Australian Academy of Health & Medical Sciences

Microplastics, forever chemicals and other contaminants

An evidence brief

March 2026

Public debate about micro and nanoplastics (MNPs), forever chemicals (PFAS) and other contaminants (including phthalates, pesticides, endocrine disrupting chemicals (EDCs) and plastic additive chemicals) has intensified in recent months, with growing media coverage and community concern about their potential health effects[i].

At the same time, scientific discussion about the reliability of current MNPs measurement techniques has highlighted important uncertainties in the evidence base. While we know that these substances are present in the environment and in human bodies, it is not yet established whether – or at what levels – they impact human health.[ii]

In this context, the Australian Academy of Health and Medical Sciences has produced this evidence brief to provide an independent, expert assessment of the current science. By evaluating and synthesising the most relevant recent research, this brief sets out what is known, what is plausible, and what remains uncertain – and identifies precautionary policy approaches that are warranted now, even as the evidence continues to develop.

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[i] *Key terms are defined in a glossary at the end of this evidence brief.*

[ii] *This evidence brief focuses primarily on MNPs and PFAS because these contaminants have received significant public attention and are supported by a comparatively more developed evidence base on human exposure, bioaccumulation and potential health impacts than many other emerging contaminants of concern, though this evidence continues to evolve.*

Glossary

Highlighted terms indicate entries explained further in the glossary; please refer to it for additional details.

Executive summary

- Microplastics, forever chemicals and other contaminants comprise a diverse set of substances and particles. Each possesses distinct properties that shape whether and how it accumulates in the human body, the techniques needed to measure its presence, and the biological mechanisms through which it may affect human health.
- Micro- and nanoplastics (MNPs, which are small polymer plastic particles) and chemical contaminants such as per- and polyfluoroalkyl substances (PFAS) are widespread across the environment, and in animal and human bodies.
- Existing analytical techniques currently limit our ability to accurately measure the exact amounts of many smaller MNPs present in environments and bodies.[1] This is one of the main reasons why the health risks of MNPs remain uncertain.
- Scientists cannot deliberately expose human subjects to toxic doses of contaminants in experimental settings, so establishing causal relationships between exposures and health outcomes depends on association evidence from observational studies in the real world, and evidence from laboratory studies of how the substances cause harm.
- There is currently insufficient evidence to say for certain that any level of exposure to MNPs, PFAS and other contaminants is safe – though this does not mean no safe level exists. However, these contaminants are being widely used across domestic, commercial, and other everyday settings. This situation demands a precautionary approach across four key areas, outlined below.

Policy approaches

1. Building the evidence base

We encourage the Federal Government to partner with the health and medical research sector to refine techniques for detecting, sampling and quantifying contaminants, and establish a national human biomonitoring (HBM) program covering PFAS and other contaminants that can be accurately measured, with MNPs incorporated once measurement techniques are sufficiently advanced. This will enable the resourcing of meaningful longitudinal human studies of representative Australian cohorts to examine potential connections between contaminant exposure and health outcomes.

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2. Prioritising vulnerable populations

Not all communities are equally exposed. Until we understand whether there are safe levels of exposure to all such contaminants, the Federal Government should partner with the health and medical research sector and affected communities to take reasonable precautionary measures. These should include immediately establishing an interim HBM program for pregnant women (similar to those operating across all G7 and many OECD countries), and partnering with lower socio-economic communities and communities living in close proximity to contaminated sites (including some Aboriginal and Torres Strait Islander communities) to design and implement culturally informed, community-led responses to PFAS and related contamination.

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3. Reviewing regulation

As scientific techniques and evidence evolve, the Federal Government should review and update regulation relating to MNPs, PFAS and other contaminants; adopt a precautionary principle where uncertainty exists; and work closely with State and Territory governments towards consistent national implementation.

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4. Supporting public awareness and exposure reduction

Australian governments should ensure that clear, accessible and evidence-based information is available to all communities on known sources of exposure to MNPs, PFAS and other contaminants.

The current science: What we do and do not know

Scientific understanding of MNPs, PFAS and other contaminants is nuanced and evolving. Our ability to measure their presence and analyse their effects varies from substance to substance. Box 1 outlines definitions of these contaminants, which are also included with other key and technical terms in the glossary at the end of this evidence brief.

We do know that MNPs, PFAS and other contaminants are present across the environment, and that animals and humans are exposed to them through different pathways.

Human exposure to MNPs occurs primarily through ingestion and inhalation.[2,3] Dermal contact could be a third potential route, though evidence for direct skin absorption remains limited and the mechanisms are not yet well understood.[4] Human exposure to PFAS occurs through ingestion of food (i.e. food that is contaminated, or packaged in or cooked with, PFAS-containing products), ingestion of contaminated drinking water, incidental ingestion of dust or soil, and everyday products such as non-stick cookware and food packaging. PFAS exposure also occurs via inhalation – including of everyday house dust, and in occupational or fire-training settings, and infants are exposed in the uterus and through breastfeeding. Skin absorption is considered minimal for most PFAS, though data for newer compounds remain limited.[5] EDCs, meanwhile, are commonly ingested and absorbed – particularly through use of everyday personal care products.

