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Forever Chemicals and Tort Law – Causation

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Abstract: The ubiquity of the thousands of artificial substances known generically as PFAS is increasingly apparent. Their presence in human blood, drinking water, commercial fisheries, soil, and consumer products, alongside their association with a range of health and environmental impacts, is stimulating tort claims globally. These claims are likely to raise recurring causal challenges for claimants and courts.

This contribution will explore some of those challenges, focusing on English law, and on core issues around, first, scientific uncertainty about the effects of these chemicals, and secondly, the existence of multiple possible causal factors in many cases. Establishing causation for disease (rather than contamination) is likely to be extremely difficult. As in other areas of complex causation, maintaining strict causal requirements might reasonably be understood either as protecting or as undermining the fundamental values of tort: even if this dilemma cannot be resolved, it is worth raising.

I Introduction

'Forever' chemicals, the category of thousands of artificial substances consisting of per- and poly-fluoroalkyl substances (PFAS), are so called because of their high persistence in the environment and the difficulty or impossibility of removing them. PFAS are also highly mobile in the environment, so as well as being 'forever', they are also 'everywhere',[1] ubiquitous in ground and surface water, in soil, air, food, animals and plants, globally including Antarctica and the Tibetan high-

1 *J van Zeben*, PFAS are forever: Regulating chronic toxicity in our living environment, in: J van Zeben/C Hilson (eds), A Research Agenda for Environmental Law (2025).

Note: I am grateful to Paul Mitchell, Barbara C Steininger and participants at the 24th Annual Conference on European Tort Law. All websites cited in this paper were last consulted in April 2025.

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lands.[2] They can be detected in the blood of almost all humans living in the developed world, and in the organs of human foetuses.[3] PFAS offer a range of industrial and consumer benefits, but are associated with severe health and environmental problems.

This paper will begin in section II with some context on PFAS and their regulation. Although covered by a dense and diverse regulatory landscape, including restrictions and bans on some PFAS, PFAS are little-regulated as a category, and new PFAS are still being developed and marketed. Section II will also touch on the existence of global tort claims.

This contribution is specifically about causation in tort, to which I turn in section III. Depending on the tort, the claimant must establish that the defendant's *activities* caused the damage, that the defendant's *negligence* caused the damage, or that the defendant's *defective product* caused the damage; and different issues are also raised depending on the type of damage, be that disease, elevated levels of PFAS in the blood, exposure to the risk of personal injury, physical damage to property or land, inability to use and enjoy one's property due to dangerous (or just scary) levels of contamination, or damage to the environment. I focus on English law, and although the legal approaches vary across jurisdictions, the causal challenges are in many respects shared. Indeed, PFAS torts may become transnational phenomena, due not only to globalised supply chains and production processes, but also transnational networks of activism and knowledge production. The *systematic* articulation of doctrines around issues such as damage and causation may (for better and worse) become as important as jurisdictional specificity.[4] I begin section III with a discussion of scientific uncertainty around the harmful effects of PFAS, before turning to a range of plausible scenarios in which claimants are faced with multiple possible causes of a harm associated with PFAS.

Finally, as in other areas of complex causation, maintaining strict causal requirements might reasonably be understood either as protecting or as undermining the fundamental values of tort. I leave this dilemma open, but touch on the implications of the discussion of causation by way of conclusion.

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2 *H Brunn et al*, PFAS: forever chemicals – persistent, bioaccumulative and mobile. Reviewing the status and the need for their phase out and remediation of contaminated sites (2023) Environmental Sciences Europ 20.

3 *Brunn et al,* ibid.

4 *N Affolder*/*GEK Dzah*, The transnational exchange of law through climate change litigation, in: F Sindico et al (eds), Handbook on Climate Change Litigation (2024).

II PFAS, their characteristics and their regulation

The category of artificial substances known generically as PFAS has multiple industrial and consumer uses, including: surface coatings for textiles, food contact materials and packaging; non-stick surfaces for pots and pans; use in cleaning agents, paints, varnishes, polishes and waxes, pharmaceuticals, cosmetics and sunscreens, medical devices and products, pesticides, and firefighting foams.[5] PFAS contaminate soil and water during manufacture and use, and on disposal. Humans are exposed primarily through food and drinking water, although air pollution is increasingly apparent, including indoors, especially from the treatment of carpets and outdoor equipment.[6] The desirable properties that make PFAS widely used (stability under intense heat; oil- and water-resistance; low chemical reactivity) are due to the exceptional strength of the carbon-fluorine bond. Those qualities are also the source of PFAS' dangers.[7] PFAS bioaccumulate, building up in organisms due to their long half-lives and their resistance to being excreted or metabolised. They are mobile in the environment, meaning that they can penetrate soil and may be carried large distances in ground water.

Some PFAS have been found to be toxic to fish, amphibians, invertebrates and insects. Some have also been found to be toxic to humans, particularly affecting the liver, kidney, thyroid and immune system; several have been classified as carcinogenic. There is growing evidence around other health effects, from effects on the hormone system of pregnant women affecting organ development in the foetus, to repression of immunoreceptors, to altered metabolism and reduced fertility.

Our understanding of levels of environmental contamination by PFAS is limited, and comes mainly from the EU and the US, due to the existence in these jurisdictions of at least some relevant drinking water standards and monitoring.[8] Remediation is complex, if it is possible at all. The removal of PFAS from drinking water, for example, is both complex and of limited effectiveness; remediating soil is even harder. So alternative supplies of drinking water, and large-scale removal of contaminated soil (which may then become contaminated waste for regulatory purposes) are more likely responses. Affected communities might be told not to drink

5 See *LGT Gaines*, Historical and current usage of per- and polyfluoroalkyl substances (PFAS): A literature review (2023) American Journal of Industrial Medicine 353.

6 Note that there have been tests in shops selling outdoor and sports textiles or carpets, *Brunn et al* (2023) Environmental Sciences Europ 20.

7 See for the following discussion of impacts, eg *Brunn et al* (2023) Environmental Sciences Europ 20; *van Zeben* (fn 1).

8 In England and Wales, there is 'precautionary' guidance on PFAS in drinking water, but no statutory standards, <https://www.dwi.gov.uk/pfas-and-forever-chemicals/>.

mains water or to take a 'precautionary' approach to home-grown vegetables;[9] commercial fishing may be restricted.[10]

A complex and potentially comprehensive web of regulation applies to PFAS, in the EU and beyond.[11] Some PFAS are banned internationally as Persistent Organic Pollutants.[12] At EU level, the chemical (or 'substance') itself is regulated under Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) Regulation.[13] If the chemical is found in a product, we could turn to general product safety law, or more specific regulation, for example on drinking water, toys or pesticides. The industrial or other activity using or producing the chemical is also likely to require licensing, for example under environmental permitting in the UK, or the Industrial Emissions Directive in the EU.[14]

Notwithstanding this dense regulatory context, PFAS are little-regulated as a *category*.[15] The category of PFAS includes thousands of individual chemicals,[16] and as one, or a group, of PFAS is regulated, its uses may be met by another, possibly with similar properties and so raising similar concerns.[17] There is increasing pressure to regulate the category of substances more comprehensively.[18] EU bodies are progressing work on a universal ban on non-essential PFAS via the restriction pro-

9 See the advice from Wyre Council, <https://www.wyre.gov.uk/news/article/513/multi-agency-investigation-update>.

10 See eg, High levels of toxic chemicals found in fish from the West Scheldt, Brussels Times, 7 December 2021.

11 *van Zeben* (fn 1).

