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Health Risk Assessment: Microplastics and Nanoplastics - UPDATED

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DISCLAIMER

CONTENTS

LIST OF TABLES

LIST OF FIGURES

EXECUTIVE SUMMARY

This report updates the information on the human health effects of microplastics and nanoplastics (MNPs) and serves as an update to the previously published report (*Health Risk Assessment: Microplastics* (FW22034))[1] prepared for the Ministry of Health in 2022. Since the original report was prepared more than 3000 peer-review studies examining MNP exposure and health effects have been published. This review synthesises current evidence on exposure pathways, biological plausibility and toxicological evidence for risks to human health. MNPs are now recognised as ubiquitous environmental contaminants arising from both intentionally manufactured particles and the fragmentation of larger plastic items. Human exposure occurs primarily through ingestion of contaminated food and water and inhalation of airborne particles, with additional exposure possible through medical procedures and dermal contact. Evidence also shows that plastics are not inert materials: they carry and release a wide range of associated chemicals, including additives and non-intentionally added substances, many of which are known to have human health effects.

Recent biomonitoring studies increasingly report the presence of MNPs in human tissues and bodily fluids, including blood, lung, placenta, liver, urine, breastmilk, semen, and gastrointestinal samples, supporting the plausibility of internal exposure and systemic distribution of smaller particles. However, interpretation of these findings remains constrained by analytical and methodological limitations, including lack of standardised detection methods, inconsistent reporting metrics and limitations around accurate detect and quantification of MNPs. As a result, the true prevalence, concentrations, and distribution of MNPs in humans remain uncertain. Despite these uncertainties, there is growing consensus across experimental studies that MNPs are biologically active and can trigger oxidative stress, inflammation, cytotoxicity, immune disruption, and tissue-specific effects, particularly in the respiratory, gastrointestinal, cardiovascular, nervous, reproductive, and endocrine systems.

At present, there is insufficient evidence to derive robust human-relevant dose-response relationships or to complete a quantitative health risk assessment for environmental MNP exposure. This reflects the heterogeneity of MNPs, uncertainty in real-world exposure characterisation, and the reliance of many studies on test materials and exposure conditions that do not fully represent weathered environmental particles or associated chemical mixtures. Accordingly, there is currently no direct evidence establishing causal adverse health outcomes in humans due to the methodological challenges presented by the ubiquity of MNPs, but the overall body of evidence supports concern that they represent a credible and emerging risk to human health. Consequently, this report concludes that a precautionary approach should be taken while critical knowledge gaps are addressed through improved analytical methods, standardised study designs, physiologically realistic models, and longitudinal human research to better inform future regulatory and public health decision-making in New Zealand. For example, through the banning of unnecessary MNPs such as glitter.

[1] Gautam, A., & Cressey, P. (2022). Health risk assessment: Microplastics (Client Report No. FW22034).

1 INTRODUCTION

1.1. MICROPLASTICS AND NANOPLASTICS

1.1.1 Definition and physical characterisation

Micro- and nanoplastic (MNPs) pollution is a growing global concern due to its ubiquity, persistence and potential impact on human and environmental health. There is continued debate about the size classification of MNPs but microplastics (MP) are widely defined as plastic particle <5 mm, and nanoplastics (NPs) as <1 µm [1, 2]. They are composed of the synthetic polymer together with functional additives, and other intentionally and unintentionally added chemicals. These definitions have been adopted by both the World Health Organization [3] and United National Environment Program [4]. They are characterised further, depending on their process of formation (Figure 1). *Primary* MNPs are those that were intentionally manufactured in that size range (e.g., preproduction pellets and glitter); and *secondary* MNPs are formed in the environment through biotic and abiotic fragmentation [5, 6] or through wear and tear of larger plastic items, such as tyre wear particles (TWP) and fibres released from synthetic textiles [7]. MNPs are characterised further, based on their morphotype, including fragment, fibre, bead, sphere, pellet, film, foam and flakes.

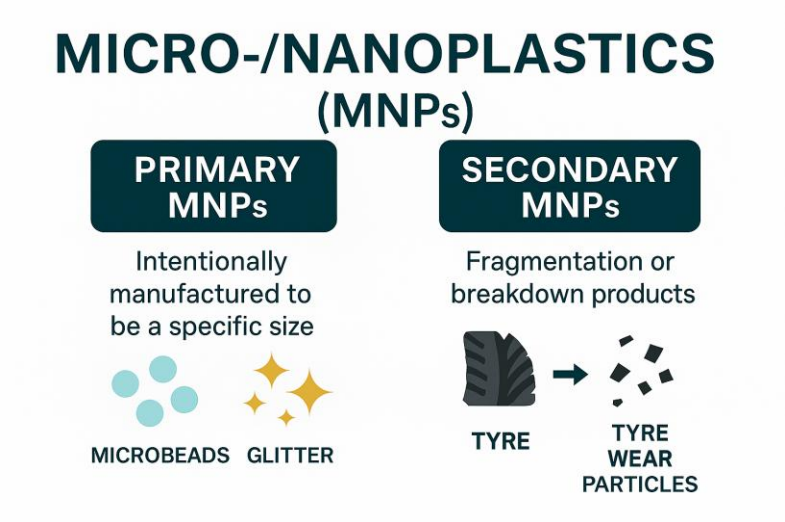

Figure 1. Subcategories of microplastics and nanoplastics (MNPs): Primary MNPs are particles intentionally manufactured at a specific size; secondary MNPs are the fragmentation or breakdown products of larger plastic items.

1.1.2 Chemical characteristics

Plastic are complex materials, composed of the solid polymer matrix and a range of functional and residual chemicals. Whilst often described as inert materials due to their resistance to biological degradation; polymers undergo abiotic transformation, release additives, interact with exogenous chemicals and fragment into MNPs, and therefore should not be considered inert [8, 9].

The synthesis of the plastic polymers (e.g., polyethylene and polypropylene) involves the polymerisation of monomers (e.g., ethylene and propylene) using catalysts and processing aids (Figure 2). To then produce the plastic items other chemicals are incorporated to achieve the desired properties (e.g., UV stabilizers, colourants, and plasticisers) and to assist manufacturing (e.g., thermal stabilisers and lubricants) [4]. The amount of these additives incorporated into the plastics can make up a considerable proportion of the plastics' mass. For example, plasticisers (e.g., phthalate esters) used in PVC dialysis bags to produce flexibility can account for up to 70% of the mass [4]. Plastics also contain non-intentionally added substances (NIAS) which include impurities carried through from polymerisation, degradation products or compounds produced during the manufacturing process, which are not deliberately added. Recycled plastics typically contain high rates of NIAS than virgin plastics due to the accumulation of contaminants and degradation products during use and recycling processes [10]. For example, pesticides, biocides and pharmaceuticals have been detected in recycled polyethylene [11, 12]. Unlike intentionally added substances (additives), NIAS are unpredictable in type and concentration, and therefore their identity is mostly unknown [13]. These chemicals are not covalently bonded to the polymer matrix and therefore migrate into the surrounding environment (e.g., gastrointestinal tract or lungs) [14-17].

