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Microplastics in the Environment and Human Health: Current State of Knowledge and Future Research Directions

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Abstract

Microplastics (MPs) are plastic particles smaller than 5 mm in size. They have become one of the most ubiquitous environmental contaminants worldwide. Their presence has been documented in all major environmental reservoirs, including oceans, rivers, soils, and the atmosphere, and in recent years they have also been detected directly in human tissues and bodily fluids. This review paper presents the current state of knowledge regarding the sources, distribution, and environmental impacts of microplastics, pathways of human exposure, and the mechanisms underlying their potential effects on health. Particular attention is devoted to evidence concerning the presence of microplastics in the respiratory, gastrointestinal, cardiovascular, and urogenital systems, as well as their influence on the gut microbiome. Existing clinical evidence is also discussed, including the landmark study published in *The New England Journal of Medicine* in 2024, which demonstrated an association between the presence of microplastics in atherosclerotic plaques and an increased risk of cardiovascular events.

Future research priorities and strategies for reducing exposure at both individual and systemic levels are also presented.

Aim: This narrative review synthesizes up-to-date international guidelines and literature (published between 2017 and 2025), focusing on health education and clinical communication as vital yet underutilized contributors to patient safety.

Keywords: microplastics, nanoplastics, human health, environmental pollution, toxicology, environmental exposure

1. Introduction

Plastics have revolutionized modern civilization, becoming an indispensable component of industry, packaging, medicine, construction, and everyday life. Since 1950, global plastic production has increased from merely 2 million tonnes per year to more than 400 million tonnes annually in the second decade of the twenty-first century, and projections indicate continued growth in plastic production [1]. It is estimated that by 2050, the cumulative mass of plastic waste will reach 12 billion tonnes [2]. One consequence of this unprecedented increase is the widespread presence of plastic debris, known as microplastics, throughout all components of Earth's ecosystems.

The term *microplastic*, introduced into the scientific literature by Thompson and colleagues in 2004, refers to plastic particles smaller than 5 mm. No universally accepted lower size limit has yet been established, although some researchers use 1 μm as the threshold below which particles are classified as nanoplastics [3]. Since the first description of microplastics in marine sediments and water samples, two decades have passed during which the number of scientific publications on this topic has grown almost exponentially-from a few dozen publications annually to several thousand.

Microplastics have been detected in all natural environments, ranging from deep ocean trenches to Arctic ice sheets, from agricultural soils to urban atmospheric air. Their ubiquity has inevitably led to human exposure, as evidenced by numerous studies published in recent years documenting the presence of microplastics in human feces [4], blood [5], lungs [6], placenta [7], breast milk [8], and even atherosclerotic plaques within carotid arteries [9]. Of particular concern is the fact that microplastics have also been detected in samples obtained from newborns and fetuses, indicating the possibility of prenatal exposure to these contaminants.

Despite the growing body of evidence, knowledge regarding the actual impact of microplastics on human health remains incomplete and controversial in many respects. The concentrations used in most experimental studies greatly exceed levels measured in human tissues, making extrapolation of findings to the general population difficult. Nevertheless, several potential mechanisms of harm-including oxidative stress, inflammation, endocrine disruption, and toxicity to the gut microbiome-have been identified, justifying intensified research and regulatory efforts.

The aim of this review is to present the current scientific understanding of the definition, classification, and sources of microplastics in the environment; pathways of human exposure; the distribution of microplastics within the human body; mechanisms of toxicity; and their effects on individual human organs and physiological systems.

Research Material and Methods

Participants

This narrative review did not involve primary recruitment of human participants. The study material comprised peer-reviewed scientific publications retrieved from the PubMed and PubMed Central databases. A total of 45 references published between January 2017 and April 2025 were included, covering original research, systematic reviews, meta-analyses, and institutional reports relevant to microplastic exposure and human health.

Procedure / Literature Search Protocol / Search Trial / Measures / Instruments

The literature search was conducted using thematic Boolean queries combining MeSH controlled vocabulary and free-text keywords (e.g., *"microplastic*"[tiab] AND "human health"[tiab]*) across nine thematic blocks: environmental distribution, exposure routes, tissue presence, toxicological mechanisms, cardiovascular, respiratory, reproductive, neurological effects, and regulatory aspects. Records were screened in two stages: title/abstract review (~420 records) followed by full-text eligibility assessment (~110 articles). Inclusion criteria required publications from 2017 onwards addressing MP sources, human exposure, biological presence, or health effects. Exclusion criteria eliminated conference abstracts, editorials, retracted articles, and studies relying solely on visual (non-spectroscopic) particle identification. Polymer identity in assessed primary studies was required to be confirmed by micro-FTIR, micro-Raman spectroscopy, or Py-GC/MS.

Data Collection and Analysis / Statistical Analysis

Data were extracted from each included publication according to a standardised schema covering: study design, biological matrix, MP type and size, analytical detection method, exposure route, and key outcomes. Findings were synthesised narratively by thematic category. No quantitative pooling of results was performed due to the heterogeneity of study designs, MP particle types, and outcome measures across included studies.

Statistical Software

Statistical processing was not applicable to the present narrative review, as no original quantitative data were collected or analysed by the authors. All numerical values cited in the text were sourced directly from the included publications and are reported in their original form. Data visualisation and tabulation were performed using Microsoft Word (Microsoft Corporation, Redmond, WA, USA).

