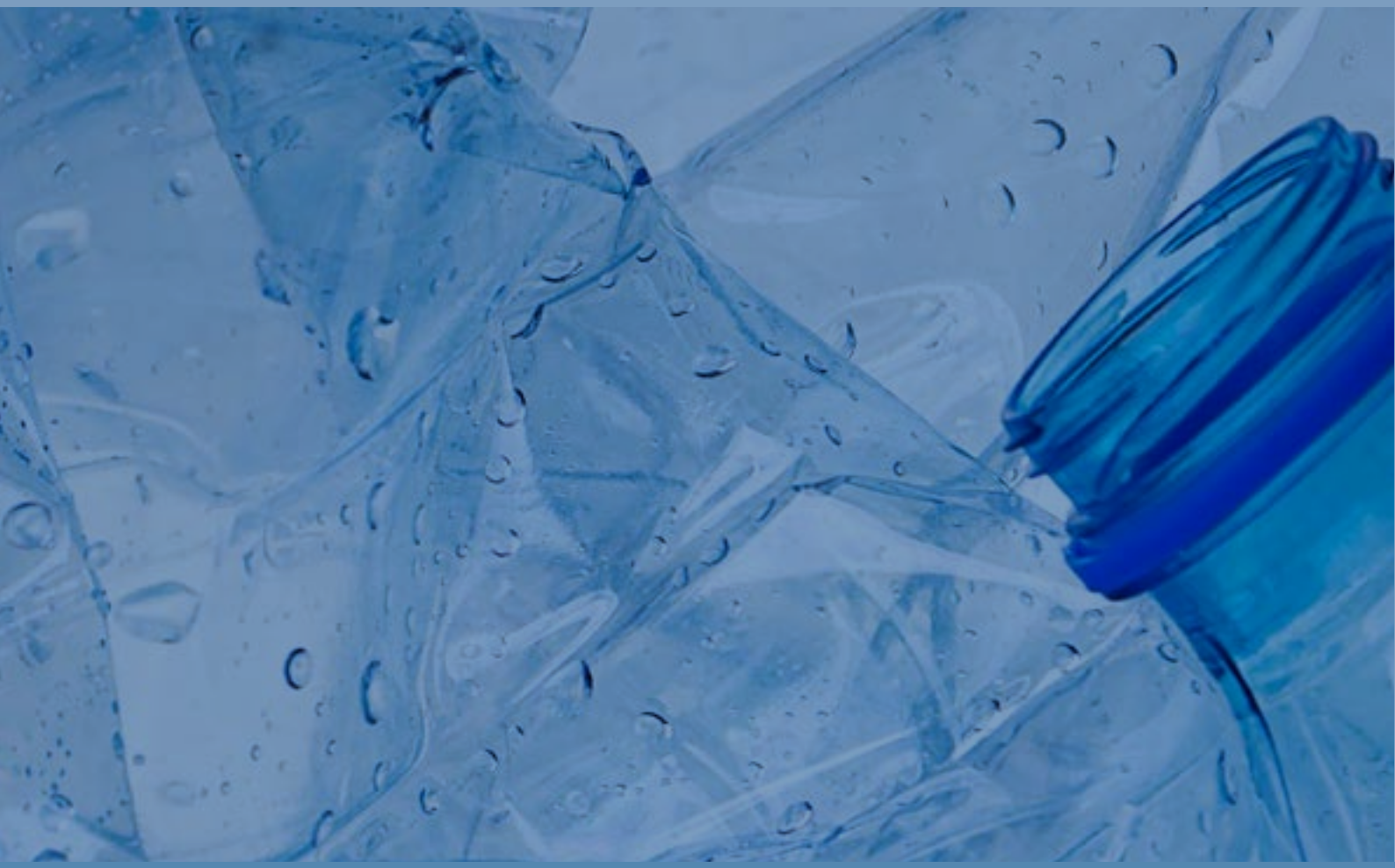
# ANNEX 1:

## The Social Cost of Plastic-Related Harms



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# ABSTRACT


## Introduction

**Plastics pervade almost all aspects of our lives, from the buildings in which we live to the water we drink. The mass of plastic produced – cumulatively more than eight billion tonnes – is already estimated to be greater than the wet biomass of all the planet's animals.**

Plastics are inexpensive to produce, lightweight, versatile, and durable. However, these same qualities underpin their capacity to harm nature, economies, and human health. Plastics, and the complex blend of performance-enhancing chemicals added to them, are highly mobile within both the environment and organisms. Plastic-related chemical additives and micro-nano-plastics (plastics less than 5 mm in diameter, "MNP") are known to cross both continents and cell membranes. They have half-lives measured in decades or centuries, and they endure in the face of the most extreme environmental conditions. This mobility and durability combine to make them ubiquitous. And it is their ubiquity that, in turn, amplifies the potential harm to plants, animals, and humans alike.

### Objective

In this Annex, we seek to further an understanding of the expected social costs of these plastic-related harms, by:

- assessing the scientific consensus that a harm is caused by plastic-related pollution
- estimating the size of the cost to society of the plastic-related harm
- assessing how likely this consensus (and size) is to change over time

### Methods

We performed a review of the literature relating to plastics to identify the specific harms that different potential plastic-related hazards (macroplastics, MNP, chemicals, carbon) may have on human health, economies and ecosystem services, and nature. We then extracted relevant data from the literature to assess the scientific consensus that the hazard causes harm, to estimate the size of social cost, and to assess how likely the consensus (and size) is to change over time. Grouping of similar hazard-harm pairs was then performed.

## Results

We identified 92 individual hazard-harm pairs, consolidated into 20 groups. Summing the estimated social costs of all identified harms (before factoring in consensus levels on causation or potential for change over time) yields a theoretical social cost of hundreds of billions of dollars. The majority of the social costs arise from harms to human health, while human health harms from chemical additives had some of the strongest consensus on causation.

After grouping the harms, we clustered them into known, indeterminate, emerging, and immature harms. Known harms are characterised by high and stable consensus on causation. Emerging harms are characterised by low or medium consensus that is very likely to move to high consensus in the near future. We found six of the 14 known and emerging harms, and most of the social costs, to be borne by harms to human health.

## Discussion

We suggest that the plastics industry, their insurers and investors, policy-makers and financial services supervisors should be proactive with respect to managing and mitigating the harms identified, with urgent priority given to those known and emerging harms with high estimated social costs. The sources of these harms are pervasive and numerous, and ubiquitous exposure to many of them is, in many cases, inevitable.

While we have attempted a comprehensive assessment, the state of our knowledge varies widely, and our assessments may change as more hazards are identified, further evidence of causation is established, and more work is undertaken to size social costs.

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*A seagull pecks at a discarded surgical gown in a trash pit at Recology on April 2, 2021 in San Francisco, California. Photo credit: Justin Sullivan/Getty Images*

# 1. INTRODUCTION

## 1.1 What do we know?

### 1.1.1 Plastics have many benefits

Plastics are synthetic carbon-based polymers mixed with a complex blend of chemical additives.[1] Over 10,500 chemical additives have been recorded including plasticisers, flame retardants, antioxidants, UV stabilisers and colourants. Of these, more than 2,486 (24 per cent) are substances that have been classified as being of potential concern because they meet one or more criteria of persistence, bioaccumulation, or toxicity.[2] A further 1,254 (12 per cent) are also high-production volume chemicals that have been classed as substances of high concern by researchers.[2] Many of these substances are seldom studied, inadequately regulated, or are not publicly disclosed, as they are held as trade secrets by their manufacturers.[3]

Despite being relatively new materials (large-scale manufacturing began in the 1950s), plastics are increasingly dominating use cases that previously belonged to traditional materials such as wood, metals, glass, and cotton.1 Today, plastics play many essential roles in the economy. Over eight billion tonnes were produced between 1950 and 2015, and today, annual production has reached more than 380 million tonnes.[3] The demand for production is likely to grow. Some forecasts estimate that plastic production will double by 2050, driven by population growth, greater per capita consumption (especially in developing nations), and the identification of more use cases.[4]

The reason for the historical and projected demand for plastics is simple: they have many benefits over traditional materials. They are inexpensive to manufacture, highly versatile, lightweight, durable, and waterproof.[5] They also claim to carry substantial benefits for reducing carbon emissions. For example, the weight of vehicles (and thereby fuel consumption) can be reduced by replacing traditional, heavier materials like steel and iron with lightweight plastic.[4]

### 1.1.2 The social costs of plastic-related harms are gaining increasing recognition

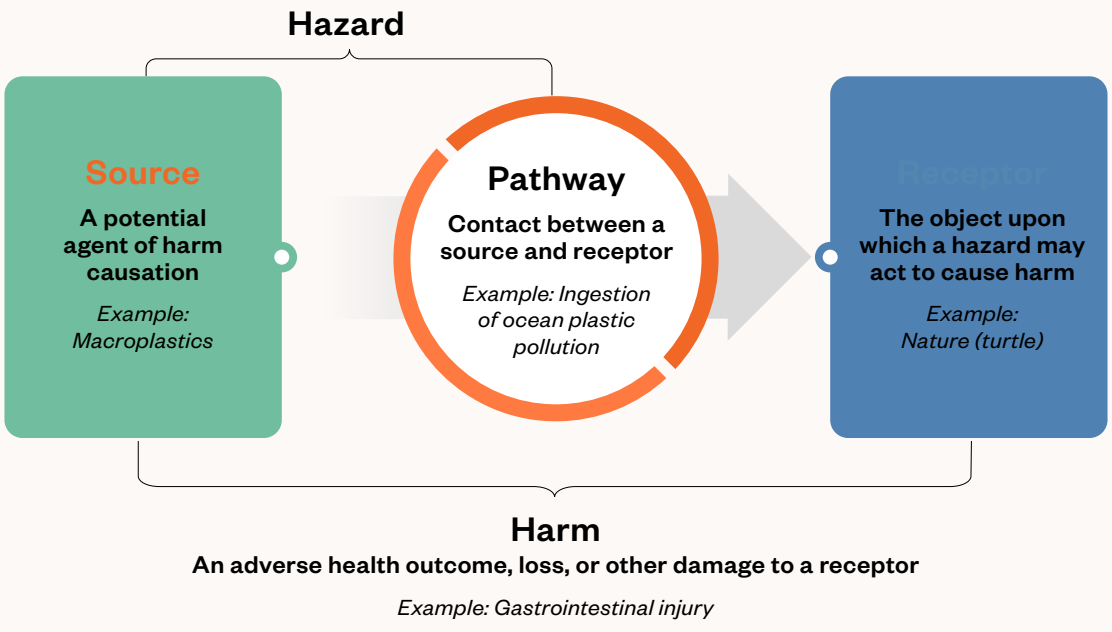
This array of properties and processes has led to the concept of 'plastic pollution', which is when plastic-related hazards – macroplastic waste, MNP, plastic-related chemicals, and gases leaked into the environment during production – cause harm to Human Health, Economies & Ecosystem Services (E&ES), and Nature. In **Figures 1** and **2**, we define the relationships between the key concepts that underpin plastic pollution and the terms used in this report.[6]

Plastic pollution is expected to increase as waste management systems fail to keep up with the increased waste generated, growing from 90 million tonnes per annum to 240 million tonnes per annum by 2040 (**Figure 3**).[7] Cumulatively, an estimated 6.3 billion tonnes of plastic-related pollution have accumulated in the environment to date.[1]

**Figure 1:** Framework for harm identification

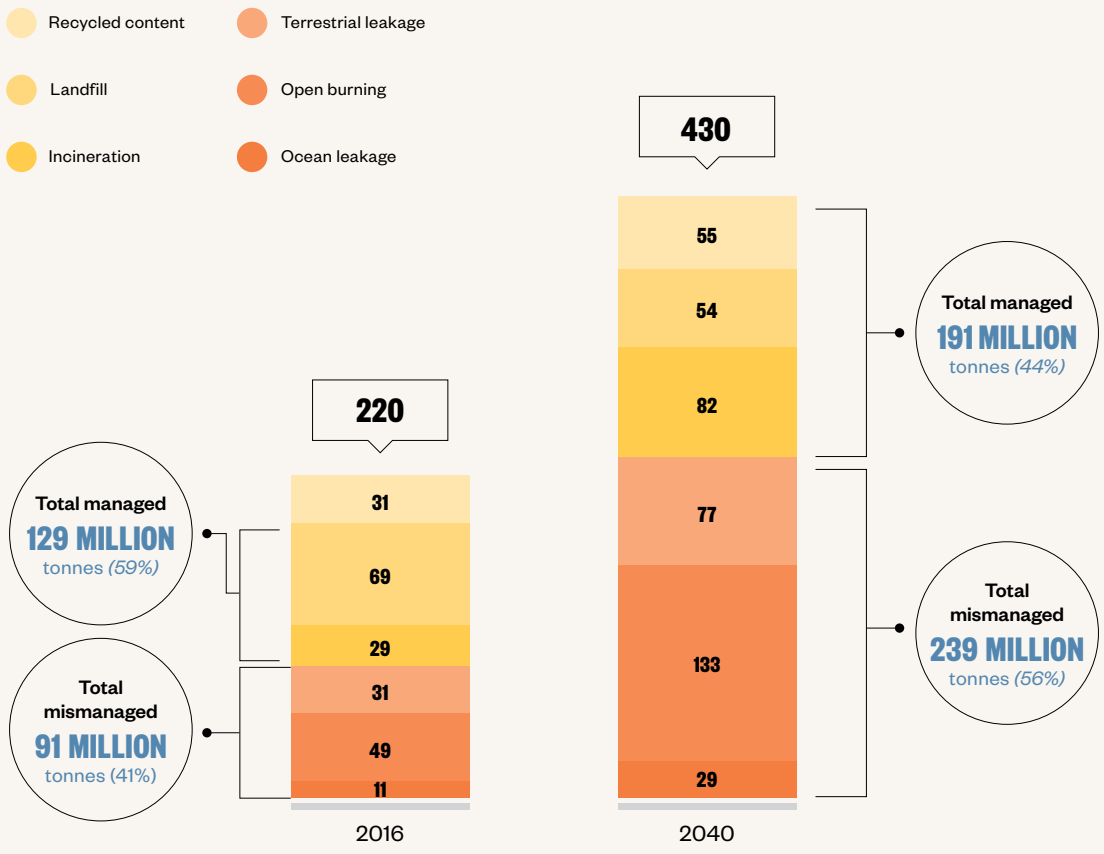
| Sources | Receptors: Human Health<br>Disease, injury, or other adverse health outcomes in humans | Economies & Ecosystem Services<br>Losses in income, asset value, or natural capital | Nature<br>Disease, injury, and other adverse health outcomes in plants & non-human animals |
|---|---|---|---|
| Chemical additives | The effects of the different types of chemicals added to plastics to give them specific properties or otherwise make them more useful for their intended purpose | | |
| Macroplastics | The effects caused by plastic products >5mm in diameter, and the direct effects of their lifecycle from production to end-of-life disposal | | |
| MNP | The effects of pieces of plastic that are <5mm in diameter created for a specific use (primary MNP) or fragmented from larger pieces of plastic (secondary MNP) | | |
| $CO_2e$ emissions and climate change | The effects of the emissions from $CO_2$ and other greenhouse gases (GHG), which are released at a number of points in the plastic lifecycle | | |

**Figure 2:** Definition of key concepts



Global concern around the accumulating evidence on the risks of plastic pollution is reflected in the recent resolution by the UN Environment Assembly to forge a global legally binding agreement to end plastic pollution.[8] The treaty aims to address "the growing problem of plastic waste in the world's oceans, rivers, and landscapes". Additional legislation on specific plastic uses has emerged in a wide variety of geographies, including single-use plastic bans in the European Union (EU), regulation of plastic bags in 127 countries, and at least a dozen countries with bans on microbeads in personal care products.[9] Likewise, many countries are focusing on better disposal of plastics: 63 countries have mandates for extended producer responsibility of single-use plastics, including elements of deposit-return schemes, product take-back schemes, and recycling targets.[9] There are also trends towards tighter regulation of additives in plastics, as well as the processes for evaluating safety. This is exemplified by a recent EU review of bisphenol A (BPA), which saw the recommended Tolerable Daily Intake drastically reduced from 4,000 ng/kg-day to just 0.04 ng/kg-day, two orders of magnitude below mean exposure levels.[10] This legislation reflects the increasing recognition that endocrine disruptors can have effects at very low concentrations/body burdens, and, more generally, of the harms posed by plastics.

**Figure 3:** Plastic leakage (mismanaged waste) now and into the future




*Data and projections taken from Lau et al. 2020.[42]

### 1.1.3 Risks of harm are shaped by the physical fundamentals and lifecycle of plastics

The qualities that make plastics so useful also underpin their known and emerging harms.[6] With 1.8 billion tonnes of greenhouse gases (GHG) emitted by plastic production and disposal,[9, 11] and only around 10 per cent of plastic waste recycled,[3] much of the emphasis in the lay literature focuses on its start and end points. However, plastic has leakage points throughout its lifecycle (**Figure 4**)[a], including:

**1**

#### Production

From raw material extraction to polymerisation (e.g., $CO_2$ emissions and industrial pollution). Plastic production generates approximately four per cent of total anthropogenic GHG emissions per year.[9, 12] In addition, some workers at plastic (primary) production and extrusion facilities experience high levels of exposure.[13]

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**2**

#### Use

For example, textile shedding, tyre dust, abrasives, coatings. A single wash of plastic garments can release millions of fibres into wastewater.[14] Polyester, rayon, and nylon microfibres are very hard to filter from water,[15] and are deposited from wastewater onto shorelines across the world.[16] Tyres release a global average of 0.81 kg/year, contributing an estimated 5–10 per cent of all plastic ending up in the ocean.[17] In air, 3–7 per cent of the particulate matter (PM2.5) is estimated to consist of tyre wear and tear. Tyre-sourced plastic particles contain a number of potentially toxic additives, and can be found in remote Arctic and ocean environments.[18, 19]

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**3**

#### Waste management

For example, incinerator pollution, landfill leachate. Incineration emits almost 100 million tonnes of GHG per year.[11] At about 900 kg $CO_2$e per tonne of plastic waste, this amounts to roughly 15 times the comparable emissions in landfill.[20] Bottom ash from municipal solid waste incinerators can generate over 100,000 micro-particles per tonne of ash.[21]

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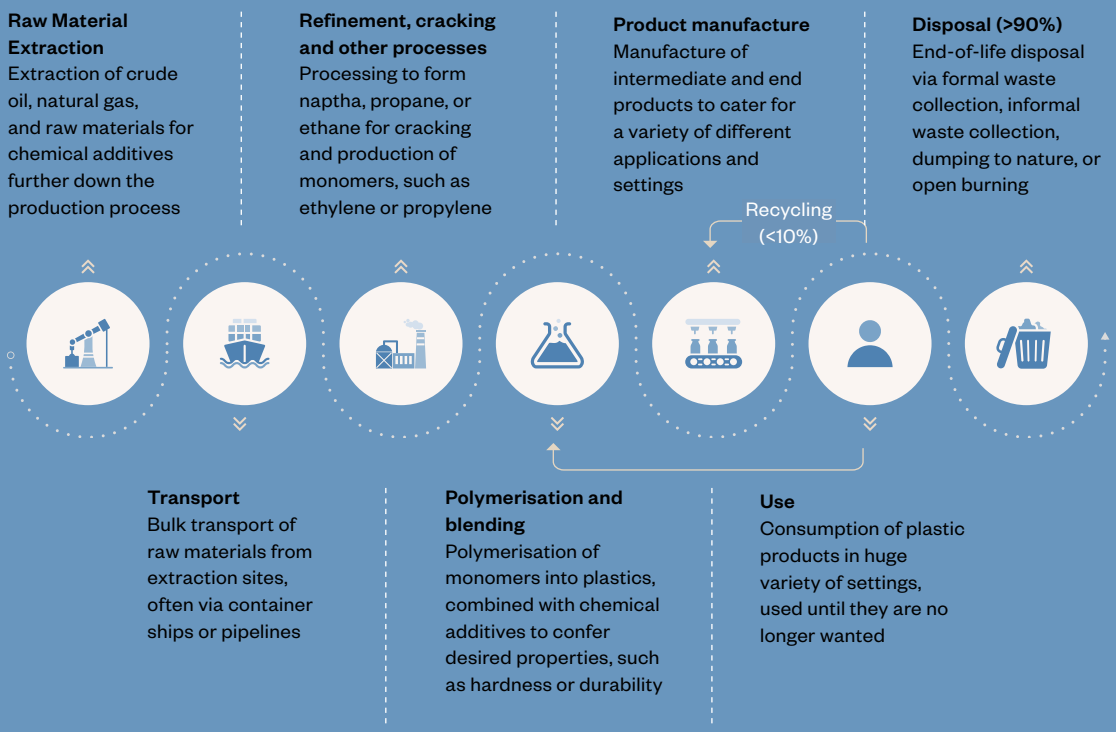
**4**

#### Post-consumer “leakage”

For example, dumping of directly into the environment. Due to a number of factors (low value of discarded plastics, high technical difficulty of recycling, capital intensity of waste management systems, and lacking producer responsibility schemes in most countries), only half of plastic waste is collected, treated, or safely stored, and less than nine per cent is recycled.[22]

[a]Adapted from Symeonides et al. 2021 under a Creative Commons Licence.[3]

**Figure 4:** The plastic lifecycle and value chain




## Mobility

The light weight of plastics means that once it enters the environment, it is highly mobile. In the environment, plastics larger than 5 mm ('macroplastics') can break down into smaller fragments, fibres, and spheres. These are classified by size into nanoplastics (less than 100 nm) or microplastics (less than 5 mm), collectively termed micro-nano-plastics (MNP).[5] The small size of MNP means that they are highly mobile both within organisms (potentially crossing cell membranes)[23] and outside organisms.