12 United Nations Stockholm Convention on Persistent Organic Pollutants 2021.

13 Reg 1907/2006/EC concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH), establishing a European Chemicals Agency [2006] Official Journal (OJ) L 396/1, as amended. For rich analysis, see *S Vaughan*, EU Chemicals Regulation: New Governance, Hybridity, and REACH (2015). A similar approach is taken in Britain, through a series of statutory instruments following Brexit, including The REACH etc (Amendment etc) (EU Exit) Regulations SI 2019 No 75.

14 Dir 2010/75/EU on industrial emissions (integrated pollution prevention and control) [2010] OJ L 334/17, as amended. Note that the proposed reversal of the burden of proof on causation in compensation claims in the 2024 revisions did not survive the process of negotiation. The Environmental Permitting (England and Wales) Regulations SI 2016/1154, as amended.

15 The European Chemicals Agency webpage on PFAS both gives a sense of the nature of the regulatory challenge and outlines steps being taken, <https://echa.europa.eu/hot-topics/perfluoroalkyl-chemicals-pfas>.

16 In an indication of the difficulty of adequately grasping PFAS with regulation, *van Zeben* (fn 1) observes that in August 2022, the US EPA lists 14,735 chemicals classified as PFAS, whilst the OECD's database only 4,700.

17 See eg, *N Hamid et al*, The untold story of PFAS alternatives: Insights into the occurrence, ecotoxicological impacts, and removal strategies in the acquatic environment (2024) Water Research vol 250 (12); *Brunn et al* (2023) Environmental Sciences Europ 20.

18 *Brunn*, ibid; *van Zeben* (fn 1).

cess under REACH, and both France and Denmark have, for example, banned all PFAS from a limited range of consumer goods. Others insist that the chemicals have different qualities and should be regulated separately,[19] and comprehensive restrictions are likely to be both legally and politically complicated.[20]

Affected individuals and communities have turned to tort. As ever a pioneer in tort, the US has seen PFAS cases since the late 1990s.[21] There have been some sizeable awards and settlements, including, for example, a $ 921 million settlement in 2017, with approximately 3,500 Ohio Valley residents, for illnesses including kidney and testicular cancer and thyroid disease. Class action settlements have also enabled epidemiological studies. There are many ongoing cases, including from public authorities seeking reimbursement of clean-up costs, as well as some important personal injury litigation. The litigation is mainly against the large manufacturers like DuPont, Chemours, 3M and others, but there are also consumer claims, for example in respect of the existence of PFAS in products marketed as 'natural' or 'organic'.[22]

Tort is being called on across Europe.[23] Many cases are still at an early stage. Many (not all) arise out of hot spots of PFAS environmental presence associated with firefighting foams, be that around manufacturing sites, defence facilities, or fire training facilities.[24] The Swedish Supreme Court, for example, held a public drinking water provider liable to local residents for providing a defective product in the form of drinking water contaminated by PFAS.[25] The contamination arose out of the use of PFAS in firefighting foam in a fire training centre. A Belgian first instance court awarded damages against 3M in respect of its production of PFAS for

19 *F Spyrakis*/*TA Dragani*, The EU's per- and polyfluoroalkyl substances (PFAS) ban: A case of policy over science (2023) Toxics 721.

20 *K Garnett*/*G van Calster*, The concept of essential use: A novel approach to regulating chemicals in the European Union (2021) Transnational Environmental Law 159.

21 For a useful review, see *CT Liljestrand*, PFAS Exposure: A Comprehensive Look at Emerging Facts and Studies, Risk and Liability Assessment, Litigation History, Evolving Regulations and Future Predictions (2022) Defense Counsel Journal 1.

22 See also *CT Liljestrand*, PFAS in Food Packaging: Product Liability Concerns for Industry Manufacturers and Sellers (2023) Defense Counsel Journal 1.

23 ChemSec, an NGO, has a useful summary of litigation, <https://chemsec.org/just-the-start-the-growing-legal-battle-over-pfas-in-europe/>. There are tort cases elsewhere in Europe, at least in the Rhone Valley, France against manufacturers Arkema, in Veneto, Italy and in the Netherlands.

24 There are also at least two Australian settlements in respect of property damage associated with firefighting foam at defence sites, see eg, *R Legg*/*J Prior*, Toxic torts as compensation: Legal geographies of environmental contamination litigation (2023) Geographical Research 234.

25 T486–23. This possibility explains the volume of public authority clean up cases in the US – presumably more will follow in Europe.

use in firefighting foam, in respect of loss of enjoyment of property.[26] The same factory at the Port of Antwerp is being targeted by groups representing Dutch commercial fishers, who have been unable to fish for certain species in the western Scheldt, due to elevated PFAS levels. The German government is being sued in respect of groundwater contamination by PFAS (from firefighting foam) at an air force base. Claims are being brought in respect of groundwater contamination by a firefighting foam manufacturer (Angus Fire) in North Yorkshire, England.[27]

III The causation challenges

I focus here on English law: whilst the doctrinal approaches are distinctive across Europe, the challenges are shared.[28] We might also note the suggestion that when the defendant is a US-domiciled parent company (such as 3M), it may be beneficial to seek to apply US state common law.[29]

A key variable in the causation analysis will be the sort of damage we are dealing with. I am not aware of any claims for personal injury in Europe, presumably precisely because of concerns about establishing causation. In the US, there have been verdicts and settlements for kidney cancer and testicular cancer,[30] although personal injury claims are clearly challenging and alternatives are sought.

Our starting point across jurisdictions will generally be 'but for' causation/*conditio sine qua non*:[31] *but for* the defendant's negligence (or activity, or defective product), the claimant would not have suffered the relevant damage. In some PFAS cases, especially if damage is generously defined, this could be straightforward: where there is only one, wrongful source of PFAS, which undoubtedly brings about the damage. For example, in the Swedish decision mentioned above, the public

26 For details, see *G van Calster*, Neighbours trip up big industry with Antwerp judgment holding 3M to account for (common law) nuisance following PFAS pollution, 2023 at <https://gavclaw.com/tag/pfas/> with a link to case (in Dutch).

27 Lawyers have been instructed, in respect of PFOA and PFOS, see <https://www.mishcon.com/news/mishcon-de-reya-instructed-by-community-group-to-bring-legal-action-over-forever-chemical-pollution-in-the-uks-most-pfas-polluted-town>.

