Review Article

Tiny Particles, Major Risks: An Integrative Review of Micro- and Nanoplastics Exposure and Its Medical Significance

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ABSTRACT

The exceptional growth in plastic production has resulted in widespread environmental contamination by micro- and nanoplastics (MPs and NPs), raising significant concerns regarding human and animal health. Once considered chemically inert, plastics undergo continuous degradation into micro- and nano-sized particles capable of crossing biological membranes and interacting with organs and organ systems at cellular, genetic, and immunological levels. In this integrative review, we summarise current evidence on the sources, physicochemical properties, analytical detection techniques, exposure pathways, and toxicological effects of MPs and NPs, with particular emphasis on their medical and public health relevance. MPs and NPs have now been detected in air, water, soil, food, and human biological matrices, including blood, placenta, lungs, and arterial plaques. Experimental and observational studies demonstrate that exposure to MPs and NPs can induce oxidative stress, inflammation, mitochondrial dysfunction, endocrine disruption, and immune dysregulation, ultimately leading to organ-specific toxicity that affects multiple organ systems. Furthermore, animal studies indicate that chronic exposure to MPs and NPs is associated with metabolic disturbances, reproductive impairment, neurotoxicity, and ecological disruption across trophic levels. Despite advances in detection methods, including FT-IR spectroscopy and artificial intelligence–assisted techniques, substantial gaps remain in exposure assessment, dose–response relationships, and long-term health outcomes. This review highlights the urgent need to recognise MPs and NPs as emerging environmental toxicants and underscores the importance of standardised detection, biomonitoring, and preventive public health strategies to mitigate future risks.

Keywords: Microplastics, Nanoplastics, Environmental Toxicology, Human Exposure Pathways, Oxidative Stress, Organ Toxicity, Public Health Risk.

INTRODUCTION

The history of plastics is key to understanding the industry, dating back to the 1800s, when natural materials like cellulose and rubber were modified into semi-synthetics, such as Parkesine and Celluloid. In 1907, Leo Baekeland developed Bakelite, the first true synthetic plastic, known for its excellent electrical insulation, heat resistance, and versatility. This breakthrough shifted industry perspectives, showing that chemicals could create materials with tailored properties and spurred research into polymers. During World War II, the need for lightweight and durable substitutes led to innovations like nylon for parachutes, Plexiglass for aircraft canopies, and low-density polyethene (LDPE) for radar cable insulation, transforming chemical manufacturing and paving the way for high-volume polymer production. [1]

After the war, industrial machinery readily transitioned from producing military goods to civilian products, marking the beginning of the "Golden Age" of plastics. Inexpensive and flexible materials like polyethene (PE), polypropylene (PP), polyvinyl chloride (PVC), and polystyrene (PS) became ubiquitous, symbolising modernity and convenience. This led to a "throwaway culture," where single-use packaging and disposable items flourished. For years, plastic's resistance to rust and quick decomposition was advantageous, propelling advancements in the medical, food safety, and construction industries. While plastic improved living standards and technology, its durability contributed to today's environmental crisis. The linear "take-make-dispose" model, combined with a staggering annual production of 400 million tons, has strained waste management systems. Discarded plastics persist in the environment, breaking down into microplastics and nanoplastics, the most harmful stage in their life cycle. This paper aims to examine the unforeseen yet substantial impact of plastics, which, although celebrated for their functional inertness in the 20th century, disintegrate into particles capable of crossing biological barriers and accumulating within the physiology of people and animals.[1].

Method

This integrative narrative review synthesised multidisciplinary evidence on MPs and NPs, focusing on exposure pathways, toxicological mechanisms, organ-specific effects, and public health implications. Although a formal systematic review was not undertaken, methodological transparency was strengthened through structured search documentation and explicit reporting of the study selection process.

A comprehensive literature search was conducted across PubMed/MEDLINE, Scopus, and Google Scholar for studies published through June 2024. Search terms included combinations of "microplastics," "nanoplastics," "plastic

pollution," "human exposure," "toxicology," "oxidative stress," "immune dysregulation," "organ toxicity," and "environmental health," using Boolean operators (AND/OR). Reference lists of eligible studies were manually screened to identify additional relevant publications. The initial search yielded 6,924 records. After removal of 1,842 duplicates, 5,082 unique records remained for title and abstract screening. Screening was performed independently by two reviewers. Studies were excluded at this stage if they were unrelated to the health effects of MPs and NPs, focused solely on polymer engineering without biological relevance, were editorials or commentaries, or were not published in English. Following title and abstract screening, 4,321 records were excluded, and 761 articles were retained for full-text assessment.