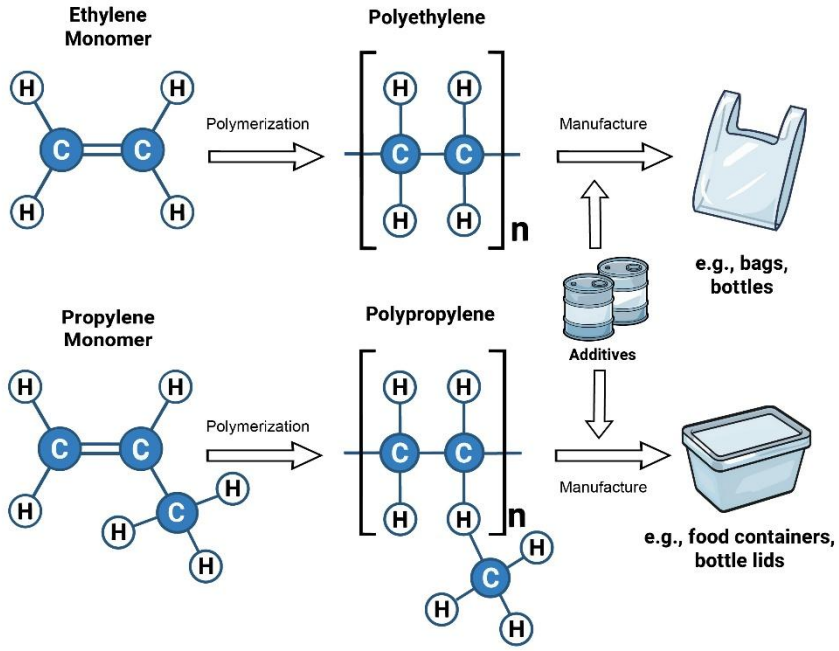

Figure 2. Schematic of the synthesis of polyethylene and polypropylene (two commodity plastic polymers) from their respective monomers, ethylene and propylene, by polymerisation to form the polymer chains, followed by the addition of additives and manufacture into final plastic products. Additives may include stabilisers, colourants, plasticisers, antioxidants, fillers, and processing aids, depending on the intended application.

A recent systematic review [18] has found that more than 16,000 chemicals have been identified as being associated with plastic materials and products. Of these, more than 4200 are considered chemicals of concern due to their hazard properties meeting one or more PBMT criteria (persistence, bioaccumulation, mobility, and/or toxicity). However, international regularity frameworks currently cover only ~6% of the >16,000 chemicals

that have been identified [18]. There is a large and growing body of evidence linking many plastic-associated chemicals to adverse health effects in humans, including well-studied groups such as phthalate esters, bisphenols and per- and polyfluoroalkyl substances (PFAS) [19, 20]. Conversely, a recent randomised controlled trial of a low-plastic diet in humans demonstrated a rapid reduction in internal concentrations of plastic-associated chemicals, which are associated with cardiometabolic risk biomarkers, providing emerging human evidence that reducing exposure results in positive health effects [21].

There are many different plastic polymers in use, but seven 'commodity' plastics dominate the market: polypropylene, low-density polyethylene, polyvinylchloride, high-density polyethylene, polyethylene terephthalate, polyurethane, and polystyrene Table 1. Other widely used polymers include synthetic rubbers in car tyres (butadiene rubber and styrene-butadiene rubber), acrylonitrile butadiene styrene, polycarbonate and nylon also being in common use [22]. Each of these major polymer types have been found to be used with >400 chemicals of concern (e.g., antimony, phthalates and bisphenols) to produce everyday plastic items [18]. Consequently, environmental MNPs that we are exposed to daily represent a diverse heterogeneous mix of polymer matrix and associated chemicals, and of different sizes and morphotypes.

Table 1. Commonly used plastic polymers and examples of their common applications.

Polymer (abbreviation)	Examples of Common Uses
Polypropylene (PP)	Food containers, bottle caps, straws, reusable containers, textiles
Low-density polyethylene (LDPE)	Plastic bags, shrink wrap, squeeze bottles, bread bags, film wrap
High-density polyethylene (HDPE)	Milk bottles, detergent bottles, water pipes, toys, storage containers
Polyvinylchloride (PVC)	Drainpipes, electrical conduit, cable insulation, flooring, window frames, medical tubing
Polyethylene terephthalate (PET)	Beverage bottles, food trays, polyester clothing
Polyurethane (PU)	Foams, insulation, mattresses, upholstery, coatings, sealants, Lycra®
Polystyrene (PS)	Disposable cutlery, foam packaging, takeaway cups, insulation, trays
Butadiene/styrene-butadiene rubber (BR/SBR)	Car tyres, shoe soles, conveyor belts, seals, artificial turf infill
Acrylonitrile butadiene styrene (ABS)	Electronics housings, toys, automotive trim, appliance parts, luggage

Polycarbonate (PC)	Safety glasses, lenses, electronic components, reusable bottles, shields
Nylon (PA	Textiles, ropes, fishing line, gears, bearings, engineering parts

1.2 REGULATION OF MNPS

1.2.2 International Action

United Nations Environment Programme (UNEP) Resolution 5/14, entitled "End plastic pollution: towards an international legally binding instrument", was adopted by consensus at the resumed fifth session of the United Nations Environment Assembly (UNEA-5.2) held in Nairobi in March 2022. The resolution mandated the establishment of an Intergovernmental Negotiating Committee (INC) to develop a legally binding international instrument to address plastic pollution across the full life cycle of plastics, including production, design, use and disposal.

The objective of the international legally binding instrument is to protect the environment and human health from the adverse effects of plastic pollution. While plastic pollution is addressed broadly across the current Chair's draft[2] treaty text, MNPs are not yet treated as a distinct or comprehensive category. In the draft text proposed by the INC Chair in August 2025, explicit references to 'microplastics' are largely limited to intentionally added microplastics (primary microplastics), which are identified alongside other problematic or avoidable plastic uses, and to general obligations to minimise releases and leakage of plastics to the environment across the life cycle. Nanoplastics are not referred to. However, the draft does not yet include dedicated definitions, source-specific controls, or binding measures addressing unintentionally generated MNPs, such as those arising from the wear and fragmentation of tyres, synthetic textiles, coatings, or environmental degradation. As a result, the regulation of MNPs in the current draft remains indirect and partial, relying primarily on upstream product design measures and general emission-reduction provisions rather than explicit, targeted controls. As treaty negotiations continue (next negotiating session (INC-5.4) is expected to take place at the end of 2026 or in early 2027), the scope and strength of future obligations related to MNPs remain uncertain. New Zealand is actively participating in the INC process, with the Ministry for the Environment and the Ministry of Foreign Affairs and Trade jointly representing New Zealand in negotiations.