28 See eg, *O Moréteau*, Basic Questions of Tort Law from a French Perspective, Part 6: The elements of liability, in: H Koziol (ed), Basic Questions of Tort Law from a Comparative Perspective (2015).

29 *G van Calster*, Forever chemicals, and suing 3M for PFAS pollution in Europe. A flag on applicable law, 2021 at <https://gavclaw.com/2021/08/31/forever-chemicals-and-suing-3m-for-pfas-pollution-in-europe-a-flag-on-applicable-law/>.

30 *Gaines* (2023) American Journal of Industrial Medicine 353.

31 Eg, Conditio sine qua non in General, in: B Winiger/L Koziol/BA Koch/R Zimmermann (eds), Digest of European Tort Law, vol 1: Essential Cases on Natural Causation (2007) 9–101.

drinking water provider supplied a defective product (water containing PFAS above the permitted level), which led to elevated levels of PFAS in the blood of the claimants, which was held to constitute personal injury.[32] In other circumstances, given the many routes by which PFAS can enter the claimants' blood, even an apparently simple case will run into difficulties. Different jurisdictions have different ways of responding (or not) to the concern that strict adherence to but for causation would cause injustice.

I address two broad issues in this section. I begin with the scientific uncertainty about the harmful effects of PFAS. I then turn to the range of scenarios in which there are multiple sources of the PFAS, or multiple other possible causes of the claimant's harm.

A Scientific uncertainty

The biggest causal issue in PFAS torts is a conceptually simple one of scientific uncertainty. Whilst this will be especially problematic in personal injury claims, it will also arise in respect of, for example, damage to crops or cattle, and scientific knowledge could also be a limiting factor in determining whether the presence of PFAS in the body, in soil or in water 'counts' as damage for the purposes of tort.[33] Only relatively few of the thousands of chemicals in the PFAS category have been studied,[34] and many of those studies are incomplete or inconclusive.[35] PFAS were first discovered by accident in 1938, but far newer substances have been, and are being, developed.[36] Two of the formerly most common PFAS (PFOA and PFOS) are no longer used, and their replacements are less well understood. Whilst PFAS share certain qualities, some argue that the different substances are distinct and should be regu-

32 In English law, we would look for the line between *Rothwell v Chemical and Insulating Company Ltd* [2007] United Kingdom House of Lords (UKHL) 39 (pleural plaques indicate exposure to asbestos, but are symptomless and do not lead or contribute to any condition that would cause symptoms) and *Dryden v Johnson Mathey Plc* [2018] United Kingdom Supreme Court (UKSC) 18 (sensitization to platinum salts is symptomless, but leads to a change to the 'physiological make-up' of the claimant, such that further exposure to platinum salts carries the risk of allergic reaction, requiring behaviour change). Only in the latter case was there 'damage'.

33 The breach of regulatory standards as to PFAS in environmental media might establish damage, see eg *M Lee*, The sources and challenges of norm generation in tort law (2018) European Journal of Risk Regulation (EJRR) 34.

34 Eg *Liljestrand* (2022) Defense Counsel Journal 1.

35 This is apparent, for example, from <https://dceg.cancer.gov/research/what-we-study/pfas>.

36 *Liljestrand* (2022) Defense Counsel Journal 1.

lated differently.[37] No doubt their distinctiveness, and the ability to analogise between different substances, will be hard fought in tort.

To establish but for causation, claimants need to show both that the level and mechanism of exposure to the substance at issue are *capable* of causing the particular disease they are suffering from ('general' or 'generic' causation), and that their disease was indeed caused in that way ('specific' or 'individual' causation).[38]

In *Holmes v Poeton*, the claimant argued that negligent workplace exposure to tricholorethylene (TCE)[39] in the cleaning and degreasing of engineering machinery had caused his Parkinson's disease. The Court of Appeal decision brings out some generally interesting issues around causation and disease. Stuart-Smith LJ described *generic* causation as 'the hypothesis that exposure to TCE can cause (or materially contribute to the causing of) Parkinson's disease'. He said that the evidence relied on by the judge to establish generic causation was 'weak',[40] since it established only that TCE is 'a risk factor for Parkinson's disease and that there is a plausible mechanism based on the rodent studies for a finding that TCE may cause or materially contribute to the development of Parkinson's disease', concluding that 'the evidence to prove generic causation is lacking.'[41] In the absence of proof of generic causation, the claimant will generally not be able to establish specific causation, that is that their harm was caused by the defendant's negligence,[42] absent possibly some exceptional situation, such as an acute reaction to massive exposure.

We return to *Holmes* for what it tells us about material contributions to damage in the following subsection. First though, the scientific uncertainties identified by the Court of Appeal are likely to be a persistent feature of PFAS claims. The absence of sufficient epidemiological evidence to support a general causal link will be a feature of many substances in the PFAS category. The discipline of epidemiology studies the distribution and determinants of disease in human populations,[43] so that, for example, a key piece of evidence in the link between smoking and lung cancer

37 *Spyrakis*/*Dragani* (2023) Toxics 721.

38 *Holmes v Poeton* [2023] Court of Appeal (Civil Division) (EWCA Civ) 1377.

39 TCE is a chlorinated solvent, which is not a PFAS but is a frequent co-contaminant with PFAS, see *F Zhang et al*, Dual effects of PFOA or PFOS on reductive dichlorination of trichloroethylene (TCE) (2023) Water Research vol 240.

40 [100].

41 [112].

42 The Court of Appeal leaves open the possibility that certain features of a particular case 'are not reflected in the generic evidence that compel a finding of causation, such as a relevant and repeated response to challenge and rechallenge by exposure to TCE', [114].

43 *T Ward*, Expert evidence, 'naked statistics' and standards of proof (2016) EJRR 580; *B Siegerink et al*, Causal inference in law: An epidemiological perspective (2016) EJRR 175.

was the study of disease in large numbers of smokers and non-smokers.[44] The novelty of some of the individual PFAS chemicals precludes long-term epidemiological studies; even if there may be some shorter-term effects, the studies take time. Even for more established substances, the complexity of PFAS exposures over a lifetime (PFAS are after all distinguished for their ubiquity and their persistence) can make observational studies difficult. And as suggested above, the extent to which it is legitimate to extrapolate knowledge from one PFAS chemical to another is also likely to be hard fought. And finally, when chronic exposure has small – but real, it causes some disease – effects, as is often the case for chronic chemical exposure, large sample sizes (and significant resources) are needed to detect it, especially if there are many cases of the disease in the general population. A significant 'background incidence' of any disease associated with PFAS creates a challenge for researchers (and claimants), since it implies other possible causes, in many cases poorly understood. For example, kidney cancer is one of the diseases associated with PFAS, but the UK National Health Service (NHS) tells us that causes of kidney cancer include smoking, obesity, high blood pressure and genetics.[45] By contrast, for example, we know that mesothelioma is always caused by asbestos, that it is a 'signature disease' of asbestos.

