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SUBSTANCE OF EXPERT EVIDENCE

OF

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in the matter of

John Brownell Hulm v James Hardie & Coy Pty Ltd

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1. I graduated in medicine and surgery from the University of Queensland in December 1962. After two years hospital experience and seven years in country general practice in Queensland (six years part-time occupational medicine), I attended the University of London as a full-time student in 1972/73 and obtained the Master of Science degree in Occupational Medicine. Since obtaining that degree I have been engaged full-time in the specialty of occupational and environmental health.

2. From August 1973 to October 1979 I was employed by the UK Health and Safety Executive (HSE) and my duties included working with the Factory Inspectorate in the administration of the 1969 Asbestos Regulations, and in the medical supervision of asbestos workers. From 1976 to my return to Australia in 1979, I held senior positions in the HSE which included overall responsibility for the medical supervision of all asbestos workers in Great Britain.

3. During the years 1973-1979 I was also appointed as a lecturer in occupational medicine at the University of London. This, and my work in the HSE required the maintenance of an up to date knowledge of the asbestos research including frequent personal contact with the leading researchers including Dr Chris Wagner and Dr Muriel Newhouse.

4. Dr Newhouse was one of the pioneers of the epidemiology of mesothelioma and described the occurrence of mesotheliomas in the wives of asbestos factory workers. Whilst this work demonstrated that the risk of mesothelioma could occur in people other than those working in asbestos factories, and at concentrations less than those needed for asbestosis, mesothelioma was still regarded as dose-related.

5. The generally held view of the researchers and the HSE during those years was that intermittent work with asbestos products in the construction industry did not constitute a risk from asbestos related diseases including mesothelioma. This view was epitomised in the HSE document: *Technical Data Note 42. Probable asbestos dust concentrations at construction processes* published in April 1973. In a section headed "Interpretation", this document stated:

When considering the need for precautions, discretion is possible if the activities are only occasional and for short duration. In a similar way if a normally steady job is interrupted for some time, it may be possible to average an exposure over a four hour period before passing judgement. When concentrations of dust exceed the Hygiene Standard of 2 fibres/ml averaged over a 4 hour period, or a 10 minute exposure to greater than 12 fibres/ml or 0.2 fibres/ml in the case of crocidolite (blue asbestos), then HM Inspectors of Factories will require precautions to be taken so as to gain compliance with the Asbestos Regulations 1969.

6. The work done by the plaintiff would not have been regarded as being subject to the 1969 Asbestos Regulations and therefore the precautions required by these Regulations would not have been expected.

7. This is how the HSE and the industrial community viewed the asbestos risks in the seventies in Britain. I do not believe that any greater levels of awareness of risks could be expected in Australia during the same time. I will support this with an outline of Australian developments later in this summary of evidence.

8. Some people have expressed the view that as little as one asbestos fibre is enough to cause an asbestos-related disease; others have expressed asbestos risks in terms of "the extreme dangers of exposure to asbestos in very small quantities". These views were not given any credibility, if indeed they were ever expressed, in the asbestos literature to the end of the 1960s.

9. The seminal papers of Cooke in 1924 and 1927 were concerned with asbestosis in asbestos textile factories and referred to the possibility of a dose effect in that *medical men have long suspected asbestos dust to be the cause of lung conditions in workers in badly ventilated factories.*

10. Merewether and Price in their 1930 report made the following comments concerning dose-response:

the increase in prevalence of fibrosis with increasing duration of employment (ie increasing exposure) was well demonstrated.

the hypothesis was generated that adequate dust suppression would eradicate the disease.

11. Merewether, in a 1933 memorandum on asbestosis (parts 6 to 9) published in Tubercle, raised the possibility of a safe dose and it was concluded that it must be below the level in one of the less dusty jobs in the factories studied. Part 10 of this memorandum described how industry was agreeable to doing everything possible to suppress dust to prevent the problems. Twenty four points of agreement were set out to achieve this aim.

12. The Dreessen paper in the 1938 US Public Health Bulletin No 241 demonstrated a relationship between dose (years exposed and dust levels) and proportion (percentage) of subjects with disease: worst in carding. Dressen considered an exposure of 2.5 million particles per cubic foot (mppcf) to be safe and suggested that 5.0 mppcf would be a reasonable threshold concentration for dust containing asbestos and suggested that this become the standard of exposure.

13. The 1955 publication by Doll in the British Journal of Industrial Medicine titled "Mortality from lung cancer in asbestos workers" described the link between lung cancer and asbestos in workers in asbestos factories. Doll concluded that:

lung cancer was a specific industrial hazard of certain asbestos workers and that the average risk among men employed for 20 or more years has been of the order of 10 times that experienced by the general population. The risk has become progressively less as the duration of employment under the old dusty conditions has decreased.

14. All the above seminal papers gave readers at the time the view that the asbestosis and lung cancer were due to exposures in dusty asbestos factories (mostly asbestos textile factories), that the diseases could be prevented by reducing the factory dusts, and that there was a safe dose of asbestos.