Box 1. Definitions

Microplastics
Plastic particles generally smaller than 5 millimetres in diameter.

Micro- and nanoplastics (MNPs)
A collective term to describe plastic particles across micro- and nanoscale ranges.

Nanoplastics
Plastic particles typically smaller than 1 micrometre (and often <100 nanometres), with distinct physiochemical properties due to their size.

Per- and polyfluoroalkyl substances (PFAS)
A group of almost 15,000 synthetic "forever" chemicals that are widespread across environments and therefore in human bodies.

Endocrine disrupting chemicals
Synthetic or naturally occurring substances found in the environment that interfere with the body's hormonal system. They can mimic, block, or alter the effects of natural hormones.

We do know that exposure to MNPs, PFAS and other contaminants can lead to many of these substances bioaccumulating in the human body.

A 2022-2024 study of 7,000 blood samples detected three types of PFAS in 85% of the Australian population.[6] In recent years, MNPs have been detected in a variety of human tissues, including the liver, kidney, placenta, blood, brains and semen.[7–11] We also know that, due to their different sizes and properties, exposure to different particles (i.e. of contaminants such as MNPs) leads to different mechanisms and degrees of bioaccumulation. For example, when it comes to plastics, each type of plastic is made from distinct polymers with specific chemical structures, and nanoplastics – because they are smaller than microplastics – move more easily through biological barriers, such as cell membranes.[12,13]

We do not know the degree to which MNPs bioaccumulate in the human body.

Detecting and measuring MNPs smaller than 20 micrometres (i.e. 0.02 millimetres) remains a major analytical challenge, and is also complicated by sample contamination and standardization issues:[1,12]

Methods of measuring MNPs, and of describing the accuracy of these measurements, are less developed than those for PFAS and PCBs.[12]

- **Analytical challenges:** Available techniques broadly fall into two categories: microscopy- and mass spectrometry-based approaches. Microscopy-based approaches can provide detailed information on particle size, morphology and localization at the individual particle level, but are currently limited in their ability to detect the smallest particles relevant to systemic exposure, typically below the micrometre scale. In addition, their particle-by-particle nature makes it difficult to estimate total exposure. Mass spectrometry-based approaches can quantify total polymer mass without a lower size limit, but require destructive sample processing (e.g. pyrolysis, where the sample is heated to break it down), and rely on indirect detection of breakdown products. A key challenge is distinguishing plastic-derived signals from overlapping signals produced by endogenous biological molecules. Ongoing methodological development is focused on improving the specificity and reliability of these approaches.[12]

- **Sample contamination**: The prevalence of MNPs in the human body, combined with the diversity of these particles' properties, makes it especially difficult to isolate a specific type of MNP in order to study its behaviour in the body.[12] In addition, techniques to separate MNPs from human tissues and from contamination introduced during the sampling process are still being refined.[12,14] Collecting human samples is highly vulnerable to MNP contamination, because the environments in which samples are typically gathered – whether a non-sterile living setting or an operating room filled with plastic equipment – are themselves sources of MNP exposure.[15]
- **Sampling standardisation**: Human tissue samples from different years and locations are collected and processed differently, under varying contamination-minimising protocols. This makes it difficult to compare samples.[14]
- **Sample availability**: Obtaining samples needed to measure bioaccumulation of MNPs in particular organs and body systems is more difficult than it is for others. For example, it is much more invasive, costly and complex to obtain egg samples from females than sperm from males.

We do know that some communities are disproportionately more likely to have higher exposure and bioaccumulation rates of certain contaminants

Including people in lower-income, rural, remote, or industrial areas; families with limited access to non-plastic consumer alternatives, some culturally and linguistically diverse (CALD) communities, and workers in certain occupations.[16–18]

We do not know the toxicity thresholds of many contaminants

i.e. the levels at which they may begin to cause harm to human health. This is because the presence of a substance in the body does not necessarily mean it is causing harm. For this reason, this evidence brief uses the term 'contaminants' rather than 'toxicants'.

We do know some of the possible biological mechanisms through which toxic concentrations of contaminants may potentially be impacting human health.

This evidence brief examines two of the most researched mechanisms – endocrine disruption, and inflammation and oxidative stress – and presents case studies that illustrate the limits of current research into the health impacts of these mechanisms (see the 'Contaminants and the human body' section below).

We do not know for certain how many contaminants may potentially be contributing to long-term health impacts across the Australian population.

We can only assess the biological risk and track the potential health impacts of contaminants where we are able to measure exposure and bioaccumulation. For this reason, the health risks of MNPs are uncertain, and will remain so until methods for detecting and measuring smaller particles are sufficiently advanced. Toxicological and epidemiological evidence for specific health outcomes in humans is limited. Much of what we know does not come from directly studying human populations, but from indirect sources such as in vitro and animal studies, which often use exposure levels that may not match those seen in the general population.[19]

Even studies that do look at human populations are more observational and therefore are more likely to show association, not necessarily causation.

