OUTER HOUSE, COURT OF SESSION

[2024] CSOH 112

PD471/21

OPINION OF LORD MALCOLM

in the cause

ALASDAIR KERR and others

Pursuers

against

MIDLOTHIAN COUNCIL and another

Defenders

Pursuer: J Brodie KC and D Swanney; Digby Brown LLP
First Defender: N McKenzie KC; Clyde & Co LLP
Second Defender: A Cowan; Kennedys Scotland

20 December 2024

Introduction

[1] In 2020 Mrs Sarah Kerr noticed symptoms of what proved to be epithelioid malignant peritoneal (abdominal) mesothelioma which, tragically, caused her death in June 2022. She had been employed by Midlothian Council as a chemistry and science teacher during two spells, first from July 1983 until late 1985 at Dalkeith High School; secondly from May 1999 until November 2003 at St David's High School, Dalkeith. She was employed by Moray Council in the same capacity at Buckie High School from late 1985 until June 1990, and again between 1992 and 1995. During her employment she required to

handle Bunsen burner heat mats and associated wire gauzes. It is claimed that as a result she was exposed to asbestos fibres capable of causing her fatal disease.

[2] This action for damages (monetary compensation) brought by her husband and family proceeds on the basis that Mrs Kerr's employers should have been aware of a significant risk of asbestos related injury arising from her duties. Statutory cases are made under regulations 4 and 8 of the Control of Asbestos at Work Regulations 1987 and regulations 5, 9 and 10 of the Control of Asbestos at Work Regulations 2002.

[3] It is said that in breach of their duty towards Mrs Kerr both employers exposed her to significant quantities of asbestos dust thereby creating a material (ie not so minor as to merit disregard) increase in her risk of contracting the disease, thus meeting the test for joint and several liability laid down in *Sienkiewicz* v *Greif (UK) Ltd* [2011] 2 AC 229, Lord Phillips of Worth Matravers at paragraph 1. As to proof that asbestos played a causative role in Mrs Kerr's death, reliance is placed on creation of this risk as opposed to demonstration that the exposure caused or contributed to the injury. Whether this approach is open to the claimants was the focus of much of counsel's submissions at the close of the evidence led at a proof which addressed only liability, the amount of any damages having been agreed by the parties.

A summary of the lay evidence as to Mrs Kerr's exposure to asbestos during her employment

[4] The principal source of evidence as to Mrs Kerr's exposure to asbestos through her work comes from her second affidavit dated 18 November 2021. She regularly had to handle "asbestos heat mats" and associated flexible wire gauzes when Bunsen burners were being used. The mats were "white fibrous asbestos" in colour. Some of the mats were old,

ragged and damaged at the corners. They would often break in half. They and the gauzes were kept in cupboards which became very dusty. Dust was deposited on her hands and clothes. It was possible that she could be using the mats and gauzes at least for one period every day, or during multiple periods on one or two days a week. She could remember seeing "flaky bits from these boards" on her hands and clothes. In about 2003 when her school moved to a new campus, the most damaged mats were replaced with new ceramic mats. Instructions were issued to remove any remaining asbestos mats or products.

[5] Rosemary Young gave evidence. Since 2000 she has been an assistant principal teacher of science, including chemistry, at St David's High School. Asked about the condition of mats in her first three years, she said some of them, perhaps three out of ten, became worn, torn and sometimes damaged at the corners. Some broke down the middle. Some gauzes were worn at the edges. Normally classes used about ten Bunsen burners and associated equipment. There was a lot of dust in the cupboards, some of it from the equipment stored there. There was more dust where the mats were kept. There was usually a bit of dust when handling the mats and sometimes part of a gauze would crumble away. She called them heatproof mats. When the term "asbestos mats" was put to her, she said it might have been used in 1994 when she started teaching. In 2016 all gauzes were replaced nationally because concerns had been raised in some schools. After Mrs Kerr's diagnosis, mats in her school were tested; one contained asbestos. Following a general asbestos survey all the mats were removed.

[6] Alasdair Kerr spoke to his recollection of conversations over the years with his wife about her work, including mention of asbestos mats, gauzes, and dusty equipment and cupboards at her school in Dalkeith in the 1980s. She talked of frayed and chipped mats. Her clothing was dirty and dusty. She regularly threw her clothes away. Had the budget

been there she would have replaced the gauzes. Conditions in Buckie High School were similar causing frustration on her part as to the state of the equipment. There was no change when she returned to Dalkeith. It was a standing joke that her clothes did not last for long. She helped in the move to the new campus in 2003, including removing and packing the equipment.

The key points in the expert reports as to Mrs Kerr's exposure to asbestos

The report from Laura E Martin, Consulting Forensic Scientist (Occupational Hygiene)

[7] The claimants' solicitors asked Ms Laura Martin to consider Mrs Kerr's cumulative asbestos dose from her employment and whether this materially increased the risk of her disease. She was also requested to comment on her employers' obligations in this regard at the time.

[8] Ms Martin stated that, given the uncertainties, she was unable to determine Mrs Kerr's lifetime occupational dose with any precision. In order to assist the court she estimated that a fairly modest time spent handling the mats and gauzes could incur an overall dose in the range of about 0.001-0.11 of a fibre per millilitre of air over her working years (f/ml years), a method of calculating and expressing cumulative exposure, sometimes termed "dose". While recognising that it was a matter for medical evidence, she offered the opinion that the exposure was not negligible and was likely to have introduced a materially increased risk of Mrs Kerr developing mesothelioma. All the guidance available from the Asbestos Regulations 1969 onwards ought to have led a prudent employer to take all necessary precautions.

[9] Of the three main different types of asbestos fibre, until about 1970 an asbestos insulating board or cement sheet might contain the most carcinogenic, namely crocidolite

fibre (blue); then possibly amosite fibre (brown) until about 1980; and then until asbestos use was banned in 1999, the least potent type, namely chrysotile fibre (white). Crocidolite and amosite are sometimes termed amphiboles. It was difficult to classify the nature of an asbestos product in a gauze; its function was to support a beaker or other receptacle over the burner.

[10] Having discussed how limit values and guidance changed over time, Ms Martin explained how she estimated a range of likely occupational exposure levels for Mrs Kerr. She had regard to data in the 1997 Medical Research Council (MRC) Institute for Health report "Fibrous Materials in the Environment" on airborne levels caused by the abrasion of asbestos boards. Health & Safety Executive (HSE) Technical Data Note 42, 1975, gave figures for the handling of cut pieces of asbestos insulating board. She also had regard to breathing zone readings she had previously taken during the handling of various materials, including asbestos insulating board. Ms Martin was inclined to adopt a typical airborne fibre concentration of 0.01-0.2 f/ml for mats in good condition, rising to 0.5-1.0 f/ml for those in poor condition. She was unable to estimate an equivalent for the handling of gauzes.