As such, MNP, along with ultra-lightweight macroplastics like plastic fibres and shopping bags, can be transported by wind and air currents to locations far from their original source.[24] MNP have been found as far away as polar and high-altitude regions, and deep in the ocean.[19, 25–27] While plastic mobility through ocean currents, rivers, and winds is well-documented,[6, 18, 28] less obvious methods of dispersal exist. For example, MNP are frequently present in wastewater, which, in developed communities, undergoes treatment in wastewater treatment systems before settling in biosolid sewage sludge. This sludge is then used to fertilise soil.[15, 29, 30] From there, MNP can make their way into agricultural crops, but can also get into waterways due to rain runoff.[31] Plastics suspended in river sediments have been found to disperse widely during flood events and settle into soils.[32]

Within organisms, nanoplastics in particular have been shown in human, animal, and *in vitro* studies to cross cell membranes, as well as specialist biological barriers such as the mammalian placental barrier and blood-brain barrier.[33, 34] To date, MNP have been detected in human faeces, lung and colectomy samples as well as the placenta.[35–38] Techniques to directly detect nanoplastics in human tissue are yet to be established.[39]

## Durability and length of exposure

Plastics are persistent, with half-lives measured in decades or centuries.[40] Physical breakdown of macroplastics to MNP occurs on the scale of years to decades.[41] Chemical breakdown is so slow that its contribution to plastic breakdown is negligible.[2] Organic pollutants such as polybrominated biphenyl, polychlorinated biphenyls (PCBs), dioxins, and furans that were previously used in great volumes as chemical additives ('legacy additives') remain highly persistent and prone to bioaccumulation, despite many not having been used in plastic production for decades.[42, 43]

Plastics termed 'biodegradable' often do not chemically degrade in the environment, instead requiring specific conditions (such as high temperatures) for microbes to speed plastic breakdown.[44] There is currently a lack of evidence that biodegradable, compostable, bio-based, and oxo-degradable plastics fully degrade in natural environments.[44]

Therefore, once in the environment, the durability of plastics means that they will persist for hundreds or thousands of years. This gives rise to the potential for cumulative, long-lasting exposure to plastics and plastic-related chemicals, with important implications for toxicity.

## Ubiquity

Plastics' ubiquity is a function of their mobility and durability. Moreover, the versatility and cost-effectiveness of plastics means that their production and consumption have outpaced the ability of humanity to collect and safely dispose of them. As a result, plastics are ubiquitous in the environment and society. Almost every person and every ecosystem on earth interacts with plastics daily, either by design or because of unintentional environmental plastic accumulation,[19, 26] which is estimated at over 30 million tonnes of terrestrial leakage per year alone.[28]

**In the environment**. Plastic pollution is widespread in the ocean, in fresh water, on land, and in the air.

- **Oceans**: Marine plastic pollution is estimated to reach 29 million tonnes per annum by 2040.[7] A significant amount of MNP is attached to oceanic organic matter ('marine snow'), and disperses widely. As macroplastics degrade into smaller pieces, and as these pieces are weighted down by flora, most plastics likely gravitate towards greater depths over time.[45]
- **Fresh water**: Factory wastewater, fertiliser runoff, and sewage all contain MNP and cause drinking water contamination, albeit indirectly.[32] Even when best available treatment technology is installed, only 90 per cent of MNP can be removed from wastewater.[46] As a result, MNP-contaminated runoff from factories, municipal sewage, and even agriculture can lead to the contamination of both groundwater and above-ground fresh water sources.[47, 48]
- **Air**: MNP, primarily from legacy production of plastic and built-up waste, have been found in outdoor air. In the United States (US), road-related pollution was the dominant source, followed by marine, agriculture, and dust emissions generated downwind of population centres.[49]

**In animals**. MNP have been detected in the bloodstream of farm animals,[50] and in the intestines, stomachs, livers, and muscle tissues of wild coastal animals.[51–53] MNP bioaccumulation has been found, but studies addressing bioaccumulation of plastic-related chemical additives remain inconclusive.[54]

Gastrointestinal (GI) exposure is well documented.[55] Plastic pollution has been found in the stomach contents of animals as diverse as earthworms, birds, turtles, dolphins, and whales.[53, 56, 57] MNP (particles range in size from 130µm to 5 mm) have been found in more than 150 fish species, as well as many other aquatic organisms.[23, 58–60] As major predators, marine mammals are commonly found with MNP in their GI tracts,[52] as well as 180 bird species.[61]

Seabirds almost universally ingest macroplastics, and plastic has been found in the faeces of more than half of the small mammalian species examined in England and Wales,[62] as well as in isolated ecosystems such as Antarctic penguins and Arctic polar bears.[18, 47, 63–67]

**In plants**. Soils are a major depository for plastic pollution, and agricultural ecosystems can thus be contaminated.[30, 31, 66, 67] Despite this potential pathway to a variety of harms, information on the distribution (and impacts) of MNP on plants remains scarce. A recent survey found only three studies on MNP in non-vascular plants, and 10 in vascular plants.[68] We know that nanoplastic particles have been found in plant cell walls, but microplastic appears to be far less present.[69, 70] We also know that MNP physically adsorb and accumulate on multiple algae species, with the attendant risk of bioaccumulation in the upper food web.[54]

**In humans**. Humans are exposed to plastics in a multitude of ways. The primary pathways are inhalation, ingestion, dermal contact, and, for infants, *in utero* exposure.[3] MNP and chemical additives from plastics have been detected in high enough concentrations in human tissue to pose serious questions about their presence in common household goods,[71] household dust,[72] personal care products,[36] food,[73] food packaging, drinking water,[33] and other beverages.[74] It is worth noting, however, that there is wide variability in density and concentration between pathways. For example, numerous studies have observed canned food as a primary pathway of human harm from BPA,[73] with concentrations as high as 0.027 mg/kg in some canned vegetables,[75] equating to more than 95 per cent of mean daily intake of BPA in adults of around 12 ng/kg-day.[76] However, non-food pathways via transdermal absorption should not be overlooked or discounted.[77–79]

## Plastic-related chemical complexity and toxicity

As outlined above, a diverse range of chemicals are often added to plastics during manufacture.[2] Current research has identified four plastic-related chemical groups as especially concerning based on their volume of use, persistence, and putative toxicity. These include bisphenols, phthalates, per- and polyfluoroalkyl substances (PFAS), and halogenated and phosphorus flame retardants.[3] All of these additives have been linked to a variety of harms, ranging from infertility and early puberty to metabolic disorders like type 2 diabetes and obesity.[80–84] The majority of these additives are endocrine disruptors. Given what is known about the central role that the endocrine system plays in the reproductive, developmental, and metabolic functions of the human body, endocrine disruption is a likely mechanism by which many harms occur.

Furthermore, because of plastics' hydrophobicity, their surface adsorbs chemicals and other agents from the surrounding environment.[85] Featuring a large surface area/volume ratio, MNP in particular can bond with large volumes of chemicals present in the environment in a process called adsorption. This can concentrate these chemicals at levels exceeding background by orders of magnitude.[85] Adsorbed chemicals can include heavy metals,[86] pharmaceuticals (if exposed to wastewater),[87] persistent organic pollutants,[88] and potentially endocrine-disrupting pollutants,[89] creating a potentially potent vector for a variety of harmful substances.

## 1.2 What are we seeking to do and how?

### 1.2.1 Objectives

In this Annex, we provide an overarching framework to categorise the expected social cost of plastic-related harms based on the scientific literature. To that end, our objectives are to:

- assess the strength of scientific consensus around specific harms being caused by plastic-related pollution; and
- provide an estimate of the size of the social cost from specific plastic-related harms;
- evaluate how this consensus (and size) might change in the future given the current trajectory of research.

### 1.2.2 Scope of data and analysis

#### Macroplastics and MNP

We included all hazards and harms from macroplastics and MNP that we could find **(see Appendix A1)**, encompassing both direct and indirect effects. Examples of direct effects include macroplastic ingestion leading to GI injury in marine animals or MNP contamination of treated water. Examples of indirect effects include air pollution from the production of macroplastics or the delivery of harmful pathogens via MNP.

#### Chemical additives

The 10,500+ chemicals added to plastics pose a particular challenge. Our initial scoping interviews with experts highlighted two issues for a subset of these chemicals:

- some are predominantly used in non-plastic production and applications
- some have not been produced and used for decades.

One of the best examples of this is PCBs. There are 209 individual chemicals (isomers) classed as PCBs.[90] Consensus around the harm they cause is well-known and long-established.[91] These include their probable carcinogenic effects in humans,[92] as well as their association with cardiovascular disease and diabetes.[82, 93]

However, PCBs were predominantly used in non-plastic applications such as capacitors, transformers, hydraulic fluids, heat transfer fluids, and lubricants.[94] Moreover, their use in plastics, as plasticisers and flame retardants, was stopped in the 1970s. Production was completely prohibited for all applications in the US in 1979, with similar bans elsewhere around the same time.[91]

As such, we excluded PCBs from our analysis. Instead, we focussed on flame retardants and plasticisers that we deemed more relevant to current and future plastic-specific social harm. This is not to say that PCBs are not a current cause of social harm; they persist in the environment and their use in legacy products is still widespread.[91] However, it is difficult to extract and quantify the contribution of plastics as a source of this persistent presence in the environment. We similarly excluded other prohibited dioxin and dioxin-like compounds for the same reasons. In contrast, we considered PFAS in scope for our analysis, given their continued use and manufacture, and available data,[95] but recognising their use being in predominantly non-plastic applications.

#### Carbon

GHG emissions across the plastics lifecycle were in scope. However, given the immensity of prior work on anthropogenic climate-related harms and their lack of plastic-specificity, the full gamut of harms for which carbon emissions and climate change potentially cause were not assessed individually. Instead they were consolidated using existing estimates of the social cost of carbon.

# 2. METHODS

**Our methodology followed three phases:**

- **Identify a long-list of emerging plastic-related harms to Human Health, to Economies & Ecosystem Services, and to Nature.**
- **Estimate the expected social cost of each harm (where possible).**
- **Present a categorisation of harms with common implications for society, the plastics industry, and other stakeholders.**

## 2.1 Identification of harms

**Goal:** identify a long-list of emerging plastic-related harms to human health, to economies and ecosystem services, and to nature.

**Steps**:

**1** **We gathered information on each of the sources of harm (chemical additives, etc.) and receptors (Human Health etc.), as described in Figure 2, at a granular level.**

- **Human Health**: we used data from a systematic algorithm-based review of the scientific literature (7,400 hazard-harm relationships studied in 5,420 studies indexed by PubMed; see Appendix A1),[b] including studies with in vitro, animal, and human subjects, and supplemented by expert interviews.
- **E&ES and Nature**: we performed a more limited review of key articles and reviews, again guided and supplemented by expert interviews.

**2** **We developed a long list of specific harms based on this information, which was characterised in terms of source, receptor, and specific injury/disease.**

[b]Collection and annotation of data by Praedicat Inc.

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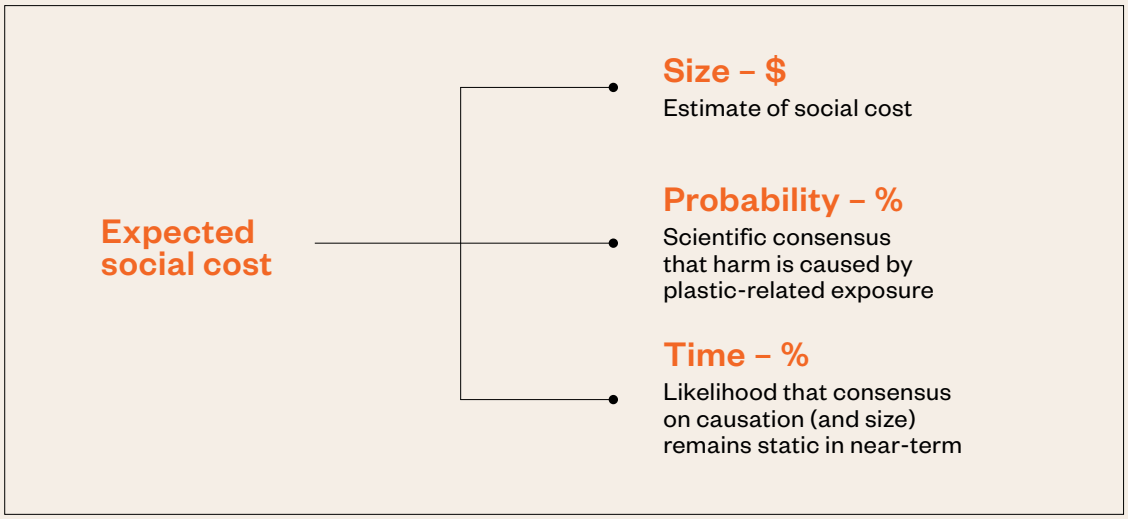
*Bales of green plastic bottles stacked at an undisclosed recycling facility circa 2009 in Toulouse. Photo credit: Shutterstock*

**Figure 5:** Overall assessment framework




## 2.2 Estimating the expected social cost of plastic-related harms

**Goal:** the expected social cost of any given harm might theoretically be calculated by taking the average across all possible magnitudes of harm over a defined time period, along with estimates of their social cost, weighted by their probability. For almost all the plastic-related harms identified,we found an absence in the literature of estimates of social cost (yet alone data needed to attempt a probability distribution for a range of harm sizes). As a result, we created a framework that uses proxies to assess the underlying principles of expected social cost – size, probability, and time (**Figure 5**).

**Steps:**

1. **Assess the size dimension: a single estimate of the social cost of each harm.**
2. **Assess the probability dimension: a view on the scientific consensus that the harm is caused through plastic-related exposure.**
3. **Assess the time dimension: a view on the likelihood of the current consensus remaining static in the near-term (and therefore whether the probability or size may change).**

## 2.2.1 Estimates of the social cost of plastic-related harms

### Human Health harms

**Following these steps, we:**

**1** **Extracted relevant data from the literature where possible (sources of data and insight provided in Appendix A1).**

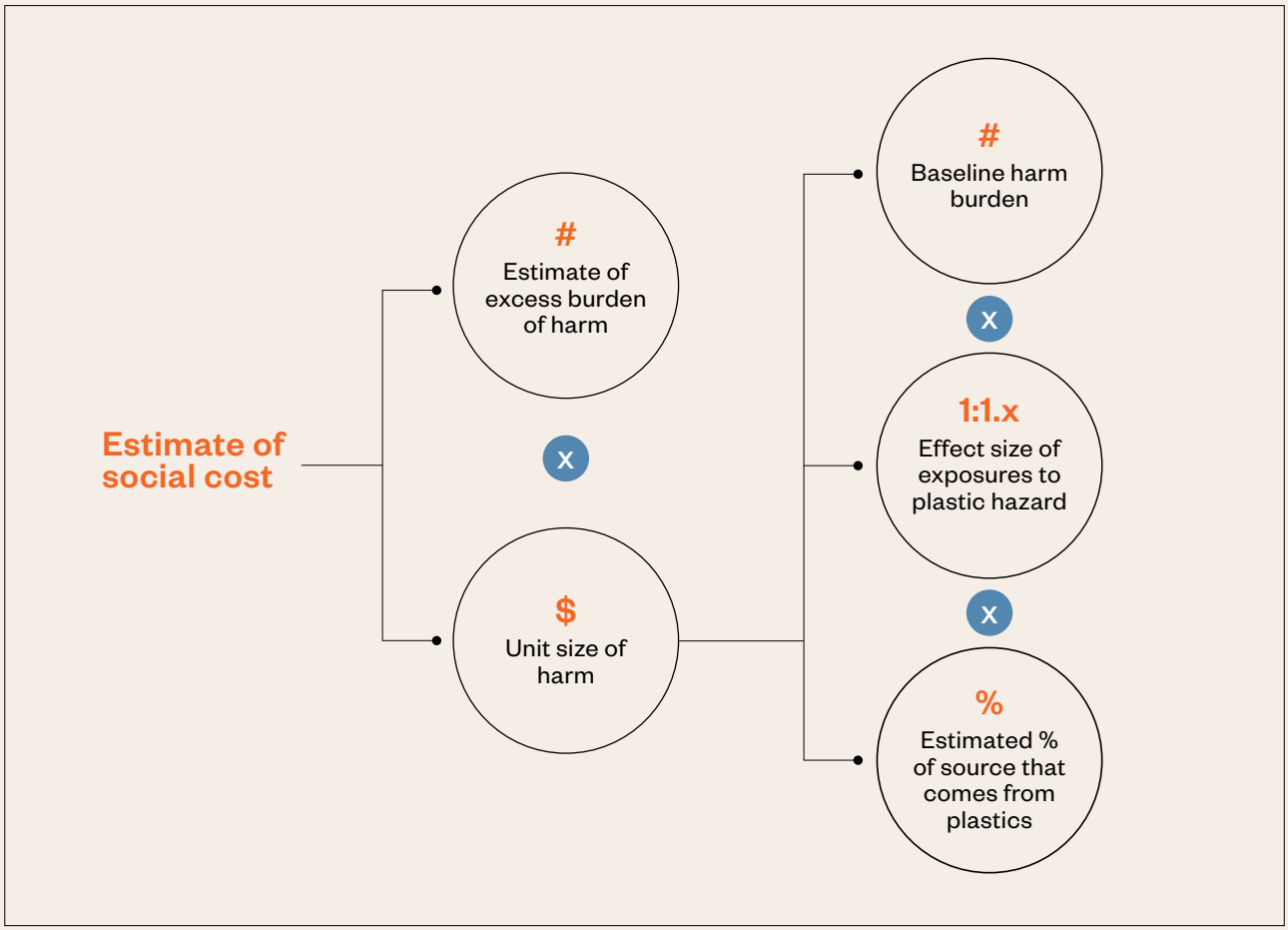
**2** **Calculated a current estimate of social cost for each harm (Figure 6) as a function of:**

- **Baseline harm burden**: we used this as the starting point before estimating excess burden caused by exposure to a plastic hazard. We assumed that the effects of plastic exposure were 'counted' within baseline burdens, given their ubiquity and decades-long use (see Appendix A3.1 for further details). With Human Health, burdens of a harm can be measured by taking the years of life lost to early death or disability from the disease caused by a harm: a disability-adjusted life year (DALY).
- **Summary of effect sizes on the general population**: we estimated the increased burden of harm attributable to each source (i.e., specific chemical additive, MNP, etc.) by extracting effect sizes from the literature. These are a measure of the association between exposure from a source and some health outcome. To account for the very different types of study populations and effect size types and to ensure we used effect sizes that were relevant to the general population (and not specific populations like factory workers or cancer patients), we did the following (see Appendix A3.1.2 for further details):
  - for those specific harms with at least one effect size from a general-population cohort study, we used that value. If a specific harm had more than one general-population cohort study and hence effect size, we took the average;
  - for specific harms with only effect sizes from more specific populations (e.g., workers, cancer patients etc.) or case-control studies, we can't definitively say how those results might translate to a general population across geographies (e.g., with different regulatory regimes and exposure levels) and different exposure routes. Therefore, in order to simulate this, we applied a discount factor by comparing effect sizes from general-population cohort studies to the effect sizes from all other studies across the entirety of the data to create a pooled value.
  - for harms that have been suggested in the literature but where an effect size on human health is yet to be determined, we made no estimate of social cost (e.g., MNP and lung injury).
- **Estimated percentage of source coming from plastics**: we included this to prevent overestimates of harm from non-plastic-specific chemical additives and to account for the potential confounding effect of plastics contributing to the baseline burden. We took these percentages from literature and expert interviews.
- **Unit size of harm (in USD)**: we used this to convert the burden of harm into financial costs. For Human Health harms, the cost of each DALY was set as the weighted global average from "willingness to pay" (WTP) surveys (US$15,700 per annum).[96]

**3** **We then transformed these USD figures into categorical values. Given the breadth of scope and, in many cases, limited research on social costs, we used a logarithmic scale for the thresholds, enabling directional comparisons between the sizes of different harms:**

- **Low**: ≤US$10 billion current estimate of social costs per year.
- **Medium**: US$10–100 billion current estimate of social costs per year.
- **High**: >US$100 billion current estimate of social costs per year.

**Figure 6:** Framework for estimate of the social cost of human health harms




## Economies and Ecosystem Services harms

For E&ES, we followed a similar logic: starting with an estimate of the total economic value of a particular service/activity (where, for Human Health harms, we had used estimates for the baseline burden of harm) then adjusting for the estimated economic impact of exposures to plastic-related hazards; and further adjusting for share of the source of the hazard that comes from plastic. Estimates were obtained from databases in peer-reviewed publications,[97] as well as credible industry and non-governmental reports. They are generally expressed in USD, so there was no need to convert the unit size of the harm.

## Nature harms

Nature's intrinsic or "heritage" value is inherently difficult to estimate, with methods ranging from "willingness to pay" (WTP) to "remedial costs". WTP estimates are low (less than US$10 billion per annum), while remediation costs are potentially so high as to be economically infeasible. Acknowledging that neither method provides for a satisfactory estimate, we have chosen the WTP estimate (also recognising that harms to nature's ecosystem services were addressed separately in this analysis – see section 3.4.3 on indeterminate harms). This assessment may well change as consensus on WTP evolves. We delve deeper into the challenges of sizing the social cost of harms to Nature in the Discussion, section 4.1.3.

### 2.2.2 Scientific consensus that a hazard from plastics causes a harm

We determined the probability of a hazard causing harm by assessing today's scientific consensus.

## Human Health harms

**Figure 7:** Framework for assessing consensus on causation

| | Score | | | | | Assesment | Sum of scores |
|---|---|---|---|---|---|---|---|
| | +0 | +1 | +2 | +3 | +4 | | |
| **Quality** Type and design of the highest scoring published study | *In vitro* experimental | Animal observational | Animal RCT/ meta analysis | Human observational cohort/ case control | Human RCT / meta-analysis | Low | [0, 4] |
| **Number** Net number of positive published studies[1,2] | < 10 studies ) 0 - 25th percentile) | - | [10, 100] studies (25th - 75th percentile) | - | ≥ 100 studies (75 - 100th percentile) | Medium | [4, 8] |
| **Relevance** Relevance of plastics as the potential pathway to harm [1,3] | No human studies, or human studies where plastics are not a pathway to harm | - | - | - | Human studies where plastics are a pathway to harm | High | [8, 12] |

1. Relevance and number are on the same scale as quality to ensure weighting
2. Number of results with effect sizes ≥1 net the number of studies with effect sizes <1
3. Plastic-related terms searched for in human-study. For macroplastics and MNP as the hazards, the maximum score of +4 will always be achieved

**To assess current consensus on causation, we:**

1. Devised an assessment **framework** (**Figure 7**). We used a scoring system rather than strict inclusion-exclusion rules. This enabled us to include and automate the assessment of ~7,800 hazard-harm relationships (derived from more than 5,500 studies indexed by PubMed[c]), while still achieving some degree of quality assurance:

- **Quality**: harms were assessed according to the design and subject of the studies
- **Volume**: harms were assessed using a logarithmic scale of the net number of studies showing a positive association between hazard and harm (number of positive associations less the number of negative associations found in studies; we did not account for null associations)
- **Relevance**: harms were assessed by ascertaining whether plastics were mentioned as a potential pathway to harm in the studies on them.