Full-text evaluation was conducted using predefined inclusion criteria. Studies were eligible if they investigated exposure to MPs or NPs through environmental, dietary, inhalational, dermal, occupational, or experimental routes and reported biological, mechanistic, or clinical outcomes. Eligible outcomes included oxidative stress, inflammation, immune modulation, organ toxicity, metabolic disruption, reproductive effects, neurotoxicity, cardiovascular implications, detection methodology, or public health relevance. During full-text assessment, 706 articles were excluded due to a lack of clear health-related endpoints, insufficient exposure characterisation, and methodological limitations. After applying refined inclusion criteria emphasising translational relevance, mechanistic clarity, and methodological robustness, 55 studies were included in the final qualitative synthesis.

The final included studies comprised 15 human epidemiological or clinical investigations, 18 animal experimental studies, 12 in vitro mechanistic studies, 5 detection and analytical methodology studies, and 5 public health or policy-focused reports. Data extracted included study design, particle size and polymer type, exposure route, target organ or system, molecular mechanisms, and reported physiological or pathological outcomes.

The evidence was grouped and summarised by themes such as physical and chemical properties, methods of detection, routes of exposure, effects on different organs, and prevention methods. Due to heterogeneity in exposure metrics, particle characterisation, and outcome assessment, quantitative meta-analysis and formal risk-of-bias scoring were not feasible. However, only peer-reviewed literature was included, and mechanistic findings were interpreted cautiously to ensure scientific rigour.

Sources and Characteristics of Micro- and Nanoplastics

The emergence of microplastics, initially documented in the 1970s, has rapidly evolved into a global concern, driven by the degradation of traditional plastics. According to Oliveira and Almeida (2019) [2], the degradation of microplastics into the even smaller nanoplastics is a cause for concern, as nanoplastics are a "potentially more pernicious form of plastic pollution." The environmental distribution, biological absorption pathways, and potential toxicological effects of particles are directly correlated with their distinguishing characteristic: size. To do research on health and the environment, you need to know the limits and definitions of these two types. The size-based distinction is crucial because, as the source literature indicates, the reported deleterious effects of MPs and NPs on biota "range from depletion of energy reserves and altered metabolism to immunological, neurotoxic, and behavioural effects." The transition from the micrometre to the nanometre scale signifies a change in biological interaction, from physical irritation to systemic, chemical, and cellular toxicity.

Table 1: Specific characteristics that define these two categories based on current scientific consensus

Feature	Microplastics (MPs)	Nanoplastics (NPs)
Definition (Range)	Particles of plastic materials are generally defined as less than *5 mm* in length or diameter, but *1µm* or greater in diameter.	Particles of plastic materials are generally defined as less than *1 µm* in diameter, down to *1 nm* (nanometre).
Typical Size Range	*1 µm to 5 mm (or 5000µm)*	*1 nm to 1000 nm (or 1 µm).*
Structure and shape	Highly diverse. Can be fibres (from textiles), fragments (from bottles/containers), spheres (from microbeads), or films. Often, irregular shapes and heterogeneous surface chemistry are due to environmental weathering.	Typically amorphous or spherical, but the shape depends on the origin. Their ultra-small size dictates a very high surface area-to-volume ratio, significantly increasing their reactivity and capacity to adsorb other environmental contaminants.
Primary Origin	**Secondary source:** Fragmentation of larger plastic waste (e.g., fishing gear, packaging). **Primary source:** Intentional production for industrial media and cosmetic exfoliants.	**Secondary source:** Further degradation/breakdown of microplastics. **Primary source:** Intentional production for advanced applications like drug delivery and industrial coatings.
Mechanism of Bio-Uptake	Primarily, ingestion (e.g., via filter feeding or trophic transfer) leads to accumulation in the gastrointestinal tract, which can cause physical effects like reduced feeding and blockage.	**Translocation** is key. Uptake often involves endocytosis or passive diffusion due to ultra-small size. Capable of crossing biological barriers (like the intestinal wall) and accumulating systemically in organs and tissues [2].