I do not wish to be too negative. Epidemiological evidence is, and will be, available, not least because funding for epidemiological research was part of the settlement in one of the early US cases.[46] I do, however, want to make the point that compelling epidemiological evidence on many PFAS is likely to be hard to come by.

In the absence of epidemiological evidence, the claimant may turn to evidence from animal testing. In *Holmes*, this also fell short. In particular, the 'inability to extrapolate the rodent studies to the impact of TCE exposure in humans' was fatal, 'not least because of the great disparity in relative exposure levels'.[47] This, again, is a commonplace problem in efforts to understand the human health impacts of novel

44 Classically, *R Doll*/*AB Hill*, The mortality of doctors in relation to their smoking habits. A preliminary report, (1954) British Medical Journal 1451.

45 <https://www.nhs.uk/conditions/kidney-cancer/causes/>.

46 <http://www.c8sciencepanel.org/>. Here is the panel's description of their task: 'One part of the Settlement was the creation of a Science Panel, consisting of three epidemiologists, to conduct research in the community in order to evaluate whether there is a probable link between PFOA exposure and any human disease. A "probable link" in this setting is defined in the Settlement Agreement to mean that given the available scientific evidence, it is more likely than not that among class members a connection exists between PFOA exposure and a particular human disease. The Science Panel recognizes that, given the many diseases we are studying, some may appear to be associated with exposure simply through chance, but we have to judge these associations individually and acknowledge the uncertainty inherent in making these judgments.'

47 [112].

chemicals. Using animal (or in vitro) experiments to establish contribution to risk of disease, or causation of disease, in humans requires contested extrapolations across species and across dosages. The most widespread human impacts are likely to arise in respect of chronic rather than acute exposure, that is low levels of exposure across many years, whilst for practical reasons, testing is often at high doses in short time periods. This is a hard task for regulators working at the population level, but in tort, even with respect to general causation, we will be asking specifically whether a particular, probably low level, chronic exposure is capable of causing a particular disease.

The scientific evidence by which general causation is established will develop. If we review the English case law on asbestos, for example, where long latency periods mean we are concerned with defendants' breaches of duty decades earlier, we are presented with an idealised story of steadily and inexorably improving knowledge: eventually, the contestation and denial around PFAS may even fall out of view.[48] Courts may moreover be willing to sidestep bespoke assessment of general causation if they can rely on the determination of an authoritative source – the World Health Organization, national governments, the EU – that a substance is capable of causing the relevant disease.[49]

If general causation can be established, however, the claimant is then likely to face further difficulties in establishing specific causation. In particular, whilst epidemiological evidence can establish general causation in appropriate cases, its role in individual causation is more problematic. I suggested above that epidemiological evidence can be hard to come by. It is also often the best evidence of disease causation that we have, so if claimants cannot use it to establish causation, they face systemic barriers to recovery.

Epidemiological evidence operates at the population level and does not explain the aetiology of disease at the individual level, only likelihoods and risks. In *Sienkiewicz*, Lord Rodger explores the distinction between 'claimant A, who proves on the balance of probability that a defendant *probably* injured him, and claimant B, who proves on the balance of probability that a defendant *actually* injured him'; the 'claimant who seeks to prove his case on the balance of probability in reliance entirely on statistical evidence will inevitably fail, since he is able to do no more than

48 *Lee* (2018) EJRR 34.

49 *Lee* (2018) EJRR 34, considering how 'official' sources of knowledge feed into questions of standard setting and breach. This may depend on the question being answered by the authority, eg internationally, substances are classified as 'carcinogenic to humans', via 'probably' and 'possibly carcinogenic to humans' to 'not classifiable as to its carcinogenicity to humans', <https://monographs.iarc.who.int/agents-classified-by-the-iarc/>.

prove on the balance of probability that the defendant probably injured him.'[50] These comments are made in the case of *Sienkiewicz v Greif*,[51] a Supreme Court mesothelioma case in which the claimants had used the decision in *Fairchild* to establish causation.[52] We return to *Fairchild* below, but essentially it allows a claimant to succeed if they can establish that the defendant's negligence increased their risk of contracting mesothelioma, which risk eventuated; *Fairchild* applies when it is impossible to prove that the defendant caused or materially contributed to the damage. The defendant employers in *Sienkiewicz* argued that in their case, proof was not impossible. They said that the very low level of workplace exposure relative to environmental exposure meant that it was more likely than not that they did *not* cause the damage (that it was caused by environmental rather than occupational exposure). Thus, *Fairchild* could not apply.

The Supreme Court, however, held that the impossibility of proving causation survived this statistical evidence on the dominance of environmental over workplace asbestos exposure. Epidemiological evidence in this case did not allow the defendant to escape liability. Whether epidemiological evidence could *ever* suffice to prove causation was not strictly decided, but the decision points towards a conclusion that 'statistical' evidence *alone* will never satisfactorily establish individual causation. Lord Dyson makes the important point that this 'is of particular relevance in relation to causation in toxic torts. It is often the basic impossibility of proving individual causation which distinguishes toxic tort cases from ordinary personal injury cases.'[53]

The role of epidemiology in English law is not categorically decided by *Sienkiewicz*: the members of the Supreme Court say slightly different things on the subject, the discussion of epidemiology is obiter,[54] and even taking the case at its strongest, epidemiology may be part of the evidential picture on specific or individual causation. Part of the discomfort with epidemiological evidence in English law (aside of course its departure from the individuals before the court) may be its interaction with the 'balance of probabilities' standard of proof. The assumption is sometimes

50 Lord Dyson, [216] and [218], summarising Lord Rodger. They both cite *S Gold*, Causation in toxic torts: burdens of proof, standards of persuasion, and statistical evidence (1986) Yale Law Journal 376 who distinguishes between 'fact probability' and 'belief probability'; on which see also *Ward* (2016) EJRR 175.

51 [2011] UKSC 10.

52 *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 Appeal Cases (AC) 32.

53 [218].

54 Both *C McIvor*, Debunking some judicial myths about epidemiology and its relevance to UK tort law (2013) 21(4) Medical Law Review (Med L Rev) and *Ward* (2016) EJRR 175 observe that no epidemiological expert was heard in this case. The statistics used in the Supreme Court were based on a calculation by the judge.

made that if epidemiological evidence is capable of establishing that the defendant's negligence (or product or activity, depending on the tort) caused the damage, tort becomes a statistical and probabilistic endeavour. And so, if the defendant doubled the claimant's risk (if there was a 51% likelihood that it caused the damage), the causal link is necessarily taken to have been proven (it is more likely than not), and vice versa.[55] Doubling the risk may be sufficient in some jurisdictions,[56] but although there are some suggestive cases,[57] it probably is not (especially post-*Sienkiewicz*) in English law. The key point of those who argue that courts should use epidemiological expert evidence with more confidence (and indeed of other scholars of causation)[58] is that the balance of probabilities is not a question of numbers.[59] The balance of probabilities is about whether the court is *convinced* that it is more likely than not that the defendant caused the damage.[60]

Further, we might note that suspicion of statistics beyond the courts is long-standing, even in now classic cases (like the pivotal 19th century John Snow cholera

55 *McIvor* (2023) 21(4) Med L Rev emphasises that epidemiologists do not assume causation on a finding of doubling of the risk, and so using epidemiological evidence does not imply that the courts will do so.