1.2.1 National Action

Control of some primary MNPs has been achieved through the *Waste Minimisation (Microbeads) Regulations 2017*[3] which banned the manufacture and sale of wash-off products[4] containing plastic microbeads in New Zealand from 7 June 2018. The purpose of the ban was to prevent intentionally added microbeads (a microbead being "a water-insoluble plastic particle that is less than 5 mm at its widest point" (reg. 3)) from entering aquatic environments, where they pose risks to wildlife and human health. Products which use primary MPs that are not covered in this ban include wipe-off products such

[2] Chair's Draft Text Proposal –15 August 2025
[3] https://www.legislation.govt.nz/secondary-legislation/pco-drafted/2017/291/en/latest/#DLM7490715
[4] Wash-off product means a product that is intended to be rinsed off during or after use. Product types include: personal care products (exfoliating cleansers, toothpaste), abrasive surface cleaners, and products where microbeads are added to alter visual appearance (e.g. glitter bubble bath).

as cosmetic makeup, crafting materials and medicines and medical devices for therapeutic purposes.

2 HAZARD IDENTIFICATION

2.1 PREVIOUS ASSESSMENTS

No previous assessments of health effects were found for MNPs in New Zealand.

2.2 HUMAN HEALTH EFFECTS

2.2.1 Exposure routes

There are multiple exposure pathways of MNPs for humans. Ingestion via contaminated food and water and inhalation of airborne particles are the major routes [23, 24]. Inhaled airborne MNPs are not confined to the respiratory tract due to particles entrapped in the airway mucus being cleared via mucociliary transport and swallowed, thereby contributing to gastrointestinal exposure. Medical interventions [25, 26] and dermal contact [27, 28] have also been identified as potential routes.

Annually 10-40 Mt of MNPs are estimated to enter the environment [29]. The equivalent of up to 2 million fully loaded garbage trucks. Under the status quo, with global plastic production projected to double by 2040 [30], MNP levels are expected to escalate significantly over the next 10-30 years, driven not only by the production, use and disposal of new plastics, but also the continued fragmentation of plastics already in existence. The resilient nature of plastic means that, without suitable remediation of existing environmental MNPs and management of whole plastic items post-consumer plastics (e.g., electronics, clothing, packaging) to mitigate the genesis of more particles due to mismanagement (littering), MNPs will accumulate and persist in the environment, posing ongoing risks to ecological integrity and human health.

Inhalation

Plastic particles are released into the air from industrial processes, abrasion of materials including from clothes, textiles and tyres, wind dispersal of degraded fragments, resuspension of fallen fibres and fragments including road dust, and via ocean-to-atmosphere transfer [3, 31-33]. Sources may be industrial, agricultural, or domestic, and include occupational settings such as nail bars [34] plastic recyclers [35]. Suspended airborne MNPs span a wide range of morphotypes, plastics and sizes, with abundance inversely correlated to size but dominated by particles within the respirable (<2.5 µm) and inhalable (2.5-10 µm) fraction.

Airborne MNPs are ubiquitous, but abundance varies from place to place, in time, and with prevailing environmental conditions [3, 36]. Abundance varies significantly, spanning more than five orders of magnitude, from 0.0065 to 1,583 particles m^{-3} [31, 36-40], and levels have increased significantly over time [41]. Some factors underpinning airborne MNP abundance include whether the air is urban or rural, indoor or outdoor, their proximity to industrial or coastal areas, and by the number of people present in any setting. Urban air typically contains higher concentrations of airborne MNPs than rural air, reflecting the greater contributions from traffic and industrial activities, including tyre wear, synthetic textiles, construction materials and roading (e.g., markings). Rural environments may still receive MNPs through atmospheric transport, with deposition

influenced by proximity to dispersed settlements and prevailing weather. They are also influenced by local sources such as the plastics used in crop production and the application of MNP-contaminated biowaste (e.g., wastewater effluent) to land. Indoor air typically contains higher concentrations of MNP particles compared with outdoor air, due to sources such as textiles, furnishings, and limited ventilation resulting in accumulation of particles.

Ingestion

Micro-/nanoplastics particles have been detected across a wide range of food groups, including seafood, meat, dairy, grains, fruit and vegetables, sugar, salt and processed foods, as well as in drinking water (bottled and tap) and other beverages. Particle abundance varies widely, within and between food matrix, with a wide range of polymer types, sizes and morphotype [3]. Their ingestion and transit through the digestive tract have been confirmed by their identification in human stool samples [42, 43].

The high prevalence and diversity of MNPs found reflects the high occurrence and repeated contact of foods across the whole produce chain. Packaged and processed foods [44, 45] tend to have higher MNP concentrations than their fresh, unprocessed counterparts [46]. Fresh produce, including plants and seafood, have been found to contain MNPs resulting from environmental contamination of their growing environment [47-51]. Plants may become contaminated due the use of MNP-contaminated soil amendments (e.g., compost/vermicompost [52, 53]), slow-release fertilisers [54] or the MNPs released from agriplastics as a result of weathering and general wear and tear [55, 56]. Aquaculture/mariculture species are exposed to MNPs originating from both infrastructure and defuse environmental pollution [47, 57, 58]. In the case of processed foods, plastic packaging is a well-recognised source of particles, with release rates increasing under conditions of mechanical stress, repeated opening and closing, ageing, and thermal treatment [e.g., 59, 60]. Elevated releases have been reported when food or beverages are heated in plastic containers, microwaved, or prepared using plastic utensils (Figure 3), such as chopping boards and mixing bowls, which may shed hundreds of particles per use [61]. Functional packaging such as tea bags, including those made wholly or partly of plastic and those marketed as biodegradable, can release millions of MPs and billions of NPs into a single cup of tea [62]. Cooking appliances may also release MNPs during use, for example new plastic kettles have been reported to release millions of microplastic particles into water during initial boiling cycles [63]. Contamination may also originate from the plastics used ubiquitously across the different processing steps, from use in equipment to the clothing worn by employees [64]. Therefore, MNP contamination is not limited to plastic considered to be ‘food grade’.