2. **Scored** the Quality, Volume, and Relevance subdimensions.

3. Summed them to give a **categorical** value (High, Medium, Low) for current consensus.

## E&ES and Nature harms

For E&ES and Nature, we qualitatively assessed harms in the literature on the same subdimensions of quality, volume, and relevance to plastic. We paid special attention to systematic reviews and meta-analyses. These were then used to inform a qualitative overall assessment of current scientific consensus.

[c]Collection and annotation of data by Praedicat Inc.

### 2.2.3 Likelihood that scientific consensus (and size) remains static

#### Human Health harms

**Figure 8:** Framework for assessing the likelihood of consensus on causation remaining static

| | Score | | | Assesment | Sum of scores |
|---|---|---|---|---|---|
| | +0 | +1 | +2 | | |
| **Stability** Latest change over 3 yrs in published study numbers[1] | ≥100% change between 2018-2021 High growth | [ 10%, 100%]change between 2018-2021 Moderate stable/ low growth | <10% change between 2018-2021 Highly stable | Low | [0, 1] |
| **Timeframe** Number of years since published studies began[2] | <5 years | [5, 20]years | ≥20 years | Medium | [2] |
| | | | | High | [3, 4] |

1. Change in the number of publications showing an association
2. Number of years since the first study showing an association published

**To assess future consensus about causation, we:**

1. Devised an assessment framework (**Figure 8**) with the following subdimensions, which we considered to be the best available measures of directionality in regard to future research:

- **Stability**: harms were assessed, on a logarithmic scale, on the change in the number of publications showing a positive association over three years. Little to no change (or even a decline) scored high on stability, meaning consensus is stable. A high growth rate scored low on stability, meaning consensus is likely to change.
- **Timeframe**: harms were assessed according to the length of time since first publication showing an association

2. **Scored** the Stability and Timeframe subdimensions
3. Summed them to give a **categorical** value (High, Medium, Low) for future consensus.

#### E&ES and Nature harms

- a non-exhaustive count of the number of studies on a harm published over the last three years to assess **stability**
- a Google Scholar search – using a harm-specific keyword search – of the earliest peer-reviewed research article (not a review) showing a positive hazard-harm association to assess **timeframe**

## 2.3 Grouping of harms

Where data were available, the specific harms were used to provide granular, comprehensive assessments.

Granular data were unavailable for many of the E&ES and Nature harms. Therefore, we grouped harms into broader categories (e.g., marine natural capital, which includes all ecosystem regulation, recovery, and resilience services) to carry out the assessment on the dimensions outlined above. Further, grouping specific Human Health harms into a larger group with common characteristics (e.g., a plastic-related chemical class, such as bisphenols) facilitated synthesis of the results.

Appendix A4 outlines the groupings alongside a justification for collapsing specific harms into these groups.

# 3. RESULTS

## 3.1 Identification of emerging harms from plastic pollution

Overall, we identified 92 specific harms in the literature (a full list with references for each is found in Appendix A2). These specific harms were grouped by receptor as follows:

- Human Health harms: we could be granular in collecting data (over 5,000 studies) and identifying human harms. The result was 48 specific harms.
- E&ES and Nature harms: we were less granular in collecting data (reviews, meta-analyses, expert interviews) to identify harms caused to terrestrial and aquatic organisms, and to the economy. The result was 22 specific E&ES harms and 22 Nature harms.

### 3.1.1 Human Health harms

Overall, there are 48 Human Health harms, comprising 31 harms from chemical additives, 9 harms from MNP, and 7 harms from macroplastics (Appendix A2).[d] **Figure 9** provides a visualisation of studies across the different sources and harms identified.

#### Chemical additives

Four classes of chemical additive account for the majority of the 1,254 chemicals of high concern that are not already banned.[2] A fifth class of other or unidentified plastic-related chemicals comprise the remainder without any reported hazard, along with unregistered classes that maybe incidentally picked up during production, use or disposal.

Phthalates are plasticisers, which are used to soften plastic. They are used in food containers, toys, plastic bags, blood bags and tubing, and vinyl flooring. They are also used in non-plastic applications. Phthalates have been shown to mimic oestrogen and induce inflammation. These phthalate-induced mechanisms have been linked to premature birth, lower testosterone levels, obesity, hypertension, diabetes, endometriosis, and changes in neurodevelopment.[81, 98, 99] Most phthalates have relatively short half-lives. As such, while they are organic pollutants, they are not listed as Persistent Organic Pollutants (POPs) under the Stockholm Convention.

Bisphenols are used to create hard plastics, such as polycarbonate, and the thermosetting

[d]Given the focus of this report is on liability risks specific to plastics, all climate-related impacts of CO2e emissions from across the lifecycle of plastics were grouped. While the plastics industry contributes significantly to global CO2e emissions, the study of climate-related liabilities is more advanced, and additional insight was determined to be beyond the bounds of this report.

••

*A picture taken on March 18, 2022 during the Global Recycling Day 2022 shows compressed waste at a recycling center in Merignac in the outskirts of Bordeaux, southwestern France. Photo credit: Philippe Lopez/Getty Images*

plastic, epoxy resin. They are found in products used for food, beverages, and general storage. Bisphenol exposure may occur occupationally during production, during product use (e.g., drinking bottles), and via wastewater in crops or aquatic ecosystems during disposal. The most produced and studied bisphenol is BPA. However, research suggests that other bisphenols "have many of the same adverse health effects" according to the European Chemicals Agency (ECHA).[100] BPA was initially developed as a synthetic oestrogen and bisphenols have been shown to mimic oestrogen and disrupt the endocrine system. BPA has also been linked to diabetes, reduced sperm quality and count, polycystic ovarian syndrome, obesity, cognitive deficits, and attention deficit hyperactivity disorder (ADHD).[98, 101]

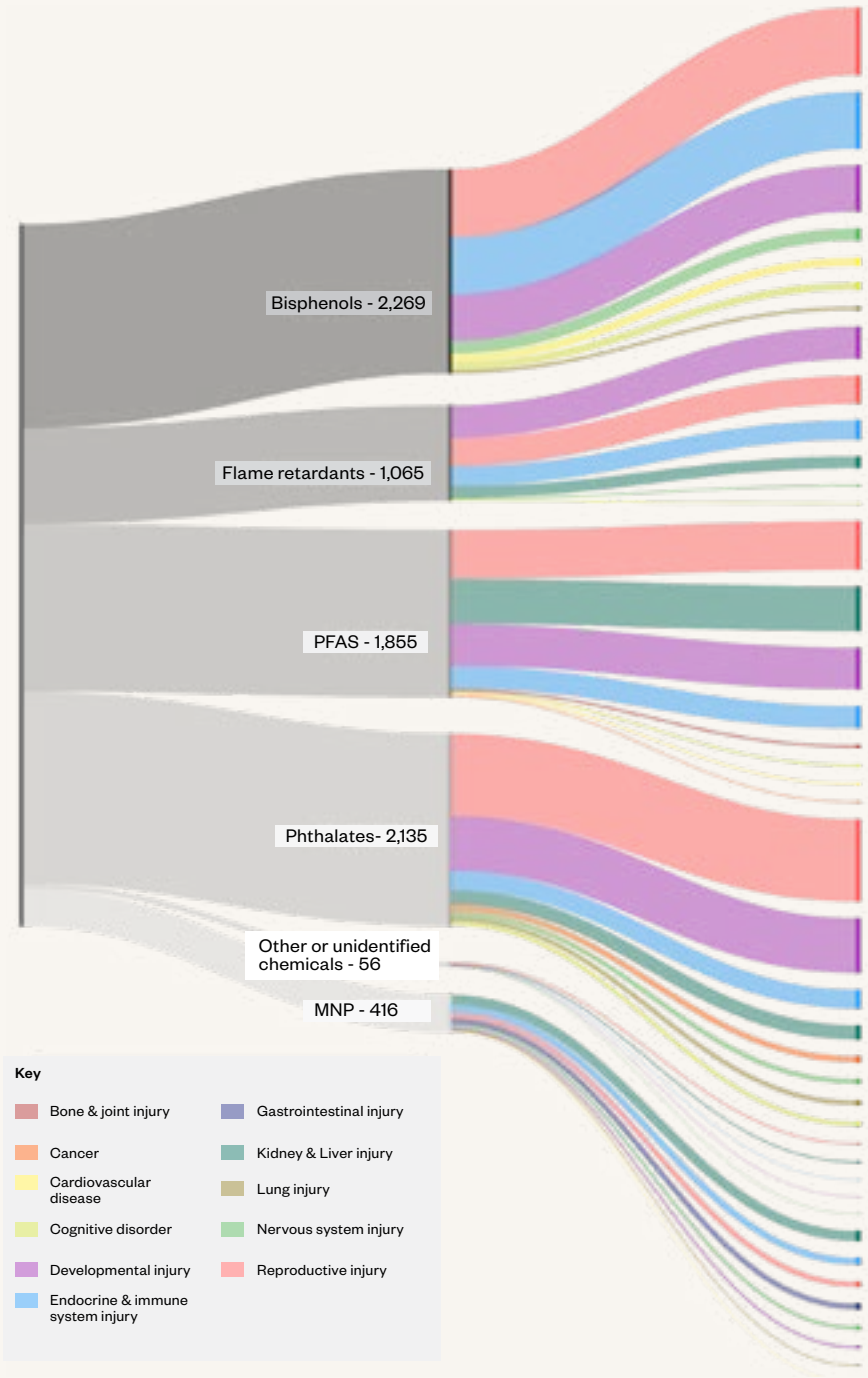
Flame retardants include several subgroups added to a variety of materials to reduce risk of fire.[102] They are used in plastics as well as textiles and electronics. Since they don't chemically bind to the products, they leach easily into air and dust during use and disposal. Some specific groups of halogenated flame retardants, such as the now phased-out polybrominated diphenyl ethers (PBDEs), have been shown to disrupt endocrine systems, and are linked to cognitive deficits, diabetes, cancer, autism spectrum disorder, and ADHD.[98] Concerningly, other halogenated flame retardants, such as tetrabromobisphenol A (TBBPA) and hexabromocyclododecane (HBCDD) seem to be hazardous as well.[103] Organophosphate flame retardants are increasingly being used to replace non-phosphate halogenated flame retardants.[104, 105] Recent findings demonstrate these have similar effects to organophosphate pesticides and insecticides: reproductive injury, endocrine disruption, carcinogenicity, neurotoxicity and developmental toxicity.[104] Many halogenated flame retardants are highly durable in organisms and the environment. Both HBCDCC and PBDEs are listed as POPs under the Stockholm Convention, and others like Dechlorane plus are under review.

PFAS comprises thousands of substances. They are used in a wide range of products due to their resistance to grease, oil, water, and heat. They may be split into two main types with very different properties: (a) fluoropolymers, and (b) non-polymers.[106] PFAS are not commonly added to plastics but can be created when polyethylene and polypropylene are exposed to fluorine gas, a common process to improve the oxygen and moisture migration qualities of plastic food packaging. PFAS are extremely persistent, with bioaccumulative properties. Non-polymeric PFAS are of most concern, since they can cross cell membranes.[106] Numerous studies have observed their toxic effects, including ADHD, cognitive deficits, diabetes, thyroid disease, hypertension, high cholesterol, obesity, and ulcerative colitis.[83] Due to their high durability and mobility, previously widely used PFAS – perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) – are listed as POPs under the Stockholm Convention.

Other/unidentified plastic-related chemicals encompass a diverse range of chemicals, including 4,000+ without any hazard classification,[2] with only a handful having undergone significant research. Examples include:

- diisononyl-cyclohexane-1,2-dicarboxylate (DINCH), which is a plasticiser and phthalate alternative used in toys and medical devices. DINCH has been shown to have some hazardous effects in animal studies, including oxidative damage.[107]
- chlorinated paraffins, which are used as plasticisers, flame retardants, and in packaging. Short-chain chlorinated paraffins (SCCPs) have shown carcinogenic effects in animal studies and are possible human carcinogens.[108]
- N-butylbenzenesulfonamide (NBBS) is a commonly-used plasticiser. NBBS lacks adequate toxicological data, with no studies on humans. Animal studies, however, have demonstrated toxicity, as well as structural similarities to known toxic chemicals.[109]

**Figure 9:** Number of publications relevant to Human Health by source and by harm




*Note: Human Health publications include all studies indexed by PubMed, comprising research conducted *in vitro*, on animals and on humans

Source: Praedicat, Inc

### Micro/nanoplastics (MNP)

**MNP may be categorised into:**

- **primary MNP**: created for a specific use (e.g., microbeads in cosmetic products) or shed during the natural usage pattern of a product (e.g., textile fibres or road tyre dust)
- **secondary MNP**: formed by the unintended degradation of larger pieces of plastic once they have entered the environment.

The high mobility and persistence of MNP make them ubiquitous, and pathways to harm are thought to be equally pervasive from ingestion of plastic teabag lining to inhalation of microfibres.[33, 112, 113]

Plastic itself is an inert material. Despite this, there are several pathways by which MNP cause harm to humans. These include induction of intracellular effects by nanoplastics, inflammatory and immune responses, mechanical injury by microplastics, and the delivery of incidental toxic contaminants.[113] In humans, there is increasing evidence that MNP cause digestive system harm[38] and lung injury.[114] For cirrhosis, it is unclear whether MNP induce chronic liver disease through inflammation or whether their presence in cirrhotic liver is a consequence of liver disease.[37]

### Macroplastics

The majority of identified macroplastic harms to Human Health stem from the release of pollutants to the environment during production and disposal. In the case of production-related emissions, these harms apply to all major industries. Disposal-related harms (principally from informal burning of plastic waste) are most prevalent in areas without formal waste collection. This leads to a disproportionate amount of harm caused in the poorer communities of low-income and lower middle-income countries.[5, 115]

## 3.1.2 Economies & Ecosystem Services harms

Overall, there are 22 harms to E&ES, comprising 9 from macroplastics, 7 from MNP and 2 from chemical additives (see Appendix A2). The literature does not support a distinction between chemical classes' harms on the economy, so we have split plastic-related chemical harms by receptor only.

### Chemical additives

Chemical additives have known toxic effects in aquatic and terrestrial organisms. However, the mechanisms by which plastic-related chemicals affect whole populations of food stocks (fisheries, livestock, etc.) are not well established. There are likely indirect means. For example, there is evidence linking plastic-related chemicals to reduced soil respiration and other harmful effects on the soil ecosystem.[66] However, the literature has not been able to separate the contributions of plastic-related chemicals from other potential causes, such as overcultvation.[116]

### MNP

In aquatic ecosystems, MNP pollution can create a physical barrier which blocks sunlight, as well as reducing the photosynthetic capacity and growth of plankton and algae.[6] This, in turn, threatens food and oxygen availability for other living species in aquatic environments. It can also have indirect climate impacts by reducing the ocean's carbon fixation capacity.[6] MNP in the form of pellets or nurdles can leak directly from ships and gather on recreational spaces such as beaches. Where such accumulation affects tourist sites, there is a potential negative impact on local economies.

In terrestrial ecosystems, MNP have been hypothesised to change soil characteristics such as structure, water holding capacity, density, temperature, and porosity, leading to reduced growth, reproductive success, seed germination, and root growth.[30, 65] MNP have also been hypothesised to harm microbial communities such as earthworms, which are crucial for soil health.[65]

Wastewater is a major pathway for contaminants entering the marine environment. It is estimated that more than 0.6 million tonnes of MNP is released into oceans from wastewater each year.[58] Furthermore, wastewater used as an input for sludge as crop fertilisers can contaminate agricultural land, and thus affect terrestrial food sources.

### Macroplastics

Harms from macroplastics on economic activity and ecosystem services are tightly linked to harms to Nature. Many communities are economically dependent on the functioning of specific ecosystems. When plastic reduces the functioning of those ecosystems by, for example, killing animals or preventing normal growth in offspring, these communities are harmed. Similarly, whenever value is either directly or indirectly gained from a place of natural beauty (e.g., tourism), it can be reduced by the presence of plastic in that area. Other economic harms include the time and cost expended by the fishing industry on repairing nets entangled with plastics and similar activities.[117]

## 3.1.3 Nature harms

Overall, there are 22 harms comprising 8 from MNP, 5 from macroplastics and 5 from chemical additives (see Appendix A2).

### Chemical additives

Chemical additives can leak into nature during production and disposal, or by migrating from plastic products as they leak into the environment. Chemicals cause endocrine disruptions in both humans and non-human animals. This disruption has been linked to changes in reproductive output, changes in neurodevelopment, delayed growth, and increased mortality.[6] Their known toxic effects and their leakage into nature have been found to have a deleterious effect on natural environments, harming both individual species and biodiversity.[118]

### MNP

Much of the work underpinning the evidence base for harms to Human Health is based on animal models. Therefore, there is a wealth of studies demonstrating that MNP cause a range of ailments in non-human animals following ingestion, including physical damage, oxidative stress, changes in gene expression, neurotoxicity, and inflammation.[6] Specific harms include mortality, non-lethal physical damage to animals, and reduced digestion of food, developmental harms, and reproductive injury.[6]

### Macroplastics

Macroplastic harms to Nature showcase the most visible adverse effects of plastic pollution and are perhaps the most deeply researched. Macroplastics affect animals when they are entangled in or ingest plastic waste. They can also affect plants and animals during the production process (raw material extraction and air pollution). Their harms can arise everywhere the raw materials for plastic are extracted and wherever plastic is produced, as well as in the areas where plastic is disposed. Some of the most overt examples of plastic harm include floating oceanic plastic debris (such as is found in the North Pacific gyre), litter along beaches, and ingestion of macroplastics by sea birds.[6,61] More than 700 species of bird are known to be affected by plastic in its various forms.[51]

## 3.1.4 Carbon emissions and climate change

Plastics release greenhouse gases at almost every phase of their lifecycle. Production results in 2.3 kg $CO_2$e released per kg and up to 2.7 kg when plastic is incinerated.[11] Total plastic-related emissions from disposal amount to almost 100 million tonnes per year.[119]

Carbon emissions lead to climate change, which in turn leads to a wide variety of harms, including rising sea levels, drought, floods, ocean acidification, and food and water insecurity. These harms are not unique to plastics. Noting the wide array of specific harms to all three receptors, we have grouped many of the specific climate change harms from production and under one group: harm from carbon emissions and climate change.

## 3.2 Expected social costs of plastic pollution

### 3.2.1 Human Health harms

For Human Health, we assessed the expected social cost for each of the 48 specific harms. These results are discussed below.

#### Estimated social cost of Human Health harms

The majority of social costs come from Human Health harms. Summing the costs of all the harms without any weighting by probability yields a theoretical social cost running into the hundreds of billions of dollars.

**The drivers of the high Human Health costs are three-fold:**

(a) the prevalence and severity of plastic-related diseases (leading to large excess DALY burdens even where effect sizes are modest)

(b) the high price that society assigns to averting a single DALY

(c) the plastic contribution to the source of harm.

#### (a) Prevalence and severity of plastic-related diseases

In **Table 1**, we show that 12 of the 48 (25 per cent) individual Human Health harms exceed 1 million DALYs per annum globally. For two of the identified harms caused by bisphenols (cardiovascular disease, and endocrine and immune disorders), two by phthalates (lung injury, and endocrine and immune disorders), plus particulate-matter release during the production of macroplastics, the DALY burden exceeds 3 million DALYs.

These large DALYs underline the high prevalence of these diseases as well as the deep impact they can have on quality of life and mortality. As a result, even relatively modest effect sizes (which have been measured on or adjusted for a general population) attributable to plastic-related hazards lead to a high excess DALY burden.

However, underlining the importance of baseline burden as a driver of societal harm is the fact that specific harms to reproductive health had high effect sizes for all chemical additives, but only small excess burdens in absolute terms. This is because infertility – while a very deep concern for those affected – has a low baseline DALY burden in comparison to diseases such as diabetes or cardiovascular disease.

#### (b) DALY cost

One million DALYs amounts to just over US$15 billion per annum in costs while 5 million DALYs is just under US$80 billion in costs. Underlying these high costs is the US$15,700 per annum that societies are willing to pay to avert a single year of disability or life lost. This number is an average weighted by the global distribution of DALY burden. As the wealth and health expectations of lower-middle income and middle-income countries rises, the per annum cost per DALY may well increase.

#### (c) Plastic contribution to source of harm

Human Health harms are dominated by harms that not only generate large DALY burdens but are also predominantly caused by a plastic pathway. As an example, MNP are 100 per cent sourced from plastics. Therefore, if the potential gastrointestinal injuries (amounting to 1.5 million excess DALYs) are directly caused by MNP particles, they are purely attributable to plastics. This contrasts with PFAS. Despite a large potential excess DALY burden (36 million unadjusted), only around 1 per cent is contributed by plastics, resulting in ~360,000 DALYs (**Table 1**). This puts PFAS via plastics into the low harm category (<$US10 billion per annum).