56 *H Koziol*, Damage caused to less than all victims by a harmful event. Comparative report, in: B Winiger/H Koziol/BA Koch/R Zimmermann (eds), Digest of European Tort Law, vol 1: Essential Cases on Natural Causation (2007).

57 *Novartis (Grimsby) v Cookson* [2007] EWCA Civ 1261, 'if occupational exposure more than doubles the risk due to smoking, it must, as a matter of logic, be probable that the disease was caused by the former', Smith LJ [74]; Lord Phillips in *AB v Ministry of Defence* [2012] UKSC 9 seems to assume that doubling the risk equates to a balance of probability, [98]. These are no more than passing comments. Smith LJ had suggested in the Court of Appeal in *Sienkiewicz* [2009] EWCA Civ 1159 that doubling the risk is the default approach to causation, albeit not in cases of mesothelioma. Dyson LJ in *Heneghan* asserts that: 'If statistical evidence shows that a tortfeasor more than doubled the risk that the victim would suffer the injury, it follows that it is more likely than not that the tortfeasor caused the injury' [8], albeit noting that the 'doubles the risk' test is contentious, citing *Sienkiewicz*. *C McIvor*, On the remit of the *Fairchild* principle and the 'doubles the risk' test for causation (2013) EJRR 559 suggests that the 'doubles the risk' test has its origin in *XYZ v Schering* [2002] England & Wales High Court (EWHC) 1420, where it was used to establish a *defect* for the purposes of product liability, but was subsequently interpreted as applying to causation.

58 S *Steel*/*D Ibbetson*, More Grief on uncertain causation in tort (2011) Cambridge Law Journal (CLJ) 451. See *RH (Minors) (Sexual Abuse: Standard of Proof)* [1996] AC 568, cited by *Ward* (2016) EJRR 580.

59 *McIvor* (2013) 21(4) Med L Rev observes that most people are unlikely to be able to distinguish between a 51% and 58% degree of belief; *Ward* (2016) EJRR 580 that even if they could, that misses their degree of confidence in that belief.

60 *McIvor* (2013) 21(4) Med L Rev; *Ward* (2016) EJRR 580. Both would think epidemiological evidence alone suffices (although epidemiological evidence is constituted of statistical and other evidence).

studies).[61] Epidemiological evidence may be more sophisticated than the Supreme Court in *Sienkiewicz* would suggest. In particular, epidemiology is not a simple matter of statistics, but uses other evidence, including, for example, biological evidence and a critical use of assumptions, in drawing causal inferences.[62] The quality of epidemiological studies will vary, and even with excellent studies, some will be more fit to answer the questions being asked in a tort trial than others. We might imagine a careful use of expert epidemiological evidence in which highly relevant studies (for example, reflecting the claimant's exposure pathways or pre-dispositions), showing strong correlations[63] might convince a court that in this case, it is more likely than not that the relevant PFAS exposure caused this claimant's damage.

In tort generally, establishing causation of disease and ill-health faces problems that are not present with respect to traumatic injury, since the causal links between injuries such as broken bones, or even acute toxic effects, are far more intuitive.[64] Some of PFAS' special qualities might exacerbate the challenges. The persistence of PFAS in the environment means that their impact may far outlast those who produced, used and disposed of them, and also creates challenges for gathering evidence, exacerbating the challenges discussed here.[65] The claimant is unlikely to have detailed records of activities and exposure prior to discovery in litigation. But

61 *A Bradford Hill*, The environment and disease: association or causation? Proceedings of the Royal Society of Medicine 1965, 295 'it will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends upon the biological knowledge of the day', 298 (of course he was not looking at establishing causation for the purposes of tort, but for the purposes of occupational health). Note also *P Laleng*, Sienkiewicz v Greif (UK) Ltd and Willmore v Knowsley Metropolitan Borough Council: A material contribution to uncertainty (2011) Modern Law Review 777 comparing *Sienkiewicz*' discussion of epidemiology with the controversy in the 1960s over using actuarial evidence in calculating damages.

62 *McIvor* (2023) 21(4) Med L Rev; *Siegerink et al* (2016) EJRR 175; *Ward* (2016) EJRR 580. The influential (and stringent) Bradford-Hill approach to epidemiology is specifically about how to '[deduce] "causation"' from association in the absence of an understanding of precise causal mechanisms, see *Bradford Hill* (fn 61), providing nine 'different viewpoints' (not criteria) from which to consider causation. See also the reports from the C8 Science Panel (fn 46).

63 The strength of the necessary correlation might depend on the nature of the epidemiological and other evidence, but might involve more than a doubling of the risk. For example, Snow's cholera study showed 14 times the death rate associated with the polluted versus the non-polluted source of water, see *Bradford Hill* (fn 61). Equally, a doubling the risk requirement might be considered overly demanding in the face of other factors of the case.

64 *J Stapleton*, Disease and the Compensation Debate (1986); *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32.

65 Note, for example, the 'vagueness' of the evidence on the claimant's exposure between 1982 and 1997 in *Holmes*.

the uncertainties discussed in this section are not unique to PFAS. Our difficulties in tort are part of a much broader, shockingly poor understanding of the potential impacts of chemicals. Notwithstanding the advances of REACH's 'no-data, no market' approach, the burden of establishing the need for regulation is (simplifying) basically on regulators and governments, and on civil society to convince them. When tort is used as a last resort, after the fact of injury, it reflects pervasive societal uncertainties and inequalities.

B Multiple possible causes

There are many ways in which multiple causal factors can arise to complicate a single case. I will not try to categorise and label them, largely because scholars and courts use the various labels in different ways that cannot always be helpfully mapped onto each other, and nor will I consider whether there might be more appropriate alternatives to the language of causation. Instead, in this section I work through some of the possibilities with respect to PFAS.

First, we can imagine a number of manufacturers and users of PFAS introducing PFAS into water, soil or the claimant's body. The claimant may not be able to establish in this case that but for any one of them (but for the defendant's negligence, activity or defective product), there would have been no damage.

