Sharp v Port Kembla RSL Club: establishing causation of laryngeal cancer by environmental tobacco smoke

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ABSTRACT

- A New South Wales Supreme Court jury has decided that environmental tobacco smoke (ETS) can cause or materially contribute to the development of laryngeal cancer.
- Evidence presented that ETS may cause or materially contribute to laryngeal cancer included the molecular genetics of tobacco-smoke-induced carcinogenesis, and two relevant epidemiological studies.
- The plaintiff's exposure to ETS was established indirectly, on the basis of occupational history involving work as a bar attendant in licensed premises.
- The jury's decision seems likely to encourage other "passive smoking" cases, and may result in measures to reduce occupational exposure to ETS.

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IN A RECENT DECISION of the Supreme Court of New South Wales, *Sharp v Port Kembla RSL Club Ltd*, an employee recovered damages from her employer on the basis that exposure to environmental tobacco smoke (ETS) in the course of her employment caused, or materially contributed to, the development of laryngeal cancer (see Box 1 for the plaintiff's occupational and clinical history). The plaintiff had brought her claim against two employers; one claim was settled out of court and the other proceeded. The decision is noteworthy not so much because it extends medicolegal recognition of cancer causation by ETS beyond the lung, but because of the kind of evidence presented to prove causation.

The case was decided by a jury and, as in all jury cases, no reasons for the decision were given. Thus, it is impossible to determine which parts of the evidence influenced the jury's decision. It may be, for example, that they preferred the evidence of the experts called by the plaintiff simply because they found them to be more believable than the other experts. The credibility of two American scientific witnesses called in the defence case came under attack by reason of their association with the tobacco industry.[1]

Nevertheless, the trial judge (whose role was to decide issues of law in the case, leaving issues of fact — including what caused the cancer — to the jury) gave the jury detailed directions over more than two days as to the way in which they were obliged by the law to assess the evidence on causation.

Having heard that summing up, the jury decided that the plaintiff's cancer was caused, or materially contributed to, by the negligence of her employer. For our discussion of the medical issues in the case we have assumed, as the law assumes, that the jury's answer to the causation question properly reflects the directions by the judge as to how causation must be determined according to law.

The law's requirement for proof of causation

To succeed in her claim for damages, the plaintiff had to prove, on the balance of probabilities, a causal connection in the legal sense between her exposure to ETS and her laryngeal cancer. The plaintiff did not have to prove that her cancer was caused solely by the defendant's wrongful conduct; it was sufficient to show that such conduct materially contributed to her injuries. A contribution is regarded as "material" for legal purposes if it is more than minimal, trivial or insignificant.

As was recently emphasised by the NSW Court of Appeal,[2] the legal concept of causation requires the court to approach the matter in a manner distinctly different from that of philosophy or science, including the science of epidemiology.

In the realm of negligence, the High Court of Australia has determined that causation is essentially a question of fact, to be determined as a matter of common sense.[3,4] This commonsense approach can be quite different to a scientist's approach. An inference of causation for the law's purposes may well be drawn when a scientist, including an epidemiologist, would not draw such an inference.[5]

In philosophy and science, the concept of causation has been developed in the context of explaining phenomena by reference to the relationship between conditions and occurrences. In law, problems of causation arise in the context of ascertaining and apportioning legal responsibility for a given occurrence.[6] In some cases, medical science cannot determine the existence of a causal relationship. However, for the purpose of attributing legal responsibility, a causal relationship might still be found to exist.

Accordingly, when the aetiology of disease is uncertain, the courts can decide as to causation on the balance of probabilities. Nevertheless, this common-law test is not satisfied by evidence which establishes only a possibility.

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Epidemiology is concerned with human populations, not individual circumstances. Therefore, in determining whether exposure to a particular substance is the legal cause of disease in a particular plaintiff, epidemiology only provides evidence of possibility.[7]

However, causation can be established in court by a process of inference that combines primary facts like "strands in a cable". An inference as to probabilities may be drawn from a number of pieces of evidence, each of which does not rise above the level of possibility.[8] Epidemiological studies and expert evidence based upon such studies can form the strands in the cable of a circumstantial case.

