ANNEX 3:

Quantifying Plastic Risk to Corporates and Their Insurers

MINDEROO FOUNDATION

Praedicat

Contributing Authors

Adam Grossman, Ph.D.,
Vice President,
Emerging Risk

Sheryll Mangahas, M.S.,
Director of Modeling
Production Operations

Arianna Libera, Ph.D.,
Environmental Scientist

CONTENTS

INTRODUCTION

The preceding annexes of this report outline the many risks posed by plastic: dozens of chemicals that can harm human health, plastic becoming a fixture in the natural environment, and the omnipresence of micro- and nanoplastic (MNP) particles becoming well established alongside the potential risks to human health and the environment.

In this annex we take the information developed in the rest of the report along with the broader scientific literature and forward-looking loss modelling to estimate the potential impacts of microplastic on economic loss to plastic-related companies and to their insurers.

First we will describe the approach to modelling and predicting economic burden and cost to industry for claims that plastic-related hazards cause bodily injury. Then we discuss individual hazards related to plastic and their modelled losses. We also discuss a scenario where companies are forced to remediate wastewater and/or drinking water contaminated with microplastic. Finally, we conclude with a discussion of how these hypothetical mass torts could affect the insurance industry.

THE MODELLING APPROACH

Risk models inevitably simplify the complex phenomena which they seek to describe, and the liability risk environment is as complex a risk as any the insurance industry covers. It involves the interaction of parties to litigation within a framework of evolving law and changing legal institutions, where injured parties (plaintiffs) are seeking compensation for an enormous range of harms allegedly caused by the activities or products of defendants occurring anywhere across the entire economy. Furthermore, in recent years the plaintiffs' bar has become more innovative within the litigation environment as their strategies have become more sophisticated and the causes of action more creative. Simultaneously, litigation funders increasingly provide capital to support the resulting litigation.

Abstracting from this complexity, our approach describes the "traditional toxic tort" mass litigation phenomenon of claims that a chemical, product, substance, or business practice caused bodily injury, claims that are built on top of scientific evidence showing that the hazard can indeed cause the harm. The canonical phenomenon is based upon asbestos litigation, which to this day remains the largest loss in insurance history. As was true for asbestos, the development of the underlying scientific evidence provides an early warning for future litigation as the scientific literatures develop, one paper at a time, over many years.

We use text mining technologies to identify these literatures in the earliest stages of scientific advances that show a commonly used chemical, product, substance, or business practice can cause bodily injury. The resulting model, built to fit the canonical case, tracks closely with many emerging litigation risks, including Roundup and talc. In addition, many recent litigation phenomena with more innovative causes of action, such as the government-driven opioids litigation, or the litigation over water contamination by PFAS, are nonetheless similarly built upon a foundation of science, and the resulting models remain highly predictive.

Latent mass actions

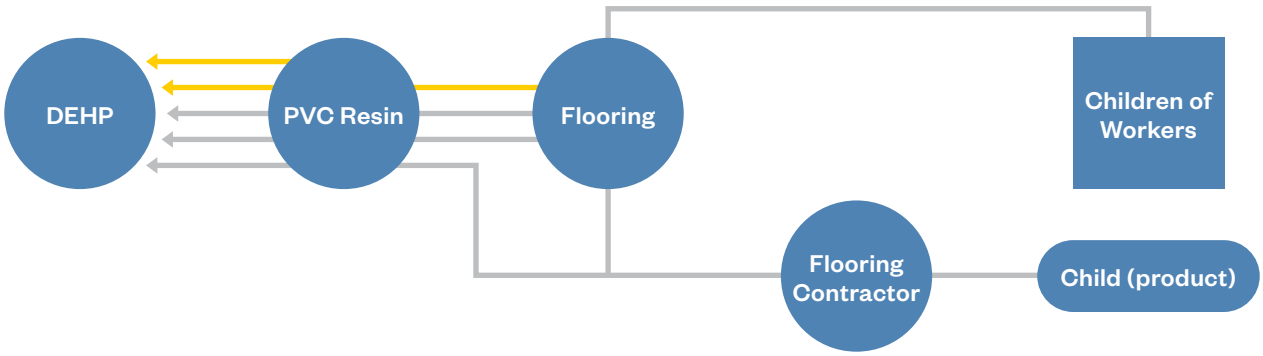
An identified hazard can be characterized by the set of hypothetical mass torts that would involve that hazard. We call these hypothetical lawsuits Latent Mass Actions, or LMAs. They are latent in the sense that they have not yet begun, and they are mass actions because they represent the possibility of a legal action involving large numbers of people.

Identifying LMAs relies on the same text mining approaches we use to identify hazards. In their research, scientists not only identify the potential links between chemicals and harms; they also identify the ways in which people are exposed to those hazards. Federal and state regulators also identify how workers and consumers are exposed to these hazards, including highlighting exposures of particular concern. We use the collected wisdom embodied in these scientific and regulatory literatures to describe each LMA according to the following properties:

1. The type of plaintiff (worker, consumer, adult, child, public entity)
2. The hazard
3. The harm caused by the hazard
4. How the hypothetical plaintiff is exposed to the hazard
5. The stream of commerce that took the hazard from its creation through to the exposure

The last two items are expressed in terms of the industries potentially implicated in each hazard's stream of commerce. See **Figure 1** for an example of a "map" of five LMAs for the plasticizer di(2-ethylhexyl) phthalate (DEHP). In the figure, each LMA is represented as a line that originates at a group of plaintiffs, such as Children of Workers, and the line passes through the stream of commerce of defendant industries named in the LMA. The colour of the line represents case strength, with higher case strength closer in colour to red. Litigation maps can be used to capture the correlated risk from mass torts across industries in an insurer's portfolio.

Figure 1: Sample litigation map segment showing five latent mass actions related to di(2-ethylhexyl) phthalate and flooring-related exposures.



Characterising case strength

The next step in our analysis assesses the strength of the plaintiffs' case for each LMA. We approach the problem by representing the stages of analysis and types of evidence a hypothetical plaintiff would need to introduce in court to prove his case.

The first step requires the plaintiff to establish that the alleged cause of the injury is capable, in general, of causing that injury. We refer to this as establishing general causation (GC). In bodily injury litigation, GC is established by having expert witnesses testify that the scientific literature supports the theory that the named hazard can cause the specified harm. We've built a [patented] GC risk score that mimics the kind of review done by expert witnesses in court using the Hill Criteria[1] to opine that the alleged cause of injury could, in principle, cause the harm. The GC risk score analyses a scientific literature by looking at the contents of each underlying peer-reviewed article and aggregates them into a score between -1 and +1 that represents the agreement in the scientific literature that the human harm hypothesis is established. For example, hypotheses that asbestos causes mesothelioma and that benzene causes leukemia have GC risk scores of +1. Scores below zero indicate rejection of the hypothesis while scores between 0 and 1 reflect growing certainty in the human harm hypothesis. Literatures with no human literature cannot exceed a GC risk score of 0.5 while literatures that only have human literature cannot exceed a score of 0.75.

After establishing the hazard involved in the LMA can, in principle, cause the harm the plaintiffs then turn to "specific causation", where they show that the individual plaintiff was, in fact, harmed by the hazard in the manner laid out in the complaint. Plaintiffs accomplish this by showing that they were exposed in a way that can cause the harm and that no other potential cause of the disease was likely to have been relevant to their case. Plaintiffs also need to establish that the defendants involved in their case are those who exposed them without appropriate warning that their product(s) could cause bodily injury.

The end result of this analysis, called Case Strength (CS), represents the likelihood that a plaintiff can marshal enough evidence to win his case. All else being equal, plaintiffs will pursue cases with higher strengths that therefore have a higher likelihood of success. Absent specific circumstances (such as discovery of explicit evidence of wrongdoing), cases with scores below 0.30 are generally fairly weak. Once the score reaches 0.70, the plaintiffs' case is quite strong, and litigation becomes far more likely. In later sections we will discuss the GC and CS scores for several LMAs. In those discussions we will label CS scores below 0.30 as "weak" and above 0.70 as "strong".

Simulating litigation events

We model the likelihood that mass litigation begins by considering the same types of things a plaintiffs' lawyer would: the strength of the case and the size of the potential financial return for successfully pursuing it. Combining the case strength as computed above along with an estimate of the number of people potentially exposed to the hazard alongside the potential recovery of each plaintiff yields the probability of litigation initiating. In each of the eight years of our forward-looking simulation, we calculate the probability litigation begins by simulating the appearance of the scientific literature and calculating its effect on case strength. In each future simulated year, we flip a coin with the given probability to determine whether litigation, in fact, begins for each group of LMAs in that year. We do this 1,000 times to generate alternative outcomes.

If a litigation event is simulated to begin, we calculate the factors necessary to determine the total amount each plaintiff will be awarded in the litigation and which insurance policy periods could be implicated by that plaintiff's case.

When a litigation becomes active in the simulation, we also simulate the accrual of defense costs. We then simulate the outcome of each litigation event according to the probability given by CS, and in simulations where the plaintiffs win the defendants then are obligated to pay indemnity in addition to the defense costs.

Once the total cost of litigation has been determined for each litigation event, we spread the responsibility for that cost to the various defendant industries involved in the litigation. We do this by first analyzing the LMAs involved in the litigation and the business activities involved in bringing the litigated hazard from its creation through the stream of commerce to the final product manufacturers exposing the plaintiffs to the hazard (see Figure 1). We also consider the size of the industry, as the potentially responsible parties need to be sufficiently large or numerous for plaintiffs to be able to recover significant settlements.

Plastic-related chemicals

The range of chemicals used to make plastic products is astounding. Plastics begin as long chains of repeating molecules that determine the plastic type and its main characteristics. Polycarbonate, for example, is made from bisphenols while polyethylene is made from linear hydrocarbons derived from petroleum. The polymer backbone, though, is only the beginning. To make a plastic that has all the properties needed for any particular use the manufacturers add other chemicals that serve a variety of functions: pigments and dyes, plasticizers, and flame retardants.

In this section of the report we discuss several groups of chemicals used in many aspects of plastic production. We will first take a look at bisphenols, commonly used to make polycarbonate plastic. Then we direct our attention to plasticizers commonly used in various plastics – phthalates and their replacements like N-butylbenzenesulfonamide (NBBS). Next, we examine the risk profiles of two categories of flame retardants: brominated flame retardants and phosphate-based flame retardants. Finally, we briefly look at per- and polyfluoroalkyl substances (PFAS), which although not commonly used directly in plastic, can be created as part of secondary processing of plastics with fluorine gas to improve their durability.

In each of the sections that follows we will describe the use of each group of chemicals and how people are exposed to them. We follow that with a summary of the main issues being investigated in the scientific literature and the resulting General Causation risk and Case Strength scores. Lastly, we give a high-level overview of the simulated losses for each of these groups and the industries at risk.

We express losses at different portions of the simulated loss distribution. We use three statistics commonly employed in property catastrophe modeling:

- **Expected Loss (EL)**: The average loss across all simulations regardless of whether a litigation event initiates.
- **Probable Maximum Loss x% (PML(x))**: The loss value exceeded with x% probability. For example, the PML(5) represents the value exceeded by 5% of simulations and therefore exceeds the other 95%. We report PML(5) and PML(1).
- **Tail Value at Risk x% (TVaR(x)):** The average loss in the most expensive x% of simulated outcomes.

All losses are expressed with respect to a concept called "all future losses", which includes any loss that could conceivably be attributed to an insurance policy written this year on the occurrence form. These numbers therefore include all people whose exposures commenced before the end of the current policy period, and all claims filed on or after the start of the current policy period, provided that the litigation is simulated to begin during the eight-year simulation our model provides.

Bisphenols

Bisphenols form the polymer backbone of polycarbonate plastic, a type of plastic used primarily because it can be made to be both clear and shatterproof. Polycarbonate plastic is often used to make containers for liquid, including baby bottles, and cases for consumer electronics. Bisphenols can leach out of their products, which is particularly concerning when it results in significant infant exposure. In 2012, the U.S. Food and Drug Administration (FDA) banned the use of bisphenol A (BPA) in infant feeding bottles and spill-proof cups.

Bisphenols are also used to make epoxy resins that are applied to metal to prevent corrosion. The most common use of epoxy resins in this vein is to line the inside of metal food cans to lengthen the viable storage time of the food within them. The FDA extended its infant bottle BPA ban to include epoxy resins used in packaging for infant formula in 2013.

While polycarbonate and epoxy are the two main uses for bisphenols, they are also found in thermal paper (like the kind used for store receipts), pesticides, polysulfone plastic, and polyvinyl chloride (PVC) plastic.

Exposures

Bisphenols are ubiquitous in the environment – they have been found in water, sediment, indoor dust, and indoor air. Bisphenols have been found in the urine and blood of nearly all people, and have also been found in infant meconium. Given that most bisphenols have a relatively short half-life in the human body this means we are exposed to them nearly continuously.

Humans are exposed to bisphenols when we ingest food or liquid from polycarbonate containers. Exposure to bisphenols also occurs from processed food from metal food cans with epoxy resin lining. We can also absorb bisphenols through the skin, a common occurrence when handling thermal paper. Higher serum levels of BPA have been found after undergoing dialysis, suggesting BPA is introduced during the dialysis process.

The scientific literature and case strength

The predominant bisphenol used in plastics has been BPA. It was the first bisphenol widely used and to be investigated for bodily injury. Bisphenol A is an endocrine disruptor with the notoriety of being an "obesogen", a chemical known to cause obesity. Bisphenol S (BPS), a common substitute for BPA, is also scientifically proven to be an obesogen. BPA and BPS also cause reproductive injury in humans resulting in infertility. As the negative effects of BPA and BPS become more broadly accepted, manufacturers have substituted other bisphenols in their products. Common substitutions include bisphenol AF, bisphenol B, and bisphenol F. The science for these to result in bodily injury is nascent; however, due to the similar structure to BPA and BPS, scientists believe that these bisphenol substitutions are also likely detrimental to human health.