The courts have been dealing with multiple polluters since at least the mid-19th century. The New Zealand Supreme Court in the recent climate tort case of *Smith v Fonterra* observed that: 'As a consequence of the long, global industrial revolution, the common law had to deal with new, widespread risk and damage caused by air and water pollution and the escape of biohazards'.[66] The orthodox position has long been that the claimant may establish causation by establishing that the defendant made a material contribution to the damage. The New Zealand Supreme Court confirms that the 19th century cases continue to apply, and rejects the defendants' argument that the 'material contribution' approach does not apply if some of the multiple polluters are not before the court.[67] This was a strike out decision, and the New Zealand Supreme Court explicitly leaves for trial issues associated with 'newer technologies and newer harms ([greenhouse gases], rather than sewage and other water pollution)'.[68] The magnitude of the complexities associated with climate change is even greater than for PFAS contamination cases.

66 [2024] New Zealand Superannuation Cases (NZSC) 5, [157].
67 This had been fatal before the Court of Appeal.
68 [166].

The UK Supreme Court has also recently reaffirmed the continued legitimacy and generosity of this industrial revolution case law. *Financial Conduct Authority v Arch Insurance* (FCA)[69] was a case about insurance for business interruptions during COVID lockdowns. Simplifying a little, coverage was for the effects on the business of the occurrence of a notifiable disease within a specified distance of the business. Many such cases of notifiable disease (COVID-19) occurred. The problem was that these 'local' cases could not be said to be a but for cause of the lockdown, and the resulting effects on the business. Whilst it is evidently not a tort case, the Court used tort cases and tort scholarship to resolve this issue.[70] The Supreme Court referred to the industrial revolution pollution cases:

> ... where multiple polluters discharge hazardous waste into a river ... each individual contribution is reasonably capable of being regarded as a cause of the harm that occurs, even though it was neither necessary nor sufficient to cause the harm by itself.[71]

Whilst in some cases, the defendant's contribution may be necessary (without it, no damage) or sufficient (without the others, damage anyway), *FCA* is wholly explicit – the defendant can be held to have materially contributed to the damage even if their contribution was neither necessary nor sufficient.

We can easily imagine the significance of this line of case law in PFAS contamination cases: defendants will not be allowed to point to existing pollution or to other sources of PFAS and say the contamination of the defendant's property or person is not their doing. And whilst the old cases are public and private nuisance cases, 'material contribution' can also apply more broadly. In its emphatic confirmation that 'but for' causation does not exhaust liability, the Supreme Court in *FCA* uses examples beyond nuisance, including:

> 20 individuals who all combine to push a bus over a cliff. Assume it is shown that only, say, 13 or 14 people would have been needed to bring about that result. It could not then be said that the participation of any given individual was either necessary or sufficient to cause the destruction of the bus. Yet it seems appropriate to describe each person's involvement as a cause of the loss. Treating the 'but for' test as a minimum threshold which must always be crossed if X is to be regarded as a cause of Y would again lead to the absurd conclusion that no one's actions caused the bus to be destroyed.[72]

69 (2021) UKSC 1; *J Stapleton*, Unnecessary and Insufficient Factual Causes (2023) 16(2) Journal of Tort Law 351.

70 *Stapleton* (2023) 16(2) Journal of Tort Law 351. But note that Lord Briggs' minority, concurring, judgment was more embedded in the construction of the contract.

71 *FCA* [185]. The Supreme Court provides a number of examples, including classic variations on two shooters, as well as the German 2023 directors' case, as adapted by Stapleton.

72 [184], see also *Stapleton* (2023) 16(2) Journal of Tort Law 351 for the source of the example.

There is also long-standing case law on the application of the 'material contribution' test to appropriate personal injury cases. In the leading case of *Bonnington Castings v Wardlaw*,[73] dust was produced in the workplace, some through the negligence of the employer and some without negligence – 'innocent' and 'negligent' dust – which combined to cause personal injury. The defendant was liable for a negligent material contribution to the plaintiff's disease.

The disease in this case was pneumonoconiosis, a *divisible* injury (where every bit of exposure makes the disease worse). The extent of the defendant's liability was not explored in *Bonnington*, but subsequent cases have clarified that with respect to a divisible injury, the defendant's liability is proportionate to their contribution.[74] I discussed the scientific uncertainty in *Holmes v Poeton* above. In terms of legal doctrine, the defendants had attempted to distinguish *Bonnington* (applied by the High Court) on the basis that Mr Holmes' Parkinson's disease is an *indivisible* disease (ie one either has the disease or does not) and that 'material contribution' cannot apply to a case of indivisible rather than divisible injury.

The Court of Appeal in *Holmes* explores *Bonnington* and later cases in depth. It confirms that *Bonnington* can indeed apply to an indivisible disease, and 'the fact that the disease actually at stake in *Bonnington* was divisible does not assist' the defendant.[75] *Bonnington* is about 'a partial cause of the whole injury, as distinct from being a cause of part of the injury'.[76] Citing Lord Rodger,[77] the Court of Appeal holds that *Bonnington* is 'classic authority' for material contribution as a more generous *alternative* to the but for test. The Court of Appeal in *Holmes* also cites Lord Rodger for the proposition that it is 'hard – and settled law – that a defendant is held liable in solidum, even though all that can be shown is that he made a material, say 5 %, contribution to the claimant's indivisible injury.'[78] So if the injury is indivisible, the defendant carries the entirety of the damage (subject to seeking contribution from others). PFAS, 'forever chemicals', may well outlast some of those who produce, use or dispose of them. This distinction between a divisible or an indivisible injury becomes decisive as to whether claimants or any remaining defendants bear the risk of absent wrongdoers.

73 [1956] AC 613. *Steel/Ibbetson* (2011) CLJ 451 on the genesis of *Bonnington* in the nuisance line of authority.

74 Eg *Thompson v Smiths Shiprepairers* [1984] Queen's Bench (QB) 405 on industrial deafness.

75 [46]; leave to appeal was refused by the Supreme Court, SC2024/022 on the grounds that there was 'no arguable point of law and not a point of law which ought to be considered at this time'.

76 *Williams v Bermuda Hospitals Board* [2016] United Kingdom Privy Council (UKPC) 4 (cited in *Holmes*).

77 In *Barker v Corus UK Ltd* [2006] 2 AC 572 (acknowledging that Lord Rodger was dissenting in the case).

78 *Holmes* [58].

PFAS defendants may well have an incentive to press the 'immateriality' of their own contribution. The threshold that must be reached for a contribution to be 'material' has never been precisely pinned down. In *Bonnington* itself, the Court took a clearly generous approach, only excluding *de minimis* contribution;[79] similarly in the quotation from Lord Rodger above, we would certainly not be excluding anything above a 5 % contribution.

