Proceedings of ICBioMed 2025 Symposium: AI for Healthcare: Advanced Medical Data Analytics and Smart Rehabilitation
DOI: 10.54254/2753-8818/2025.AU28680

Microplastic Exposure and Human Health: Advancing Risk Assessment and Future Research Directions

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Abstract. Microplastics (MPs), defined as plastic fragments smaller than 5 mm, have become ubiquitous environmental contaminants. Their pervasive presence across terrestrial, aquatic, and atmospheric systems has led to increasing human exposure. Recent evidence indicates MPs are present in human blood, respiratory tissues, placental structures, and gastrointestinal waste, raising significant concerns about their potential toxicological implications. This paper synthesizes recent advances in microplastic toxicology, focusing on human exposure pathways, tissue distribution, experimental toxicological evidence, and critical knowledge gaps. It aims to provide a coherent risk assessment framework for understanding the health consequences of microplastic exposure, highlighting the imperative for standardized methodologies and long-term epidemiological studies.

Keywords: Microplastics, Human Health, Exposure Pathways, Toxicology, Risk Assessment

1. Introduction

Plastics, celebrated for their durability, cost-effectiveness, and versatility, have become indispensable across numerous industries. However, the exponential growth in global plastic production and consumption has resulted in an escalating environmental crisis, primarily manifested by the widespread presence of microplastics (MPs) [1]. These microscopic particles, typically defined as fragments smaller than 5 mm, are now pervasive contaminants found in terrestrial ecosystems, aquatic environments, and atmospheric systems globally [2].

The past decade has seen an alarming accumulation of evidence documenting the presence of MPs not only in environmental matrices but also within human biological systems. MPs have been detected in critical human tissues, including blood circulation, respiratory tissues, placental structures, and gastrointestinal waste, prompting profound concerns about their potential toxicological implications for human health [3, 4]. Such discoveries have catalyzed urgent scientific inquiry into the pathways through which these particles enter biological systems and the mechanisms by which they may compromise physiological function.

This report systematically evaluates the routes, mechanisms, and health consequences associated with microplastic exposure in human populations. Mounting evidence from both in vivo and in vitro experimental studies reveals concerning signs of cellular toxicity, inflammatory responses, and organ dysfunction [5]. Therefore, synthesizing these findings within a coherent risk assessment framework is imperative. The primary objective of this analysis is to consolidate recent advances in

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microplastic toxicology research, with particular emphasis on human exposure pathways, tissue distribution patterns, experimental toxicological evidence, and the significant knowledge gaps that currently impede accurate health risk characterization.

2. Background: sources, persistence, and environmental distribution of microplastics

Microplastic contamination originates from two distinct sources. Primary microplastics are intentionally manufactured particles, such as microbeads in personal care products. Secondary microplastics arise from the degradation and fragmentation of larger plastic debris due to environmental weathering processes like ultraviolet radiation, mechanical abrasion, and thermal cycling [1].

The inherent chemical stability and resistance to biological decomposition that make plastics commercially valuable also enable MPs to persist in environmental systems for extended periods, potentially spanning decades or centuries. Their diminutive size facilitates easy ingestion or inhalation by living organisms across taxonomic groups. Furthermore, their surface properties allow them to act as vectors for the transport and bioaccumulation of heavy metals, persistent organic pollutants, and pathogenic microorganisms [6].

Comprehensive environmental monitoring initiatives have demonstrated the pervasive distribution of MPs across diverse ecosystems, including oceanic waters, freshwater systems, terrestrial soils, atmospheric compartments, and complex food webs. Aquatic organisms, from commercially important fish species to crustaceans and mollusks, readily ingest these particles, leading to bioaccumulation and biomagnification processes that can ultimately transfer concentrated MP loads to human consumers [7]. More alarmingly, recent human biomonitoring studies have confirmed the presence of MP particles in various biological matrices, including circulating blood, placental tissues, pulmonary structures, and fecal matter, providing unequivocal confirmation of widespread human exposure [3, 4].

