometimes selected or simply those that happened to come to the attention of the writer (Asbestos and Disease by Selikoff and Lee, published in 1978 at p.31).'

36. The Merewether & Price Report bore no relationship to the use by carpenters of asbestos-cement which contained 10-15% asbestos and who were exposed daily to less airborne asbestos in a different environment.

37. Item 34 of Professor Musk's report refers to the 1955 Article by Richard Doll. However, Musk omits the important words:

> 'Lung cancer was a specific industrial hazard of certain (my emphasis) asbestos workers' (p.86 of Doll's paper). (before 1933...)

UK factory regs never applied to insulators

There is no suggestion in Doll's paper that carpenters who used asbestos-cement were some of these 'certain asbestos workers' and at risk for lung cancer.

38. Musk's item 35 demonstrates the different incidence for disease between different groups of workmen. In the early 1970's mesothelioma had been found (or thought to have been found) amongst insulators but not amongst miners; it might equally have been absent amongst carpenters who used asbestos-cement. Musk's item 37 refers to insulation workers; insulation workers used quite a different material from asbestos-cement.

39. In items 40-45 of his report, Musk refers to the development of the history of mesothelioma and exposure to crocidolite. However, he fails to comment that his item 41 is described by the authors:

> 'This is a preliminary publication and the problem is being intensively investigated' (p.260)
>
> and also
>
> 'The pathological evidence for associating these tumours with asbestos exposure is not conclusive' (p.269).

Musk makes no mention of the absence of mesothelioma amongst amosite workers although JC Wagner (one of the authors) sought it.

40. Musk refers to the situation after 1964. However, during the relevant period, it was not accepted that small doses of asbestos could cause mesothelioma. It was accepted only that small doses of crocidolite could cause mesothelioma. It was not until after the end of the relevant period that the NH&MRC recommended different occupational exposure limits for amosite and chrysotile. The British asbestos industry did not abandon the use of amosite until after the end of the relevant period.

41. Musk refers to standards, regulations and legislation. Most of his commentary is irrelevant to the issue of mesothelioma and carpenters who used asbestos-cement. For example, the Dreesen standard was associated with the substance 'asbestos' and it measured 'asbestos particles' not the 'concentration' of dust which contained some asbestos particles. The DLG Thomas article is irrelevant to carpenters who used asbestos cement. Asbestos sheeting is not 'asbestos cement' - it probably means 'asbestos millboard' which was a soft board and contained about 85% asbestos. In 1969 the NH&MRC did not recommend 'an exposure limit of 4 fibres per cubic centimetre', it recommended that (Hygiene Standards for Contaminants of the Air of the Workplace; NH&MRC 15-16 May 1969):

> 'The long-term, average fibre concentration of the air breathed by the worker should not significantly exceed four fibres per cubic centimetre of air as measured by the membrane filter method of the British Occupational Hygiene Society or by any other method proven equivalent to this method'. [The word 'long-term' was not defined but it was taken to mean about 3 months following the B.O.H.S., Hygiene Standards for Chrysotile Asbestos Dust; British Occupational Hygiene Society, 1968 & 1973].

42. The first sentence of Dr Joseph's (first) item 23 is irrelevant - the unscheduled occupations did not include carpenters who used asbestos-cement which did not contain crocidolite. Although the cases included some carpenters they were ship's carpenters and shipwrights who had worked in poorly ventilated naval ships while insulators were working, including spraying and removing crocidolite.

43. In item 24, Joseph writes that Gordon Thomas 'drew attention to the dangers of asbestos to workers in the construction industry handling asbestos products'. However, all that is in the article concerning the construction industry are the words '... and various insulating materials; tearing down old lagging - this is a very dangerous process, even in the open air; spraying asbestos and walls and ceiling as an insulator.' That Thomas makes no mention of asbestos cement demonstrates that he did not consider these workers to be at risk for asbestosis - the only asbestos-related disease he mentions.