**Table 1:** Estimate of excess burden and social cost of Human Health Harms

| Specific harm | Current estimate estimate of excess burden of harm/ (milions DALYs) | Current estimate of social cost / (billion USD) | Assessment |
|---|---|---|---|
| Bisphenol from plastics leading to **cardiovascular disease** | 4.9 | 76 | Medium |
| Bisphenol from plastics leading to **cognitive disorder** | 0.3 | 5 | Low |
| Bisphenol from plastics leading to **developmental injury** | 1.5 | 23 | Medium |
| Bisphenol from plastics leading to **endocrine & immune system injury** | 4.8 | 75 | Medium |
| Bisphenol from plastics leading to **lung injury** | 2.2 | 34 | Medium |
| Bisphenol from plastics leading to **nervous system injury** | 0.1 | 2 | Low |
| Bisphenol from plastics leading to **reproductive injury** | 0.3 | 4 | Low |
| Flame retardant from plastics leading to **cognitive disorder** | 0.1 | 1 | Low |
| Flame retardant from plastics leading to **developmental injury** | 1.7 | 26 | Medium |
| Flame retardant from plastics leading to **endocrine & immune system injury** | 3.0 | 47 | Medium |
| Flame retardant from plastics leading to **kidney & liver injury** | 0.5 | 7 | Low |
| Flame retardant from plastics leading to **nervous system injury** | 0.0 | 1 | Low |
| Flame retardant from plastics leading to **reproductive injury** | 0.3 | 5 | Low |
| PFAS from plastics leading to **bone joint injury** | 0.05 | 0.8 | Low |
| PFAS from plastics leading to **cardiovascular disease** | 0.114 | 1.8 | Low |
| PFAS from plastics leading to **cognitive disorder** | 0.00 | 0.00 | Low |
| PFAS from plastics leading to **developmental injury** | 0.03 | 0.5 | Low |
| PFAS from plastics leading to **endocrine & immune system injury** | 0.1 | 1.5 | Low |
| PFAS from plastics leading to **kidney & liver injury** | 0.06 | 1.0 | Low |
| PFAS from plastics leading to **reproductive injury** | 0.002 | 0.003 | Low |
| Phthalate from plastics leading to **cognitive disorder** | 1.0 | 16 | Medium |
| Phthalate from plastics leading to **developmental injury** | 2.3 | 36 | Medium |
| Phthalate from plastics leading to **endocrine & immune system injury** | 3.7 | 58 | Medium |
| Phthalate from plastics leading to **kidney & liver injury** | 0.3 | 4 | Low |
| Phthalate from plastics leading to **lung injury** | 3.3 | 52 | Medium |
| Phthalate from plastics leading to **nervous system injury** | 0.1 | 2 | Low |
| Phthalate from plastics leading to **reproductive injury** | 0.1 | 2 | Low |
| Unidentified or other chemicals from plastics leading to **endocrine & immune system injury** | 2.2 | 34 | Medium |
| MNP leading to **gastrointestinal injury** | 1.5 | 23 | Medium |
| MNP indirect delivery of other harmful agents | 0.3 | 5 | Low |
| End-of-life burning of macroplastics leading to harm[3] | 0.7 | 11 | Medium |
| Production of macroplastics leading to harm[b] | 3.5 | 54 | Medium |

Estimates of excess burden were calculated using effect sizes from studies on human subjects only. This means that results for only 32 of the 48 specific human-health harms (43 excluding those consolidated as in footnotes a. and b. below) are shown.

a. 'End-of-life burning of macroplastics leading harm' is consolidated from five specific harms: end-of-life burning of macroplastics leading to cancer; cardiovascular disease; endocrine & immune system injury; lung injury; nervous system injury

b. 'Production of macroplastics leading to harm' is consolidated from two specific harms: production of macroplastics leading to PM2.5 release; production of macroplastics leading to environmental contamination.

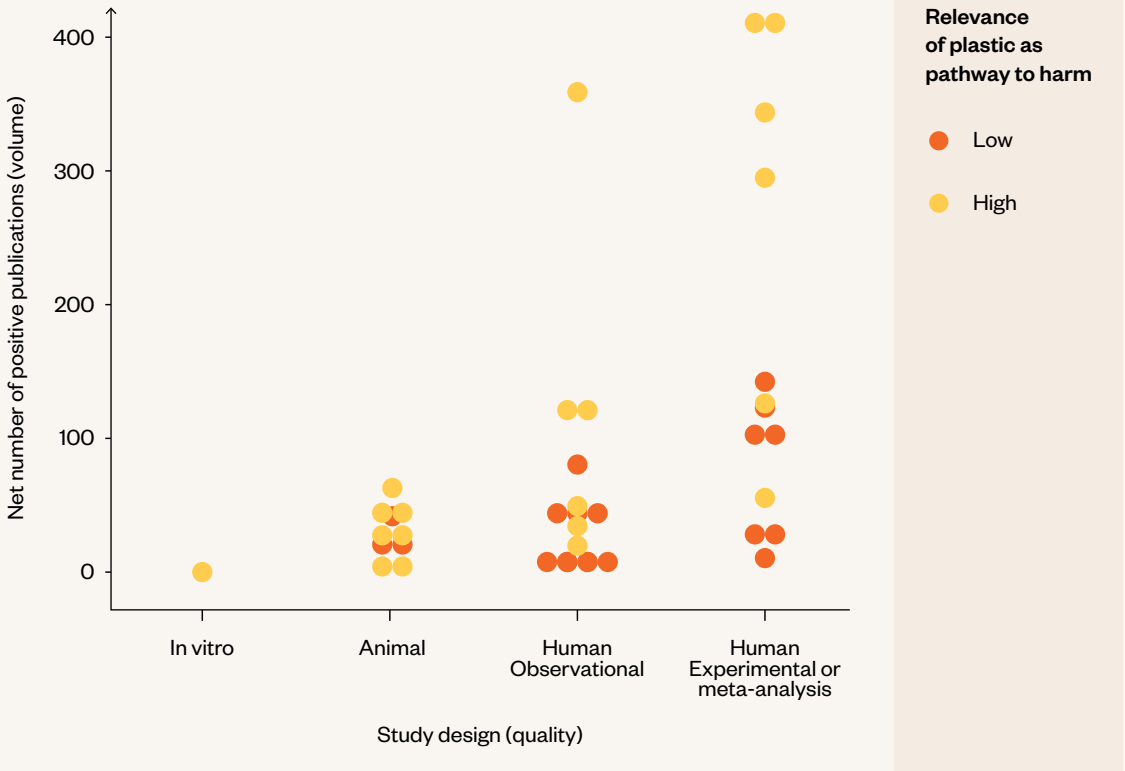
## Consensus on causation for Human Health harms

Human Health harms have variable levels of consensus. High-quality, plastic-relevant human studies cause certain plastic-related chemicals (bisphenols, phthalate, and flame retardants) and macroplastics to score high. By contrast, PFAS scores medium due to a lack of evidence demonstrating plastic as a viable, real-world route to harm. Despite high prevalence, direct effects from MNP also score medium because there is just one human study. Both indirect MNP harms (where MNP act as an incidental vector for other contaminants) and other/unidentified plastic-related chemicals score low due to a lack of evidence.

Unsurprisingly, we found the highest quality study for a given harm correlates with the volume of positive-association publications on harm causation (**Figure 10**). For example, bisphenol and phthalate Human Health harms have the greatest number of net-positive studies on average as well as the highest quality due to at least one meta-analysis. This makes sense: as more studies are conducted into a topic, more studies are available for meta-analyses.

There are exceptions. For example, there are several human observational studies examining whether PFAS causes high cholesterol (medium-high quality).[120] However, the net number of positive studies is zero (number of positive associations less the number of negative associations found in studies). As such, volume and quality diverge (although this may be due to publication bias). We found plastic relevance to be uncorrelated with quality and volume (**Figure 10**). The prime example of this is MNP, which have fewer studies of lower quality in general. However, plastic as the pathway to MNP harm is relevant by definition.

**Figure 10:** Correlation between study quality and volume for human health harms



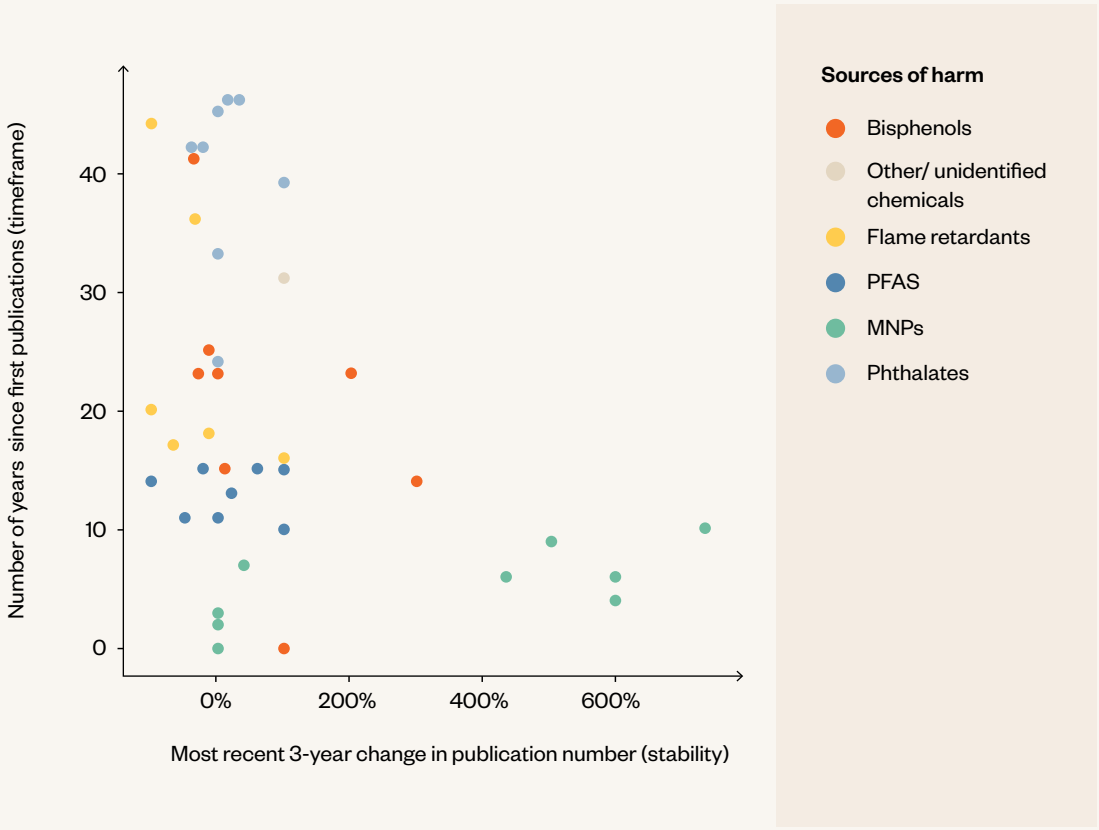
## Likelihood of consensus on causation remaining static in the near-term for Human Health harms

Human Health harms vary from low likelihood of causation consensus remaining static (MNP harms) to relative stability in consensus (harms from macroplastics and chemical additives). This is driven by the relatively recent explosion of interest in MNP as direct mediators of harm and as incidental vectors of other contaminants in human-related studies using *in vitro*, animal, and human subjects.

We found the latest three-year percentage change in publication numbers (stability) to be inversely correlated with the numbers of years since published studies first appeared (timeframe). In other words, as the number of years since first publication increased, the greater the recent "stability" in publication numbers (**Figure 11**). For example, endocrine and immune system injury from bisphenol has been studied for over 25 years and has a -13 per cent last three-year change in publication numbers (in decline/stable). By contrast, MNP as a direct cause of kidney and liver injury has only been studied in the past five years and has undergone a more than 500 per cent change in publication numbers (growth) across *in vitro*, human, and animal subjects. This makes sense: harms that have been studied for a long time are less likely to garner an explosion in interest.

There are exceptions. For example, there are several MNP harms that have only recently been studied. However, they have yet to receive the same level of interest as other MNP harms. This may be a result of latency.

**Figure 11:** Inverse correlation between stability and timeframe



## Grouping Human Health harms

As discussed in the Methods, given the large number of individual Human Health harms, we grouped harms by common source (e.g., plastic-related chemical class) to facilitate the synthesis and visualisation of the results (see section 3.4 below).

For estimated social cost, we summed the estimates for each individual harm into a total estimate for the group. We then re-mapped these aggregate costs to the low-medium-high size thresholds. For consensus on causation and likelihood of consensus remaining static in the near-term, we took the average of the results for the individual harms. Despite a handful of exceptions, the relative consistency on consensus for a given source supported the value of rolling up the specific Human Health harms into grouped harms.

### 3.2.2 Harms to Economies & Ecosystem services and Nature

For E&ES, Nature, and Climate, we performed the assessment of expected societal cost at a broader level, where harms with common characteristics were grouped (e.g., plastic-related chemical harms to fish stocks, soil degradation, and livestock were grouped into plastic-related chemical harms to food sources). This less systematic, higher-level approach was adopted for these non-Human Health harms due to the limited availability of comprehensive, granular data to analyse within the time constraints of the research. The results are discussed below.

## Estimated social cost of harms to E&ES and Nature

E&ES harms are driven by those that have large baseline economic costs (baseline burden) and are predominantly caused by a plastic pathway (plastics contribution). Contamination of treated water from MNP (discussed below) stands out on both counts (**Table 2**).

Nature harms are uniformly low. This is driven by our assessment of their low baseline burden of harm (**Table 2**). In the Discussion, we describe the issues surrounding our conservative approach to this, the result, and its implications.

**Table 2**: Estimate of social cost for E&ES and Nature harm

| Receptor | Source | Grouped harm | **Baseline burden** Baseline size of harm upon which plastic acts | **Effect size** Size of effect plastic harms may have on baseline burden | **Plastics contribution** Estimated % of source coming from plastics | **Current estimate of social cost** |
|---|---|---|---|---|---|---|
| Economies & Ecosystem Services | Chemical additives | Harm to food sources from chemical additives | High | Low | Low | Low |
| | Macroplastics | Harm to food sources from macroplastics | High | Low | High | Low |
| | | Harm to tourism from macroplastics | High | Low | High | Low |
| | MNP | Harm to food sources from MNP (direct) | High | Medium | High | Medium |
| | | Harm to water sanitation from MNP (direct) | High | High | High | High |
| | | Harm to food sources from MNP (indirect) | High | Low | High | Low |
| | All plastics | Harm to marine natural capital from all plastics | High | Medium | High | High |
| Nature | Chemical additives | Harm to organisms from chemical additives | Low | High | Low | Low |
| | Macroplastics | Harm to organisms from macroplastics | Low | High | High | Low |
| | MNP | Harm to organisms from MNP | Low | High | High | Low |
| All | $CO_2e$ emissions | Carbon emissions and climate change | High | High | Low | High |

## Consensus on causation for harms to E&ES and Nature

Harms to E&ES are the least well studied. While there is a wealth of evidence showing that plastics cause harm to animals and plants, there is far less robust evidence demonstrating second-order economic impacts (**Table 3**).

Harms to Nature are generally well established. This is driven by both quality and net volume of positive studies scoring high (**Table 3**). These in turn are driven by the high number of controlled, well-powered experiments demonstrating harm in animal models. Such harms to Nature (especially for MNP, see above) can be viewed as leading indicators for Human Health, as well as for E&ES harms.

**Table 3**: Scientific consensus that harm is caused by plastic-related exposure for each grouped harm

| | | | Consensus on causation | | | |
|---|---|---|---|---|---|---|
| **Receptor** | **Source** | **Grouped harm** | **Quality** Type & design of the highest scoring published study | **Volume** Net number of positive published studies | **Relevance** Relevance of plastics as an established pathway to harm | **Total** Sum of scores |
| E&ES | Chemical additives | Harm to food sources from chemical additives | Medium | Medium | Low | Medium |
| | Macroplastics | Harm to food sources from macroplastics | Medium | Medium | High | Medium |
| | | Harm to tourism from macroplastics | Low | Low | High | Low |
| | MNP | Harm to food sources from MNP (direct) | Medium | Medium | High | Medium |
| | | Harm to water sanitation from MNP (direct) | Medium | Medium | High | Medium |
| | | Harm to food sources from MNP (indirect) | Low | Low | High | Low |
| | All plastics | Harm to marine natural capital from all plastics | Low | Low | Medium | Low |
| Nature | Chemical additives | Harm to organisms from chemical additives | High | High | Medium | High |
| | Macroplastics | Harm to organisms from macroplastics | High | High | High | High |
| | MNP | Harm to organisms from MNP | High | High | High | High |
| All | $CO_2e$ emissions | Carbon emissions and climate change | High | High | Medium | High |

## Likelihood of consensus on causation remaining static in the near-term for E&ES and Nature harms

Harms to E&ES show signs of a potential rapid rise in research interest across plastic sources of harm. As ever, the difficulty will be in translating investigations into first order impacts on marine animals and plants, soil, recreational areas, and other fundamental units of an ecosystem into second-order impacts where there is tangible economic loss. However, there are relatively recent attempts to do this (albeit qualitatively)[121] which are being combined with efforts to collect more robust datasets.[122]

As discussed, harms to Nature are generally well established and hence unlikely to change (**Table 4**).

**Table 4**: Likelihood that consensus on causation (and size) remains static for E&ES and Nature harm

| Receptor | Source | Grouped harm | Likelihood of consensus remaining static | | Total Sum of scores |
|---|---|---|---|---|---|
| | | | **Stability** Latest change over 3 years in published study numbers | **Timeframe** Number of years since published studies began | |
| E&ES | Chemical additives | Harm to food sources from chemical additives | Medium | Medium | Medium |
| | Macroplastics | Harm to food sources from macroplastics | Low | Medium | Low |
| | | Harm to tourism from macroplastics | High | Low | Medium |
| | MNP | Harm to food sources from MNP (direct) | Low | Medium | Low |
| | | Harm to water sanitation from MNP (direct) | Low | High | Low |
| | | Harm to food sources from MNP (indirect) | Low | Low | Low |
| | All plastics | Harm to marine natural capital from all plastics | Medium | Low | Medium |
| Nature | Chemical additives | Harm to organisms from chemical additives | Medium | High | High |
| | Macroplastics | Harm to organisms from macroplastics | High | High | High |
| | MNP | Harm to organisms from MNP | Medium | Medium | Medium |
| All | $CO_2e$ emissions | Carbon emissions and climate change | Medium | High | High |

## 3.3 Comparison across sources of harm

### 3.3.1 Size of social cost

Across sources, chemical additives contribute the most potential harm. Two of the seven (29 per cent) grouped chemical additive harms – phthalates and bisphenols – are estimated to cost society >US$100 billion per annum globally. As noted above, chemical additive harms are mostly driven by the effects on Human Health across chemical classes. Chemical additive harms on E&ES are considerably lower. Despite the well-documented effects of chemical additives on animals and plants, the translation of this into second-order economic losses is dwarfed by other factors. For example, economic losses to the terrestrial food industry can be primarily attributed to overgrazing and deforestation.[123] Likewise, much of the economic losses in the aquatic food industry are attributable to overfishing, bycatch, and spoilage during transportation.[124]

Macroplastic harms are uniformly low by comparison. Only Human Health harms from macroplastics exceed US$10 billion per annum globally. The DALYs associated with respiratory and cardiovascular disease from particular-matter air pollution (PM2.5) have been estimated at around 210 million.[97] Therefore, for air pollution during production, plastics' estimated contribution of less than 2 per cent to PM2.5s still results in ~3.5 million of plastic-attributable DALYs (just over US$50 billion per annum). It should be noted that the costs of plastic waste management are not included in the scope of our analysis, which only considers the social costs of plastic-related pollution (i.e., mismanaged waste).

MNP harms, by contrast, are dominated by the medium and large potential harms to food sources and Human Health, and water sanitation, respectively. With respect to water sanitation, we have estimated the size of the social cost attributable to MNP based on the cost of remediation. Water remediation costs for MNP are estimated at US$200–300 billion per annum globally[125] and we have attributed the full cost of water remediation to MNP regardless of whether or not other sources of water pollution are present.[18]

Carbon emission-induced climate change that is attributable to plastic production and disposal is around four per cent of total GHG emissions (1.8 billion CO2e tonnes per annum out of just under 50 billion $CO_2e$ tonnes[20] per annum).[9, 12] The large estimated unit social cost of carbon (US$100 per tonne),[4] means that the current estimated social cost attributable to plastics is greater than US$100 billion per annum globally.

### 3.3.2 Consensus on causation

Chemical additives as a cause of harm have either medium or high consensus. This is driven largely by the attention that individual chemical classes have received in the scientific community with respect to Human Health harms. This, in turn, has resulted in numerous studies, some including human subjects, which score highly for Quality.

We found a reasonable degree of consistency for causation consensus within plastic-related chemical classes. As examples, over two-thirds of the specific harms for PFAS and MNP are categorised as medium consensus on causation (see Methods 2.2.2). Similarly, over 60 per cent of specific harms from bisphenols and phthalates have a high consensus on causation. However, there is some heterogeneity. For halogenated flame retardants, we found that liver injury scores low despite all other harms scoring medium or high. While only animal experiments have demonstrated that PDBEs and other brominated flame retardants can cause liver pathology,[126–128] other flame retardant-linked harms, such as developmental injury, have human evidence.[129, 130] On the other end of the spectrum, GI injury, which scored high, was the exception to the medium/low scoring harms for MNP. This was a result of one study on human subjects in which the severity of inflammatory bowel disease was found to be correlated with the faecal concentration of microplastics.[38]

Macroplastics' consensus ranges from high to low. While all these harms score high for relevance, only harms to Human Health and to Nature have been studied well enough to push their consensus to high. Examination of second-order effects on economies has been comparatively less well studied. In particular, robust evidence of an impact on tourism is particularly lacking, despite anecdotal reports.

MNP follow a similar pattern to macroplastics. Of special note are the indirect harms from MNP. Despite a lot of linked evidence showing MNP can adsorb a range of contaminants to their surface,[85] and such contaminants have been shown to cause harm when artificially mixed with MNP,[131] there is no direct evidence that MNP cause harm in the real world as incidental vectors of other agents.

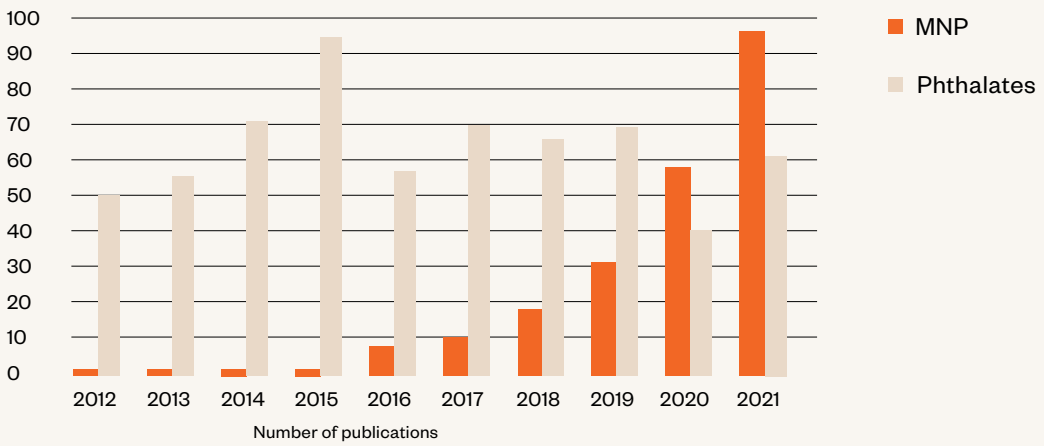
### 3.3.3 Likelihood that consensus (and size) remain static in the near-term

Chemical additives: results are primarily driven by receptor. Consensus that plastic-related chemicals cause first-order impacts on Human Health and Nature are likely to remain static given the wealth of data to date. Halogenated flame retardants, PFAS and phthalates are all relatively stable, with a body of at least 10 years of research and stagnant/declining publication rates. Within these three classes, each of the specific harms broadly conforms with this finding. By contrast, Human Health harms from bisphenols exhibit a range of different study durations despite a uniformly low change in publication rate.