Bisphenols have attracted significant scientific interest, with scientists having investigated at least 10 different types of harms including cardiovascular disease, cognitive disorders, nervous system injury, and endocrine disruption. BPA has also been studied intensively for its effects on reproduction, which may be due to its endocrine disrupting effects.

Figure 2: Historical General Causation risk scores for bisphenol A

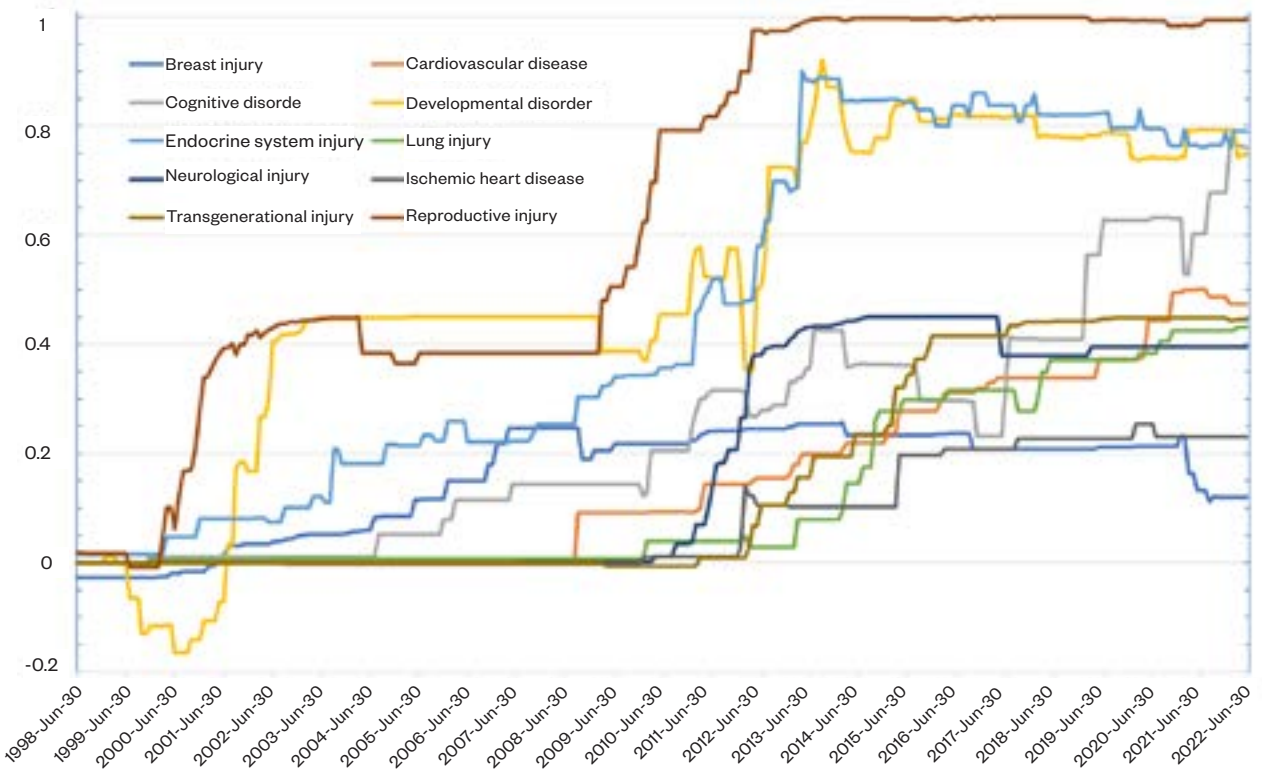


Figure 2 shows a time series plot of the GC risk scores for each of ten harm hypotheses as of mid-year from 1998 until 2022. As discussed above in more detail, the General Causation (GC) risk score measures the level of acceptance in the scientific community that (in this case) BPA can cause 10 different kinds of bodily injury. Scores near zero indicate equivocal evidence while scores approaching +1 indicate strong acceptance of the hypothesis.

All the GC scores for BPA are positive, suggesting causal relationships of varying strength between BPA and these ten harms. The reproductive injury hypothesis is strong; there is no scientific doubt BPA alters the coordination of this system due to a combination of *in vitro*, animal and human literature. A study on male infertility showed BPA has been linked to poor semen quality and DNA damage in multiple studies.[2]

In women, BPA has been linked to precocious puberty.[3] The GC score for BPA exposure resulting in endocrine injury is high, this is due to literature affirming BPA is an obesogen. Embryos, fetuses, and babies exposed to BPA via their parents may experience particularly harmful effects. The GC score for developmental injury is almost as high as endocrine injury with effects in human offspring ranging from lower mental development index scores,[4] low birth weight,[5] lower body mass index (BMI)[6] and development of lung diseases including wheezing and asthma.[7] The cognitive injury GC score for BPA rose the past couple of years due a high publication rate of human studies. One linked BPA as well as BPS and bisphenol F to attention deficit hyperactivity disorder (ADHD).[8]

Figure 3: Current case strength distribution for bisphenols and their substitutes

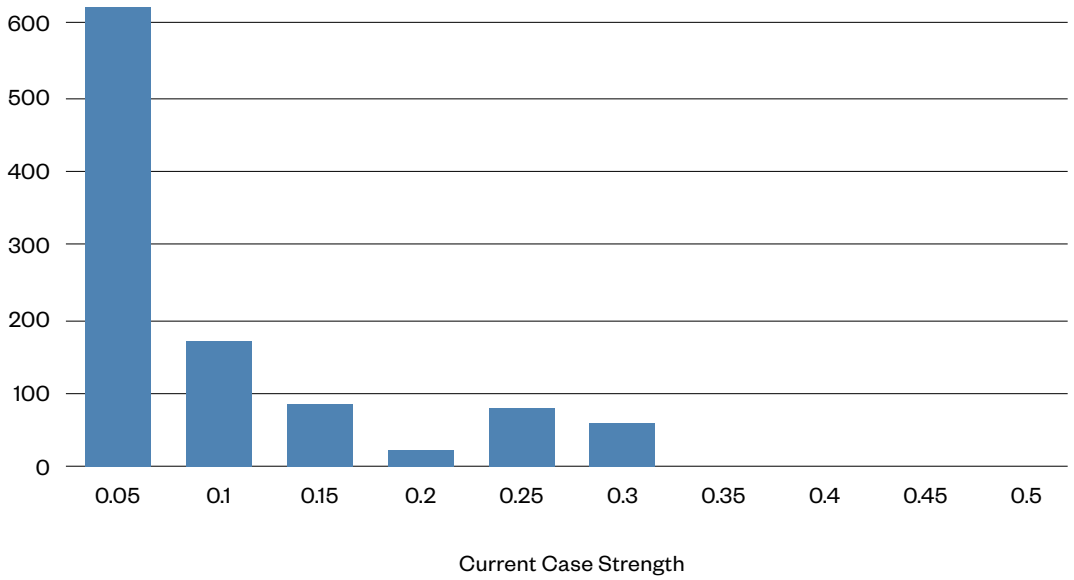


Figure 3 shows the number of Latent Mass Actions (LMAs) for bisphenols that have case strength scores in each of the intervals listed above. While the approach to modelling CS is described fully above, we reiterate here that it models the way a plaintiff must prove his case, first by "ruling in" that the plaintiff's injury could have been caused by the claimed hazard and then by "ruling out" all the other possible causes of his injury, followed by showing that the defendant(s) were responsible for the exposure and therefore injury. The CS score represents the probability that the hypothetical plaintiff could meet that evidentiary burden.

The distribution of baseline case strength for bisphenols is weighted heavily toward zero. This is common, despite the relative strength of GC risk scores showing that BPA and some other bisphenols enjoy broad scientific consensus that human exposure can lead to harm. The ubiquity of bisphenols in the environment and in our bodies means that it's difficult to show that a specific commercial product led to enough exposure to cause disease, and the common nature of many of the harms renders it quite difficult to rule out other potential causes of a hypothetical plaintiff's disease. A large portion of the low case strength LMAs is also due to the inclusion of many BPA substitutes in this plot, most of which have much less scientific agreement that they cause harm.

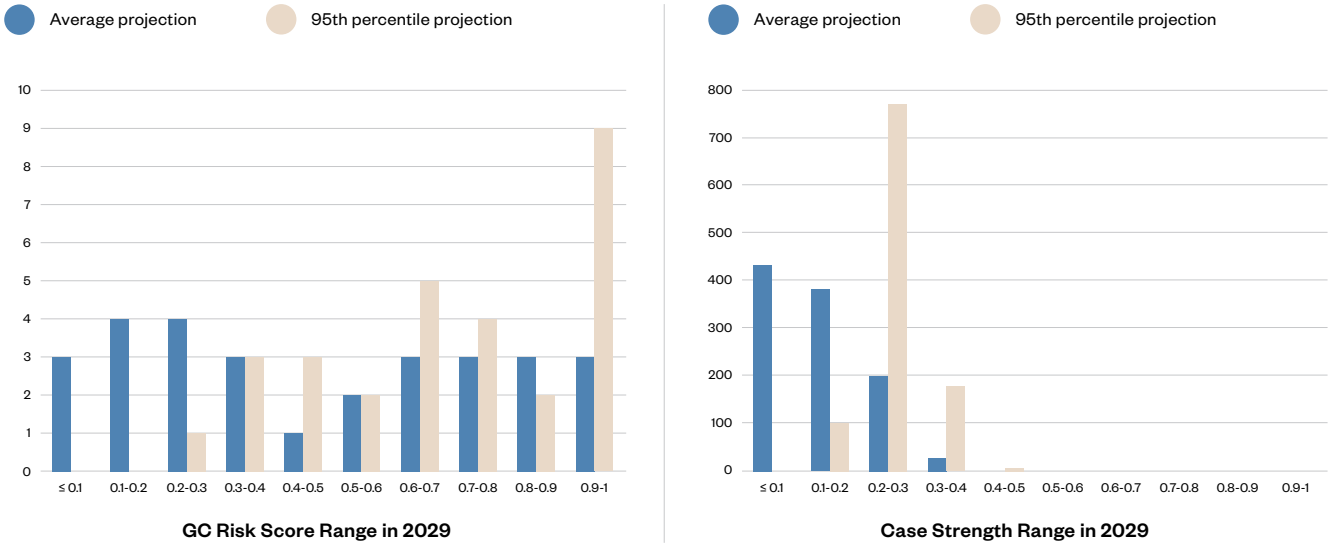
Simulated losses

As discussed in the methods section above, we simulate the potential costs of litigation by projecting 1,000 different future trajectories of the scientific literature and modelling the consequences of that evolution for case strength and the likelihood that litigation begins around any given group of LMAs.

The data presented in this section reflect three stages of that projection: GC risk scores, CS, and overall statistics of litigation likelihood and cost.

Bisphenols have a long history of being studied in the scientific literature. As shown above, this leads to fairly high GC risk scores and low to moderate CS values today. Projecting GC risk and CS scores eight years into the future yields the distributions shown in **Figure 4**.

Figure 4: Histograms of projected General Causation risk (left) and Case Strength (right) for bisphenols. Blue bars represent the average projection while beige bars show the 95th percentile projection.



The left panel of **Figure 4** shows the number of scientific hypotheses that bisphenols cause bodily injury within each band of GC risk score. The blue bars represent the count in each score range at the average projection while the beige bars show the count of hypotheses in each score range at the 95th percentile of projections in eight years' time. Similarly, the right panel of Figure 4 shows the number of LMAs within each CS range in eight years. As in the left-hand panel, blue bars show the count of LMAs at the average projection while beige bars show the same count of LMAs in each CS score range at the 95th percentile of the projection.

We immediately see that while average case strengths rise moderately compared to the distribution shown in **Figure 3**, there is a significant increase in CS at the upper end of the simulated distribution. That means that in a small percentage of possible futures the hypothetical cases against BPA become significantly stronger and therefore more likely to attract the attention of plaintiffs' lawyers. When looking to simulated litigation events, this translates into a relatively high litigation probability and a moderate likelihood of plaintiffs succeeding in recovering damages from the defendants.

Table 1: Litigation model results for bisphenols

Probability of litigation	High	
Litigation success rate	Moderate	
	Total cost	**Defense**
Expected loss	$1.8B	32%
PML(5)	$8.7B	32%
PML(1)	$36.2B	12%
TVaR(5)	$23.8B	16%

The majority of these losses stem from claims alleging that BPA causes various developmental disorders due to exposure before birth or during breast feeding. These diseases include type 2 diabetes, autism, asthma, and morbid obesity. This respects the fact that the scientific literature has reached a relatively strong consensus that the endocrine disruptive effects of bisphenols coupled with the high likelihood of early life exposure can cause a variety of diseases.

Table 2: Top five responsible industries for simulated bisphenols litigation

Bisphenol manufacturers	Expoy resin manufacturers	Food & food containers	Plastic manufacturers	Automobile manufacturers

As described in detail in the methods section, we distribute the total cost of litigation to industrial sectors and specific business activities using the structure of each LMA (example in **Figure 1**). **Table 2** shows the top industrial sectors expected to sustain losses related to bisphenols.

A sizable portion of the cost attributable to bisphenol-related bodily injury litigation is due to its use in thermal paper – a non-plastic use of this chemical group. Much of the remaining liability arises from the use of bisphenols to make epoxy resins. Epoxy resins, while not often discussed as part of the global plastic problem, are commonly used plastics that have found significant use in lining food cans to help preserve the food for longer periods of time by preventing it from reacting with the metal in the can itself.