[11] If it were accepted that Mrs Kerr was exposed over the full range of about 0.01-1.0 f/ml for, say, half an hour a week over the typical 39 weeks of a school year, her time at Dalkeith High school from 1983-1985 contributed an exposure of 0.0002- 0.02 f/ml years. The equivalent for Buckie High School between 1985 and 1990 would be 0.0005-0.05 f/ml years. Further supply teaching would have made a contribution depending on how many days were worked. Her time at St David's High School over the next four years would contribute asbestos exposure of 0.0004-0.04 f/ml years. Using these figures, the indicative cumulative overall lifetime dose was in the range 0.001-0.11 f/ml years. In the absence of a known safe level of exposure to asbestos, Ms Martin considered this to be a meaningful exposure. She

noted that ranging from rural to urban environments the likely airborne asbestos levels in the general outdoor environment have been estimated at 0.000001-0.0001 f/ml. It is about 0.0005 f/ml for buildings with asbestos materials in good condition. Long-term low-level asbestos exposure is a widely recognised profile of mesothelioma causation.

[12] From 1965 it was known that any exposure to asbestos could cause mesothelioma. Initially Mrs Kerr's employment would have been subject to the 1969 Regulations. From March 1988 the 1987 Regulations required employers to conduct risk assessments and reduce exposure to the lowest practicable level. Non-asbestos equipment was available from at least the early 1990s.

The report from Dr Alan D Jones, Senior Consultant, Senior Physicist and member of the Institute of Occupational Medicine's Research Board

[13] Dr Jones was instructed by agents for Moray Council. He considered it likely that the mats and gauzes in use in many schools at the relevant time contained asbestos. 1976 guidance issued to schools warned of the dangers of asbestos, particularly crocidolite. It indicated that chrysotile was the predominant form in use. Soft asbestos mats and wire gauzes with asbestos centres should not be used in science classes and elsewhere. The guidance stated that hard asbestos products, whether as mats or boards, were much less likely to cause dust, nevertheless hard mats should be replaced wherever possible. From 1986 the advice to educational establishments was to reduce exposure to any form of asbestos to the lowest reasonably practicable level. Materials such as asbestos millboard should be replaced. Asbestos cement was less hazardous but should be replaced if damaged or worn. Reference was made to guidance indicating that particles and fragments from gauzes did not represent an airborne risk.

[14] Mats used with Bunsen burners would be hard mats, not insulation boards. If mats or gauzes did contain asbestos, it was probably chrysotile which was the main type used in asbestos cement. As to likely airborne levels, reference was made to concentration figures recorded in HSE guidance regarding asbestos cement used in construction processes. In Dr Jones' opinion the handling of small heat pads and gauzes was likely to lead to a much lower airborne concentration level, which he estimated at 0.001 f/ml. Ms Martin had regard to readings taken from asbestos insulation board, but asbestos cement had a lower fibre content and the fibres would be bound more tightly in the denser material. He was content with Ms Martin's adoption of a half hour exposure for each of 39 weeks. For Buckie High School's overall contribution, Dr Jones' estimate was about 0.00005 f/ml years. The estimate for Mrs Kerr's combined cumulative occupational exposure was 0.0001 f/ml years. This can be compared with Ms Martin's equivalent suggestion of a range from 0.001-0.11 f/ml years.

[15] As to the level of risk which would flow from his estimated overall dose, reference was made to an influential epidemiological study published in 2000, namely *The Quantitative Risks of Mesothelioma and Lung Cancer in relation to Asbestos Exposure* by John Hodgson and Andrew Darnton of HSE's Epidemiology and Medical Statistics Unit (Ann Occup Hyg vol 44, no 8, pp 565-601). It stated that for an exposure to chrysotile of 0.005 f/ml years, the risk of this causing mesothelioma was "insignificant". At 0.01 f/ml years it was "probably insignificant". As to distinguishing pleural (lung) and peritoneal (abdominal) mesotheliomas, the finding that the risk of the latter is proportional to the square of cumulative exposure meant that the abdominal disease is very unlikely to occur at low dose levels. It was notable that peritoneal mesotheliomas are associated with high levels of exposure to amosite and crocidolite fibres, which are respectively 100 and 500 times more potent than chrysolite.

The report from Professor Stephen R Jones, Professor of Environmental and Occupational Toxicology, University of Central Lancashire

[16] Professor Jones was asked to consider the scientific evidence available as to the risk of peritoneal mesothelioma from exposure to asbestos, and how that would translate into estimates of risk for Mrs Kerr. Again reference was made to the Hodgson and Darnton study and the indications suggesting a non-linear exposure response for peritoneal mesothelioma. For those occupational cohorts exposed to crocidolite or amosite, excess cases of peritoneal mesothelioma were seen mostly in those with higher levels of exposure. There was no good evidence for excesses in those exposed only to chrysotile – only one case of peritoneal mesothelioma. Thus calculations had to be limited to the two more potent types of asbestos. For amosite exposure of 0.1 f/ml years the predicted excess mortality from peritoneal mesothelioma was less than one thousandth of that from pleural mesothelioma; at 0.0001 f/ml years it was less than one millionth. In the absence of sufficient data across a large population with long term follow up, it was assumed that there was no threshold dose below which there was zero risk of developing mesothelioma from asbestos exposure.

[17] A 2023 review of the Hodgson and Darnton study was broadly consistent with the original analysis. Despite including three additional cohorts with substantial chrysotile exposure alone, only one further case of peritoneal mesothelioma was identified. In the 2000 paper the authors explained the extreme rarity of the disease as follows:

> "If the route by which asbestos reaches the peritoneum is from the pleural cavity, it may well be that chrysotile fibres do not survive long enough in body tissues to make the journey in sufficient numbers" (page 587).

In his report Professor Jones explained that chrysotile fibres differ in nature and structure from the other types in a way which may support this thesis.

[18] While subject to well recognised uncertainties, it was considered that the Hodgson and Darnton study provided a wide range of risks that might be attributed to low levels of exposure to asbestos. However, as to the development of peritoneal mesothelioma Professor Jones expressed the view that exposure to chrysotile asbestos did not add to the risk. And for exposure to amosite at levels well below those seen in the occupational cohorts, the additional risks were vanishingly small – negligible for all practical purposes.

[19] Having regard to the estimated exposures spoken to by Ms Martin and Dr Jones, if it was only to chrysotile asbestos the literature suggests no additional risk of the development of peritoneal mesothelioma. If entirely amosite, the risk would be low in the extreme. Professor Jones provided an estimated cumulative asbestos exposure from background sources for Mrs Kerr, which would include all types of asbestos, of 0.046 f/ml years.