44. As to the last section of Joseph's report, in my opinion, few (if any) power tools used by carpenters were electrically safe when used with water and none were equipped with 'an effective exhaust system' during the relevant period. In my opinion, had carpenters working with asbestos-cement which did not contain crocidolite been at risk for mesothelioma during the relevant period, respiratory protection would not have been effective, principally because the degree of protection required was not known so that the selection of 'an effective mask' was impossible. It was not foreseeable that carpenters during the relevant period were at risk for mesothelioma and that respiratory protection was required to protect them from asbestosis and its complication, cancer of the bronchus.

45. The Plaintiff's experts refer to the Dreesen Report. However, the report bears little relevance to carpenters

who used asbestos-cement in the outdoors environment. On page 91 of the Dreesen Report we read:

> 'From a practical standpoint, one of the most important results of a medical and engineering study such as this is the definition of safe working conditions in the industry under study (my emphasis). Ideally, a threshold concentration of dust [of the type in the industry under study] should be the highest dust concentration that would not produce pneumoconiosis in originally healthy workmen during their entire working life.'

46. Clearly the dust in the asbestos textile industry is the dust which arises from asbestos and the minor amount of cotton introduced to impart strength to the yarn being made. The dust in the asbestos textile industry was essentially 'asbestos dust' and the 5 million particles per cubic foot 'tentative threshold' established by Dreesen applied to asbestos dust and not to 'any dust which contains some asbestos'.

47. The 5 mppcf 'threshold' or 'maximum allowable concentration' or 'occupational exposure limit' or some other synonym was never meant to apply to asbestos cement as used by carpenters because the airborne dust was not 'asbestos dust' or 'asbestos particles' or 'the substance asbestos'.

48. Walter E Fleischer et al in Jour Industr Hyg & Tox, vol 28 no 1 Jan 1946 at p.13 writes:

> 'There are no established figures for permissible or safe dustiness in pipe covering operations [the American term for the application of thermal insulation (lagging to steam pipes]. Dreesen at al in their study of asbestosis in the asbestos textile industry suggested 5 million particles of total dust by impinger as a threshold for that

industry (my emphasis). We should like to point out that the asbestos textile and asbestos pipe covering industries differ widely in their dust exposures'.

49. 'The present TLV for asbestos (my emphasis) was recommended by Dreesen at al in 1938, after epidemiologic studies in textile mills using chrysotile asbestos. It was not intended for extrapolation to all forms of asbestos use under all circumstances of exposure.' (J LeRoy Balzer & W Clark Cooper in American Industrial Hyg Assoc J. May-June 1968 p222-7 at p.227).

50. The 1945 Victorian Regulations referred to the 'substance' asbestos and, indeed, 'asbestos particles' not asbestos-cement (DO Shiels, Letter to Editor, M.J.A. Nov 13 1976) .
5mppcf applied to dust from the substance asbestos as can be seen in the heading of the table in the Regulations; the 5 mppcf applied to 'asbestos particles' not dust generated by cutting asbestos-cement. The use of asbestos-cement by carpenters was not declared a dangerous trade in Victoria.

51. Many scientific articles are irrelevant to carpenters who used asbestos-cement which did not contain crocidolite; they are mostly directed towards insulation workers who worked with the more friable materials which contained a higher percentage of asbestos than asbestos-cement. Similarly, many articles refer to asbestos products not manufactured by the First Defendant.
Asbestos boards or sheets were not asbestos-cement boards or sheets and were either millboard or asbestos-insulation board such as Marinite. For example asbestos insulation board is a friable board which contained much more asbestos than the type of asbestos-cement manufactured by your client. 'Asbestos acoustical board' as used in the British navy was of two types - one was the soft type of insulation board which

contained about 25% asbestos and the other pure asbestos stiffened with sodium silicate (water glass). Neither type resembled the asbestos-cement manufactured by the First Defendant. Millboard was a soft board which contained about 85% asbestos, mostly chrysotile but some was made of amosite.

52. Dr Kilpatrick's statement that 'There has been extensive documentation on the hazards of asbestos exposure ... from the mid 1920's' should be read in conjunction with Selikoff's first sentence in his paper IJ Selikoff et al in Annals of NY Acad of Sci written in 1964 (Supra) p.139.

> 'Information currently available concerning asbestosis has been derived largely from studies of employees of asbestos textile factories and should properly be referred to such individuals.'