Bisphenols are used in the manufacture of several other plastics – polycarbonates, polyvinyl chloride, polyether sulfone, polyolefin, and high-impact polystyrene. Most of the loss to this sector is to the polycarbonate and PVC manufacturers. The use of bisphenols in polycarbonate plastic implicates baby bottles, one of the higher profile uses of BPA in particular, and the subject of consumer fraud lawsuits in 2008.

Phthalates and other plasticizers*

Plasticizers are a group of chemicals that increase flexibility and resilience in plastic. Absent these chemicals, many kinds of plastic would be very brittle and easily breakable. While many chemicals can function as plasticizers, the most commonly used type of chemical for this function are esters of phthalic acid, called phthalates. Di(2-ethylhexyl) phthalate (DEHP, sometimes also called bis(2-ethylhexyl) phthalate or BEHP) was the first phthalate to market and is the most studied.

Scientists became concerned that phthalates could cause human injury over 50 years ago, prompting the US Occupational Safety and Health Administration (OSHA) to institute occupational exposure limits for DEHP in 1970. Examples of other phthalates include butyl benzyl phthalate (BBP), dibutyl phthalate (DBP), diethyl phthalate (DEP), diisononyl phthalate (DINP), and dimethyl phthalate (DMP). These other phthalates, particularly DBP, DEP, and DMP, have found uses outside of plastics although DINP is often used as a direct replacement for DEHP. Scientists have hypothesized that these phthalates also cause human injury, although the underlying mechanisms may differ.

Non-phthalate chemicals have been used as plasticizer substitutes for phthalates in plastic. DINCH is a direct replacement for phthalates like DEHP and DINP and is used similarly to them. N-Butylbenzenesulfonamide (NBBS) is a newer plasticizer that has not yet attracted significant attention in the scientific community with respect to its potential to cause bodily injury. However, NBBS persists in the environment and is mobile in water, suggesting that NBBS may become an environmental concern.

Exposures

Humans are exposed to phthalates because of their wide use as plasticizers in polyvinyl chloride (PVC) and polyvinyl butyral plastics. These plastics are used in a wide range of products including bottles, food packaging, construction materials, containers for personal care products, and textiles. They are also used in rubber and commonly found in tires.

Phthalates and their breakdown products have been found and quantified in outdoor air, water, marine sediments, and fish. Indoor air and dust commonly contain phthalates as well. Humans can therefore ingest phthalates through their diets, and we also breathe phthalates in the air and dust. Phthalate metabolites have consistently been found in adult and child urine samples worldwide, suggesting, just as with bisphenols, that we are constantly exposed to phthalates.

As a phthalate replacement, DINCH may be integrated into a variety of products that use phthalates, leaving us similarly exposed. NBBS currently does not have as large a commercial footprint as phthalates. When used as a plasticizer, NBBS is found primarily in cooking utensils, food-contact film, bottles, and textiles.

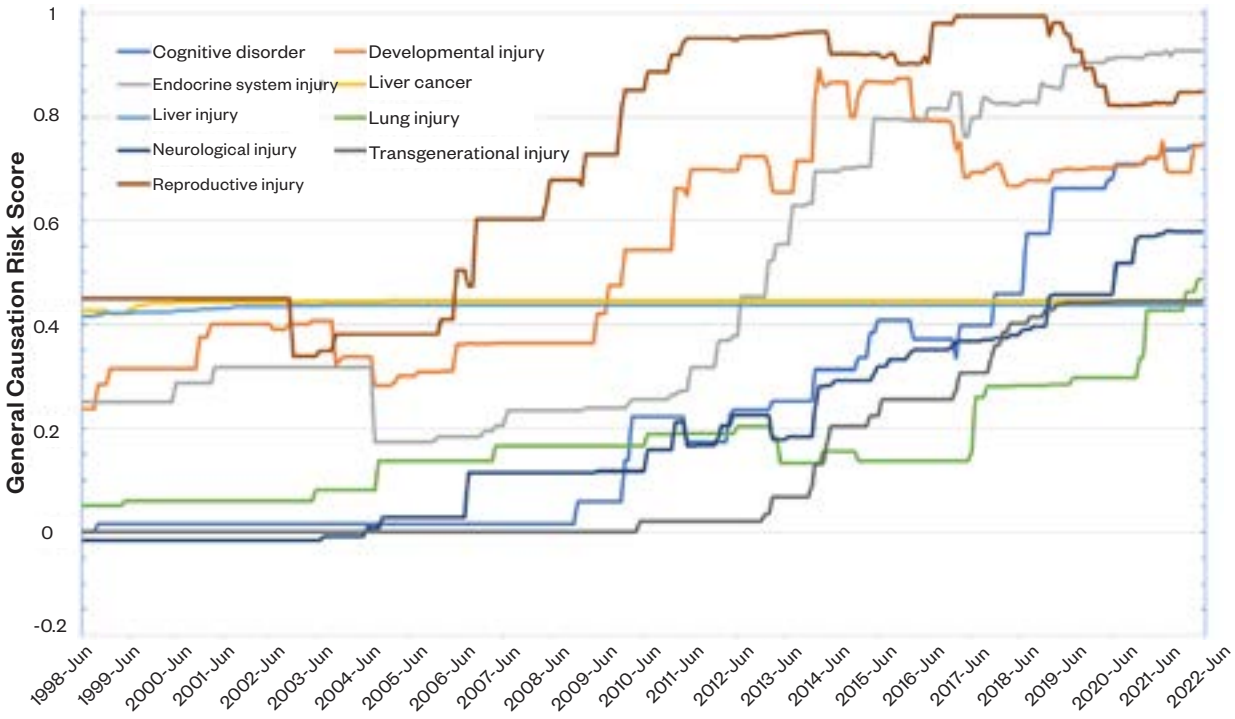
The scientific literature

Most of the research on phthalates has focused on how DEHP disrupts the endocrine system and the consequent effects. This research has led to DEHP being a proven obesogen. Perhaps unsurprisingly, given the importance of endocrine functionality in reproduction, research on DEHP exposure is strongly connected to both female and male infertility. Because many different phthalates are used as plasticizers, scientists have hypothesized that those with a similar chemical structure to DEHP may also be linked to human infertility. In men, for example, both BBP and DEP (in addition to DEHP) were associated with decreased sperm concentration and motility, two strong indicators of infertility.[9] A study in adolescent females correlated early menarche with higher BBP metabolite levels alongside a greater body burden of all phthalate ester metabolites collectively.[10]

Scientists have also established connections between DBP and infertility. Diminished semen quality was associated with high levels of DBP metabolite.[11] In females, recurrent pregnant loss was significantly associated with urinary levels of DBP metabolites.[12]

*Note: in this section and those that follow, please refer to the section on bisphenols for explanations of the figures and to the methods section for detailed descriptions of the underlying modelling process.

Figure 5: Historical General Causation risk scores for di(2-ethylhexyl)phthalate



The GC scores shown in Figure 5 confirm that scientists have reached fairly strong agreement that DEHP exposure can cause disease. The strongest causal association is that of endocrine dysfunction, with *in vitro*, animal, and human studies all demonstrating that DEHP exposure causes both obesity and type II diabetes. Reproductive problems often result from endocrine dysfunction, although recent human studies investigating DEHP and infertility have not shown an effect of DEHP exposure, leading to the 2018-2020 decrease in the GC score for reproductive injury. However, the GC score reversed course recently when a study was published showing that women undergoing *in vitro* fertilization with high DEHP exposure may contribute to the development of polycystic ovary syndrome.[13]

The *in utero* and perinatal exposure to DEHP captured in the "developmental injury" GC score results in a wide range of diseases in human offspring. This includes increased child adiposity,[14] which can lead to teenage and adult obesity, and decreased skeletal muscle mass in children.[15] The scientific consensus that DEHP exposure can cause cognitive injuries is equally strong in 2022 as the developmental injury hypothesis. One study, for example, shows that markers of high exposure to DEHP, DBP, and BBP are all associated with ADHD behavior in children.[16]

In contrast to the fast-moving GC scores discussed above, it appears that scientists have lost interest in studying whether DEHP exposure causes liver injury or cancer.

Simulated losses

The simulated evolution of case strength for phthalates reaches well into the "strong" range in the coming eight years, so it is unsurprising that phthalate litigation is projected to begin in a relatively high fraction of simulated futures.

Table 3: Litigation model results for phthalates

Probability of litigation	High	
Litigation success rate	High	
	Total cost	**Defense**
Expected loss	$17.7B	33%
PML(5)	$57.5B	21%
PML(1)	$129B	28%
TVaR(5)	$90.9B	23%

Phthalate litigation, if it starts, has a high likelihood of succeeding, and could get quite expensive. As we are exposed to phthalates directly from plastic and indirectly from food, wall coverings, paint, cosmetics, and more, phthalate litigation could easily spread to broad swaths of the economy as shown in **Table 4.**

Table 4: Top five responsible industries for simulated plasticizer litigation

Phthalate manufacturers	Food-related uses	Polyvinyl chloride manufacturers	Plastic glove manufacturers	Building products

Phthalates' use in flexible food packaging materials leads to this industry taking the largest loss alongside the PVC manufacturing industry. The food industry's exposure includes a diverse array of more specific business activities, including manufacturers of processed food, bottled beverages, packaged food, and the businesses that serve food and drinks.

As a diverse array of phthalates are used in the products at the centre of these litigation events, we note that the manufacturers of sixteen specific phthalates and replacement chemicals sustain losses in these simulated litigation events.

Several common building products are made from phthalate-containing materials, most prominently vinyl flooring, wallpaper, and paint. As phthalates can migrate out of their original products they can find their way into house dust leading to significant residential exposures. Although the consequences of bodily injury litigation from these products may be manageable, we note that the property damage costs associated with having to remove phthalate-containing building materials from houses could dwarf the indemnification costs of bodily injury litigation.

Lastly, we note that the medical industry uses many products that contain phthalates, ranging from plasticizers for blood bags and intravenous fluid storage/delivery to enteric coatings used to make pills that provide controlled release of the active ingredient.

Brominated flame retardants

Brominated flame retardants release bromine atoms when heated, helping prevent ignition and slowing the spread of fires. They are often incorporated into plastic for these reasons, at concentrations reaching 15%.

Pentabromodiphenyl ether (pentaBDE), a mixture of chemicals containing 5 bromine atoms per molecule on average, was the first brominated flame retardant on the market in the 1950s. PentaBDE has an affinity to lipids, allowing the human body to store it for a long time and also increasing its potential to bioaccumulate. PentaBDE is also classified as a persistent organic pollutant and has been banned since 2004.

Since bromine-containing chemicals make effective flame retardants, replacements for pentaBDE are commonly other brominated chemicals that are still manufactured and used in commerce. Among the first replacements for pentaBDE were other polybrominated diphenyl ethers (PBDEs) – chemicals with the same basic structure as pentaBDE but with different bromine content. The two most common of those are octabromodiphenyl ether (octaBDE) and decabromodiphenyl ether (decaBDE), the latter of which is fully brominated and extremely stable in the environment and in animals. DecaBDE is linked to cognitive and developmental injuries in humans, resulting in its being listed as a substance of very high concern under REACH in the EU. DecaBDE has largely been replaced by a similar molecule: decabromodiphenyl ethane.

The class of brominated flame retardants contains several other commonly used chemicals: 2-ethylhexyl-2,3,4,5-tetrabromobenzoate (TBB), bis(2-ethylhexyl) tetrabromophthalate (TBPH – a brominated analogue of the phthalate DEHP), hexabromocyclododecane (HBCDD, also a persistent organic pollutant), tetrabromobisphenol A (TBBPA – a brominated bisphenol), and tris(2,3-dibromopropyl) phosphate (TDBPP).

Exposures

Before pentaBDE was phased out, it was added to flexible polyurethane foam in airplane seating, automotive seating, upholstered furniture, mattresses, and carpet padding. The replacement brominated flame retardants are used in the same products. HBCDD is the primary flame retardant in expanded and extruded polystyrene which are formed to make insulation boards for various construction products. Along with decaBDE and decabromodiphenyl ethane, HBCDD is also integrated into high-impact polystyrene used for consumer electronics and automotive interiors.

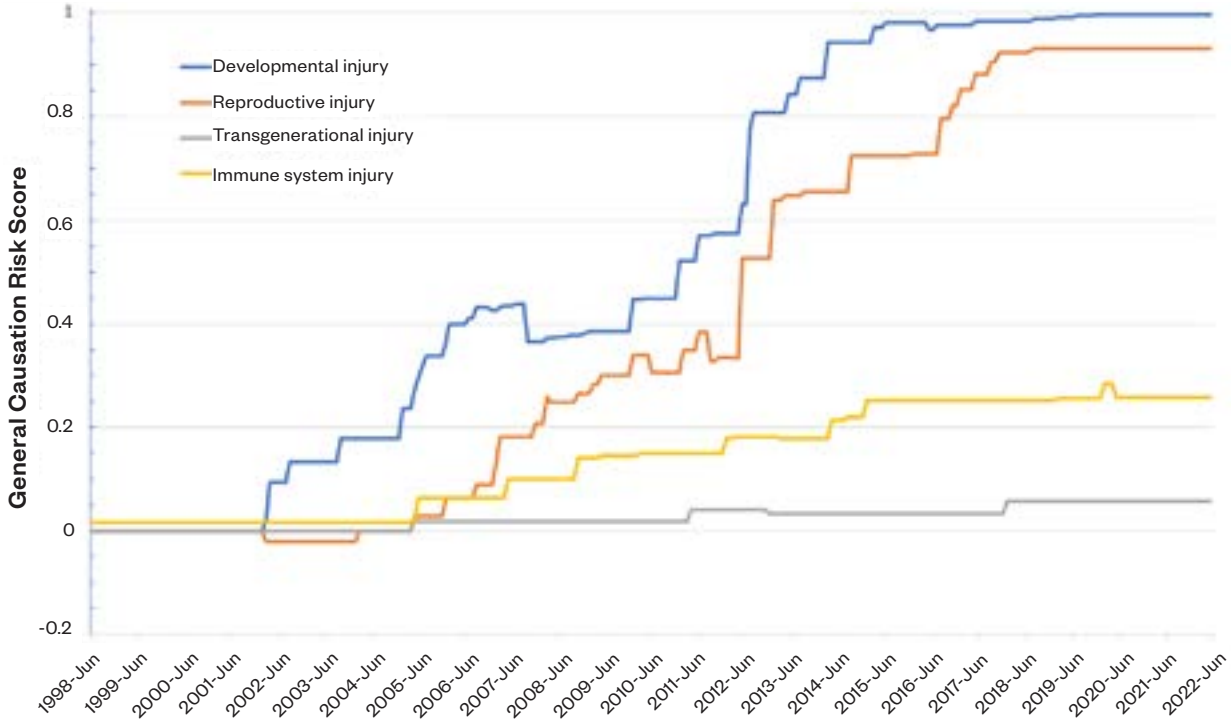
The wide array of products that contain brominated flame retardants has led scientists to look for them in indoor dust, where they've found it provides a near constant source of human exposure. The lipophilicity of brominated flame retardants means that they may be retained in lipids and stored for years. They have also been found in blood, hair, and breast milk.