Despite these significant discoveries, scientific understanding of the health risks posed by MP exposure remains in its nascent stages. Most available toxicological data derive from controlled laboratory studies using cell culture systems or rodent models to infer potential toxic mechanisms relevant to human health. Given the substantial limitations in current exposure assessment methodologies, the absence of established dosage threshold values, and the lack of comprehensive long-term epidemiological monitoring programs, existing risk assessment strategies remain fundamentally incomplete and inadequate for regulatory decision-making [1, 5].

3. Human exposure pathways and tissue translocation

Humans encounter MP contamination primarily through three key routes: ingestion, inhalation, and dermal contact [8].

3.1. Ingestion

Ingestion is considered the primary exposure pathway, with MPs ubiquitous in human diet components. Particles contaminate various water sources, including bottled and tap water. Seafood harbors significant MP loads from marine organism ingestion, while crops can accumulate particles through contaminated irrigation and atmospheric deposition. Food packaging also contributes to exposure via particle migration, especially under thermal or mechanical stress. Indoor dust

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represents a major source of unintentional ingestion, with estimates suggesting thousands of MP particles consumed annually [8, 9].

3.2. Inhalation

Inhalation occurs through airborne MPs, with indoor concentrations typically exceeding outdoor levels due to synthetic materials. Polyester fibers often dominate indoor airborne MPs, with concentrations varying by ventilation, occupancy, and cleaning. Assessments indicate that humans can inhale tens of thousands of MP particles daily, with variations depending on location, urbanization, and industrial activity [10, 11]. Particles smaller than 10 μm are generally retained in the upper respiratory tract, while smaller particles can penetrate deeper into the lungs [12].

3.3. Dermal contact

Dermal contact is a potentially significant but under-researched pathway. MPs in personal care products, synthetic textiles, and medical devices may interact with the skin. Nanoplastics (<100 nm) can potentially penetrate intact skin via follicles or defects in the stratum corneum, while larger particles tend to remain surface-localized. Concerns also focus on MP release from contact lenses, masks, and synthetic clothing [8, 13]. Studies have shown that toxic chemicals from MPs can be absorbed through the skin [14].

3.4. Tissue translocation and vulnerable populations

MPs can translocate to diverse tissues and organs via mechanisms dependent on particle size, charge, and composition. Nanoplastics, in particular, can cross epithelial barriers through endocytic pathways, accumulating in the liver, neural, and adipose tissues. For instance, 20 nm polystyrene particles have been shown to cross the blood-brain barrier, preferentially accumulating in hippocampal regions associated with learning and memory [4]. This neurotoxicity is a growing concern, as MPs have been linked to neuroinflammation and Parkinson's-like behavioral changes in animal models [4].

Bioaccumulation concerns are heightened in vulnerable populations, notably infants and pregnant women, who may have enhanced susceptibility to MP toxicity. Pediatric populations exhibit elevated fecal MP concentrations compared to adults, likely due to hand-to-mouth behaviors and incomplete barrier development. Documented placental MP transfer suggests prenatal exposure during fetal development, necessitating attention to developmental toxicity and long-term offspring consequences [4, 15]. Studies have confirmed the presence of MPs in human placenta, meconium, and breast milk, indicating potential early-life exposure and intergenerational transfer [2, 15].

4. Toxicity evidence: in vitro and in vivo studies

Experimental studies have provided substantial evidence regarding the toxic effects of MPs. In vitro studies using human cell lines (e.g., THP-1 macrophages, HeLa, respiratory epithelium) consistently demonstrate MP-induced oxidative stress through the generation of reactive oxygen species (ROS), which can overwhelm antioxidant capacity [5, 16]. MPs can damage cellular membranes, mitochondria, and lysosomes, compromising energy metabolism and waste processing. Inflammatory responses include the upregulation of pro-inflammatory cytokines (e.g., TNF-α, IL-6), indicating innate immune activation [5, 17]. Genotoxic effects, such as DNA breaks and chromosomal aberrations, have also been observed, raising mutagenic and carcinogenic concerns [5,

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18]. Smaller plastic particles are generally more cytotoxic and penetrate cells more easily, leading to increased ROS formation [19].

