

# PLASTICS, HEALTH, AND ONE PLANET

AN EVIDENCE-BASED CALL FOR GLOBAL RULES



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In partnership with



UNIVERSITY OF  
BIRMINGHAM



Birmingham Institute  
for Sustainability  
and Climate Action

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MICRO- AND NANOPLASTICS AND THE CHEMICALS THEY CONTAIN ARE FOUND IN AIR, WATER, AND FOOD—POSING SERIOUS RISKS TO HUMAN, ANIMAL, AND ENVIRONMENTAL HEALTH. STRONG GLOBAL RULES ARE URGENTLY NEEDED TO TACKLE THIS GROWING CRISIS.



# 1. EXECUTIVE SUMMARY

Plastic pollution is not only an environmental issue—it is a growing global health crisis. Plastics are leaking into and harming water and land-based ecosystems. As a result, micro- and nanoplastics (MnPs) are now entering our bodies too, exposing people and animals everywhere to potentially serious health risks. They are found in the air we breathe, the water we drink, and the food we eat. The potential negative impacts on major organ systems of the human body are wide-ranging and long-lasting, with evidence of their severity continuing to emerge.

This pollution by microscopic plastic particles is not only a physical threat, it is also a chemical one. In addition to the plastic particles themselves, toxic additives and other chemicals added to plastics are increasingly linked to a wide range of health impacts, from infertility and cancer to respiratory conditions, cardiovascular disease, and impaired childhood development.

Understanding the health impacts of plastic pollution requires a *One Health* approach, which recognises the deep interconnections between human, animal, and environmental health.

These harms are not confined to humans, but are mirrored across ecosystems, where plastic pollutants and their associated chemicals are harming wildlife and livestock, triggering biological disruptions through similar mechanisms to those seen in humans. Understanding the health impacts of plastic pollution requires a *One Health* approach, which recognises the deep interconnections between human, animal, and environmental health. Studies on both humans and animals have been instrumental in revealing how plastics can cause harm, underscoring that plastic pollution is a systemic threat to the health of all living beings and the ecosystems we share.

Global and national regulations have yet to catch up with the rapidly emerging scientific evidence. While research continues to evolve, several studies already show consistent and concerning links between many plastic additives and serious health effects. Some of the most concerning include endocrine-disrupting chemicals like phthalates and bisphenols, which interfere with reproductive hormones and brain development, and PFAS (Per- and Polyfluoroalkyl Substances)—known as “forever chemicals”—that are linked to cancer, immune suppression, and metabolic disease. These risks are especially concerning during pregnancy and childhood, where early exposure may lead to lasting or even multigenerational health effects.

This growing body of evidence must serve as a foundation for strong, science-based legislation to minimise exposure to MnPs and the toxic chemicals currently found in many plastics. Adopting the precautionary principle—taking action where credible risks are identified, even in the absence of absolute scientific certainty—is essential to avoid long-term harm. The Montreal Protocol offers a powerful precedent: by phasing out ozone-depleting substances (chlorofluorocarbons or CFCs), based on emerging scientific evidence, governments prevented millions of cases of skin cancer<sup>1</sup> and facilitated the restoration of the ozone layer.



Plastic pollution transcends borders. The production, use, and disposal of plastics form a global value chain, and the resulting pollution, including MnPs and plastic-related chemicals of concern, spans continents.

Tackling this complex and interconnected crisis requires coordinated global action: no single country can solve it alone. Given the pervasive presence of plastics in our daily lives and widespread plastic pollution, global action—grounded in the latest scientific research and a *One Health* approach—is urgently needed. Such an approach can provide a globally-harmonised response to address the plastic crisis, not only protecting our ecosystems and biodiversity but also greatly reducing health risks now and in the future.

As momentum builds for a much-needed and legally binding global treaty to end plastic pollution, it is vital that health impacts are front and centre of the negotiations. WWF urges governments and negotiators to seize this moment and deliver an effective and ambitious global plastics treaty that protects both people and the planet. Governments must agree on a treaty that delivers real change. This means securing global bans and phase-outs of the most harmful and avoidable plastic products and the chemicals of concern they contain.

The longer we delay, the higher the costs. A treaty that tackles plastic pollution at its source is not only environmentally essential, but a public health imperative. Now is the time for bold, coordinated global action.



### AT A MINIMUM, THE TREATY MUST INCLUDE:



**1. Global bans and phase-outs** of the most harmful and avoidable plastic products and chemicals of concern



**2. Harmonised requirements for safe and circular plastic product design** and enabling systems for a non-toxic circular economy in practice and at scale



**3. Measures to align financial flows and mobilise resources** for an equitable and just transition



**4. Mechanisms to strengthen and adapt measures over time**



### ABOUT THIS PAPER

This paper was developed in partnership with the University of Birmingham, combining scientific and policy expertise. The University of Birmingham led on the research and evidence review, with WWF contributing strategic direction and policy framing relevant to global efforts to end plastic pollution.

It is based on a comprehensive review of the latest scientific evidence on plastics and health. It draws on a wide array of peer-reviewed research papers and scientific resources—from human and animal studies to systematic reviews, observational studies, and expert analyses, as well as insights from respected organisations such as the World Health Organization and the Endocrine Society.

The evidence base on micro- and nanoplastics (MnPs) and plastic-related chemicals of concern is rapidly evolving. Early research on plastics and human health focused largely on occupation exposures, particularly the effects of additives on worker health and safety. Since then, scientific attention has broadened significantly. In recent decades, there has been a steep rise in studies investigating the health impacts of microplastics and plastic-associated chemicals in the general population. Many of these studies, particularly in the field of ecotoxicology, have used concentrations that are relatively high in order to identify potential mechanisms of harm. However, there is a growing body of work that is beginning to use environmentally realistic concentrations. At present, determining what constitutes ‘realistic’ exposures remains challenging. With monitoring still being inconsistent and incomplete, the true variability of environmental MnP concentrations and resulting peaks in human exposures remain largely unknown.<sup>2,3</sup>

Proving causality in public health science is rarely straightforward and plastics are no exception. Nearly all people are now exposed to MnPs and plastic additives, making it difficult to identify unexposed control groups or clear baselines for assessing health impacts. This complexity means



that researchers rely on a combination of methods, including animal models, statistical correlation, and observational studies, to build a comprehensive picture of the risks.

While this paper reflects the current state of scientific knowledge, it is not an exhaustive review. But despite these limitations, the prevailing conclusions of studies across disciplines and geographies provide a compelling basis for precautionary policy action.

The aim of this paper is to distil complex, evolving science into clear insights for policymakers, with a focus on the health impacts of MnPs and six key groups of chemicals commonly used in plastic products. It outlines how these substances enter the human body, the direct health implications and biological mechanisms through which they cause harm, and the wider risks to animal and environmental health. The conclusion outlines policy recommendations based on the weight of current scientific understanding.



## 2. INTRODUCTION

Due to the widespread use of plastics, MnPs are now almost omnipresent, found across every part of the planet, from oceans and mountains to air, and indoor environments, food, and drinking water.

Plastics are deeply embedded in modern life, but their increasing unintended presence in our environment and bodies is a cause of growing concern. Plastic pollution is not only damaging the environment, it is increasingly recognised as a serious threat to both human and ecosystem health. Due to the widespread use of plastics, MnPs are now almost omnipresent, found across every part of the planet, from oceans and mountains<sup>4</sup> to air<sup>5, 6</sup> and indoor environments,<sup>7, 8</sup> food, and drinking water.<sup>9, 10, 11, 12</sup> From microplastics found in our food and water, to toxic additives leaching from plastic products, the health implications are becoming increasingly clear.

Exposure to plastic pollution is largely involuntary and nearly universal. Micro- and nanoplastics can enter the human body through actions as unavoidable as breathing air and drinking water. A vast legacy of plastic pollution has already accumulated in the environment over decades, steadily compounding exposure levels and deepening the risks to human and ecological health.

This paper brings together the latest science to examine how exposure to MnPs and associated chemicals affects human, animal, and environmental health. It traces how these particles and chemicals enter the body, their origins, and what happens once they are inside—highlighting the most concerning health effects and setting out clear policy recommendations for action. A detailed Technical Appendix provides an in-depth review of the health evidence for six major groups of chemicals of concern related to plastics. While many of these chemicals are not exclusive to plastics, plastics serve as a major and growing source of exposure due to their widespread use, persistence in the environment,<sup>13</sup> and capacity to be transported through air, oceans, and other cross-border pathways.



Reducing plastic pollution is therefore central to reducing the overall toxic burden on people and the planet and mitigating its far-reaching health and ecological consequences. To fully understand these risks, this paper takes a *One Health* research approach, looking at human and animal health through an integrative framework that recognises the multiplicity of ways in which these factors are interconnected. Due to these interconnections, animal studies have helped clarify how plastic exposure may impact human health, and ecosystem findings help us understand how and why this exposure occurs.

### WHAT IS ONE HEALTH, AND WHY DOES IT MATTER?

One Health<sup>14</sup> is an integrated, unifying concept that aims to sustainably balance and optimise the health of people, animals, and ecosystems. In its approach, it acknowledges that the health of humans, domestic and wild animals, and the environment are closely linked and interdependent.

Conservation efforts targeted at this intersection have the ability to improve human health by strengthening wildlife and ecosystem resilience. A One Health approach can prevent, predict, detect, and respond to global health threats and can enable the development of new ideas that address root causes and create long-term, sustainable solutions. It is therefore particularly well-suited to addressing the interconnected challenges of plastic pollution and its health impacts.

This approach is gaining traction in global policy frameworks. For example, the Convention on Biological Diversity (CBD) explicitly incorporates One Health in the Kunming-Montreal Global Biodiversity Framework and its Global Action Plan on Biodiversity and Health, adopted at COP 16, setting an important precedent for its use in future international environmental agreements.

While this paper focuses intentionally on the risks posed by MnPs and plastic additives, it is important to acknowledge that plastics pose health hazards across their entire lifecycle. From fossil fuel extraction and chemical production to manufacturing, use, and disposal, each stage carries potential harm to both people and the environment, particularly for workers and frontline communities exposed to higher levels of pollution.

At the same time, it is important to recognise that some plastic products offer crucial benefits in a number of contexts, particularly in medicine and safety. This paper does not argue for blanket bans, but aims to highlight the complexity and encourages a more targeted approach to eliminate the plastic products and chemicals with high risks of harms to human health and the environment.



An aerial photograph showing a polluted waterway. A large, dense pile of plastic waste, including bottles, bags, and other debris, is visible on the left side of the water. The water is murky brown. To the right of the water, there are some small structures and more debris. The background shows a mix of green vegetation and some buildings. The overall scene depicts environmental pollution and its impact on water quality.

PLASTIC POLLUTION IS INCREASINGLY LINKED TO WIDE-RANGING HEALTH EFFECTS—FROM CELL DAMAGE TO HORMONAL DISRUPTION. A ONE HEALTH APPROACH HELPS EXPLAIN THESE INTERCONNECTED IMPACTS AND WHY PROTECTING HUMAN HEALTH REQUIRES SYSTEMIC SOLUTIONS.



### 3. UNDERSTANDING THE HEALTH RISKS OF PLASTIC POLLUTION

Scientific evidence is rapidly accumulating on the serious and wide-ranging health risks posed by plastic pollution. These harms stem from two interrelated but distinct sources: MnPs and the hazardous chemicals added to or associated with plastic materials. This section explores the latest science, looking at the major potential health risks that have been linked to each.

### 3.1 HOW MICROPLASTICS ENTER THE ENVIRONMENT

Microplastics are generally defined as plastic fragments smaller than 5 millimetres, while nanoplastics are typically less than 1 micrometre (1/1000<sup>th</sup> of a mm).<sup>15</sup> The majority of MnPs found in the environment are secondary particles, formed as larger plastic products break down, degrade, or fragment over time. Everyday products—from packaging and textiles to carpets and furniture<sup>16, 17, 18</sup>—shed MnPs into the environment throughout their lifecycle. In some cases, primary MnPs, manufactured at microscopic scales, are intentionally added to consumer products, such as cosmetics—a practice that is increasingly being restricted through emerging regulations.

**FIGURE 1:**  
**Anatomy of Plastic: Selection of**  
**the most harmful plastic-related**  
**additives and chemicals of concern.**

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## Per- and polyfluoroalkyl substances (PFAS)

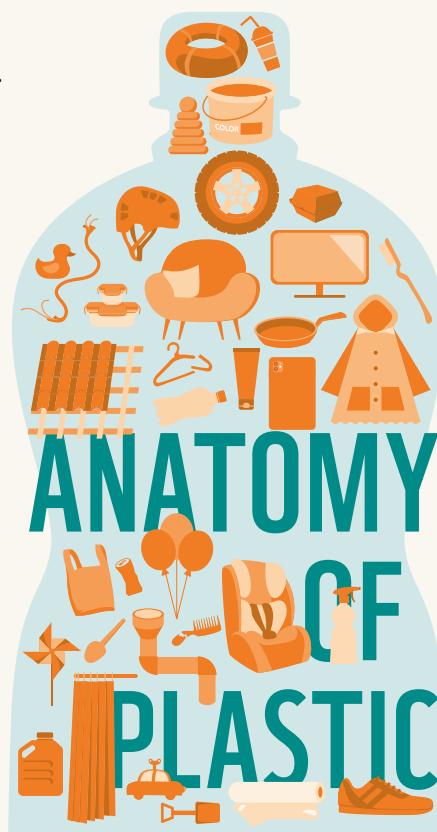
Substances added for water and stain resistance, which persist in nature and pollute water and soil.

## Flame retardants

Chemicals added to products to reduce the flammability of the desired materials that can accumulate in the environment and human body.

## Metals, metalloids, and metal compounds

Additives including lead, cadmium, and antimony that boost colour and heat resistance but can pollute soil and water, harming wildlife and humans.



## Micro- and nanoplastics

Tiny plastic particles that pollute ecosystems and can enter living organisms.

## Phthalates

Chemical additives used to give plastics desired properties, such as flexibility, that can leach into the environment and may disrupt human hormones.

## Bisphenols

Chemicals (including BPA and BPS) used in plastic production that may interfere with hormone function.

## UV stabilisers and alkylphenols

Additives that help plastic resist sunlight and can be toxic to wildlife and humans.



## 3.2 PLASTIC POLLUTION'S ECOLOGICAL TOLL

Micro- and nanoplastics are already causing widespread harm to nature. In marine environments, MnP pollution is a well-documented threat, found in the guts of marine mammals, seabirds, turtles,<sup>19</sup> and fish.<sup>20</sup> Marine animals often mistake plastic for food—a study found evidence that over 56% of marine animals and 40% of seabirds have ingested plastic, which likely releases MnPs in their bodies, while smaller organisms like plankton can ingest MnPs alongside the tiny particles they consume for food.<sup>21, 22</sup> Once ingested, MnPs can travel through other parts of the body and transfer through food webs, with numerous health consequences, including changes in food uptake, physiological impacts, behavioural change, and mortality.<sup>23</sup>

**Marine animals often mistake plastic for food—a study found evidence that over 56% of marine animals and 40% of seabirds have ingested plastic, which likely releases MnPs in their bodies.**

On land too, the risks are becoming increasingly clear. MnPs disrupt the health and behaviour of soil organisms that are essential to ecosystem functioning and food production. Studies have shown that polyethylene (PE) microplastics can damage the immune system of earthworms, polyvinyl chloride (PVC) microplastics restrict the movement of springtails (insect-like creatures that play a key role in breaking down organic matter in soil), and polyethylene terephthalate (PET) particles cause oxidative damage in roundworms.<sup>24</sup> All these organisms play vital roles in soil health and represent key parts of the food chain,<sup>25</sup> meaning the ripple effects of plastic pollution extend deep into food systems and ecosystem functioning.

Plastics can also release harmful chemicals into the environment, with serious implications for wildlife and ecosystem functioning. Flame retardants, which often come from plastics, have been detected in polar bears, chimpanzees, and red pandas.<sup>26</sup> Documented impacts of this particular group of chemicals include thyroid dysfunction in polar bears, altered heart and brain size in American kestrels, and reproductive and immune impacts in orcas.<sup>27</sup> Even microorganisms are affected. *Prochlorococcus*—the ocean's most abundant photosynthetic bacteria and a major oxygen producer<sup>28</sup>—shows reduced growth and photosynthetic abilities<sup>29</sup> when exposed to plastic-derived chemicals. Disrupting these microbes therefore risks destabilising the global carbon and oxygen cycles, with cascading effects for planetary health.

FIGURE 2:

Chemicals and micro- and nanoplastics enter nature and the human body through various pathways and are mostly invisible to the eye



# SILENT SPREAD:

How plastic threatens human and environmental health





## 3.3 PLASTICS AND HUMAN HEALTH

It is becoming increasingly clear that the same plastics that harm wildlife are also a threat to human health. MnPs, along with the chemicals added to plastics during manufacture, are being found in human bodies, from our lungs to our bloodstreams. The consequences can be significant.