Even where the methods for detecting and measuring a class of chemical contaminant are more advanced, larger-scale, representative datasets are required to enable the longitudinal research needed to better understand possible connections between exposure and multiple health outcomes. To understand the prevalence of PFAS and other chemical contaminants across the Australian population, and to explore potential links between exposure and health outcomes, we must begin tracking levels of PFAS (which bioaccumulates in the human body) as well as other substances that may be rapidly metabolised but are still relevant to human health. Unlike comparable nations such as Canada, the US, Germany, South Korea and Japan, Australia does not currently operate a HBM program.[20,21]

We do know that strengthening Australia's measuring, monitoring and reporting techniques and infrastructure will enable us to understand better the potential health impacts of contaminants.

We also know that there are proportionate precautionary policy levers that can be used now to mitigate these. These measures include the Federal Government working with the Australian Academy of Health and Medical Sciences and other relevant stakeholders to develop and implement a comprehensive national HBM, as recommended by the Senate Select Committee on PFAS in November 2025 (see Box 2 below).[22] This could start with smaller, easily established programs targeted at priority populations such as pregnant women and some First Nations communities (see the 'Precautionary action' section of this evidence brief below).[22–24]

Box 2. Developing an Australian human biomonitoring program

December 2024

AAHMS called for the development of a national human biomonitoring (HBM) program to track exposure to PFAS and other contaminants.[23]

August 2025

AAHMS published a policy statement explaining how Australia could establish an interim and longer-term HBM program to monitor exposure to PFAS and other contaminants.[24]

November 2025

The Senate Select Committee on PFAS recommended that "the Australian Government consults with the Australian Academy of Health and Medical Sciences and other relevant stakeholders on designs for a national, longitudinal chemicals biomonitoring program [...] and associated longitudinal PFAS health research projects."[22]

Contaminants and the human body: Potential pathways to health impacts

Limitations of existing techniques for accurately detecting and measuring MNPs mean it is challenging to identify doses at which these contaminants may cause harm, making it difficult to establish causal links between exposure and disease.

Techniques for tracing and quantifying PFAS and EDCs in humans are more advanced, but the challenge of linking exposure to health outcomes remains. However, a growing body of mechanistic (how the contaminants act in the body), toxicological and epidemiological research points to biologically plausible pathways through which certain contaminants – if sufficient doses are reached – could contribute to adverse health outcomes.

Two of the most studied potential pathways are:

- Endocrine disruption.
- Inflammation and oxidative stress.

These pathways do not exist in isolation – they can be caused by processes and factors unrelated to contaminant exposure, and their effects can vary across life stage and co-existing environmental and social determinants of health.

Endocrine disruption

The endocrine system is a complex network of glands and organs that synthesise, store, and secrete hormones directly into the bloodstream. These hormones act as chemical messengers, regulating essential functions including metabolism, growth, reproduction, sleep and mood.[25] By maintaining hormonal balance, the endocrine system supports stability across every organ system in the body.

While this Evidence Brief focuses primarily on MNPs and PFAS, these groups intersect with a broader class of contaminants for which a more substantial evidence base exists: endocrine-disrupting chemicals (EDCs).

Endocrine-disrupting chemicals (EDCs) are a broad category of external substances that can interfere with internal hormone signaling. PFAS are recognised as EDCs, and many MNPs contain EDCs.

Present in air, dust, water, food, consumer products and manufactured materials, at certain doses, EDCs can disrupt the body's finely balanced endocrine system.[26] This interference can occur even at relatively low doses, and effects may vary depending on when the exposure happens and at what stage of life, as well as environmental or social factors.

A substantial body of largely animal-based toxicological and epidemiological research has identified associations between EDC exposure and a broad range of adverse health outcomes. These include developmental, reproductive, neurological, cardiovascular, immune, metabolic and endocrine disorders, including fertility, thyroid and pancreas dysfunction, with concerns that some effects may extend to future generations via intergenerational or epigenetic, prenatal and early life exposure.[26–29]

PFAS are among the chemical groups recognised as EDCs, with a substantial and growing body of evidence linking exposure to a range of adverse health outcomes. This includes consistently observed correlations in human populations, supported by mechanistic evidence from laboratory studies that reveal show how PFAS act in the body.[30,31] Uncertainties remain regarding the magnitude of effects at different exposure levels and across populations. However, despite these uncertainties, and although much of the current evidence around the health impacts of EDCs remains observational, mechanistic laboratory findings and consistency across a growing number of studies indicate the strong possibility of causal relationships between exposure to some EDCs and certain health outcomes, which warrants further investigation.

Case study: Reproductive health and fertility

Summary: The current evidence has not established whether (or the degree to which), PFAS and other sources of most types of EDCs may impair fertility, although it does suggest a plausible biological mechanism that warrants further investigation. However, it also does not determine safe levels of exposure or rule out reproductive harm.[34] Improving our understanding of the impacts of EDC-containing contaminants on fertility will require better methods of quantifying bioaccumulation and more longitudinal human research.