Detection of micro- and nanoplastics

This review gives a comparison of the main analytical and microscopic methods used to detect and characterise MPs and NPs. These techniques differ in their operational principles, analytical resolution, and suitability based on particle size and sample matrix. Stereo (dissecting) microscopy (3, 4) and fluorescence microscopy (5, 6, 7) are two methods that quickly identify MPs based on physical traits such as size, shape, and colour. For more detailed morphological evaluations, scanning electron microscopy (SEM) (8,7), transmission electron microscopy (TEM) [7], and atomic force microscopy (AFM) (9, 10) offer high-resolution images and analyse surface topography. However, these procedures often do not provide adequate information. Determining the type of polymer may require lengthy processes or complex sample preparation. Fourier-transform infrared (FTIR) [11] and Raman spectroscopy are two spectroscopic techniques used for polymer identification. They work by detecting specific molecular vibrations or absorption bands unique to each polymer, allowing for accurate identification without damaging the samples. However, factors such as fluorescence interference, uneven surfaces, and the high cost of equipment can limit the effectiveness of these methods. Additional techniques, such as thermal analysis (TGA, DSC, Py-GC/MS) and Nile Red fluorescence staining [12, 13], can provide further insight into the composition and quantity of polymers. Thermal analysis determines polymer types by examining their thermal degradation or melting points, whereas Nile Red staining is a quick and cost-effective method for screening for MPs, particularly in field studies. New technologies, such as digital holography (DH), employ artificial intelligence to capture three-dimensional holographic images of MPs directly from water samples without the need for labels. Although still in the early stages of research with low technology readiness levels, these methods show considerable promise for real-time, in situ monitoring of MPs in complex environments [14].

Table 2: Comparative overview of analytical techniques used for detection and characterisation of microplastics (MPs) and nanoplastics (NPs), outlining their principles, advantages, limitations, and estimated Technology Readiness Levels (TRLs).

Method	Principle / Description	Advantages	Limitations	Estimated TRL [15]	Key References
Stereo (Dissecting) Microscopy	Uses reflected light to visualize 3D structure of large MPs fragments (≥100 µm).	- Fast and easy screening- Identifies shape, size, and colour	- Not confirmative of polymer type- Poor for transparent/small particles	5–7	[3, 4]
Fluorescence Microscopy	Detects emitted fluorescence from stained or intrinsic fluorophores in plastics (e.g., Nile Red).	- Detects transparent MPs- Rapid visualization- Can combine with imaging	- Fluorescence interference from additives- UV lasers may damage samples	4–6	[5, 6, 7]
Scanning Electron Microscopy (SEM)	Produces high-resolution images of MP surfaces using electron beams.	- High magnification and detail- Combined EDS gives elemental info	- Time-consuming- Expensive- Limited polymer identification	4–6	[8, 7]
Transmission Electron Microscopy (TEM)	Uses electron transmission through ultrathin samples for nanoscale visualization.	- Sub-nanometer resolution- Chemical analysis possible (EELS)	- Complex sample prep- Limited for amorphous polymers	1–2	[7]
Atomic Force Microscopy (AFM)	Measures topography by scanning a fine tip over the sample surface.	- 3D images- High resolution (0.3 nm)- No sample coating required	- Contamination risk- Tip may damage samples	2–4	[9, 10]
FTIR Spectroscopy (including µ-FTIR)	Detects infrared absorption bands specific to polymer bonds.	- Confirms polymer type- Non-destructive- Detects MPs <20 µm	- Time-consuming- Costly instruments- Interference on rough surfaces	5–7	[11],
Raman Spectroscopy (including µ-Raman)	Uses inelastic light scattering to identify molecular vibrations of polymers.	- Detects MPs/NPs (1 µm or less)- Non-destructive- No false positives	- Fluorescence interference- Expensive- Slow for large samples	5–7	[16,17]
Thermal Analysis (TGA, DSC, Py-GC/MS)	Analysis polymers by thermal degradation or melting behaviour.	- Identifies low-solubility polymers- Detects additives- Quantitative	- Destructive- Lacks particle size info- Complex data	3–5	[18]
Nile Red Fluorescent Staining	Stains plastics to fluoresce under blue light for rapid quantification.	- Fast, low-cost screening- Can quantify small MPs- Suitable for field use	- May co-stain organic matter- Requires acid cleaning (e.g., nitric acid)	6–8	[12, 13]
Digital Holography (DH)	AI-assisted 3D holographic imaging for identifying MPs in unfiltered water.	- Label-free 3D imaging- Differentiates MPs from biotic materials- In situ monitoring	- New technique; limited validation- Requires AI processing	2–4	[14]

TLR* – Technology Readiness Level (1 = early stage, 9 = full deployment), EELS = Electron Energy Loss Spectrometry, EDS = Energy-Dispersive Spectroscopy, UV = Ultra Violet, AI = Artificial Intelligence.

In summary, Table 2 illustrates that a combination of imaging, spectroscopic, and thermal methods is often essential for accurately identifying and fully characterising MPs and NPs, as no single method can provide complete information regarding morphology, polymer type, and concentration.