Studies of the toxicological impacts of plastic-related substances such as bisphenols, phthalates, and flame retardants have linked these chemicals to a range of illnesses, including:

- hormone-related cancers (including breast and testicular)
- reduced fertility and reproductive disorders
- chronic respiratory conditions such as asthma
- cardiovascular diseases such as heart disease and stroke
- metabolic conditions such as diabetes and obesity
- neurological conditions including ADHD, autism, and dementia

These wide-ranging effects stem from the ways plastic particles and their chemical additives interact with the body. For example, plastic particles can carry chemical additives, increasing their bioavailability and making it easier for them to enter cells and tissues.

The following sections explore these two main sources of harm: first, the physical effects of plastic particles themselves; and second, the health risks linked to the chemicals used in plastics.



#### INVISIBLE HARM: HOW MICROPLASTICS AFFECT THE BODY

Once released into the environment, MnPs can be inhaled, ingested, or enter the skin.<sup>30</sup> Inside the body, they can cause substantial harm.

Firstly, as biologically foreign and non-biodegradable materials, MnPs can provoke inflammatory responses, trigger dysregulated immune responses, disrupt cellular function, and damage tissues<sup>31</sup> simply through their physical presence. Nanoplastics are especially concerning, because of their ability to cross biological barriers, penetrate deeply into tissues and organs, and accumulate in the body. These particles have been detected in vital organs such as the lungs, brain, intestine, and placenta, as well as in blood, breast milk, and stool.<sup>32, 33, 34</sup>

Inhalation of MnPs can irritate and inflame the airways and lungs, contributing to respiratory problems and aggravating existing conditions like asthma or chronic obstructive pulmonary disease (COPD).<sup>35</sup> Emerging evidence suggests that they can also accumulate in the arteries, exacerbating inflammation<sup>36, 37</sup> and potentially increasing the risk of severe cardiovascular diseases such as heart attack (myocardial infarction), stroke,<sup>38</sup> and atherosclerosis, a chronic inflammatory disease caused by a buildup of fatty material in the arteries. Studies in both humans and mice have demonstrated that nanoplastics, such as polystyrene (PS), can trigger inflammation<sup>39</sup> and even cause cell death.<sup>40</sup>

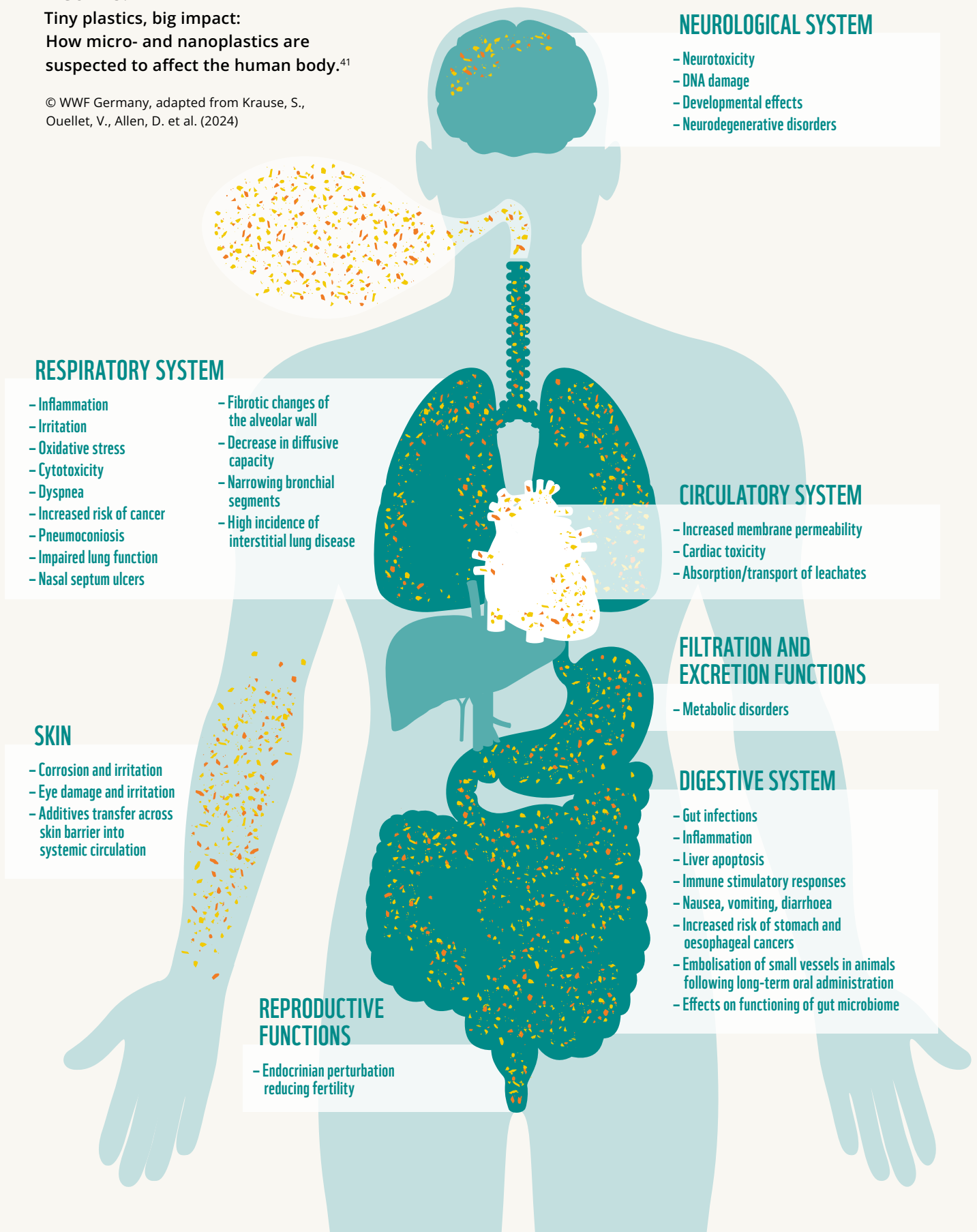




FIGURE 3:

**Tiny plastics, big impact:**  
How micro- and nanoplastics are suspected to affect the human body.<sup>41</sup>

© WWF Germany, adapted from Krause, S., Ouellet, V., Allen, D. et al. (2024)



In the digestive system, microplastics have been found in human faeces and colorectal cancer tissue,<sup>42, 43</sup> raising concerns about their role in gastrointestinal inflammation and tumour development. The presence of plastic particles in human brains<sup>44</sup> indicates that they can cross into the bloodstream and reach the brain. Notably, higher levels of MnPs have been found in dementia patients' brains, suggesting potential links to neurodegenerative diseases—a pattern also observed with other ultrafine particles, such as those from vehicle combustion, which are known to cross the blood-brain barrier and contribute to cognitive decline.

Secondly, plastic particles can carry harmful microbes, serving as vectors for pathogens and drivers of antimicrobial resistance. The surfaces of many plastic particles provide ideal conditions for microbial colonisation, allowing fungi, algae, and bacteria to accumulate. These plastic particles then effectively function as floating reservoirs of potential infection, especially when consumed by humans or animals.<sup>45</sup>

Even more concerning is their role in accelerating antimicrobial resistance, one of the most urgent public health threats worldwide. A recent lab-based study suggests that certain MnPs can facilitate the transfer of antibiotic resistant genes between bacteria—a process known as horizontal gene transfer—increasing the spread of antibiotic resistance by up to 200 times.<sup>46</sup>

This potentially undermines the effectiveness of antibiotics, fuels the rise of resistant superbugs, and erodes the efficacy of life-saving medicines, although more evidence is needed to confirm these effects.

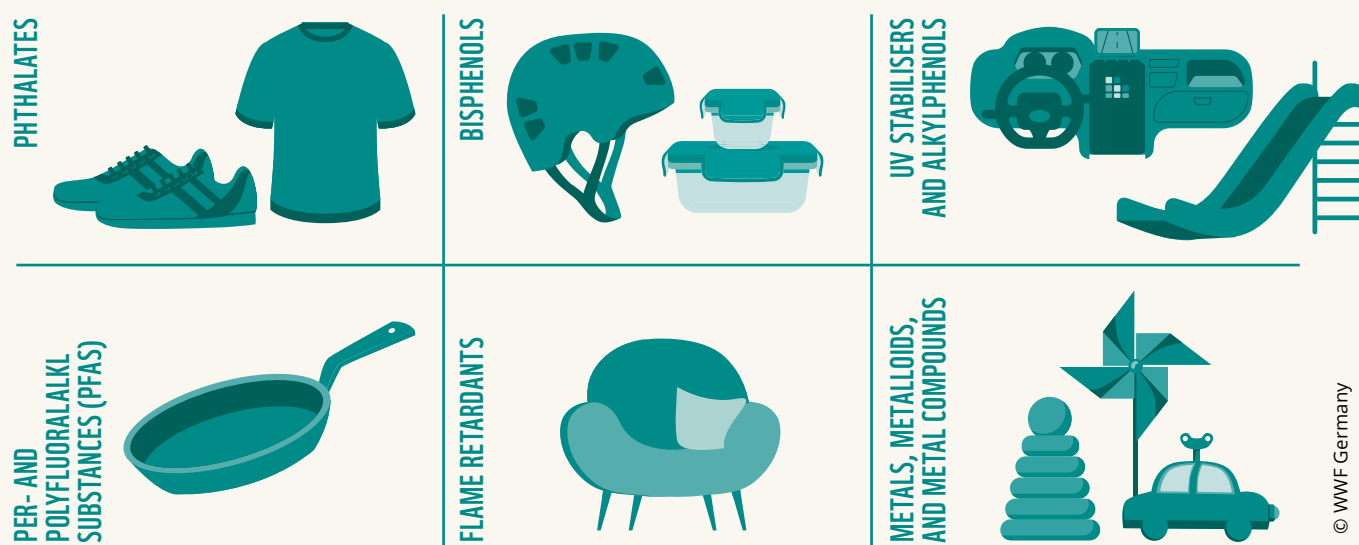
Thirdly, MnPs can transport and release a cocktail of toxic chemicals and additives that interfere with biological processes. The next section explores these chemical processes in more detail, highlighting how they interact with the body, along with the risks they pose to human health.





FIGURE 4 :

Plastics are everywhere and carry toxic chemicals that harm humans and wildlife



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#### INSIDE THE BODY: HOW PLASTIC-RELATED CHEMICALS OF CONCERN CAUSE HARM

Most plastics contain a complex mix of additives, often introduced during production to give plastics specific properties, such as flexibility, durability, or resistance to fire and UV light. Many of these additives are known to be harmful, including chemicals in the six following groups which are commonly found in many plastic products. Not all plastics contain all the chemicals discussed in this list, but these examples illustrate the varied use of harmful chemicals of concern across the plastic economy. Although it is not always possible to prove that the presence of these chemicals in the human body comes exclusively from plastics, their widespread use in plastic products, combined with their detection in human tissues, makes plastics a likely and preventable source of exposure.

- **Phthalates**—commonly used to soften PVC in product like vinyl flooring, shower curtains, and medical tubing
- **Bisphenols**—found in products such as food and drink packaging, thermal paper receipts, and reusable water bottles
- **UV stabilisers and alkylphenols**—used in outdoor furniture, automotive plastics, and packaging to prevent degradation from sunlight
- **Per- and polyfluoroalkyl substances (PFAS)**—found in grease-resistant food packaging, non-stick cookware, and waterproof clothing
- **Flame retardants**—widely used in textiles, electronics, and building insulation materials
- **Metals, metalloids, and metal compounds**—used as stabilisers, pigments or catalysts in plastic production and found in products like PVC pipes, toys, and synthetic turf

Critically, some of these chemicals—such as Bisphenol A and phthalates—can leach into the environment,<sup>47</sup> increasing the risk of human exposure. These chemicals can then be released over time and interfere with human biology in multiple interconnected ways,<sup>48, 49, 50, 51</sup> disruptions that are linked to a wide range of illnesses and health conditions.<sup>52</sup> In addition, MnPs can act as carriers for these chemicals, transporting them into the body. In some cases they can “smuggle” harmful substances past the body’s natural defences and carry them deep into tissues, cells, and organs, a mechanism known as the “Trojan Horse Effect”.<sup>53, 54, 55</sup>

**MnPs can “smuggle” harmful substances past the body’s natural defences and carry them deep into tissues, cells, and organs.**

One of the most well-documented health effects induced by plastic-related chemicals of concern is endocrine disruption. Many of the additives found in plastics—particularly phthalates and bisphenols—are endocrine-disrupting compounds (EDCs), which interfere with the body’s finely tuned hormone system that regulates functions such as growth, reproduction, metabolism, and body weight.<sup>56</sup> These chemicals can mimic natural hormones like oestrogen and testosterone, binding to receptors and disrupting normal hormonal signalling.<sup>57</sup> Endocrine disruption can contribute to long-term problems, including fertility issues, premature puberty, thyroid disorders,<sup>58</sup> and hormone-sensitive cancers such as breast cancer.<sup>59</sup> These chemicals are also linked to metabolic conditions such as diabetes and obesity.<sup>60, 61</sup>

The same chemicals also pose risks to brain development. By disrupting neurotransmitter systems and key developmental hormones, they can impair how the brain forms and functions. Phthalates, for example, have been shown to interfere with neurotransmitter systems during critical phases of brain development. The consequences can be reduced grey matter volume, impaired cognitive and motor function, and lower IQ.<sup>62, 63</sup> These impacts are especially concerning during the prenatal period and early childhood, when the brain is developing rapidly. The exposure of children to bisphenols in crucial development periods has been shown to contribute to the emergence of serious neurological disorders like attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorder (ASD), depression, and anxiety.<sup>64</sup>

MnPs can also cause harm by triggering oxidative stress. Some plastic-related chemicals of concern can increase the production of reactive oxygen species (ROS) or free radicals—unstable molecules that can react easily with other molecules in a cell.<sup>65</sup> While ROS have normal biological functions and are managed by the body using antioxidants, excessive production impairs their balance, leading to oxidative stress. This can damage DNA and other molecules in the cell and promote aging and disease.<sup>66, 67</sup> For example, phthalates have been shown to interfere with antioxidant activity, tipping the balance in favour of oxidative stress.<sup>68</sup>



There is a need for developing joined-up solutions that protect public health, safeguard biodiversity, and support planetary resilience, recognising the deep links between human, animal, and environmental health.

In addition, several of these chemicals interfere with how genes work, affecting whether they are switched on or off, which leads to long-term health effects that may be passed on to future generations.<sup>69</sup> This kind of epigenetic modification can change how the body develops or functions, especially during early life. For instance, prenatal exposure to Bisphenol A can interfere with a gene involved in brain function, which increases the risk of neurodevelopmental disorders, particularly in girls.<sup>70</sup>

These plastic-related chemicals of concern can also disrupt the immune system—the body’s defense against disease and harmful substances—by interfering with cytokines, signalling substances which coordinate the immune response. Inappropriate immune response signalling can lead to chronic inflammation, a persistent state of immune activation that has been linked to a wide range of conditions, from respiratory diseases to cancer.<sup>71</sup>

Many of these disease mechanisms are interconnected. Endocrine-disrupting chemicals can alter gene expression,<sup>72</sup> and altered expression of inflammatory genes influences inflammation.<sup>73</sup> Oxidative stress leads to DNA damage, which can trigger inflammation and subsequent diseases.<sup>74</sup> This cumulative effect can increase the overall risk of a wide range of chronic diseases, underscoring the urgency of precautionary action to reduce exposure to plastic-related chemicals.

More detailed evidence is presented in the *Technical Appendix*, where the risks posed to people and wildlife of six key chemical groups are explored in depth, but the science is increasingly clear: MnPs and plastic-related chemicals can impact human—and environmental—health through multiple biological pathways. These findings underscore the need to view plastic pollution through a *One Health* lens, not only to understand the full scope of harm, but for developing joined-up solutions that protect public health, safeguard biodiversity, and support planetary resilience, recognising the deep links between human, animal, and environmental health.

While research will continue to evolve, a substantial body of evidence already exists that points to credible and consistent harm. The time for action is now. The next section will outline how this evidence can and must inform ambitious, precautionary policy measures to address the plastics crisis at its root.

## HAZARD VS RISK: WHAT'S THE DIFFERENCE?

A **hazard** is something that has the potential to cause harm, such as a chemical or microplastic that, under certain conditions, can affect human or environmental health.

A **risk** is the likelihood that harm will actually occur and its severity, based on how much, how often, and in what way people (or other organisms) are exposed to that hazard.

For example, a chemical may be hazardous at high concentrations, but the risk to people or the environment is dependent on how much they are exposed to and in what way.