Associations between EDCs exposure and reproductive health and fertility are increasingly supported by population-level observations of reduced semen quality, altered levels of estradiol (the primary female sex hormone), luteinizing hormone or LH (which triggers ovulation in women and testosterone production in men), and follicle-stimulating hormone or FSH (which regulates egg and sperm development), diminished ovarian reserve (a reduction in the number or quality of a woman's remaining eggs), polycystic ovary syndrome (a hormonal condition in which the ovaries produce excess male hormones), spontaneous abortion, preterm birth, and less favourable assisted reproduction outcomes.[32,33] However, causality has not been established in most of these cases.[iii,32,33] An observed association between EDC exposure and a health outcome does not, on its own, confirm that the exposure caused the outcome – other factors may also play a role and are difficult to fully account for in observational research. However, findings brought together in systematic reviews, and supportive experimental studies to demonstrate a biological mechanism, provide compelling provisional evidence.

Emerging human biomonitoring research has detected multiple synthetic chemicals, including PFAS, parabens, plastic additives, UV filters and antimicrobials, within human follicular and seminal fluids (i.e. the microenvironments where eggs and sperm develop).[34] Sunscreen ingredients and industrial chemicals have been shown to cross the blood-follicle barrier (i.e. a barrier in the ovary that regulates the exchange of substances between the blood and developing ovarian follicles) and blood-testis barrier, structures previously assumed to offer meaningful protection, and blood PFAS concentrations reliably reflect chemical levels within the ovarian environment.[34]

Findings across studies of semen quality, reproductive hormones and time taken to become pregnant, are mixed, with some reporting positive associations, some negative and others finding no association with exposure to EDCs. [32] Studies have found no consistent exposure threshold that reliably predicts impaired fertility.[32]

US data suggests that exposure to EDCs tends to be higher among non-white populations. These populations tend to carry higher concentrations of EDCs (including phthalates, parabens, flame retardants, and related chemicals) found in everyday products.[35] These populations also experience higher rates of earlier puberty, uterine fibroids (non-cancerous growths in the womb that can affect fertility and pregnancy), infertility, and pregnancy complications.[35] It is not clear whether EDC exposure itself drives these disparities, or whether co-occurring social and structural determinants are the primary contributors, with both exposure profiles and health outcomes strongly shaped by socially patterned product use, diet, housing and environmental conditions.[35]

[iii] *Comparatively stronger evidence for detrimental reproductive health and fertility outcomes is available for a minority of EDCs – notably BPA, DEHP and PCB.*[32]

Inflammation and oxidative stress

Both human and laboratory studies have shown that MNPs and chemicals used to make plastics flexible (i.e. phthalates) may lead to inflammation and oxidative cellular damage.[36–38]

People with more plastic particles in their blood had higher levels of a marker of systemic inflammation (C-reactive protein/CRP), and a clotting protein that also rises during inflammation (fibrinogen), suggesting the body's immune system may be treating the presence of these particles as a threat.[38] Elevated inflammation markers (e.g. (interleukin-6, tumour necrosis factor-alpha, and reactive oxygen species) have also been observed, suggesting immune and cellular disruption.[36]

People with greater amounts of phthalates in their urine also showed higher levels of hsCRP (high-sensitivity C-reactive protein) and a liver enzyme that rises under oxidative stress (GGT - gamma-glutamyl transferase), though these associations were inconsistent across different groups of people and varied based on individual factors such as age, weight, and diet.[37]

Because most human studies capture only a single moment in time rather than following people across years, it is not yet possible to say with confidence that plastics and phthalates are the direct cause of these changes, and no known safe level of exposure currently exists.[36–38]

MNPs have been identified in brain tissue at concentrations 7–30 times higher than in the liver or kidney.[39] One study using postmortems in the US found that brain MNP concentrations increased by approximately 50% between 2016 and 2024, a trend temporally consistent with rising global environmental MNP exposure.[39] Higher MNP levels have been reported in individuals diagnosed with dementia compared with unaffected controls, but again there is no clear evidence that MNPs have a causal role in neurodegeneration, they might simply rise in parallel with ageing or cumulative exposure.[39] However, given current challenges surrounding accurately measuring smaller MNPs, these findings are not uncontroversial.

Similar patterns are seen for plastic-associated chemicals. Small molecules of a new, "safer" phthalate substitute used in flexible PVC (di(2-ethylhexyl) terephthalate) are linked with changes in general inflammatory markers, liver stress markers and immune system activation in adults.[37] These associations, though suggestive of ongoing inflammation and oxidative stress, are observational – the underlying causal pathways and potential health implications not yet established.[37,40]

In the US, contamination from a PFOA manufacturing plant in West Virginia has been linked to elevated blood PFAS concentrations among nearby residents and higher rates of kidney and testicular cancer.[41] PFOA is a long-chain PFAS formerly used in non-stick coatings, waterproof fabrics, and industrial applications, which is now banned in Australia and listed for global phase-out.[42,43]

Case study: Cardiovascular health

Summary: Experimental studies show that exposure to MNPs, particularly when they act as carriers of PFAS, can induce oxidative stress and inflammatory responses in multiple organ systems in vitro and in animals.[28] However, their effects in humans have been insufficiently investigated to understand their potential implications for disease risk.

Emerging evidence from preclinical studies suggests that MNPs may affect cardiovascular health. However, these studies must be interpreted with caution, since current techniques for measuring smaller MNPs are still being developed.