In terms of the factual situation, the High Court had found that Mr Holmes' Parkinson's disease was caused not *either* by genetic *or* by workplace exposure, but that there is a 'complex interplay between genetic and environmental factors and that both factors work together to produce the condition'.[80] 'Material contribution' can apply if some of the contributing factors are non-negligent, or contingent in some way. So genetic pre-dispositions of the claimant to a cancer associated with PFAS exposure, or other sources of exposure, would not in themselves prevent the *application* of the 'material contribution' test (although of course it may not be satisfied). There is, for example, evidence that certain PFAS can initiate prostate cancer in individuals where a first-degree family member has prostate cancer.[81]

In these cases, we see a defendant whose negligence (or activity or defective product) can be said to have contributed to the harm. A quite different case arises where it is impossible for the claimant to prove that the defendant's negligence *either* caused *or* materially contributed to the harm. In the cases above, the defendant was causally involved in a way that makes it appropriate to allow causation: here the claimant cannot establish this, since something else entirely may have caused the damage. This is certainly plausible for PFAS. It might be, for example, that genetic susceptibility and PFAS operate independently, so that one or the other caused the disease; or it might be that a low level of PFAS exposure causes (or may cause) the disease, so that any one of a number of exposures could have caused the disease.

This brings us to the famous/notorious *Fairchild* exception in English law. In *Fairchild,* a number of claimants, all of whom died of mesothelioma, were negligently exposed to asbestos by more than one employer. Mesothelioma is a signature disease, caused only by asbestos. The parties accepted that the greater the quantity of asbestos dust and fibres a person inhales, the greater the risk of developing mesothelioma; at the time, the scientific evidence was that the disease could have been caused by many asbestos fibres, just a few fibres or a single fibre,[82] and the way in which the impact of different fibres might interact was not known. The claimants

79 *Bonnington* at 621.

80 [96].

81 *Brunn et al* (2023) Environmental Sciences Europ 20.

82 The single fibre theory no longer seems to be accepted.

could not therefore prove on the balance of probabilities that any single defendant or combination of defendants had either caused or materially contributed to the damage. The claimants lost for that reason before the Court of Appeal.

The House of Lords held, however, that a sufficient causal link exists if the claimant can establish that the defendant materially contributed to the *risk* faced by the claimant, if that risk subsequently materialises. *Fairchild* applies only in cases where it is *impossible*, because of the limits of science, to prove causation. Subsequent case law has added both refinements and 'wrinkles' to the *Fairchild* exception. First, it is clear that *Fairchild's* innovation is in causation, not in the definition of damage: the damage is the mesothelioma, not the risk.[83] Secondly, *Fairchild* can apply when there is a single tortious possibility, alongside other non-tortious potential causes,[84] including if some of the asbestos exposure is down to the contributory negligence of the claimant.[85] Further, as with material contribution to damage, the 'material' contribution to risk by the defendant does not have to be significant, just more than *de minimis*.[86] Thirdly, in an unorthodox approach to compensation for an indivisible injury, the Supreme Court in *Barker* held that if *Fairchild* applies, defendants are responsible for damages proportionate to the risk imposed by the tort (rather than full damages).[87] That has been overturned by legislation in respect of mesothelioma,[88] but continues to apply in respect of other diseases, and explains why the claimant in *Holmes* had not claimed under *Fairchild*.[89] Again, given the persistence of PFAS in the environment, and the likely latency of disease, some potential defendants are very likely to be missing by the time of a successful tort claim; proportionate liability places that risk on the claimant rather than the defendant.

The application of *Fairchild,* even if environmental exposure to asbestos is a possible alternative to the defendant's negligence, is particularly pertinent for PFAS, given the ubiquity of PFAS in the environment. PFAS are, however, even more complicated than the mesothelioma at stake in *Sienkiewicz*, where this proposition was established.[90] All of the possible causes or agents of the mesothelioma were asbestos

83 Eg *Durham v BAI (Trigger Litigation)* [2012] 1 Weekly Law Reports (WLR) 867. That had been open to question following *Barker v Corus UK Ltd* [2006] 2 AC 572.

84 *Sienkiewicz*; *Barker*, where the contributory negligence of the claimant during a period of self-employment was not a barrier to applying *Fairchild*.

85 *Barker* [83].

86 *Sienkiewicz* [51].

87 Speaking to the discussion of scientific uncertainty above, *R Merkin/JC Steele*, Causation and Proportional Recovery, in: K Barker/R Grantham (eds) Apportionment in Private Law (2018).

88 Compensation Act 2006.

89 *Holmes* [3].

90 The claimants in *Fairchild* were almost certainly also exposed environmentally, but the decision assumes that the entire risk was created by the defendants.

exposure, whilst the bodily harms at stake with respect to PFAS (kidney or testicular cancer, immune system problems, etc) have many possible causes other than PFAS. If these factors can be shown to interact, the 'material contribution to damage' line of case law may be an option; if they are, or could be, independent, that is not possible.

Whether *Fairchild* could be applied to the multiple possible causes of disease associated with PFAS is open to question. The 'single agent' (all the potential agents of the damage are the same – asbestos dust) notion was understood to be an important (albeit pragmatic rather than principled) limitation on the application of *Fairchild* in its early years. The main confounding factor that has arisen in occupational disease cases has been the claimant's smoking. Lord Hoffmann in *Barker* says explicitly that:

> I do not think that the exception applies when the claimant suffers lung cancer which may have been caused by exposure to asbestos or some other carcinogenic matter, but may also have been caused by smoking and it cannot be proved which is more likely to have been the causative agent.[91]

In *Heneghan v Manchester Dry Docks Ltd*,[92] the possible causes of the claimant's lung cancer included not only the negligent exposure to asbestos by the six defendant employers and some other employers not before the court, but also the claimant's smoking. The Court of Appeal applied *Fairchild* without engaging with the single agent criterion. The defendants, however, had conceded liability, that asbestos had caused the lung cancer, and *Fairchild* was used to decide which defendants should pay for what. But although the smoking was not therefore discussed by the Court of Appeal, the upshot of *Heneghan* was to apply *Fairchild* to a case where there was more than one potential agent of the disease.

If that is followed generally, it could of course be helpful for claimants in PFAS cases. It is however challenging for tort law. As Sarah Green observes, employers and others create risks all of the time, whenever they do anything that is capable of leading to an untoward outcome, and so *Heneghan* is potentially an extension without limit.[93] She argues that the 'single agent' constraint on *Fairchild* ensures that the

91 [24].

92 [2016] EWCA Civ 86.