Dr Owen Dempsey's report

[20] Dr Dempsey is a consultant respiratory physician. While the bulk of his report is concerned with matters not in dispute, the claimants' solicitors asked him to advise as to whether Mrs Kerr's peritoneal mesothelioma was caused or contributed to by previous exposure to asbestos, and to discuss whether her exposure as a teacher materially increased her risk of developing the disease. Dr Dempsey noted that it is rare for mesothelioma to occur other than in the lining of the lungs. For this reason most studies have concerned pleural mesothelioma. All forms of mesothelioma are strongly associated with industrial pollutants, of which asbestos is the principal carcinogen. Having reviewed Mrs Kerr's medical records, Dr Dempsey found no evidence of alternative causes such as radiation therapy or environmental exposure to other mineral fibres. Thus, on the balance of

probabilities he believed that her occupational exposure resulted in Mrs Kerr developing mesothelioma.

The report of Dr John Moore-Gillon, Consultant Respiratory Physician

[21] Dr Moore-Gillon noted that, as with pleural mesothelioma, there is a strong association between peritoneal mesothelioma and asbestos exposure. As to mesotheliomas in general, the relevant studies indicate that for women, up to one third would have occurred even if asbestos had never been imported into this country. For chrysotile cumulative exposure at the upper limit of Ms Martin's estimate (which is 100 times greater than her lower limit, and 1,000 times greater than that of Dr Jones) Hodgson and Darnton described it as "probably insignificant" (table 11). By this they meant a lifetime risk of 1 in 100,000, which equates to an annual risk of around 1 in 5 million. HSE describes annual risks of 1 in 1 million as being "generally regarded as insignificant and adequately controlled". If one adopts Ms Martin's lower limit, or that of Dr Jones, the annual risks are respectively 1 in 500 million and 1 in 5,000 million.

[22] Dr Moore-Gillon recognised the potential limitations of the Hodgson and Darnton risk estimates. There are uncertainties as to Mrs Kerr's actual occupational exposure, and the validity of Hodgson and Darnton's work depends on the rigour of the original studies upon which it is based. And as discussed by the Working Group on Action to Control Chemicals, February 2011, caution is required since Hodgson and Darnton had to extrapolate beyond the limits of the observed data pertaining to risk. Furthermore, while it is conventional to use the authors' best estimate of risk range for any given exposure, the actual risk might be higher or lower. Nonetheless Hodgson and Darnton is the best

available tool for assessing levels of risk of mesothelioma (and lung cancer) associated with asbestos exposure. Dr Moore-Gillon described it as an authoritative meta-analysis.

[23] At very low exposure levels the risk of peritoneal mesothelioma is far lower than that for pleural mesothelioma. Thus the Hodgson and Darnton figures, which include both types, very markedly over-estimate the chance of developing the rarer tumour, which accounts for only about 10-15% of mesotheliomas as a whole. The numbers associated with chrysotile exposure were very low thus preventing statistical analysis. The data did demonstrate that at high levels of amphibole exposure peritoneal mesotheliomas make up a greater proportion of the total than at low exposures.

[24] At the low levels of exposure figured here, peritoneal mesotheliomas would make up far less than 10/15% of the total, however Dr Moore-Gillon decided to adopt what he described as "the very cautious" estimate that the relevant risk is one tenth of the overall figures in Hodgson and Darnton. On that basis, to present a medically significant risk of developing mesothelioma Mrs Kerr's exposure during her teaching career would require to be 50 times greater than Ms Martin's upper figure. (The reference to 10 times in the report was an error which was corrected during evidence.) By "medically insignificant" Dr Moore-Gillon had in mind a dose which a medical practitioner would advise a patient not to worry about. The top of the range figure gives an annual risk of mesotheliomas in general of less than 1 in 5 million, and the cautious translation to the risk of peritoneal mesotheliomas is 1 in 50 million. This is 3,000 times lower than HSE estimates of the annual risk of being killed in a road accident; 12,500 times lower than the annual risk of death from all types of accidents and all other external causes; a third of the risk of being killed by lightning; and equivalent to the risk of being killed by an accident during a single train journey.

The report of Professor Pat Price, Consultant Oncologist

[25] Midlothian Council asked Professor Price for advice on diagnosis and causation from an oncological perspective. Mrs Kerr suffered from epithelioid malignant peritoneal mesothelioma which is an extremely rare condition. The link with exposure to asbestos is weaker for peritoneal mesothelioma, with a third to a half of patients reporting no known history. Historically these diagnoses have been associated with heavy amphibole commercial exposure. Such exposures are now uncommon and the current epidemiological evidence suggests that a much smaller fraction of such tumours in men are related to asbestos, and very few in women. While asbestos exposure is a recognised risk factor, other possible causes have been identified, including exposure to mineral fibres, therapeutic radiation, chronic inflammation, and familial genetic abnormalities. However the vast majority of these tumours are idiopathic, meaning the cause is unknown.

[26] Reference was made to a paper (Attanoos, Churg et al, 2018) which presents evidence supporting a background rate of spontaneous mesotheliomas (non-asbestos related) arising particularly in women. Professor Price expressed the view that since the paper indicated that fewer than 50% of epithelioid mesotheliomas in females are related to asbestos exposure, the probability is that Mrs Kerr's tumour was unrelated to any such exposure.

A summary of additional matters raised in the oral evidence of the expert witnesses

Ms Laura Martin

[27] When giving evidence all of the experts adopted their reports and were examined and cross-examined on them. Ms Martin noted that while the use of amphibole asbestos

was wound down from the mid-1970s and prohibited in 1985, chrysotile was popular after 1970 and continued in use until the late 1990s. Older products could continue to be used for many years. A Dalkeith High School report in 1992 recorded that the previous year Bunsen tiles, type white (chrysotile), had been removed from use. A 2003 survey of equipment at Kirkwall High School reported that several asbestos cement Bunsen burner mats were in use. They were to be removed and replaced with a non-asbestos equivalent. Such would have been available from the early 1980s onwards. A 2022 survey at St David's RC School, Dalkeith, has a photograph of a bag containing science mats. No asbestos was detected in the one mat sampled, but nonetheless they were all to be treated as positive. There is also a photograph of a mat on a workbench with a fibre identification of "chrysotile".

[28] In the 1980s, mats might still have white fibres. For asbestos mats the possibilities were the use of asbestos cement, millboard or asbestos insulation board. The majority would be asbestos cement. Millboard was too soft, but some might be asbestos insulation board which would be more prone to fibre release. One would need to be rough with asbestos cement boards for there to be asbestos dispersal. Ms Martin had carried out a lot of airborne concentration monitoring, including with products which would behave in a similar manner to asbestos cement and asbestos insulation board mats. Her estimated range of possible occupational exposure covered, at the lower end, the use of asbestos cement mats in good condition; at the upper end the assumption is of entirely asbestos insulation board mats, all in poor condition.

[29] In a supplementary report Ms Martin questioned Professor Jones' methodology when calculating Mrs Kerr's overall background asbestos cumulative exposure. She used a divider of the total number of hours in a calendar year rather than those typically spent

working. As a result her figure is 0.01 f/ml years; Professor Jones' figure was 0.046 f/ml years. However she did not know why it was thought that the exercise might be useful. She had not previously seen it deployed.