In vitro (cell and tissue) studies have shown that MNPs contribute to oxidative stress, inflammation, and premature cell death in the cells lining blood vessels, while animal studies have shown scarring of the heart muscle and impaired blood vessel function.[44] Human studies indicate that the presence of MNPs within carotid artery plaque (fatty deposits that build up inside the carotid arteries, which supply blood to the brain and neck) was associated with an approximately 4.5-fold increased risk of serious cardiovascular events, including non-fatal heart attack, non-fatal stroke, or death for any reason.[44] MNPs presence within plaques was also linked to higher concentrations of inflammatory markers suggesting that MNPs may worsen cardiovascular disease by driving the inflammation that accelerates the build-up of fatty deposits in the arteries.[44] However, causality has not been established, and the clinical relevance of these findings remains unclear, as other factors such as environmental pollution, human behaviour, and lifestyle may be contributing to these associations, and the results may not be generalisable to the broader population.[44,45]

Evidence from human studies have also linked the exposure to fine particulate matter (PM2.5), phthalates, BPA, PFAS, and heavy metals to cardiovascular disease through metabolic dysregulation, oxidative stress, and inflammation.[46–48] Preclinical studies further suggest that MNPs, by acting as carriers for these chemicals, facilitate their transport into cells and organ tissues.[46–48] Smaller MNPs can more easily enter the body, cross biological barriers, accumulate in organ tissues, and interact with cells, thereby increasing reactive oxygen species (ROS) and lipid oxidation events leading to cell death.[49] In preclinical models, high levels of ROS and lipid oxidation have been shown to damage cells, including their membranes, proteins, and genetic material.[47]

However, findings across studies are inconsistent, with some reporting higher toxicity from larger particles and others finding no association between particle size and toxicity.[47] Size-dependent toxicity also appears to vary across biological systems, both in nature and magnitude, highlighting that results may differ depending on the organism or organ system being studied.[47] These discrepancies may be attributed to differences in particle properties; metrics used for reporting such as weight, number of particles, or surface area; dose and exposure duration; endpoint selection and detection methods; and the biological systems studied.[47]

[iv] *PFOA is banned in Australia effective 1 July 2025, with the manufacture, import, export and use of PFOA, PFOS and PFHxS prohibited under the Industrial Chemicals Environmental Management Standard (IChEMS), subject to limited exemptions (e.g. trace contamination, legacy articles and some therapeutic uses).*[42]

Toxic interactions: Co-accumulation and complex chemical mixtures

Many chemical contaminants and their effects co-accumulate and interact, and some particle types can act as carriers for other substances.

The effects of individual contaminants can accumulate even when they occur in low concentrations, often as part of complex chemical mixtures.[50] The combined effects of these mixtures are not well understood; PFAS have been more extensively studied than MNPs, but PFAS–MNP interactions are an emerging area of research.[28] While only some EDCs bioaccumulate, even those with short-lived presence in the body may act synergistically or antagonistically with other contaminants during the period of exposure.

Initial studies in cell-based and animal models suggest that MNPs can disrupt cell membrane integrity, trigger oxidative stress and inflammation, and interfere with hormone and lipid signalling.[51] Early studies examining co-exposure to MNPs and PFAS suggest these effects may be synergistically amplified; however, this evidence is largely confined to cell models and the broader evidence base remains limited.[51,52]

In addition to augmenting potential mechanisms of harm, co-accumulation of multiple contaminants may alter modes and rates of bioaccumulation. One mechanism that is widely discussed but under-investigated is the 'Trojan horse effect' whereby MNPs – due to their small size and large surface areas – may act as vectors (i.e. carriers) of chemicals, proteins, toxins or pathogens into the human body.[53,54] However, while this mechanism is biologically plausible, evidence for it in humans remains limited. Another related co-accumulation mechanism is found in the fact that many MNPs contain unintended contaminants such as chemicals left behind on plastics during their production.

Precautionary action: What we can do now to understand and manage potential risks

Although the techniques to measure different contaminants vary in their maturity, and the type of large-scale, longitudinal research needed to track potential connections between exposure and health outcomes is currently limited, uncertainty is not a reason for inaction. Rather, these knowledge gaps warrant a precautionary approach: reducing plastics pollution, phasing out highly biopersistent PFAS where safer alternatives exist, strengthening mixture-focused (i.e. multiple contaminants) risk assessment, and investing in refined detection and measuring techniques along with longitudinal human research studies – prioritising groups who may be at the highest risk of exposure and bioaccumulation.

Priority populations

All Australian communities are exposed to contaminants such as MNPs and PFAS, but not all communities are equally exposed. While the scientific techniques and evidence base are evolving, it is vital that we prioritise research and measures to protect communities that are more likely to experience higher cumulative exposure and fewer protections – which often risk compounding existing health inequities.