Brominated flame retardants' persistence and bioaccumulation potential are a significant environmental concern. They have been found in aquatic environments around the world, even in the Arctic.

The scientific literature

Like with many other chemicals that affect the endocrine system, two harm categories garner the most scientific interest for brominated flame retardants: reproductive and developmental injuries. Most of the literature establishing these harms from brominated flame retardants have been done *in vitro* and in animals. Some human epidemiological studies have been conducted, though, demonstrating that exposure to brominated flame retardants can result in bodily injury. High brominated flame retardants levels in house dust have been linked to altered reproductive hormone levels in men[17]. Prenatal exposure to TBBPA was correlated with decreased newborn weight[18]. There is also interest in endocrine injury for some brominated flame retardants – HBCDD is linked to increased risk of type 2 diabetes in women[19].

Figure 6: General Causation risk score for pentabromodiphenyl ether (pentaBDE)



PentaBDE has high GC scores **(Figure 6)**, demonstrating near universal scientific consensus that it can cause reproductive and developmental injuries. These hypotheses reached consensus five and ten years ago, respectively, due to a strong combination of *in vitro*, animal, and human studies investigating these two harms. Scientists have kept studying older brominated flame retardants because of their potential to bioaccumulate and newer ones because we are still exposed to them. A paper from 2016 found adverse reproductive effects in women, including lower levels of follicle stimulating hormone – a hormone critical to ovulation – and higher rates of threatened abortion* in women with higher blood levels of pentaBDE[20].

In utero exposure to pentaBDE specifically and PBDEs more generally have been strongly linked to impaired neurodevelopment in babies[21]. A newly developing area of interest for PBDE-related bodily injury is the immune system. An interesting recent paper linked a component of octaBDE (along with other persistent organic pollutants) to the development of celiac disease in genetically susceptible people[22].

Simulated losses

The moderate amount of scientific interest being paid to the class of brominated flame retardants suggests that the risk of litigation could increase over the coming eight years. These scores are low enough (and are considered fairly "weak") that they do not usually attract litigation, but as we see below there are enough cases that are strong enough that our model projects a moderate probability of litigation beginning, with a moderate success rate.

*Threatened abortion (or miscarriage) is defined as vaginal bleeding without cervical dilation occurring in the first 20 weeks of pregnancy. Its presence is a strong indicator that spontaneous abortion or miscarriage may occur.
See: https://www.merckmanuals.com/professional/gynecology-and-obstetrics/abnormalities-ofpregnancy/spontaneous-abortion

Table 5: Litigation model results for brominated flame retardants

Probability of litigation	Moderate	
Litigation success rate	Moderate	
	Total cost	**Defense**
Expected loss	$363M	46%
PML(5)	$1.7B	70%
PML(1)	$10B	27%
TVaR(5)	$6.2B	41%

Despite the simulation finding a moderate probability of litigation beginning, litigation targeting brominated flame retardants is not projected to be particularly expensive across the entire economy. The marginal case strength projections lead to the observation that cost of defending the litigation is as large or larger than the indemnity cost, even when plaintiffs win the case.

Table 6: Top five responsible industries for simulated brominated flame retardant litigation

Brominated flame retardant manufacturers	Aircraft manufacturer and operator	Polystyrene manufacturer	Electronics manufacturer	Flame-retardant polymer manufacturer

As with most toxic tort litigation, the plurality of the responsibility falls to the originators of the chemicals at issue. The use of brominated flame retardants in both polystyrene and polyurethane materials leads to the aerospace industry suffering a significant portion of the total cost of these litigations. The broad usage of these flame retardants across the economy leads to a broad distribution of the remaining responsibility in these simulated litigations.

Phosphate flame retardants

Phosphate flame retardants are an alternative to brominated flame retardants. They work by helping release water, creating a layer of charred material on plastic surfaces, and by interrupting the chemical process of burning. Since they are generally less effective as flame retardants than their brominated cousins, higher amounts are used in plastic. Compared to brominated flame retardants, phosphate-based flame retardants seem to be less toxic to humans. Many phosphate-based flame retardants are used today, some brominated or chlorinated, and others that are not halogenated. These include tris(2-chloroisopropyl) phosphate, triphenyl phosphate (TPHP), tris(1,3-dichloro-2-propyl) phosphate (TDCIPP), tris(2,3-dibromopropyl) phosphate, tris(2-butoxyethyl) phosphate, tris(2-chloroethyl) phosphate, and tris(methylphenyl) phosphate.

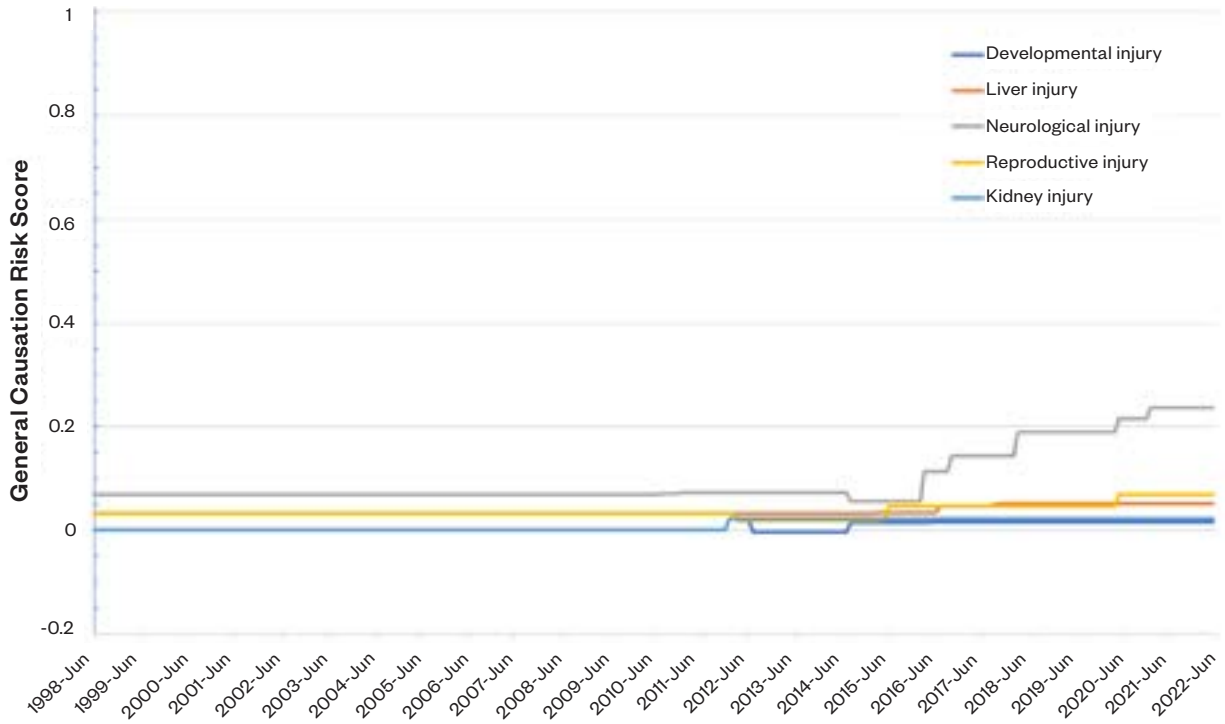
Exposures

Phosphate flame retardants are used in food-contact material, consumer electronics, and automobiles. Since they are incorporated into polyurethane foam, phosphate flame retardants can also be found in insulation, mattresses, upholstered furniture, and children's napping mats. Their presence in this wide array of products has led them to be found in indoor dust and inside automobiles. Drinking water treatment plants have found phosphate flame retardants in their water as part of the testing during their purification processes. Biomonitoring studies on phosphate flame retardants have found that many humans have these chemicals in their urine and blood. Phosphate flame retardants have, unsurprisingly, been found all around the world in various bodies of water and in aquatic life.

The scientific literature

Scientific interest in phosphate flame retardants is lower than that of several of the other chemical groups we discuss in this report. Interest in the last few years has remained stable and moderate. The majority of the literature investigating bodily injury hypotheses to date consists of animal and *in vitro* studies. Prenatal exposure to phosphate flame retardants, specifically TDCIPP and TPHP, are linked to adverse behavioral development in children.[23] Organophosphate esters, the broad chemical category these flame retardants belong to, may also be a linked to breast and cervical cancers via hormone disruption.[24]

Figure 7: General Causation risk score for tris(2-chloroethyl) phosphate



For tris(2-chloroethyl) phosphate (TCEP), 5 harms have been investigated for injury with mostly *in vitro* and animal literature. The highest GC score for TCEP **(Figure 7)** is nervous system injury consisting entirely of *in vitro*, rodent, and fish studies. In mice, TCEP has been associated with neurotoxicity.[25]

Simulated losses

Although there is sustained scientific interest in the potential health effects of phosphate-based flame retardants, the average projection of case strength does not move significantly from today's relatively "weak" values. A small number of case strength simulations, however, rise to the level where litigation will occasionally activate **(Table 7).**

Table 7: Litigation model results for phosphate flame retardants

Probability of litigation	Low	
Litigation success rate	Moderate	
	Total cost	**Defense**
Expected loss	$300M	20%
PML(5)	n/a	n/a
PML(1)	$4.2B	39%
TVaR(5)	$6.0B	20%

Although we simulate that litigation begins less often than for brominated flame retardants, we see that the expected loss is similar in both cases. This is driven partly by the fact that defense costs mount quickly in any mass litigation, even if defendants ultimately prevail.

The TVaR(5) measure for both phosphate-based and brominated flame retardants is similar, at approximately $6 billion. This reflects the fact that both types of flame retardant are hypothesized to cause similar harms to similarly large numbers of people.

Table 8: Top five responsible industries for simulated phosphate flame retardant litigation

Phosphate flame retardant manufacturers	Flame retardant product manufacturers	Baby carrier manufacturer	Flexible polyurethane foam manufacturer	Paints and coatings manufacturer

The industrial footprint of phosphate-based flame retardant responsibility in our simulated litigation **(Table 8)** differs from that of brominated flame retardants. This reflects the differences in uses, where phosphate flame retardants are used more commonly in polyurethane foams than some of the harder plastics like high-impact polystyrene, leading to increased exposure via furniture and padded surfaces, including those often used by babies in mattress pads, car seats, and carriers.

Per- and polyfluoroalkyl substances (PFAS)

Per- and polyfluoroalkyl substances (PFAS) are chemicals used for their ability to impart water, heat, oil, and chemical resistance to other materials. They are persistent in animals and the environment, are found in the blood of residents of industrialized countries, and some members of this chemical class are known to be toxic to humans. While most of the industrially used PFAS are not used directly in plastic, some uses of PFAS intersect with plastics at some point in their life cycles. For example, PFAS are commonly used to make carpets stain resistant and can sometimes be found on microfibres released from those carpets. Recent investigation has also shown that fluorination of simple plastics like polyethylene can lead to PFAS being present in the finished product.

Litigation has been ongoing for some specific PFAS chemicals and specific uses of PFAS, neither of which directly implicate the plastic industry. The first of these is the litigation involving both bodily injury and property damage from discharges of PFOA from a DuPont/Chemours plant into the Ohio River. The second litigation stems from water contamination and high levels of human exposure to a variety of PFAS chemicals used in Class B firefighting foam, also known as aqueous film forming foam (AFFF).

Since most potential PFAS liability arises from non-plastic uses we do not discuss this class of chemicals further in this report.

Microplastic

As discussed elsewhere in this report, plastics are polymers, chains of repeated small building blocks, constructed to give plastic their myriad properties. One way to classify the different types of plastic is by their chemical composition, giving rise to the most commonly used plastic types: polystyrene, PVC, polyethylene, etc. As relevant to this section of the report, another important distinction has emerged in classifying plastics – their size. Scientists now recognise differences between macroplastic, microplastic, and nanoplastic.

While there is no universally agreed-upon definition, scientists and regulators have all but converged on 5 millimetres (in the longest dimension of the plastic fragment) being the dividing line between macro and microplastic. The smallest microplastics are classified as nanoplastics, although this categorisation also suffers from lack of uniform definitions. According to the European Chemicals Agency (ECHA), nanoplastics are considered a subset of microplastics so they do not have a specified size range. The International Organization for Standardization considers particles to be nanoplastic when they are smaller than 1 micrometre. Many scientists have drawn the line delineating micro- and nanoplastic at 100 nanometres, a scale that changes how those particles interact with biological tissue.

