IN THE HIGH COURT OF AUSTRALIA

SYDNEY REGISTRY

No. S 219 of 2011

BETWEEN

AMACA PTY LTD (ACN 000 035 512)

(UNDER NSW ADMINSITERED WINDING UP)

Appellant

JOHN WILLIAM BOOTH

First Respondent

AMABA PTY LTD (ACN 000 387 342)

(UNDER NSW ADMINISTERED WINDING UP)

Second Respondent

APPELLANT'S SUBMISSIONS IN REPLY

Part I: Internet Publication

1. These submissions are in a form suitable for publication on the internet.

Part II: Argument

2. Amaca makes the following eight general submissions in response to Mr Booth's submissions.

3. *First*, Mr Booth repeatedly suggests that Amaca has referred selectively to the evidence; omitting reference to material that does not support its case (see, e.g., RS [23], [25], [38], [44]). In fact, rather than refer to every single occasion upon which the medical witnesses used a verbal formula capable of suggesting that every exposure to asbestos is causative of mesothelioma, Amaca referred to those portions of the evidence that could reasonably be regarded as representing the high point of the material in Mr Booth's favour. The additional extracts of the evidence to which Mr Booth has referred in his submissions do not add anything to the material dealt with in Amaca's submissions. Indeed, the additional passages upon which Mr Booth relies are subject to the same qualifications or explanations given by each of the medical witnesses set out in Amaca's submissions. Once those general qualifications and

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explanations are taken into account, the true import of all the evidence, including those additional passages to which Mr Booth refers, is revealed.

4. *Secondly*, Mr Booth appears to assert that there is "some evidence" supporting the "every exposure is causative theory" provided that the evidence of one or more of the medical witnesses contained verbal formulas consistent with such a finding. Mr Booth is accordingly dismissive of Amaca's attempts to place the evidence containing those verbal formulas in context, to elucidate the meaning sought to be conveyed by expert witnesses using technical language, and to point to the underlying evidence that explains or qualifies the witness' evidence, as an impermissible attempt to "re-characterise" that evidence (RS [23]). Yet the question whether there is evidence to support a finding must be answered by reference to the substance of the evidence, not mere verbal formulas.

5. Thus, in the case of Professor Henderson, to understand his opinion one must consider its stated basis. The basis is primarily twofold:

   a. *First*, reliance on a "dose-response causal relationship" given by "the Peto Model and its various modifications" which generates the conclusion that each increment of exposure within a latency period produces an increment in the "risk/incidence" of mesothelioma.[1] Usually, but not always, there will be an exposure above background exposure if mesothelioma develops.[2]

   b. *Secondly*, although the epidemiological studies on brake lining work in particular are inconclusive, studies on chrysotile asbestos in general plus other reasoning allows a cautious conclusion that asbestos released from brake linings has the "capacity" to induce mesothelioma.[3] That is, exposure to dust from brake linings becomes a relevant "increment to risk" within the overall Peto Model of "causation".[4]

6. None of this basis provides a reason to conclude which of the various exposures which would create across a population an "increment in risk" have in fact, on their own or in any combination, caused Mr Booth's mesothelioma.

7. Mr Booth's repeated references to a "biological", "pathobiological", or "aetiology" explanation (RS [24], [26], [27], [29], [30], [31], [32], [33]) do not take matters any further. The evidence cited at RS [37] was in answer to a question seeking to explain the Peto Model.[5] Nothing in that model allows a conclusion that every asbestos fibre inhaled in fact plays a role in the contraction of mesothelioma in those people who develop the disease, or that every fibre not cleared from the lungs plays such a role (c.f., RS [34]).

8. *Thirdly*, and similarly, Mr Booth asserts that Dr Leigh has provided a "biological" explanation for the "every exposure is causative" theory (RS [43]). Even taken at its highest (i.e., that there can be interaction between various asbestos fibres to which a person is exposed and his or her mesothelial cells, over various generations of cells, leading to the development of mesothelioma) that biological explanation does not

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[1] CA Blue(1) at 67-8.

[2] CA Blue(1) at 75.