Other/unidentified plastic-related chemicals stand out. Despite low consensus on causation due to the low volume of studies on chemicals outside the main classes identified, there is only weak evidence to suggest that this will change in the near term. There is likely a lot of heterogeneity around plastic specificity (for example, unidentified plastic-specific chemicals versus SCCPs, which are used in many different applications) and levels of regulation (SCCPs are heavily regulated versus DINCH, which is not).[107, 132] For highly regulated, non-plastic-specific sources that are already known to cause harm independent of plastic-specific pathways, there is unlikely to be any impetus to discover more about the contribution of plastic-specific uses to the harm they cause. Likewise, for less-regulated sources – which are either known to be plastic-specific, or for which the contribution of plastics as a pathway to harm is less clear – there are few studies being conducted on cataloguing their harms, especially for any unidentified chemicals and the 4,000+ chemicals without any hazard classification.[2] This heterogeneity, together with the high number, drives our assessment of this category being static in terms of whether future consensus is likely to change.

Macroplastics' likelihood for causation consensus remaining static varies with receptor in a similar fashion to chemical additives. MNP results also vary according to receptor but with a much wider gap between harms to Nature and to humans. This underscores the recent interest in this plastic source of harm. As discussed, MNP are relatively recent topics of research interest, which contrasts sharply with chemical additives (**Figure 12**: with phthalates as an illustrative chemical additive[e]).

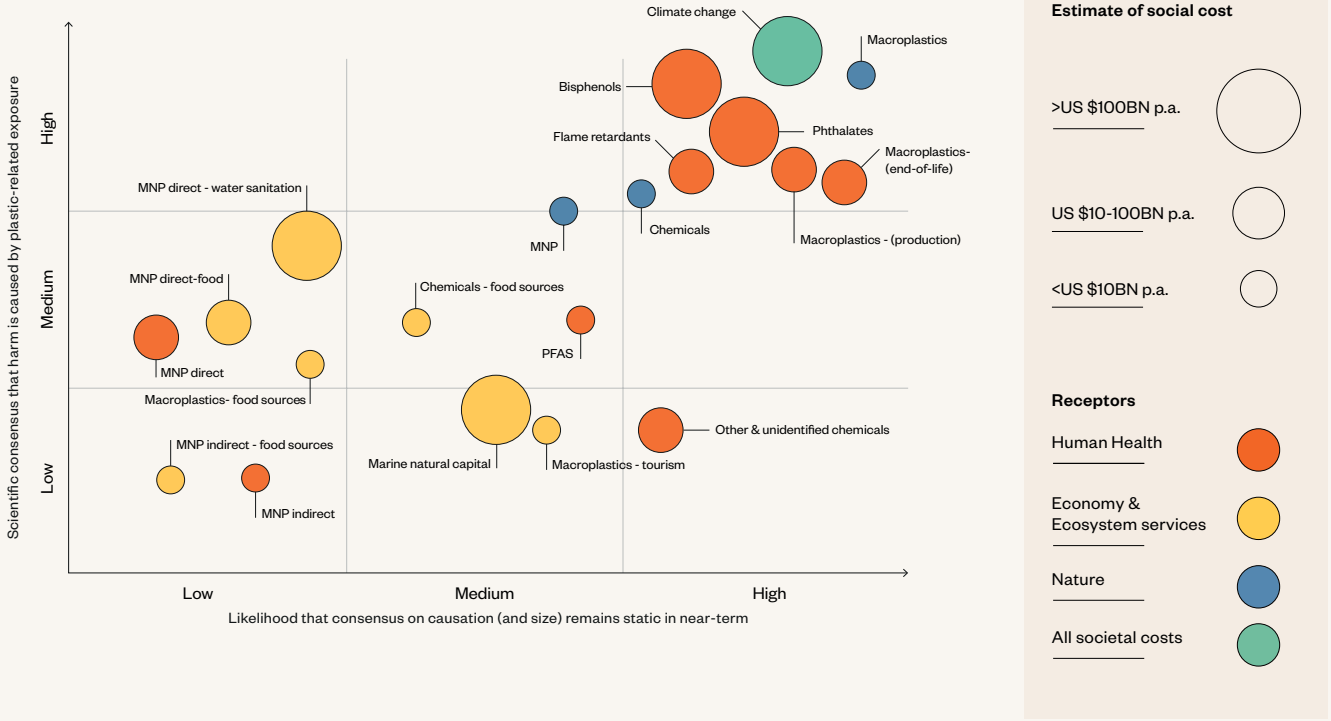
**Figure 12**: Number of positive-association publications related to Human Health per year for MNP and phthalates




[e]Publications related to Human Health include all publications indexed by PubMed, and include research conducted *in vitro*, on animals and on humans.

## 3.4 Four clusters of plastic-related harms

**Figure 13**: Estimate of social cost, and current and future consensus on causation




We plotted the results of the 20 grouped harms (Methods 2.3) (**Figure 13**) to uncover clusters and develop insights and implications (for the plastic industry, its shareholders and insurers, regulators and policymakers, civil society groups and scientific researchers). Based on the probability of harm (consensus on causation) and trend pathway of knowledge (likelihood that consensus remains static), we identified four clusters of harms.

### 3.4.1 Known harms

In the top-right corner are the known harms, which are defined by a well-known and relatively long-established scientific consensus on causation.

**Human Health harms** include chemical additives that have received a great deal of scientific attention: phthalates, phosphate-based and halogenated flame retardants, and bisphenols. Bisphenols and phthalates have current estimated social costs in excess of US$100 billion per annum globally.

**Harms to Nature** from the three main types of sources (plastic-related chemicals, macroplastics, and MNP) are also in this cluster. This visually illustrates the role of Nature harms as a leading indicator for certain Human Health and ecosystem service harms (for MNP and macroplastics). However, as described in the Methods and Discussion, the current estimated social cost of these is <US$10 billion per annum.

Finally, **climate change-related harms** fall into this cluster given the well-established effects of carbon emissions on climate change, and of climate change on a variety of harms to Human Health, E&ES, and Nature. Current estimated social cost is >$100 billion per annum globally.

### 3.4.2 Emerging harms

Below and to the left of the known harms are a cluster of five harms. These harms have a moderate degree of consensus on causation and are experiencing rapid growth in scientific support. The harms in this cluster have been the subject of study in the past decade or so.

The lone **Human Health harm** is the direct impact of MNP as mediators of inflammation and mechanical damage. The current estimated social cost of the harm exceeds US$10 billion per annum globally. **E&ES** harms comprise two MNP-related harms: one to food sources and the other to water sanitation, the latter entailing current estimated social costs of >US$100 billion per annum globally based on remediation costs to remove nano-sized plastic particles.

### 3.4.3 Indeterminate harms

To the bottom right and middle of the chart are four harms defined by a relatively stable but incomplete scientific understanding of causation. The consensus on causation seems unlikely to change in the near future due to a recent decline/stagnation in publications. Arguably, these harms should be an active area of research given that there is already moderate evidence of harm causation.

**Human Health harms** include those from other/unidentified plastic-related chemicals and PFAS, which are both low consensus due to the lack of evidence demonstrating plastics as the route to causing harm. Dominating the **E&ES** part of this cluster is the large harm (>US$100 billion per annum globally) to marine natural capital.

### 3.4.4 Immature harms

In the bottom-left corner, a cluster is defined by low consensus around causation but exhibiting signals of change in the near term. Both harms in this cluster relate to the indirect effects of MNP acting as incidental vectors for other contaminants (heavy metals, pharmaceuticals, etc.). Both are <US$10 billion per annum in current estimated social cost.

# 4. DISCUSSION

## 4.1. Implications of plastic pollution costs for society

We identified several potential sources of harm (five groups of chemical additives, macroplastics, MNP and carbon emissions). These sources of harm originate at multiple points in the plastics lifecycle, from production and consumption to disposal.[9] They impact several receptors: Human Health, E&ES, and Nature.[9]

Putting together the combinations of sources, receptors, and specific health and economic impacts results in 92 specific harms. The sources of these harms are pervasive, the hazards are numerous, and our exposure to them is inevitable. We attempted to comprehensively assess each of these specific harms, but the state of our knowledge varies widely. These specific harms are almost certain to grow in number, and our assessments may change with identification of more hazards and further evidence of harm causation.

### 4.1.1 Human Health harms

Much of the conversation around plastic pollution harms in civil society has focussed on the damage that plastics can cause to the environment, especially marine ecosystems.[28, 117, 121] By comparison, there has been arguably less public attention on the human health harms of plastic pollution.

Based on our assessment, the potential societal burden from human-health harms could be even greater than non-human-health harms.[3] We estimate the social cost across Human Health harms runs into hundreds of billions of dollars per year.[133] By contrast, the size of harm for E&ES has a wide range that may exceed US$100 billion per annum, while harms to Nature are less than US$100 billion per annum (**Figure 13)**.

Most chemicals that are added to plastics are known endocrine disruptors. The endocrine system plays a central role in the reproductive, developmental, and metabolic functions of the human body. Much is still needed to tie all this together, but a sound inference can be made that endocrine disruption is a driver of many of the harms we have observed in the literature, and synthesised in this report.

Our results underline the opportunity for a rebalancing in the narrative around plastic pollution that should raise civil society's awareness of the direct Human Health costs of plastics production, use, and disposal.

### 4.1.2 Economies & ecosystem services

Research on the non-Human Health effects of plastic pollution has hitherto focussed on first-order effects such as harm to fish and other marine organisms.[121, 134, 121, 134] There have been few robust and definitive links to second-order economic impacts. Some studies have estimated very large numbers, especially for the cost of harms to the recovery, resilience, and regulation of marine ecosystem services.[121] However, more research is needed to robustly establish causation and fully determine the impacts of plastic pollution on economies, especially on terrestrial systems (such as harm to soil and other land-based food sources from chemical additives).[30, 65]

### 4.1.3 Nature harms

Calculating the current estimated social cost of harms to Nature – i.e., after excluding all E&ES benefits (such as marine natural capital and biodiversity's impact on ecosystems and food sources) – is based on heritage or intrinsic value. Putting a monetary figure on the amenity of wildlife and landscapes is challenging. In many regards, a vibrant oceanic ecosystem is invaluable to humans, and a potential plastic pollution-driven reduction of primary productivity could have incalculable consequences. The currently available options for making such estimates are unsatisfactory: (a) WTP for redress[135, 136] and (b) total cost of remediation. The former is not a reliable indication of true societal costs. For the latter, in almost every jurisdiction globally, Nature lacks a legal standing to enforce remediation without there being some associated bodily injury, or damage to property or livelihood.[f]

Not surprisingly, given its conceptual flaws, estimates for WTP have extremely high variability across contexts. This contrast with WTP-estimated costs for averting a human DALY, for which there are widely used, standardised estimates that exhibit comparatively less contextual variability.[135–137] Certain observed current levels put the WTP cost for Nature in the Low threshold (<US$10 billion per annum).[138] Remediation costs, on the other hand, are likely to be High (>US$100 billion per annum), especially for MNP.[125]

We present the cost of harms to Nature in this report as Low, based on heritage value and observed current WTP for redress. However, this could flip to High should there be a step-change in the legal or public resolve to pay for the remediation of nature back to its original state.

## 4.2 Implications of the social cost of pollution for the plastics industry

### 4.2.1 Contribution and relevance of plastics

Not all the hazards we have identified have pathways to harm that are primarily from plastics. This highlights how two important and distinct variables impact our results:

- for the social cost of a harm: the estimated proportion of the source coming from plastics (versus other materials and applications)
- for the scientific consensus on causation: the degree to which plastics are established as a specific pathway from source to harm.

[f]Although not undermining the general observation, there are exceptions to this. For example, in 2016, Colombian courts ruled that the Atrato River basin has rights to "protection, conservation, maintenance, and restoration." https://celdf.org/2017/05/press-release-colombia-constitutional-court-finds-atrato-river-possesses-rights

### Estimated proportion of source coming from plastics

#### PFAS

As detailed in the Results, we found plastics contribute only a very small proportion of PFAS: we estimate only three per cent are used in plastics. Of this, we calculated that just over two-thirds of PFAS by mass are the less harmful fluoropolymers.[139] The remaining third, therefore, are non-polymers, which means only ~1 per cent of total PFAS known to cause harm derives from plastics. This result caused the small sizing of harm for PFAS despite its well-publicised problems.[140, 141]

### Degree to which plastics is established as a specific pathway to harm

#### PFAS

Similar to sizing, we found plastic-specific pathways had little relevance to PFAS' causation of harm. We used a comprehensive keyword search of abstracts of human studies in the scientific literature to derive a plastic-specific relevance score. We found no human studies examining plastic as a pathway to harm for PFAS. This contrasts with other chemicals for which at least one study for each looked at plastic as a pathway to harm. Therefore, while PFAS harms are known, plastic-specific PFAS harms are less well-established.[83, 142, 143] Therefore, the low relevance score of PFAS contributed to its medium assessment of causation of harm.

#### MNP and macroplastics

By comparison, the relevance of MNP and macroplastics is self-evident – they are plastic materials. As such, we scored their relevance as high.

## 4.2.2 Implications for liability risk to the plastics industry

The clusters of harms outlined in section 3.4 (**Figure 13**) lay the groundwork for corporates and insurers to parse the myriad different risks and harms associated with plastic pollution. However, this clustering serves only as a foundation. Scientific consensus on causation does not begin to address legal issues surrounding attribution and culpability, both of which are necessary building blocks in assessing liability risk.

**This report has two main implications for the plastics industry as it concerns liability risk:**

- **Be proactive** in managing and mitigating the known and emerging harms, especially those with large estimated social cost. Even where there is no pathway to litigation, consideration should be given to self-regulation in advance of potential legislation.
- **Be aware** and monitor changes in the science and legal thinking on immature and indeterminate harms. There may even be a case for funding scientific research into potentially medium- or large-size indeterminate and immature harms.

### Be proactive

Known and emerging harms with large current estimated social burdens are likely to already pose sufficient risk to merit consideration by the legal and insurance systems (the focus of Annexes 2 and 3, respectively). Given the high consensus on causation and the large current estimated social cost of chemical additives and their harms to Human Health, they are of immediate interest to insurers. Bisphenols are already a highly regulated hazard in some jurisdictions.[80, 144, 145] Meanwhile for phthalates, multiple petitions have been filed with the FDA and class actions filed against food packaging companies in the US,[146, 147] indicating more stringent blanket regulation of the large family of phthalates is likely. To a lesser extent, halogenated and phosphate flame retardants are catching up. A lawsuit was filed against the EPA in the

US for inadequate protection against the toxic effects of decabromodiphenyl ether (DecaBDE) in 2021.[148] Furthermore, a number of bills are going through US state legislatures that prohibit the use of halogenated flame retardants more generally.[149]

The consensus around harm causation by MNP on Human Health and water sanitation is moderate.[23, 29, 150–152] However, the rapid rise in publications combined with a large baseline burden of harm merits special consideration by insurers.

The last two years have seen several "firsts" with regards to MNP as sources of Human Health harm. These include the first time MNP have been detected in human placenta, cirrhotic liver tissue, and lungs.[35–37] This opens up the possibility of congenital and transgenerational harm as well as digestive and respiratory injury, respectively.[114, 153]

- Even though the consensus on causation is not quite established, these are non-zero probabilities. When multiplied by the potential for catastrophic losses, the expected risk of such emerging harms from MNP is too big to ignore. This necessitates a proactive approach of the same kind suggested for chemical additives' harms on Human Health.
- Due to long latency periods, however, there may be no litigation on bodily injury or property damage for some time. For example, asbestos took 45 years from the first scientific paper to the first bodily injury litigation claim.[154]

## Be aware and monitor changes

While our assessment of the likelihood of consensus remaining the same are medium/high for indeterminate harms, this trajectory can change rapidly. Only a handful of studies are needed to inject momentum behind whether a plastic-related source causes a certain harm. A harm can quickly move from indeterminate to emerging.

This is especially the case for the other or unidentified plastic-related chemicals grouping. Our first attempt at comprehensive categorisation only scratches the surface of what harms may emerge. Any of the unidentified chemicals categorised within this grouped harm could "break out" as a consequence of identification and further research that bolsters scientific consensus. An example of this is our separation of phthalates – a known harm for their effect on Human Health – from other plasticisers. This separation is based on different current consensus around Human Health harm. As consensus evolves, this grouping could change to identify a class of non-phthalate substitutes worth their own grouping.[155]

Given the possibility of step changes or fast-moving incremental changes, it behoves insurers and corporates to monitor the research landscape closely. There may also be a role to play in funding research in certain neglected areas.

### 4.2.3 Implications for regulation

#### Plastic-related chemicals

Many chemical additives are regulated, but approaches vary greatly between jurisdictions.

#### Regulation in Europe

The EU has some of the most stringent regulations globally on chemical additives used in plastics, especially as they apply to Human Health. The most stringent regulations applicable to plastic chemical additives are the ECHA's REACH Authorisation List and its Persistent Organic Pollutants (POPs) regulations.[156] The Authorisation List effectively prohibits the manufacture, importation, or sale of substances of very high concern (SVHC) without special exemption from the European Commission.[157] This Authorisation List is fed by a Candidate List, with less stringent restrictions placed on these substances.[158]

Among the plastic-related chemicals on the Authorisation List are 14 phthalates. The Candidate List contains PFOA, PFOS, and BPA.[157, 158]

However, in identifying a list of plastic-related harms, we observed that many of the replacements for these restricted chemical additives may cause similar harms. These have been called "regrettable substitutions". For example, both PFNA and bisphenol S – replacements for PFOA and BPA, respectively – have growing evidence of similar harms to Human Health.[159–161] However, they remain unregulated.

Therefore, new regulations should focus more on evaluations at a wider group level, based on structural and functional similarity.[162]

#### Regulation in the US

In the US, regulation is complicated by the interlocking series of laws and regulations between the federal and state levels. Federal regulations tend to be less stringent than those found in some states. For example, phthalates intentionally added to food packaging in Maine are to be prohibited.[163] This contrasts with the federal level, where at least nine phthalates are authorised for use as additives in food-contact products.[111]

However, as we have noted, plastic pollution – including plastic-related chemical pollution – is mobile, ubiquitous, and persistent.[18, 19] They do not abide by state boundaries. Therefore, state-level regulations are insufficient to address the problem of mobile, pervasive pollutants. Taking this logic further implies federal-level regulations are similarly insufficient for many hazards that are a global problem and require international regulation.

#### International regulation and policies

In terms of international regulations, 2022 saw a United Nations Environment Assembly resolution to negotiate a legally binding global treaty on plastic pollution by 2024.[8] Other selected international treaties or resolutions relevant to plastic-related chemicals include:

- the Stockholm Convention, which regulates and lists some PFAS, three halogenated flame retardants (PBDEs HBCDD, and HBB), and SCCPs (previously used in great volume as plasticisers and flame retardants in PVC) as POPs, and restricts their use in most applications.[164]
- the International Convention for the Prevention of Pollution from Ships 1973 (MARPOL), which prohibits dumping of food, domestic, and other operational waste from ships, and covers plastic-related chemicals.[165]
- the Basel, Rotterdam, and Stockholm Conventions' Conference of the Parties (BRS COPs) resolved in 2022 to eliminate, globally, the use of perfluorohexane sulfonic acid, a type of PFAS.[166]

Our findings show that the full range of harms needs to be addressed. Harms to humans from chemical additives often receive a lot less attention than, for example, macroplastic pollution in the marine environment. Any future international regulations, such as the UN Environment Programme resolution on global plastic pollution, should ensure these less-visible harms are addressed.

### Macroplastics and MNP

Global agreements on macroplastics are relatively rare. The only legally binding global agreements address marine plastic pollution solely. These are the London Dumping Convention and MARPOL,[165] which prohibit ships from dumping plastic waste into the ocean. The United Nations Convention on the Law of the Sea seems to have little application to marine plastics and states are only required to reduce marine pollution "in accordance with their capabilities".[167]

However, some national and regional legislation does exist. The EU's Single-use Plastic Directive from 2019 addresses the issue by applying different measures to the ten most common single-use plastic products found as marine litter.[168] This is targeted at reducing the quantity of plastic waste generated, and 60+ countries have adopted similar legislation to phase out specific single-use plastic products like bags and cutlery.[156, 169, 170]

MNP are regulated at various levels globally, and proposed new legislation is increasing, such as prohibitions on the manufacture, packaging, and distribution of MNP in cosmetic products (intentionally added microbeads). The Netherlands was the first country to introduce such legislation, after which others like the US, UK, and Australia followed.[171] The ECHA has proposed an EU-wide regulation prohibiting any product designed to release primary MNP, and is expected to be adopted in the near future. Related is the proposed Plastic Pellet Free Waters Act,[172] which would prohibit plastic pellet discharge and other pre-production plastic pieces into waterways.

### Carbon emissions and climate change

Carbon emissions are governed internationally by the Paris Agreement, a legally binding treaty on climate change, on which most countries base their national laws.[173] The goal of limiting global warming by achieving net-zero emissions by 2050 forces countries and companies to make real effort towards transitioning to climate-neutral practices.

## 4.3. Limitations and challenges

### 4.3.1 Methodological limitations

### Lack of evidence hinders assessment

#### Risk

Certain plastic harms suffer from large data and evidence gaps. This is especially the case for harms to E&ES. While the literature does consider the fundamental science of a harm, there is a distinct lack of evidence on sizing the economic costs to society or at least sizing up impacts on an ecosystem or population level. An example of this is the effect of plastic-specific chemical additives, such as BPA, on soil health. The physical fundamentals of the harm that BPA may have on soil health have been established,[67, 174] but their ecological effects and economic costs have not.

For Human Health, one of the larger limitations was our assessment of the current estimated social cost of MNP harms. This relied on only one study's effect size, to which we applied our pooled-data discount factor. The social costs of Human Health harms have also been neglected in research terms, albeit to a lesser extent than E&ES. There have been some studies quantifying the cost of harms due to lost productivity from specific hazards. These have validated correlative studies/natural experiments on independent datasets or with alternative methodologies.[175]

### Impact

The lack of data for some harms drove our rationale to group certain specific harms together when carrying out our assessment. This meant we were unable to undertake assessments at a roughly equal level of granularity across receptors. We were also unable to robustly size the excess burden of some specific harms. This lack of robustness was particularly evident for MNP harms to Human Health, relying, as it did, on a single study.