Scientific studies often use higher concentrations to understand the potential for harm and how it occurs. These studies help identify and characterise hazards, however they don't always reflect real-world conditions, where exposures are lower but may still be chronic and widespread. While exact exposure levels are still difficult to measure, there is still growing concern that long-term, low-level exposure to MnPs and associated chemicals of concern could contribute to serious health effects.







SCIENTIFIC CONCERNS ARE GROWING—AND SO IS THE  
NEED FOR ACTION. A STRONG UN PLASTIC TREATY  
WITH GLOBAL RULES IS ESSENTIAL TO REDUCE  
EXPOSURE AND PREVENT LONG-TERM HEALTH HARMS.

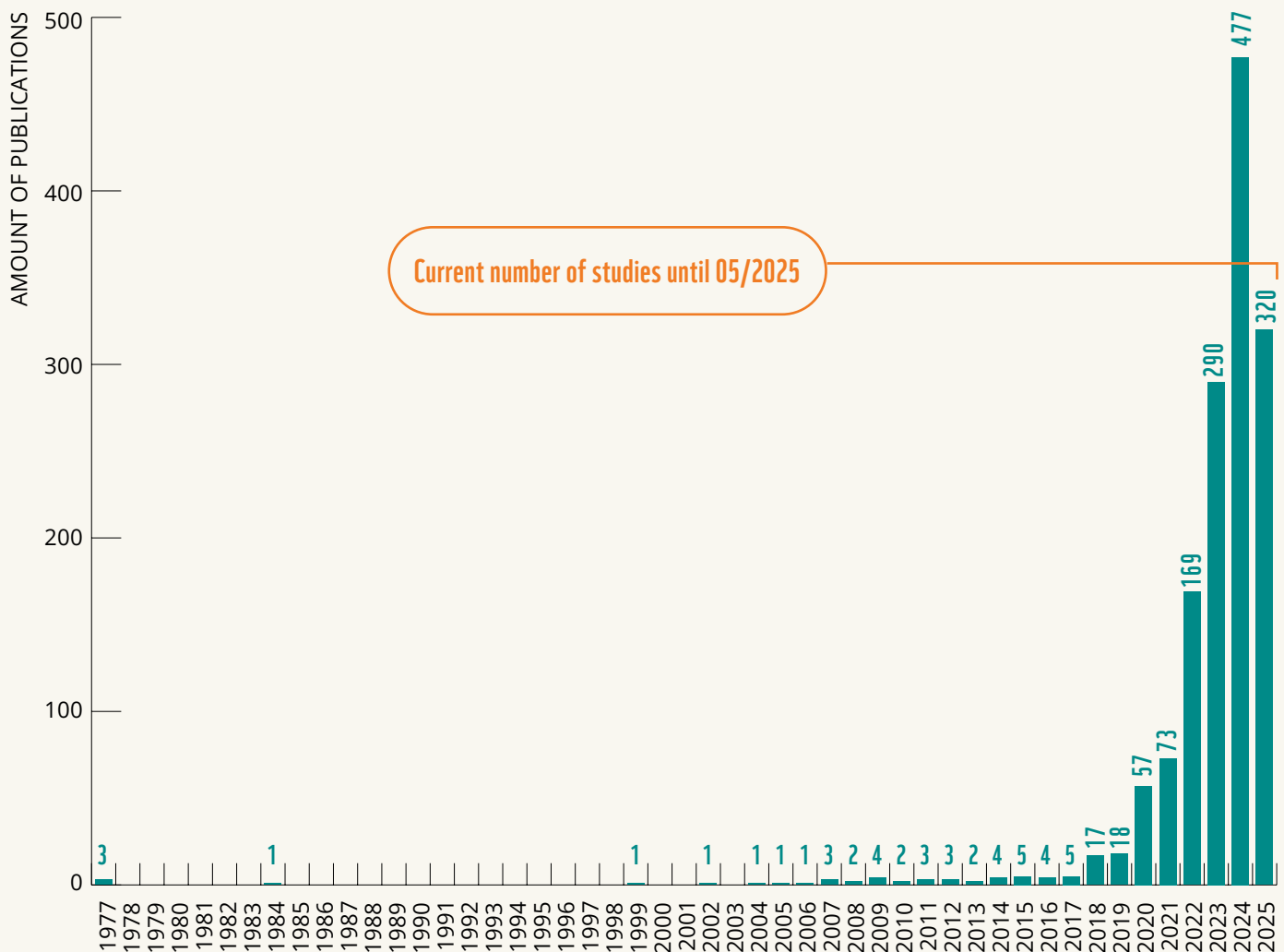


## 4. FROM EVIDENCE TO ACTION: POLICY PRIORITIES FOR CHANGE

The scientific evidence is now substantial. Thousands of peer-review studies point to more and more consistent and troubling associations. The volume of research in this field has increased dramatically in recent years—with almost 1,500 studies published since 2018 alone—a reflection not just of mounting concern, but of the increasing availability of data and technological capacity to trace these harms. This growing body of evidence demands an equally ambitious political and regulatory response.

FIGURE 5:  
**Number of studies identifying the impact of micro- and nanoplastics on human health between 1977 until May 2025**

Studies demonstrating the impact of micro and nanoplastics on human health have increased over the last decades (and since the first WHO report on human health risks from 2019). A total of 1,467 studies were identified (05/2025) in a search performed using Scopus and individually combining the keywords “microplastics” or “nanoplastics” with “human health”, public health, lung, heart, gut, brain, and cancer.



© WWF Germany, Source: Analysis by University of Birmingham



### THE NEED FOR DECISIVE, EFFECTIVE, AND PRECAUTIONARY GLOBAL POLICY

While scientific evidence will continue to evolve, the risks are credible and the stakes are high. Exposure to plastic pollutants is near-universal and largely involuntary. The health impacts are not limited to humans. MnPs and the toxic chemicals contained in plastics may also damage wildlife and ecosystems. And because many of the effects (like cancers and fertility disruption) can take years to emerge, there is a high risk of long-term and potentially irreversible consequences if action is delayed.

In such contexts, the precautionary principle<sup>75</sup> becomes essential. This well-established principle is a foundational tenet of environmental and public health law, and asserts that where credible risks exist, the lack of scientific certainty should not be a reason to postpone action. It has guided multiple successful international agreements, such as the 1987 Montreal Protocol, which acted decisively on ozone-depleting substances before the science was fully settled. This treaty has since prevented millions of cases of skin cancer and eye cataracts, and—instead of a projected tenfold increase in ozone depletion by 2050—the ozone layer is now projected to recover by the middle of the century,<sup>76</sup> despite the fact that chlorofluorocarbons (CFCs, the chemicals responsible) persist in the atmosphere for decades to centuries, with half-lives ranging from 50 to 500 years.



### WHAT IS THE PRECAUTIONARY PRINCIPLE?

The precautionary principle means taking preventative action in the face of uncertainty. If an action or policy has a suspected risk of causing harm to the public or the environment, the burden of proof falls on those advocating for the activity, not on the public to demonstrate harm. It is a principle that enables us to protect people and nature before damage is done.

Multiple international treaties have successfully applied the precautionary principle, for example, the Montreal Protocol, the Rio Declaration on Environment and Development, the Basel Convention, and the UN Framework Convention on Climate Change.

A similar approach is now needed for plastic pollution, which poses a direct threat to people, nature, and future generations. Like ozone-depleting substances, many plastics and their associated chemicals are highly persistent, remaining in the environment for decades or even centuries.<sup>77</sup> This persistence makes delayed action especially costly. Strong, proactive measures to address the negative impacts of plastics are essential. A truly effective response to the plastic pollution crisis must take an integrated One Health approach, recognising that impact on one domain is deeply linked to harm in others; reducing exposure in humans, animals, or the environment can reduce risk across the board.





## THE GLOBAL PLASTIC TREATY: A ONCE-IN-A-LIFETIME OPPORTUNITY

A robust global plastic treaty offers a vital opportunity to turn scientific evidence into policy action and reduce health risks from MnPs and plastic-related chemicals of concern. After five rounds of negotiations, a large majority of countries were supportive of ambitious measures,<sup>78</sup> with nearly 100 countries supporting the “Nice wakeup call for an ambitious treaty.”<sup>79</sup>

To effectively protect human and environmental health, the treaty must include, at minimum, the following four elements:<sup>80</sup>

FIGURE 6:

Must-haves for an ambitious global plastics treaty and one health approach



**Scientific evidence shows that out of the 16,000 chemicals that are used or present in plastic materials and products, less than 1,000 (6%) are currently subject to international regulations, even though more than 4,200 are known to be hazardous.<sup>81</sup>**

To minimise health risks, it is essential to ban and phase out the most high-risk plastic products—those with characteristics that increase their likelihood of ending up in our environments and cause harm as pollutants. This includes single-use items that are prone to littering and escaping waste management systems, as well as plastic products containing toxic substances and intentionally added microplastics. These products represent the most significant risks in the plastic pollution crisis and must be the first to go.

At the same time, addressing the chemical risks embedded in plastics is critical. Scientific evidence shows that out of the 16,000 chemicals that are used or present in plastic materials and products, less than 1,000 (6%) are currently subject to international regulations, even though more than 4,200 (more than 26%)—including those discussed in this paper—are known to be hazardous.<sup>81</sup> Closing this regulatory gap is crucial to reduce the potential harms of plastics to human and environmental health.





Measures to redesign plastic products and relevant systems provide another critical tool to mitigate health impacts. Harmonised global criteria on product design and performance would require producers across jurisdictions to increase the safety and minimise the risks of plastic products, as well as keep them in circulation through reuse and recycling. For example, product standards could minimise plastic products' releases of MnPs or require the exclusion of harmful additives. Such measures would ensure that plastics in circulation are safer by design and less likely to pollute environments and impact ecosystems and human health.

Lastly, comprehensive finance and implementation assistance, as well as mechanisms that allow the treaty to be strengthened over time, are fundamental to ensuring the long-term effectiveness of these measures and the treaty's overall mission of protecting human health and the environment from plastic pollution. Beyond these must-have elements, the treaty needs to include robust provisions for reporting, transparency, and trade, all of which are essential to enable effective implementation.



### NATIONAL LEADERSHIP CAN PAVE THE WAY

**While global rules are essential, national and regional governments don't need to wait.** Many of the measures now on the global negotiating table have already been tested at national level, such as national bans of specific plastic products and chemicals, national reforms of regulations on packaging, and eco-modulation in mandatory Extended Producer Responsibility schemes. For example, the EU's Packaging and Packaging Waste Regulation (PPWR), Kenya's EPR policy and bans on single-use plastics, and China's restrictions on certain plastic additives show that countries can feasibly phase out harmful products and chemicals.

Strong domestic policies can reduce immediate risks at the local level, while inspiring stronger commitments and actions at the global scale. By acting now, countries can protect their populations, drive innovation and raise the bar for international negotiations and the establishment of global rules. But to truly solve plastic pollution, national action must be matched by ambitious, binding global rules that ensure a level playing field and address global drivers of harm.

### FROM EVIDENCE TO ACTION

The severity of the crisis calls for urgent and coordinated action. As global power dynamics shift and new economic players emerge, there is a unique opportunity to reframe sustainable development through a One Health lens. States must recognise that the health of people, animals, and the environment are inextricably linked, and that greater international cooperation and regulatory harmonisation are critical to realising the long-term socio-economic benefits of this integrated approach.

Against the alarming threats of plastic pollution to human health and the environment, WWF and its partners call on governments and negotiators to deliver a robust and legally binding global treaty, with specific, enforceable rules capable of holding industry accountable. The ambitious majority of states must take the lead in shaping a treaty that is fair, effective, and uncompromising in its protection of the environment and public health. Governments should choose courage over compromise.

WWF calls on governments to demonstrate the courage to lead, the vision to build lasting systems change, and the responsibility to protect people and nature. The global treaty to end plastic pollution is within reach.

**The world is watching—now is the time to act.**







SCIENTIFIC FINDINGS ON SIX GROUPS OF CHEMICALS  
COMMONLY FOUND IN PLASTICS SHOW HOW  
THEY ENTER OUR BODIES AND CAUSE HARM—  
UNDERSCORING THE NEED FOR PRECAUTIONARY  
ACTION AND STRONGER REGULATION.



## 5. TECHNICAL APPENDIX

This technical appendix provides supporting detail on a selection of chemicals commonly found in plastics and plastic-associated pollution. While the main body of this paper summarises the overarching health risks associated with plastic exposure, this appendix offers a closer look at six specific chemical groups, a brief explanation of the use of these chemicals in plastics, the mechanisms by which they cause harm, and the scientific evidence linking them to adverse effects in humans and animals. The following summaries reflect findings from peer-reviewed research, international health agencies, and recent reviews. Where available, evidence from both human and animal studies is included to illustrate the weight of scientific concern.

While many of these chemicals have overlapping effects, this appendix organises the scientific evidence under the key health impact categories. Some plastic-related chemicals of concern, like phthalates and bisphenols, have been the focus of a significant wave of recent studies; others, like lead, have been under scrutiny for decades.

**While the volume and recency of research varies across these substances, the collective scientific picture is robust and deeply concerning.**

Many toxicological studies use concentrations of chemicals that are higher than what people may typically encounter in everyday environments. This approach helps researchers identify potential mechanisms of harm, but can limit direct comparisons to real-world exposure. Nevertheless, the patterns emerging across the literature present a clear signal that precautionary action is warranted, and there is growing scientific effort to assess health risks using more environmentally realistic concentrations.



## PHTHALATES



### What they are

Phthalates are used as plasticisers—substances that make a material softer and more flexible—in the manufacturing process across applications ranging from toys to medical devices to vinyl flooring, and are commonly found in household plastic waste.<sup>82</sup>

### Chemical examples

- DEHP (Di(2-ethylhexyl) phthalate) (CAS number 117-81-7)
- DBP (Dibutyl phthalate) (CAS number 84-74-2)
- BBP (Benzyl butyl phthalate) (CAS number 85-68-7)
- DIBP (Diisobutyl phthalate) (CAS number 84-69-5)
- DEP (Diethyl phthalate) (CAS number 84-66-2)
- MEP (Monoethyl phthalate) (CAS number 2306-33-4)
- MBP (Monobutyl phthalate) (CAS number 131-70-4)
- MIBP (Mono-isobutyl phthalate) (CAS number 30833-53-5)
- DINP (Diisononyl phthalate) (CAS numbers 28553-12-0 and 68515-48-0)
- DIDP (Diisodecyl phthalate) (CAS numbers 26761-40-0 and 68515-49-1)

How they affect human health	Phthalates are widely used, easily absorbed, and one of the most well-documented plastic-associated threats to human health. Their effects span multiple human systems, making them a key concern in the health impacts of plastic-related chemicals.	
<b>Hormonal and reproductive disruption</b>  As one of the most commonly encountered endocrine disrupting chemicals (EDCs), phthalates interfere with the body's hormone production system, often by mimicking natural hormones, binding to their receptors, and blocking the normal hormonal response. <sup>83</sup> Their impact is particularly notable during pregnancy and early childhood when hormonal balance is critical for brain and body development.	<b>Reduced male fertility</b>	Globally, male fertility is on the decline. Phthalates interfere with testosterone, a hormone essential for male reproductive development. There is robust evidence linking these chemicals to reduced fertility, <sup>84,85</sup> principally due to: <ul style="list-style-type: none"> <li>• <i>Reduced sperm quality:</i> exposure to higher phthalate concentrations is linked to decreased sperm motility (the sperm's ability to swim toward and fertilise an egg, with decreased motility increasing the likelihood of infertility).<sup>86</sup> A Chinese study found that phthalate-exposed men had a 6% reduction in total sperm count, a 5% reduction in sperm concentration, and a 3% decrease in motility, effects that partially reversed when the men moved to areas with lower phthalate exposure.<sup>87</sup> Similar findings have been observed in Sweden<sup>88</sup> and Russia, where men with higher DINP exposure during late puberty exhibited 32% lower total sperm count, 30% lower sperm concentration, and 30% reduced motility.<sup>89</sup></li> <li>• <i>Genital abnormalities:</i> phthalate exposure is associated with abnormal male reproductive development, increased risks of cryptorchidism (undescended testes) and hypospadias (misplacement of the urethral opening), and shorter anogenital distance (a marker for reduced fertility).<sup>90</sup> These abnormalities contribute to male infertility. Significant associations have been observed between prenatal exposure and these genital abnormalities.<sup>91</sup></li> </ul>
	<b>Female reproductive disruption</b>	While the evidence is less robust than in males, phthalates have also been linked to an adverse array of effects on female reproductive health. A number of studies have shown that phthalate exposure is linked with endometriosis, <sup>92</sup> uterine fibroids, <sup>93</sup> decreased ovarian reserve, <sup>94</sup> reduced rates of pregnancy, <sup>95</sup> increased rates of miscarriage, <sup>96</sup> and other poor pregnancy outcomes. <sup>97</sup>