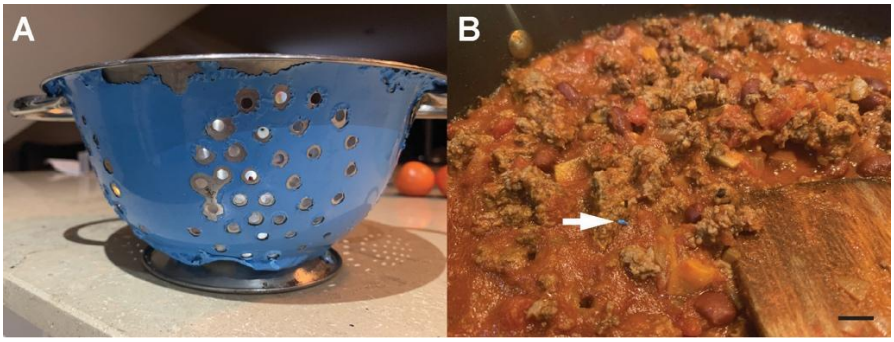

Figure 3. Microplastic contamination observed during food preparation. (A) Breakdown and flaking of the plastic coating on a colander during use, was found to be the source of the microplastic particle found in the food (B). Scale bar = 1 cm. (Source: Author)

Drinking water represents another predominant source of dietary exposure, both due to direct ingestion but also due to its use in the production and processing of foods and beverages. Plastic particles have been detected in both tap and bottled water of 30 countries to date, with concentrations spanning several orders of magnitude, from 0 to 1800 MPs L^{-1}, with NP counts reaching in to the millions per litre [65]. Bottled water on average contains higher particle counts than tap water, attributable to the contact with plastic bottles and caps and to mechanical stresses during bottling, transport and consumer use. The origin of the source water (rainwater, groundwater or surface waters) and treatment processes employed influences the levels and characteristics of MNPs, with surface waters generally considered at greatest risk of elevated concentrations due to direct and cumulative pollution inputs, including land-based runoff, wastewater discharge and atmospheric deposition. Removal rates for MNPs typically range from 45% to over 99%, while NPs are more challenging to eliminate, with rates of removal often below those for MPs. Larger particles are removed more efficiently compared to smaller MNPs which can evade conventional treatments and persist in the treated water. Desalinated water, which uses advanced membrane technologies like reverse osmosis, in general have lower levels if MNPs. Most large-sized MPs are removed, but trace amounts of smaller MPs and NPs may pass through, and post-treatment steps (such as remineralization) may reintroduce MNPs into the final product water [65-70]. Exposure levels are therefore dependent on the levels of contamination of the source water, the level and type of treatment and whether the water is packaged. Consequently, estimated daily intakes vary significantly both between and within countries. However, it has been consistently demonstrated that children, due to physiological and behavioural factors, are at a greater risk of ingesting higher amounts of MNPs relative to their body weight. For example, a study of Fijian tap water estimated that children ingested 0.0031-0.1813 MPs kg^{-1} bw day^{-1} compared to adults 0.0021-0.0829 MPs kg^{-1} bw day^{-1} [71]; and a similar study of bottled water in India estimated daily MP intake was four times higher for children (1.194 ± 1.314 MPs kg^{-1} bw day^{-1}) than for adults[5] (0.326 ± 0.358 MPs kg^{-1} bw day^{-1}) [72].

Estimates of overall ingestion exposure vary considerably due to differences in diet, consumer behaviour, and methodological approaches to quantification of MNPs used in exposure studies [3, 42, 43]. Reported annual intakes range from tens of thousands to over one hundred thousand particles per person, with higher intakes associated with greater consumption of processed foods, bottled beverages, and foods prepared or heated in plastic [3, 73]. Infants and young children may experience higher ingestion exposures on a body-weight basis due to formula preparation, use of plastic feeding bottles, teats, food pouches [74] and potential exposure via MNP-contaminated breastmilk [75, 76].

Other sources of MNPs not considered to be food but result in the exposure to MNPs via ingestion include products such as: dental care products ranging from daily oral care products (floss and toothpaste) and products used in dental procedures (e.g., orthodontic aligners, acrylic resin fillings) [77]; and toys for children [78] of ages that frequently put things in their mouths due to their unique hand-to-mouth behaviour, and chewing gum [79, 80].

[5] Estimated intakes may differ by gender, and values shown represents female intake.

Other exposure routes

Medical procedures present a potential exposure pathway that bypass the body's usual protective barriers, such as intravenous, intramuscular, or intradermal drug administration, enteral feeding, exposure through open surgical wounds, and prolonged direct contact between tissues and implants or surgical mesh. An example of medical intervention that results in direct internal exposure is that of dialysis due to the extensive use of plastics in the procedure, including PVC fluid bags, plastic tubing, dialyser membranes, and plastic syringes used for fluid handling and medication delivery. MNP concentrations of 0.29 ± 0.16 particles L^{-1} have been identified in haemodialysis solutions and 0.34 ± 0.02 particles L^{-1} in peritoneal dialysis solutions [81].

Dermal contact presents a potential exposure route for MNPs, particularly through skincare and therapeutic products which are applied directly to the skin, however there is currently limited evidence of penetration occurring. Intact human skin is an effective barrier, resulting in MNPs larger than ~2-5 µm are largely confined to the outermost layer (stratum corneum) and accumulating in hair follicles. Smaller particles (<2 µm) have been reported to penetrate deeper into the epidermal layers under specific experimental conditions, notably when the skin barrier is compromised or the matrix the MNPs were applied in enhance permeability [27].

2.2.2 Observations in humans

There is an increasing body of evidence of the presence of plastic particles throughout the human body. Presence in lung tissues, the gastrointestinal tract and faeces, reflecting the exposure via the two main exposure routes (inhalation and ingestion); as well as in a wide range of tissues and bodily fluids (Table 2). The detection of MNPs in tissues, for example brain, placenta, blood [82-84] and bodily fluids, for example blood and breastmilk [76, 83] with no direct exposure to environmental MNPs indicating internal exposure and systemic distribution.

Table 2. Overview of human organs, tissues, and bodily fluids in which MNPs have been identified, including reported concentration ranges and analytical method used.