2. Definition, Classification, and Physicochemical Properties of Microplastics

2.1. Definition and Size Limits

Microplastics are defined as solid particles composed of synthetic polymers ranging in size from 1 μm to 5 mm, although the lower size threshold varies among researchers and regulatory organizations [3]. Particles smaller than 1 μm are referred to as **nanoplastics (NPs)** and exhibit distinct physicochemical properties, including greater surface reactivity, enhanced ability to cross biological barriers, and increased cytotoxicity. In 2023, the European Commission defined microplastics as synthetic materials containing polymers at concentrations exceeding 1% by weight, with the largest particle dimension ranging from 1 nm to 5 mm [3].

Based on shape, five principal categories of microplastics are distinguished: **fragments** (irregular pieces of plastic), **fibers**(microfibers originating from synthetic textiles), **pellets** (industrial feedstock granules), **beads** (microbeads used in cosmetic products), and **foams** (fragments of expanded polystyrene). Each of these forms exhibits distinct aerodynamic and hydrodynamic properties, which determine their transport and deposition in the environment as well as their biological effects [10].

2.2. Classification into Primary and Secondary Microplastics

Primary microplastics are intentionally manufactured at microscopic sizes for direct use. They include microbeads used in cosmetics, oral hygiene products, and exfoliants; industrial resin pellets employed as raw materials in plastic manufacturing; pharmaceutical microparticles; and granules used as infill material for artificial turf fields [10,11]. It is estimated that primary microplastics account for approximately 15-28% of total microplastic emissions into the oceans.

Secondary microplastics are generated through the fragmentation of larger plastic waste under the influence of physical factors (UV radiation, mechanical abrasion, wave action), chemical processes (hydrolysis, oxidation), and biological degradation. Major sources of secondary MPs include tire wear from road traffic (estimated at 1.3-2.0 million tonnes annually worldwide), laundering of synthetic textiles, fragmentation of plastic bags, PET bottles and packaging materials, and degradation of fishing gear [11,12]. During a single washing cycle of a fleece jacket, approximately 0.1-0.5 grams of microfibers may be released into the environment [12].

2.3. Chemical Composition and Adsorptive Properties

The most commonly identified polymers constituting microplastics include polyethylene (PE, approximately 28.5% of all plastics), polypropylene (PP, approximately 16.7%), polyvinyl chloride (PVC), polystyrene (PS), polyethylene terephthalate (PET), polyamide (PA/nylon), and polycarbonate (PC) [10]. Each polymer differs in density, hydrophilicity, resistance to degradation, and capacity to adsorb chemical substances.

One of the key properties of microplastics is their ability to adsorb persistent organic pollutants such as polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), dichlorodiphenyltrichloroethane (DDT), and heavy metals including lead, cadmium, and mercury [4]. Microplastics may concentrate these substances at levels up to one million times higher than those found in surrounding water, thereby acting as vectors for toxic xenobiotics within food chains. Furthermore, plastics themselves contain numerous additives introduced during manufacturing-including plasticizers (phthalates, bisphenol A), flame retardants (polybrominated diphenyl ethers, PBDEs), UV stabilizers, and pigments-which may leach into biological tissues and fluids [4,13].

3. Distribution of Microplastics in the Environment

3.1. Marine and Freshwater Environments

The oceans constitute one of the largest reservoirs of microplastics on Earth. It is estimated that by 2016, the total mass of plastic floating on the ocean surface ranged from 93,000 to 236,000 tonnes, while the annual influx of new plastic waste into marine environments reached approximately 11 million tonnes [1]. Particular attention has been drawn to plastic accumulation zones, commonly known as *garbage patches*, the largest of which is the **Great Pacific Garbage Patch**, covering an area of approximately 1.6 million km^2. Microplastics have even been detected at depths approaching 10,000 meters in the Mariana Trench, highlighting the global extent of this contamination [5].

Freshwater ecosystems are similarly affected. Microplastics have been detected in rivers, lakes, reservoirs, and groundwater systems on every continent. Rivers are estimated to transport between 70,000 and 2.4 million tonnes of plastic into the oceans annually, serving as major pathways through which terrestrial pollution reaches marine environments [14]. Wastewater treatment plants, although effective at removing a substantial proportion of microplastics from water, simultaneously act as points of accumulation. Sewage sludge may contain up to 56,000 MP particles per kilogram of dry weight, and because such sludge is often applied as agricultural fertilizer, it can facilitate the transfer of MPs into soils [15].

3.2. Terrestrial and Soil Environments

In recent years, increasing scientific attention has been directed toward terrestrial ecosystems, which are estimated to accumulate between four and twenty-three times more microplastics than marine environments [15]. Major sources of MPs in soils include sewage sludge used as fertilizer, plastic mulches employed in agriculture, atmospheric deposition of airborne microplastics, and fragmentation of plastic waste discarded in the environment. Studies have shown that agricultural soils amended with sewage sludge may contain between 0.3 and 67,500 MP particles per kilogram of soil, with synthetic fibers predominating [15].

