or other period of employment may well have led to the fatal cellular transformation, it was not possible to say which, and he was therefore unable to find either responsible.

In coming to his judgement, Mr Justice Curtis made a distinction between causation and risk factors. In his words "the only relevance of the number of fibres is in connection with the risk of contracting the disease". He was thus dissuaded from being influenced by any evidence that might have shown a relationship between risk of mesothelioma and dose of asbestos, although there is much such evidence from studies both of human lungs and of animals.

I have heard the view expressed before that one fibre causes mesothelioma. It depends what you mean by "cause". It is in one sense obvious nonsense. We all have millions of asbestos fibres in our lungs and the likelihood of us developing mesothelioma depends on how many millions. This means that the disease is dose related. The problem in this case arose from confusing the disease *mesothelioma* with transformation in a *cell*, which may be a factor in the development of the disease. Take the case of the butterfly flapping its wings in the Amazon rain forest. It may be possible for an ingenious QC to prove that the hurricane that hit the coast of west Africa was caused by that insect's action, but the other side would surely point to other risk factors that, taken with the action of the butterfly, contributed significantly to the disaster.

Is it really possible to say that the only and necessary cause of mesothelioma is transformation in a cell? Are we sure that the *milieu* in which that cell lives and divides is not influential? Are we sure that inflammation in the tissue involved is not an important precondition for the development of the disease? Are we sure that the action of asbestos on other cells does not interfere with the natural defences that would otherwise eliminate the transformed cell? Are we sure that genetic factors and viruses do not also determine whether the transformation occurs or succeeds in overcoming the body's defences? Or looked at another way, experimentally, how many rats would have to be used to produce one mesothelioma after injection of one fibre into the peritoneum? Of course, no one has ever shown that one fibre causes mesothelioma. All that has been conjectured is that the malignant cells that form part of the tumour may be the genetic offspring of one transformed cell.

The judge appears in this case to have been persuaded to accept a naïve view of causation—that disease has one ultimate cause. Most who have studied the causation of disease would argue that the likelihood of disease occurring in any individual is influenced by multiple factors, the outcome of inherited and acquired susceptibility and environmental precipitants. In the case of mesothelioma, a very heavily asbestos exposed individual may have a one in 10 lifetime risk of the disease. Most of us, with very small incidental exposures, have about a one in 1 million annual risk. The risk varies with the length and intensity of exposure, as assessed by the individual's occupational history. We know from animal studies that asbestos fibres do indeed cause mesothelioma, so this evidence of a dose related association strongly suggests that factors other than transformation of one cell are also, and critically, responsible for the disease. Among these is likely to be inflammation in the pleura initiated by the presence of many fibres. Such inflammation may not only result in malignant transformation of many cells, but may also inhibit the

natural mechanisms whereby such cells are eliminated. If this is true, and it is certainly more plausible than the one fibre theory, then mesothelioma is caused by the access of large numbers of fibres to the mesothelial tissue. Since it is a dose related disease, it may be argued that all exposures to asbestos before a critical time would be expected to have contributed to the causation of the disease. Thus, attribution of blame should be on the basis of relative intensity and duration of exposure in different trades.

The moral of this story is that lawyers are clever people and part of their business is the meaning of words. The word "cause" is one that requires a bit of thought. My *Shorter Oxford Dictionary* devotes a column to it.

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Mesothelioma

We write as the three medical witnesses who provided evidence (all in writing, two orally) to the Court in the case referred to by Professor Seaton. Essentially we agree with his analysis. The medical evidence presented to the Court made it clear that the risk of mesothelioma increases in relation to the dose of asbestos and that it is not possible to identify the particular fibre or fibres involved in the genesis of a particular mesothelioma. From an epidemiological standpoint it is therefore appropriate to regard all sources of significant exposure as having contributed to causation of the disease, in the same way that all cigarettes smoked would be considered to have contributed to causation of a lung cancer.

Mr Justice Curtis, however, accepted the invitation of Leading Counsel for one of the defendants to adopt a strictly mechanistic approach to causation. He decided that, because the claimant could not show whether the fibre or fibres actually involved in the genesis of the tumour were derived from either or both of two sources of exposure, causation could not be established against either of two defendants.

More recently, a different view has been taken in a similar case by Mr Justice Mitting (Queen's Bench Division C20010111). He considered that there was "no substantial difference between saying that what the defendant did materially increased the risk of injury to the claimant and saying that what the defendant did made a material contribution to his injury". It would be "wholly artificial to require a claimant to prove which fibre or fibres, inhaled in whose employment in precisely what circumstances, caused or set off or contributed to the process by which one or more mesothelial cells become malignant". He concluded that breach of duty on the part of both defendants caused the mesothelioma.

Both cases are soon to be considered by the Court of Appeal and the outcome will determine whether the many mesothelioma victims who happen to have derived their asbestos exposure from more than one source are to be left without redress.

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Statement on malignant mesothelioma in the UK

We would like to provide the following additional material to Appendix 3 "Sources of information and help available for patients and carers" which appeared on pages 263–4 of the BTS statement on malignant mesothelioma in the UK published recently in *Thorax*.¹

The following Asbestos Support Groups are the major practical sources of information in the UK for people with asbestos related diseases. Most provide a drop in and telephone service, giving confidential free advice and support to patients and families. They also have particular expertise in the field of industrial injury benefits and government and civil compensation claims. Although most of the groups are in the north of England, telephone queries from any part of Great Britain are acceptable to them.

Manchester (tel: 0161 953 4037)

Sheffield (tel: 0114 282 3212 or 01709 513 587)

Liverpool (tel: 0151 236 1895)

Bradford (tel: 01274 393 949)

West Yorks (tel: 0113 243 9979)

Cheshire (tel: 01928 576641)

Nottingham (tel: 0115 927 5108)

In Scotland:

Clydeside Action on Asbestos (tel: 0141 552 8852)

Clydebank Asbestos Group (tel: 0141 951 1008)

Other important sources of help and information are:

The Macmillan Mesothelioma Information Line (tel: 0113 206 6466; email: mavisro@ulth.northy.nhs.uk) and the Occupational and Environmental Diseases Association (OEDA), both of which were mentioned in the original statement.

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Reference

- 1 British Thoracic Society Standards of Care Committee. Statement on malignant mesothelioma in the United Kingdom. *Thorax* 2001;56:250–65.

Asymptomatic pulmonary involvement in RA

Dawson *et al*¹ found HRCT evidence of fibrosing alveolitis (FA) in 19% of 150 outpatients with rheumatoid arthritis (RA). The presence of FA did not relate to previously described predisposing factors such as male sex, nodular and/or extra-articular disease, disease duration and severity. Moreover, the authors did not find any relation with respiratory symptoms such as dyspnoea or cough, chest radiographic appearance of FA, or restrictive pattern at pulmonary function tests. The only features significantly associated with FA on the HRCT scan were the presence of bibasal crackles and the reduction in carbon monoxide transfer factor (Tlco). These findings are more difficult to explain, especially considering that FA was defined as an HRCT pattern