Chemical and Physical Properties of Micro- and Nanoplastics

The various physical and chemical characteristics of MPs and NPs impact their bioavailability, toxicity, and environment. The dimensions, form, density, colouration, and surface morphology of MPs and NPs influence their dispersion and interactions with biological and abiotic systems. Nanoplastics possess a greater surface area-to-volume ratio and increased reactivity as compared to microplastics, which measure between 1 µm and 5 mm. The disintegration of larger plastics generates fragments, fibres, films, foams, and pellets that affect sedimentation and ingestion. In aquatic environments, low-density polymers such as polyethene (PE), polypropylene (PP), polystyrene (PS), and polyvinyl chloride (PVC) remain buoyant. High-density polymers submerge in sediments. Stabilisers, plasticisers, flame retardants, colourants, and antioxidants are included in synthetic polymers to enhance the durability and performance of MPs and NPs. With the passage of time, these components leach away, rendering MPs and NPs chemically perilous. MPs and NPs function as vectors for secondary pollutants by adsorbing organic pollutants, heavy metals, and persistent organic contaminants from their environment due to their hydrophobic surfaces and elevated specific surface areas. Photo-oxidation, hydrolysis, and mechanical abrasion generate reactive functional groups that alter charge and hydrophilicity. These alterations affect MPs' and NPs' aggregation and enhance their interaction with natural colloids, dissolved organic matter, and cellular membranes. Owing to their intricate physical morphology and chemical structure, MPs and NPs exhibit significant dynamism and might experience ongoing environmental and biological modifications, complicating their identification and risk evaluation [7].

Exposure Pathways of Microplastics and Nanoplastics in Human

MPs and NPs are increasingly recognised as emerging environmental pollutants due to their widespread presence in air, water, and food. This text describes the primary ways these synthetic particles can enter the human body, their interactions with living organisms, and the potential health impacts they may have. The primary route of exposure to MPs and NPs is through consumption. These particles have been detected in various food sources, including seafood, bottled water, and table salt, which indicates that the global food supply is contaminated in numerous areas. Once ingested, MPs and NPs can interact with the epithelial cells in the gut, damaging the mucosal barrier and affecting oxidative stress and inflammation. Smaller, positively charged NPs can penetrate cell membranes via endocytosis or phagocytosis, allowing them to reach the liver and kidneys, among other organs. Additionally, MPs may absorb harmful chemicals, such as bisphenol A, phthalates, and heavy metals, increasing their toxicity [19, 20]. Inhalation is another significant exposure route, particularly in urban environments. Airborne MPs can originate from sources such as synthetic fabrics, tyre wear, industrial emissions, and household dust. Fine particles smaller than 10 micrometres can penetrate deep into the alveolar region of the lungs, leading to increased oxidative stress and inflammation, as well as mitochondrial issues. The accumulation of MPs in lung tissue is associated with long-term respiratory irritation, fibrosis, and potential systemic toxicity, as these particles can enter the bloodstream [21]. Dermal contact is generally considered a minor exposure pathway, occurring mostly through cosmetics, exfoliating scrubs, personal care products, and synthetic textiles. Nanoplastics can enter the body through the skin through sweat ducts, hair follicles, or small wounds, potentially causing localised oxidative stress and inflammation. While systemic absorption through intact skin is minimal, compromised or damaged skin may allow for deeper penetration, leading to chronic low-level exposure [19, 22].

Exposure of Microplastics and Nanoplastics in Animals.

The various environmental occurrences, exposure pathways, and toxicological aftereffects of MPs and NPs in aquatic, terrestrial, and animal systems. These particles originate from the breakdown of larger plastic items; they have infiltrated the entire food chain [23, 24]. In aquatic ecosystems, microplastics become part of the food web through ingestion by primary consumers such as plankton and filter feeders. Their size, density, and shape affect their distribution in surface waters, sediments, and deep-sea layers, which in turn influences their bioavailability at different trophic levels. Aquatic species that continuously consume these particles often experience reproductive difficulties, reduced energy levels, and feeding challenges [25–27]. Moreover, MPs act as chemical vectors, capable of adsorbing and transporting harmful substances, such as bisphenol A, phthalates, polycyclic aromatic hydrocarbons (PAHs), and heavy metals. This increases the risk of systemic poisoning in species that come into contact with these pollutants [24]. Land animals can also be exposed to MPs and NPs through contaminated feed and water, allowing these contaminants to enter the food chains of agricultural settings. Ingestion of these particles has been linked to gut inflammation, disruption of the intestinal barrier, and dysbiosis of the gut microbiota. In cattle and other animals, MPs can induce hepatic inflammation, oxidative stress, and an altered cytokine balance, resulting in increased levels of IL-6 and TNF-α, and decreased levels of IL-10, ultimately leading to metabolic and immunological dysfunction [25–27]. Reproductive toxicity is well-documented, showcasing damage to ovarian and testicular tissues. The granulosa cell death and reduced fertility have been observed in rodents, aquatic animals, and cattle. Additionally, microplastics can hinder the movement of birds and soil organisms. It is more difficult for them to find food and survive. The interaction between MPs and microbial populations heightens

ecological risks. The surfaces of microplastics facilitate the development of biofilms, enabling bacteria, fungi, and viruses to adhere and spread antibiotic resistance genes. Long-term exposure to MPs can lead to systemic inflammation, fibrosis, and degeneration of vital organs, including the liver, kidneys, and spleen. Table 4 illustrates the impact of micro- and nano-plastics on animals and the environment.