Of particular concern is the ability of microplastics to enter plant tissues through root systems and subsequently enter the food chain. Microplastics have been detected in the leaves, fruits, and grains of wheat, lettuce, carrots, and other crops cultivated in contaminated soils [16]. The primary mechanism of uptake involves pores on root surfaces through which particles smaller than 200 nm may pass, creating a potential route of human exposure through the consumption of fruits and vegetables.

3.3. Atmospheric Environment

The atmosphere represents a less obvious but increasingly well-documented reservoir and transport pathway for microplastics. Atmospheric microplastics may be transported over distances of thousands of kilometers, and their presence has been documented even in Antarctic and Arctic snow, as well as on remote mountain peaks in the Pyrenees, far from any identifiable point sources [17]. In urban environments, concentrations of airborne MPs may range from several to several hundred particles per cubic meter. Synthetic fibers originating from textile wear and laundering, along with particles derived from tire wear and road surfaces, are the predominant forms detected [17].

An important but often overlooked source of indoor airborne microplastics includes carpets, curtains, upholstered furniture, and even clothing. Consequently, concentrations of microplastics within indoor environments may exceed those measured outdoors [18].

4.1. Ingestion Through Food and Drinking Water

Dietary intake is considered the primary route of human exposure to microplastics (MPs). Cox and colleagues estimated that the average American ingests between 39,000 and 52,000 MP particles annually through food and beverages alone, with this figure increasing to 74,000-121,000 particles per year when inhalation exposure is also taken into account [19]. Fish and seafood represent particularly important sources of MPs; annual intake through shellfish consumption may reach approximately 11,000 particles, while fish consumption may contribute around 8,300 particles [20]. MPs have been detected in both freshwater and marine fish, squid, shrimp, oysters, mussels, and even in table salt, honey, sugar, beer, wine, milk, and other dairy products [10].

Drinking water-both tap water and bottled water-is also widely contaminated with microplastics [21]. A meta-analysis encompassing 21 studies demonstrated the presence of MPs in drinking water samples from all continents. Concentrations were generally higher in bottled water than in tap water, suggesting that plastic bottles and bottling processes themselves may contribute additional contamination [21]. Kosuth et al. reported that more than 81% of tap water samples collected from 14 countries worldwide contained MPs [22]. A World Health Organization (WHO) report published in 2019 confirmed the widespread occurrence of MPs in drinking water while concluding that available evidence was insufficient to determine adverse health effects, emphasizing the need for further research [22].

Plastic packaging constitutes another significant source of exposure. Studies have shown that single-use plastic bottles release thousands of polymer particles into water. Of particular concern is the heating of food in plastic containers: one minute of microwaving a polypropylene food container can release more than 4 million microplastic particles and billions of nanoplastic particles into food [23]. Similarly, tea brewed in plastic tea bags has been shown to release more than 11 billion microplastic particles into a single cup of tea [23].

4.2. Inhalation

Inhalation represents the second most important route of MP exposure, particularly in urban and industrial environments. Airborne MPs can be inhaled and deposited throughout the respiratory tract depending on their size, shape, and density. Particles measuring 10-100 µm are primarily retained in the nasal cavity and pharynx, particles between 1 and 10 µm can reach the bronchi, while particles smaller than 1 µm may penetrate into the alveoli and potentially enter systemic circulation [24]. Daily inhalation exposure in adults has been estimated to range from several dozen to several thousand particles, depending on living conditions and environmental characteristics [18].

Evidence supporting inhalation as a significant exposure pathway comes from studies demonstrating the presence of MPs in human lung tissues obtained during autopsy and surgical procedures. Amato-Lourenço et al. [25] detected MPs in 13 of 20 lung tissue samples, predominantly polyethylene (PE) and polypropylene (PP) particles measuring less than 5.5 µm. Jenner et al. (2022) identified MPs in 11 of 13 patients undergoing surgery, with particles detected in both the upper and lower respiratory tract [26]. Huang et al. (2022) identified MPs in the sputum of patients with respiratory diseases, reporting up to 565 particles per 10 mL of sputum and identifying 21 different polymer types [27].

4.3. Dermal Contact

Dermal (transcutaneous) contact represents a third potential route of exposure to MPs; however, its significance remains the least understood to date [28]. The skin, with its keratinized barrier, constitutes an effective obstacle to most particles. Penetration through intact skin appears possible only for nanoparticles and ultrafine particles smaller than 100 nm. Some evidence suggests that polystyrene nanoparticles may penetrate damaged or diseased skin. In practical terms, dermal exposure may result from contact with cosmetic products containing microbeads (which are increasingly being restricted through legislation), synthetic textiles, and various personal care products [28].

5. Distribution of Microplastics in the Human Body

5.1. Blood, Tissues, and Organs

One of the most significant discoveries in recent years has been the documentation of MPs in human blood. Leslie et al. (2022), in a study involving healthy adult volunteers in the Netherlands, detected MPs in 77% of 22 blood samples analyzed, with an average concentration of 1.6 µg/mL [5]. The identified polymers consisted primarily of polyethylene terephthalate (PET), polystyrene (PS), and polyethylene (PE). These findings raised important questions regarding the potential for hematogenous transport of MPs to internal organs.