### Mitigation

However, our framework was flexible enough to allow assessment of harms at different levels of granularity – depending on the data and evidence available – and then group them into roughly the same level of granularity for comparison. Lastly, in deriving our current estimated social cost results, we triangulated our results with analogue studies and through expert interviews. This validated, for example, our assessment of MNP harms to Human Health.

## Evidence for many harms relies on observational studies

### Risk

There are three experimental studies in which test groups of human subjects were exposed to chemical additives while corresponding control groups were not.[178, 179, 180] Given the ethical considerations, the vast majority of studies that support Human Health harms are from observational human studies, animal models, or *in vitro* experiments. Likewise, the evidence for E&ES harms comes from observational studies and, at most, natural experiments, which often lack high-quality controls. Furthermore, such experimental studies are unlikely to happen soon. Ethical concerns prevent human subjects and economies from being intentionally exposed to potential hazards. The three studies above are exceptions to the rule, as exposure to these chemicals was happening outside the context of the experiment.

### Impact

Without direct evidence from experimental studies or an accumulation of many observational studies, Human Health harms from MNP and macroplastics, and E&ES harms are unlikely to move into high consensus on causation.

### Mitigation

Many of the known harms we have described have not needed direct experimental evidence. For example, climate change, macroplastics' impact on Nature, and flame retardants' impact on Human Health all have a high consensus on causation. This is a consequence of an accumulation of high-quality, observational studies with consistent results. One telling macro-observation that supports, albeit very weakly, plastic as a cause of Human Health harm in general is the rapid rise in the incidence rate of cardiovascular diseases, autism, and Parkinson's disease among many others.[137] While some of this can be explained by improved diagnostic capabilities and capacities, as well as heightened awareness of such diseases, much of it cannot. Indeed, it may not be a coincidence that this rise has overlapped with the exponential accumulation of plastic usage and prevalence.

## Comparability of sizing across receptors is difficult

### Risk

Following on from the above, assessments of the three receptors of harm are inherently difficult to compare. We assessed the receptors using broadly the same framework, but we carried out a more systematic survey of the literature for Human Health than for the other two. This was due to access to exhaustive PubMed data, which provided a single reliable source for assessing Human Health harms.[g]

### Impact

Similar to our first limitation, we were unable to perform the assessment at a similar level of granularity across receptors. This was particularly conspicuous for sizing, where we had a solid evidence base on which to calculate baseline and excess burdens of harm with our use of DALYs for Human Health harms.

[g]Collection and annotation of data by Praedicat Inc.

### Mitigation

Our transformation of numerical values into categorical values for overall assessment provided us with a wide enough margin of error to help mitigate this and provide order-of-magnitude estimates rather than precise values.

## 4.3.2 Challenges with the science

### Establishing causation of harm and sizing

#### Relevance to receptors of interest

Most scientific studies on harm are carried out on laboratory animal models, and interpreting the real-world impact from these studies is challenging. An example are studies that have shown that laboratory mice have higher mortality rates following MNP exposure. Such studies may not translate into Human Health or even real-world Nature harm.[181, 182]. This may be due to differences between species and even differences between laboratory and wild organisms within a species. Furthermore, the artificial nature of such experiments in terms of context, exposure pathway, and dosage (discussed below) present a challenge in making the results more general. There is a real need to corroborate such studies with evidence in the field.

Similarly, for E&ES, plastics can be shown to harm fish (a Nature receptor), but it is difficult to translate that into an assessment of the second-order effects on the economic impact on fisheries and aquaculture.[124, 134]

#### Relevance of real-world dosages

The validity of laboratory studies is also questionable due to the differences between the concentrations of hazards found in the real world and in the laboratory. Furthermore, there are technical and technological limitations in detecting and measuring nanoparticles in non-isolable environment, such as a river or soil.[30, 31, 65] This presents difficulties in knowing whether they are present and causing damage, and if so, the extent of that damage. In general, doses present in laboratory experiments often exceed those present in the real world, so question marks hang over the real-world validity of such studies' conclusions, especially with regard to effect sizes.[182, 183]

### Real-world attribution of harm to plastics

#### Multiple pathways to harm

Chemical additives may cause harm, but not necessarily via plastics. A prime example of this is PFAS. PFAS is known to cause harm[83] but our analysis showed that such harm is very likely to originate from non-plastic sources. Given the number of toxicology studies that rely on laboratory results, there are many knowledge gaps in the literature regarding the sources and pathways by which chemical additives cause harm in the real world.[95, 183]

Moreover, observational studies that do look at real-world effects often consider associations with their presence (and concentration) and the harm they are purported to cause. They do not consider their source and pathway, and whether they are plastic-related.[183]

#### Complex mixtures

The science on chemical additives suffers from lack of real-world attribution. It is challenging to attribute harm to a plastic-related chemical when many of the same and different chemicals are present in the environment and causing similar harms.

#### Ubiquity of hazard and harm

Finally, certain sources of hazard (e.g., MNP in the environment) and of harm (e.g., cardiovascular disease) are highly ubiquitous. This makes any real-world attribution of harm to these sources problematic.[182] This challenge is reflected, for example, in our assessment of MNP, which score medium for consensus on causation for a range of different receptors.

## 4.4. Future scientific directions

### 4.4.1 Establishing causation of harm

**Emerging harms** require further high-quality studies into causation before scientific consensus is established, but this is happening. As our analysis of the publication rates of MNP Human Health harms showed, there has been a surge in studies. Below we outline the kinds of studies that need to be performed for different emerging harms.

#### Human Health harms from MNP

Study design. At present, there are only a handful of studies suggesting a role for MNP in human disease.[37, 38, 114] Only one of these studies suggests causation through a type of dose-response in which higher levels of microplastics were correlated with more severe inflammatory bowel disease.[38] Other correlative studies in humans may well indicate microplastic presence as a consequence rather than a cause of disease. Therefore, further human observational studies are needed.

Ideally, we would conduct prospective cohort studies. Such studies might follow groups of people who have been unintentionally exposed to higher levels of MNP through their occupation (e.g. for cosmetics)[184] or daily life (e.g. from bottled water). The study would also include a control group drawn from the general population. These studies could use follow-up disease data to assess the potential that disease rates in the two groups differed due to MNP exposure. Such studies could provide valuable insights.

Enablers of such research include funding for granular data collection, as well as improved technologies & techniques to detect MNP in human blood and solid tissues.[39]

#### Economies and ecosystem service costs from MNP and from chemical additives

**Study design**. These categories encompass harms to food sources and water pollution. To date, much of the research into these harms has focussed on two things: first-order effects on fish, soil, or water[51–53, 60, 70, 185] and more speculative sizing studies that do not add to evidence of causation.[121]

Therefore, natural experiments that examine the economic impact of natural variation in plastic exposure occurring across geography (cross-sectional) or across time (longitudinal) might help. The large number of variables that might confound any association between plastic levels and economic outputs make such studies extremely challenging, but not impossible.

**Enablers**, therefore, will be more granular data to enable researchers to account for potential confounders. Linked to the enablers suggested above, better technologies and techniques for detecting MNP and chemical additives in water and in animals and plants will also be critical.

### 4.4.3 Attribution of harm

As we discuss in section 4.3, there are significant challenges around attributing harm to a particular hazard in the real world.[181–183]

Attribution challenges are especially relevant for chemical additives to plastic. There are at least three primary types of challenge: existence of non-plastic sources, direct/indirect routes, and complex mixtures.

To address this, plastics research can learn from climate science. One of the methodological subfields of climate science is 'attribution science'.[190] The goal is to determine how much more likely or severe a specific event is in today's world versus the counterfactual, where there was no human-induced climate change. If the counterfactual and factual likelihood and severity are similar, the event cannot be attributed to human-induced climate change. Adapting and applying methodologies from attribution science may lead to further breakthroughs in the science around plastics.

# APPENDIX

## A1. SOURCES OF DATA AND INSIGHT

Peer-reviewed academic literature serves as the foundation for the identification and assessment of harms we carried out and detailed above. In all, we used three sources:

- peer-reviewed academic literature (both research and review articles)
- reports (without formal academic peer review) and publicly accessible databases
- interviews with academic experts.

### A1.1 Peer-reviewed academic literature

#### A1.1.1 Definition and scope

We used the PubMed database to search the academic literature for publications related to Human Health; these included *in vitro*, animal, and human studies. For plastic pollution studies with a focus on E&ES and Nature, we expanded our search to include Google Scholar and Web of Science.

#### A1.1.2 Search strategy

We searched published articles in the aforementioned databases with no time constraints. We also performed "reference mining" by searching the bibliographies of retrieved articles looking for additional relevant publications. These searches were last updated in June 2022.

For our Human Health assessment, the search strategy was conducted by the analytics firm Praedicat Inc. Their search of the PubMed database was exhaustive.

For other literature sources for E&ES and Nature, we took only a representative sample of recent publications.

#### A1.1.3 Data extraction and classification

With the assistance of Praedicat Inc., the abstracts of all PubMed-indexed publications were reviewed and the following data points recorded for each: effect type name, effect size, confidence intervals, and statistical significance. We extracted 7,400 hazard-harm relationships from 5,420 studies indexed by PubMed.

Using these extracted data, we classified the studies by:

- year of publication
- hazard type, which was based on a search of commonly used chemical additives in plastics,[191] as well as micro- and nanoplastics
- harm type, which was based loosely on PubMed's Medical Subject Headings (MeSH)
- study design, with options ranging from reviews, observational (cohort), case control, meta-analysis, and experimental/randomised controlled trial (RCT)
- study subject, with options including *in vitro*, animal, and human subjects.

## A1.2 Reports

(without formal academic peer review) and publicly accessible databases

### A1.2.1 Definition and scope

In order to obtain a more rounded view of the issues involved around plastics, and gather real-world socioeconomic data to inform our assessments, we leveraged reports from outside the academic peer-reviewed literature, as well as databases from reputable organisations.

Where we used such resources, they are cited and included in our list of references. They comprise reportsby intergovernmental organisations like the Food & Agricultural Organisation (FAO), economic data from the World Bank, and reports by non-profit organisations such as the Center for International Environmental Law (CIEL).

### A1.2.2 Search strategy

We searched intergovernmental organisation websites in a non-systematic way for reports addressing plastic production, use, disposal, and pollution. If we came across a comprehensive, well-referenced report and database, such as the Organisation for Economic Co-operation and Development's (OECD) Global Plastics Outlook,[192] we reference mined this to find other sources of reputable information.

### A1.2.3 Classification

These resources broadly comprise one-off reports, recurring reports (such as bulletins released annually), or databases, and are sourced from the following types of organisations:

- intergovernmental
- corporate or private sector
- non-profit and non-governmental
- governmental or public sector.

## A1.3 Expert interviews

### A1.3.1 Definition and scope

We also conducted interviews with academic researchers active in the environmental and health sciences. This further guided the literature review and provided the latest state of research as of July 2022.

### A1.3.2 Search strategy

In total, we interviewed 17 experts. The selection of interviewees was initially guided by scientists at Minderoo Foundation's Plastics & Human Health program, and expanded with the literature review and recommendations from the initial group of interviewees.

# A2. IDENTIFICATION OF HARMS

The long list of plastic-related specific harms we identified is given below.

**Table A1:** Long list of specific harms

| Source type | Source | Receptor | Specific harm |
|---|---|---|---|
| Chemical additives | Bisphenols | Human health | Bisphenol from plastics exposure leading to cognitive disorder |
| Chemical additives | Bisphenols | Human health | Bisphenol from plastics exposure leading to lung injury |
| Chemical additives | Bisphenols | Human health | Bisphenol from plastics exposure leading to cardiovascular disease |
| Chemical additives | Bisphenols | Human health | Bisphenol from plastics exposure leading to endocrine & immune system injury |
| Chemical additives | Bisphenols | Human health | Bisphenol from plastics exposure leading to reproductive injury |
| Chemical additives | Bisphenols | Human health | Bisphenol from plastics exposure leading to nervous system injury |
| Chemical additives | Bisphenols | Human health | Bisphenol from plastics exposure leading to developmental injury |
| Chemical additives | Bisphenols | Human health | Bisphenol from plastics exposure leading to cancer |
| Chemical additives | Flame retardants | Human health | Flame retardant from plastics exposure leading to reproductive injury |
| Chemical additives | Flame retardants | Human health | Flame retardant from plastics exposure leading to cognitive disorder |
| Chemical additives | Flame retardants | Human health | Flame retardant from plastics exposure leading to endocrine and immune system injury |
| Chemical additives | Flame retardants | Human health | Flame retardant from plastics exposure leading to kidney and liver injury |
| Chemical additives | Flame retardants | Human health | Flame retardant from plastics exposure leading to developmental injury |
| Chemical additives | Flame retardants | Human health | Flame retardant from plastics exposure leading to nervous system injury |
| Chemical additives | PFAS | Human health | PFAS from plastics exposure leading to cardiovascular disease |
| Chemical additives | PFAS | Human health | PFAS from plastics exposure leading to endocrine & immune system injury |
| Chemical additives | PFAS | Human health | PFAS from plastics exposure leading to kidney and liver injury |
| Chemical additives | PFAS | Human health | PFAS from plastics exposure leading to reproductive injury |
| Chemical additives | PFAS | Human health | PFAS from plastics exposure leading to developmental injury |
| Chemical additives | PFAS | Human health | PFAS from plastics exposure leading to cognitive disorder |
| Chemical additives | PFAS | Human health | PFAS from plastics exposure leading to cancer |
| Chemical additives | PFAS | Human health | PFAS from plastics exposure leading to bone or joint injury |
| Chemical additives | Phthalates | Human health | Phthalate from plastics exposure leading to kidney and liver injury |
| Chemical additives | Phthalates | Human health | Phthalate from plastics exposure leading to developmental injury |
| Chemical additives | Phthalates | Human health | Phthalate from plastics exposure leading to reproductive injury |
| Chemical additives | Phthalates | Human health | Phthalate from plastics exposure leading to endocrine & immune system injury |
| Chemical additives | Phthalates | Human health | Phthalate from plastics exposure leading to lung injury |
| Chemical additives | Phthalates | Human health | Phthalate from plastics exposure leading to nervous system injury |

| Source type | Source | Receptor | Specific harm |
|---|---|---|---|
| Chemical additives | Phthalates | Human health | Phthalate from plastics exposure leading to cancer |
| Chemical additives | Phthalates | Human health | Phthalate from plastics exposure leading to cognitive disorder |
| Chemical additives | Other chemicals | Human health | Unidentified or other chemicals exposure from plastics leading to significant human harms |
| Macroplastics | Macroplastics | Human health | End-of-life burning leading to cancer |
| Macroplastics | Macroplastics | Human health | End-of-life burning leading to cardiovascular injury |
| Macroplastics | Macroplastics | Human health | End-of-life burning leading to endocrine and immune system injury |
| Macroplastics | Macroplastics | Human health | End-of-life burning leading to nervous system injury |
| Macroplastics | Macroplastics | Human health | End-of-life burning leading to lung injury |
| Macroplastics | Macroplastics | Human health | Air pollution from PM 2.5 release during plastic production |
| Macroplastics | Macroplastics | Human health | Air pollution from contamination of production made from recycled plastics |
| MNP | MNP | Human health | MNP direct exposure leading to developmental injury |
| MNP | MNP | Human health | MNP direct exposure leading to kidney and liver injury |
| MNP | MNP | Human health | MNP direct exposure leading to cardiovascular injury |
| MNP | MNP | Human health | MNP direct exposure leading to lung injury |
| MNP | MNP | Human health | MNP direct exposure leading to gastrointestinal injury |
| MNP | MNP | Human health | MNP direct exposure leading to reproductive injury |
| MNP | MNP | Human health | MNP direct exposure leading to nervous system injury |
| MNP | MNP | Human health | MNP direct exposure leading to endocrine & immune system injury |
| MNP | MNP | Human health | MNP indirect delivery of other harmful agents (heavy metals, etc.) |
| $CO_2$e emissions | $CO_2$e emissions | Human health | Air pollution |
| $CO_2$e emissions | $CO_2$e emissions | E&ES | Global warming |
| $CO_2$e emissions | CO2e emissions | E&ES | Air pollution |
| CO2e emissions | $CO_2$e emissions | E&ES | Ocean acidification |
| $CO_2$e emissions | $CO_2$e emissions | E&ES | Extreme weather |
| Chemical additives | Chemical additives | E&ES | Chemicals from plastic leading to marine dead zones; affecting fish stock |
| Chemical additives | Chemical additives | E&ES | Chemicals from plastic leading to soil degradation |
| Macroplastics | Macroplastics | E&ES | Ingestion of macroplastics leading to starvation/suffocation of animals |
| Macroplastics | Macroplastics | E&ES | Animal entanglement in macroplastic waste |
| Macroplastics | Macroplastics | E&ES | Environmental destruction in oil extraction for plastic production |
| Macroplastics | Macroplastics | E&ES | Environmental destruction in actual plastic production |
| Macroplastics | Macroplastics | E&ES | Reduction of biodiversity in areas surrounding landfills |
| Macroplastics | Macroplastics | E&ES | Reduction in livelihood due to macroplastic waste (e.g., beach resorts) |
| Macroplastics | Macroplastics | E&ES | Product recall due to greenwashing claims |
| Macroplastics | Macroplastics | E&ES | Drop in share price due to inability to meet claims on recycled content |

| Source type | Source | Receptor | Specific harm |
|---|---|---|---|
| Macroplastics | Macroplastics | E&ES | Drop in share price due to inability to meet claims on recyclability/ biodegradability |
| MNP | MNP | E&ES | MNP direct exposure leading to reduced marine food sources (fish, molluscs, etc.) |
| MNP | MNP | E&ES | MNP direct exposure leading to reduced land animal food sources (livestock, etc) |
| MNP | MNP | E&ES | MNP direct exposure leading to reduced land plant food sources (crops, etc) |
| MNP | MNP | E&ES | MNP direct exposure leading to reduced land animal food sources (fodder crops, etc) |
| MNP | MNP | E&ES | MNP indirect delivery of other harmful agents leading to reduced land food sources |
| MNP | MNP | E&ES | MNP indirect delivery of other harmful agents leading to reduced marine food sources |
| MNP | MNP | E&ES | MNP indirect delivery of antibiotic-resistant microorganisms |
| Chemical additives | Chemical additives | Nature | Chemicals exposure from plastic leading to aquatic dead zones |
| Chemical additives | Chemical additives | Nature | Chemicals exposure from plastic leading to soil degradation |
| Chemical additives | Chemical additives | Nature | Exposure to chemicals from plastic leading to endocrine disruption in animals |
| Chemical additives | Chemical additives | Nature | Exposure to chemicals from plastic leading to direct toxicity in animals |
| Chemical additives | Chemical additives | Nature | Exposure to chemicals from plastic leading to developmental problems in animals |
| Macroplastics | Macroplastics | Nature | Ingestion of macroplastics leading to starvation/suffocation of animals |
| Macroplastics | Macroplastics | Nature | Animal entanglement in macroplastic waste |
| Macroplastics | Macroplastics | Nature | Environmental destruction in oil extraction for plastic production |
| Macroplastics | Macroplastics | Nature | Environmental destruction in actual plastic production |
| Macroplastics | Macroplastics | Nature | Reduction of biodiversity in areas surrounding landfills |
| MNP | MNP | Nature | MNP direct harm to terrestrial plants |
| MNP | MNP | Nature | MNP direct harm to aquatic plants |
| MNP | MNP | Nature | MNP direct harm to terrestrial animals |
| MNP | MNP | Nature | MNP direct harm to aquatic animals |
| MNP | MNP | Nature | MNP indirect delivery of other harmful agents to terrestrial plants |
| MNP | MNP | Nature | MNP indirect delivery of other harmful agents to aquatic plants |
| MNP | MNP | Nature | MNP indirect delivery of other harmful agents to terrestrial animals (heavy metals, etc.) |
| MNP | MNP | Nature | MNP indirect delivery of other harmful agents to marine animals (heavy metals, etc.) |
| CO2e emissions | CO2e emissions | Nature | Global warming |
| CO2e emissions | CO2e emissions | Nature | Air pollution |
| CO2e emissions | CO2e emissions | Nature | Ocean acidification |
| CO2e emissions | CO2e emissions | Nature | Extreme weather |

# A3. ASSESSMENT OF HARMS

## A3.1 Estimating size of social cost

Our approach was to calculate the product of the unit size of a harm in dollar terms and the current estimated excess burden due to the harm.

### A3.1.1 Baseline harm burden

We sourced data from the literature to give a current baseline estimate of the burden of a harm in financial terms:

- **for Human Health harms**, we mapped the harms to Global Burden of Disease (GBD) causes,[193] and mapped these to the standardised methodology for estimating disease burden using disability-adjusted life years (DALYs), which are a measure of years of life lost to ill health or early death
- **for harms to E&ES**, we took the estimated value of the market for which the harm was most likely to affect.

### A3.1.2 Effect size

Harms were assessed based on the possible effect they may have on the baseline burden:

#### Human Health

For Human Health harms, we extracted dichotomous studies (studies that class an association as either positive or negative) with effect-size data. The steps taken were as follows.

1. **Identifies the type of effect size used in a study, and convert results shown as odds ratios into risk ratios, where population or sample sizes available**
2. **Classified studies into either those:**
   - With cohorts selected, either randomly or as part of a consecutive series, from the general population ("general-population cohort studies"), or
   - That were not cohort studies (e.g. case-control studies), or did not select their study population from a general population (e.g. only factory workers or only cancer patients) ("narrow-population or non-cohort studies")
3. **For specific harms with:**
   - One effect size from a general-population cohort study, we used that value
   - More than one effect size from general-population cohort studies, we took the average
   - No effect size from a general-population cohort study, we:
     - Calculated a discount factor pooled across different sources' data as follows

$$\text{Discount factor} = \frac{\text{"Average effect size of 'general-population studies'"-1}}{\text{"Average effect size of 'narrow-population or non-cohort studies'"-1}}$$

     - Applied the discount factor to the effect sizes of specific hazard-harm relationships from "narrow-population or non-cohort studies"
     - Took the average discounted effect sizes for each specific harm

4. **Assumed the effect sizes extracted or calculated represent the following (given that plastics have been in widespread use for decades, baseline burden of harm already incorporates the potential harm from plastic exposure):**

$$\text{Effect size} = \frac{\text{Baseline harm burden}}{\text{“Baseline harm burden”- “Excess from exposure”}}$$

5. **Rearranged average effect size for a specific harm into the following, so that it could be multiplied by baseline burden of harm and estimated % source of exposure from plastic to calculate current estimate of excess burden of harm**

$$\text{Adjusted effect size} = \text{"1"} - \frac{\text{"1"}}{\text{"Effect size"}}$$

## E&ES

For harms to E&ES, we took a more qualitative approach, estimating the impact of plastic harms as a proportion of the size of the market or service.