[30] When asked about the causative impact of different doses, Ms Martin deferred to medical opinion. Historically there has been an attitude that chrysotile is safe, but in her view there remains an inherent risk. 1976 guidance from the Dept of Education and Science suggested that chrysotile was predominant, however amosite could be used up to 1980. Ms Martin proceeds on the basis that any exposure to asbestos may be associated with the development of mesothelioma. She accepted that her range of possible occupational exposures was subject to a number of assumptions and uncertainties, including that asbestos was involved at all; if so, the type; that dust was created and inhaled; and as to the duration and frequency of exposure. The 20 minute toothbrush abrasion data she used from the MRC report was the closest equivalent activity she could find for estimating an airborne concentration which Mrs Kerr might have experienced.

Dr Alan D Jones

[31] After guidance issued in 1976, replacements mats were likely to be non-asbestos products. Earlier advice had been to replace soft mats with hard (asbestos cement) mats in which the main fibre was chrysotile. There would be no need to use amphiboles in such thin mats. If Mrs Kerr handled asbestos insulation board mats he could understand Ms Martin's estimates. However, he doubted that such products would be used as they were not suitable for the purpose, and were more expensive. Millboard was very unlikely. The data used by Ms Martin derived from the MRC report was compiled from tests in a closed box. This was a good method for comparing different materials but would elevate airborne

concentrations. Some of the examined items were weathered, but mats will not become weathered. Dr Jones supported Professor Jones' method for calculating cumulative background exposure.

Professor Stephen Jones

[32] Chrysotile fibres are said by some to be relatively soluble and thus are not retained in the lung for as long as the more carcinogenic types. However there is no pure chrysotile; it will always have some naturally occurring other fibres. There is no good evidence that exposure to chrysotile causes peritoneal mesothelioma. This is perhaps because the white fibres are not readily transportable to the abdomen. However, as with ionising radiation, the conventional assumption is that there is no safe threshold for asbestos exposure. There are likely to be a number of other causes of mesotheliomas, and it is generally accepted that cancers can happen without the intervention of an outside agency.

Dr Owen Dempsey

[33] There is no full understanding as to how asbestos fibres cause mesothelioma. It is thought that after inhalation they cause cellular damage and resultant abnormalities progress to the cancer. Some individuals may have a genetic susceptibility. It is unclear how fibres might come to damage the abdomen, perhaps through ingestion, or becoming trapped in the lymphatic system. Reference was made to a 2021 paper from H Richard Alexander Jr, MD, on various matters concerning malignant peritoneal mesothelioma, including epidemiology and risk factors. It notes that the link between exposure to asbestos and this disease is less strong than it is for pleural mesothelioma, particularly among women. Chrysotile is one of the safer varieties, but in Dr Dempsey's view it still presents

risks. Reference was made to a 2020 paper concerning Italian miners exposed only to white asbestos. (It has not been produced.) Any dose, however small, might cause mesothelioma, including of the peritoneum. All asbestos fibres are carcinogens capable of causing cancer. There may well be a mechanism whereby white fibres move from the lung to the abdomen.

[34] Dr Dempsey noted that we have no tissue pathology for Mrs Kerr; it might have revealed whether fibres were present in her abdomen. He considered Professor Price's reference to "idiopathic" cancers to be a route to lazy thinking. Every condition has a cause, and asbestos is a recognised risk factor. Dr Moore-Gillon's approach was very epidemiology slanted. Dr Dempsey deals with risk every day, for example, are the benefits of an X-ray worth running the risk of it causing harm? He cannot say whether a risk is significant or insignificant. A statistician would say there is no risk in passing a piece of asbestos around, but he would not touch it.

Dr John Moore-Gillon

[35] There was nothing of relevance in Mrs Kerr's medical history. Dr Moore-Gillon described how cancers develop through faulty mutations at a cellular level. These are occurring in very large numbers all the time in all of us, but the vast majority are dealt with by the body's immune system. Unfortunately very occasionally this does not happen and sometimes results in a malignancy. The abdomen is relatively large so there may be greater scope for non-asbestos related problems. It is thought that asbestos increases the number of faulty mutations thereby creating a greater risk of some not being caught by the body's defences.

[36] With regard to pleural mesotheliomas, chrysotile is less potent because it is cleared more rapidly from the body. Physical and chemical differences may also contribute to the

greater risk posed by amosite, and even more so by crocidolite. The level of risk is much lower for peritoneal mesotheliomas at low levels of exposure. Fibres have to reach the diaphragm, and at high levels this is more likely since more will be ingested. For example in the 1950s and 1960s insulation laggers said they could taste it.

[37] If the Hodgson and Darnton study is correct, and it seems reliable, there is no demonstrable risk of peritoneal mesothelioma at occupational levels of chrysotile exposure. Chrysotile is much less carcinogenic; in his view the risk is exceptionally small at any exposure level. Ms Martin's estimated levels of exposure were very low, and "twiddling at the edges" would have no impact for present purposes. It would be irrelevant to compare them with environmental exposure (as to which Dr Moore-Gillon supported Professor Jones' methodology). Dr Moore-Gillon would regard a level of risk as insignificant if a reasonable person would not worry about it. He observed that simply by entering the court building he had increased his risk. He would not have advised Mrs Kerr that she was under an increased risk. Such as it was, it was much lower than cooking by gas.

Professor Pat Price

[38] There are only about 40 cases of peritoneal mesothelioma each year in the UK. She has not seen one, but how tumours grow and behave was her main research interest. She described how all tumours in any tissue of the body develop from faulty mutations at the molecular level with cells becoming abnormal. This usually occurs from a source of inflammation or injury which sets up a chain reaction that damages the DNA. If not repaired by the body's natural defences, this can cause a cancer. It is now known that inflammation is really important in causing cancers. It is the common thread.

[39] The extreme rarity of peritoneal mesothelioma in the cohorts exposed to chrysotile examined by Hodgson and Darnton might be explained by fibres not surviving in the body for long enough for sufficient numbers to make the journey from the lungs to the abdomen. There is no direct anatomical connection. It seems likely that some individuals will have an individual susceptibility to asbestos exposure.

A summary of the parties' submissions

Mr Kerr and family

[40] Mrs Kerr's evidence as to occupational exposure to respirable asbestos should be accepted, as should that of Mrs Young and Mr Kerr. While the proportions cannot be determined, many of the mats and gauzes she handled contained asbestos. Ms Martin accepted that the majority of asbestos mats would be made from asbestos cement, but asbestos insulation board was also used. It is likely that in the 1980s most mats and gauzes contained asbestos. It is not possible to determine the number of asbestos mats and gauzes still in use by Mrs Kerr thereafter, but her evidence was that they were retained till not usable. Thus Mrs Kerr was exposed to chrysotile, and also to some level of amosite and of tremolite (another type of asbestos fibre). Professor Jones said chrysotile was frequently contaminated with the latter. Dr Jones conceded in cross that mats might contain amosite.