MPs have also been detected in the human liver, spleen, lymphatic tissue, and intestinal contents [4]. In 2021, Ragusa and colleagues made a landmark discovery by demonstrating the presence of MPs in human placental tissues. Their study, referred to as the "Plasticenta" study, identified MPs on both the maternal and fetal sides of the placenta [7]. Subsequent investigations confirmed and expanded these findings, detecting MPs in neonatal meconium, infant stool, breast milk, and infant formula products [8]. Zhao et al. (2023) further confirmed the presence of MPs in human testes and semen, raising concerns regarding potential effects on male fertility [29].

5.2. Placenta and Prenatal Exposure

The detection of MPs on the fetal side of the placenta implies the possibility of prenatal exposure to microplastics. Several mechanisms support this hypothesis: sufficiently small MP particles may cross the placental barrier, while nanoplastics have demonstrated such capability even in vitro using placental perfusion models [7]. Liu et al. (2023) [8] conducted a prospective pilot study in which various MP types were detected in samples obtained from mother-infant pairs, including placenta, meconium, breast milk, and infant feces collected at six months of age. These findings suggest continuous exposure beginning during fetal development and extending into infancy.

Particular concern arises from the fact that fetuses and newborns may be especially susceptible to the toxic effects of MPs because of the immaturity of detoxification systems and biological barriers during early development.

5.3. Respiratory System

Microplastics inhaled from the air may be deposited throughout the respiratory tract. Their presence has been documented in lung tissue, bronchoalveolar lavage fluid (BAL), and bronchial mucosa [25–27]. The study by Jenner et al. (2022) detected MPs in every one of the 11 lung regions examined among 11 of 13 patients undergoing surgical procedures [26]. The most frequently identified polymers were polypropylene (PP), polyethylene terephthalate (PET), and polyamide-materials commonly used in textiles and packaging.

The detection of MPs in the lower respiratory tract, where mechanical filtration mechanisms were previously believed to effectively prevent particle deposition, suggests that smaller airborne plastic particles may penetrate deeper into the lungs than previously assumed.

6. Mechanisms of Microplastic Toxicity

6.1. Oxidative Stress and DNA Damage

Oxidative stress is among the best-documented mechanisms underlying MP-induced cytotoxicity. Exposure to MPs can lead to excessive production of reactive oxygen species (ROS), which damage membrane lipids, enzymatic proteins, and nucleic acids [30]. Polypropylene, polystyrene, polyethylene, and plastic-derived phthalates have all been shown to induce elevated ROS production both in vitro in cell cultures and in vivo in animal models [30].

The resulting DNA damage-including double-strand breaks and oxidative base modifications-may contribute to mutations, apoptosis, and carcinogenesis. Chen et al. demonstrated that exposure to polypropylene MPs increased biomarkers of oxidative stress, including malondialdehyde (MDA) and 8-hydroxy-2′-deoxyguanosine (8-OHdG), while simultaneously decreasing the activity of key antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) in liver cells [30].

6.2. Inflammatory Response

Microplastics activate cellular immune mechanisms, triggering both local and systemic inflammatory responses. Macrophages and dendritic cells recognize MPs as foreign bodies and release pro-inflammatory cytokines-including tumor necrosis factor alpha (TNF-α), interleukin-1 beta (IL-1β), interleukin-6 (IL-6), and interleukin-8 (IL-8)-thereby amplifying inflammatory signaling cascades [31].

Chronic exposure to MPs may promote persistent low-grade inflammation, a well-established risk factor for cardiovascular disease, type 2 diabetes, cancer, and neurodegenerative disorders [31]. MPs can be phagocytosed by macrophages; however, due to their non-biodegradable nature, they may form plastic granulomas that resist enzymatic degradation and sustain chronic inflammatory processes.

6.3. Effects on the Gut Microbiome

The gut microbiome-a complex ecosystem consisting of trillions of microorganisms inhabiting the gastrointestinal tract-appears to be particularly sensitive to MP exposure. Animal studies have demonstrated that MPs alter both the composition and diversity of the intestinal microbiota, reducing populations of beneficial genera such as *Lactobacillus*and *Bifidobacterium* while increasing the abundance of potentially pathogenic bacteria, including *Escherichia coli* and *Clostridium* species [32].

MPs may also physically irritate the intestinal epithelium, causing microinjuries that increase intestinal permeability [32]. Damage to the gut barrier facilitates the translocation of bacterial endotoxins, particularly lipopolysaccharides (LPS), into the bloodstream, thereby initiating systemic inflammatory responses.

MP-induced dysbiosis may additionally disrupt the metabolism of short-chain fatty acids (SCFAs), secondary bile acids, and neurotransmitters, potentially affecting the gut-brain axis. A systematic review by Ghosh et al. (2024) demonstrated that MPs alter the gut microbiota composition of mice in a manner dependent on both exposure dose and polymer type, with polystyrene producing the most pronounced effects [32]. Patients with inflammatory bowel disease (IBD) have been found to exhibit higher concentrations of MPs in intestinal tissues than healthy individuals, suggesting a possible relationship between MP exposure and disease exacerbation [32].

6.4. Endocrine Effects

Microplastics, as well as chemical substances contained within or adsorbed onto them, possess the capacity to disrupt hormonal homeostasis [33]. Among the compounds released from plastics, particular concern surrounds:

- **Bisphenol A (BPA)**, which mimics endogenous estrogens and interferes with the hypothalamic-pituitary-gonadal axis;
- **Phthalates** such as DEHP and DBP, which disrupt steroidogenesis and affect reproductive system development;
- **Alkylphenols** and **polybrominated diphenyl ethers (PBDEs)**, which exhibit endocrine-disrupting properties.