### A3.1.3 Estimated percentage of source coming from plastics

For both E&ES and Human Health harms, we sourced data on the relevant contribution made to those harms by the hazards via plastic-related pathways. These percentage contributions were used to scale the harm burdens calculated from the product of the effect size and baseline burden.

### A3.1.4 Unit size of harm

We converted the product of the baseline burden, effect size, and percentage of source coming from plastics (the current estimated excess burden of harm) into financial costs.

- for Human Health harms, the cost of each DALY was set as the weighted global average from willingness to pay ("WTP") surveys (US$15,700 per annum).[194]
- harms to E&ES were already expressed in US$ amounts.

### A3.1.5 Harms to Nature

For harms to Nature specifically, the monetary value of wildlife and companion animals can be estimated by willingness to pay, remediation costs, and even commercial value to tourism and other ecosystems services (for wildlife and biodiversity).[5]

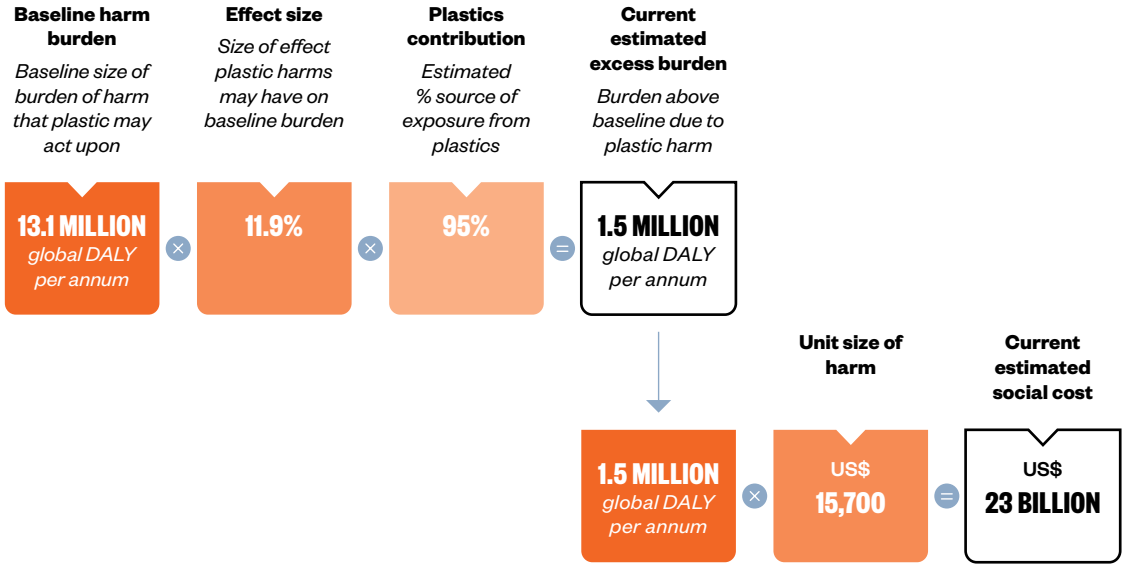
However, estimates for WTP vary widely across contexts (in contrast to costs for averting a human DALY, for which there are widely used, standardised estimates). Meanwhile, commercial value to tourism is already incorporated into harms to E&ES. For remediation costs, which are likely to be high (>US$100 billion per annum globally), many might argue restoring nature to its original state can (and should) be quantified. By contrast, others believe that remediation is of low societal priority.[195]

As such, given the inability to enforce remediation in many jurisdictions, we took the blanket view that the size of societal harm is small. We expand upon this in section Discussion 4.1.3.

## A3.1.6 Worked examples

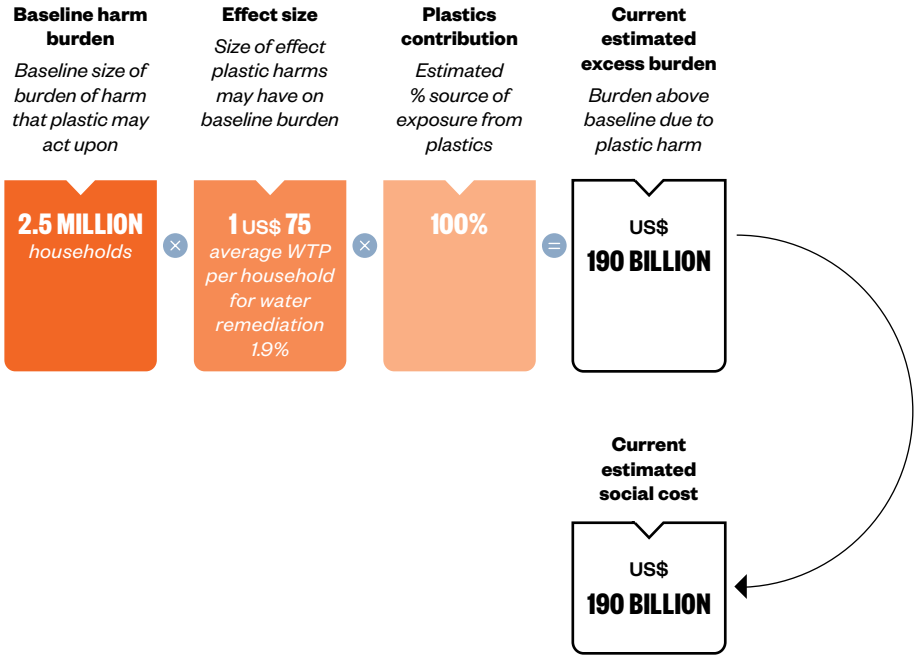
To supplement the steps outlined in Methods section 2.2.1, we provide two worked examples.

**Box A1:** Current estimated social cost from bisphenols from plastic exposure leading to developmental injury




- We chose bisphenol's harm on developmental injuries because it has a well-established cause with many papers and a large size of harm due to large effect size in the literature;[196] we also deemed it an important and representative harm to Human Health.
- We collected data on the global burden of developmental disorders (disorders that affect people during infanthood or childhood), measured in DALYs to serve as a baseline for harm burden.[193]
- We then derived a figure attributable to the size of the effect of bisphenol on the baseline burden of harm; taking this from the average effect sizes extracted from the literature (Praedicat data).
- We found an estimate of the proportion of bisphenol coming from plastics: 95%.[197]
- Multiplying the three factors, baseline burden, effect size, and plastic-specific contribution gave us the current estimated excess burden attributable to bisphenol.
- We then sized the global cost of 1 DALY in USD to arrive at a current estimated social cost of US$23 billion.
- Our component data points would collectively have to be wrong by more than 55% for our assessment to fall into a lower category, giving an assessment safety factor.

**Box A2:** Current estimated social cost from MNP contamination of water sanitation




- We chose MNP contamination of treated water, given its global relevance.
- We considered the potential exposed population for wastewater treatment to be roughly equivalent to the number of households globally at ~2.5 billion households.
- The effect of wastewater pollution is remediation costs; separate Swiss and Kazakh studies have found WTP for remediating water sanitation equates to around $75 (after scaling up and down for GDP per capita, respectively)
- Plastic-specific contribution to MNP is 100%.
- Given the ubiquity of MNP in wastewater,[198–200] we assume that the scale of remediation would remain the same even if MNP were the only pollutant.
- Similar to bisphenols above, at ~$200 billion estimated size of harm, our component data points would collectively have to be wrong by 50% for our assessment to fall into the medium category.

## A3.2. Assessing consensus on causation of harm

Our approach for assessing the current confidence or probability that a hazard causes harm based on the scientific literature was to score harms via the following subdimensions, and sum them to give a categorical value (High, Medium, Low).

### A3.2.1 Quality of studies

**Harms were assessed based on their study type and design:**

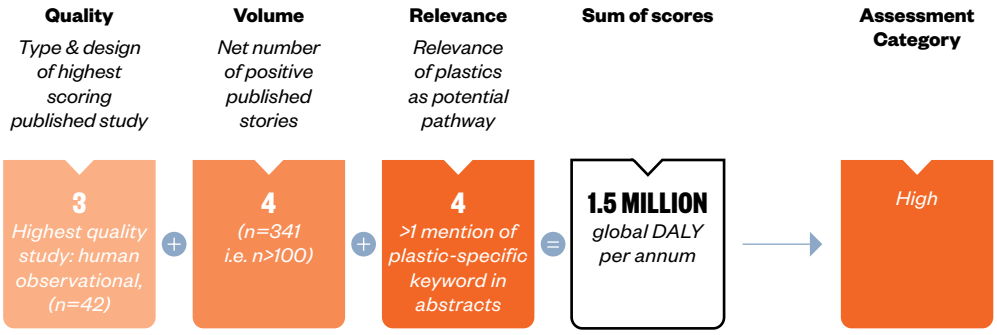
- for Human Health harms, we scored each extracted publication according to its study type, design, and subject, and found the highest scoring study for each specific harm. Publications with human RCTs or meta-analysis scored highest and *in vitro* studies scored lowest.
- For harms to E&ES and Nature, we qualitatively assessed the quality of the literature on the specific harm.

### A3.2.3 Plastic relevance of studies

**Harms were assessed based on whether plastic was a typical pathway; all harms due to macroplastic and MNP sources were assessed as high by definition:**

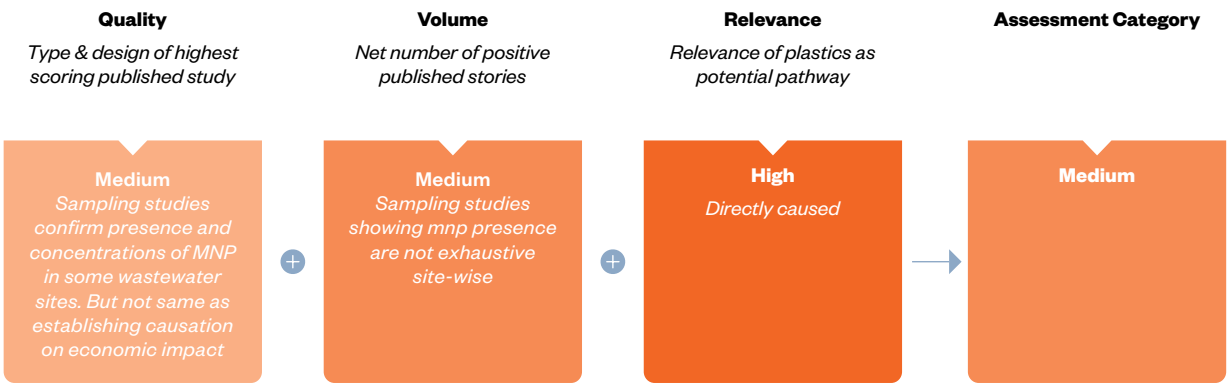
- for Human Health harms, we performed keyword searches for "plastic", "plastics", "macroplastic*", "microplastic*", "nanoplastic*", "polyethylene", "polypropylene", "polyvinyl*", "polystyrene", "polycarbonate", "plasticiz*", "plasticis*", and scored harms in a binary manner (albeit on the same scale as quality and volume) according to whether at least one of those keywords were present or whether they were all absent.
- for harms to E&ES and Nature, we reviewed the literature to score each harm based on its pathway.

**Box A3:** Current consensus of harm causation for bisphenols from plastic exposure leading to developmental injury




- We chose bisphenol's harm on developmental injuries because it has a well-established cause with many papers and a large size of harm due to large effect size in the literature;[196] we also deemed it an important and representative harm to Human Health.
- With the help of Praedicat Inc., we exhaustively collected data on all studies indexed by PubMed looking at a relationship between bisphenol and developmental injuries.
- Harms under developmental injuries were classified by Praedicat Inc., loosely based on MeSH subheadings and mapped onto GBD causes.
- The highest-scoring studies found for bisphenol's effect on developmental injuries were observational studies on humans, which in our scoring system were assigned a score of 3.
- We found 387 studies that observed a positive association between bisphenol and developmental injuries and 46 studies that found no or a negative association. The net number resulted in 341 hazard-harm associations. This was well in excess of our logarithmic scale that assigned a high score to more than 100 studies showing net positive hazard-harm associations.
- Our keyword search of abstracts for relevance to plastic pathways of exposure found many instances, meaning we assigned a score of 4 in our binary scoring system for plastic relevance.
- Summing the scores yields a total of 11, which put the harm in the High category (see **Figure 7** in Methods section 2.2.2).

**Box A4:** Current consensus of harm causation for MNP contamination of water sanitation




- We chose MNP contamination of treated water given its global relevance.
- We found numerous studies confirming the presence of MNP in wastewater treatment plants, with a study in the US estimating 4 million microparticles were released per facility per day.[10] However, while the presence of MNP is confirmed, the economic impact of their presence has not been assessed.
- There was not a broad enough geographical cross-section of studies confirming the presence of MNP in all treated water – not just wastewater – to be able to assess volume of studies as High.
- MNP are, by definition, sourced from plastics, thus scoring High for plastic relevance.
- Our overall assessment of the scientific consensus that MNP contamination of water sanitation causes harm is Medium.

## A3.3. Assessing likelihood consensus on causation (and size) remains static

Our approach for assessing the confidence or likelihood that a hazard causing harm would remain stable in the near-term was to score harms via the following subdimensions, and sum them to give a categorical value (High, Medium, Low).

### A3.3.1 Stability

Harms were assessed on the change in the number of studies on specific hazard-harm associations published over the last three years (2018–2021). Harms that experienced a doubling of publications scored the lowest (low stability), and those with no growth or with fewer studies (high stability) published scored highest (to indicate that the consensus or willingness to research the harm may be stabilising):

- for Human Health harms, due to our exhaustive download of PubMed studies from Praedicat Inc., we were able to perform an exhaustive search of the number of studies on a harm published over the last three years
- for harms to E&ES and Nature we performed a non-exhaustive count of the number of studies on a harm published over the last three years.

### A3.3.2 Timeframe

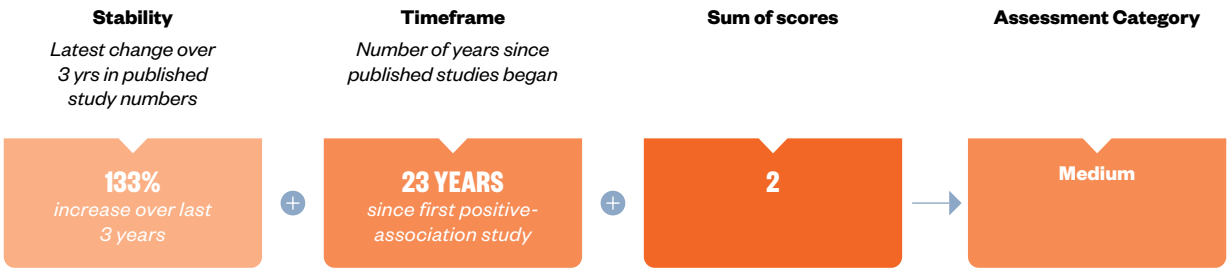
Harms were assessed on the duration for which they had published studies demonstrating a positive hazard-harm association:

- for Human Health harms, due to our exhaustive download of PubMed studies from Praedicat Inc., we were able to perform an exhaustive search of the earliest study on a harm showing a positive hazard-harm association
- for harms to E&ES and Nature, we performed a Google Scholar search – using a harm-specific keyword search – of the earliest peer-reviewed research article (not a review) showing a positive hazard-harm association.

### A3.3.3 Worked examples

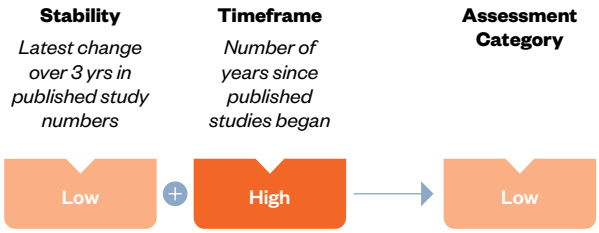
To supplement the steps outlined in Methods section 2.2.2, we provide two worked examples.

**Box A5:** Future consensus of harm causation for bisphenols from plastic exposure leading to development injury




- We chose bisphenol's harm on developmental injuries because it has a well-established cause with many papers and a large size of harm due to large effect size in the literature;[6] we also deemed it an important and representative harm to Human Health.
- With the help of Praedicat Inc., we exhaustively collected data on all studies indexed by PubMed looking at a relationship between bisphenol and developmental injuries.
- Harms under developmental injuries were classified by Praedicat Inc., loosely based on MeSH subheadings and mapped onto GBD causes.
- We found a 133% increase in the number of studies between 2018 and 2021 looking at bisphenol as a source of harm on developmental injuries. This was more than double the number of studies over the three years showing high growth.
- We found it was 23 years since the first study showing a positive hazard-harm association.[201]
- Summing the scores yields a total of 2, which put the harm in the Medium category (see Exhibit 8 in Methods section 2.2.2).
- While bisphenol's effect on developmental injury seemed to experience an increase in interest over the last three years, in general, studies on bisphenol harms were relatively stable, showing growth under 10% between 2018 and 2021 and 41 years since the first publication on bisphenol's adverse effects on reproductive health.[202]

**Box A6:** Future consensus of harm causation for MNP contamination of water sanitation




- We chose MNP contamination of treated water given its global relevance.
- A non-systematic review of the literature showed many studies conducted since 2019 on the presence of MNP in wastewater,[198, 199, 203–15] and, to a lesser extent, on treated drinking water.[206, 207] This "explosion" of interest led to us scoring stability Low, positing that consensus on harm causation – especially with studies on the second-order economic impacts currently lacking – is likely to evolve rapidly in the near future.
- It has been 24 years since the first study detecting the presence and analysing the concentration of plastic microfibres in wastewater and wastewater-derived biosolids used as fertiliser.[208] As such, we assessed the length of time the harm had been studied as High. However, while noting this, studies have only begun to increase after a seminal study in 2011 that looked at the microfibres in washing machine wastewater and its pollution of beaches.[209]
- Given these factors, our overall assessment of the future consensus that MNP contamination of water sanitation causes harm would remain static is Low. This is driven by the increased interest in the area as a topic of research and the fact that much of the economic-impact research is relatively immature.

# A4. RESULTS IN DETAIL

## A4.1 Estimated social cost of harm

### Human Health

**Table A2:** Detailed results of current estimated social cost of plastic harms on Human Health

| Source | Specific harm | Baseline burden / (DALYS) | Effect size | Estimated % source from plastics | Current estimated social cost / (billion USD) |
|---|---|---|---|---|---|
| Bisphenols | Bisphenol from plastics exposure leading to cardiovascular disease | 30,265,804 | 1.20 | 95% | 76.5 |
| | Bisphenol from plastics exposure leading to cognitive disorder | 5,902,625 | 1.06 | 95% | 4.7 |
| | Bisphenol from plastics exposure leading to developmental injury | 13,056,564 | 1.14 | 95% | 23.1 |
| | Bisphenol from plastics exposure leading to endocrine & immune system injury | 22,906,564 | 1.28 | 95% | 74.9 |
| | Bisphenol from plastics exposure leading to lung injury | 21,550,977 | 1.12 | 95% | 34.0 |
| | Bisphenol from plastics exposure leading to nervous system injury | 4,263,390 | 1.04 | 95% | 2.3 |
| | Bisphenol from plastics exposure leading to reproductive injury | 981,036 | 1.39 | 95% | 4.1 |
| Flame retardants | Flame retardant from plastics exposure leading to cognitive disorder | 5,902,625 | 1.01 | 80% | 1.0 |
| | Flame retardant from plastics exposure leading to developmental njury | 13,056,564 | 1.19 | 80% | 26.5 |
| | Flame retardant from plastics exposure leading to endocrine and immune system injury | 22,906,564 | 1.20 | 80% | 46.9 |
| | Flame retardant from plastics exposure leading to kidney and liver injury | 20,019,387 | 1.03 | 80% | 7.3 |
| | Flame retardant from plastics exposure leading to nervous system injury | 4,263,390 | 1.01 | 80% | 0.5 |
| | Flame retardant from plastics exposure leading to reproductive injury | 981,036 | 1.79 | 80% | 5.4 |
| PFAS | PFAS from plastics exposure leading to bone or joint injury | 18,948,965 | 1.38 | 1% | 0.8 |
| | PFAS from plastics exposure leading to cancer | 4,052,817 | 1.18 | 1% | 0.10 |
| | PFAS from plastics exposure leading to cardiovascular disease | 30,265,804 | 1.60 | 1% | 1.8 |
| | PFAS from plastics exposure leading to cognitive disorder | 5,902,625 | 1.01 | 1% | 0.0 |
| | PFAS from plastics exposure leading to developmental injury | 13,056,564 | 1.30 | 1% | 0.5 |
| | PFAS from plastics exposure leading to endocrine & immune system injury | 22,906,564 | 1.69 | 1% | 1.5 |
| | PFAS from plastics exposure leading to kidney and liver injury | 20,019,387 | 1.46 | 1% | 1.0 |
| | PFAS from plastics exposure leading to reproductive injury | 981,036 | 1.23 | 1% | 0.03 |

| Source | Specific harm | Baseline burden / (DALYS) | Effect size | Estimated % source from plastics | Current estimated social cost / (billion USD) |
|---|---|---|---|---|---|
| Phthalates | Phthalate from plastics exposure leading to cognitive disorder | 5,902,625 | 1.29 | 90% | 18.5 |
| | Phthalate from plastics exposure leading to developmental injury | 13,056,564 | 1.24 | 90% | 35.8 |
| | Phthalate from plastics exposure leading to endocrine & immune system injury | 22,906,564 | 1.22 | 90% | 57.6 |
| | Phthalate from plastics exposure leading to kidney and liver injury | 5,902,625 | 1.05 | 90% | 4.0 |
| | Phthalate from plastics exposure leading to lung injury | 21,550,977 | 1.21 | 90% | 52.0 |
| | Phthalate from plastics exposure leading to nervous system injury | 4,263,390 | 1.03 | 90% | 1.6 |
| | Phthalate from plastics exposure leading to reproductive injury | 981,036 | 1.18 | 90% | 2.1 |
| Other chemicals | Unidentified or other chemicals exposure from plastics leading to significant human harms | 22,906,564 | 1.23 | 50% | 33.9 |
| MNP | MNP direct exposure leading to cardiovascular injury | 30,265,804 | 0.00 | 100% | - |
| | MNP direct exposure leading to developmental injury | 13,056,564 | 0.00 | 100% | - |
| | MNP direct exposure leading to endocrine & immune system injury | 22,906,564 | 0.00 | 100% | - |
| | MNP direct exposure leading to gastrointestinal injury | 19,802,461 | 1.08 | 100% | 23.5 |
| | MNP direct exposure leading to kidney and liver injury | 20,019,387 | 0.00 | 100% | - |
| | MNP direct exposure leading to lung injury | 21,550,977 | 0.00 | 100% | - |
| | MNP direct exposure leading to nervous system injury | 4,263,390 | 0.00 | 100% | - |
| | MNP direct exposure leading to reproductive injury | 981,036 | 0.00 | 100% | - |
| MNP | MNP indirect delivery of other harmful agents (heavy metals, etc.) | 403,088,030 | 1.20 | 0.5% | 5.3 |
| Macroplastics | Human harm from air pollution (PM2.5) from macroplastic production | N/A | N/A | 1.7% | 54.4 |
| | Human harm from burning of end-of-life macroplastics | N/A | N/A | 100% | 10.6 |