[41] It is accepted that no more than indicative levels of Mrs Kerr's exposure can be given, however it is only necessary to establish a material increase in risk, not specific measurements, see *Cox* v *Rolls Royce Industrial Power (India) Ltd*, [2007] EWCA Civ 1189, at paragraph 21. The practical difficulties militate against requiring reliable evidence as to actual exposure levels. Ms Martin's estimated range can be accepted. Dr Jones supplied less by way of an explanation for his figure. He adopted an arbitrary approach designed to

produce a low level. Ms Martin's methodology for calculating cumulative background exposure should be preferred, but the exercise forms no part of the claimants' case.

[42] As in *Fairchild* v *Glenhaven Funeral Services Ltd*, [2003] 1 AC 32, it is not possible to prove what caused Mrs Kerr's cancer. However her employers' breaches of duty in exposing her to respirable asbestos fibres was conduct capable of causing her an injury, including that which she sustained. The common law test for foreseeability is whether the employer should have appreciated that the exposure created a material risk of asbestos-related injury, *Bussey* v *Anglian Heating Ltd*, [2018] EWCA 243, at paragraphs 20, 63 and 65. Reference was also made to *Jeromson* v *Shell Tankers (UK) Ltd*, [2001] ICR 1223, at paragraphs 33, 34, 51 and 52. By the mid-1970s the risk of inhalation of asbestos fibres was known to schools, and that heat mats and wire gauzes contained asbestos and so should be replaced if damaged or worn. Reference was made to the relevant statutory duties in the 1987 and 2002 Regulations, including regulation 8(1) in the former which required every employer to prevent the exposure of employees to asbestos so far as was reasonably practicable.

[43] The rest of counsel's submissions focussed on the issue of causation and what was described as almost an asbestos jurisprudence driven by policy considerations. The test is as stated by Lord Phillips in *Sienkewicz* at paragraph 1:

> "… when a victim contracts mesothelioma each person who has in breach of duty been responsible for exposing the victim to a significant quantity of asbestos dust and thus creating a 'material increase in risk' of the victim contracting the disease will be held to be jointly and severally liable for causing the disease."

A special rule of causation has been devised because of ignorance as to the biological cause of the disease. The loss in cases of wrongful exposure to asbestos is that of contributing to the risk of developing the cancer. It is not a matter of whether the exposure has caused it.

The term "material" in the test excludes a *de minimis* increase in risk, a matter for a judge to assess on the particular facts of a case. Any real risk of a fatal injury, even if statistically small, is significant, *Bussey* at paragraph 63. There is no requirement to make a comparison with any other source of exposure, *Williams* v *The University of Birmingham,* [2012] PIQR P4 (CA), at paragraphs 71-72.

[44] The circumstances in which the special rule of causation, sometimes called the *Fairchild* exception, applies were summarised in *Sanderson* v *Hull,* [2009] PIQR P7 (CA), at paragraph 53. It must be shown that in the current state of scientific knowledge it is impossible to prove exactly how the injury was caused. The defendant's conduct must have been capable of causing the injury and have materially increased the risk of it. The claimant must prove that the injury was caused by the eventuation of the kind of risk created by his conduct, and it must be caused by the same agency as was involved in the defendant's wrongdoing (or by an agency which operates in a similar way).

[45] Counsel for the claimants candidly recognised that these last two requirements presented a difficulty for him. Indeed the suggested findings in fact in his written submissions did not include a finding that asbestos exposure caused Mrs Kerr's cancer. (Had they done so, I would not have made such a finding; the evidence does not permit it.) He contended that it is not necessary to establish that asbestos exposure played a causative role in the normal or traditional sense. As in *Fairchild* and *Sienkiewicz*, it is enough to show that the occupational exposure was a possible cause and materially increased the risk of developing mesothelioma.

[46] The factors relied upon in this regard can be summarised as follows. The issue should not be decided by reference to the epidemiology. Its limitations were recognised in the evidence. Given the rarity of the disease there are few studies considering peritoneal

mesothelioma and asbestos exposure. There is substantial uncertainty as to Mrs Kerr's actual exposure levels. Individual susceptibilities may be relevant. While we do not know if she had such, we do know that Mrs Kerr developed the disease having been wrongfully exposed by her employers over her teaching career, principally to chrysotile, but with the possibility of some amosite. Breach of duty and causation are distinct matters, but they are linked. The period of exposure is consistent with the known latency period. It is accepted that there is a strong association between asbestos exposure and peritoneal mesothelioma. There is at least some evidence that chrysotile exposure might lead to the development of peritoneal mesothelioma. There is no known safe level. Risk increases with dose. Any medical advice would have been to avoid working with the mats and gauzes. Mrs Kerr's occupational exposure was orders of magnitude higher than background levels. No other source dwarfs her occupational dose. There is no evidence of exposure to other agents implicated in peritoneal mesothelioma.

The submissions for Midlothian Council

[47] There is a world of difference between this case and other asbestos claims which have come before the courts, including the low level of the alleged exposure and the type of the disease. The claimants have failed to prove (1) the extent of any exposure, (2) negligence, and (3) that any exposure caused Mrs Kerr's illness. The attempt to extend the *Fairchild* exception to this case is flawed. In that case there was no issue as to whether asbestos caused the injury. There can be cases where the evidence adduced is too weak to prove anything to an appropriate standard, and judges should not allow the harrowing nature of the illness and its devastating consequences to lead to a lax approach to proof of the essential elements, *Sienkiewicz* at paragraphs 166 and 193.

[48] By 1999 it is probable that Mrs Kerr was using asbestos free equipment. Removal instructions had been in place since the mid-1970s. Mrs Young did not speak to the use of asbestos equipment. The 1992 survey recorded that asbestos Bunsen mats had been removed the previous year. Any exposure was in Mrs Kerr's first period of employment in Dalkeith when she was likely to have used both asbestos and non-asbestos mats. The latter were available by the mid-1970s. The evidence as to dust on hands and clothes is consistent with the use of cement mats. Soft mats were prohibited from an early stage. Any exposure was likely to be to white asbestos. Dr Jones' evidence was that tremolite contamination depended on where the chrysotile had been mined, and would in any event be a fraction of one per cent.

[49] Given all the uncertainties, any attempt to estimate an airborne concentration level is no more than speculation. On any view, any cumulative occupational exposure to respirable asbestos was small, perhaps as per Dr Jones' estimate of about 0.0001 f/ml years. Asbestos cement is dense and does not readily release fibres.

[50] Given the paucity of the evidence, the court should not make a finding of negligence. There was guidance in the 1970s and 1980s which included the replacement of damaged mats. The evidence suggests that the guidance was followed. In any event the use of asbestos cement mats did not create a significant risk of an asbestos related injury. It has not been established that any negligent exposure was causative of Mrs Kerr's disease.