Microplastic's origin provide another important distinction. Primary microplastics are produced intentionally as pellets, powders, and beads and are intended for use as microplastics or as precursors, called nurdles, for producing plastic or plastic-containing products. Primary microplastics are widely used in a variety of applications, including skin care products, air blasting, and drug delivery. In 2015, the US banned the use of primary microplastics in rinse-off cosmetics containing plastic microbeads. Secondary microplastics are not intentionally produced but are the result of industrial and environmental breakdown and fragmentation of macroplastic. Microfibres are a type of secondary microplastic that are fibrous in shape and less than or equal to 5 millimeters in length.

Exposures

Polyethylene is used to make kitchen cutting boards and plastic bags. Retail food containers are commonly made from polypropylene, while food take out containers can be expanded polystyrene, originally branded as Styrofoam. Polyester and polyamide (nylon is an example) are common clothing textiles. Polyethylene terephthalate is a polyester frequently used to make single-use drink bottles. Polyvinyl chloride is a type of halogenated plastic with various uses including construction, cable and wires, flooring, and pipes.

All these types and uses of plastic can contribute to both human exposure and the global burden of microplastic.

Microplastic has been found everywhere researchers have looked, from the north to south poles, from the heights of Mount Everest to the depths of the ocean floor. It's unsurprising, then, that human exposure to microplastic arises from multiple routes, amounting to as much as several grams of microplastic per week.

The exposures include our drinking water, whether bottled or from municipal water supplies. Microplastic can be consumed in food packaged in plastic containers or wrapped in plastic material. Microplastic has, surprisingly, been found in fruits and vegetables. A study on produce bought from an Italian farmers market found microplastic in all the produce.[26] Higher amounts were found in tree fruits like apples while less was found in fast-growing crops like lettuce. Scientists think that plants take up microplastic from their roots and transport them through their tissues, meaning that microplastic content in plants is likely correlated with how long the plant grows in the ground.

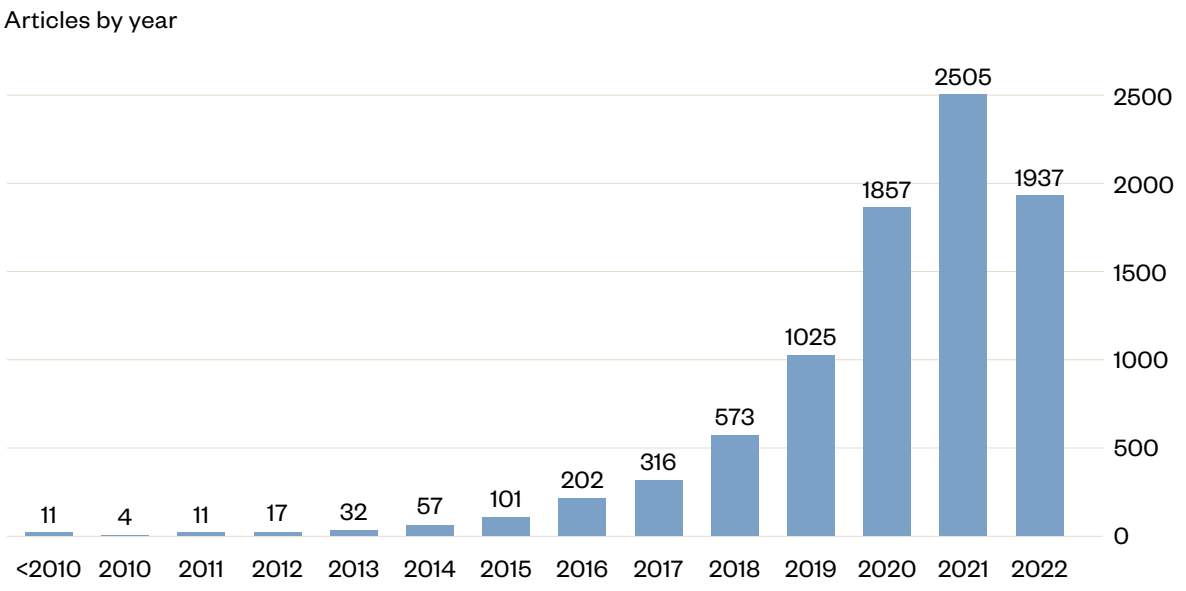
High levels of microplastic have been detected in wastewater treatment plants, often attributed to microfibres from laundering clothes. Wastewater treatment plants are effective at removing larger pieces microplastic from wastewater, but then the microplastic becomes a component of wastewater sludge that's commonly used to fertilise crops. This leads to significant introduction of microplastic into the soil, which also contributes to the presence of microplastic in produce as described above.

Humans inhale microplastic in both indoor and outdoor air. Microplastic is a component of the indoor dust we frequently breathe, with clothing being a major contributor to that burden. In outdoor air, the majority of inhaled microplastic is from tire wear. Microplastics that get released into the environment eventually end up in the ocean. Once there, they can make their way back into the atmosphere as part of sea spray, leading to microplastic presence in outdoor air and its transport on air currents.

The scientific literature and case strength

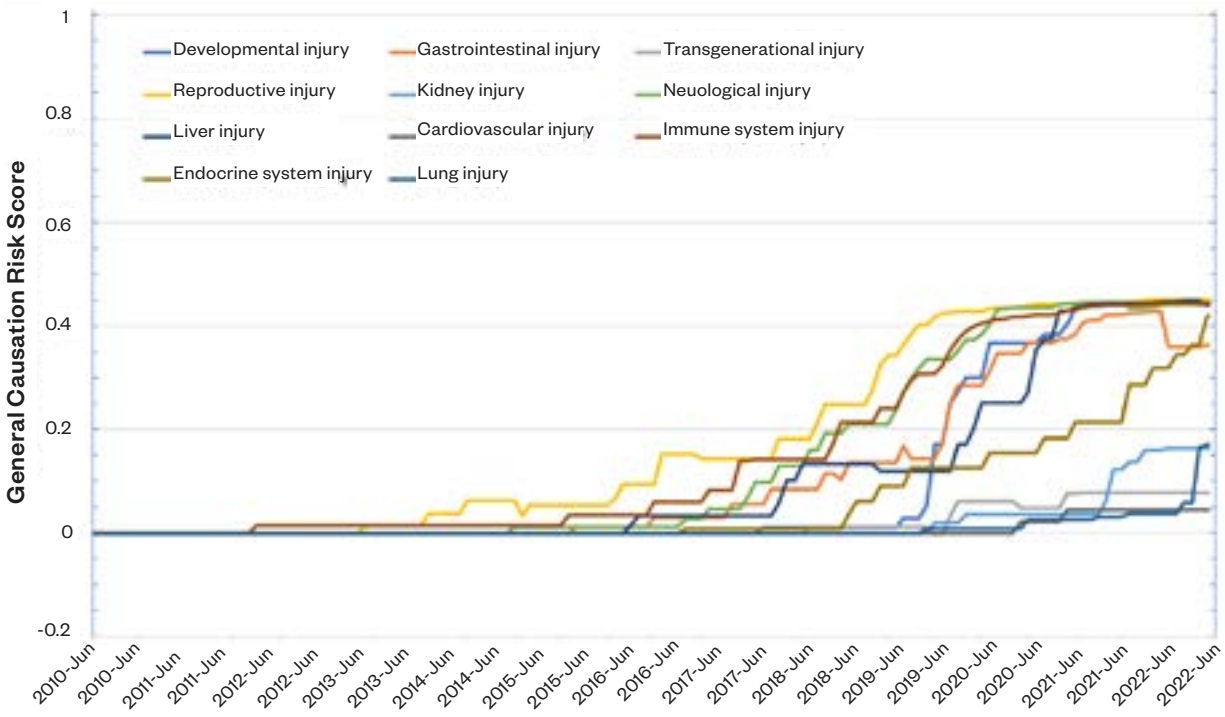
There is intense scientific interest in microplastic as evidenced by the volume of abstracts published since 2020 (**Figure 8**). Halfway through 2022 there are as many abstracts published about microplastics in PubMed than were published in the entirety of 2020. The majority of microplastic literature revolves around the environmental presence and impact of microplastic. Scientists have intensively and repeatedly studied the quantities and types of microplastic in water and land.

Figure 8: Microplastic and nanoplastic publishing by year, from PubMed as of 1 July 2022



The microplastic literature for bodily injury consists primarily of studies done *in vitro* and in animals, most of which are aquatic creatures. The *in vitro* studies are helpful for developing an understanding of the mechanisms by which microplastic exposure can cause injury. In some cases, the organisms used for these studies are important parts of the food web, making their study useful in better understanding the ecological (and eventually human) effects of microplastic presence in the environment. Also related to our food supply, ever since scientists determined that many fertilisers contain microplastic, they have investigated microplastic's effect on soil-dwelling organisms like earthworms.

Figure 9: General Causation risk scores for microplastic



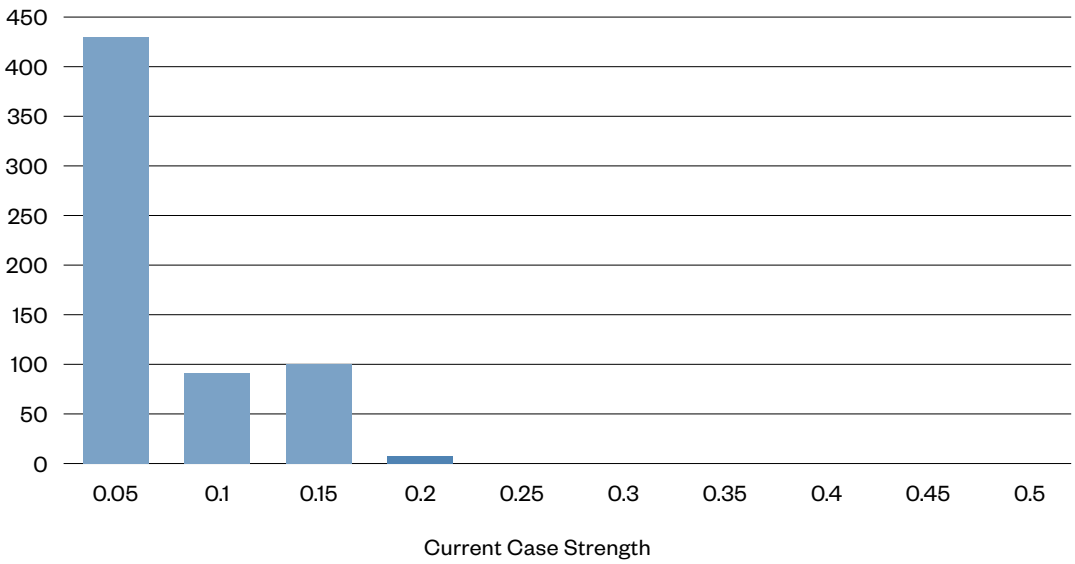
Scientists are investigating whether microplastic can cause a broad array of human harms, as seen in the figure above. Despite the breadth of study, the recency of the discovery that humans are constantly exposed to microplastic means that human health research is still in its early stages. That results in the literature investigating whether microplastic causes bodily injury being entirely composed of *in vitro* and animal studies.

One quickly notes that five harms share the highest GC risk score that can be reached absent human epidemiology studies: reproductive, neurological, immune system, liver, and developmental injuries. The recent uptick in score for endocrine system injury is driven by the recognition that plastic particles can deliver endocrine disrupting compounds to our bodies. This has led to a recent spate of rodent studies that, as one example, show microplastic can cause insulin resistance in mice.[27]

The first human epidemiology study of microplastics in humans was published at the end of 2021.[28] It measured the amount of microplastic in faeces and determined that people with inflammatory bowel disease had noticeably more microplastic particles in it. They further concluded that the concentration of microplastic in faeces was correlated with the severity of disease. As this was an observational cross-sectional study design, the authors were unable to determine whether the faecal microplastic caused the disease or if the disease caused the patients to have more microplastic in their faeces. Further research will no doubt shed light on this putative link.

The second human study,[29] a small epidemiology study published in July 2022, found that six patients with liver cirrhosis had microplastics in their liver tissue while five control patients without liver disease had microplastic-free liver tissue. As with the first human study, it is unclear whether liver disease increases microplastic uptake or the reverse.

Figure 10: Current case strength distribution for microplastic

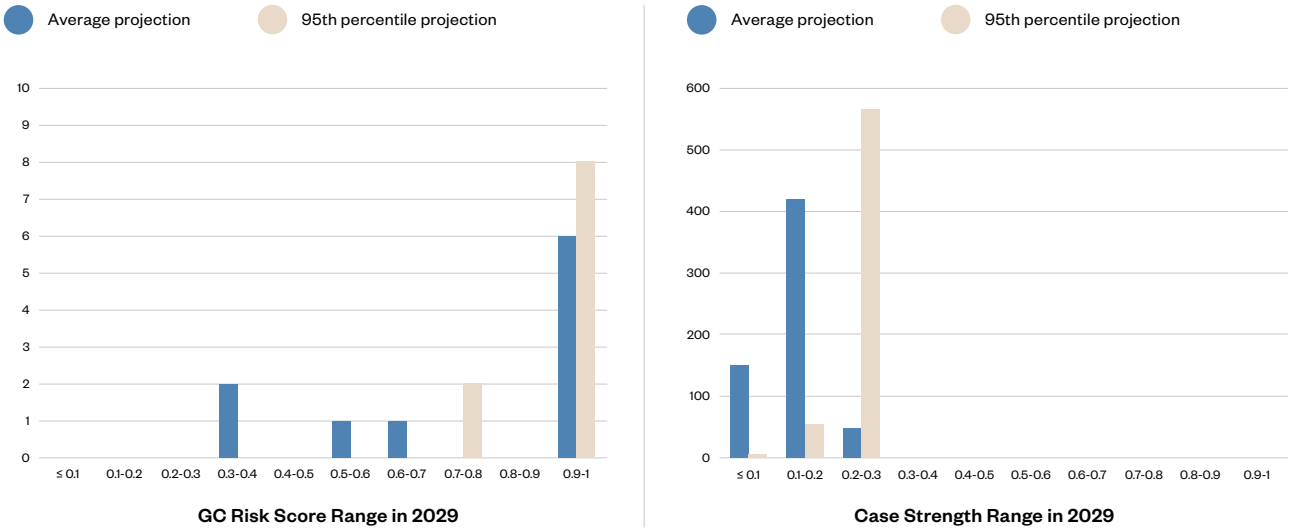


Somewhat surprisingly, the current case strength values for some hypothetical lawsuits is as high as 0.15-0.2 – still generally weak cases but stronger than one might think given the early stage of MNP research. While this is not generally considered a strong enough case to trigger a lawsuit it indicates that scientists are quite concerned about microplastic exposure's potential to cause bodily injury. It also suggests that identifying microplastic as the cause of at least some types of bodily injury is possible with current methods.