In the *Sharp* case, because of the rarity of this kind of cancer, the epidemiological strands of the cable were tenuous. The trial judge instructed the jury on the issue *inter alia* as follows:

> The epidemiology may prove useful to you, but you have heard how the scientists approach those studies and how many studies may be required before the scientists are able to say that the issue has been proved. [*Sharp*, summing up, p 21]

However, when combined with evidence of the current understanding of the molecular mechanisms by which the disease develops, the cable of causation became sufficiently strong for the law's purposes; the totality of the evidence showed a connection which was close enough to justify the jury's conclusion that the possible was the actual cause.

As the judge went on to direct the jury:

> You must have regard to all of the evidence, the opinions for and against, which includes the clinicians and the scientists. It is open to you to determine the issue in favour of the plaintiff or the second defendant, even if the epidemiological evidence does not confirm that outcome. [*Sharp*, summing up, p 21]

Case for the plaintiff

Cancer in an individual is attributable to the impact of a particular agent or environment upon determination of two issues:

- that the agent or environment presents a carcinogenic hazard to humans, and (if so)
- that the individual in question was exposed to the carcinogenic hazard in such circumstances as may result in the development of the malignancy.

Thus, the first issue is whether ETS causes laryngeal cancer. In addition to lung cancer, smoking causes cancer of the larynx, pharynx, oral cavity, oesophagus, kidney, bladder and pancreas.[9] Other malignancies associated with smoking include stomach cancer, cervical cancer and leukaemia.[10] Of all the types caused by smoking, laryngeal cancer appears to exhibit the highest attributable risk. In Australia, 83% of laryngeal cancer in men and 78% in women is attributable to active smoking[11] and similar figures have been consistently reported worldwide.[12]

In the *Sharp* case, the epidemiological evidence was augmented by an explanation of the current understanding of carcinogenesis. The argument was predicated on biological mechanisms responsible for malignant transformation and the consideration that tobacco smoke is known to be carcinogenic via processes now understood at the molecular level (Box 2).[13-15]

1: Plaintiff's occupational and clinical history

The plaintiff was born in 1939 in Sydney and moved to Port Kembla when aged nine years. At age 18, she was diagnosed with pernicious anaemia and treated with vitamin B_{12} injections. She had an appendicectomy in early life, and was taking hormone replacement therapy at the time of recent surgery. She was employed from 1963–1972 at a Wollongong factory making brassieres, which did not involve any significant exposure to environmental tobacco smoke. From 1972 to 1984, she was employed (17 hours/week) as a bar attendant at the Port Kembla Hotel. The plaintiff was similarly employed 25 hours per week at Port Kembla RSL Club during 1984–1995. The building was air conditioned, but the plant was often out of service. There were no windows in bar areas and no smoke-free zones; the atmosphere was described as "smoky". She variously attributed eye irritation, dry throat and intermittent cough to conditions at work, and her children noted that after she returned from work she smelt of cigarettes. The plaintiff did not smoke. Her father had been a light smoker, but did not smoke in the house. Her second husband smoked "casually" between 1978 and 1984, but not in her presence. Her alcohol intake was 2–3 drinks per week.

In May 1995, she noted a small lump on the right side of her neck. Biopsy showed malignant cells. Further investigation indicated a primary tumour involving the right side of the epiglottis. In August 1995, she underwent right neck dissection and a supraglottic laryngectomy. The tumour was described as a moderately differentiated squamous cell carcinoma of the base of the tongue, with extension into the base of the epiglottis and right epiglottic vallecula. She received postoperative radiotherapy over six weeks. The patient was seen regularly in the course of follow-up. She is described as having rehabilitated very well, has excellent oral function and has a good voice.