## PHTHALATES

<b>Neurological development</b>  Phthalates are linked to disruptions in hormones critical to brain development. Phthalates have been shown to negatively impact the function of tyrosine, a building block for hormones linked to mood regulation, attention, and impulse control, and thyroxine, a hormone critical for neurodevelopment. 98, 99	<b>Impaired early cognitive development</b>	A study found that specific phthalates were associated with drastic developmental reductions between the ages of 4.5 and 7.5 months, including an 85% decrease in problem-solving ability in female babies and a 52% decrease in personal-social skills and 39% decrease in fine motor skills in male babies. <sup>100</sup> Early-life exposure, either in the womb or during childhood, has been shown to negatively impact cognitive development. <sup>101</sup> This aligns with findings from at least 25 studies linking maternal and prenatal phthalate exposure to reduced neurodevelopment and cognitive function in children. <sup>102</sup>
	<b>Reduced grey matter volume</b>	Higher maternal levels of phthalates (monoethyl phthalate, MEP) during pregnancy were associated with lower total grey matter volume in children at age 10, linked to impaired cognitive and motor function. <sup>103</sup>
	<b>Slower information processing</b>	Prenatal exposure to phthalates led to slower information processing in infancy. <sup>104</sup>
<b>Respiratory effects</b>  Phthalate exposure during pregnancy can elevate the risk of breathing issues in children. <sup>105</sup>	<b>Asthma</b>	One study found children of mothers with higher levels of the urinary metabolites of BBP and DBP during pregnancy were up to 78% more likely to develop asthma between the ages of 5 and 11, compared to children of mothers with lower levels. <sup>106</sup>
	<b>Allergic rhinitis (allergy-related breathing issues)</b>	Phthalates may trigger coughing and airway inflammation by disrupting immune function, contributing to chronic respiratory irritation and compromising lung function. <sup>107,108</sup> This may occur because they stimulate nerves <sup>109</sup> or modify gene activity in key immune cells, which could enhance allergic lung inflammation. <sup>110</sup>
<b>Cardiovascular impacts</b>  Phthalates can also pose serious risks to cardiovascular health, <sup>111</sup> possibly by damaging the gene expression of mitochondria. <sup>112</sup>	<b>Heart disease</b>	A study of over 10,000 adults suggested that phthalate levels are associated with an increase in cardiovascular disease. <sup>113</sup>
	<b>Hypertension</b>	Several studies have found phthalate exposure (specifically MEP, MBP, and MIBP) is associated with high blood pressure (hypertension), especially in pregnant women. <sup>114</sup>
	<b>Atherosclerosis (plaque build-up)</b>	Phthalates are believed to damage the mitochondria, <sup>115</sup> the engines of our cells that manage energy; malfunctioning mitochondria can cause inflammation and lead to atherosclerosis <sup>116</sup> , where arteries become narrowed and hardened due to a build-up of fatty deposits (plaque) on their inner walls. Atherosclerosis is a key cause of heart attack (myocardial infarction), heart failure, and stroke.



## PHTHALATES

## How they affect animal health

Male dogs exposed to DEHP showed similar declines in sperm quality to humans.<sup>117</sup> In mice, a mixture of phthalates was found to significantly reduce the motility of sperm, leading to reduced fertilisation rates.<sup>118</sup> A recent study of goats fed with phthalates throughout pregnancy reported significantly decreased levels of critical hormones oestrogen, progesterone, luteinizing hormone and thyroxine, compared to controls.<sup>119</sup>

Rodents and zebrafish exposed to phthalates show altered brain development, cognitive impairment, oxidative stress, and behavioural changes. Rat offspring exposed to phthalates in utero performed notably worse in cognitive tests.<sup>120</sup> Some phthalates, including BBP, DEHP and DBP, have also been shown to alter brain development and disrupt cognitive-behavioural function in adult zebrafish and rodents.<sup>121</sup> For instance, a particular study found that DEHP was developmentally toxic to zebrafish, with neurotoxicity shown through the inhibition of tail coiling and reduced activity. DEHP also induced oxidative stress and apoptosis in zebrafish larvae.<sup>122</sup> In a mouse model study, pregnant mice exposed to DEHP had offspring with up to 14% incidence of congenital heart disease; this study linked this to occurred by DEHP suppressing the key genes involved in the cardiogenesis (the development and formation of the heart).<sup>123</sup>



## BISPHENOLS



## What they are

Bisphenols are chemical compounds used to harden plastics and make them shatterproof. The most common is Bisphenol A (BPA), which is used in the production of polycarbonate plastics—transparent plastics often used as an additive in plastics for roofing and glazing, but also in CDs, safety helmets, and baby bottles. BPA has been phased out in some regions and is nowadays often replaced by other bisphenols such as BPB and BPS, which may pose the same (eco)toxicological risks. There are at least 34 other bisphenols in use that may exert similar harmful effects.<sup>124</sup>

## Chemical examples

- BPA (Bisphenol A) (CAS number 80-05-7)
- BPB (Bisphenol B) (CAS number 77-40-7)
- BPS (Bisphenol S) (CAS number 80-09-1)

## How they affect human health

BPA has raised public health concerns and has been classified as both an endocrine disruptor and a reproductive toxin.<sup>125</sup> The disruption of hormone pathways impacts numerous elements of human health, underscoring the systemic impact of bisphenols. As awareness of BPA's toxicity has grown, it has often been replaced by alternatives, such as bisphenol S. However a recent meta-analysis has demonstrated that all bisphenols have a strong impact on several hormones in animals, including those of the thyroid and reproductive systems. While the evidence base is still evolving, it is suggested that BPA alternatives have as great or greater effects on hormones as BPA.<sup>126</sup>

## Hormonal and reproductive disruption

By mimicking oestrogen, BPA alters genetic and hormonal pathways, linking it to increased risk of oestrogen-dependent diseases in women.<sup>127</sup>

## Breast cancer

BPA is suspected to increase the risk of breast cancer by interfering with oestrogen pathways and inducing genetic mutations that can lead to tumour formation.<sup>128</sup> In one study, individuals with breast cancer had significantly higher levels of urinary BPA compared to those without the disease.<sup>129</sup>

## Female reproductive issues

BPA exposure is associated with a range of reproductive issues, including polycystic ovary syndrome (PCOS—a condition which can lead to infertility)<sup>130</sup>, premature onset of puberty, low birth weight, and preeclampsia (a serious pregnancy complication characterised by high blood pressure).<sup>131</sup>

## Thyroid disruption

The thyroid gland is essential for regulating hormonal balance and orchestrating the growth and development of the nervous and skeletal systems. BPA can mimic thyroid hormone pathways and disrupt their normal function. These effects are especially pronounced in girls due to the interaction with female developmental processes.<sup>132,133</sup>

## Neurological development

BPA can interfere with oestrogen receptors throughout the body, including the brain, leading to long-term consequences. Disruption of oestrogen signalling during key developmental stages, such as in utero and puberty, can alter brain structure and function.

## Impaired neuro-development

By altering gene expression and brain function, BPA can have long-term consequences for neurodevelopment including ADHD, ASD, depression, anxiety, emotional instability, and cognitive deficits.<sup>134</sup>

## Adult neurological disease

BPA-related neurotoxicity has also been linked to an increased risk of neurological diseases including stroke, Alzheimer's disease, and Parkinson's disease.<sup>135</sup>



## BISPHENOLS

### Cardiovascular impacts

BPA exposure has been associated with cardiovascular disease and hypertension. Although not all mechanisms are fully understood yet, BPA has been shown to disrupt signalling within the cardiovascular system.<sup>136</sup>

### How they affect animal health

BPA has similar endocrine-disrupting effects on animals. In rodent studies, oestrogen interference led to altered sexual behaviour and heightened anxiety.<sup>137</sup> BPA has been identified as a direct carcinogen in breast tissue in mice and rats and has induced PCOS in rats.<sup>138</sup> Animal studies also found evidence of bisphenol exposure impeding thyroid activity in hamsters and disrupting tadpole metamorphosis.<sup>139</sup> Exposure of pregnant sheep to BPA was associated with hypothyroidism in newborn lambs.<sup>140</sup> Neurological effects have also been observed. Mice exposed to BPA in utero showed decreased learning ability, reduced long-term memory, and increased anxiety;<sup>141</sup> the brains of male rats exposed in utero were also negatively impacted.<sup>142</sup>



## UV STABILISERS AND ALKYLPHENOLS



### What they are

UV stabilisers are used to prevent degradation of plastics by heat and light, especially during processing. Alkylphenols are a category of stabilisers that make plastics more pliable and durable. They are found in products including medical plastics, automotive components, and outdoor furniture, as well as in cosmetic products.

<b>Chemical examples</b>	<ul style="list-style-type: none"> <li>• Benzophenone-3 (BP-3)</li> <li>• Nonylphenols</li> <li>• Octylphenols</li> </ul>	
<b>How they affect human health</b>	<p>UV stabilisers are endocrine disruptors that interfere with the body's hormonal systems, leading to a wide range of adverse health outcomes. BP-3 is one of the most well-documented harmful UV stabilisers. Alkylphenols are also endocrine disruptors; they are a broad category of complex chemicals, but they all share a similar molecular structure to oestrogen.</p>	
<b>Hormonal and reproductive disruption</b>  The hormonal impacts of UV stabilisers and alkylphenols mean they are linked to a number of serious hormone-related conditions, including several related to the reproductive system.	<b>Hormone-related cancers</b>	<p>Due to their impact on female sex hormones, UV stabilisers have been linked to hormone-sensitive cancers, including breast and endometrial cancer. A study involving more than 1,500 breast cancer cases found higher cases among people working in jobs with regular exposure to alkylphenols, including plastics manufacturing,<sup>143</sup> while certain UV stabilisers have been shown to promote the growth of breast cancer cells.<sup>144</sup> In another study, women with endometrial cancer had significantly higher urinary concentrations of two types of alkylphenol compared to those without the disease.<sup>145</sup></p>
	<b>Endometriosis and other uterine disorders</b>	<p>Eight regional studies linked UV stabilisers to an increased risk of hormone-related reproductive conditions such as uterine fibroids and endometriosis. In one study, women with the highest concentration of urinary BP-3 presented a 65% higher chance of endometriosis than other groups.<sup>146</sup></p>
	<b>Gestational diabetes</b>	<p>Alkylphenol exposure results in a significantly increased risk of gestational diabetes in pregnant women, particularly those carrying female fetuses.<sup>147</sup> While evidence is still emerging, one study of pregnant women in China found that this could be due to alkylphenol exposure during pregnancy interfering with the mother's liver function.<sup>148</sup></p>
	<b>Testosterone disruption in males</b>	<p>Exposure to BP-3 is associated with lower levels of testosterone in adolescent males, which can impede puberty, decrease bone and muscle density, and cause infertility.<sup>149</sup></p>
	<b>Congenital disease</b>	<p>Prenatal exposure to BP-3 is associated with Hirschsprung's disease, a congenital gut disorder caused by gene mutations that causes intestinal blockage and chronic constipation.<sup>150</sup></p>
<b>Other impacts</b>	<b>Bone health</b>	<p>BP-3 interferes with SPARC, a protein that influences bone formation, maintenance, and repair. Elevated levels of urinary BP-3 are correlated with osteoarthritis.<sup>151</sup></p>
<b>How they affect animal health</b>	<p>The impacts of UV stabilisers are not only seen in humans but have also been found to have negative health effects in fish and rodents.<sup>152</sup> Animal studies have shown that alkylphenols can damage liver and kidney tissue, particularly in rats.<sup>153</sup> Exposure in pregnant rats was linked to liver damage for both mother and offspring, providing a basis for studying similar impacts in humans.<sup>154, 155</sup></p>	



## PER- AND POLYFLUOROALKYL SUBSTANCES (PFAS)



### What they are

Per- and polyfluoroalkyl substances (PFAS) are used widely for consumer products. They are added also to plastic products to make them resistant to heat, oil, stains, grease, and water. They can be found in food packaging, cookware, building materials, and electronics.

### Chemical examples

There are potentially thousands of chemicals classified as PFAS. Studies tend to approach them as a category rather than individual compounds.

### How they affect human health

PFAS—often referred to as “forever chemicals”—are synthetic compounds that persist in the environment, wildlife, and human bodies for years. These chemicals are known endocrine disruptors, interfering with hormonal pathways and particularly affecting thyroid function. PFAS do not target a single organ or system—they can disrupt the body at multiple levels, over long periods of time.

### Hormonal and reproductive disruption

PFAS can mimic oestrogen and testosterone, impairing the production, transport, and breakdown of these key hormones, which has significant implications for reproductive and overall health.<sup>156</sup>

#### Adverse pregnancy and birth outcomes

Prenatal exposure to PFAS is linked to adverse impacts including increased risks of preeclampsia, low birth weight, and damage to the placenta, an organ critical to fetal development and nutrient transfer.<sup>157</sup>

#### Impaired lactation and breastfeeding

Two studies have linked PFAS exposure to shortened breastfeeding duration, with implications for early life nutrition.<sup>158</sup>

#### Cancer

PFAS have been linked to several types of cancer, particularly thyroid, given its interference with thyroid hormones. One study found a 56% increased rate of thyroid cancer diagnosis in patients with double the amount of PFOS (a type of PFAS) in their blood.<sup>159</sup> There is also evidence linking PFAS to kidney and testicular cancers: 11 studies found an association between overall PFAS exposure and a higher risk of kidney cancers, while high-dose exposure is associated with a higher risk of testicular cancer.<sup>160</sup>

### Respiratory effects

PFAS exposure can damage lung tissue and trigger inflammation.

#### Chronic obstructive pulmonary disease (COPD)

PFAS exposure is emerging as a risk factor for COPD, a major global health issue identified as the third leading cause of death by the WHO in 2019.<sup>161</sup>

#### Reduced lung function

Exposure can damage lung tissue, triggering inflammation, and has been linked to reduced lung function in children and adolescents, as well as increased rates of asthma and allergies.<sup>162, 163</sup>

### Other impacts

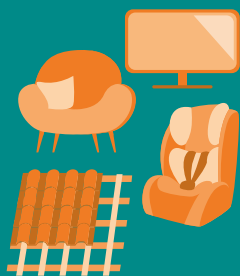
#### Immune suppression

PFAS can suppress immune function more broadly, leading to reduced vaccine efficacy and increasing risk of childhood infectious disease.<sup>164</sup> These immune system disruptions underscore the systemic nature of PFAS toxicity in the body.

### How they affect animal health

In rabbits, which have placental structures similar to humans, PFAS exposure led to maternal weight gain, high blood pressure (hypertension), kidney damage, and altered placental responses.<sup>165</sup> In rodents, PFAS have been shown to reduce the production and transport of lactation-related hormones and delay mammary gland development, mirroring findings in human studies linking PFAS exposure to shorter breastfeeding duration.<sup>166</sup>

## FLAME RETARDANTS



### What they are

Flame retardants are used to make plastics resistant to fire, stopping them from combusting and degrading; they are used for applications in clothing, construction, electric appliances, wires, and cables.

Organophosphates, once considered safer alternatives to common brominated flame retardants like HBCD and PBDEs, have recently come under scrutiny themselves.

### Chemical examples

- TBBPA (Tetrabromobisphenol A) (CAS number 79-94-7)
- HBCD (Hexabromocyclododecane) (CAS number 3194-55-6)
- TDCPP (Tris(1,3-dichloro-2-propyl)phosphate) (CAS number 13674-87-8)
- PBDEs (polybrominated diphenyl ethers)

### How they affect human health

Many flame retardants are known endocrine disruptors, impacting the way hormones behave in the body and impairing normal physiological processes.<sup>167</sup>

#### Hormonal and reproductive disruption

The hormonal impacts induced by exposure to flame retardants raise serious concerns about hormone-related cancers.

#### Increased cancer risk

HBCD can interfere with oestrogen activity, thus increasing the proliferation of breast cancer cells,<sup>168</sup> and has also been found to enhance the progression of prostate cancer.<sup>169</sup> PBDEs (polybrominated diphenyl ethers) disrupt thyroid hormones and are linked to thyroid cancer.<sup>170</sup>

TDCPP, an organophosphate flame retardant (OPFR), has been shown to be carcinogenic in human liver cells.<sup>171</sup>

#### Neurological development

Flame retardants have been shown to negatively affect neurological development by disrupting hormone regulation, altering neurotransmitter levels, and impairing cognitive function during critical periods of brain growth.

#### Developmental impairment

Prenatal and early childhood exposure to PBDEs has been linked to cognitive impairments and reduced IQ levels in children, and possible links to autism.<sup>172, 173</sup>

#### Parkinson's disease

BFR exposure has also been associated with a higher risk of Parkinson's disease.<sup>174</sup>

#### Respiratory effects

Flame retardants can adversely impact respiratory health by reducing lung function and increasing susceptibility to respiratory conditions, particularly due to the inhalation of particles.