93 *S Green*, *Fairchild* and the single agent criterion (2017) Law Quarterly Review 25. In *Holmes*, the Court of Appeal 'agrees' with Green that *Heneghan* is an extension of *Fairchild*, but does not engage with her concern that the extension is effectively without limit. Note also that Dyson LJ in *Heneghan* [45] relied on Lord Hoffmann's discussion of 'risk' (not mesothelioma) as damage in *Barker*, which is no longer sustainable, *Merkin*/*Steele* (fn 87).

very risk to which the defendant exposed the claimant materialises.[94] We might add that it takes us beyond any sense that *Fairchild* was designed to avoid a number of negligent employers effectively hiding behind each other's wrongdoing to avoid liability,[95] although the same applies of course to the environmental exposure in *Sienkiewicz* or the contributory negligence in *Barker*. It is at least open to question whether *Heneghan* would be applied to the diseases currently associated with PFAS, given the factual agreement that the Court of Appeal was dealing with. Perhaps the earlier case of *Novartis v Cookson*[96] provides some middle ground. The claimant alleged that his bladder cancer had been caused by negligent workplace exposure to aromatic amines. The point was not taken by the parties, but the Court of Appeal suggests that *Fairchild* can apply in this case because the amines in cigarette smoke 'operate in the same way' as the amines in occupational exposure.[97]

Whether seeking to apply *Heneghan* or the more measured flexibility in *Novartis,* we might bear in mind the Supreme Court's cautious approach to *Fairchild*: Lord Hoffmann, who gave an important speech in *Fairchild*, has, for example, suggested that it teaches a 'lesson of caution ... in relation to future invitations to depart from conventional principles of causation.'[98] *Fairchild* is important, however, not only for the scope of the exception it creates, but also for indicating that the courts recognise that sometimes a strict application of the rules on causation would lead to injustice. When courts intervene in evidential or conceptual aspects of causation out of fairness for claimants, there may be further complexities and disagreement around the scope of responsibility for damages: *Fairchild, Barker* and the Compensation Act 2003 are evidence of that.[99] These questions in some cases will simply push the need for courts to grapple with uncertainty elsewhere in the analysis, although the English courts seem largely to have taken a pragmatic approach to allocating responsibility between defendants.[100]

94 The single agent was an important way of distinguishing the earlier case of *Wilsher v Essex Area Health Authority* [1988] AC 1074: 'there was nothing to show that the risk which the defendant's staff had created – that the plaintiff would develop [retrolental fibroplasia] because of an unduly high level of oxygen – had eventuated', Lord Rodger in *Fairchild* [149]. See also Lord Hoffmann in *Barker* [24].

95 *McIvor* (2013) 21(4) Med L Rev. In Europe, note also *Winiger*/*Koziol*/*Koch*/*Zimmermann* (fn 31) to the effect that 'each of the potential tortfeasors would then be in a position to exonerate himself by referring to the other tortfeasors', 523.

96 [2007] EWCA Civ 1261 (fn 57).

97 [72].

98 *Sienkiewicz* [189]. Note also *AB v MoD* [157].

99 *Stapleton* (2023) 16(2) Journal of Tort Law 351.

100 *Merkin*/*Steele* (fn 87) argue that any apportionment is necessarily rough and ready, although noting that whilst contribution between tortfeasors under the Civil Liability (Contribution) Act 1978

IV Conclusions

A feature that PFAS litigation shares with other 'toxic torts', not unrelated to causation, is its David and Goliath nature. Communities of 'ordinary' workers, residents and consumers are pitted in lengthy proceedings against some of the world's most powerful global organisations. This has, for example, been very apparent in long-running litigation against Shell, around the appalling pollution of the delta region that is the claimants' home and livelihood. The Court of Appeal has observed 'what may politely be called an asymmetry of information' and notes that the 'very substantial sums [spent] on the litigation so far' would be 'relatively trifling' for Shell.[101] This asymmetrical pattern underpins not only PFAS torts, but also the scientific uncertainty that pervades the broader regulatory context.[102]

Our discussion of causation suggests that the focus of tort in this area is likely to remain on property damage, and in some jurisdictions on elevated levels of PFAS in the blood.[103] Scientific evidence develops, as does the law, and unusual cases with clearer causal mechanisms may arise, for example with respect to more established, better understood and (probably) banned substances. But as things stand, it is difficult to imagine many claimants successfully establishing causation in respect of diseases associated with PFAS, certainly in English law; the absence of such cases across Europe suggests similar calculations. The focus on property is the result of sensible litigation tactics, but it is not a neutral fact. When a key tool for justice in an area of public harm and scandal focuses on property over bodies, sometimes to the dismay or bewilderment of the communities concerned,[104] it sends messages about what is valued and what is discounted.

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or contributory negligence under the Law Reform (Contributory Negligence) Act 1945 are explicitly inexact, the common law *Fairchild* approach seems to be appealing in principle.

101 *Alame v Royal Dutch Shell Plc* [2024] EWCA Civ 1500 [5] and [81]. This case is part of ongoing pretrial litigation which includes causation disputes. The jurisdiction of the English courts was established by the Supreme Court *Okpabi v Royal Dutch Shell Plc* [2021] UKSC 3.

102 *W Wagner*, When a corporation's deliberate ignorance causes harm: Charting a new role for tort law (2022) DePaul Law Review 413 argues that the defendants in toxic tort cases are able to 'control the information environment relevant to causation', at 415. Note also *N Gaber*/*L Bero*/*TJ Woodruff*, The devil they knew: Analysis of industry influence on PFAS science (2023) Annals of Global Health 1.

103 In English law, fn 32.

104 Note the comments of the interviewees in *Legg*/*Prior* (2023) Geographical Research 234. The dismay of the community at the exclusion of one of the campaigners from litigation for tactical reasons was dramatised in *Toxic Town* (Netflix 2025); Tracey Taylor, mother of Shelby-Anne who died a few days after birth, was interviewed on the BBC, <https://www.bbc.co.uk/sounds/play/m0028stn> (at about 32 minutes). See *The Claimants Appearing on the Register of the Corby Group Litigation v Corby District Council* [2009] EWHC 1944 (TCC). For an earlier appeal on a point of law, [2008] EWCA Civ 443.

In these sorts of cases, tort law is in part a tool in a broader search for political as well as legal strategic impacts, aiming for a form of justice that is broader than the parties before the court. For some, this is obviously anathema. Beyond concern about the integrity and autonomy of tort, however, there may be risks in devoting energy and resources to courts, rather than political or regulatory change – and our knowledge problems with PFAS are not just about tort, they are fundamentally about the way our world is shaped economically and politically. Further, social resources may be devoted to the justiciable over the significant – in this case to property over bodies. On the other hand, the shift in perspective that goes along with seeing PFAS as something more than a series of cases within the vast canon of tort can highlight systemic gaps with respect to chemicals. Even if the (causal) connection between the parties is generally considered crucial to the 'correlative' form of justice that tort provides,[105] when causation poses such a consistent barrier in the interests of the powerful, it is worth asking questions about who should bear the cost of ignorance. Our lack of knowledge and understanding of chemicals is one element of the imbalance between corporations using PFAS and individuals harmed by them. This may be at least a relevant issue when considering the structure of the legal response when things go wrong. In the absence of special state-mandated schemes, what happens (legally) when things go wrong usually depends on tort.

The challenges faced in respect of PFAS are different from other diseases perhaps only because of the potential scale of the impact of PFAS, which are widely used, poorly understood, and both ubiquitous and persistent in the environment. This has, of course, just been a sketch of possible issues.

105 *P Cane*, The Anatomy of Tort Law (1997).