Organ/Tissue/Fluid	Abundance	Analytical Method*	Reference
Blood	170-2490 ng/mL blood	Pyr-GC/MS	[85]
	0-19,765 particles/L blood	µFTIR	[83]
Urine	0.53-7.83 mg/kg urine 0-62.30 particles/kg urine	Pyr-GC/MS LDIR	[86]
	0-9600 particles/L urine	µFTIR	[87]
Liver	0.3-11.9 particles/g tissue	µ-Raman	[88]
	22.31-1264 µg/g tissue	Pyr-GC/MS	[89]‡
Brain	0-308.14 particles/g tissue 0.80-299.45 µg/g tissue	LDIR Pyr-GC/MS	[82]
	1,267-5,608 µg/g tissue	Pyr-GC/MS	[89]‡
	Particle presence not quantified	µFTIR	[90]
Lung	1.42-1.50 particles/g tissue	µFTIR	[91]
	0.56±0.53 particles/g tissue	µ-Raman	[92]
	14.19±14.57 particles/g tissue	LDIR	[93]
Gastrointestinal tract (incl. faeces)	0.046-0.96 particles/g stool	µFTIR	[42]
	1-39 particles/stomach	µ-Raman	[94]
	9.45±13.13 particles/g tissue large intestine 7.91±7.00 particles/g tissue small intestine	LDIR	[93]
Placenta	0-5 particles per placenta	µ-Raman	[95]
	0-6 particles per placenta	µFTIR	[96]
	1.24-13.41 particles/g tissue	µ-Raman	[84]
Vagina	1-51 MPs/g lavage fluid	µ-Raman	[97]
Breastmilk	0-2.72 particles/g milk	µ-Raman	[76]
Semen	0-5 particles per sample†	µ-Raman	[98]
	0.23±0.45 particles/ml semen	LDIR	[99]
Carotid plaque	PE: 21.7±24.5 µg/mg plaque tissue PVC: 5.2±2.4 µg/mg plaque tissue	Pyr-GC/MS	[100]

* Pyr-GC/MS – pyrolysis gas chromatography-mass spectrometry; µ-Raman – micro-Raman spectroscopy; LDIR – laser direct infrared spectroscopy; µFTIR – micro-Fourier-transform infrared spectroscopy.
† Sample volume not provided.
‡ This study [89] has received feedback of substantive concerns about the validity of this study's findings, citing limitations in analytical specificity and potential artefacts associated with the methods used; accordingly, the results should be interpreted with caution [101].

Direct quantitative comparison between studies must be done with caution due to the lack of standardised methodologies, with substantial differences in sample collection, contamination control, particle isolation efficiency, and analytical techniques [102]. These methodological variations, including the use of techniques such as micro-Fourier

transform infrared (μ-FTIR) spectroscopy, μ-Raman spectroscopy, laser direct infrared (LDIR) spectroscopy, and pyrolysis–gas chromatography–mass spectrometry (Pyr-GC/MS), as well as the use of differing reference libraries for polymer identification, result in method-dependent size detection limits, polymer-specific biases, and inconsistent reporting metrics (particle-based vs mass-based), which collectively contribute to discrepancies between studies.

Significant methodological limitations constrain the interpretation of current human biomonitoring studies of MNPs. The choice of analytical technique fundamentally determines the size range of particles detected, with the predominantly used methods (e.g., μ-FTIR, μ-Raman, LDIR) limited to micrometre-scale resolution, thereby failing to capture smaller nanoplastics that are more biologically relevant for permeation of biological barriers. Consequently, the absence of particles below certain size threshold but physiologically feasible, likely reflects analytical limitations rather than their true absence [103, 104]. In addition, techniques such as Pyr-GC/MS are highly susceptible to matrix interferences in complex biological samples, including contributions from endogenous lipids and other compounds, which can lead to false positives and overestimation of polymer concentrations, particularly for polyethylene and polyvinyl chloride [104]. This lack of specificity, combined with limited method sensitivity, high detection limits, and low or variable recovery rates, raises concerns regarding the validity and biological plausibility of some reported concentrations and particle sizes.

2.2.3 Absorption, distribution, metabolism and excretion (ADME)

Due to MNPs not being a single well-defined chemical, but instead a complex contaminant, comprising particles with diverse physical properties and chemical compositions, their toxicokinetics can be considered as two overlapping processes: (i) the fate of the particles themselves and (ii) the fate of associated chemicals (e.g., additives, NIAS, and sorbed contaminants) that may leach in to surrounding tissues or fluids. Whilst a biological effect will be determined by the combined physical and chemical nature of the MNP the fate of only the particle is discussed below.

Absorption

Direct evidence for absorption of MNPs in humans remains indirect, with the detection of particles in blood and other tissues lacking direct environmental exposure pathways suggesting translocation across biological barriers such as the lung-blood, gut-blood, and blood-brain interfaces [103, 105].

Inhalation represents a potential route of systemic absorption for smaller particles deposited in the respiratory tract. While many inhaled MNPs are cleared via mucociliary transport and subsequently swallowed, nanoscale and fine MP particles that reach the deep lung (alveolar region) may be internalised and, in some cases, cross the air-blood barrier into pulmonary capillaries. Additional pathways that have been proposed include nasal-neural transport and translocation across respiratory epithelia [90, 92]. However, as with other exposure routes, evidence is largely derived from *in vitro* and *in vivo* animal studies and remains poorly understood for humans.

Ingested MNPs may be absorbed through physicochemically mediated interactions within the gastrointestinal tract, although this is subject to strict biological constraints [103, 105]. Particle size is a key determinant of uptake potential, with only smaller particles considered biologically plausible for interaction with mucus and epithelial cells [103]. Proposed upper size limits for uptake span from the nanoscale (via

endocytosis[6]-dominated processes) to small microplastics (e.g., ≤150 µm) under limited conditions such as "persorption" through transient epithelial gaps; larger particles are expected to remain in the gut lumen and be excreted [103]. Following ingestion or inhalation, MNPs rapidly acquire a biomolecular "corona" of proteins, lipids, and organic matter that alters surface properties and influences cellular recognition, immune interactions, and uptake efficiency [103, 105]. Translocation is thought to occur primarily via transcellular pathways (including endocytosis, transcytosis, micropinocytosis, or phagocytosis) as well as uptake by microfold (M) cells and transport by migratory phagocytes into lymphatic pathways. Paracellular transport appears limited and is more likely under conditions of compromised epithelial integrity. If particles cross epithelial barriers in the gut or lung, they may enter the hepatic portal or systemic circulation and potentially reach secondary organs such as the liver; however, their subsequent distribution, persistence, and clearance remain largely unknown.

Dermal uptake is considered limited under normal conditions. Healthy, intact human skin acts as an effective barrier, with most particles remaining on the surface or confined to the stratum corneum and hair follicles. Experimental studies, primarily using in vitro and ex vivo models, show only limited penetration, generally restricted to very small particles (<1–2 µm) under specific conditions influenced by particle characteristics and skin integrity (e.g., hydration, inflammation, or physical damage) [27]. There is no robust evidence demonstrating systemic availability of intact MNPs following dermal exposure, and observed penetration does not equate to toxicokinetic absorption.