#### Childhood breathing issues

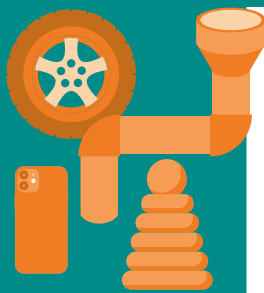
Exposure to OPFRs in inhaled dust was found to be associated with a higher risk of wheezing, respiratory infection, and hay fever or allergies in children aged one year old.<sup>175</sup>



FLAME RETARDANTS		
Other impacts	Metabolic syndrome	In men, OPFRs are associated with metabolic syndrome – a group of conditions linked to heart disease, stroke, and diabetes that include high blood pressure, high blood sugar, and high cholesterol. <sup>176</sup>
How they affect animal health	TBBPA (tetrabromobisphenol A) has been found to cause uterine cancer in rats. <sup>177</sup> In zebrafish, HBCD has been found to interfere with thyroid hormones, resulting in liver damage, and cause body malformations. <sup>178, 179</sup> At least three rodent studies show that HBCD impedes neurodevelopment, resulting in disturbed behaviour in adult animals. <sup>180</sup>	



## METALS, METALLOIDS AND METAL COMPOUNDS



### What they are

Heavy metals and metalloids are used as colour pigments or to increase the density of plastic.<sup>181</sup> Cadmium and lead are the two highest risk metal additives. Cadmium is banned for use in the EU as an additive in 16 types of plastic, including PVC and polypropylene, due to its health impacts, but it is allowed in other polymers.<sup>182</sup> There are high concentrations of lead in historical items that are still in use or circulation (toys, construction plastics, wiring insulation), and it has contaminated some modern plastics through mechanical recycling.<sup>183</sup>

<b>Chemical examples</b>	<ul style="list-style-type: none"> <li>• Cadmium (CAS number 7440-43-9)</li> <li>• Lead (CAS number 7439-92-1)</li> </ul>	
<b>How they affect human health</b>	Cadmium and lead are linked to significant health risks impacting the hormonal, neurological, and cardiovascular systems.	
<b>Hormonal and reproductive disruption</b>  Lead and cadmium can cause hormonal and reproductive disruption by interfering with endocrine system function and impairing fertility mechanisms.	<b>Reduced male fertility</b>	Lead exposure reduces sperm concentration, volume, and motility, contributing to a decline in male fertility. <sup>184</sup>
	<b>Cancer</b>	Cadmium is a known carcinogen, with the main pathway being oestrogen and oestrogen receptor disruption, increasing the risk of endometrial cancer. <sup>185</sup> It is also associated with an elevated risk of thyroid, <sup>186</sup> lung, kidney, prostate, and pancreatic cancer. <sup>187</sup>
<b>Neurological impacts</b>  Lead is a well-documented neurotoxin, causing damage to brain functioning and development through several mechanisms.	<b>Nervous system development</b>	Lead can alter the development of the nervous system, impacting the formation of important neuronal cells and affecting the function of neurotransmitters. It also impacts nitric oxide activity, which affects blood vessels in the brain and alters the transmission of serotonin. <sup>188</sup>
	<b>Cognitive outcomes</b>	Lead exposure has serious implications for fetal neurodevelopment, with prenatal and early life lead exposure linked to various neurological disorders and effects on behavioural and cognitive outcomes such as lower IQ, dyslexia, ADHD, and antisocial behaviour. <sup>189</sup>
<b>Cardiovascular impacts</b>  Lead and cadmium are associated with harmful impacts on the cardiovascular system.	<b>Cardiovascular disease</b>	Lead exposure has been linked to hypertension, coronary heart disease, and stroke mortality, as well as peripheral arterial disease. <sup>190</sup> Cadmium can also increase the risk of cardiovascular disease, and urinary cadmium level is a predictor of mortality in cardiovascular disease patients. <sup>191</sup>



## METALS, METALLOIDS AND METAL COMPOUNDS

Other impacts	Diabetes	Cadmium can increase the risk of diabetes due to its interactions with the metabolic system. <sup>192</sup>
	DNA damage	The stress and DNA damage inflicted on the body by cadmium exposure can also induce kidney damage, liver injury, neurodegenerative disease, and osteoporosis. <sup>193,194</sup>
How they affect animal health	Lead has many of the same harmful effects on animals. It has been linked to brain damage, hypertension, lower fertility, and liver injury in rats. <sup>195</sup> Cadmium has been found to be carcinogenic in rats, with laboratory studies observing links to leukaemia, kidney, prostate, and testicular cancers. <sup>196</sup> It is also associated with osteoporosis and bone density in rats. <sup>197</sup>	



# BIBLIOGRAPHY

- 1 United Nations Environment Programme Ozone Secretariat (2020). Montreal Protocol likely to avert 443 million skin cancer cases in the United States. Retrieved from: <https://ozone.unep.org/montreal-protocol-likely-avert-443-million-skin-cancer-cases-united-states>
- 2 Kukkola, A., Schneidewind, U., Haverson, L., Kelleher, L., Drummond, J. D., Smith, G. S., Lynch, I., Krause, S. (2024). Snapshot sampling may not be enough to obtain robust estimates for riverine microplastic loads, *ACS ES&T Water*, 4/5: 2309-2319. DOI: 10.1021/acsestwater.4c00176
- 3 Wazne, M., Schneidewind, U., Haverson, L., Mermillod-Blondin, F., Simon, L., Nel, H. A., Krause, S. (2024). Does what we find depend on how we sample? Measured streambed microplastic concentrations can be affected by the choice of sampling method, *The Science of the Total Environment*, 958: 178096. DOI: 10.1016/j.scitotenv.2024.178096
- 4 Jamieson, A. (2020). The new face of the plastics crisis, *Newcastle University, Eurythenes plasticus*. Retrieved from: <https://www.ncl.ac.uk/press/articles/archive/2020/03/eurythenesplasticus>
- 5 Materić, D., Ludewig, E., Brunner, D., Röckmann, T., Holzinger, R. (2021a). Nanoplastics transport to the remote, high-altitude alps, *Environmental Pollution*, 288: 117697. DOI: 10.1016/j.envpol.2021.117697
- 6 Zhang, Y., Kang, S., Allen, S., Allen, D., Gao, T., Sillanpää, M. (2020a). Atmospheric microplastics: A review on current status and perspectives, *Earth-Science Reviews*, 203: 103118. DOI: 10.1016/j.earscirev.2020.103118
- 7 Vethaak, A. D., Legler, J. (2021a). Microplastics and human health, *Science*, 371/6530: 672-4. DOI: 10.1126/science.abe5041
- 8 Zhang, J., Wang, L., Kannan, K. (2019). Microplastics in house dust from 12 countries and associated human exposure, *Environment International*, 134: 105314. DOI: 10.1016/j.envint.2019.105314
- 9 Peixoto, D., Pinheiro, C., Amorim, J., Oliva-Teles, L., Guilhermino, L., Vieira, M. N. (2019). Microplastic pollution in commercial salt for human consumption: A review, *Estuarine Coastal and Shelf Science*, 219: 161-168. DOI: 10.1016/j.ecss.2019.02.018
- 10 World Health Organization. (2022). Dietary and inhalation exposure to nano- and microplastic particles and potential implications for human health. Retrieved from: <https://iris.who.int/handle/10665/362049>
- 11 Diaz-Basantes, M. F., Conesa, J. A., Fullana, A. (2020). Microplastics in honey, beer, milk and refreshments in Ecuador as emerging contaminants, *Sustainability*, 12/14: 5514. DOI: 10.3390/su12145514
- 12 Rubio-Armendáriz, C., Alejandro-Vega, S., Paz-Montelongo, S., Gutiérrez-Fernández, Á. J., Carrascosa-Iruzueta, C. J., La Torre, A. H.-D. (2022). Microplastics as emerging food contaminants: A challenge for food safety, *International Journal of Environmental Research and Public Health*, 19/3: 1174. DOI: 10.3390/ijerph19031174
- 13 Thompson, R. C., Courteney-Jones, W., Boucher, J., Pahl, S., Raubenheimer, K., Koelmans, A. A. (2024). Twenty years of microplastics pollution research—what have we learned?, *Science*, 386/6720. DOI: 10.1126/science.adl2746
- 14 WWF (o. J.). Implementing a One Health approach to conservation. Retrieved from: <https://www.panda.org/discover/our-focus/wildlife-practice/one-health>
- 15 Rodrigues, A. C. B., De Jesus, G. P., Waked, D., Gomes, G. L., Silva, T. M., Yariwake, V. Y., Da Silva, M. P. et al. (2022). Scientific Evidence about the Risks of Micro and Nanoplastics (MNPLs) to Human Health and Their Exposure Routes through the Environment, *Toxics*, 10/6: 308. DOI: 10.3390/toxics10060308
- 16 Ageel, H. K., Harrad, S., Abdallah, M. A.-E. (2021). Occurrence, human exposure, and risk of microplastics in the indoor environment, *Environmental Science Processes & Impacts*, 24/1: 17-31. DOI: 10.1039/d1em00301a
- 17 Soltani, N. S., Taylor, M. P., Wilson, S. P. (2021a). Quantification and exposure assessment of microplastics in Australian indoor house dust, *Environmental Pollution*, 283: 117064. DOI: 10.1016/j.envpol.2021.117064
- 18 Zhang, Q., Zhao, Y., Du, F., Cai, H., Wang, G., Shi, H. (2020). Microplastic fallout in different indoor environments, *Environmental Science & Technology*, 54/11: 6530-6539. DOI: 10.1021/acs.est.0c00087
- 19 Pfaller, J. B., Goforth, K. M., Gil, M. A., Savoca, M. S., Lohmann, K. J. (2020). Odors from marine plastic debris elicit foraging behavior in sea turtles, *Current Biology*, 30/5: R213-4. DOI: 10.1016/j.cub.2020.01.071
- 20 NOAA Marine Debris Program, Ingestion | Why Marine Debris Is a Problem. Retrieved from: <https://marinedebris.noaa.gov/why-marine-debris-problem/ingestion>
- 21 Kühn, S., Van Franeker, J. A. (2020a). Quantitative overview of marine debris ingested by marine megafauna, *Marine Pollution Bulletin*, 151: 110858. DOI: 10.1016/j.marpolbul.2019.110858
- 22 Jeong, E., Lee, J.-Y., Redwan, M. (2024). Animal exposure to microplastics and health effects: A review, *Emerging Contaminants*, 10/4: 100369. DOI: 10.1016/j.emcon.2024.100369
- 23 Tekman, M. B., Walther, B. A., Peter, C., Gutow, L., Bergmann, M. (2022). Impacts of plastic pollution in the oceans on marine species, biodiversity and ecosystems. *Zenodo* (CERN European Organization for Nuclear Research). DOI: 10.5281/zenodo.5898684
- 24 Musa, I. O., Auta, H. S., Ilyasu, U. S., Aransiola, S. A., Makun, H. A., Adabara, N. U., Abioye, O. P. et al. (2023). Micro- and nanoplastics in environment: degradation, detection, and ecological impact, *International Journal of Environmental Research*, 18/1. DOI: 10.1007/s41742-023-00551-9
- 25 Kavle, R. R., Nolan, P. J., Carne, A., Agyei, D., Morton, J. D., Bekhit, A. E.-D. A. (2023). Earth Worming—An Evaluation of Earthworm (*Eisenia andrei*) as an Alternative Food Source, *Foods*, 12/10: 1948. DOI: 10.3390/foods12101948
- 26 ChemTrust (2023). Flame retardant chemicals contaminate over 150 species of wildlife. Retrieved from: [chemtrust.org/news/flame-retardant-map](https://chemtrust.org/news/flame-retardant-map)
- 27 ChemTrust (2023). Flame retardant chemicals contaminate over 150 species of wildlife. Retrieved from: [chemtrust.org/news/flame-retardant-map](https://chemtrust.org/news/flame-retardant-map)



- 28 West, J. B. (2022). The strange history of atmospheric oxygen, *Physiological Reports*, 10/6. DOI: 10.14814/phy2.15214
- 29 Tetu, S. G., Sarker, I., Schrameyer, V., Pickford, R., Elbourne, L. D. H., Moore, L. R., Paulsen, I. T. (2019). Plastic leachates impair growth and oxygen production in *Prochlorococcus*, the ocean's most abundant photosynthetic bacteria, *Communications Biology*, 2/1. DOI: 10.1038/s42003-019-0410-x
- 30 Luo, Q., Tan, H., Ye, M., Jho, E. H., Wang, P., Iqbal, B., Zhao, X. et al. (2025). Microplastics as an emerging threat to human health: An overview of potential health impacts, *Journal of Environmental Management*, 387: 125915. DOI: 10.1016/j.jenvman.2025.125915
- 31 Mahmud, F., Sarker, D. B., Jocelyn, J. A., Sang, Q.-X. A. (2024). Molecular and Cellular Effects of Microplastics and Nanoplastics: Focus on Inflammation and Senescence, *Cells*, 13/21: 1788. DOI: 10.3390/cells13211788
- 32 Krause, S., Ouellet, V., Allen, D., Allen, S., Moss, K., Nel, H. A., Manaseki-Holland, S. et al. (2024). The potential of micro- and nanoplastics to exacerbate the health impacts and global burden of non-communicable diseases, *Cell Reports Medicine*, 5/6: 101581. DOI: 10.1016/j.xcrm.2024.101581
- 33 Koelmans, A. A., Redondo-Hasselerharm, P. E., Nor, N. H. M., De Ruijter, V. N., Mintenig, S. M., Kooi, M. (2022). Risk assessment of microplastic particles, *Nature Reviews Materials*, 7/2: 138–152. DOI: 10.1038/s41578-021-00411-y
- 34 Landrigan, P. J., Raps, H., Cropper, M., Bald, C., Brunner, M., Canonizado, E. M., Charles, D. et al. (2023). The Mindereroo-Monaco Commission on Plastics and Human Health, *Annals of Global Health*, 89/1. DOI: 10.5334/aogh.4056
- 35 Vasse, G. F., Melgert, B. N. (2024). Microplastic and plastic pollution: impact on respiratory disease and health, *European Respiratory Review*, 33/172: 230226. DOI: 10.1183/16000617.0226-2023
- 36 Xuan, L., Wang, Y., Qu, C., Yi, W., Yang, J., Pan, H., Zhang, J. et al. (2024). Exposure to polystyrene nanoplastics induces abnormal activation of innate immunity via the cGAS-STING pathway, *Ecotoxicology and Environmental Safety*, 275: 116255. DOI: 10.1016/j.ecoenv.2024.116255
- 37 Liu, S., Wang, C., Yang, Y., Du, Z., Li, L., Zhang, M., Ni, S. et al. (2024). Microplastics in three types of human arteries detected by pyrolysis-gas chromatography/mass spectrometry (Py-GC/MS), *Journal of Hazardous Materials*, 469: 133855. DOI: 10.1016/j.jhazmat.2024.133855
- 38 Marfella, R., Prattichizzo, F., Sardù, C., Fulgenzi, G., Graciotti, L., Spadoni, T., D'Onofrio, N. et al. (2024). Microplastics and Nanoplastics in Atheromas and Cardiovascular Events, *New England Journal of Medicine*, 390/10: 900–910. DOI: 10.1056/nejmoa2309822
- 39 Xuan, L., Wang, Y., Qu, C., Yi, W., Yang, J., Pan, H., Zhang, J. et al. (2024). Exposure to polystyrene nanoplastics induces abnormal activation of innate immunity via the cGAS-STING pathway, *Ecotoxicology and Environmental Safety*, 275: 116255. DOI: 10.1016/j.ecoenv.2024.116255
- 40 Guanglin, L., Shuqin, W. (2023). Polystyrene nanoplastics exposure causes inflammation and death of esophageal cell, *Ecotoxicology and Environmental Safety*, 269: 115819. DOI: 10.1016/j.ecoenv.2023.115819
- 41 Adapted from: Krause, S., Ouellet, V., Allen, D., Allen, S., Moss, K., Nel, H. A., Manaseki-Holland, S. et al. (2024). The potential of micro- and nanoplastics to exacerbate the health impacts and global burden of non-communicable diseases, *Cell Reports Medicine*, 5/6: 101581. DOI: 10.1016/j.xcrm.2024.101581
- 42 Yan, Z., Liu, Y., Zhang, T., Zhang, F., Ren, H., Zhang, Y. (2021). Analysis of Microplastics in Human Feces Reveals a Correlation between Fecal Microplastics and Inflammatory Bowel Disease Status, *Environmental Science & Technology*, 56/1: 414–421. DOI: 10.1021/acs.est.1c03924
- 43 Roslan, N. S., Lee, Y. Y., Ibrahim, Y. S., Anuar, S. T., Yusof, K. M. K. K., Lai, L. A., Brentnall, T. (2024). Detection of microplastics in human tissues and organs: A scoping review, *Journal of Global Health*, 14. DOI: 10.7189/jogh.14.04179
- 44 Nihart, A. J., Garcia, M. A., El Hayek, E., Liu, R., Olewine, M., Kingston, J. D., Castillo, E. F. et al. (2025). Author Correction: Bioaccumulation of microplastics in decedent human brains, *Nature Medicine*. 31, 1367. DOI: 10.1038/s41591-025-03675-x
- 45 Cverenkárová, K., Valachovičová, M., Mackulak, T., Žemlička, L., Bírošová, L. (2021). Microplastics in the Food Chain, *Life*, 11/12: 1349. DOI: 10.3390/life11121349
- 46 Yang, Q. E., Lin, Z., Gan, D., Li, M., Liu, X., Zhou, S., Walsh, T. R. (2025). Microplastics mediates the spread of antimicrobial resistance plasmids via modulating conjugal gene expression, *Environment International*, 195: 109261. DOI: 10.1016/j.envint.2025.109261
- 47 Segovia-Mendoza, M., Nava-Castro, K. E., Palacios-Arreola, M. I., Garay-Canales, C., Morales-Montor, J. (2020). How microplastic components influence the immune system and impact on children health: Focus on cancer, *Birth Defects Research*, 112/17: 1341–1361. DOI: 10.1002/bdr2.1779
- 48 National Institute of Environmental Health Sciences, Endocrine disruptors. Retrieved from: <https://www.niehs.nih.gov/health/topics/agents/endocrine>
- 49 Pérez-Albaladejo, E., Solé, M., Porte, C. (2020). Plastics and plastic additives as inducers of oxidative stress, *Current Opinion in Toxicology*, 20–21: 69–76. DOI: 10.1016/j.cotox.2020.07.002
- 50 Nadeem, A., Al-Harbi, N. O., Ahmad, S. F., Alhazzani, K., Attia, S. M., Alsanea, S., Alhoshani, A. et al. (2021). Exposure to the plasticizer, Di-(2-ethylhexyl) Phthalates during juvenile period exacerbates autism-like behavior in adult BTBR T + tf/J mice due to DNA hypomethylation and enhanced inflammation in brain and systemic immune cells, *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 109: 110249. DOI: 10.1016/j.pnpbp.2021.110249
- 51 Prado, Y., Aravena, C., Aravena, D., Eltit, F., Gatica, S., Riedel, C. A., Simon, F. (2023). Small plastics, big inflammatory problems, *Advances in Experimental Medicine and Biology*, 101–127. DOI: 10.1007/978-3-031-26163-3\_6
- 52 Seewoo, B. J., Goodes, L. M., Mofflin, L., Mulders, Y. R., Wong, E. V., Toshniwal, P., Brunner, M. et al. (2023). The plastic health map: A systematic evidence map of human health studies on plastic-associated chemicals, *Environment International*, 181: 108225. DOI: 10.1016/j.envint.2023.108225