**Note:** See Box A1 for a worked example of calculation. Unit size is US$15,700, and is a global estimate of the cost of averting 1 DALY

## Economies & Ecosystem Services and Nature

**Table A3:** Detailed results of current estimated social cost of plastic harms to E&ES and Nature

| Source | Receptor | Grouped harm | Total economic burden | Estimated losses | Estimated % source from plastics | Current estimated social cost / (billion USD) |
|---|---|---|---|---|---|---|
| Chemical additives | E&ES | Harm to aquatic and terrestrial food sources from chemical additives | US$5 trillion Global annual size of crop, fisheries & aquaculture markets | High | Low | <10 |
| | Nature | Harm to aquatic and non-human terrestrial organisms from chemical additives | Low* | High | Low | <10 |
| Macroplastics | E&ES | Harm to aquatic and terrestrial food sources from macroplastics | US$5 trillion Global annual size of crop, fisheries & aquaculture markets | Low | High | <10 |
| | | Harm to tourism from macroplastics | US$1.7 trillion Global annual size of tourism market | Low | High | |
| | Nature | Harm to aquatic and non-human terrestrial organisms from macroplastics | Low | High | High | <10 |
| MNP | E&ES | Harm to aquatic and terrestrial food sources from MNP (direct) | US$5 trillion Global annual size of crop, fisheries & aquaculture markets | Low | High | <10 |
| | | Harm to water sanitation from MNP (direct) | 2.5 billion households Global households potentially affected | US$75 | High | >100 |
| | | Harm to aquatic and terrestrial food sources from MNP (indirect) | US$5 trillion Global annual size of crop, fisheries & aquaculture markets | US$10 billion | Low | <10 |
| | Nature | Harm to organisms from MNP (direct and indirect) | Medium | Low | High | <10 |
| All plastics | E&ES | Harm to marine natural capital from all plastics | High | High | High | >100 |
| $CO_2e$ emissions | All | Harm to all receptors from carbon emissions and climate change | US$180 billion Estimated global annual cost of emissions from plastic-attributable carbon | High | Low | >100 |

**Note:** See Box A2 for a worked example

* All Nature harms are considered low (see section 4.1.3 in Annex 1 for explanation)

## A4.2 Scientific consensus on causation

### Human Health

**Table A4:** Detailed results of current scientific consensus of plastic harms to Human Health

| Source | Specific harm | Quality of publications | Cumulative number of net positive-association studies | Relevance of plastics as a potential pathway | Current scientific consensus |
|---|---|---|---|---|---|
| Bisphenols | Bisphenol from plastics exposure leading to cardiovascular disease | High | 56 | High | High |
| | Bisphenol from plastics exposure leading to cognitive disorder | High | 47 | Low | Medium |
| | Bisphenol from plastics exposure leading to developmental injury | High | 341 | High | High |
| | Bisphenol from plastics exposure leading to endocrine & immune system injury | High | 279 | High | High |
| | Bisphenol from plastics exposure leading to lung injury | High | 21 | High | High |
| | Bisphenol from plastics exposure leading to nervous system injury | High | 118 | High | High |
| | Bisphenol from plastics exposure leading to reproductive injury | High | 395 | High | High |
| Flame retardants | Flame retardant from plastics exposure leading to cognitive disorder | High | 14 | Low | Medium |
| | Flame retardant from plastics exposure leading to developmental njury | High | 145 | High | High |
| | Flame retardant from plastics exposure leading to endocrine and immune system injury | High | 80 | Low | Medium |
| | Flame retardant from plastics exposure leading to kidney and liver injury | High | 42 | Low | Medium |
| | Flame retardant from plastics exposure leading to nervous system injury | High | 15 | Low | Medium |
| | Flame retardant from plastics exposure leading to reproductive injury | High | 94 | Low | Low |
| PFAS | PFAS from plastics exposure leading to bone or joint injury | High | 27 | Low | Medium |
| | PFAS from plastics exposure leading to cancer | High | 11 | Low | Medium |
| | PFAS from plastics exposure leading to cardiovascular disease | High | 126 | Low | Medium |
| | PFAS from plastics exposure leading to cognitive disorder | High | 10 | Low | Medium |
| | PFAS from plastics exposure leading to developmental injury | High | 192 | Low | High |
| | PFAS from plastics exposure leading to endocrine & immune system injury | High | 126 | Low | High |
| | PFAS from plastics exposure leading to kidney and liver injury | High | 217 | Low | High |
| | PFAS from plastics exposure leading to reproductive injury | High | 160 | Low | High |

| Source | Specific harm | Quality of publications | Cumulative number of net positive-association studies | Relevance of plastics as a potential pathway | Current scientific consensus |
|---|---|---|---|---|---|
| Phthalates | Phthalate from plastics exposure leading to cognitive disorder | High | 31 | Low | Medium |
| | Phthalate from plastics exposure leading to developmental injury | High | 410 | High | High |
| | Phthalate from plastics exposure leading to endocrine & immune system injury | High | 131 | High | High |
| | Phthalate from plastics exposure leading to kidney and liver injury | High | 83 | High | High |
| | Phthalate from plastics exposure leading to lung injury | High | 28 | Low | Medium |
| | Phthalate from plastics exposure leading to nervous system injury | High | 43 | Low | Medium |
| | Phthalate from plastics exposure leading to reproductive injury | High | 489 | High | High |
| Other chemicals | Unidentified or other chemicals exposure from plastics leading to significant human harms | High | 9 | Low | Low |
| MNP | MNP direct exposure leading to cardiovascular injury | Medium | 2 | High | Medium |
| | MNP direct exposure leading to developmental injury | Medium | 22 | High | Medium |
| | MNP direct exposure leading to endocrine & immune system injury | Medium | 64 | High | Medium |
| | MNP direct exposure leading to gastrointestinal injury | Medium | 35 | High | Medium |
| | MNP direct exposure leading to kidney and liver injury | Medium | 42 | High | Medium |
| | MNP direct exposure leading to lung injury | Medium | 9 | High | Mediium |
| | MNP direct exposure leading to nervous system injury | Medium | 34 | High | Medium |
| | MNP direct exposure leading to reproductive injury | Medium | 47 | High | Medium |
| MNP | MNP indirect delivery of other harmful agents (heavy metals, etc.) | Low | 0 | High | Medium |
| Macroplastics | Human harm from air pollution (PM2.5) from macroplastic production | High | Medium | High | High |
| | Human harm from burning of end-of-life macroplastics | High | Medium | High | High |

**Note:** See Box A3 for a worked example

## Economies & Ecosystem Services and Nature

**Table A5:** Detailed results of current scientific consensus of plastic harms to E&ES and Nature

| Source | Receptor | Grouped harm | Quality of publications | Volume of publications | Relevance of plastics as a potential pathway | Current scientific consensus |
|---|---|---|---|---|---|---|
| Chemical additives | E&ES | Harm to aquatic and terrestrial food sources from chemical additives | Medium<br>Lots of animal experimental studies, but no link directly to plastics[6,7] | Medium<br>Lots of studies on first-order effects but not on showing impact on economy | Low<br>Not the primary cause of economic loss plus lots of other sources for chemicals that cause harm to food sources | Medium |
| | Nature | Harm to aquatic and non-human terrestrial organisms from chemical additives | High<br>Lots of animal experimental studies | High<br>Lots of animal experimental studies | Medium<br>Some chemicals primarily from plastics, e.g. phthalates | High |
| Macroplastics | E&ES | Harm to aquatic and terrestrial food sources from macroplastics | Medium<br>Lots of animal experimental studies, but few on economic impact | Medium<br>Few academic studies on economic impact | High<br>Directly caused | Medium |
| | | Harm to tourism from macroplastics | Low<br>Only observational or anecdotal reports | Low<br>Very few academic studies | High<br>Directly caused | Low |
| | Nature | Harm to aquatic and non-human terrestrial organisms from macroplastics | High<br>Lots of high-quality observational studies | High<br>Lots of studies | High<br>Directly caused | High |
| MNP | E&ES | Harm to aquatic and terrestrial food sources from MNP (direct) | Medium<br>Animal experimental studies show various MNP harms | Medium<br>Lots of studies on first order effects but not on showing impact on economy | High<br>Directly caused | Medium |
| | | Harm to water sanitation from MNP (direct) | Medium<br>Biomonitoring studies confirm presence and concentration, but not economic impact | Medium<br>Studies not exhaustive | High<br>Directly caused | Medium |
| | | Harm to aquatic and terrestrial food sources from MNP (indirect) | Low<br>Animal experimental studies but with lots of confounders on real-world impact on overall stock and soil quality | Low<br>Some studies but few showing impact on economy | High<br>Directly caused | Low |
| | Nature | Harm to organisms from MNP (direct and indirect) | High<br>Animal experimental studies showing various MNP harms | High<br>Lots of studies | High<br>Directly caused | High |
| All plastics | E&ES | Harm to marine natural capital from all plastics | Medium<br>Some high-quality studies in specific areas, some lower quality studies on wider economic impact | Low<br>Very few showing impact on economy | Medium<br>Difficult to disentangle plastic-specific sources from non-plastic-specific sources | Low |
| $CO_2e$ emissions | All | Harm to all receptors from carbon emissions and climate change | High<br>Clear causal relationship between GHG emissions and climate change | High<br>Lots of studies | Medium<br>Plastic contribution is fairly well defined in terms of pathway to causation | High |

**Note:** See Box A4 for a worked example

## A4.3 Likelihood of scientific consensus remaining static

### Human Health

**Table A6:** Detailed results of future scientific consensus of plastic harms to Human Health

| Source | Specific harm | Stability (rate of change over 3 years in volume of publications) | Timeframe (number of years since first publication) | Likelihood of scientific consensus remaining static |
|---|---|---|---|---|
| Bisphenols | Bisphenol from plastics exposure leading to cardiovascular disease | 0% | 14 | High |
| | Bisphenol from plastics exposure leading to cognitive disorder | 0% | 23 | High |
| | Bisphenol from plastics exposure leading to developmental injury | 133% | 23 | Medium |
| | Bisphenol from plastics exposure leading to endocrine & immune system injury | 0% | 25 | High |
| | Bisphenol from plastics exposure leading to lung injury | 50% | 23 | High |
| | Bisphenol from plastics exposure leading to nervous system injury | 0% | 15 | High |
| | Bisphenol from plastics exposure leading to reproductive injury | -18% | 41 | High |
| Flame retardants | Flame retardant from plastics exposure leading to cognitive disorder | 0% | 16 | High |
| | Flame retardant from plastics exposure leading to developmental njury | -100% | 20 | High |
| | Flame retardant from plastics exposure leading to endocrine and immune system injury | 0% | 28 | High |
| | Flame retardant from plastics exposure leading to kidney and liver injury | 0% | 36 | High |
| | Flame retardant from plastics exposure leading to nervous system injury | 0% | 44 | High |
| | Flame retardant from plastics exposure leading to reproductive injury | -50% | 18 | High |
| PFAS | PFAS from plastics exposure leading to bone or joint injury | -60% | 11 | High |
| | PFAS from plastics exposure leading to cancer | 0% | 10 | High |
| | PFAS from plastics exposure leading to cardiovascular disease | -75% | 37 | High |
| | PFAS from plastics exposure leading to cognitive disorder | 0% | 11 | High |
| | PFAS from plastics exposure leading to developmental injury | 33% | 15 | Medium |
| | PFAS from plastics exposure leading to endocrine & immune system injury | -11% | 15 | High |
| | PFAS from plastics exposure leading to kidney and liver injury | -100% | 15 | High |
| | PFAS from plastics exposure leading to reproductive injury | 14% | 13 | Medium |

| Source | Specific harm | Stability (rate of change over 3 years in volume of publications) | Timeframe (number of years since first publication) | Likelihood of scientific consensus remaining static |
|---|---|---|---|---|
| Phthalates | Phthalate from plastics exposure leading to cognitive disorder | -33% | 24 | High |
| | Phthalate from plastics exposure leading to developmental injury | 143% | 42 | Medium |
| | Phthalate from plastics exposure leading to endocrine & immune system injury | 150% | 42 | Medium |
| | Phthalate from plastics exposure leading to kidney and liver injury | 0% | 46 | High |
| | Phthalate from plastics exposure leading to lung injury | 0% | 45 | High |
| | Phthalate from plastics exposure leading to nervous system injury | 0% | 33 | High |
| | Phthalate from plastics exposure leading to reproductive injury | 233% | 46 | Medium |
| Other chemicals | Unidentified or other chemicals exposure from plastics leading to significant human harms | 0% | 41 | High |
| MNP | MNP direct exposure leading to cardiovascular injury | 0% | 2 | Medium |
| | MNP direct exposure leading to developmental injury | 600% | 4 | Low |
| | MNP direct exposure leading to endocrine & immune system injury | 733% | 10 | Low |
| | MNP direct exposure leading to gastrointestinal injury | 433% | 6 | Low |
| | MNP direct exposure leading to kidney and liver injury | 600% | 6 | Low |
| | MNP direct exposure leading to lung injury | 0% | 3 | Medium |
| | MNP direct exposure leading to nervous system injury | 40% | 7 | Medium |
| | MNP direct exposure leading to reproductive injury | 500% | 9 | Low |
| MNP | MNP indirect delivery of other harmful agents (heavy metals, etc.) | 0% | 0 | Low |
| Macroplastics | Human harm from air pollution (PM2.5) from macroplastic production | High | High | High |
| | Human harm from burning of end-of-life macroplastics | High | High | High |

**Note:** See Box A5 for a worked example

## Economies & Ecosystem Services and Nature

**Table A7:** Detailed results of future scientific consensus of plastic harms to E&ES and Nature

| Source | Receptor | Grouped harm | Stability of publications | Timeframe since first publications | Likelihood of scientific consensus remaining static |
|---|---|---|---|---|---|
| Chemical additives | E&ES | Harm to aquatic and terrestrial food sources from chemical additives | Medium<br>Further evidence being gathered[8] | Medium<br>Research spans decades for animals; economic impact quite recent[8,9] | Medium |
| | Nature | Harm to aquatic and non-human terrestrial organisms from chemical additives | Medium<br>Further evidence being gathered[8] | High<br>Research spans decades[10] | High |
| Macroplastics | E&ES | Harm to aquatic and terrestrial food sources from macroplastics | Low<br>Lots of evidence being gathered[2] | Medium<br>Research spans decades for animals; economic impact studies quite recent[8,9] | Low |
| | | Harm to tourism from macroplastics | High<br>Not a specific, active area of research[8] | Low<br>Lack of empirical data | Medium |
| | Nature | Harm to aquatic and non-human terrestrial organisms from macroplastics | High<br>Lots of research establishing causation[11] | High<br>Research spans decades[12] | High |
| MNP | E&ES | Harm to aquatic and terrestrial food sources from MNP (direct) | Low<br>Further evidence being gathered[13] | Medium<br>Only past decade have studies started[14] | Low |
| | | Harm to water sanitation from MNP (direct) | Low<br>Lots of evidence on economic harm being gathered[13] | Medium<br>Only past decade have studies started[15] | Low |
| | | Harm to aquatic and terrestrial food sources from MNP (indirect) | Low<br>Lots of evidence being gathered[16] | Low<br>Very recent linking MNP as a vector of agents | Low |
| | Nature | Harm to organisms from MNP (direct and indirect) | Medium<br>Further evidence being gathered[13] | Medium<br>Only past decade have studies started[14] | Medium |
| All plastics | E&ES | Harm to marine natural capital from all plastics | Medium<br>Further evidence on economic harm being gathered[8] | Low<br>Lack of empirical data[17] | Medium |
| $CO_2e$ emissions | All | Harm to all receptors from carbon emissions and climate change | Medium<br>Lots more research coming out but not establishing causation - rather more on quantifying the impacts from different scenarios[2] | High<br>Research spans decades | High |

**Note:** See Box A6 for a worked example

# A5. GROUPING OF HARMS

Our specific harms were used to provide granular, comprehensive assessments where the data were available to do so. However, for many of our Economies & Ecosystem and Nature harms, granular data were unavailable. Therefore, we required less granular harms to carry out the assessment on the dimensions outlined above. We also needed to:

- develop a manageable, intuitive grouping of harms
- find common characteristics across specific harms in terms of the outputs of the assessment.

**Table 2** is the result of our grouping, along with an explanation of each.

**Table A5:** Group harms with justification

| Vector | Grouped harm | Explanation |
|---|---|---|
| Chemical additives | Human harm from bisphenols | Eight bisphenol harms to human health all have the same plastic-specific exposure routes |
| | | Cardiovascular disease and diabetes dominate, making up more than 70 per cent of expected harm |
| | | This justifies consolidating these eight harms into one |
| | Human harm from flame retardants | Six flame retardant harms to human health all have the same plastic-specific exposure routes |
| | | Developmental disorders in offspring and endocrine disorders dominate, making up almost 90 per cent of expected harm |
| | | This justifies consolidating these six harms into one |
| | Human harm from PFAS | Eight PFAS harms to human health have very few plastic-specific exposure routes |
| | | This lack of plastic specificity puts the specific harms at a low individual level of harm via plastic exposure |
| | | This justifies aggregating the impact of these eight harms into one |
| | Human harm from phthalates | Eight phthalate harms to human health have the same plastic-specific exposure routes |
| | | Developmental disorders in offspring and endocrine disorders dominate, making up more than 75 per cent of expected harm |
| | | This justifies consolidating these eight harms into one |
| | Human harm from unidentified or other chemicals | There are more than 4,000 chemical additives used in plastics for which there are no hazard classifications and a lack of data |
| | | While the effects of these chemicals are likely to be highly heterogeneous with different exposure routes to causing human harm, it is uncertain to what extent |
| | | This high uncertainty and unknown variability justify consolidating all chemicals outside the main classes known to cause harm into one group, on the shared basis that they lack information about effects |
| MNP | Human harm from MNP (direct) | Eight MNP direct harms to human health are likely to have similar exposure routes |
| | | While the science is only just emerging, digestive disorders (including GI and kidney and liver injuries) are posited to dominate, making up more than 70 per cent of expected harm |
| | | This justifies consolidating these eight harms into one |
| | Human harm from MNP (indirect) | Given the lack of knowledge around the indirect harms posed by MNP, we identified this as one specific harm mapping onto one grouped harm |
| Macroplastics | Human harm from burning (end-of-life macroplastics) | Five end-of-life-mediated macroplastic harms to human health all have the same exposure route |
| | | Given the localised effects of most of these harms, they are likely to cause harms in the same exposed population |
| | | This justifies consolidating these five harms into one |
| | Human harm from air pollution (macroplastics production) | Two production-mediated macroplastic harms to human health have the same exposure route |
| | | Particulate matter release and contamination of the environment are likely to cause similar harms in similar exposed populations |
| | | This justifies consolidating these two harms into one |

| Vector | Grouped harm | Explanation |
|---|---|---|
| Chemical additives | Harm to food sources from chemical additives | Eight phthalate harms to human health have the same plastic-specific exposure routes |
| | | Developmental disorders in offspring and endocrine disorders dominate, making up more than 75 per cent of expected harm |
| | | This justifies consolidating these eight harms into one |
| Macroplastics | Harm to food sources from macroplastics | Macroplastics harm is likely to be similarly small across terrestrial and aquatic food sources |
| | | Therefore, we consolidated several exposure pathways – including entanglement and ingestion by livestock or fish stock for example – positing that distinguishing between the harms would not affect our assessment |
| | Harm to tourism from macroplastics | Many of macroplastic's harms to tourism share the same exposure pathways as its harms to food sources |
| | | However, the exposed populations are very different, with non-food megafauna and landscapes with visual amenity value affected |
| | | This justifies separating these two harms |
| MNP | Harm to food sources fro m MNP (direct) | MNP harms are likely to be similarly sized and have similar consensuses across terrestrial and aquatic food sources |
| | | Therefore, we consolidated several exposure pathways – including, soil degradation and ingestion by livestock or fish stock for example – positing that distinguishing between the harms would not affect our assessment |
| | Harm to water sanitation from MNP (direct) | This was considered a harm in and of itself |
| | Harm to food sources from MNP (indirect) | Likewise, with the direct effects of MNP, we considered the harms from the indirect effects to be similarly sized and have similar consensuses |
| | | Therefore, we consolidated several exposure pathways |
| All plastics | Harm to marine natural capital from all plastics | This harm was consolidated from several sources and pathways for which there were insufficient evidence to derive consensuses on the economic impact of their first-order effects |
| Chemical additives | Harm to organisms from chemical additives | Chemical additive harms are likely to have similar consensus and size across plants and animals |
| | | Therefore, we consolidated these harms, positing that distinguishing between the harms would not affect our assessment |
| Macroplastics | Harm to organisms from macroplastics | The harms to plants and animals from macroplastics are similarly sized and all have strong consensus on causation |
| | | Therefore, we consolidated these harms, positing that distinguishing between the harms would not affect our assessment |
| MNP | Harm to organisms from MNP | The harms to plants and animals from MNP are similarly sized and most have medium-strong consensus on causation |
| | | Therefore, we consolidated these harms, positing that distinguishing between the harms would not affect our assessment |
| $CO_2e$ emissions | Carbon emissions and climate change | Climate change harms are not unique to plastics |
| | | Therefore, we grouped many of the specific climate change harms from production and disposal under one grouped harm: harm from carbon emissions and climate change. |

**KEY:**

Harms to Human Health

Harms to Economies & Ecosystem Services

Harms to Nature

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