There was no 'documentation on the hazards of exposure to dust generated by carpenters using asbestos-cement' before 1964 nor, indeed, before the relevant period.

53. The reference by Dr Kilpatrick to 'women washing their husband's work clothes' is irrelevant because it was known only amongst workmen exposed to crocidolite.

54. The issue of visible dust has arisen out of a most unscientific theory that it is possible to determine quantitatively the airborne dust concentration by visual observations. This is scientific nonsense and indicates a fundamental ignorance of the optical properties of dusts.

The visibility of a dust cloud depends on the nature of illumination, the nature of the particles, the number of particles, the size of the particles and the particle size distribution, i.e. the relative number of big particles to small particles and those of intermediate size. One particle ten micrometres in diameter reflects

as much light as 100 particles one micrometre in diameter - or one particle ten micrometres in diameter contributes as much to the visibility of a light beam as one hundred particles one micrometre in diameter. Hence the absurdity of trying to estimate with the naked eye the number of particles in a dust cloud by comparison with measurements made of quite different clouds of different particle size and concentration.

To that may be added the comment that the obscuring power of light by dust particles is also increased by the dust concentration, the square of the diameter of the particles and the particles size distribution. INDUSTRIAL DUST by Drinker & Hatch, McGraw Hill 1936 at page 19 comments:

> 'It is frequently suggested that this [obscuring power] phenomenon could be used as a simple method for determining the concentration of dust in an industrial establishment. The application of the above equation to a practical problem, however, indicates the fallacy of this contention.'

Particles 100 micrometres and larger are present in mechanically generated airborne dust such as when asbestos-cement sheets are cut with power saws and grinders and these, no doubt, contribute markedly to the visible dust seen around the saw when cutting.

55. Airborne dust generated when asbestos-cement was cut was not asbestos dust; it was asbestos-cement dust. Asbestos cement contained asbestos particles and particles which arose out of the sand (silica) and cement which comprised about 85% by weight of the raw material. The commercial asbestos used in asbestos-cement was, itself, not pure fibre; it contained up to 6% by weight grit and rock which originated from the rock of the mine from which the fibre was extracted. Airborne dust from commercial

asbestos was referred to by some as a mixture of motes and fibres; it was termed 'asbestos dust'. The number of motes seem to have exceeded the number of fibres but this probably differed by, amongst other things, the grade of asbestos used. In my opinion nobody seriously attempted to determine the amount of asbestos fibre in the dust which arose out of the use of asbestos cement, particularly when it was cut with a power saw (or grinder). I do not think that it would have been profitable for any person to try to determine the fibre and non-fibrous components in an airborne dust cloud which arose out of work by carpenters with asbestos-cement, if only because no disease was foreseeable.

O Kilpatrick 1969

If, after the development of the so-called membrane filter method for estimating airborne asbestos dust, any person had been interested in determining the airborne asbestos concentration to which a carpenter was exposed when using asbestos-cement, he/she would have disregarded the non-fibrous particles because they are inert.

56. I reject entirely any suggestion by Dr Kilpatrick that, during the relevant period, it was possible to guess even approximately the concentration of an airborne dust generated by carpenters while working with asbestos-cement in the open air by visual sensation because the parameters vary so much. This would be true particularly for asbestos-cement dust in which, amongst other things, the particle size distribution could not be expected to remain constant from time to time, from job to job, from workman to workman. There were, of course, no objective measurements against which different visual sensations could have been compared.

What data are there now on these exposures & visibility of the dust?

Whilst I argue that it was impossible to quantify any dust concentration by visual sensation, it is even more absurd to assert that it is possible to estimate the fibre level from the visual observations of the airborne

mixed asbestos-cement dust as described by another person. It is equally absurd to suggest that visual observations of a markedly varying concentration can be averaged over a period of eight hours.

57. Dr Kilpatrick refers to the issue of warnings. The British did not place warning labels on asbestos-cement voluntarily until 1976. really?

58. In final conclusion, during the relevant period, there was no foreseeable danger of asbestos-related disease amongst carpenters who used asbestos-cement which did not contain crocidolite.

(NOTE - Substance of Expert Evidence is yet to be settled by Mr Major)