15. Other papers published in 1955 supported the view that the asbestos diseases would be prevented by reducing factory exposures. Sander's paper in the American Journal of Surgery, 1955, 90: 115–119 was titled "Silicosis and asbestosis. Diagnosis, prevention and treatment", and it concluded:

silicosis and asbestosis are preventable diseases. Experience in the past ten years has shown not only a decreasing incidence of new cases as recognised by X-Ray, but also fewer and fewer affected workers are becoming disabled. This is due not only to lower and lower dust exposures in most industries but also to lessened opportunities for tuberculosis contacts in our communities and industries, and to an even greater extent to

an increasing optimism on the part of the medical profession on the prognosis of these dust diseases. The pessimism of the past is being replaced by the realisation that a diagnosis of silicosis or asbestosis is comparative with good health and a feeling of well-being.

16. Also in 1955, K W Smith's report in the Archives of Industrial Health, 12: 198-203 titled "Pulmonary disability in asbestos workers" concluded:

Respiratory disease experience among several thousand male and female asbestos workers in the United States and Canada is reported. Of all workers exposed to the fibers, very few develop asbestosis. An X-Ray survey of 708 employees in a milling operation showed that the majority had normal X-Ray patterns, that 10 or more years of exposure were necessary to produce X-Ray changes, and that no cases of asbestosis were found who had worked less than 20 years in the dust.

17. In Australia in 1956, the information on asbestos resulted in the listing of "asbestos lagging or spraying" and "asbestos works or factories in which asbestos is used manipulated, crushed or pulverised" as dangerous trades under the Health Acts in Victoria. No mention was made of the use of asbestos products. It was to be a further 22 years before the asbestos regulations were enacted in Victoria. The 1978 Victorian regulations included reference to the use of asbestos products, but the regulations only applied to the use of power tools on asbestos containing products and only if the periods of exposure exceeded forty hours in any three consecutive months.

18. The 1957 paper by Gordon Thomas in the Medical Journal of Australia titled "Pneumonokoniosis in Victorian industry" described the prevalence of asbestosis in Victorian asbestos factories, including factories manufacturing and processing asbestos products. The paper did not refer to risks associated with the use of asbestos products in the construction industry and it was more than twenty years before such exposures were included in Victorian asbestos regulations.

19. Similarly, the 1960 paper by Maurice Joseph in the Medical Journal of Australia "Occupational diseases of the chest, excluding silicosis" described cases of asbestosis in people heavily exposed to asbestos in lagging, mattress making and emptying bags of asbestos in an asbestos factory. There was no mention of the risks involved in the use of asbestos products in the construction industry, nor was there any suggestion of "the extreme dangers of exposure to asbestos in very small quantities".

20. The first reference to any concern about the possibility of risks associated with the use of asbestos products in the construction industry appeared in the "Report and recommendations of the working groups on asbestos and lung cancer". The Working Group was convened under the auspices of the International Union Against Cancer (UICC) and met in New York in October 1964. Delegates included the world's leading researchers, but the Australian invitee was unable to attend. Among the "Recommendations or problems requiring epidemiological study" was the following:

5. That studies of morbidity and mortality be extended to asbestos-exposed populations that have not so far been widely investigated.

(1) It is recommended that special attention be directed to surveys in:
(a) the insulating industry including that in ships
(b) the asbestos cement industry
(c) asbestos products industry
(d) other plants in which asbestos is regularly used, such as certain paper, paint, and plastic factories.

(2) It is also recommended that, since incidental exposure to asbestos dust may occur in certain trades and occupations, attention be directed to:
(a) handling and transporting asbestos
(b) the building industry
(c) pipe fitting
(d) ship building and breaking

21. Following that 1964 meeting, and in response to the Wagner and Selikoff papers, there was a gradual increase in concern about asbestos risks. This resulted in the 1969 Asbestos Regulations in Britain which applied to: "Every process involving asbestos or any article composed wholly or partly of asbestos, except a process in connection with which asbestos dust cannot be given off". As stated in paragraphs 5 and 6 above, the view in the late 1960s and early 1970s was that the use of asbestos products in the construction industry generally was of such low risk as not to require the precautions set out in the 1969 Asbestos Regulations.

22. The first paper on "Dust problems in the use of asbestos products" was presented to the 1969 International Pneumoconiosis Conference in Johannesburg by E Walther. His paper summarised clearly the widely held views on the use of asbestos products as follows:

Problems associated with using asbestos products were discussed. It was noted that even under adverse conditions it is very unlikely that consumer exposure to asbestos would reach the levels of intensity that exist in occupational environments. Much of the controversy in recent years about the risk of asbestos to the general public stems from the lack of precise information about environmental asbestos exposures and extrapolating exposure data from the occupational environment to that of the general public. Risks associated with consumer exposures to asbestos cement products, brake linings, floor tiles, insulating materials, asbestos containing textiles, and battery boxes were discussed. The author concludes that asbestos containing products under conditions of normal use do not present an abnormal health hazard. Experience gained in industrial exposures cannot be transfered to the consumer environment. Additional research is needed to determine the extent of environmental contamination with asbestos, the exact dust concentrations and length of exposure required to initiate disease, and how long asbestos fibers persist in the environment.