[3] CA Blue(1) at 93.

[4] CA Blue(1) at 94.

[5] CA Black (1) at 97C; CA Blue (1) at 67.

support a finding that every exposure of a person to asbestos fibres is causative of his or her mesothelioma. In particular:

a. The experts accepted that Mr Booth's exposures to asbestos *prior* to his exposures by Amaca, or later Amaba, were *capable* of causing his mesothelioma on their own.[6]

b. The experts provided no opinion as to *when* Mr Booth developed mesothelioma; i.e., whether it was before or after the Amaca exposure; or whether he would have developed it regardless of the later Amaca exposure.

c. If the disease commenced prior to the Amaca exposure, or would have developed regardless of the later exposures, then those later exposures are causally irrelevant to the development of his disease.

9. In other words, even if the evidence supported a view (which it did not) that every fibre to which Mr Booth was exposed up until the point at which he developed mesothelioma, or was sufficiently damaged that he would inevitably develop mesothelioma, was causative of his disease, that does not mean that every exposure over his lifetime was causative. Dr Leigh's "biological explanation" does not permit a finding that it is more probable than not that the Amaca exposure was causally relevant.

10. *Fourthly*, on various occasions throughout his submissions Mr Booth sets out wrong or misleading accounts of the evidence. For example:

a. Mr Booth submits that the evidence of Professor Berry supported the "every exposure is causative" theory (RS [18], footnote 38), asserting that Professor Berry agreed that it was "the lifetime load of all asbestos exposure which causes the illness in the individual". In fact, that is a quotation from a question by Mr Booth's counsel with which Professor Berry did *not* agree.[7] Rather, Professor Berry answered that "it's the total lifetime exposure and the components that make up that total lifetime exposure that *increase the risk*" (emphasis added). Later in that answer, when he went on to deal with the question of cause, Professor Berry said "we would then be saying well which bits of exposure is it due to and *we wouldn't really be able to determine that except on probability grounds* based on the sizes of the different types of exposure" (emphasis added). Professor Berry's evidence was thus directly inconsistent with the "every exposure is causative" theory.

b. Mr Booth submits that Dr Leigh's evidence that every exposure to asbestos fibres of a person with mesothelioma was causative of the disease was supported by the conclusion of "definitive text" of Dodson and Hammar (RS [42]). That is a surprising submission given that Dr Leigh himself conceded that he was unable to identify a single study or authority that supported the "every exposure is causative" theory.[8] The ellipsis in the passage quoted from Dodson and Hammar by Mr Booth replaces nearly an entire page of text, with the result that the abbreviated quotation does not accurately convey the meaning of the text. In fact, the quoted passage concerns the comparative carcinogenic qualities of short versus long asbestos fibres. Moreover, it is the

[6] CA Black(1) at 117-119, 263-269.

[7] CA Black 552V.

[8] CA Black at 482D-G.

various pathological events that "can" be caused by short fibres that are said to "cumulatively" cause asbestos-related diseases, not the cumulative exposure to asbestos fibres.[9]

11. *Fifthly*, despite a suggestion that the causation finding could be supported by evidence other than the medical evidence in the case (RS [19]), in fact the only possible basis for that finding is the medical evidence. The only other evidence suggested by Mr Booth to be capable of supporting the "every exposure contributes" theory was the evidence of Professor Berry (RS [10], [18]). For the reasons given above, that evidence provided no support for the theory.

12. *Sixthly*, Mr Booth suggests that Amaca failed to put necessary propositions to Professor Henderson (and perhaps by implication, other medical witnesses) in cross-examination (RS [13], [30], [31]). Two answers may be made to that contention:

    a. *First*, Amaca's case was clearly and sufficiently put to Professor Henderson in cross-examination. The inability of medical (or any other) science to identify the cause of Mr Booth's particular mesothelioma, as opposed to identifying exposures that increased his risk of contracting the disease, was fully exposed in Professor Henderson's cross-examination.[10]

    b. *Secondly*, Mr Booth's criticism assumes that Professor Henderson's evidence, properly understood, supported the "every exposure is causative" theory. For the reasons given in Amaca's primary submissions, that is not the case. If that is right, then Professor Henderson's evidence was not "outside the same body of international learning" upon which *Sienkiewicz v. Grief (UK) Ltd* [2011] 2 WLR 523 and other English cases were decided. There was, therefore, no need to put that particular proposition to Professor Henderson.