Distribution

Few studies have investigated the mechanisms involved in the translocation of particles throughout the human body [106]. Particle size is considered to be the primary determinant of internal distribution, influencing both the ability to cross biological barriers and undergo systemic distribution, with larger particles more likely to be retained at the site of deposition or eliminated [103, 104, 107]. Evidence for systemic distribution remains largely indirect but is supported by analytical studies detecting MNPs in human tissues and fluids, including urine, blood, lungs, liver, placenta, and other organs [84, 86, 100, 108, 109].

Substantial uncertainties remain, as the current evidence base is limited by the small number of studies on human distribution, variability between analytical methods, and methodological constraints, including insufficient sensitivity for nanoplastics and reliance on non-representative experimental materials [103, 104]. Critical knowledge gaps also persist regarding the influence of environmental weathering and *in vivo* surface modification (e.g. biomolecular corona formation) on particle distribution, persistence, tissue interactions, and the efficiency of translocation, limiting a comprehensive assessment of tissue-specific accumulation, long-term fate, and the associated health implications of internal MNP exposure.

Metabolism

Synthetic plastic polymers are generally considered to be recalcitrant and are not readily enzymatically metabolised the way other small molecules are. Emerging evidence indicates that certain members of the human gut microbiome may possess a limited capacity to degrade or transform synthetic plastic polymers [110]. This is supported by studies showing that gut microbiomes of other organisms, such as insects, possess

[6] Endocytosis is a process by which a cell actively engulfs substances from its external environment.

comparable biodegradation capacity, which occurs at similarly low rates [111, 112]. However, there is currently no evidence that the human physiological system itself is capable of enzymatically metabolising the plastic particles [113]. The particles may still undergo biotransformation *in situ*, including surface modification in biological fluids (e.g., formation of a protein corona [114]), oxidation or weathering-like changes which may result in fragmentation and the release of the associated chemicals [17] or desorption of environmental pollutants acquired during use or in the environment [115-119].

Excretion

Particle size strongly influences clearance mechanisms. The presence of MNPs in faeces and sputum confirms the elimination of particles internalised via the two predominant exposure pathways. Ingested particles which are too large to be absorbed are expected to pass directly through the gastrointestinal tract, and be eliminated in faeces [42]. Foetal clearance has also been documented via meconium [84, 120]. Inhaled particles deposited in the central and upper airways are removed via sputum and mucociliary clearance [109]; these particles are typically swallowed and subsequently enter the gastrointestinal tract. Smaller particles, including fine microplastics (<5 μm) and nanoplastics, are able to penetrate deeper into the bronchioles and alveolar regions. Clearance from these regions is slower and relies primarily on alveolar macrophage phagocytosis, immune-mediated transport to lymphatic pathways, or in some cases translocation across the alveolar epithelium [92, 121]. Together, these processes generally limit long-term pulmonary retention of inhaled MNPs, although clearance rates vary widely depending on particle size, shape, and physicochemical properties. Plastic particles identified in other bodily excretions such as urine and breastmilk [76, 86, 87], are thought to result from the translocation of small particles across the gut-blood and air-blood barriers [103]. Other potential routes of expulsion of smaller particles include sweat, tears and menstruation, although currently there is no confirmed evidence for significant elimination of microplastic via these routes.

The extent to which internalised particles are cleared, remains uncertain, with existing evidence largely derived from animal models or indirect observations. While these findings support the plausibility of multiple excretion routes, significant knowledge gaps remain [106, 122].

Overall, although clearance is expected to reduce internal exposure, critical knowledge gaps remain regarding size-specific retention, clearance rates, and the relationship between exposure, persistence, and toxicity, limiting a comprehensive assessment of the health impacts of MNPs.

2.2.4 Toxicity Studies

Toxicity studies of MNPs are dominated by *in vitro* cell line experiments and *in vivo* animal models, with relatively limited human data. Since 2022, the evidence base has expanded; however, much of this remains focused on experimental and preclinical *in vitro* studies, making it difficult to translate findings to real-world human exposures, doses, and durations, particularly at the nano-scale. These studies consistently demonstrate that MNPs can induce a range of biological responses at the cellular and organism level, including oxidative stress, inflammation, cytotoxicity, apoptosis, immune modulation, and metabolic disruption.

In vitro studies

Since 2022, there has been >620 peer-reviewed studies[7] published that have investigated the biological effects of MNPs on human and animal cell models relevant to human toxicology. *In vitro* studies have become a cornerstone for elucidating the mechanisms of plastic particle toxicity, offering insights into cellular uptake, intracellular fate, and the activation of stress and inflammatory pathways in both human and animal-derived cells [123].

Key findings from in vitro studies:

- The internalisation of MNPs have been reported for a variety of human and animal cell types, including intestinal, placental, hepatic, lung, and brain cells, with the extent of uptake influenced by particle size, shape, and physicochemical properties [124-127]. Cellular uptake often occurs via endocytosis and is frequently associated with localisation in lysosomes and other organelles, sometimes resulting in accumulation and intracellular stress responses [128, 129].
- Cytotoxicity is a common endpoint, with several studies reporting dose-dependent reductions in cell viability upon exposure to MNPs [130-133]. Mechanisms underlying cytotoxicity often involve oxidative stress, mitochondrial dysfunction, increased reactive oxygen species (ROS) production, and activation of apoptosis pathways [134-136].
- Inflammatory responses are frequently observed, with increased expression of cytokines and chemokines indicating immune activation in exposed cells [137, 138]. Some studies also report genotoxic effects, such as DNA damage and micronucleus formation, especially at higher concentrations and with smaller-sized particles [131].
- The physicochemical properties of particles, such as size, surface charge, and the presence of additives or contaminants, significantly influence biological outcomes, with NPs generally inducing stronger effects than MPs [126, 129, 139]. Additionally, shape (e.g., fibres vs. spheres) and co-exposure with other pollutants (e.g., plasticizers or environmental toxins) can modulate toxicity, sometimes synergistically [133, 134, 140].
- Emerging models such as organoids and advanced microfluidic systems are increasingly used to better mimic *in vivo* conditions and reveal more physiologically relevant responses to plastic particle exposure [125, 141].

In vivo studies

Since 2022 >1000 peer-reviewed articles[7] have been published on *in vivo* studies examining the impacts if MNPs on human health. The result of which have continued to raise significant concerns regarding their potential impacts on human and animal health [123]. This has been particularly highlighted by studies which have confirmed MNPs can enter the body through various routes-such as ingestion, inhalation, and dermal contact-accumulate in different organs [142-144], and induce a variety of biological responses, including inflammation, oxidative stress, and even tissue-specific dysfunction [145-147].