Table 3: Summary of environmental occurrence, biological pathways, and toxicological impacts of micro- and nanoplastics (MPs and NPs) in aquatic, terrestrial, and animal systems, including their ecological and health implications.

Aspect	Details	Key References
Environmental Occurrence & Entry	Microplastics (MPs) are widespread in aquatic environments, entering the food chain through ingestion by organisms at the base of the food web. Their size, shape, and density determine distribution across surface waters, sediments, and deep layers, affecting bioavailability across trophic levels.	[23], [24]
Feeding & Digestive Disruption	MPs disrupt feeding and digestion in algae and filter feeders, leading to reduced energy intake, weight loss, reproductive failure, and mortality.	[23]
Chemical Vector Effects	MPs act as carriers for toxic additives and environmental contaminants (e.g., carcinogenic, mutagenic, and endocrine-disrupting compounds), which can leach and cause systemic toxicity.	[24]
Routes of Ingestion	Direct ingestion (mistaken for food) or indirect ingestion via trophic transfer (contaminated prey consumed by higher predators). MPs may also adhere to organism surfaces, causing physical irritation and stress.	[23 – 24]
Terrestrial & Animal Exposure	MPs and NPs enter terrestrial ecosystems through animal feed and aquatic products, posing risks to food safety and potential transmission to humans.	[25–27]
Gastrointestinal Effects	Induce gut inflammation, intestinal barrier damage, dysbiosis of gut microbiota, and impaired growth in exposed animals.	[25,26]
Hepatic Effects	Cause liver inflammation and metabolic disorders, marked by increased IL-6, TNF-α, decreased IL-10, immune dysfunction, and oxidative stress.	[25,26]
Reproductive Toxicity	Damage to ovarian and testicular tissues, granulosa cell death, and reduced fertility in rodents, aquatic organisms, and cattle.	[25–27]
Mechanical Obstruction in Birds & Soil Organisms	MPs cause digestive blockage, reduced gizzard size, impaired foraging, and stunted development in poultry and soil fauna.	[26]
Microbial & Biofilm Interactions	MP surfaces promote biofilm formation, facilitating bacterial, fungal, and viral colonization and spreading antibiotic resistance genes.	[26]
Systemic & Organ Effects	Chronic MPS AND NPS exposure causes inflammation, fibrosis, and degeneration in liver, kidney, and spleen of rodents and livestock.	[26,27]
Neurotoxicity	MPs and NPs cross the blood–brain barrier, leading to neuroinflammation, altered neurotransmitter levels, and cognitive impairment.	[27]
Genotoxicity & Epigenetic Effects	Induce DNA damage, oxidative stress, and alterations in gene expression and methylation patterns, leading to genotoxic and epigenetic disruptions.	[27]
Bioaccumulation in Edible Tissues	MPs and additives persist in consumable tissues (muscle, liver, milk, eggs, shellfish), raising human dietary exposure risks.	[25–27]
Metabolic Disturbances	Elevated blood glucose and cholesterol observed in rats and sheep, indicating disrupted glucose and lipid metabolism pathways.	[25,26]
Overall Implication	MPs and MPs and NPs are pervasive environmental and biological threats causing oxidative stress, immune dysregulation, genetic impairment, and reproductive dysfunction. Calls for global monitoring, regulatory action, and sustainable waste management.	[25–27]

Organ-Specific Effects

This text outlines the effects of MPs and NPs on humans and animals. These synthetic particles can enter the body through various environmental routes and affect multiple organ systems, leading to oxidative, inflammatory, and metabolic issues. In urban areas, there are multiple ways of contamination with MPs and NPs from sources like synthetic fabrics, tyre wear, and household dust, which is a key exposure route discussed in Table 4.

Table 4: Organ-specific exposure routes, molecular mechanisms, and physiological effects of microplastics (MPs) and nanoplastics (NPs) in humans and animals.