One population that can be particularly vulnerable to higher exposure levels is lower income groups. US-based research has found a correlation between lower socio-economic status and higher exposure to EDCs, phthalates, BPA and other contaminants.[55]

Another cohort who are more vulnerable to contaminant exposure and bioaccumulation are geographically exposed groups such as those residing next to landfill sites or recycling plants, and occupationally exposed groups such as those working in in plastic manufacturing, synthetic textile production, recycling facilities, or – in the case of PFAS – firefighting and Defence.[19,22,56]

Aboriginal and/or Torres Strait Islander communities living or engaging in cultural practices on Country are often at heightened risk of PFAS exposure. Defence sites, airports and industrial facilities, which are significant sources of PFAS pollution, have often been established on or near the lands of these communities.[57]

The Wreck Bay Aboriginal community in New South Wales, for example, has been impacted by PFAS contamination from Defence activities, which have spread pollutants into water, soil and sacred sites – making traditional practices like fishing, hunting and gathering bush foods and medicines unsafe, and exerting a heavy psychological and cultural toll.[22,58]

In addition to being more likely to live or practice on land that has been contaminated by Defence, aeronautical or industry development, Aboriginal and/or Torres Strait Islander people may be more likely to rely on second-hand goods, experience poor housing conditions, and have lower access to comparatively expensive non-plastic cooking utensils and food storage options.[57]

Differing exposure levels are not the only factor that may contribute to the degree of vulnerability to bioaccumulation and the potential health impacts of MNPs, PFAS and other contaminants. Developmental life stages, including fetal life and early childhood, are likely to be periods of increased susceptibility to such contaminants.[19] MNPs and associated chemicals have been reported in human placenta, meconium (i.e. a baby's first stool) and cord blood, demonstrating exposure during critical developmental periods.[19] Higher prenatal exposure to BPA has been associated with greater autism-related symptoms in early childhood and a higher likelihood of an autism diagnosis by age 9, particularly in boys with a genetic predisposition to lower hormonal activity in the brain. However, these associations were based on limited exposure measurements during pregnancy. These findings are observational and cannot confirm a direct causal link, and BPA was studied in isolation rather than as part of the complex chemical mixtures to which people are typically exposed.[59] A growing body of research is exploring possible connections between exposure to EDC mixtures in pregnancy and early childhood and effects on childhood neurodevelopment.[50,60] While direct causal links to specific developmental outcomes remain difficult to establish, this evidence, combined with emerging mechanistic and epidemiological research, raises plausible concerns about potential impacts on early-life development which warrant further investigation.

Policy approaches

While safe exposure levels of MNPs, PFAS and other contaminants are not yet known, Australian policy approaches should focus on building the evidence base, starting with priority populations and refining regulation. The "safe levels" of MNPs, PFAS and other contaminants enshrined in legislation – and even the existence of regulation at all – currently varies globally.

1. Building the evidence base

Determining what (if any) levels of exposure to MNPs, PFAS and other contaminants are safe requires that we first refine scientific techniques and methodologies where these are currently limited (as is the case for smaller MNPs), establish large-scale bioaccumulation datasets (for PFAS and other measurable and reportable contaminants), and fill current research gaps. Understanding the nature of possible health impacts of these contaminants requires the Federal Government to partner with the health and medical research sector to:

a. **Refine techniques and methods for detecting, sampling and quantifying a range of contaminants.** This will enable us to understand with greater certainty the extent to which certain contaminants bioaccumulate in the human body – particularly MNPs, which pose unique challenges for analytical chemistry.

b. **Establish a National Human Biomonitoring (HBM) Program** covering PFAS and other contaminants of interest that can be accurately and reliably measured, with MNPs subsequently incorporated into this Program once the techniques for isolating and quantifying them have been sufficiently advanced. We cannot deliberately expose humans to toxic doses of chemicals in experimental settings, but we can analyse incidental environmental exposures. However, to do this, we must first collect data relating to these exposures through a national HBM. The Academy stands ready to work with Government and all key stakeholders to develop and implement the National HBM Program that we have recommended since 2024, a recommendation which the Senate Inquiry on PFAS adopted in its report last year.[22–24]

c. **Resource longitudinal human studies** of representative Australian cohorts to identify and examine potential connections between exposure to contaminants and health outcomes across all Australian communities.[v]

2. Focusing on priority populations

All Australian communities are exposed to contaminants such as MNPs and PFAS, but not all communities are equally exposed. Until we understand whether there is such a thing as "safe" levels of exposure to all such contaminants, Federal Government should partner with the health and medical research sector, and with priority populations, to take reasonable precautionary measures, including:

a. **As an initial and efficient first step towards establishing a National HBM Program, the Government should establish an interim HBM stream through existing antenatal care mechanisms and settings, while working with AAHMS and other key stakeholders to design and implement the broader National HBM Program.** Some MNPs, PFAS and other contaminants can spread to the placenta and fetal tissues. Human data for pregnancy outcomes (e.g. preterm birth, fetal growth, pregnancy complications, infant health and development) in relation to MNPs, PFAS and related substances exposure is currently limited. The Academy suggests that an interim HBM for pregnant women could be efficiently established while Government works with us and other key stakeholders to establish the broader national HBM Program.[23,24]

b. **Partner with Aboriginal and Torres Strait Islander communities and Aboriginal Community Controlled Health Organisations (ACCHOs)** to design and implement responses to, and management of, PFAS and related contamination – including by resourcing community-led initiatives. This will help ensure that action is culturally safe, equitable and grounded in community leadership.[57]

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v *Notably, the 1996-2012 National Health Priority Areas guided policy attention and targeted research initiatives across key health challenges, including healthy environments for population health. Today, most HMR funding is investigator-initiated and demand-driven, and environmental exposure research does not operate under a comparable policy priority framework.*

3. Reviewing regulation

MNPs, PFAS and other contaminants can only be effectively regulated to the extent that their bioaccumulation and potential health impacts can be measured and understood.