Epidemiological studies have demonstrated associations between urinary concentrations of BPA and phthalates and impaired fertility, earlier pubertal onset, obesity, diabetes, and neurodevelopmental disorders [33]. Importantly, endocrine disruption may not be limited to adsorbed contaminants alone; certain polymers themselves may exhibit hormonal activity. For example, polystyrene has been reported to possess weak estrogen-like properties.

6.5. Physical Effects: Particle Toxicity

In addition to their chemical effects, MPs may exert direct physical toxicity arising from their particulate nature. Similar to other non-organic particles such as silica, asbestos, and coal dust, MPs can mechanically damage cell membranes, impair the function of pulmonary macrophages, and induce granulomatous reactions.

Microplastic fibers are particularly problematic because their elongated morphology makes them difficult for macrophages to phagocytose effectively. This promotes accumulation and persistent inflammatory responses resembling the pathological processes associated with asbestos exposure [26]. Occupational exposure to polypropylene and polyethylene dust has long been linked to an increased risk of respiratory diseases among workers in the plastics industry [26].

7. Impact of Microplastics on Human Health: Clinical and Epidemiological Evidence

7.1. Cardiovascular System

The most significant clinical report in recent years concerning the health effects of microplastic (MP) exposure is the study by Marfella et al., published in *The New England Journal of Medicine* in March 2024 [9]. In this study, atherosclerotic plaque tissue obtained from 257 patients undergoing carotid endarterectomy was analyzed. Microplastics and nanoplastics (MNPs) were detected in 58.4% of the samples using pyrolysis gas chromatography-mass spectrometry (Py-GC/MS). Patients whose plaques contained MNPs exhibited a 4.53-fold higher risk of a composite endpoint (myocardial infarction, stroke, or death) during a median follow-up period of 34 months. Importantly, tissues containing MNPs showed elevated concentrations of inflammatory biomarkers, including IL-18, IL-1β, and C-reactive protein (CRP), suggesting that MNPs may enhance inflammatory activity within atherosclerotic plaques and accelerate plaque destabilization [9].

The findings of Marfella et al. generated considerable interest but also methodological debate, as some researchers highlighted the possibility of sample contamination during surgical procedures. Nevertheless, this study represents the first prospective clinical evidence linking the presence of microplastics in cardiovascular tissue with an increased risk of adverse cardiovascular events. Yang et al. (2023) also detected microplastics in cardiac tissues collected from patients undergoing cardiac surgery, identifying 15 different polymer types [9, 34]. In vitro and animal studies provide plausible mechanistic explanations for these observations: microplastics induce endothelial dysfunction, increase oxidative stress in cardiomyocytes, impair vascular integrity, and promote a pro-thrombotic platelet phenotype [34].

7.2. Respiratory System

Inhalation of microplastics is recognized as an important route of occupational exposure, particularly in the plastics-processing and textile industries. Workers exposed to polypropylene dust exhibit higher rates of asthma, chronic obstructive pulmonary disease (COPD), and pulmonary fibrosis than the general population [35]. The phenomenon known as *flock worker's lung*-an interstitial lung disease affecting workers involved in the manufacture of nylon-based carpeting and textiles-is an established occupational pathology in which inhaled synthetic microfibers play a central etiological role [35].

Within the general population, evidence regarding the respiratory effects of inhaled microplastics remains limited. Observational studies have reported that children living near artificial turf facilities and playgrounds are exposed to higher concentrations of airborne rubber microgranules in the nasopharyngeal region and display a greater prevalence of allergic symptoms [35]. Microplastics may also act as carriers for allergens and fungal spores, thereby exacerbating allergic respiratory reactions. Experimental studies have demonstrated that microplastics can activate mast cells and eosinophils, enhancing the inflammatory response characteristic of asthma [35]. Many questions concerning the long-term health consequences of inhalational exposure in the general population remain unanswered and require large-scale cohort studies with extended follow-up periods.

7.3. Gastrointestinal Tract and Liver

Following ingestion, microplastics enter the gastrointestinal tract, where they may exert both local and systemic effects. Animal studies have demonstrated that microplastics can damage the intestinal epithelium, induce mucosal inflammation, increase intestinal permeability, and activate intraepithelial lymphocytes [32]. In mice, polypropylene microplastics caused inflammatory changes in the gastric and small intestinal mucosa and, following prolonged exposure, resulted in micro-ulcerations and microscopic hemorrhages [32].

The liver, as the primary organ responsible for detoxification, may be particularly susceptible to the adverse effects of microplastics and the chemicals they transport. Yan et al. (2022) detected microplastics in liver tissue obtained from patients with cirrhosis, with concentrations exceeding those observed in healthy liver tissue [36]. Phthalates and bisphenol A (BPA) released from microplastics disrupt hepatic metabolic pathways, promoting hepatic steatosis and oxidative stress in hepatocytes. Administration of large polyethylene microplastics to mice resulted in hepatic lipidosis and disturbances in lipid homeostasis [36].