Simulated bodily injury losses

Turning now to the projected evolution of the scientific literature and case strength, we see that the rapid publishing rate increases of today tell us to expect significant evolution of the literature in the coming eight years.

Figure 11: Histograms of projected General Causation risk (left) and Case Strength (right) for microplastic. Blue bars represent the average projection while beige bars show the 95th percentile projection



It is particularly impressive that the average projection of general causation risk has six human harm hypotheses moving all the way to consensus. We attribute this to the strong and rapid literatures emerging on microplastic and bodily injury. Given the relatively recent understanding that we are continually exposed to microplastic the model predicts that scientists continue to study their effects in humans, with increasing publications over the coming years.

The projected large increases in general causation risk scores lead to significant possibility that case strength evolves enough to, in rare cases, support litigation.

Table 9: Litigation model results for microplastic

Probability of litigation	Low	
Litigation success rate	**Low**	
	Total cost	**Defense**
Expected loss	$103M	53%
PML(5)	n/a	n/a
PML(1)	$2.8B	92%
TvaR(5)	$2.1B	53%
TvaR(0.5)	$13.6B	33%

Despite the large projected increases in both mean and 95th percentile GC and case strength scores, the cases rarely get to the point where we simulate litigation beginning, and where it does only a low percentage of simulated outcomes contain successful microplastic litigation. That means that microplastic litigation, if it begins, under today's projections we would expect it to be a defense-only event more likely than not.

We included in the above table the TvaR(0.5) statistic. Because this corresponds to the probability of litigation both beginning and succeeding, this result also indicates the expected value of successful microplastic litigation given our knowledge today. At $13.6 billion, the litigation is certainly a significant event but it does not approach – again, with today's knowledge – the severity of asbestos or many of the other possible plastic-related litigations discussed above. As our understanding of the kinds of harms MNP can cause evolves, these numbers could increase significantly.

Table 10: Top five responsible industries for simulated microplastic litigation

Plastic resin manufacturers	Commercial fishery	Custom compounded resins	Plastic bottle manufacturer	Apparel & textile manufacturer

Turning our attention to the distribution of responsibility in these simulated bodily injury litigation events, we note that the plastic industry absorbs much of the responsibility. As we've seen in the other industry-level results above it's common that the original producers of the litigated hazard take much of the responsibility.

We also see that significant responsibility goes to the fishing industry. This is due to the well-established fact that much of our exposure can be traced back to seafood. While defendants in this industry may have better defenses than others (similar to produce growers) any mass litigation results in substantial defense costs from the outset and seafood companies and their insurers need to be aware of this risk should they not test their products for the presence of microplastic.

Remediating nanoplastic from water

Scientists have extensively documented the presence of microplastic in ocean water,[30,31] wastewater,[32] fresh water,[33,34] and drinking water.[35] According to one analysis,[36] 83% of tap water samples from major metropolitan areas around the world were contaminated with plastic fibers and over 90 percent of the world's most popular bottled water brands contain microplastic. A large study[37] examined 107 water samples from 29 Great Lakes tributaries in five U.S. states and found that all of them contained microplastics. The highest concentration was found in the Huron River at Ann Arbor, Michigan, at 32 particles per cubic metre. Other studies have found that the concentration of microplastic in fresh and drinking water spanned ten orders of magnitude (1×10^{-2} to 10^{8} particles per cubic metre) across individual samples and water types.[38] One U.S. and international study[39] found an average concentration of 5,450 microplastic particles per cubic metre and found a maximum of 61,000 particles per cubic metre. Wastewater treatment plant effluent has also been shown to contain microplastic, although at lower concentrations than drinking water.[40]

Collectively, these studies tell us that MNP are ubiquitous in wastewater, drinking water, and the freshwater sources used for our drinking water. When problems of this scale are identified it's prudent for an insurer to assess the risk of litigation and the potential outcomes of litigation seeking compensation for increased water treatment costs, as has happened in PFAS litigation.

Any such litigation, however, must determine which companies and industries are responsible for the presence of MNP in their water sources. Scientists have identified several major sources: tire wear debris, fibres released from textiles due to wear-and-tear and clothes washing, and paint, mainly from road markings. Microplastic contribution to drinking water extends to other sources, too. Wastewater effluents, agricultural run-off, "city-dust" (a complex mixture of abraded particles attributed to things like synthetic shoe soles and artificial turf), and the fragmentation and degradation of macroplastic entering freshwater systems have also been classified as potential sources of microplastic.

Wastewater treatment plants usually capture larger microplastic particles. Unfortunately, most of the captured microplastic is trapped in the produced sludge that is often sold as fertiliser, thereby putting those captured microplastic back into the environment. Drinking water treatment also captures larger microplastic particles but the smaller nanoplastic particles often escape the treatment and make their way to our taps.

In the deterministic scenario we describe below, we posit that, as the science continues to evolve towards an agreement that MNPs cause bodily injury, the U.S. government will restrict the presence of MNP in treated wastewater and drinking water. Given that current drinking water and wastewater treatment techniques do not assure a complete removal of MNPs, wastewater treatment plants and drinking water utility operators will need to upgrade their treatment protocols. Like with PFAS water remediation lawsuits, the entities burdened by these extra costs will sue the industries that released MNPs into water to cover the costs of upgrading their plants and operating them for several years.

Economy-wide remediation cost

The first step in building our scenario estimate is computing, under several plausible outcomes, how much the plaintiffs will have to pay to remedy the underlying problem. We term this the "economy-wide burden". We compute the economy-wide burden in two steps. First, we determine the cost of removing nanoplastic from water per unit of volume that requires treatment by assessing the operating and capital cost of building new treatment facilities. Then we assess the volume of water that needs treatment and over what time scale. Given estimates of these two numbers we proceed to calculate the economy-wide burden by multiplying the volume by the remediation cost. We use the lower and higher ends of our estimates to determine a range of costs for these scenarios.

Based on a review of the scientific literature,[41, 42, 43, 44] advanced membrane technologies like ultrafiltration or nanofiltration appear to be the most cost-effective way to remove nanoplastic from water. These are more efficient to operate than technologies with smaller pore sizes like reverse osmosis but have small enough pores

to capture the vast majority of nanoplastic particles. Our literature survey allowed us to select a volumetric unit cost for installing and operating these membrane technologies.

Then we proceed to estimate the volumes of drinking water and wastewater contaminated with nanoplastic that would require remediation. While comprehensive data on nanoplastic in drinking and wastewater are not available, we were able to estimate the percentage of water treatment plants already using advanced membrane technology.[45] Based on these data, and knowing that some existing membrane treatment techniques will still need to be upgraded, we estimated the upper and lower bounds of the amount of drinking and wastewater that could need treatment, while also accounting for uncertainty in the stringency of future regulation.

We combine this with annual drinking water and wastewater volumes processed in the US based on USGS data[46] to obtain a range of estimated total annual water volumes needing nanoplastic remediation. Based on remediation plans from the PFAS litigation and observations from the scientific literature, we were also able to estimate the necessary duration of water treatment for nanoplastic removal.

The product of annual water volumes requiring nanoplastic remediation, the unit cost of treatment, and the length of treatment yields estimates of the economy-wide burden.

Table 11: Economy-wide cost to remediate nanoplastic from drinking water and wastewater

	Min	Max
Wastewater	$176B	$470B
Drinking water	$162B	$431B
Total	$338B	$901B

Our estimates (Table 11) range from $176 billion to $470 billion for wastewater remediation, and from $162 billion to $431 billion for drinking water remediation. These cost estimates only include the costs to remediate the water and do not, at this stage of the analysis, take into account the cost of defending litigation in court. The amount actually recovered in mass litigation hinges on the ability of the responsible parties to be able to pay their full share of the cost they are responsible for. In scenarios like this with large economic costs, much of that cost is not recovered in litigation, instead being passed on to the customers of the affected businesses.

Industry responsibility

As we've done above, we assess the ways in which potential litigation could evolve and what that means for the distribution of costs to different sectors of the economy. We model two different theories that plaintiffs' lawyers might press as to which parties are responsible. Undoubtedly other theories could be presented, and this discussion is not meant to be an exhaustive legal analysis. Rather, it's meant to describe how we applied both past precedent and lessons from the ongoing PFAS litigation to the hypothetical case where water companies seek remuneration for having to remediate their effluent.

Option 1: Lawyers target those most directly responsible for nanoplastic pollution

A review of the scientific literature suggests that some industries are more responsible for directly releasing MNPs to drinking water sources and wastewater than others. In this example of how plaintiffs could approach the case, they use the scientific literature as a guide for which companies and industries to target in this litigation. For the sake of brevity, we will refer to this as the "direct pollution" theory.

As described above, run-off from the breakdown of road-marking paints, debris from tire wear, and microplastic fibres released from textiles are dominant sources of microplastics in water, while other sources like wastewater effluent, agricultural run-off, city-dust, and fragments of degraded macroplastic also contribute to the contamination.[47, 48, 49] It's reasonable, then, in this scenario, for the eventual resolution of the litigation to mirror this observation.

One feature of the logic underlying this theory is that only the direct polluters pay. The upstream contributors to the global plastic problem are not targeted because plaintiffs' lawyers believe them to be too far removed from the water contamination at issue to bring them to court. This leads us to apportion responsibility for

nanoplastic water cleanup to products responsible for directly releasing MNPs to water.

Each product category is composed of multiple business activities involved in bringing the products themselves to market. After we determine the share of the problem attributable to each product type, we distribute the responsibility to the linked business activities identified as contributing to the nanoplastic problem. Because, in the U.S., the Microbead-Free Waters Act of 2015 prohibited the use of microbeads in rinse-off cosmetics and non-prescription drugs,[50] we assume plaintiffs do not target these sources for their historical contribution to drinking water and wastewater contamination.

Option 2: Lawyers successfully target the broader plastic industry

Plaintiffs' lawyers may attempt to bring in a broader array of economic actors who would then be able to share the responsibility for the remediation more broadly, likely enabling larger recoveries of plaintiffs' costs if successful. In a sense, in this theory of the litigation the plaintiffs target the entire plastic industry for their role in the global plastic problem by using this water remediation litigation as a proxy. We will refer to this as the "plastic proxy" theory.

This prosecutorial theory leads to responsibility being spread to the plastic-based economy, regardless of the direct and/or imminent release of nanoplastic to water. This litigation theory also allows the plaintiffs' lawyers to focus a hypothetical jury's ire on several very large companies that produce and use large amounts of plastic – a strategy the lawyers may feel can yield favourable verdicts and settlements.

It is well known that different beverage corporations have been at the center of the news for multiple years and some groups have cited them as the main "plastic polluters". [51,52] At the same time, plastic-producing companies are attracting attention. The Minderoo Foundation published a list of 100 plastic producers that are responsible for 90% of global single-use plastic waste,[53] with petrochemical and primary plastic producers topping the list.

Because of this approach to the case, costs are spread to a broad set of business activities based on how much plastic they make and/or use and proxies for how much they contribute to the global plastic pollution problem. This wider set includes, in addition to all the direct polluters, the industries that produce plastic and the personal care product and related sectors.

In Table 12 we show the top five implicated business activities based on total defense plus indemnity costs to each. Defense costs are calculated as a percentage of indemnity costs, except for the business activities that successfully defend their cases in the failed plastic proxy litigation.

Table 12: Top five business activity losses for two modelled outcomes of MNP water remediation scenarios

Direct pollution	Plastic proxy
Clothing manufacturing and laundering	Plastic manufacturing
Paint and coating manufacturing	Clothing manufacturing and laundering
Tire manufacturing	Plastic packaging manufacturing
Agricultural use	Plastic bottle manufacturing
Bottled drink manufacturing	Synthetic fibre manufacturing

Securities litigation

The results in the remainder of this annex are those damages we estimate defendants in mass litigation pay to resolve litigation claiming bodily injury and/or property damage. As industry observers have pointed out[54,55,56] event-driven securities litigation has emerged as a significant risk both for companies involved in mass litigation and their insurers.

While in depth discussion of securities litigation is provided in Annex 2, we have previously estimated the potential effects of securities litigation targeting the defendants in bodily injury and property damage litigation. Modelling this litigation is out of scope for this report, but insights gleaned from other models provide useful context for companies and insurers who are exposed to this risk.

Our model for securities litigation estimates the potential stock drop for public companies due to mass litigation. Using examples and studies of prior securities litigation settlements we established that settlements tend to be small relative to both the size of the stock drop and the amount paid in the underlying mass tort litigation. We can expect that trend to continue, particularly with the larger simulated and modelled events we discuss above like removing nanoplastic from water systems and large litigations claiming bodily injury from plasticizers.