Exposure Pathway	Primary Sources	Mechanism of Entry and Retention	Cellular & Molecular Effects	Physiological/Health Implications	Key References
Inhalation	Airborne MPs and NPs from synthetic textiles, tyre wear, industrial emissions, and household dust.	- Inhaled particles reach alveoli and alveolar ducts.- Partially cleared via mucociliary transport, macrophage phagocytosis, or lymphatic drainage.- Smaller fibres and nanoparticles evade clearance and accumulate in deep lung tissue.	- Induce oxidative stress, chronic inflammation, and fibrotic responses. - Trigger TNF-α–mediated apoptosis in alveolar cells (A549). - Cause ROS generation, MMP-9 and SP-A upregulation, and disruption of redox homeostasis.- Elevate IL-6, IL-1β, and TNF-α in animal models. - Increase susceptibility in individuals with pre-existing respiratory diseases (e.g., cystic fibrosis).	- Cytotoxicity, airway inflammation, and loss of epithelial barrier integrity. - Potential for asthma, fibrosis, and COPD development. - Persistent retention in lung tissue indicates possible systemic distribution. - Long-term effects remain under-researched.	28,29
Gastrointestinal Tract	Contaminated food, beverages, and packaging materials.	- Ingestion introduces MPs to digestive enzymes, mucus, and epithelial lining. - Particles cross intestinal barrier and enter circulation via portal vein to the liver. - MPs adsorb toxic substances and disrupt gut microbiota.	- Induce oxidative stress, apoptosis, and inflammatory cytokine release. - Impair intestinal barrier and increase permeability. -Elevate CYP2E1 expression and suppress PPAR, promoting lipid peroxidation and energy dysregulation.- Activate TGF-β and Wnt/β-catenin pathways, leading to hepatic fibrosis. - Damage β-cells, reducing insulin synthesis and altering glucose metabolism.	- Intestinal inflammation, gut dysbiosis, and metabolic disorders. - Risk of hepatic fibrosis, insulin resistance, and energy imbalance. - Limited human data; effects depend on polymer type, size, and dose. - Highlights need for standardized exposure and biomonitoring studies.	30
Liver System	Ingestion via contaminated food, water; inhalation of airborne particles	Translocation through the portal vein; accumulation in hepatocytes, Kupffer and sinusoidal endothelial cells	ROS production, mitochondrial damage, activation of p53/Bcl-2/Bax and PERK pathways; CYP2E1 enzyme disruption; ATP depletion	Oxidative stress, hepatocyte apoptosis, steatosis, fibrosis, cirrhosis, and metabolic dysfunction resembling MAFLD	31–35
Reproductive System	Dietary exposure, inhalation, medical plastics, skin absorption	Crosses blood–testis and placental barriers; accumulates in gonadal tissues	Oxidative stress, endocrine disruption, mitochondrial dysfunction; activation of MAPK, Nrf2/HO-1/NF-κB, and LHR/cAMP/PKA/StAR pathways	Reduced sperm count and motility, decreased testosterone, follicular atresia, impaired fertility, embryotoxicity	36,37 38 39,40 41
Nervous System	Ingestion, inhalation, dermal absorption from polluted air, food, or water	Crosses blood–brain barrier via tight junction disruption; retained in neurons and glial cells	ROS and lipid peroxidation, mitochondrial dysfunction, neurotransmitter imbalance (ACh, DA, 5-HT), activation of NLRP3 inflammasome	Neuroinflammation, neuronal death, cognitive impairment, behavioural alterations, Parkinson-like neurodegeneration	42–45
Immune System	Ingestion, inhalation, dermal exposure to MPs/NPs	Uptake by macrophages, neutrophils, dendritic and epithelial cells; formation of protein corona	Activation of TLR and NF-κB pathways, cytokine release (IL-1β, TNF-α, IL-6), oxidative stress, and apoptosis; epigenetic reprogramming ("trained immunity")	Chronic inflammation, impaired immune regulation, autoimmune susceptibility, and altered pathogen response	46–48

Public Health Control Measures

Effective public-health management of MPs and NPs requires. This will be achieved by coordinated primary, secondary, and tertiary interventions to reduce human exposure and prevent long-term health consequences. Primary prevention focuses on eradicating sources of plastic pollution. This involves banning non-essential single-use plastics, restricting microbeads in cosmetics, requiring biodegradable or compostable packaging, and implementing strict regulations on harmful plastic additives, such as bisphenol A (BPA) and phthalates, which can disrupt endocrine function and cause metabolic disorders [49].