7.4. Reproductive System and Fertility

Experimental evidence suggests that microplastic exposure may have significant adverse effects on the reproductive systems of both males and females. In male animals exposed to polystyrene microplastics (PS-MPs), researchers observed reduced sperm motility and viability, morphological abnormalities of the sperm head and tail, decreased testosterone concentrations, testicular atrophy, and impaired spermatogenesis [29]. Proposed mechanisms include oxidative stress within testicular tissue, apoptosis of spermatocytes, disruption of the blood–testis barrier, and dysregulation of the hypothalamic-pituitary-gonadal axis [29]. Zhao et al. (2023) were the first to identify microplastics directly in human semen, detecting them in 34 of 45 participants. Higher microplastic concentrations were associated with lower sperm motility and viability [29].

In females exposed to microplastics, studies have reported disturbances of the ovulatory cycle, ovarian follicle dysfunction resulting from granulosa cell apoptosis, reduced fertility, impaired embryo implantation, and increased embryonic mortality [37]. Hu et al. (2021) demonstrated that polyethylene microplastics disrupted maternal-fetal immune balance in pregnant mice, increasing the risk of fetal resorption [37]. BPA and phthalates released from microplastics can mimic endogenous estrogens, interfering with normal oocyte maturation and endometrial cell proliferation [33, 37]. Particular concern surrounds the possibility that the effects of microplastic exposure may be transmitted across generations through epigenetic mechanisms, resulting in transgenerational toxicity [37].

7.5. Nervous System

Current evidence regarding the neurotoxicity of microplastics is derived primarily from experimental studies; however, the findings raise significant concerns. Nanoplastics have been shown to cross the blood-brain barrier, as demonstrated in both murine and fish models [38]. Exposure to polystyrene microplastics and nanoplastics has been associated in rodents with behavioral abnormalities, cognitive deficits, anxiety-like behavior, and depressive symptoms. These changes were accompanied by neurochemical alterations, including reduced serotonin and dopamine levels, activation of microglial cells, and increased biomarkers of oxidative stress within brain tissue [38]. Wang et al. (2024) reported that exposure to PS-MPs accelerated the progression of Alzheimer-like pathology in mice, suggesting a potential association between microplastics and neurodegenerative disease processes [38].

Particular concern surrounds prenatal and early-life exposure, as the developing brain is substantially more vulnerable to neurotoxic agents than the mature adult brain. Studies using *Caenorhabditis elegans* and zebrafish models have demonstrated transgenerational neurotoxicity associated with polystyrene exposure, with offspring of exposed parents exhibiting behavioral and neurochemical abnormalities across successive generations [38]. Proposed mechanisms of microplastic-induced neurotoxicity include oxidative stress in neurons, disruption of neurotransmitter synthesis and transport, activation of neuroinflammatory pathways, and accumulation of aggregated proteins such as α-synuclein, which is implicated in the pathogenesis of Parkinson's disease [38].

7.6. Effects on Children and Other Vulnerable Populations

Children experience proportionally greater exposure to microplastics than adults due to several factors, including a higher respiratory minute volume relative to body weight, frequent hand-to-mouth behaviors, consumption of food and beverages from plastic packaging and bottles, participation in activities on artificial turf surfaces, and prolonged contact with floor-level environments where concentrations of dust and microplastics are generally higher [39]. Sripada et al. (2022) conducted a comprehensive assessment of children's exposure to nano- and microplastics and estimated that children aged 3-6 years may ingest up to 3,000 microplastic particles per day through dust ingestion alone [39].

Developing physiological systems - including the nervous, endocrine, immune, and reproductive systems - are particularly susceptible to disruption by endocrine-disrupting chemicals released from plastics. An important area of ongoing research concerns the impact of prenatal microplastic exposure on neurodevelopment, physical growth, and the long-term metabolic health of children.

Table 1

Summary of key human studies reporting the detection of microplastics (MP) in biological matrices

Author (Year)	Country	n	Biological Matrix	Analytical Method	Key Finding	Ref.
Leslie et al. (2022)	Netherlands	22 healthy adults	Peripheral blood	micro-FTIR, Py-GC/MS	MP detected in 77% of samples; mean 1.6 µg/mL; PET, PS and PE most common	**[5]**
Ragusa et al. (2021)	Italy	6 term placentas	Human placenta (maternal & foetal side)	micro-Raman spectroscopy	MP confirmed in all 6 placentas; 12 particles identified; PP and PE/PC polymers	**[7]**
Jenner et al. (2022)	United Kingdom	13 surgical patients	Lung tissue (11 regions per donor)	micro-FTIR	MP detected in 11/13 patients across all lung regions; PP, PET and PA dominant	**[26]**
Amato-Lourenço et al. (2021)	Brazil	20 autopsy cases	Lung parenchyma	micro-Raman spectroscopy	MP found in 13/20 samples; PE and PP < 5.5 µm; higher load in urban residents	**[25]**