Key findings:

- *In vivo* studies consistently show that MNPs are bioavailable, capable of crossing biological barriers (e.g., intestinal, blood-testis, blood-brain, ocular surface), and accumulating in organs such as the liver, kidneys, brain, heart, reproductive tissues, and even the eye [142, 148-150]. Distribution patterns are influenced by particle size, shape, chemical composition, and exposure route [151, 152].

[7] Determined based on results obtained from the Web of Science database (June 2026).

- Exposure can induce a range of toxic effects, including oxidative stress, mitochondrial dysfunction, apoptosis, inflammation, and disruption of tissue barriers or homeostasis [148, 152-155]. These effects have been documented in multiple organ systems, including the cardiovascular, renal, hepatic, nervous, and reproductive systems [148, 156-158].
- The reproductive system is particularly sensitive, with studies showing that MNPs disrupt spermatogenesis, induce mitochondrial damage in sperm, and alter hormone levels in exposed animals [145, 148]. Similar findings of placental and foetal accumulation have been reported, raising concerns about developmental toxicity [147].
- Recent research has highlighted the role of MNPs in promoting or exacerbating inflammatory diseases, such as colitis, and their potential to contribute to carcinogenesis when combined with plasticizer additives like tributyl citrate [159]. Oxidative stress and inflammation are central mechanisms, often mediated by mitochondrial dysfunction and activation of signalling pathways [153, 154].
- While some studies in rodents show minimal acute effects at environmentally relevant doses, chronic or high exposures often produce marked toxicities, including histological changes and biomarker alterations [160, 161]. Zebrafish and *Drosophila* models corroborate these findings and are useful for high-throughput mechanistic studies [161, 162].
- Emerging evidence links MNPs exposure to neurotoxicity and behavioural changes, with animal studies revealing blood-brain barrier penetration, neuroinflammation, and protein aggregation relevant to neurodegenerative diseases [149, 158].
- Human evidence, although limited, confirms the presence of MNPs in various tissues (e.g., placenta, blood, lungs, semen, and eye) and suggests associations with inflammation, gut microbiome dysbiosis, and functional impairment [145, 147, 150].

Next-generation in vitro studies

Recent advances in organ-on-a-chip (OOC) technology have provided more physiologically relevant *in vitro* platforms for investigating the impacts of MNPs on human health, allow for the more accurate modelling of human organ systems offering insights into the uptake, transport and toxicity. Whilst studies are limited their use is increasing and demonstrate the benefits of being able to reproduce complex biological responses and dose-dependent effects of MNPs in ways that traditional methods cannot, reflecting both acute and chronic exposure scenarios relevant to real-world environmental contamination [163, 164].

Microfluidic lung-on-a-chip and advanced airway epithelial models indicate that MNPs can perturb alveolar/airway barrier function and trigger inflammatory and injury-associated signalling, with high-dose polystyrene nanoplastics producing clearer injurious responses and certain fibres/fragments (e.g., nylon fibres, cleaned HDPE) disrupting epithelial integrity even without overt cytotoxicity [164, 165]. Advanced intestinal and human colon tissue models show reduced tissue functionality and viability changes after exposure to selected particles, alongside evidence that some particles can across intestinal epithelia, disrupt tissue functionality and trigger immune activation [165, 166]. Microfluidic thrombosis platforms have demonstrated that MNPs can disrupt blood clotting and promote thrombus instability, matching previous work using animal models [167].

Research gaps

Key research gaps largely reflect the relatively early stage of research in this field, which is characterised by a lack of standardised methods and limited availability of human-relevant data. A major shared gap is the lack of standardised methodologies, including inconsistent particle characterisation and experimental conditions in *in vitro* studies [168, 169] and a lack of harmonised protocols for exposure, detection, and characterisation in *in vivo* studies [144, 146, 170], which together hinder cross-study comparability and robust risk assessment.

There is currently insufficient consideration of realistic exposure complexities, including the effects of weathered or aged plastics, environmentally relevant mixtures, and interactions with associated chemical additives or co-contaminants, despite emerging evidence of potential synergistic toxicity [133, 140, 159, 170]. Although this reflects more the magnitude of the complexity of MNP contamination, studies are now beginning to incorporate this complexity. Similarly, whilst studies have often examined acute or high-dose exposures, limiting the relevance to real-world conditions more studies are investigating the impacts of chronic, low-level, environmentally relevant exposure scenarios that better reflect human conditions [143, 145, 171, 172].

Critical gaps still exist in the understanding of impacts on the sensitive biological systems and populations, including specialised or vulnerable cell types (e.g. nervous, reproductive, and developing foetal tissues) [128, 141] and broader considerations of life stage, sex differences, and susceptible populations [147, 149], underscoring the need for more physiologically relevant models and targeted, human-relevant research.

Conclusions

Collectively, evidence from *in vitro* and *in vivo* studies indicate that MNPs are biologically active and can elicit adverse responses across multiple organ systems. *In vitro* studies consistently show that they can be internalised by human and animal cells and trigger cytotoxic, genotoxic, and inflammatory responses, with outcomes strongly influenced by particle size, polymer type, surface chemistry, and exposure conditions [130, 131, 172]. *In vivo* animal studies corroborate these mechanisms at the organism level, demonstrating bioavailability, barrier crossing and distribution to organs, and downstream effects including oxidative stress, inflammation, and tissue dysfunction, particularly for smaller particles [142, 148, 153]. Despite rapid growth in the evidence base, major uncertainties remain in translating experimental findings to real-world human health risk, driven by non-representative test materials and doses, limited environmentally relevant mixtures and polymer-associated chemical combinations, and inconsistent exposure and measurement protocols.

To address these knowledge gaps, future studies must prioritise standardised methods, physiologically realistic models, including organ-on-a-chip systems, human-derived tissues; coupled with longitudinal biomonitoring studies to support physiologically relevant exposure models, and long-term human studies [103, 141, 143, 144, 168].

3 DOSE-RESPONSE INFORMATION

Biomonitoring studies increasingly demonstrate internal exposure to MNPs in human tissues and fluids, supporting biological plausibility for systemic interactions. However, due to analytical limitations, lack of standardisation of analysis (e.g. particle number versus mass, differing size cut-offs) these studies rarely capture dose metrics most relevant to toxicity, including for example the characteristics of the associated chemical contaminants [14, 173]. As a result, biomonitoring data cannot yet be reliably translated into quantitative risk estimates [123].