23. In Australia, the National Health and Medical Research Council (NH&MRC) was the government body maintaining an active interest in the occupational health aspects of asbestos. In 1968 the NH&MRC recommended a dust standard of 5 mppcf, the same standard which Dreessen had recommended in 1938. In the years 1973-75 the NH&MRC developed the "Model Asbestos Regulations".

24. Queensland was the first Australian state to enact asbestos regulations in 1971 as The Asbestos Rule (Rule 9) of the Factories and Shops Act 1960. Other states followed: South Australia 1976, NSW February 1978, Western Australia August 1978, Victoria December 1978, and Tasmania 1979. In 1982, lung cancer caused by asbestos was added to the list of occupational diseases (3rd schedule) Workers' Compensation and Assistance Act Western Australia.

25. It was not until 1979 that the NH&MRC published its document "Statement on health hazards associated with the use of asbestos in the construction industry". Even in 1979 the text of the document did not suggest extreme dangers of exposure to asbestos in very small quantities, though it did recommend a reduction in the then asbestos standards of 0.2 f/ml for crocidolite and 2.0 f/ml for amosite and chrysotile. The following extracts demonstrate these views:

Materials which contain asbestos are extensively used in the construction industry and because of their special properties will continue to be used in the foreseeable future. If these materials are used carelessly, excessive exposure of workers to airborne asbestos fibres may occur.

The diverse work situations encountered in the construction industry and the difficulties faced by persons attempting to quantify the extent of asbestos dust exposure of building workmen are recognised. Concern is also expressed regarding the adequacy of the current hygiene standards for asbestos dust in the workplace. In the light of the present knowledge, it is advised:

Recommendation 7

The Sub-committee recommends that all products containing asbestos be labelled by the manufacturer or importer so as to alert the user to potential hazards. For example:

- *CAUTION*
- *contains asbestos fibre*
- *avoid creating dust*
- *breathing asbestos dust may cause serious damage to health including cancer*
- *smoking greatly increases the risk*

29. I believe that all the above examples indicate that concern about possible health risks from the use of asbestos products in the construction industry is a relatively recent development. The concern was first raised at the UICC Workshop in 1964, but only converted into health recommendations by the NH&MRC in the late 1970s.

30. I also believe that some statements should be made about the causation of mesothelioma. Whilst it is possible that the plaintiff's mesothelioma is due to asbestos, it is by no means certain.

31. In 1972, an advisory committee on asbestos cancers reported to the Director of the International Agency for Research on Cancer:

Evidence for an important difference in risk in different occupations and with the type of asbestos has increased. The risk is greatest with crocidolite, less with amosite, and apparently less with chrysotile. With amosite and chrysotile there appears to be a higher risk in manufacturing than in mining or milling. There is also evidence from population studies that a proportion of cases of mesothelioma have no known association with exposure to asbestos.

32. Two recent papers, one co-authored by Dr J C Wagner who did the pioneering work on mesothelioma in the late 1950s, have cast doubt on the currently held views in Australia that all mesothelioma are caused by asbestos and that even minute exposures are enough to cause mesothelioma.

33. The first paper is: Ilgren E B, Wagner J C. "Background incidence of mesothelioma: animal and human evidence". Regulatory Toxicology and Pharmacology. 1991; 13: 133-149:

Evidence is presented showing that mesotheliomas can have causes other than exposure to asbestos dust in both experimental animals and humans. In experimental animals, for example, results from two major experimental laboratories suggest that at least 10% may be taken for background incidence whereas a third laboratory suggests that the experimental group must have a rate exceeding 30%. "Background" also includes mesotheliomas found in association with non-fibrous and fibrous non-asbestiform agents. Mesotheliomas in humans can be broadly classified in a manner similar to those of experimental animals: (1) spontaneously occurring, (2) those with a latent period less than ten years, (3) childhood mesotheliomas, (4) familial cases, (5) cases before the 20th century, (6) mineralogically negative mesotheliomas, and (7) mesotheliomas caused by non-asbestiform agents. The importance of the acceptance of these "background" cases lies in the fact that a basis is provided for the study of the incidence of disease associated with various types of asbestos.

34. The second paper is: Ilgren E B, Browne K. "Asbestos-related mesothelioma: evidence for a threshold in animals and humans". Regulatory Toxicology and Pharmacology. 1991, 13: 116-132:

A threshold for mesothelioma for the major asbestos fibre types becomes not only plausible but also very likely in view of the existence of a distinct background incidence of spontaneously occurring and non-asbestos related mesotheliomas; the high occupational doses associated with the appearance of mesotheliomas in humans, and the

large number of "tumourigenic" fibres required to produce significant numbers of mesotheliomas in animals. Even when the duration of exposure associated with the appearance of mesotheliomas in humans has been brief, the exposure itself has been intense. The review of the relevant animal and human literature cited herein supports the concept of mesothelioma threshold."

35. Before regarding the plaintiff's mesothelioma as probably due to his work exposures to asbestos, it is important to have knowledge of the asbestos burden in his lungs, and more detailed information on his actual asbestos exposures.

Dr David Douglas
2 December 1991