The assessment and regulation of MNPs, PFAS and other contaminants is a complex policy area – particularly where scientific techniques and evidence is evolving, and where coordination is required across different State/Territory jurisdictions, and between State/Territory governments and the Federal Government.[vi]

Australia's 2021 National Plastics Plan outlines an approach to find alternatives to phase out certain plastic materials, find alternatives to unnecessary plastics, and reduce the impact of plastic on the environment.[61] However, most legislation aimed at reducing MNPs pollution, and therefore exposure, is being made at the State and Territory level. This has resulted in substantial jurisdictional variation. For single-use plastics, jurisdictions differ not only in the items they regulate, but also in the timing and pathway of phase-outs. This means a product may be banned or fully phased out in one jurisdiction well before comparable measures are introduced elsewhere. Even where the same items appear across multiple State and Territory lists, the timeframe for implementation can still vary, highlighting the lack of a nationally consistent approach.

Australia is not starting from scratch. Nationally agreed technical guidance already exists, including the PFAS National Environmental Management Plan co-developed by Australian and New Zealand governments in 2024 and the NHMRC guidance on PFAS in drinking water, updated in 2025.[vii,62,63] The policy challenge is therefore less the absence of evidence-based advice than the lack of a mechanism to translate that advice into nationally consistent, enforceable regulation across shared Commonwealth–state responsibilities. This need was highlighted in the 2025 Senate inquiry on PFAS, which recommended 47 actions, including that the Australian Government "provides additional resources to the Australian Industrial Chemical Introduction Scheme to fast-track chemical assessment, including for PFAS and other persistent organic pollutants (POPs) to ensure toxic chemicals are not allowed on the market" (Recommendation 39).[22]

Australia can also review regulatory approaches and monitoring frameworks already being implemented internationally. The EU, for example, has introduced increasingly precautionary reporting requirements and restrictions in relation to PFAS and other long-lasting chemicals through frameworks such as the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH).[64] Aligning Australian regulatory approaches to those of comparable nations where appropriate would help reduce fragmentation and duplication of effort, facilitate the sharing and pooling of scientific evidence and approaches, and support the development of globally consistent benchmarks for safe exposure levels.

As scientific techniques and evidence evolve, the Federal Government should review and update regulation relating to MNPs, PFAS and other contaminants; adopt a precautionary principle where uncertainty exists; and work closely with State and Territory governments to consistent national implementation.

4. Supporting public awareness and exposure reduction

While scientific techniques and understanding continue to evolve, Australian governments should ensure that clear, accessible and evidence-based information is available to all communities on known sources of exposure to MNPs, PFAS and other contaminants. This could include practical ways that individuals can reduce exposure where feasible, alongside transparent communication about what is known, what remains uncertain, and how government and the health and medical research sector are responding through monitoring, research and – where appropriate – regulation.

[vi] *While State/Territory governments are responsible for everyday regulation of pollution prevention and contamination management, the Commonwealth Department sets national standards relating to protection of the environment though nationally consistent risk management.*[22]

[vii] *The PFAS National Environmental Plan relates to a small number of PFAS and excludes the newer short-chain class. This is in contrast to, for example, the EU, which takes a whole PFAS class approach.*

Glossary

Arterial plaque
A build-up of fats, cholesterol, blood cells, and other substances from blood on the inner walls of arteries (the blood vessels that carry oxygen-rich blood from the heart to the rest of the body). Over time, plaque can narrow the arteries and increase the risk of serious cardiovascular events.

Bioaccumulation
The process whereby MNPs and related toxicants build up in the human and animal bodies.

Bisphenol A (BPA) / BPA analogues (BPS, BPF)
Industrial chemicals used to make certain plastics and resins, including food and beverage containers. BPS and BPF are structurally similar chemicals that have been introduced as substitutes for BPA; however, they may carry similar health concerns.

Blood-follicle barrier / blood-testis barrier
Specialised protective barriers that separate the bloodstream from the ovarian follicles (where eggs develop) and testicular seminiferous tubules (where sperm develop), respectively. These barriers restrict the entry of most circulating chemicals, toxins and immune cells to safeguard egg and sperm maturation.

C-reactive protein (CRP) / high-sensitivity CRP (hs-CRP)
Proteins produced by the liver in response to inflammation. Elevated levels in blood tests are used as markers of ongoing inflammation in the body. Hs-CRP is a more precise version of the same test.

Contaminants
Introduced substances that – at certain concentrations – can be harmful to living organisms or cells.

DEHP
Di(2-ethylhexyl) phthalate, one of the most widely used phthalate plasticisers, commonly found in flexible PVC products such as medical devices, food packaging, and flooring.