Liu et al. (2023)	China	17 mother-infant pairs	Placenta, meconium, breast milk, infant faeces	micro-FTIR, Py-GC/MS	MP detected in all four matrices; PE, PP and PS most prevalent; transfer confirmed	**[8]**
Zhao et al. (2023)	China	45 male volunteers	Testicular tissue & seminal fluid	micro-FTIR	MP in 34/45 semen samples; higher MP load correlated with lower sperm motility	**[29]**
Yang et al. (2023)	China	15 cardiac surgery patients	Cardiac tissue (atrium, pericardium)	micro-FTIR	15 polymer types identified; PP, PE, PVC detected; mean 8.52 particles/g tissue	**[34]**
Marfella et al. (2024)	Italy	257 endarterectomy patients	Carotid atherosclerotic plaque	Py-GC/MS + TEM/EDX	MNP in 58.4% of plaques; 4.53× higher risk of MACE over 34 months follow-up	**[9]**
Yan et al. (2022)	China	50 IBD patients, 50 controls	Colonic mucosal tissue & faeces	micro-FTIR	Higher MP concentration in IBD vs. controls; MP load correlated with disease severity	**[36]**

Huang et al. (2022)	China	28 patients (respiratory disease)	Sputum	micro-FTIR	Up to 565 MP particles/10 mL sputum; 21 polymer types; fibres predominant	**[27]**

Note. MP = microplastics; MNP = micro- and nanoplastics; MACE = major adverse cardiovascular events; IBD = inflammatory bowel disease; PE = polyethylene; PP = polypropylene; PS = polystyrene; PET = polyethylene terephthalate; PA = polyamide; PVC = polyvinyl chloride; PC = polycarbonate; micro-FTIR = micro-Fourier transform infrared spectroscopy; Py-GC/MS = pyrolysis gas chromatography-mass spectrometry; TEM/EDX = transmission electron microscopy with energy-dispersive X-ray spectroscopy. References in brackets correspond to the bibliography of the main review.

8. Methods for the Identification and Quantification of Microplastics in Biological Samples

Standardization of methods used to detect microplastics in tissues and biological fluids represents one of the major scientific challenges in this field. The absence of harmonized protocols makes comparison between studies difficult. The principal analytical techniques currently employed include:

Raman Spectroscopy and Fourier-Transform Infrared Spectroscopy (FTIR)

These non-destructive techniques enable the identification of polymer chemical composition without destroying the sample. Micro-FTIR can identify particles as small as 10–20 µm, while micro-Raman spectroscopy can detect particles smaller than 1 µm [40]. These methods are considered the gold standard for microplastic analysis in biological tissues.

Pyrolysis Gas Chromatography-Mass Spectrometry (Py-GC/MS)

Py-GC/MS is a destructive analytical technique that allows precise determination of the polymer composition of a sample. It was employed by Marfella et al. in their analysis of atherosclerotic plaques [9]. The method provides quantitative mass-based measurements but destroys the sample and does not yield information regarding particle size or morphology.

Scanning Electron Microscopy with Energy-Dispersive X-Ray Spectroscopy (SEM-EDX)

SEM-EDX enables high-resolution visualization of particle morphology and elemental composition. However, it cannot directly identify polymer type.

Fluorescence Microscopy and Lipophilic Dyes (e.g., Nile Red)

This approach represents a less expensive alternative that allows rapid screening of samples. However, it is less specific than spectroscopic techniques and is more susceptible to analytical artifacts.

A major methodological challenge in all studies investigating microplastics in biological samples is the risk of contamination from laboratory materials, clothing, and airborne particles. Without rigorous blank controls and contamination-prevention procedures, results may be artificially inflated. Many researchers have therefore emphasized the urgent need for improved methodological quality and standardization of sampling and analytical protocols [40].

9. Legal Regulations, Treatment Technologies, and Exposure-Reduction Strategies

9.1. Regulatory Frameworks

Regulation of microplastics remains relatively underdeveloped and fragmented worldwide. In 2023, the European Chemicals Agency (ECHA) introduced restrictions on intentionally added microplastics, limiting the use of microbeads in cosmetics, detergents, fertilizers, and lawn-care products. These measures are estimated to prevent the release of approximately 500,000 tonnes of microplastics into the environment over a 20-year period [41].

At the international level, the United Nations is currently negotiating a legally binding global treaty on plastic pollution, which could provide a comprehensive framework for reducing microplastic emissions worldwide [41].

In the United States, the *Microbead-Free Waters Act of 2015* prohibited the use of plastic microbeads in rinse-off cosmetic products. Many states have additionally implemented regulations targeting single-use plastic bags and packaging. However, no federal standards currently exist for allowable concentrations of microplastics in drinking water, and the U.S. Environmental Protection Agency (EPA) has yet to establish binding regulatory limits [41].

9.2. Water and Wastewater Treatment Technologies

Municipal wastewater treatment plants remove between 70% and 99% of microplastics from wastewater. However, given the enormous volumes of water processed, even these high removal efficiencies still result in billions of microplastic particles being discharged daily into receiving water bodies from a single facility [42]. Treatment sludge often contains high concentrations of microplastics, and its agricultural application as fertilizer represents a common pathway for secondary soil contamination.

Promising technologies for improving microplastic removal include membrane ultrafiltration, coagulation–flocculation processes, advanced oxidation processes (AOPs) such as ozonation, and membrane bioreactors [42].

Drinking water treatment facilities also remove microplastics with varying effectiveness depending on the technologies employed. Coagulation and sedimentation can eliminate up to 80% of particles, sand filtration approximately 40%, and membrane ultrafiltration more than 95% [42]. The consistently higher concentrations of microplastics reported in bottled water compared with tap water suggest that treated municipal water may represent a lower-exposure option than bottled water.