- 53 Katsumiti, A., Losada-Carrillo, M. P., Barros, M., Cajaraville, M. P. (2021). Polystyrene nanoplastics and microplastics can act as Trojan horse carriers of benzo(a)pyrene to mussel hemocytes in vitro, *Scientific Reports*, 11/1. DOI: 10.1038/s41598-021-01938-4
- 54 Zhang, M., Xu, L. (2020). Transport of micro- and nanoplastics in the environment: Trojan-Horse effect for organic contaminants, *Critical Reviews in Environmental Science and Technology*, 52/5: 810–846. DOI: 10.1080/10643389.2020.1845531
- 55 Zhang, Y., Goss, G. G. (2021). The “Trojan Horse” effect of nanoplastics: potentiation of polycyclic aromatic hydrocarbon uptake in rainbow trout and the mitigating effects of natural organic matter, *Environmental Science Nano*, 8/12: 3685–3698. DOI: 10.1039/d1en00738f
- 56 La Merrill, M. A., Vandenberg, L. N., Smith, M. T., Goodson, W., Browne, P., Patisaul, H. B., Guyton, K. Z. et al. (2019). Consensus on the key characteristics of endocrine-disrupting chemicals as a basis for hazard identification, *Nature Reviews Endocrinology*, 16/1: 45–57. DOI: 10.1038/s41574-019-0273-8
- 57 There are several proposed mechanisms whereby endocrine-disrupting chemicals operate. One of these is to activate hormone receptors and interrupt important downstream processes, others are through alteration of production, distribution, metabolism and clearance of hormones, and some can epigenetically (by turning on or off genes without changing the DNA sequence) modify hormone producing or hormone-responsive cells (La Merrill, M. A., Vandenberg, L. N., Smith, M. T., Goodson, W., Browne, P., Patisaul, H. B., Guyton, K. Z. et al. (2019). Consensus on the key characteristics of endocrine-disrupting chemicals as a basis for hazard identification, *Nature Reviews Endocrinology*, 16/1: 45–57. DOI: 10.1038/s41574-019-0273-8).
- 58 Gore, A. C., La Merrill, M. A., Patisaul, H. B., Sargis, R. (2024). Endocrine Disrupting Chemicals: Threats to Human Health, *The Endocrine Society and IPEN*.
- 59 Casiano, A. S., Lee, A., Tete, D., Erdogan, Z. M., Treviño, L. (2022). Endocrine-Disrupting Chemicals and Breast Cancer: Disparities in Exposure and Importance of Research Inclusivity, *Endocrinology*, 163/5. DOI: 10.1210/endocr/bqac034
- 60 Pang, L., Wei, H., Wu, Y., Yang, K., Wang, X., Long, J., Chen, M. et al. (2024). Exposure to alkylphenols during early pregnancy and the risk of gestational diabetes mellitus: Fetal sex-specific effects, *Ecotoxicology and Environmental Safety*, 287: 117270. DOI: 10.1016/j.ecoenv.2024.117270
- 61 Völker, J., Ashcroft, F., Vedø, Å., Zimmermann, L., Wagner, M. (2022). Adipogenic Activity of Chemicals Used in Plastic Consumer Products, *Environmental Science & Technology*, 56/4: 2487–2496. DOI: 10.1021/acs.est.1c06316
- 62 Ghassabian, A., Van den Dries, M., Trasande, L., Lamballais, S., Spaan, S., Martinez-Moral, M.-P., Kannan, K. et al. (2023). Prenatal exposure to common plasticizers: a longitudinal study on Phthalates, brain volumetric measures, and IQ in youth, *Molecular Psychiatry*, 28/11: 4814–4822. DOI: 10.1038/s41380-023-02225-6
- 63 Martínez-Martínez, M. I., Alegre-Martínez, A., Cauli, O. (2021). Prenatal exposure to Phthalates and its effects upon cognitive and motor functions: A systematic review, *Toxicology*, 463: 152980. DOI: 10.1016/j.tox.2021.152980
- 64 Costa, H. E., Cairrao, E. (2023). Effect of bisphenol A on the neurological system: a review update, *Archives of Toxicology*, 98/1: 1–73. DOI: 10.1007/s00204-023-03614-0
- 65 National Cancer Institute. reactive oxygen species. Retrieved from: <https://www.cancer.gov/publications/dictionaries/cancer-terms/def/reactive-oxygen-species>
- 66 Yang, J., Luo, J., Tian, X., Zhao, Y., Li, Y., Wu, X. (2024). Progress in Understanding Oxidative Stress, Aging, and Aging-Related Diseases, *Antioxidants*, 13/4: 394. DOI: 10.3390/antiox13040394
- 67 Sicińska, P., Kik, K., Bukowska, B. (2020). Human Erythrocytes Exposed to Phthalates and Their Metabolites Alter Antioxidant Enzyme Activity and Hemoglobin Oxidation, *International Journal of Molecular Sciences*, 21/12: 4480. DOI: 10.3390/ijms21124480
- 68 Brassea-Pérez, E., Hernández-Camacho, C. J., Labrada-Martagón, V., Vázquez-Medina, J. P., Gaxiola-Robles, R., Zenteno-Savín, T. (2021). Oxidative stress induced by Phthalates in mammals: State of the art and potential biomarkers, *Environmental Research*, 206: 112636. DOI: 10.1016/j.envres.2021.112636
- 69 Nilsson, E. E., Maamar, M. B., Skinner, M. K. (2022). Role of epigenetic transgenerational inheritance in generational toxicology, *Current Zoology*, 8/1. DOI: 10.1093/eep/dvac001
- 70 Alavian-Ghavanini, A., Lin, P.-I., Lind, P. M., Rimfors, S. R., Lejonklou, M. H., Dunder, L., Tang, M. et al. (2018). Prenatal Bisphenol A Exposure is Linked to Epigenetic Changes in Glutamate Receptor Subunit Gene *Grin2b* in Female Rats and Humans, *Scientific Reports*, 8/1. DOI: 10.1038/s41598-018-29732-9
- 71 Cleveland Clinic, Inflammation. Retrieved from: <https://my.clevelandclinic.org/health/symptoms/21660-inflammation>
- 72 Singh, D. D. (2024). Epigenetic Mechanisms of Endocrine-Disrupting Chemicals in Breast Cancer and Their Impact on Dietary Intake, *Journal of Xenobiotics*, 15/1: 1. DOI: 10.3390/jox15010001
- 73 Nadeem, A. et al. (2021). Exposure to the plasticizer, Di-(2-ethylhexyl) Phthalates during juvenile period exacerbates autism-like behavior in adult BTBR T + tf/J mice due to DNA hypomethylation and enhanced inflammation in brain and systemic immune cells, *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 109: 110249. DOI: 10.1016/j.pnpbp.2021.110249
- 74 Kay, J., Thadhani, E., Samson, L., Engelward, B. (2019). Inflammation-induced DNA damage, mutations and cancer, *DNA Repair*, 83: 102673. DOI: 10.1016/j.dnarep.2019.102673
- 75 Pinto-Bazurco, J. F. (2020). The Precautionary Principle, *International Institute for Sustainable Development*. Retrieved from: [www.iisd.org/articles/deep-dive/precautionary-principle](http://www.iisd.org/articles/deep-dive/precautionary-principle)
- 76 United Nations Environment Programme, About Montreal Protocol. Retrieved from: [www.unep.org/ozonaction/who-we-are/about-montreal-protocol](http://www.unep.org/ozonaction/who-we-are/about-montreal-protocol)
- 77 Thompson, R. C., Courteney-Jones, W., Boucher, J., Pahl, S., Raubenheimer, K., Koelmans, A. A. (2024a). Twenty years of microplastics pollution research—what have we learned?, *Science*, 386/6720. DOI: 10.1126/science.adl2746



- 78 WWF (n.d.). WWF Global Plastic Navigator, Must-haves in the treaty. Retrieved from: [plasticnavigator.wwf.de/#/en/policy/?st=0&ch=0&ui-info=0&ui-mpol=show%3Atrue&layers=surface-concentration%7Cpolicy-commitments\\_10&info=policy-commitments\\_10](https://plasticnavigator.wwf.de/#/en/policy/?st=0&ch=0&ui-info=0&ui-mpol=show%3Atrue&layers=surface-concentration%7Cpolicy-commitments_10&info=policy-commitments_10)
- 79 Ministère de l'Aménagement du territoire et de la Décentralisation (n.d.). The Nice wake up call for an ambitious plastics treaty. Retrieved from: [www.ecologie.gouv.fr/sites/default/files/documents/The%20Nice%20wake%20up%20call%20for%20an%20ambitious%20plastics%20treaty.pdf](https://www.ecologie.gouv.fr/sites/default/files/documents/The%20Nice%20wake%20up%20call%20for%20an%20ambitious%20plastics%20treaty.pdf)
- 80 WWF (2024). A Global Treaty To End Plastic Pollution—That People and Nature Need. Retrieved from: [wwfint.awsassets.panda.org/downloads/a-global-treaty-to-end-plastic-pollution-must-haves.pdf](https://www.wwf.org.uk/policy/a-global-treaty-to-end-plastic-pollution-must-haves.pdf)
- 81 Wagner, M., Monclús, L., Arp, H. P. H., Groh, K. J., Løseth, M. E., Muncke, J., Wang, Z., Wolf, R., Zimmermann, L. (2024). State of the science on plastic chemicals—Identifying and addressing chemicals and polymers of concern, *Zenodo*. <https://doi.org/10.5281/zenodo.10701705>
- 82 Pivnenko, K., Eriksen, M. K., Martín-Fernández, J. A., Eriksson, E., Astrup, T. F. (2016). Recycling of plastic waste: Presence of Phthalates in plastics from households and industry, *Waste Management*, 54: 44–52. DOI: 10.1016/j.wasman.2016.05.014
- 83 Gore, A. C., La Merrill, M. A., Patisaul, H. B., Sargis, R. (2024). Endocrine Disrupting Chemicals: Threats to Human Health. *The Endocrine Society and IPEN*.
- 84 Radke, E. G., Braun, J. M., Meeker, J. D., Cooper, G. S. (2018). Phthalates exposure and male reproductive outcomes: A systematic review of the human epidemiological evidence, *Environment International*, 121: 764–793. DOI: 10.1016/j.envint.2018.07.029
- 85 Eales, J., Bethel, A., Galloway, T., Hopkinson, P., Morrissey, K., Short, R. E., Garside, R. (2021). Human health impacts of exposure to Phthalates plasticizers: An overview of reviews, *Environment International*, 158: 106903. DOI: 10.1016/j.envint.2021.106903
- 86 Thurston, S. W., Mendiola, J., Bellamy, A. R., Levine, H., Wang, C., Sparks, A., Redmon, J. B. et al. (2015). Phthalates exposure and semen quality in fertile US men, *Andrology*, 4/4: 632–638. DOI: 10.1111/andr.12124
- 87 Chen, Q., Yang, H., Zhou, N., Sun, L., Bao, H., Tan, L., Chen, H. et al. (2017). Phthalates exposure, even below US EPA reference doses, was associated with semen quality and reproductive hormones: Prospective MARHCS study in general population, *Environment International*, 104: 58–68. DOI: 10.1016/j.envint.2017.04.005
- 88 Axelsson, J., Rylander, L., Rignell-Hydbom, A., Jönsson, B. A. G., Lindh, C. H., Giwercman, A. (2015). Phthalates exposure and reproductive parameters in young men from the general Swedish population, *Environment International*, 85: 54–60. DOI: 10.1016/j.envint.2015.07.005
- 89 Mínguez-Alarcón, L., Burns, J., Williams, P. L., Korricks, S. A., Lee, M. M., Bather, J. R., Kovalev, S. V. et al. (2022). Urinary Phthalates metabolite concentrations during four windows spanning puberty (prepuberty through sexual maturity) and association with semen quality among young Russian men, *International Journal of Hygiene and Environmental Health*, 243: 113977. DOI: 10.1016/j.ijheh.2022.113977
- 90 Yu, C., Lu, J., Zhao, J., Zhao, T., Long, C., Lin, T., Wu, S. et al. (2022). Maternal Phthalates exposure during pregnancy and male reproductive disorders: a systematic review and metaanalysis, *The Turkish Journal of Pediatrics*, 64/2: 187–209. DOI: 10.24953/turkjped.2020.2060
- 91 Bustamante-Montes, L. P., Hernández-Valero, M. A., Flores-Pimentel, D., García-Fábila, M., Amaya-Chávez, A., Barr, D. B., Borja-Aburto, V. H. (2013). Prenatal exposure to Phthalates is associated with decreased anogenital distance and penile size in male newborns, *Journal of Developmental Origins of Health and Disease*, 4/4: 300–306. DOI: 10.1017/s2040174413000172
- 92 Cai, W., Yang, J., Liu, Y., Bi, Y., Wang, H. (2019). Association between Phthalates Metabolites and Risk of Endometriosis: A Meta-Analysis, *International Journal of Environmental Research and Public Health*, 16/19: 3678. DOI: 10.3390/ijerph16193678
- 93 Iizuka, T., Yin, P., Zuberi, A., Kujawa, S., Coon, J. S., Björvang, R. D., Damdimopoulou, P. et al. (2022). Mono-(2-ethyl-5-hydroxyhexyl) Phthalates promotes uterine leiomyoma cell survival through tryptophan-kynurenine-AHR pathway activation, *Proceedings of the National Academy of Sciences*, 119/47. DOI: 10.1073/pnas.2208886119
- 94 Hu, W., Jin, Z., Wang, H., Wang, F., Qu, F. (2024). Relationship between Phthalates exposure, risk of decreased ovarian reserve, and oxidative stress levels, *Toxicology and Industrial Health*, 40/4: 156–166. DOI: 10.1177/07482337241229761
- 95 Nobles, C. J., Mendola, P., Kim, K., Pollack, A. Z., Mumford, S. L., Perkins, N. J., Silver, R. M. et al. (2023). Preconception Phthalates Exposure and Women's Reproductive Health: Pregnancy, Pregnancy Loss, and Underlying Mechanisms, *Environmental Health Perspectives*, 131/12. DOI: 10.1289/ehp12287
- 96 Ji, H., Wu, Z., Chen, D., Miao, M., Chen, H., Shuai, W., Liang, H. et al. (2023). Individual and joint effects of Phthalates exposure on the risk of early miscarriage, *Journal of Exposure Science & Environmental Epidemiology*, 34/4: 620–628. DOI: 10.1038/s41370-023-00533-1
- 97 Hoffman, S. S., Tang, Z., Dunlop, A., Brennan, P. A., Huynh, T., Eick, S. M., Barr, D. B. et al. (2025). Impact of prenatal Phthalates exposure on newborn metabolome and infant neurodevelopment, *Nature Communications*, 16/1. DOI: 10.1038/s41467-025-57273-z
- 98 Land, K. L., Ghuneim, S. M., Williams, B. A., Hannon, P. R. (2024). Phthalates Disrupt Female Reproductive Health: A Call for Enhanced Investigation into Mixtures, *Reproduction*. DOI: 10.1530/rep-24-0117
- 99 Van Wassenaer-Leemhuis, A., Ares, S., Golombok, S., Kok, J., Paneth, N., Kase, J., LaGamma, E. F. (2014). Thyroid Hormone Supplementation in Preterm Infants Born Before 28 Weeks Gestational Age and Neurodevelopmental Outcome at Age 36 Months, *Thyroid*, 24/7: 1162–1169. DOI: 10.1089/thy.2013.0618
- 100 Sprowles, J. L. N., Dzwilewski, K. L. C., Merced-Nieves, F. M., Musaad, S. M. A., Schantz, S. L., Geiger, S. D. (2022). Associations of prenatal Phthalates exposure with neurobehavioral outcomes in 4.5- and 7.5-month-old infants, *Neurotoxicology and Teratology*, 92: 107102. DOI: 10.1016/j.ntt.2022.107102