DEHTP
Di(2-ethylhexyl) terephthalate, a newer phthalate introduced as a "safer" substitute for older phthalates in flexible PVC products.

Endocrine system
A complex network of glands and organs that synthesise, store and secrete hormones directly into the bloodstream

Endocrine disruption
The interference with the body's normal hormonal signalling which can alter or impair the essential functions that hormones regulate.

Endocrine disrupting chemicals (EDCs)
Synthetic or naturally occurring substances found in the environment that interfere with the body's hormonal system. They can mimic, block, or alter the effects of natural hormones.

Epidemiological study
A research design that examines the distribution and determinants of health outcomes in human populations.

Epithelial barriers
The body's primary physical and chemical defence, made up of layers of cells lining body surfaces and internal organs.

Estradiol
The primary and most potent female sex hormone, produced mainly by the ovaries. It plays a key role in the menstrual cycle, reproductive health, bone density and cardiovascular health.

Exogenous
Originating from outside the body, rather than being produced by the body itself.

Follicle-stimulating hormone (FSH)
A hormone responsible for sexual development and functioning, and the development of eggs in women and sperm in men.

Follicular fluid / seminal fluid
The biological fluids that directly surround eggs (follicular fluid, found in the ovaries) and sperm (seminal fluid). These are the microenvironments where eggs and sperm develop.

Forever chemicals
A non-technical term commonly used to describe PFAS and other chemicals whose strong carbon-fluoride bonds make them highly durable and persistant.

Human biomonitoring (HBM)
A means of assessing exposure levels to chemical substances in human bodies through the analysis of samples such as blood, gametes or urine.

Inflammation
The body's biological immune response triggered by a variety of harmful factors including foreign objects and pathogens.

Interleukin-6 (IL-6) / Tumour necrosis factor-alpha (TNF-α)
Signalling proteins produced by the immune system when the body detects injury, infection, or stress. Elevated levels may suggest the body is in a state of inflammation.

In vitro
Research or experiments conducted in a controlled laboratory environment outside a living organism.

Longitudinal study
A research design/method that involves collecting data from the same individuals over an extended period, allowing researchers to observe changes or trends in relation to ongoing exposures.

Luteinising hormone (LH)
A hormone that triggers ovulation in women and stimulates testosterone production in men.

Mass spectrometry
A technique that identifies and quantifies chemical substances by measuring the mass-to-charge ratio of ionised molecules.

Meconium
The first stool passed by a newborn, which forms in the gut during pregnancy. Because it accumulates material from the foetal environment, it can be used as a record of in-utero exposures.

Microscopy
A set of techniques that use microscopes to magnify and visualise small structures or particles.

Microplastics
Plastic particles generally smaller than 5 millimetres in diameter.

Morphology
The physical form, shape, and structure of a particle or organism.

Micro- and nanoplastics (MNPs)
A collective term to describe plastic particles across micro- and nanoscale ranges.

Nanoplastics
Plastic particles typically smaller than 1 micrometre (and often <100 nanometres), with distinct physiochemical properties due to their size.

Observational study
A research design/method where researchers observe and record data without intervention or treatment. These studies can identify associations between exposures and health outcomes but generally cannot prove causation on their own.

Ovarian reserve
A measure of the number and quality of eggs remaining in a woman's ovaries. A low ovarian reserve means fewer eggs are available for conception.

Oxidative stress
An imbalance between free radicals and the body's ability to neutralise them with antioxidants, which can lead to cell and tissue damage.

Parabens
A group of synthetic preservatives widely used in cosmetics, pharmaceuticals, and food products.

Polychlorinated biphenyls (PCBs)

A group of synthetic chemicals now banned in many countries due to their toxicity. PCBs are classified as persistent organic pollutants (POPs), meaning they resist environmental degradation and accumulate in the food chain and human body tissue.

Per- and polyfluoroalkyl substances (PFAS)

A group of almost 15,000 synthetic chemicals that are ubiquitously present in the environment (including in drinking water, the food supply, and household products) and therefore in human bodies.

PFOA / PFOS / PFHxS

Three of the most studied long-chain PFAS. PFOA (perfluorooctanoic acid) was formerly used in non-stick coatings; PFOS (perfluorooctane sulfonate) in stain-resistant products and firefighting foam; PFHxS (perfluorohexane sulfonate) in firefighting foam and industrial applications.

Phthalates

A family of synthetic chemicals used to make plastics flexible and durable, found in products including PVC packaging, medical devices, and personal care products.

Plastic

A wide range of substances made from polymer-containing compounds.

Polycystic ovary syndrome (PCOS)

A hormonal condition where the ovaries produce excess male hormones. It is associated with irregular periods and reduced fertility.

Polymer

A long, shapable chain of repeating molecules.

Pyrolysis

A thermochemical technique that breaks down polymer samples in the absence of oxygen.

Toxicant

A synthetic contaminant with confirmed potential to cause harm to living organisms or cells at certain exposure levels.

Toxicity

The extent to which a substance may be able to harm living organisms or cells.

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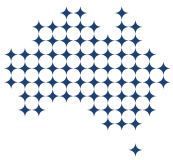

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