9.3. Individual-Level Exposure Reduction Strategies

At the individual level, exposure to microplastics can be reduced through changes in consumer behavior. Recommended measures include:

- Avoiding the heating of food in plastic containers.
- Replacing plastic bottles with glass or stainless-steel alternatives.
- Reducing consumption of heavily processed foods packaged in plastic.
- Using water filtration systems based on activated carbon or reverse osmosis.
- Choosing natural, non-synthetic textiles and fabrics.
- Regularly ventilating indoor spaces and vacuuming to reduce dust-associated microplastics [19, 41].

The use of laundry filters designed to capture synthetic microfibers (e.g., Guppy Friend-type filtration systems) may reduce microfiber emissions into wastewater by more than 86% during washing [41].

Although diets rich in fruits and vegetables may inadvertently increase exposure to microplastics originating from contaminated soils, thermally processed vegetables generally contain lower concentrations of microplastics than raw produce [19].

10. Knowledge Gaps and Future Research Directions

Despite the rapid growth of microplastic research, many fundamental questions remain unresolved. Key priorities for future investigation include:

10.1. Biomonitoring and Exposure Assessment

Large-scale, representative epidemiological studies are needed to quantify microplastic concentrations in human tissues and biological fluids across different populations, geographical regions, and lifestyles. Standardization of analytical methodologies is essential for meaningful interlaboratory comparisons [40].

10.2. Cohort and Prospective Studies

Current clinical evidence regarding the health effects of microplastics is largely correlational or cross-sectional. Long-term prospective cohort studies incorporating accurate exposure assessment and hard clinical endpoints - including chronic diseases, mortality, fertility outcomes, and pregnancy outcomes - are urgently required [9, 34].

10.3. Nanoplastic Toxicology

Nanoplastics (<1 μm) are considerably more difficult to detect and characterize than larger microplastics. Their biological effects may be more pronounced due to their ability to cross biological barriers, including the blood-brain barrier, intestinal barrier, and placenta. Improved analytical methods and systematic toxicological investigations are therefore needed [38].

10.4. Pregnancy Exposure and Neurodevelopmental Outcomes

The identification of microplastics in the placenta, meconium, and breast milk highlights the urgent need to investigate prenatal and early-life exposure. Particular attention should be directed toward understanding the long-term consequences for neurodevelopment, endocrine function, growth, and metabolic health in children [39].

10.5. Mixture Effects

Humans are simultaneously exposed to hundreds of different microplastic types and thousands of associated chemical compounds, including plasticizers, stabilizers, and dyes. Interactions among different microplastics, as well as between microplastics and other environmental contaminants such as heavy metals, pesticides, and pharmaceuticals, may produce synergistic effects that cannot be predicted from studies examining individual substances alone [4].

10.6. Biodegradation and Environmental Remediation

The development of effective and environmentally safe methods for the degradation and removal of microplastics remains an active area of research. Promising approaches include the use of plastic - degrading microorganisms and photocatalytic degradation technologies [42].

10.7. Regulation and Public Policy

Translating scientific evidence into effective regulatory policy requires close collaboration among researchers, policymakers, industry stakeholders, and the public. Evidence-based standards for permissible concentrations of microplastics in drinking water, food, and occupational environments are urgently needed and should be grounded in robust toxicological and epidemiological data [41].

11. Conclusion

Microplastics represent one of the most pervasive and persistent environmental pollutants in human history. Over the past two decades, scientific research has made remarkable progress in documenting their environmental distribution and human exposure, leading to the conclusion that virtually no individual on Earth - regardless of age, geographic location, or lifestyle - is completely free from exposure to microplastics.

Current scientific evidence identifies several mechanisms through which microplastics may adversely affect human health, including oxidative stress and DNA damage, chronic inflammation, intestinal dysbiosis, endocrine disruption, reproductive toxicity, and potential neurotoxicity. The landmark 2024 study published in *The New England Journal of Medicine* [9] provided the first prospective clinical evidence linking the presence of microplastics in atherosclerotic plaques with an increased risk of cardiovascular events, opening a new chapter in research on the health significance of microplastic exposure.

At the same time, caution is warranted in interpreting the available evidence. Much of the current knowledge is derived from experimental studies employing microplastic concentrations substantially higher than those typically detected in human tissues. Large, representative epidemiological studies with long-term follow-up are still lacking. Standardized analytical methods and population-level biomonitoring programs are urgently needed to clarify the true extent of human exposure and its health implications.

Addressing the challenge of microplastic pollution will require coordinated action at multiple levels - from reducing plastic production and emissions at the source, to developing innovative water and wastewater treatment technologies, to implementing regulatory measures that limit the use of single-use plastics and non-recyclable materials. International cooperation is essential, as studies conducted in the Arctic, Antarctic, and deep-ocean environments clearly demonstrate that microplastic pollution transcends national boundaries.

In many respects, the current situation resembles the early stages of research on tobacco smoke and lead exposure - hazards that were initially underestimated but later recognized as major public health threats. The challenge now is to act preventively before incontrovertible evidence of harm accumulates, while simultaneously pursuing the highest standards of scientific rigor in generating that evidence

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