- 101 Martínez-Martínez, M. I., Alegre-Martínez, A., Cauli, O. (2021a). Prenatal exposure to Phthalates and its effects upon cognitive and motor functions: A systematic review, *Toxicology*, 463: 152980. DOI: 10.1016/j.tox.2021.152980
- 102 Zhang, Q., Chen, X.-Z., Huang, X., Wang, M., Wu, J. (2019). The association between prenatal exposure to Phthalates and cognition and neurobehavior of children-evidence from birth cohorts, *NeuroToxicology*, 73: 199–212. DOI: 10.1016/j.neuro.2019.04.007
- 103 Ghassabian, A., Van den Dries, M., Trasande, L., Lamballais, S., Spaan, S., Martínez-Moral, M.-P., Kannan, K. et al. (2023a). Prenatal exposure to common plasticizers: a longitudinal study on Phthalates, brain volumetric measures, and IQ in youth, *Molecular Psychiatry*, 28/11: 4814–4822. DOI: 10.1038/s41380-023-02225-6
- 104 Dzwilewski, K. L. C., Woodbury, M. L., Aguiar, A., Shoaff, J., Merced-Nieves, F., Korrick, S. A., Schantz, S. L. (2021). Associations of prenatal exposure to Phthalates with measures of cognition in 7.5-month-old infants, *NeuroToxicology*, 84: 84–95. DOI: 10.1016/j.neuro.2021.03.001
- 105 Navaranjan, G., Diamond, M. L., Harris, S. A., Jantunen, L. M., Bernstein, S., Scott, J. A., Takaro, T. K. et al. (2021). Early life exposure to Phthalates and the development of childhood asthma among Canadian children, *Environmental Research*, 197: 110981. DOI: 10.1016/j.envres.2021.110981
- 106 Whyatt, R. M., Perzanowski, M. S., Just, A. C., Rundle, A. G., Donohue, K. M., Calafat, A. M., Hoepner, L. A. et al. (2014). Asthma in Inner-City Children at 5–11 Years of Age and Prenatal Exposure to Phthalates: The Columbia Center for Children's Environmental Health Cohort, *Environmental Health Perspectives*, 122/10: 1141–1146. DOI: 10.1289/ehp.1307670
- 107 Kim, Y.-M., Kim, J., Cheong, H.-K., Jeon, B.-H., Ahn, K. (2018). Exposure to Phthalates aggravates pulmonary function and airway inflammation in asthmatic children, *PLoS ONE*, 13/12: e0208553. DOI: 10.1371/journal.pone.0208553
- 108 Oh, Y., Hong, S., Park, Y. J., Baek, I. (2024). Association between Phthalates exposure and risk of allergic rhinitis in children: A systematic review and meta-analysis, *Pediatric Allergy and Immunology*, 35/9. DOI: 10.1111/pai.14230
- 109 Bolaji, J. A., Bonvini, S. J., Wortley, M. A., Adcock, J. J., Dubuis, E., Carlsten, C., Tetley, T. D. et al. (2017). Phthalates trigger respiratory reflexes, *European Respiratory Journal*, PA4785. DOI: 10.1183/1393003.congress-2017.pa4785
- 110 Yu, Y., Wang, J. Q. (2022). Phthalates exposure and lung disease: the epidemiological evidences, plausible mechanism and advocacy of interventions, *Reviews on Environmental Health*, 39/1: 37–45. DOI: 10.1515/reveh-2022-0077
- 111 Mariana, M., Castelo-Branco, M., Soares, A. M., Cairrao, E. (2023). Phthalates' exposure leads to an increasing concern on cardiovascular health, *Journal of Hazardous Materials*, 457: 131680. DOI: 10.1016/j.jhazmat.2023.131680
- 112 Kabekkodu, S. P., Gladwell, L. R., Choudhury, M. (2024). The mitochondrial link: Phthalates exposure and cardiovascular disease, *Biochimica Et Biophysica Acta (BBA)—Molecular Cell Research*, 1871/4: 119708. DOI: 10.1016/j.bbamcr.2024.119708
- 113 Zhu, X., Yin, T., Yue, X., Liao, S., Cheang, I., Zhu, Q., Yao, W. et al. (2021). Association of urinary Phthalates metabolites with cardiovascular disease among the general adult population, *Environmental Research*, 202: 111764. DOI: 10.1016/j.envres.2021.111764
- 114 Mariana, M., Castelo-Branco, M., Soares, A. M., Cairrao, E. (2023a). Phthalates' exposure leads to an increasing concern on cardiovascular health, *Journal of Hazardous Materials*, 457: 131680. DOI: 10.1016/j.jhazmat.2023.131680
- 115 Kabekkodu, S. P., Gladwell, L. R., Choudhury, M. (2024a). The mitochondrial link: Phthalates exposure and cardiovascular disease, *Biochimica Et Biophysica Acta (BBA)—Molecular Cell Research*, 1871/4: 119708. DOI: 10.1016/j.bbamcr.2024.119708
- 116 Nevoit, G., Jarusevicius, G., Potyazhenko, M., Mintser, O., Bumblyte, I. A., Vainoras, A. (2025). Mitochondrial Dysfunction and Atherosclerosis: The Problem and the Search for Its Solution, *Biomedicines*, 13/4: 963. DOI: 10.3390/biomedicines13040963
- 117 Sumner, R. N., Tomlinson, M., Craigon, J., England, G. C. W., Lea, R. G. (2019). Independent and combined effects of diethylhexyl Phthalates and polychlorinated biphenyl 153 on sperm quality in the human and dog, *Scientific Reports*, 9/1. DOI: 10.1038/s41598-019-39913-9
- 118 Amjad, S., Rahman, M. S., Pang, W.-K., Ryu, D.-Y., Adegoke, E. O., Park, Y.-J., Pang, M.-G. (2021). Effects of Phthalates on the functions and fertility of mouse spermatozoa, *Toxicology*, 454: 152746. DOI: 10.1016/j.tox.2021.152746
- 119 Hasan, S., Mustari, A., Rafiq, K., Miah, M. A. (2025). Phthalates plasticizer affects blood electrolytes, hormones, and reproductive parameters of black Bengal goats, *Journal of Advanced Veterinary and Animal Research*, 0: 1. DOI: 10.5455/javar.2024.k856
- 120 Kougiyas, D. G., Sellinger, E. P., Willing, J., Juraska, J. M. (2018). Perinatal Exposure to an Environmentally Relevant Mixture of Phthalates Results in a Lower Number of Neurons and Synapses in the Medial Prefrontal Cortex and Decreased Cognitive Flexibility in Adult Male and Female Rats, *Journal of Neuroscience*, 38/31: 6864–6872. DOI: 10.1523/jneurosci.0607-18.2018
- 121 Morales-Grahl, E., Hilz, E. N., Gore, A. C. (2024). Regrettable Substitutes and the Brain: What Animal Models and Human Studies Tell Us about the Neurodevelopmental Effects of Bisphenol, Per- and Polyfluoroalkyl Substances, and Phthalates Replacements, *International Journal of Molecular Sciences*, 25/13: 6887. DOI: 10.3390/ijms25136887
- 122 Huang, W., Xiao, J., Shi, X., Zheng, S., Li, H., Liu, C., Wu, K. (2022). Effects of di-(2-ethylhexyl) Phthalates (DEHP) on behavior and dopamine signaling in zebrafish (*Danio rerio*), *Environmental Toxicology and Pharmacology*, 93: 103885. DOI: 10.1016/j.etap.2022.103885
- 123 Shi, H., Zhang, Z., Shen, A., Ding, T., Zhao, R., Shi, Y., Zhao, J. et al. (2025). Maternal di(2-ethylhexyl) Phthalates exposure increases the risk of congenital heart disease in mice offspring, *Pediatric Research*. DOI: 10.1038/s41390-025-03997-z
- 124 European Chemicals Agency (o. J.). Bisphenols. Retrieved from: <https://www.echa.europa.eu/hot-topics/bisphenols>



- 125 Gonkowski, S., Makowska, K. (2022). Environmental Pollution with Bisphenol A and Phthalates—A Serious Risk to Human and Animal Health, *International Journal of Environmental Research and Public Health*, 19/21: 13983. DOI: 10.3390/ijerph192113983
- 126 Rubin, A. M., Seebacher, F. (2022). Bisphenols impact hormone levels in animals: A meta-analysis, *The Science of The Total Environment*, 828: 154533. DOI: 10.1016/j.scitotenv.2022.154533
- 127 Chitakwa, N., Alqudaimi, M., Sultan, M., Wu, D. (2024). Plastic-related endocrine disrupting chemicals significantly related to the increased risk of estrogen-dependent diseases in women, *Environmental Research*, 252: 118966. DOI: 10.1016/j.envres.2024.118966
- 128 Wang, Z., Liu, H., Liu, S. (2016). Low-Dose Bisphenol A Exposure: A Seemingly Instigating Carcinogenic Effect on Breast Cancer, *Advanced Science*, 4/2. DOI: 10.1002/advs.201600248
- 129 Keshavarz-Maleki, R., Kaviani, A., Omranipour, R., Gholami, M., Khoshayand, M. R., Ostad, S. N., Sabzevari, O. (2021). Bisphenol-A in biological samples of breast cancer mastectomy and mammoplasty patients and correlation with levels measured in urine and tissue, *Scientific Reports*, 11/1. DOI: 10.1038/s41598-021-97864-6
- 130 Chowdhury, E.-U.-R., Banu, H., Morshed, M. S., Jahan, I. A., Kharel, S., Hasanat, M. A. (2025). Raised Bisphenol A has a Significant Association with Adverse Reproductive Manifestations rather than Biochemical or Hormonal Aberrations in Women with Polycystic Ovary Syndrome, *Journal of The ASEAN Federation of Endocrine Societies*. DOI: 10.15605/jafes.040.01.14
- 131 Gore, A. C., La Merrill, M. A., Patisau, H. B., Sargis, R. (2024). *Endocrine Disrupting Chemicals: Threats to Human Health*. The Endocrine Society and IPEN.
- 132 Liu, J., Tian, M., Qin, H., Chen, D., Mzava, S. M., Wang, X., & Bigambo, F. M. (2024). Maternal bisphenols exposure and thyroid function in children: a systematic review and meta-analysis, *Frontiers in Endocrinology*, 15. DOI: 10.3389/fendo.2024.1420540
- 133 Viguié, C., Collet, S. H., Gayrard, V., Picard-Hagen, N., Puel, S., Roques, B. B., Toutain, P.-L. et al. (2012). Maternal and Fetal Exposure to Bisphenol A Is Associated with Alterations of Thyroid Function in Pregnant Ewes and Their Newborn Lambs, *Endocrinology*, 154/1: 521–528. DOI: 10.1210/en.2012-1401
- 134 Costa, H. E., Cairrao, E. (2023a). Effect of bisphenol A on the neurological system: a review update, *Archives of Toxicology*, 98/1: 1–73. DOI: 10.1007/s00204-023-03614-0
- 135 Ebd.
- 136 Moon, S., Yu, S. H., Lee, C. B., Park, Y. J., Yoo, H. J., Kim, D. S. (2020). Effects of bisphenol A on cardiovascular disease: An epidemiological study using National Health and Nutrition Examination Survey 2003–2016 and meta-analysis, *The Science of the Total Environment*, 763: 142941. DOI: 10.1016/j.scitotenv.2020.142941
- 137 Prins, G. S., Patisaul, H. B., Belcher, S. M., Vandenberg, L. N. (2018). CLARITY-BPA academic laboratory studies identify consistent low-dose Bisphenol A effects on multiple organ systems, *Basic & Clinical Pharmacology & Toxicology*, 125/S3: 14–31. DOI: 10.1111/bcpt.13125
- 138 Wang, Z., Liu, H., Liu, S. (2016a). Low-Dose Bisphenol A Exposure: A Seemingly Instigating Carcinogenic Effect on Breast Cancer, *Advanced Science*, 4/2. DOI: 10.1002/advs.201600248
- 139 Kitamura, S., Kato, T., Iida, M., Jinno, N., Suzuki, T., Ohta, S., Fujimoto, N. et al. (2004). Anti-thyroid hormonal activity of tetrabromobisphenol A, a flame retardant, and related compounds: Affinity to the mammalian thyroid hormone receptor, and effect on tadpole metamorphosis, *Life Sciences*, 76/14: 1589–601. DOI: 10.1016/j.lfs.2004.08.030
- 140 Viguié, C., Collet, S. H., Gayrard, V., Picard-Hagen, N., Puel, S., Roques, B. B., Toutain, P.-L. et al. (2012a). Maternal and Fetal Exposure to Bisphenol A Is Associated with Alterations of Thyroid Function in Pregnant Ewes and Their Newborn Lambs, *Endocrinology*, 154/1: 521–528. DOI: 10.1210/en.2012-1401
- 141 Wang, Zhihao, Alderman, M. H., Asgari, C., Taylor, H. S. (2020). Fetal Bisphenol-A Induced Changes in Murine Behavior and Brain Gene Expression Persisted in Adult-aged Offspring, *Endocrinology*, 161/12. DOI: 10.1210/endocr/bqaa164
- 142 Morsy, M. M., Ahmad, M. M., Hassan, N. H. (2024). Maternal exposure to low-dose bisphenol A and its potential neurotoxic impact on male pups: A histological, immunohistochemical, and ultrastructural study, *Tissue and Cell*, 90: 102503. DOI: 10.1016/j.tice.2024.102503
- 143 Peremiquel-Trillas, P., Benavente, Y., Martín-Bustamante, M., Casabonne, D., Pérez-Gómez, B., Gómez-Acebo, I., Oliete-Canela, A. et al. (2018). Alkylphenolic compounds and risk of breast and prostate cancer in the MCC-Spain study, *Environment International*, 122: 389–399. DOI: 10.1016/j.envint.2018.12.007
- 144 He, S., Xiao, H., Luo, S., Li, X., Zhang, J.-D., Ren, X.-M., Yang, Y. et al. (2022). Benzotriazole Ultraviolet Stabilisers Promote Breast Cancer Cell Proliferation via Activating Estrogen-Related Receptors  $\alpha$  and  $\gamma$  at Human-Relevant Levels, *Environmental Science & Technology*, 56/4: 2466–2475. DOI: 10.1021/acs.est.1c03446
- 145 Wen, H.-J., Chang, T.-C., Ding, W.-H., Tsai, S.-F., Hsiung, C. A., Wang, S.-L. (2020). Exposure to endocrine disruptor alkylphenols and the occurrence of endometrial cancer, *Environmental Pollution*, 267: 115475. DOI: 10.1016/j.envpol.2020.115475
- 146 Mustieles, V., Balogh, R. K., Axelstad, M., Montazeri, P., Márquez, S., Vrijheid, M., Draskau, M. K. et al. (2023). Benzophenone-3: Comprehensive review of the toxicological and human evidence with meta-analysis of human biomonitoring studies, *Environment International*, 173: 107739. DOI: 10.1016/j.envint.2023.107739
- 147 Pang, L., Wei, H., Wu, Y., Yang, K., Wang, X., Long, J., Chen, M. et al. (2024a). Exposure to alkylphenols during early pregnancy and the risk of gestational diabetes mellitus: Fetal sex-specific effects, *Ecotoxicology and Environmental Safety*, 287: 117270. DOI: 10.1016/j.ecoenv.2024.117270
- 148 Chen, M., Liang, J., Wei, H., Mu, C., Tang, Y., Wu, X., Jiang, Q. et al. (2024). Association of alkylphenols exposure with serum liver function markers in pregnant women in Guangxi, China, *Ecotoxicology and Environmental Safety*, 282: 116676. DOI: 10.1016/j.ecoenv.2024.116676