[51] As to the attempt to use an exception to the usual need for proof of factual causation by relying on the creation of a risk of injury, this special rule was first expressed in *Fairchild* where Lord Bingham made it clear that a necessary first stage is to establish that the mesothelioma was caused by the inhalation of asbestos fibres, see paragraph 2. If and when that is done, anyone who created a material increase in the risk of that injury bears legal

responsibility. Here the claimants have not established that first stage. On the contrary the evidence indicates that low level exposure to chrysotile does not cause peritoneal mesothelioma. The most likely explanation is an endogenous (or spontaneous) cancer caused by mutations and errors at a cellular level uninfluenced by an outside agency. Reference was made to *Heneghan* v *Manchester Dry Docks Ltd and others*, [2016] 1 WLR 2036, at paragraphs 8 and 9.

The submissions for Moray Council

[52] There is doubt in the evidence as to whether Mrs Kerr's employment as a teacher between 1992 and 1995 was on a supply or a full-time basis, and if the former, for how many days. There are contradictions in Mrs Kerr's two affidavits, for example as to whether gauzes were handled. Mr Kerr's evidence should be treated with caution. The evidence of Dr Moore-Gillon and Dr Jones can be accepted. Both were well-qualified and impressive experts in their field. Ms Martin's experience is primarily in surveys and monitoring. Dr Dempsey's evidence was vague and of limited assistance. The only paper he specifically referred to in evidence has not been produced.

[53] As to asbestos exposure, cement has relatively low fibre content and high density thus reducing the likelihood of fibre release. There was no evidence that asbestos insulation boards were used. Such was inherently unlikely. On the evidence, if asbestos mats were used, they would likely be made of chrysotile. Any contaminant would be taken into account in the various epidemiological studies.

[54] As to risk, cumulative exposure is the most important factor. Ms Martin over-estimated her range. Her top end is an extreme scenario proceeding on a worst case for which there is no evidence. And it is based on test results from an exercise which bears no

relation to the circumstances here, including reliance on abrasion of an old and weathered product. Dr Jones' approach was logical and preferable. Midlothian Council's submissions on breach of duty were adopted.

[55] As to causation, *Fairchild* was a case where any cause other than the inhalation of asbestos could be discounted, but it could not be proved which defendant's wrongdoing caused the mesothelioma. Rather than the claim fail, it was decided that any employer who breached a duty to protect against asbestos exposure and thereby materially increased the risk of harm should bear responsibility and provide compensation. In the present case it cannot be said, and is not said, that Mrs Kerr's disease was caused by any breach of duty on the part of either or both employers.

[56] As to the epidemiological evidence, there is no good objection to its use in helping as to whether asbestos caused the injury, see *Heneghan*, paragraph 8; *Sienkiewicz*, paragraphs 163 and 172. As several witnesses explained, it does not support an association between chrysotile exposure, particularly at low exposures, and peritoneal mesothelioma. Even with pleural mesothelioma the link is weak. The Hodgson and Darnton analysis questions the association between low levels of chrysotile exposure and all mesotheliomas. It has not been established that any likely level of asbestos exposure caused Mrs Kerr's disease.

[57] If the court decides to apply the *Fairchild* exception, it addresses not the actual injury, but rather the creation of a risk of injury, and it must be shown to be material. In recent cases south of the border various methods have been suggested for assessing materiality. The approach discussed in *Bannister* v *Freemans Public Ltd Co*, [2020] EWHC 1256 (QB), is attractive. It involves putting all the relevant evidence into the mix, including the epidemiology, in which event this court should hold that any occupational asbestos

exposure did not create a material increase in Mrs Kerr's risk of developing peritoneal mesothelioma.

Discussion

[58] As will become apparent, I take the view that the *Fairchild* exception was not designed for a case where it is neither admitted nor established that asbestos exposure caused the injury. Nonetheless, before elaborating on this, it is appropriate that I address the evidential issues in dispute.

Mrs Kerr's exposure to asbestos at work

[59] I have little difficulty in concluding that it is likely that Mrs Kerr did handle asbestos Bunsen mats (in all probability made of asbestos cement) and gauzes, and this particularly during her earlier years as a teacher. Even if one assumes that guidance was followed, it would not mean that all such products were quickly removed. The 1992 Dalkeith High School report recorded that white asbestos mats were in use till the previous year, and in 2003 they were found in Kirkwall High School. In 2022 a chrysotile mat was photographed on a workbench in St David's RC School, Dalkeith. Mrs Young said that thereafter all mats were removed. In her affidavit Mrs Kerr spoke of "white fibrous asbestos" mats. As she and Mrs Young said, one would expect some to be in relatively poor condition and thus more prone to fibre release when moved, handled and stored in cupboards.

[60] It is much more difficult to assess the amount of respirable fibres, likely to be chrysotile, to which Mrs Kerr was exposed. Indeed Ms Martin as good as accepted that the imponderables are such that it is not possible to do so. Asbestos cement mats were more

resistant to fibre dispersal. The indicative range Ms Martin offered stretched from her best case with all mats made of asbestos cement and in good order, to the worst scenario, namely all mats made of the more porous asbestos insulation board and in poor condition. She was unable to offer anything regarding a contribution from the gauzes. I agree with the submission that the evidence provides no support for the worst scenario, or indeed anything approaching it.

[61] There are obvious limitations in the utility of the data from the test results relied on by both Ms Martin and Dr Jones. In reality both witnesses were offering a judgement based on their general experience rather than anything which can be objectively justified. All that can be said with any confidence is that in comparison with levels commonly discussed in claims of this kind, Mrs Kerr's exposures to respirable asbestos and overall dose were of a very low level. If one was limiting oneself to the cumulative range offered by Ms Martin, it was likely to be at the lower end, namely 0.001 f/ml years. It is also of note that it was probably to chrysotile fibres, or at least predominantly chrysotile, which has by far the lowest potency for malignancies.

[62] In the submissions there was little reference to the estimates (they can be no more than that) of Mrs Kerr's cumulative exposure to background levels of all types of asbestos fibres. As to the dispute as to how this should be calculated, the clear weight of the expert evidence favoured Professor Jones' route to 0.046 f/ml years. If and in so far as it may be of any importance, I accept that figure.

Is the Fairchild exception test met; did Mrs Kerr's occupational exposure to asbestos create a material risk of developing peritoneal mesothelioma?