While the raw dollar amounts may not be eye-popping at the economy level, these nonetheless could be highly material events to those insurers writing D&O policies for the affected companies.

Implications for the insurance industry

Insurers have much to consider with respect to managing the risks of plastic and related products. The array of chemicals is dizzying and nearly everybody in the United States and around the world has been exposed to plastic and its related chemicals. The hypothetical lawsuits discussed above show the myriad ways insurers can be exposed to the liabilities stemming from plastic production and use. Nonetheless, many questions remain that insurers must consider in developing strategies to manage these risks.

A useful reference point is the asbestos litigation. From the earliest cases in the 1970s through today, over 8,000 defendants have been implicated and over a million plaintiffs compensated. Estimates of ultimate plaintiff recoveries range from $200 billion to $275 billion, with estimates of insured loss around $100 billion, implying that somewhere between three-eighths and one-half of “ground-up” losses are paid as claims.

For plastics, there are several factors that would point toward a smaller level of coverage. Most significantly, a large fraction of exposure has occurred since the mid-1980s, when widespread adoption of pollution exclusions began. As a significant amount of exposure to plastic may be deemed to come from its presence as a pollutant, coverage under General Liability policies may not be provided under the later policy language. In cases where General Liability coverage is deemed inapplicable, though, other coverages may apply, such as Environmental Liability.

Offsetting this, the tendency toward later exposure would result in later policy years – where limits purchased were considerably higher – responding to claims. Courts may also rule that the insurer has a duty to defend these claims, as they have the potential to be covered by the policy. So while the insured fraction of plastics claims may well end up considerably lower than for asbestos, there is still potential for significant insurer losses for plastics.

Bodily injury lawsuits

For insurance written using the standard occurrence form, the basic insuring agreement requires that the bodily injury or property damage must arise from an occurrence during the policy period. For most situations, where injury is the result of an accident or other event, the event occurs at a point in time (or over a narrow interval of time), and there is no question of what the occurrence was and whether it occurred during the policy period except in the case of an event occurring at or near the moment of policy inception or expiration. In contrast, when there is a significant interval of time from initial hazard exposure until the manifestation and discovery of harm (i.e., long latency), there is significant uncertainty and room for opposing assertions regarding what the occurrence is and exactly when it occurred. Courts have found in these cases that the occurrence may take place over many years, with the consequence that policies issued across many underwriting years may incur losses.

Reflecting this uncertainty, a wide range of precedents for interpretation of policy language has evolved around key questions regarding which policies have the potential to respond to claims and how losses are allocated among the responding policies. Asbestos litigation is again instructive here, as many of the precedents for finding of facts, or their being supplanted by evidentiary presumptions, as well as multi-year coverage allocation methods, originated in the context of asbestos litigation.

Depending on the jurisdiction, any policy written from the commencement of human exposure through to the manifestation of a plaintiff's disease may potentially incur losses. With claims spread across all U.S. states and people of variable age, it is likely that the litigation discussed above could in some circumstances trigger the maximum number of policy periods, from the distant past when exposure first occurred through the present and into the future, where additional disease manifestation and ongoing "injury-in-fact" is continuing. The specific policies triggered will depend on how individual states interpret the occurrence form, as each state has adopted its own precedents on key coverage questions, as well as the specific language of the policies.

The net effect of the policy period precedents discussed above is that so-called "limit stacking" across multiple policy years is a significant risk for plastic-related liability. The chemicals and products discussed above have been in use for many decades, and the harms that are likely to be the subject of litigation can also take years to develop after exposure commences. This is the same situation as asbestos, and could lead to similar aggregation risk due to interpretations of the occurrence policy that can lead to multi-year limit stacking.

Another consideration in the effect of plastic-related liabilities on insurers' portfolios is the presence of exclusions and/or other policy terms that limit losses payable for these injuries. Exclusions may be written on a substance basis or a product basis and will almost certainly be tested in court. When faced with the multiple exposures from plastic and related chemicals the court may also determine which chemical(s) or exposure(s) caused the plaintiffs' injuries and those determinations may affect insurance coverage, in terms of applicable policy periods and other policy terms.

Another important feature in most General Liability policies is a policy provision commonly referred to as the "pollution exclusion". Rapid evolution of the pollution exclusion occurred between mid-1980s and the late 1990s, precipitated by adverse interpretation of the then-prevailing "sudden and accidental" pollution coverage language. A typical pollution exclusion today explicitly excludes cleanup and remediation costs. Furthermore, bodily injury and property damage arising from the release of pollutants (a term defined quite broadly in the policy) are only covered in limited circumstances. With a few explicit exceptions, bodily injury and property damage arising from the release of pollutants on the insured's premises or resulting from ongoing operations off-premises is not covered. Bodily injury and property damage arising from the release of pollution from products or completed operations is not excluded except in specific circumstances, such as when the pollutants are considered waste. This coverage for bodily injury and property damage arising from the release of pollutants not otherwise excluded is often referred to as "product pollution". Further complications may arise if plaintiffs' bodily injury is due to ingesting, e.g., microplastic from apples. These novel problems for the insurance industry bring significant coverage uncertainty.

Older policies – many of which may still respond to plastic-related claims – will contain older incarnations of pollution exclusions. A common version prior to the mid-1980s distinguished "sudden and accidental" discharges – which are generally covered – from gradual discharge, which was generally not covered. Some courts interpreting these contracts construed "sudden and accidental" to be effectively synonymous with "expected and intended", ruling that discharges taking place over many decades still qualified as "sudden and accidental".

When managing complex risks like plastic and related chemicals it must be noted that the industrial footprint spans a wide range of business activities and industries. Plastic liability risks are spread through large sections of the economy because of the ubiquity of plastic in modern life, leading to widespread exposure and significant cross-industry clash potential.

Water remediation

The treatment of widespread drinking water remediation under general liability insurance policies is not yet settled. While pollution cleanup and related remediation claims are generally excluded from GL policies through the pollution exclusion, exceptions do remain, most notably for "product pollution". A key question in coverage determination will be whether drinking water remediation is deemed to be property damage, as opposed to cleanup or remediation, which would be a more likely determination for remediation of water source locations.

PFAS remediation lawsuits will be instructive here as coverage parameters are determined. While some releases are certainly excluded via the pollution exclusion, others, including contamination from firefighting foam, may be found to be covered as property damage under the relevant insuring agreements.

The legal causes of action may also determine whether nanoplastic remediation is covered by insurance. In the wake of the opioid litigation some insurers are clarifying that their contracts are not meant to cover claims that an insured's product caused a public nuisance nor pay for the abatement costs. On the other hand, plaintiffs may allege that the presence of nanoplastic in water is not a public nuisance but is property damage, in which case coverage is more likely to be applicable.

Risk management

The preceding discussion makes clear that the contours of potential plastic litigation are similar in scope to other large casualty risks past and present such as asbestos and PFAS. Insurers must manage the risk their insureds will be named in lawsuits alleging bodily injury, property damage, or financial harm attributable to the manufacture, use, and disposal of plastic. Named insureds will need to defend themselves and, in some cases, pay settlements. Policy language may eliminate coverage in some cases, but insurers need to prepare for the possibility it will not.

Proper risk management begins by estimating an insurer's exposure to plastic mass litigation in the same manner they might estimate their exposure to a natural catastrophe that has yet to occur. No carrier writes property insurance in Florida without first considering their exposure were a hurricane to make landfall in the coming policy year. Similarly, casualty insurers should not be writing companies in the plastics stream of commerce without examining their potential exposure in the event science strengthens to the point that the plaintiffs' bar believes plastics litigation has a sufficiently high risk-adjusted rate of return. The virtual landscape of plastic liability is perhaps more complex than say the physical map of a flood, but the risk management principle in both cases is the same. One cannot diversify against what one cannot see.

Armed with an estimate – ideally a probabilistic estimate – of how their in-force and legacy casualty books – general liability, D&O, environmental impairment liability – are exposed to a plastics mass litigation event, carriers can then steer their ships via underwriting and capital management. This does not mean excluding all claims stemming from plastics litigation. After all, property insurers continue to write coastal property in Florida despite the risk of hurricane. Property insurers accumulate hurricane exposure with their eyes wide open knowing a loss is a distinct possibility, but that they have charged an appropriate premium for the risk that allows them to maintain sufficient capital to withstand a catastrophic event. Casualty carriers must do the same, writing plastics with an eye toward shifting their portfolios away from plastic risks that have accumulated beyond current risk tolerances.

As in any competitive market, individual casualty carriers cannot dictate price. But as carriers become aware of their aggregations of plastic risk, they may mitigate future exposure by writing less limit, attaching higher in the tower, applying targeted exclusions, or getting off risks entirely. They may also purchase additional reinsurance, maintain higher reserves, or seek to transfer legacy plastic risks to the run-off market. All these activities effectively reduce the supply of liability coverage for plastics, driving prices up. Forced to bear more risk themselves or pay more to transfer it to capital markets, manufacturers and downstream users of plastics have greater incentive to move away from business as usual.

The extent to which insurance facilitates the internalization of plastic risks in this manner, of course, depends heavily on the extent to which society seeks to address the risks of plastics through the courts rather than the legislature. This report has highlighted many barriers to establishing plastics liability, but the law is ever shifting and where liability is stymied today, it may flow in the future if society cannot find other means of reducing the risk plastic poses to human health and the environment.

Endnotes

1 The Hill Criteria, named after Sir Austin Bradford Hill, describe the factors one should use to establish whether correlative evidence as found in epidemiology can establish causation. See Hill AB. The Environment and Disease: Association or Causation? Proc R Soc Med. 1965 May;58(5):295-300. PMID: 14283879; PMCID: PMC1898525.

2 Omran GA, Gaber HD, Mostafa NAM, Abdel-Gaber RM, Salah EA. Potential hazards of bisphenol A exposure to semen quality and sperm DNA integrity among infertile men. Reprod Toxicol. 2018 Oct;81:188-195. doi: 10.1016/j.reprotox.2018.08.010. Epub 2018 Aug 10. PMID: 30103013.

3 Zhou F, Jin Z, Zhu L, Huang F, Ye A, Hou C. A preliminary study on the relationship between environmental endocrine disruptors and precocious puberty in girls. J Pediatr Endocrinol Metab. 2022 Jun 14;35(8):989-997. doi: 10.1515/jpem-2021-0691. PMID: 35692072.

4 Jiang Y, Li J, Xu S, Zhou Y, Zhao H, Li Y, Xiong C, Sun X, Liu H, Liu W, Peng Y, Hu C, Cai Z, Xia W. Prenatal exposure to bisphenol A and its alternatives and child neurodevelopment at 2 years. J Hazard Mater. 2020 Apr 15;388:121774. doi: 10.1016/j.jhazmat.2019.121774. Epub 2019 Nov 28. PMID: 32001102.

5 Huang YF, Chang CH, Chen PJ, Lin IH, Tsai YA, Chen CF, Wang YC, Huang WY, Tsai MS, Chen ML. Prenatal Bisphenol a Exposure, DNA Methylation, and Low Birth Weight: A Pilot Study in Taiwan. Int J Environ Res Public Health. 2021 Jun 7;18(11):6144. doi: 10.3390/ijerph18116144. PMID: 34200176; PMCID: PMC8201193.

6 Hoepner LA, Whyatt RM, Widen EM, Hassoun A, Oberfield SE, Mueller NT, Diaz D, Calafat AM, Perera FP, Rundle AG. Bisphenol A and Adiposity in an Inner-City Birth Cohort. Environ Health Perspect. 2016 Oct;124(10):1644-1650. doi: 10.1289/EHP205. Epub 2016 May 17. PMID: 27187982; PMCID: PMC5047776.

7 Tang N, Wang D, Chen X, Zhang M, Lv W, Wang X. Maternal bisphenol A and triclosan exposure and allergic diseases in childhood: a meta-analysis of cohort studies. Environ Sci Pollut Res Int. 2022 Jun 28. doi: 10.1007/s11356-022-21575-2. Epub ahead of print. PMID: 35764729.

8 Kim JI, Lee YA, Shin CH, Hong YC, Kim BN, Lim YH. Association of bisphenol A, bisphenol F, and bisphenol S with ADHD symptoms in children. Environ Int. 2022 Mar;161:107093. doi: 10.1016/j.envint.2022.107093. Epub 2022 Jan 22. PMID: 35077929.

9 Chang WH, Wu MH, Pan HA, Guo PL, Lee CC. Semen quality and insulin-like factor 3: Associations with urinary and seminal levels of phthalate metabolites in adult males. Chemosphere. 2017 Apr;173:594-602. doi: 10.1016/j.chemosphere.2017.01.056. Epub 2017 Jan 16. PMID: 28152410.

10 Park O, Park JT, Chi Y, Kwak K. Association of phthalates and early menarche in Korean adolescent girls from Korean National Environmental Health Survey (KoNEHS) 2015-2017. Ann Occup Environ Med. 2021 Feb 1;33:e4. doi: 10.35371/aoem.2021.33.e4. PMID: 34754465; PMCID: PMC7952777.

11 Yuan G, Zeng Y, Hu G, Liu Y, Wei L, Liu P, Liu G, Cheng J. Inverse association of certain seminal phthalate metabolites with semen quality may be mediated by androgen synthesis: A cross-sectional study from the South China. Environ Int. 2021 Jun;151:106459. doi: 10.1016/j.envint.2021.106459. Epub 2021 Mar 6. PMID: 33684682.

12 Liao KW, Kuo PL, Huang HB, Chang JW, Chiang HC, Huang PC. Increased risk of phthalates exposure for recurrent pregnancy loss in reproductive-aged women. Environ Pollut. 2018 Oct;241:969-977. doi: 10.1016/j.envpol.2018.06.022. Epub 2018 Jun 14. PMID: 30029331.