Thus, cancer induced by genotoxic carcinogens is a consequence of critical changes in gene structure and expression. One corollary of this understanding is that there is no level of exposure below which an increased risk of cancer cannot be anticipated (the "no safe dose" principle[16]). As it is established that cigarette smoke is carcinogenic for humans, the molecular genetic data suggest that inhalation of tobacco smoke presents a carcinogenic hazard irrespective of the amount inhaled. Exposure to ETS results in inhalation of a lesser amount of smoke than is inhaled by a smoker.[17] A large body of epidemiological evidence, and its evaluation, has provided confirmation of the expected increased risk of lung cancer among non-smokers who share breathing space with smokers at home and/or at work.[18]

On the same basis, ETS is reasonably expected to cause laryngeal cancer, although the risk can be expected to be less than that for a smoker.[17] Moreover, as there are fewer laryngeal cancers in comparison with lung cancers among smokers, the number of cases of laryngeal cancer caused by ETS will be small. Accordingly, quantification of the risk by epidemiology is difficult. Funding for such studies may be difficult to obtain for this reason and because no additional data are necessary to justify public health measures to reduce exposure to ETS.

Nevertheless, the impact of ETS has been investigated in squamous-cell carcinoma of the head and neck (HNSCC),

which includes the larynx. In 1997, a retrospective analysis comparing 59 non-smoking HNSCC patients with matched non-HNSCC non-smoking control subjects showed that the HNSCC patients had a significantly higher risk of ETS exposure both in the home and workplace.[19] Subsequently, a study of 173 HNSCC patients and 176 cancer-free controls returned a crude odds ratio for ETS exposure of 2.4 (95% CI, 0.9–6.8) after controlling for age, sex, race, education, alcohol consumption and other variables. Dose response was evident after categorisation to either moderate (crude OR, 1.8) or heavy (crude OR, 4.3) ETS exposure.[20]

Therefore, a relationship between exposure to ETS and increased risk of head and neck cancer, anticipated on the basis of the carcinogenicity of ETS and the susceptibility of the upper respiratory tract to this carcinogen, is supported by the available epidemiology.[21,22]

On the issue of whether the plaintiff was exposed to sufficient ETS to cause cancer, the evidence was indirect. Direct evidence of such exposure is exemplified by the presence of nicotine metabolites in urine.[23] Indirect evidence is provided by the occupational history (Box 1). Levels of tobacco-derived pollutants have been measured in various indoor spaces. Typical amounts of particulate matter are < 120 $\mu g/m^3$ in aircraft, 130–960 $\mu g/m^3$ in workrooms and 233–986 $\mu g/m^3$ in taverns.[12] The plaintiff was exposed to ETS as a bar attendant in a hotel between 1972 and 1984, and between 1984 and 1995 in a licensed club.

The plaintiff and other employees and patrons of the club gave evidence of their observations of a high level of ETS in the plaintiff's workplace. There was no evidence before the jury of comparable, consistent exposure of the plaintiff to ETS anywhere else. Accordingly, it was submitted to the jury that the more than 11 years of employment at the club in a bar room situation provided the necessary duration and intensity of exposure for it to be concluded that ETS materially contributed to the development of her malignancy.

2: Elucidation of the carcinogenicity of tobacco smoke[13-15]

Whole organism (1950s)

- Association between smoking and lung cancer based on analysis of groups.
- Causation of tumours in rodents by polycyclic aromatic hydrocarbons, *N*-nitroso compounds and other chemicals in tobacco smoke.

Cell and tissue (1970s)

- Metabolism of polycyclic hydrocarbons and *N*-nitroso compounds demonstrated in rodent and human tissue.
- Malignant transformation of human cells in culture by polycyclic hydrocarbons and *N*-nitroso compounds.

Macromolecule (1970s)

- Covalent binding of polycyclic hydrocarbons and *N*-nitroso-derived alkyl groups to DNA.
- Persistence of carcinogen adducts in DNA caused by failure of DNA repair processes, correlated with the site of tumour development.

Gene (1980s)

- Activation of *ras* oncogene following alkylation by *N*-nitroso compounds able to cause malignant transformation in cultured cells.
- Evolution of malignant cells in human cancer, via hyperplastic premalignant and benign lesions, correlated with multiple discrete changes in oncogenes and tumour-suppressor genes.

Codon (1990s)

- Role of p53 tumour-suppressor gene ("Guardian of the Genome") in mediating cell-cycle arrest and apoptosis established.
- Inactivation of p53 by mutation (or chromosome deletion) recognised as the most common specific genetic alteration encountered in human malignancy.
- Sequence-specific mutation of p53 in cultured cells exposed to a polycyclic hydrocarbon described.
- Sequence-specific mutation of p53 in lung cancer from individual smokers is the same as that observed in cultured cells exposed to a tobacco-smoke-derived polycyclic aromatic hydrocarbon.