13. *Seventhly*, Mr Booth submits that he is entitled to succeed merely upon the basis that every exposure to asbestos adds to the overall risk of the development of mesothelioma, and that risk has materialised (RS [35], [46]). That submission is based on a simplistic, and wrong, reading of Kiefel J's judgment in *Roads & Traffic Authority v. Royal* (2008) 82 ALJR 870 at 898 [144]. In circumstances where the risk of an event is attributable to multiple different factors, the occurrence of the event does not permit an inference that any one or more of those factors was the cause (unless, contrary to the assumption upon which Mr Booth makes this submission, it is known that the event can only be caused by all factors operating together). In other words, unless one assumes the validity of the "every exposure is causative" theory (the very thing Mr Booth, in this alternative submission, is trying to avoid), the development of mesothelioma does not permit an inference that the various exposures that added to the total risk were each causative.

14. *Eighthly*, Mr Booth submits that the "every exposure is causative" theory was somehow recognised in *Sienkiewicz* (RS [15]). That is not the case. The passage to which Mr Booth refers is contained in a discussion by his Lordship as to the unreliability of drawing inferences as to causation in particular cases from epidemiological evidence as to risk in populations generally (at [94]-[106]). One aspect of his Lordship's reasoning on that topic was the existence of various limitations on the reliability of epidemiological evidence (at [98]-[102]). His Lordship's reference to the discrediting of the "single fibre theory", and the

[9] CA Blue(1) at 426.

[10] For example, CA Black(1) at 114-115, 116-117

"possibility (but no more)" raised by the Peto Report that there may be a "synergistic interaction between early and later exposures", was to demonstrate that it was not possible to infer from epidemiological studies that a particular exposure to asbestos caused mesothelioma. His Lordship did not suggest that *all* exposures to asbestos were thus to be regarded as causative of mesothelioma. Indeed, the question his Lordship posed ("which exposures in an individual case may have contributed to causing the disease?") (at [102]), his affirmation of the special rule in *Fairchild* (at [103]-[105]), and his recognition of "the gaps in our knowledge" (at [106]), demonstrate that the "every exposure is causative" theory was neither urged upon, let alone accepted by, his Lordship.

Conclusion

15. Mr Booth does not contend for any *Fairchild* exception, reversal of the onus of proof, or other alteration to the ordinary principles of burden of proof in a civil suit for damages. Accordingly, the verdicts below against both appellants can stand only if there was a basis in the evidence for the all of the following three propositions in respect to this particular plaintiff:

    a. The various exposures to asbestos which Mr Booth faced prior to 1953, while *capable*, alone or in combination, of causing his contraction of mesothelioma, did not in fact do so - the entirety of this exposure to asbestos fibre prior to 1953 must either have played no, or no sufficient, role in his subsequent contraction of mesothelioma such that as at 1953 he was uninjured or at worst only potentially injured;

    b. That exposure to some asbestos fibre during the period 1953-1962 was a *necessary* step in his ultimate contraction of mesothelioma, and this fibre can be identified as, or as including, Amaca asbestos fibre, as opposed to being, say, background risk – with the result that as at 1962 he was a person *potentially* injured (*potentially*, not actually, because if his exposure to this point was both necessary and sufficient to cause his ultimate contraction of mesothelioma, then Amaba should have been held not liable);

    c. That exposure to some asbestos fibre during the period post 1962 was both a *necessary and sufficient* step in his ultimate contraction of mesothelioma, and this fibre can be identified as, or as including, Amaba asbestos fibre, as opposed to being, say, background risk –with the result that it was exposure to Amaba fibre that turned his potential injury into actual injury.

16. Neither the judgments below, not Mr Booth's submissions, identify where in the body of evidence (whether the Peto Model or otherwise) a foundation for such propositions was laid.

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