13 Al-Saleh I. The relationship between urinary phthalate metabolites and polycystic ovary syndrome in women undergoing *in vitro* fertilization: Nested case-control study. Chemosphere. 2022 Jan;286(Pt 1):131495. doi: 10.1016/j.chemosphere.2021.131495. Epub 2021 Jul 14. PMID: 34293567.

14 Kupsco A, Wu H, Calafat AM, Kioumourtzoglou MA, Cantoral A, Tamayo-Ortiz M, Pantic I, Pizano-Zárate ML, Oken E, Braun JM, Deierlein AL, Wright RO, Téllez-Rojo MM, Baccarelli AA, Just AC. Prenatal maternal phthalate exposures and trajectories of childhood adiposity from four to twelve years. Environ Res. 2022 Mar;204(Pt B):112111. doi: 10.1016/j.envres.2021.112111. Epub 2021 Sep 23. PMID: 34563522; PMCID: PMC8678304.

15 Lee DW, Lim YH, Shin CH, Lee YA, Kim BN, Kim JI, Hong YC. Prenatal exposure to di-(2-ethylhexyl) phthalate and decreased skeletal muscle mass in 6-year-old children: A prospective birth cohort study. Environ Res. 2020 Mar;182:109020. doi: 10.1016/j.envres.2019.109020. Epub 2019 Dec 12. PMID: 31863942.

16 Wang LJ, Huang YH, Chou WJ, Lee SY, Chang HY, Chen CC, Chao HR. Interrelationships among growth hormone, thyroid function, and endocrine-disrupting chemicals on the susceptibility to attention-deficit/hyperactivity disorder. Eur Child Adolesc Psychiatry. 2022 Feb 4. doi: 10.1007/s00787-021-01886-4. Epub ahead of print. PMID: 35119524.

17 Johnson PI, Stapleton HM, Mukherjee B, Hauser R, Meeker JD. Associations between brominated flame retardants in house dust and hormone levels in men. Sci Total Environ. 2013 Feb 15;445-446:177-84. doi: 10.1016/j.scitotenv.2012.12.017. Epub 2013 Jan 16. PMID: 23333513; PMCID: PMC3572297.

18 Liang J, Liu S, Liu T, Yang C, Wu Y, Jennifer Tan HJ, Wei B, Ma X, Feng B, Jiang Q, Huang D, Qiu X. Association of prenatal exposure to bisphenols and birth size in Zhuang ethnic newborns. Chemosphere. 2020 Aug;252:126422. doi: 10.1016/j.chemosphere.2020.126422. Epub 2020 Mar 4. PMID: 32199162.

19 Ongono JS, Dow C, Gambaretti J, Severi G, Boutron-Ruault MC, Bonnet F, Fagherazzi G, Mancini FR. Dietary exposure to brominated flame retardants and risk of type 2 diabetes in the French E3N cohort. Environ Int. 2019 Feb;123:54-60. doi: 10.1016/j.envint.2018.11.040. Epub 2018 Nov 26. PMID: 30496982.

20 Gao Y, Chen L, Wang C, Zhou Y, Wang Y, Zhang Y, Hu Y, Ji L, Shi R, Cui C, Ding G, Jin J, Tian Y. Exposure to polybrominated diphenyl ethers and female reproductive function: A study in the production area of Shandong, China. Sci Total Environ. 2016 Dec 1;572:9-15. doi: 10.1016/j.scitotenv.2016.07.181. Epub 2016 Jul 30. PMID: 27485910.

21 Ji H, Liang H, Wang Z, Miao M, Wang X, Zhang X, Wen S, Chen A, Sun X, Yuan W. Associations of prenatal exposures to low levels of Polybrominated Diphenyl Ether (PBDE) with thyroid hormones in cord plasma and neurobehavioral development in children at 2 and 4 years. Environ Int. 2019 Oct;131:105010. doi: 10.1016/j.envint.2019.105010. Epub 2019 Jul 18. PMID: 31326823.

22 Gaylord A, Trasande L, Kannan K, Thomas KM, Lee S, Liu M, Levine J. Persistent organic pollutant exposure and celiac disease: A pilot study. Environ Res. 2020 Jul;186:109439. doi: 10.1016/j.envres.2020.109439. Epub 2020 May 11. PMID: 32409013.

23 Doherty BT, Hoffman K, Keil AP, Engel SM, Stapleton HM, Goldman BD, Olshan AF, Daniels JL. Prenatal exposure to organophosphate esters and behavioral development in young children in the Pregnancy, Infection, and Nutrition Study. Neurotoxicology. 2019 Jul;73:150-160. doi: 10.1016/j.neuro.2019.03.007. Epub 2019 Apr 3. PMID: 30951742; PMCID: PMC6635002.

24 Liu Y, Li Y, Dong S, Han L, Guo R, Fu Y, Zhang S, Chen J. The risk and impact of organophosphate esters on the development of female-specific cancers: Comparative analysis of patients with benign and malignant tumors. J Hazard Mater. 2021 Feb 15;404(Pt B):124020. doi: 10.1016/j.jhazmat.2020.124020. Epub 2020 Oct 2. PMID: 33049558.

25 Wang C, Chen Z, Lu Y, Wang L, Zhang Y, Zhu X, Song J. Neurotoxicity and related mechanisms of flame retardant TCEP exposure in mice. Toxicol Mech Methods. 2020 Sep;30(7):490-496. doi: 10.1080/15376516.2020.1765060. Epub 2020 May 13. PMID: 32397869.

26 Oliveri Conti G, Ferrante M, Banni M, Favara C, Nicolosi I, Cristaldi A, Fiore M, Zuccarello P. Micro- and nano-plastics in edible fruit and vegetables. The first diet risks assessment for the general population. Environ Res. 2020 Aug;187:109677. doi: 10.1016/j.envres.2020.109677. Epub 2020 May 20. PMID: 32454310.

27 Huang D, Zhang Y, Long J, Yang X, Bao L, Yang Z, Wu B, Si R, Zhao W, Peng C, Wang A, Yan D. Polystyrene microplastic exposure induces insulin resistance in mice via dysbacteriosis and pro-inflammation. Sci Total Environ. 2022 Sep 10;838(Pt 1):155937. doi: 10.1016/j.scitotenv.2022.155937. Epub 2022 May 16. PMID: 35588841.

28 Yan Z, Liu Y, Zhang T, Zhang F, Ren H, Zhang Y. Analysis of Microplastics in Human Feces Reveals a Correlation between Fecal Microplastics and Inflammatory Bowel Disease Status. Environ Sci Technol. 2022 Jan 4;56(1):414-421. doi: 10.1021/acs.est.1c03924. Epub 2021 Dec 22. PMID: 34935363.

29 Horvatits T, Tamminga M, Liu B, Sebode M, Carambia A, Fischer L, Püschel K, Huber S, Fischer EK. Microplastics detected in cirrhotic liver tissue. EBioMedicine. 2022 Aug;82:104147. doi: 10.1016/j.ebiom.2022.104147. Epub 2022 Jul 11. PMID: 35835713; PMCID: PMC9386716.

30 Boucher J, Friot D. (2017). Primary Microplastics in the Oceans: A Global Evaluation of Sources. IUCN, Gland, Switzerland. https://portals.iucn.org/library/sites/library/files/documents/2017-002-En.pdf

31 Paruta P, Pucino M, Boucher J. (2021). Plastic Paints the Environment, EA-Environmental Action, Lausanne, Switzerland. https://www.e-a.earth/_files/ugd/425198_6f553fe5eb444350bb61b4acee3aef14.pdf

32 Dyachenko A, Mitchel J, Arsen N. Extraction and identification of microplastic particles from secondary wastewater treatment (WWTP) effluent. Anal. Methods. 2017;(9):1412-1418. https://doi.org/10.1039/C6AY02397E

33 Koelmans AA, Mohamed Nor NH, Hermsen E, Kooi M, Mintenig SM, De France J. Microplastics in freshwaters and drinking water: Critical review and assessment of data quality. Water Res. 2019 May 15;155:410-422. doi: 10.1016/j.watres.2019.02.054. Epub 2019 Feb 28. PMID: 30861380; PMCID: PMC6449537.

34 Hendrickson E, Minor E, Schreiner K. Microplastic Abundance and Composition in Western Lake Superior as Determined via Microscopy, Pyr-GC/MS, and FTIR. Environ Sci Technol. 2018 Jan18;52(4), 1787-1796 https://doi.org/10.1021/acs.est.7b05829

35 Microplastics in drinking-water. (2019 August 28). World Health Organization https://www.who.int/publications/i/item/9789241516198

36 McCarthy, N. (2018, March 16) Study Finds Microplastics In 93% Of Bottled Water. Statista. https://www.statista.com/chart/13255/study-finds-microplastics-in-93-of-bottled-water/

37 Baldwin AK, Corsi SR, Mason SA. Plastic Debris in 29 Great Lakes Tributaries: Relations to Watershed Attributes and Hydrology. Environ Sci Technol. 2016 Oct 4;50(19):10377-10385. doi: 10.1021/acs.est.6b02917. Epub 2016 Sep 14. PMID: 27627676.

38 Koelmans AA, Mohamed Nor NH, Hermsen E, Kooi M, Mintenig SM, De France J. Microplastics in freshwaters and drinking water: Critical review and assessment of data quality. Water Res. 2019 May 15;155:410-422. doi: 10.1016/j.watres.2019.02.054. Epub 2019 Feb 28. PMID: 30861380; PMCID: PMC6449537.

39 Kosuth M, Mason SA, Wattenberg EV. Anthropogenic contamination of tap water, beer, and sea salt. PLoS One. 2018 Apr 11;13(4):e0194970. doi: 10.1371/journal.pone.0194970. PMID: 29641556; PMCID: PMC5895013.

40 Dyachenko A, Mitchel J, Arsen N. Extraction and identification of microplastic particles from secondary wastewater treatment (WWTP) effluent. Anal. Methods. 2017;(9):1412-1418. https://doi.org/10.1039/C6AY02397E

41 Costa AR, de Pinho MN. Performance and cost estimation of nanofiltration for surface water treatment in drinking water production. Desalination. 2006 Sept;196(1-3):55-65 https://doi.org/10.1016/j.desal.2005.08.030

42 Amouamouha M, Gholikandi GB. (2018). Performance investigation and cost evaluation of nanofiltration membranes in groundwater remediation. 5th international conference on small and decentralized water and wastewater treatment plants. https://www.researchgate.net/publication/341984702_Performance_investigation_and_cost_evaluation_of_nanofiltration_membranes_in_groundwater_remediation

43 Shahmansouri A, Bellona C. Nanofiltration technology in water treatment and reuse: applications and costs. Water Sci Technol. 2015;71(3):309-19. doi: 10.2166/wst.2015.015. PMID: 25714628.

44 Guo T, Englehardt J, Wu T. Review of cost versus scale: water and wastewater treatment and reuse processes. Water Sci Technol. 2014;69(2):223-34. doi: 10.2166/wst.2013.734. PMID: 24473289.

45 "Clean Watersheds Needs Survey 2012 Data and Reports" United States Environmental Protection Agency, https://ordspub.epa.gov/ords/cwns2012/f?p=241:29::::RP::. Accessed 07 Sept. 2022

46 Dieter CA, Maupin MA, Caldwell RR, Harris MA, Ivahnenko TI, Lovelace JK, Barber NL, and Linsey KS. (2018). Estimated use of water in the United States in 2015. U.S. Geological Survey Circular, 1441. https://pubs.usgs.gov/circ/1441/circ1441.pdf

47 Microplastics in drinking-water. (2019 August 28). World Health Organization https://www.who.int/publications/i/item/9789241516198

48 Boucher J, Friot D. (2017). Primary Microplastics in the Ocean: A Global Evaluation of Sources. IUCN, Gland, Switzerland. https://portals.iucn.org/library/sites/library/files/documents/2017-002-En.pdf

49 Paurta P, Pucino M, Boucher J. (2021). Plastic Paints The Environment: A global assessment of paint's contribution to plastic leakage to Land Ocean & Waterways. EA – Environmental Action, Lausanne, Switzerland. https://www.e-a.earth/_files/ugd/425198_6f553fe5eb444350bb61b4acee3aef14.pdf

50 United States Food and Drug Administration. (2022, February 25). The Microbead-Free Waters Act: FAQs https://www.fda.gov/cosmetics/cosmetics-laws-regulations/microbead-free-waters-act-faqs Accessed 07 Sept. 2022

51 Breakfreefromplastic. (2021 October 25). The Coca-Cola Company and PepsiCo named top plastic polluters for the fourth year in a row. https://www.breakfreefromplastic.org/2021/10/25/the-coca-cola-company-and-pepsico-named-top-plastic-polluters-for-the-fourth-year-in-a-row/

52 Waste Management World. (2021 December 2). The top plastic polluters 2021. https://waste-management-world.com/materials/the-top-plastic-polluters/

53 Minderoo Foundation. (2021 November 22). Top 100 Polymer Producers. https://www.minderoo.org/plastic-waste-makers-index/data/indices/producers/

54 Coffee J. (2018 March 19). Securities Litigation in 2017: "It Was the Best of Times, It Was the Worst of Times". The CLS Blue Sky Blog. https://clsbluesky.law.columbia.edu/2018/03/19/securities-litigation-in-2017-it-was-the-best-of-times-it-was-the-worst-of-times/

55 LaCroix K. (2017 July 17). Turning Events into Securities Suits. The D&O Diary. https://www.dandodiary.com/2017/07/articles/securities-litigation/turning-events-securities-suits/

56 LaCroix K. (2018 March 27). Scrutinizing even driven securities litigation. The D&O Diary. https://www.dandodiary.com/2018/03/articles/securities-litigation/scrutinizing-

MINDEROO.ORG