Arguments of the defence

Apart from denying that there was sufficient ETS at the club to cause cancer, the principal defence was that the plaintiff's malignancy was not correctly categorised as "laryngeal". Rather, it was contended that it was "oro-pharyngeal". It was therefore suggested that epidemiological evidence regarding the association between laryngeal cancer and cigarette smoke was irrelevant. Alternatively, if the tumour were indeed "laryngeal", other risk factors which could be established from the plaintiff's history provided a more likely explanation for carcinogenesis and/or reduced confidence that the disease resulted from the effects of ETS.

Characterisation of the neoplasm as "laryngeal" cancer was disputed on anatomical grounds. The tumour was described as a squamous-cell carcinoma of the base of the tongue with extension into the base of the epiglottis and the right epiglottic vallecula. Differences between the epithelia at various sites in the upper respiratory tract were subject to detailed discussion. The distinctions drawn were arguably of little application, as no part of the upper respiratory or upper alimentary tract is considered refractory to smoke-induced carcinogenesis and the relative sensitivities are difficult to establish because data (for both "active" and "passive" smoking) are often based on "cancer of the head and neck".

The defence argued that the plaintiff should be considered at above average risk of HNSCC because she lived at Port Kembla, specifically on the basis of the "Wollongong leukaemia cluster".[24] Cross-examination was predicated on the cluster investigation being concerned with levels of particular atmospheric pollutants in the area (which is true) and argument that the cluster itself, among other things, was evidence that Port Kembla residents are at increased risk of pollution-associated cancer (which is false[25]). In cross-examination, which focused wholly on the plaintiff, there was scant opportunity to refute generalisations about the whole population, and any resident could have been left with serious concerns. The irony is that the effect of local pollution on cancer risk is almost impossible to discern against the background level of cigarette-induced disease. Head and neck cancer (in men or women) is no more common in the Illawarra region than it is in the whole of NSW.[25]

Alcohol drinking is a risk factor for laryngeal cancer.[26] The plaintiff drank only very moderately. Virtually all data concerning alcohol-associated cancer involve consumption at a level of several drinks per day. More importantly, the risk of laryngeal cancer in people who both smoke and drink alcohol daily increases multiplicatively compared with the risk from either factor operating alone.[27] The most reasonable conclusion was that any impact of alcohol would have been to increase the risk that may be inferred from the two studies of the impact of ETS on HNSCC.

The plaintiff was exposed occupationally to significant levels of ETS between 1972 and 1995. However, because she settled her case against the first employer, the jury was only concerned with the exposure between 1984 and 1995. The defendant argued that carcinogenesis could be wholly attributed to ETS exposure during the earlier period, 1972 to 1984. However, among active smokers who quit, the risk of lung cancer decreases within five years.[12] This finding is crucial to an understanding of tobacco smoke carcinogenesis. The relatively immediate decrease of risk upon quitting suggests a progressive process in which each year of exposure increases the likelihood of tumorigenesis, a scenario which is incompatible with tumorigenesis being the irreversible outcome of exposure during some initial period. Accordingly, exposure to ETS up to the point when cancer was diagnosed may be judged as contributing to the plaintiff's risk.

The verdict and its implications

By their verdict, the jury showed they accepted that, more probably than not, ETS at the Port Kembla RSL Club caused, or materially contributed to, Mrs Sharp's cancer. No appeal has been lodged by the club. The insurance and licensed clubs industries have already taken steps to reduce the likelihood of similar cases and the extent of future human exposure to ETS. The decision will undoubtedly encourage other "passive smoking" cases, although there is unlikely to be a deluge owing to the cost of such litigation and the difficulties of proving causation given the current epidemiology.

Because juries do not give reasons for their decisions, it is impossible to say how much the *Sharp* jury was influenced by epidemiological as opposed to biological, mechanistic considerations in determining causation. However, given the state of the evidence and the judge's directions to the jury, it is likely that both played a significant part.

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