Experimental studies demonstrate that MNPs can elicit biological effects across a range of doses and endpoints [123]. However, robust human-relevant dose-response relationships suitable for quantitative risk assessment are not yet established. This is largely due to the intrinsic complexity of MNPs as contaminants, substantial uncertainties in exposure characterisation, and the reliance of toxicological studies on monodisperse, spherical or pristine polymer particles that do not account for co-exposure of the physical particles and associated chemical contaminants. Consequently, these studies are not representative of real-world exposure [3, 123] and this lack of harmonisation restricts comparability between studies and limits the derivation of meaningful exposure-effect relationships.

A key challenge in establishing dose–response relationships and causal links between MNP exposure and adverse health outcomes in humans is the ubiquity of these particles in the environment. Given that exposure to MNPs is widespread through air, food, water, consumer products and even via medical intervention, identifying truly unexposed populations for use as control groups in epidemiological or experimental studies is extremely difficult, limiting the applicability of traditional approaches used to establish causality in environmental health research. A recent study (The PERTH Trial [21]) minimised plastic exposure to be able to determine the effects of the associated plastics chemicals on health exposure, which would also have also to some degree reduced MNP exposure. However, applying a similar approach to MNPs would be logistically very challenging given their pervasive presence across environmental media and the difficulty of fully eliminating exposure. Consequently, assessment of potential health impacts is likely to rely on a weight-of-evidence approach integrating mechanistic, toxicological, biomonitoring, and epidemiological data alongside precautionary public health principles.

Collectively, the available evidence supports biological plausibility for dose-related effects under experimental conditions, but the heterogeneity of test materials, inconsistent dosing metrics, and persistent limitations in human exposure assessment mean that human-relevant dose-response relationships for environmental micro- and nanoplastics remain unresolved. Uncertainty in internal dosimetry continues to pose a major challenge for interpreting animal studies and extrapolating findings to human health risk.

4 CONCLUSIONS

Since the previous review (*Health Risk Assessment: Microplastics*, FW22034) was conducted research output on the health risks posed by MNPs has increased rapidly, reflecting the growing concern over their ubiquity and potential to induce a broad range of adverse health effects. Approximately 3000 additional peer-reviewed studies examining the effects of MNPs on human health have been published, of which >1600 investigated the interactions with, and impacts on, tissues and organ systems; this body of literature continues to expand. Studies have continued to highlight the increasing scientific and public health concern regarding the ubiquity of MNPs, identify routes and magnitude of exposure, and their potential to trigger adverse health effects. It is now well established that the two predominant exposure pathways are ingestion and inhalation, and that dermal contact and medical procedures present other potential routes although evidence is still limited.

There is growing evidence of the presence of MNPs in human tissues and organs. Recent studies increasingly report MNPs in a wide range of tissues and bodily fluids, supporting the conclusion that size-dependent elimination of MNPs occurs with smaller particles able to enter the circulatory system and be translocated to other areas not directly exposed to the environment. While their internalisation and translocation are increasingly considered biologically plausible, substantial analytical limitations continue to generate uncertainty. Consequently, even where translocation across biological barriers is mechanistically plausible, the true prevalence, concentrations, and systemic distribution of MNPs in humans remain uncertain. Limitations of current analytical techniques also results in uncertainty of particle exposure levels, proportion of sizes and morphotypes and the associated chemical cocktail which hinders assessment of health risk. Resulting in much of the mechanistic evidence supporting barrier crossing is derived from *in vitro* and animal studies using virgin, fluorescent or surface-modified particles, which may not reflect real-world MNP exposure.

There is currently no direct evidence linking MNPs to adverse human health outcomes; however, the evidence base indicates that they are ubiquitous, biologically active, and capable of interacting with multiple physiological systems, with the potential to cause harm [14, 144, 145, 174-177]. Experimental studies consistently identify key mechanisms of toxicity, including inflammation, oxidative stress, genotoxicity, immune dysregulation, metabolic disruption, and the ability of smaller particles to cross biological barriers (e.g., placenta and blood-brain barrier), with effects observed across the respiratory, gastrointestinal, cardiovascular, nervous, immune, reproductive, and endocrine systems. MNPs act not only as physical stressors, but also as vectors for the transport and internal delivery of the plastic-associated chemicals, potentially facilitating their distribution to tissues that would otherwise be less exposed. This raises concern for combined or synergistic effects arising from both the physical properties of the particles and the chemical toxicity of associated substances. Overall, while direct human health effects have not been conclusively demonstrated, establishing causal evidence for complex issues like MNPs is inherently challenging. The existing evidence supports biologically plausible pathways for harm and uncertainty about real-world, low-dose exposure and long-term outcomes, supporting a precautionary approach. An additional dimension influencing risk, not in scope of this report due to its focus on biophysical and toxicological dimensions of health, is that of the cultural context, and including environmental connectedness (ki uta ki tai), and the potential for unequal distribution of

exposure associated with mahinga kai, and how contamination affects these relationships. Incorporating a wider range of perspectives in future work, including holistic health frameworks and place-based perspectives (central to te ao Māori), would support a more comprehensive understanding of risk.

Addressing uncertainties will require coordinated, transdisciplinary research that integrates advanced analytical methods, standardised methodologies, physiologically realistic exposure models informed by real-world environmental exposure levels, and longitudinal human studies alongside advanced laboratory models, including cell-based and tissue-based systems. This will be critical to fully elucidate risks, better characterise long-term health effects, and support evidence-based risk assessment, regulatory decision-making and effective mitigation strategies.

There is broad international agreement that both microplastics and nanoplastics represent a credible and emerging risk to human health, warranting a precautionary approach while critical knowledge gaps are addressed. The World Health Organisation (WHO) has emphasised that although the risks are not yet fully understood, these particles do not belong in the environment and that measures should be taken to mitigate exposure. Such measures may include eliminating unnecessary intentionally added primary MNPs, strengthening controls on chemicals of concern in plastics, and adopting lifecycle measures to reduce the release of secondary MNPs from large plastic items.

Establishing a national monitoring programme for MNPs across environmental domains (air, water, soil, biota, including food) and human populations would provide critical evidence to understand sources, transport pathways, environmental fate, and exposure routes. Such monitoring would support the identification of priority exposure pathways, inform mitigation and prevention strategies, and provide the foundation for robust risk assessments. Human biomonitoring would deliver direct evidence of internal exposure and generate the long-term, standardised datasets needed to track trends, evaluate the effectiveness of interventions, address key knowledge gaps, and support evidence-based policy and regulatory decisions. Ongoing monitoring and adaptive policy responses will be essential to ensure that management strategies remain effective as scientific understanding of MNP exposures and impacts continues to evolve.

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