- 149 Scinicariello, F., Buser, M. C. (2016). Serum Testosterone Concentrations and Urinary Bisphenol A, Benzophenone-3, Triclosan, and Paraben Levels in Male and Female Children and Adolescents: NHANES 2011–2012, *Environmental Health Perspectives*, 124/12: 1898–904. DOI: 10.1289/ehp150
- 150 Huo, W., Cai, P., Chen, M., Li, H., Tang, J., Xu, C., Zhu, D., et al. (2015). The relationship between prenatal exposure to BP-3 and Hirschsprung's disease, *Chemosphere*, 144: 1091–1097. DOI: 10.1016/j.chemosphere.2015.09.019
- 151 Nie, Y., Liu, H., Wu, R., Fan, J., Yang, Y., Zhao, W., Bao, J. et al. (2024). Interference with SPARC inhibits Benzophenone-3 induced ferroptosis in osteoarthritis: Evidence from bioinformatics analyses and biological experimentation, *Ecotoxicology and Environmental Safety*, 274: 116217. DOI: 10.1016/j.ecoenv.2024.116217
- 152 Sakuragi, Y., Takada, H., Sato, H., Kubota, A., Terasaki, M., Takeuchi, S., Ikeda-Araki, A. et al. (2021). An analytical survey of benzotriazole UV stabilisers in plastic products and their endocrine-disrupting potential via human estrogen and androgen receptors, *The Science of The Total Environment*, 800: 149374. DOI: 10.1016/j.scitotenv.2021.149374
- 153 Shi, R., Liu, Z., Liu, T. (2021). The antagonistic effect of bisphenol A and nonylphenol on liver and kidney injury in rats, *Immunopharmacology and Immunotoxicology*, 43/5: 527–535. DOI: 10.1080/08923973.2021.1950179
- 154 Kim, J., Kang, E.-J., Park, M.-N., Kim, J.-E., Kim, S.-C., Jeung, E.-B., Lee, G.-S. et al. (2015). The adverse effect of 4-tert-octylphenol on fat metabolism in pregnant rats via regulation of lipogenic proteins, *Environmental Toxicology and Pharmacology*, 40/1: 284–291. DOI: 10.1016/j.etap.2015.06.020
- 155 Yu, J., Luo, Y., Yang, X. F., Yang, M. X., Yang, J., Yang, X. S., Zhou, J. et al. (2016). Effects of perinatal exposure to nonylphenol on delivery outcomes of pregnant rats and inflammatory hepatic injury in newborn rats, *Brazilian Journal of Medical and Biological Research*, 49/12. DOI: 10.1590/1414-431x20165647
- 156 Gore, A. C., La Merrill, M. A., Patisau, H. B., Sargis, R. (2024). *Endocrine Disrupting Chemicals: Threats to Human Health*. The Endocrine Society and IPEN.
- 157 Blake, B. E., Fenton, S. E. (2020). Early life exposure to per- and polyfluoroalkyl substances (PFAS) and latent health outcomes: A review including the placenta as a target tissue and possible driver of peri- and postnatal effects, *Toxicology*, 443: 152565. DOI: 10.1016/j.tox.2020.152565
- 158 Gore, A. C., La Merrill, M. A., Patisau, H. B., Sargis, R. (2024). *Endocrine Disrupting Chemicals: Threats to Human Health*. The Endocrine Society and IPEN.
- 159 Van Gerwen, M., Colicino, E., Guan, H., Dolios, G., Nadkarni, G. N., Vermeulen, R. C. H., Wolff, M. S. et al. (2023). Per- and polyfluoroalkyl substances (PFAS) exposure and thyroid cancer risk, *EBioMedicine*, 97: 104831. DOI: 10.1016/j.ebiom.2023.104831
- 160 Seyyedsalehi, M. S., Boffetta, P. (2023). Per- and Polyfluoroalkyl Substances (PFAS) Exposure and Risk of Kidney, Liver, and Testicular Cancers: A Systematic Review and Meta-Analysis, *La Medicina Del Lavoro*, 114/5: e2023040. DOI: 10.23749/mdl.v114i5.15065
- 161 Wang, Y., Zhang, J., Zhang, J., Hou, M., Kong, L., Lin, X., Xu, J. et al. (2024). Association between per- and polyfluoroalkyl substances exposure and prevalence of chronic obstructive pulmonary disease: The mediating role of serum albumin, *The Science of The Total Environment*, 925: 171742. DOI: 10.1016/j.scitotenv.2024.171742
- 162 Dragon, J., Hoaglund, M., Badireddy, A. R., Nielsen, G., Schlezinger, J., Shukla, A. (2023). Perfluoroalkyl Substances (PFAS) Affect Inflammation in Lung Cells and Tissues, *International Journal of Molecular Sciences*, 24/10: 8539. DOI: 10.3390/ijms24108539
- 163 Wang, Y., Zhang, J., Zhang, J., Hou, M., Kong, L., Lin, X., Xu, J. et al. (2024a). Association between per- and polyfluoroalkyl substances exposure and prevalence of chronic obstructive pulmonary disease: The mediating role of serum albumin, *The Science of The Total Environment*, 925: 171742. DOI: 10.1016/j.scitotenv.2024.171742
- 164 Von Holst, H., Nayak, P., Dembek, Z., Buehler, S., Echeverria, D., Fallacara, D., John, L. (2021). Perfluoroalkyl substances exposure and immunity, allergic response, infection, and asthma in children: review of epidemiologic studies, *Heliyon*, 7/10: e08160. DOI: 10.1016/j.heliyon.2021.e08160
- 165 Crute, C. E., Hall, S. M., Landon, C. D., Garner, A., Everitt, J. I., Zhang, S., Blake, B. et al. (2022). Evaluating maternal exposure to an environmental per and polyfluoroalkyl substances (PFAS) mixture during pregnancy: Adverse maternal and fetoplacental effects in a New Zealand White (NZW) rabbit model, *The Science of The Total Environment*, 838: 156499. DOI: 10.1016/j.scitotenv.2022.156499
- 166 Rickard, B. P., Rizvi, I., Fenton, S. E. (2021). Per- and poly-fluoroalkyl substances (PFAS) and female reproductive outcomes: PFAS elimination, endocrine-mediated effects, and disease, *Toxicology*, 465: 153031. DOI: 10.1016/j.tox.2021.153031
- 167 Gore, A. C., La Merrill, M. A., Patisau, H. B., Sargis, R. (2024). *Endocrine Disrupting Chemicals: Threats to Human Health*. The Endocrine Society and IPEN.
- 168 Zainab, B., Ayaz, Z., Rashid, U., Farraj, D. A. A., Alkufeidy, R. M., AlQahtany, F. S., Aljowaid, R. M. et al. (2021). Role of Persistent Organic Pollutants in Breast Cancer Progression and Identification of Estrogen Receptor Alpha Inhibitors Using In-Silico Mining and Drug-Drug Interaction Network Approaches, *Biology*, 10/7: 681. DOI: 10.3390/biology10070681
- 169 Kim, S.-H., Nam, K.-H., Hwang, K.-A., Choi, K.-C. (2016). Influence of hexabromocyclododecane and 4-nonylphenol on the regulation of cell growth, apoptosis and migration in prostatic cancer cells, *Toxicology in Vitro*, 32: 240–247. DOI: 10.1016/j.tiv.2016.01.008
- 170 Gorini, F., Iervasi, G., Coi, A., Pitto, L., Bianchi, F. (2018). The Role of Polybrominated Diphenyl Ethers in Thyroid Carcinogenesis: Is It a Weak Hypothesis or a Hidden Reality? From Facts to New Perspectives, *International Journal of Environmental Research and Public Health*, 15/9: 1834. DOI: 10.3390/ijerph15091834
- 171 Saquib, Q., Al-Salem, A. M., Siddiqui, M. A., Ansari, S. M., Zhang, X., Al-Khedhairi, A. A. (2022). Organophosphorus Flame Retardant TDCPP Displays Genotoxic and Carcinogenic Risks in Human Liver Cells, *Cells*, 11/2: 195. DOI: 10.3390/cells11020195



- 172 Costa, L. G., De Laat, R., Tagliaferri, S., Pellacani, C. (2013). A mechanistic view of polybrominated diphenyl ether (PBDE) developmental neurotoxicity, *Toxicology Letters*, 230/2: 282–94. DOI: 10.1016/j.toxlet.2013.11.011
- 173 Poston, R. G., Saha, R. N. (2019). Epigenetic Effects of Polybrominated Diphenyl Ethers on Human Health, *International Journal of Environmental Research and Public Health*, 16/15: 2703. DOI: 10.3390/ijerph16152703
- 174 Lv, J.-J., Zhang, Y.-C., Li, X.-Y., Zhang, L.-J., Yixi, Z.-M., Yang, C.-H., Wang, X.-H. (2024). The association between brominated flame retardants exposure with Parkinson's disease in US adults: a cross-sectional study of the National Health and Nutrition Examination Survey 2009–2016, *Frontiers in Public Health*, 12. DOI: 10.3389/fpubh.2024.1451686
- 175 Mendy, A., Percy, Z., Braun, J. M., Lanphear, B., La Guardia, M. J., Hale, R., Yolton, K. et al. (2023a). Exposure to dust organophosphate and replacement brominated flame retardants during infancy and risk of subsequent adverse respiratory outcomes, *Environmental Research*, 235: 116560. DOI: 10.1016/j.envres.2023.116560
- 176 Luo, K., Zhang, R., Aimuzi, R., Wang, Y., Nian, M., Zhang, J. (2020). Exposure to Organophosphate esters and metabolic syndrome in adults, *Environment International*, 143: 105941. DOI: 10.1016/j.envint.2020.105941
- 177 Dunnick, J. K., Sanders, J. M., Kissling, G. E., Johnson, C. L., Boyle, M. H., Elmore, S. A. (2014). Environmental Chemical Exposure May Contribute to Uterine Cancer Development: studies with tetrabromobisphenol A, *Toxicologic Pathology*, 43/4: 464–473. DOI: 10.1177/0192623314557335
- 178 Guo, Z., Zhang, L., Liu, X., Yu, Y., Liu, S., Chen, M., Huang, C. et al. (2019). The enrichment and purification of hexabromocyclododecanes and its effects on thyroid in zebrafish, *Ecotoxicology and Environmental Safety*, 185: 109690. DOI: 10.1016/j.ecoenv.2019.109690
- 179 Usenko, C., Abel, E., Hopkins, A., Martinez, G., Tijerina, J., Kudela, M., Norris, N. et al. (2016). Evaluation of Common Use Brominated Flame Retardant (BFR) Toxicity Using a Zebrafish Embryo Model, *Toxics*, 4/3: 21. DOI: 10.3390/toxics4030021
- 180 Reffatto, V., Rasinger, J. D., Carroll, T. S., Ganay, T., Lundebye, A. -K., Sekler, I., Hershinkel, M. et al. (2017). Parallel in vivo and in vitro transcriptomics analysis reveals calcium and zinc signalling in the brain as sensitive targets of HBCD neurotoxicity, *Archives of Toxicology*, 92/3: 1189–1203. DOI: 10.1007/s00204-017-2119-2
- 181 Turner, A., Filella, M. (2021). Hazardous metal additives in plastics and their environmental impacts, *Environment International*, 156: 106622. DOI: 10.1016/j.envint.2021.106622
- 182 James M. Brown Ltd., EU Restrictions on the use of Cadmium Pigments (n.d.). Retrieved from: <https://www.jamesmbrown.co.uk/regulatory-affairs/eu-restrictions>
- 183 Turner, A., Filella, M. (2020). Lead in plastics—Recycling of legacy material and appropriateness of current regulations, *Journal of Hazardous Materials*, 404: 124131. DOI: 10.1016/j.jhazmat.2020.124131
- 184 Giulioni, C., Maurizi, V., De Stefano, V., Polisini, G., Teoh, J. Y.-C., Milanese, G., Galosi, A. B. et al. (2023). The influence of lead exposure on male semen parameters: A systematic review and meta-analysis, *Reproductive Toxicology*, 118: 108387. DOI: 10.1016/j.reprotox.2023.108387
- 185 Chitakwa, N., Alqudaimi, M., Sultan, M., Wu, D. (2024a). Plastic-related endocrine disrupting chemicals significantly related to the increased risk of estrogen-dependent diseases in women, *Environmental Research*, 252: 118966. DOI: 10.1016/j.envres.2024.118966
- 186 Chung, H.-K., Nam, J. S., Ahn, C. W., Lee, Y. S., Kim, K. R. (2015). Some Elements in Thyroid Tissue are Associated with More Advanced Stage of Thyroid Cancer in Korean Women, *Biological Trace Element Research*, 171/1: 54–62. DOI: 10.1007/s12011-015-0502-5
- 187 Peana, M., Pelucelli, A., Chasapis, C. T., Perlepes, S. P., Bekiari, V., Medici, S., Zoroddu, M. A. (2022). Biological effects of human exposure to environmental cadmium, *Biomolecules*, 13/1: 36. DOI: 10.3390/biom13010036
- 188 Mason, L. H., Harp, J. P., Han, D. Y. (2014). Pb Neurotoxicity: Neuropsychological Effects of Lead Toxicity, *BioMed Research International*, 2014: 1–8. DOI: 10.1155/2014/840547
- 189 Parithathi, A., Choudhary, N., Dsouza, H. S. (2024). Prenatal and early life lead exposure induced neurotoxicity, *Human & Experimental Toxicology*, 43. DOI: 10.1177/09603271241285523
- 190 Navas-Acien, A., Guallar, E., Silbergeld, E. K., Rothenberg, S. J. (2006). Lead Exposure and Cardiovascular Disease—A Systematic Review, *Environmental Health Perspectives*, 115/3: 472–482. DOI: 10.1289/ehp.9785
- 191 Lin, H.-C., Hao, W.-M., Chu, P.-H. (2021). Cadmium and cardiovascular disease: An overview of pathophysiology, epidemiology, therapy, and predictive value, *Revista Portuguesa De Cardiologia (English Edition)*, 40/8: 611–617. DOI: 10.1016/j.repce.2021.07.031
- 192 Hong, H., Xu, Y., Xu, J., Zhang, J., Xi, Y., Pi, H., Yang, L. et al. (2021). Cadmium exposure impairs pancreatic  $\beta$ -cell function and exaggerates diabetes by disrupting lipid metabolism, *Environment International*, 149: 106406. DOI: 10.1016/j.envint.2021.106406
- 193 Ma, Y., Su, Q., Yue, C., Zou, H., Zhu, J., Zhao, H., Song, R. et al. (2022). The Effect of Oxidative Stress-Induced Autophagy by Cadmium Exposure in Kidney, Liver, and Bone Damage, and Neurotoxicity, *International Journal of Molecular Sciences*, 23/21: 13491. DOI: 10.3390/ijms232113491
- 194 Verzelloni, P., Urbano, T., Wise, L. A., Vinceti, M., Filippini, T. (2024). Cadmium exposure and cardiovascular disease risk: A systematic review and dose-response meta-analysis, *Environmental Pollution*, 345: 123462. DOI: 10.1016/j.envpol.2024.123462
- 195 Assi, M. A., Hezmee, M. N. M., Haron, A. W., Sabri, M. Y., Rajion, M. A. (2016). The detrimental effects of lead on human and animal health, *Veterinary World*, 9/6: 660–671. DOI: 10.14202/vetworld.2016.660-671
- 196 Huff, J., Lunn, R. M., Waalkes, M. P., Tomatis, L., Infante, P. F. (2007). Cadmium-induced Cancers in Animals and in Humans, *International Journal of Occupational and Environmental Health*, 13/2: 202–212. DOI: 10.1179/oeh.2007.13.2.202
- 197 Bhattacharyya, M. H. (2009). Cadmium osteotoxicity in experimental animals: mechanisms and relationship to human exposures, *Toxicology and applied Pharmacology*, 238/3: 258–265. DOI: 10.1016/j.taap.2009.05.015







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