Strengthening waste-management systems, especially in low- and middle-income countries, is crucial; improvements include the development of advanced material-recovery facilities, plastic-to-fuel conversion units, and the implementation of extended producer responsibility (EPR) policies. Secondary prevention aims to reduce ongoing exposure to risk factors. This involves routine monitoring of MPs/NPs in drinking water, seafood, table salt, atmospheric particulate matter, and indoor air, where synthetic textiles and tire-wear particles significantly contribute to airborne MPs [49]. Adding microfibre filters to washing machines, upgrading wastewater treatment plants with membrane bioreactors, and installing HEPA filters in high-risk industrial settings can substantially reduce environmental release. Similarly, food industries, particularly those in the seafood and dairy sectors, should implement periodic testing for plastic contamination.

Tertiary prevention focuses on improving health outcomes in populations exposed to risk factors. Occupational health guidelines must mandate the use of protective equipment for workers in textile mills, plastic manufacturing, waste recycling, and construction. Public health authorities should establish national or regional databases that document suspected plastic-related illnesses, enabling clinicians to correlate exposures with respiratory, metabolic, reproductive, and cardiovascular disorders. As microplastics have recently been detected in human blood and arterial plaques [50], integrating microplastic surveillance into environmental health programs is now considered essential.

What Should Have Been Done Earlier

Many of the health and environmental risks associated with MPs/NPs could have been significantly mitigated if proactive decisions had been taken during the early expansion of global plastic production. Historically, plastic manufacturers and regulatory bodies assumed that synthetic polymers were chemically inert and posed negligible health risks to humans. However, evidence dating back to the 1970s already indicated that plastics fragment into smaller particles and accumulate in marine organisms (51). Had policymakers acted at that time, several interventions could have prevented the widespread contamination of human tissues today.

Firstly, international regulators should have implemented early bans on microbeads and restricted the production of plastics containing hazardous additives such as BPA, phthalates, and brominated flame retardants, compounds now linked to endocrine dysfunction, infertility, and metabolic disorders [52]. Secondly, wastewater treatment plants worldwide should have adopted microplastic-removal technologies as early as the 1990s when microfibres from synthetic clothes were first reported in effluent streams. Thirdly, governments should have introduced global treaties, similar to the Montreal Protocol, to limit the production of non-recyclable plastics, mandate circular economy approaches, and enforce corporate accountability. Equally important, medical and scientific communities should have integrated environmental toxicology into training curricula, enabling earlier identification of MPs/NPs as emerging pollutants. Research funding for plastic toxicology, particularly its metabolic, reproductive, and neurological effects, should have been prioritised decades ago. The absence of these early interventions has contributed to today's scenario, where MPs/NPs are detected in human blood, placenta, lungs, stool, and recently, in cardiovascular plaques, indicating deep systemic penetration that might have been prevented with timely action [50,53].

Additional Supporting Information and New Evidence

Recent scientific advances have significantly transformed our understanding of the human health implications of MPs and NPs. In 2022, Leslie et al. provided the first evidence of polymer particles circulating in human blood, including polyethylene terephthalate (PET) and polyethylene (PE), confirming systemic absorption following ingestion and inhalation [50] In 2023, Jenner et. al. discovered plastic nanoparticles lodged within human carotid atherosclerotic plaques, raising concerns that chronic exposure may accelerate vascular inflammation, endothelial dysfunction, and plaque instability, potentially increasing risks of stroke or myocardial infarction [53]. Additional studies reveal that MPs and NPs accumulate in the placenta and can cross the maternal, fetal barrier, suggesting possible long-term implications for fetal development, immune imprinting, and metabolic programming [54]. Furthermore, MPs and NPs have shown significant neurotoxic potential, with experimental studies demonstrating their ability to cross the blood–brain barrier, activate microglia, induce oxidative stress, and disrupt neurotransmitter balance, mechanisms linked to cognitive impairment and early neuroinflammation [55]. In the reproductive system, both male and female animal models show reduced sperm motility, testicular oxidative stress, ovarian follicular apoptosis, and impaired fetal growth following chronic exposure [56,57]. Evidence is also emerging that chronic exposure to microplastics alters the human microbiome, potentially contributing to the development of metabolic syndrome, obesity, inflammatory bowel diseases, and liver dysfunction [58].

Future aspects of micro- and nanoplastics, prevention roadmap

These new findings suggest that MPs and NPs are physiologically active particles that can overcome physiological barriers, accumulate in organs, influence metabolism, and potentially contribute to the development of chronic illnesses. Based on this information, the medical community should treat MPs and NPs as an urgent environmental toxicant class, similar to heavy metals or persistent organic pollutants, and call for accelerated research, monitoring, and prevention efforts. To prevent MPs and NPs from repeating these mistakes in the future, we need to take several steps, starting with observation and standardisation. Create globally standardised sampling and analytical methods for microplastics in wastewater treatment facilities (WWTPs), as FTIR and Raman spectroscopy exhibit significant variability across studies, to facilitate accurate risk assessment. MPs and NPs biomonitoring in public health surveillance can identify high-exposure groups and inform targeted treatments by collecting blood, placenta, faeces, indoor air, and other human matrices [59,60].