[63] The bulk of Dr Dempsey's report dealt with matters of diagnosis, prognosis (he was instructed while Mrs Kerr was alive), and medical history. He also provided a supplementary report on cause of death and life expectancy absent the disease. His expertise and experience is in lung disease, and on such matters he was on solid ground. Entirely understandably, he seemed less assured on the specific medical/legal issues in dispute, particularly in respect of Mrs Kerr's abdominal cancer. The view on causation set out in his report was based on the strong association between asbestos exposure in general and all forms of mesothelioma, of which he accepted the bulk damage the lungs. Ultimately it was not pressed by counsel for the claimants. The H Richard Alexander Jr paper appended to Dr Dempsey's report states that "in contrast to pleural mesothelioma, chrysotile has not been convincingly shown to cause (malignant peritoneal mesothelioma)". Dr Dempsey did not address such matters in his report. Indeed he expressly invited "more expert comment" from an oncologist on this rare condition (paragraph 10.5.3).

[64] In his oral evidence Dr Dempsey was invited to go beyond his report. He mentioned a paper concerning Italian miners, however it has not been produced and thus was not put to the other relevant witnesses. In essence Dr Dempsey said that there is no known safe threshold and that all asbestos fibres are capable of causing cancer. His zero tolerance to any unnecessary risk, for example touching asbestos, is understandable, but the legal test of "material risk" does not allow the court to adopt it. As will become apparent, I do not share his criticism of reliance on the epidemiology, nor his view that Professor Price was guilty of lazy thinking. I have no doubt that Dr Dempsey is an excellent consultant respiratory physician, and he gave his evidence in an engaging and attractive manner. I am sure he was

doing his best to assist the court, however I suspect he would acknowledge that on the key issues he was being asked to stray into areas where others were better qualified.

[65] When asked about causation, Laura Martin stated that she deferred to medical opinion. In her report, given that there is no known safe level, she described her range of cumulative occupational exposure as "meaningful", which is not a particularly helpful phrase. She acknowledged that historically chrysotile has been regarded as safe; however in her view there remains an inherent risk. As someone predominantly employed to monitor and advise as to safety practices, it is understandable that Ms Martin proceeds on the assumption that any exposure to asbestos might be associated with the development of mesothelioma. That is a sensible precautionary approach, but as she would no doubt recognise, it is of little direct assistance on whether in fact Mrs Kerr's occupational exposure created a material risk of her developing peritoneal mesothelioma.

[66] Professor Price provided the suggested oncological perspective. It was based on many years practice as a consultant oncologist, currently visiting Professor at Imperial College, London, and a long-standing research interest in cancer biology. She is a past President of the British Oncological Association, has published over 300 research papers, and is co-editor of the standard UK textbook on the treatment of cancer. She does not claim expertise in peritoneal mesothelioma (given its rarity perhaps no one does), but states that she is fully familiar with its biology and management.

[67] Professor Price's evidence was summarised earlier. She spoke to the epidemiology which suggests that Mrs Kerr's condition was probably not caused by asbestos exposure. While there are a number of other possible causes, in the vast majority of cases the trigger is not identified. Professor Price gave a possible explanation for the extreme rarity of peritoneal mesotheliomas in cohorts exposed to chrysotile, and described how it is now

thought that a source of inflammation or injury may be the most common explanation for the abnormal changes at a cellular level which ultimately lead to a malignancy. I have identified no good reason to question her evidence. Its importance is in rebutting any notion that any exposure to asbestos fibres from the equipment handled by Mrs Kerr was likely to be the explanation for her abdominal cancer.

[68] Like Dr Dempsey, Dr Moore-Gillon is a consultant respiratory physician and therefore predominantly a specialist in lung conditions. However, even leaving aside his distinguished catalogue of clinical, academic and regulatory practice, his extensive involvement in medico-legal matters, including in many if not most of the leading cases, has given him a detailed appreciation of the issues to be addressed in the unusual circumstances of the present claim. His ability to address complex matters in a clear and comprehensible way both in writing and in oral evidence was as impressive as his CV.

[69] Dr Moore-Gillon's evidence pointed up the importance of the particular feature of this case, namely a rare condition not shown to be associated with low levels of exposure to asbestos, and certainly not to chrysotile. He confirmed that there was nothing of relevance in Mrs Kerr's medical notes, and that it cannot be said that she had a particular susceptibility. The limitations inherent in the Hodgson and Darnton study were recognised and explained, but it remains the best tool available when trying to assist the court. He adopted a cautious approach likely to over-estimate the relevant risks. His description of how cancers occur was particularly illuminating. (For a detailed exposition see the annex to Lord Phillips' judgment in *Sienkiewicz*.) While asbestos can increase the number of faulty mutations, many occur otherwise or for no apparent reason.

[70] Dr Moore-Gillon was able to offer rational explanations for (a) the epidemiological data, for example, as to why peritoneal mesothelioma predominated in cohorts involved in

large commercial exposure, (b) for the almost non-existent evidence of it at low dose levels, and (c) for the much higher potency of amphibole fibres. The *Fairchild* exception depends on proof of a material increase in risk of harm. Dr Moore-Gillon persuasively explained why in his view, at the very low levels of exposure involved here, and even if one adopted Ms Martin's upper range figure, any risks were so small that a reasonable person would have no concerns. I accept all of his evidence.

[71] The evidence from Dr Alan Jones was broadly consistent with Dr Moore-Gillon's analysis and the conclusion that if there was any risk, it was insignificant. The same can be said of that from Professor Stephen Jones. Both effectively excluded any causative role from such a low occupational exposure to chrysotile.

[72] It was submitted that little weight should be placed on the epidemiological evidence. I could understand that proposition had there been anything, for example from tissue pathology, to suggest that Mrs Kerr's disease was asbestos related, or that she had a particular susceptibility. I agree with Dr Moore-Gillon that, recognising its limitations, absent such direct evidence, this source can be used. If one takes it away, there is little left beyond an assertion that one should assume that any occupational exposure presents a material risk.

[73] The approach I am taking is similar to that adopted by the deputy judge in *Bannister*, see paragraphs 185-186. In *Sienkewicz* Lord Nicholls of Birkenhead defended the potential role of epidemiological evidence given that matters are determined by proof on a balance of probabilities. Some of the justices were more cautious. However, the context of the discussion was whether in a "single exposure" case, where the only competing explanation was environmental asbestos fibres, a claimant could and should be required to prove that the wrongful exposure caused the harm by showing that it doubled his risk of the disease.

The decision was to affirm the test for liability of creating a material increase in the risk, and for Lord Rodger of Earlsferry it was obvious that "epidemiology is likely to lie behind much of the evidence on which a court determines whether an exposure has materially increased the risk of the claimant developing a disease", paragraph 163. Lady Hale thought that when deciding a case most judges would put "everything into the mix", including statistical probabilities, see paragraph 172. It can be noted that in the lung cancer case of *Heneghan*, the Court of Appeal had no difficulty with epidemiological evidence establishing that the injury was caused by asbestos on the "doubles the risk" test. This was described as the "what" question, as opposed to the "who" question; the latter arising when the issue is which contributor's exposure was responsible for the disease (paragraphs 8 and 9).