Legislation and product design should include bans or strict limits on intentionally added microplastics (like cosmetic microbeads) and require companies to tell customers about plastic additives. Extended Producer Responsibility (EPR) models may encourage producers to make products that are less likely to break, use safer chemicals, and collect materials that are no longer useful. Microfibers released during the washing of clothes are a significant source of MPs and NPs emissions. People can reduce microfiber release by installing filters in their washing machines, which have been shown to be effective over time. Adding more built-in filters to new machines and retrofit kits to old ones would significantly reduce pollution [61,62]. Changing how we handle waste is also important, along with the actions we take at home. To prevent MPs and NPs from entering effluent or sludge, WWTPs must install high-efficiency membrane modules, coagulation-based separation, and new sorbents. Conventional treatment facilities are unable to detect or eliminate all nanoplastics, and numerous microplastics persist in sludges. Bio-inspired adsorbents, materials incorporating enzymes, and hybrid systems that utilise physical, chemical, and biological processes should be the primary focus of research on remediation. We need to test these changes in real-world WWTPs and greywater systems to see how well they work, how much they cost, how much energy they use, and what their long-term effects are. A strong international plastics treaty with clear, enforceable goals for reducing MPs and NPs through design standards, limits on production, bans on dangerous materials, and recycling requirements is needed for regulation and global governance. National governments can do this by making action plans with measurable goals (like cutting down on microfibre emissions or upgrading the filtration system at a treatment plant) and using procurement or EPR frameworks [63]. Long-term cohort studies assessing MPS and NPS exposure (quantified by biomarkers) and outcomes such as respiratory illnesses, metabolic health, neurodevelopment, and reproductive effects are crucial for public health and research. Clinical recommendations should also include information about MPS and NPS exposure in the workplace, such as during the manufacturing of textiles, the production of tyres, or the recycling of waste. This should include safety measures, exposure limits, and monitoring [64,65]. Educating consumers could lead to changes in behaviour and the market, such as washing clothes less frequently, choosing fabrics that shed less, using packaging that can be reused or recycled, and disposing of trash properly. These methods can influence customer behaviour, alongside eco-labels, product transparency, and social nudges. Prize funds or public-private partnerships that pay for and guarantee the purchase of high-potential technology pilots (like new filters and sorbent systems) can also encourage new ideas. Three important areas should finally guide research in the near future: (1) Lowering detection limits and developing methodologies for complicated environmental matrices to improve nanoplastics detection, (2) advancing toxicological studies on human health, including mechanism of action, dose–response, and long-term effects, and (3) conducting real-world intervention trials (in homes, WWTPs) for removal technologies with socioeconomic and life-cycle assessments to ensure sustainability and equity.

CONCLUSION

MPs and NPs are now widespread pollutants with growing concerns for environmental, animal, and human health. Their persistence, fragmentation, and long-range movement have led to their widespread detection in air, water, soil, food systems, and human biological matrices, including blood, placenta, lungs, and arterial plaques. MPs and NPs may cross biological boundaries and induce oxidative stress, endocrine disruption, immunological modulation, and alterations in the metabolic, reproductive, neurological, and cardiovascular systems in animal models and cells. In vitro and animal studies provide much of the existing mechanistic data; however, exposure concentrations may not adequately match real-world human exposure scenarios. Thus, while these findings are concerning, direct causal links and long-term health repercussions in humans are poorly understood and warrant caution. Chronic low-dose exposure, cumulative effects over the lifetime, acceptable exposure limits, and interactions between MPs and NPs and co-existing environmental contaminants are poorly understood. A coordinated, multi-level strategy is needed to address these uncertainties. Exposure assessment must improve by strengthening environmental and biological surveillance systems, standardising detection and characterisation methods, and increasing human biomonitoring. Restricting the purposeful addition of microplastics, encouraging safer material design, and extending producer accountability are crucial to reducing microplastic pollution. Improved wastewater treatment technology, household source-control methods (e.g., washing machine filtration), and sustainable remediation studies may further reduce environmental buildup.

Coordinated worldwide regulatory frameworks, well-designed long-term human cohort studies, and integrated public health programs are needed to assess hazards and inform policy. While doubts exist, the accumulating amount of research suggests that MPs and NPs contamination is an environmental and public health issue that requires proactive and cautious intervention.

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