[74] The Hodgson and Darnton study is eloquent of the difficulty in establishing any link between predominantly chrysotile exposure and the development of peritoneal mesothelioma, let alone at such a low dose, and I consider that the witnesses were well-founded in relying upon this material. There is no inconsistency with the evidence that the assumption is that there is no safe level. That is a wise precautionary approach for employers and others to adopt, but it does not follow that, whatever the specific circumstances, any exposure to asbestos will lead to a material risk in terms of the *Fairchild* exception.

[75] It was submitted that medical advice would have been to avoid working with mats and gauzes. Perhaps if asked, some doctors, for example Dr Dempsey, would have adopted this approach, but it is unlikely that it would have been derived from any considered assessment of the actual risk involved. In summary, I accept the evidence to the effect that if Mrs Kerr's work as a teacher exposed her to any risk of developing peritoneal mesothelioma, a doubtful proposition in itself, the risk was so small as to merit disregard

(sometimes referred to as being *de minimis*). In other words it was not material. In these circumstances there can be no liability, see *Prescott* v *The University of St Andrews*, [2016] CSOH 3, per Lord Pentland at paragraph 64. It also follows that even if the *Fairchild* exception could be in play, it would not be applied in the claimants' favour.

[76] The issue of breaches of common law and statutory duties hardly figured in the submissions, I think on the footing that they would turn on whether a material risk had been demonstrated. And there was nothing said as to any need to make an apportionment between the two employers should damages be awarded.

Is the Fairchild exception designed for a case such as this?

[77] Although now not necessary for the decision, I will offer some observations on whether the *Fairchild* exception is designed for a case such as the present. To date it has been applied only where non-asbestos related causes of the injury can be discounted. In such cases liability is established against anyone who in breach of duty has exposed a claimant to asbestos fibres and has thereby materially increased the risk of the development of mesothelioma ("materially" meaning more than minimally), see *Sienkiewicz.* If that is demonstrated, it is not necessary to prove that the exposure caused or contributed to the disease. The injury must result from the kind of risk created by the employer's wrongdoing (or one operating in the same way), see *Fairchild* at paragraph 170, and *Sanderson* v *Hull*, paragraph 53. If the exception cannot be used, factual causation must be established in the normal manner.

[78] The special rule devised in *Fairchild* is justified on grounds of policy and fairness by the inability of current medical science to identify which exposure to asbestos, whether environmental or occupational, caused the disease to take hold, rendering proof on

traditional principles impossible for claimants. Thus it is understandable that it has not been applied where asbestos exposure is no more than one of a number of potential explanations, though no doubt that will be rare in claims involving pleural mesothelioma, the type of disease with which the leading cases are concerned. The peculiarity of the present case is that it relates to the rarer condition of peritoneal (abdominal) mesothelioma where the association with asbestos exposure, especially at low doses such as that involved here, is much weaker.

[79] Counsel for the claimants accepted, in my view correctly, that the need to show that Mrs Kerr's peritoneal mesothelioma was linked to asbestos exposure presents a difficulty for him. It was not entirely clear how he proposed to deal with it. As I understood it, the submission came to this: if Mrs Kerr's occupational exposure created a material risk of peritoneal mesothelioma, the special rule should be available if the true cause of the disease cannot be known. Whether this is such a case was not a specific topic of discussion in the evidence. But even if it had been established, I see no proper basis for advancing the law in this direction. Whatever else, it would be hard to reconcile with the decision in the non-asbestos case of *Wilsher* v *Essex Area Health Authority*, [1998] AC 1074.

[80] Of course *Fairchild* was decided after *Wilsher*, and it set out the material increase in the risk of harm test. However the context was cases where the only impediment to success for the claimants was the inability to prove which defendant was responsible for the asbestos exposure which caused the lung disease. Given the particular nature of the illness, sometimes described as "indivisible", exposures before and after the damage is done make no difference to the outcome. Lord Bingham made it clear that the necessary pre-conditions for the application of the special rule included the exclusion of other possible causes of the

pleural mesothelioma. (He mentioned a requirement to rule out environmental asbestos exposure, but this has been removed in subsequent decisions.)

[81] In *Barker* v *Corus UK Ltd*, [2006] 2 AC 572, it was held that the *Fairchild* exception could be applied when part of the overall asbestos exposure was not wrongful, but it was confirmed that the other exposure(s) must involve the same risk or something which operates in the same way, see Lord Hoffman at paragraphs 17 and 23-24. Thus it would not be enough if all that was proved was that a lung cancer was caused by either asbestos or smoking. A translation to the present case would be the possibility of non-asbestos related faulty genetic mutations causing abdominal mesothelioma, variously described as endogenous, spontaneous or idiopathic.

[82] In *Barker* Lord Scott of Foscote asked whether the special rule can apply where more than one injurious agent subjected the victim to a risk of the outcome but it cannot be determined which was responsible, see paragraph 58. His answer was no; *Fairchild* "established a narrow exception to the causation requirements applicable to single agent cases", paragraph 64. So far as mesothelioma is concerned, Lord Walker of Gestingthorpe limited the exception to cancers caused by the inhalation of asbestos fibres, paragraph 114. Reference can also be made to *Williams* v *Bermuda Hospitals Board*, [2016] AC 888, at paragraph 40 where the Board of the Privy Council distinguished "disparate factors" cases from those where the injury was caused by a single known process.

[83] In *Sienkewicz*, Lord Phillips commented that the *Fairchild* exception case law equated "materially increasing the risk" with "contributing to the cause" in circumstances which he described as "specified and limited", see paragraph 70. Later he affirmed that the special rule should be confined to cases involving the same agent, or one acting in the same causative way (paragraph 104). While he also commented that the common law is capable

of further development, it seems to me that it is difficult to apply the policy reasons driving the *Fairchild* exception to a case where it has not been established that the injury was caused by asbestos exposure. In such circumstances the law's resistance to making a party pay compensation unless it is shown that he wrongfully caused or materially contributed to the injury will hold sway. In *Fairchild* at paragraph 43 Lord Nicholls emphasised that to avoid injustice "considerable restraint" is called for in any relaxation of the normal test for a causal connection.

[84] No doubt recognising that the *Fairchild* exception as developed by the courts, and by Parliament through the Compensation Act 2006, can result in full liability being imposed on someone who probably did not cause the mesothelioma, in *Sienkewicz* Lord Brown of Eaton-under-Heywood stressed that the circumstances for its application were "narrowly circumscribed" and that the policy rationale depended on the harm having been caused by that against which the employers should have afforded protection, see paragraph 178. His Lordship was clearly concerned about the impact of the special treatment of mesothelioma cases on the coherence of the law, and sternly counselled against the courts thinking of any further special rules or anomalies "in an area of law which benefits above all from clarity, consistency and certainty in its application", paragraph 187. In the same passage he mentioned academic opinion to the effect that special rules of causation are more suited to "single agent" cases.

Disposal

[85] For the above reasons decree will be pronounced absolving Mrs Kerr's employers from any liability in damages for her very sad demise.