In vivo rodent studies offer insights into system-level toxicity. Digestive effects include intestinal inflammation, barrier dysfunction, microbiota disruption, and hepatic lipid disorders, potentially predisposing to metabolic syndrome [5]. Nervous system impacts show blood-brain barrier crossing, neuroinflammation, and Parkinson's-like behavioral changes [4]. Respiratory exposure can lead to pulmonary deposition, fibrotic changes, and alveolar damage, compromising gas exchange [10]. Male reproductive effects include decreased sperm quality, hormonal alterations, and testicular inflammation, impairing fertility [5].

However, critical limitations exist due to discrepancies between laboratory and environmentally relevant exposure concentrations, highlighting the need for more realistic experimental designs [1, 5].

5. Risk assessment and research gaps

Comprehensive MP risk assessment faces substantial methodological and knowledge limitations impeding accurate health risk characterization. Absence of standardized exposure models creates study inconsistencies with varying particle types, sizes, and dosages complicating comparative analysis.

Insufficient human-specific data represents critical limitations, as most toxicological evidence derives from animal/cell models potentially not reflecting human responses. Longitudinal epidemiological studies establishing causal MP exposure-health outcome relationships remain rare, limiting regulatory decision-making basis.

Missing established adverse effect threshold values represent fundamental assessment gaps, as minimum toxic MP doses remain unknown across exposure scenarios and populations. Complex combined toxicity from MPs as co-contaminant vectors (heavy metals, pharmaceuticals, antibiotic-resistant bacteria) substantially complicates evaluation.

Addressing limitations requires urgent research priorities: establishing physiologically relevant exposure scenarios, standardizing experimental protocols, and implementing comprehensive biomonitoring programs tracking population exposure trends and health outcomes.

Primary obstacles included overwhelming research heterogeneity in particle compositions, sizes, experimental models, and endpoints, making synthesis into coherent health risk perspectives exceptionally challenging. Limited human-specific toxicological data severely restricted meaningful quantitative risk assessment and regulatory threshold development.

Under advisor guidance, I developed effective strategies organizing findings by exposure routes and target organs, enabling structured comparative analysis. We prioritized identifying and discussing knowledge gaps rather than overstating limited evidence conclusions, maintaining scientific rigor. Initial literature retrieval/synthesis difficulties improved through strategic advanced database utilization (PubMed filtering, Scopus analytics), significantly enhancing research efficiency and comprehensiveness.

6. Conclusion

The pervasive presence of microplastics in the environment and their increasing detection in human biological systems underscore an urgent need for comprehensive risk assessment. While significant progress has been made in identifying exposure pathways and toxicological effects through in vitro and in vivo studies, substantial knowledge gaps remain. Future research must prioritize the

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development of standardized methodologies, physiologically relevant exposure models, and long-term epidemiological studies to accurately characterize the health risks of microplastic exposure. This interdisciplinary effort is crucial for informing public health policies and safeguarding human and environmental well-being.

Moving forward, future research should delve deeper into the long-term health consequences of chronic low-dose microplastic exposure, particularly in vulnerable populations such as children and pregnant women, by establishing robust epidemiological cohorts. Furthermore, a critical area for investigation involves elucidating the precise molecular mechanisms by which microplastics interact with biological systems at the cellular and subcellular levels, utilizing advanced multi-omics approaches to identify novel biomarkers of exposure and effect. This will enable a more accurate understanding of dose-response relationships and the development of targeted interventions to mitigate adverse health outcomes. Finally, research into innovative solutions for microplastic remediation and prevention, alongside comprehensive policy frameworks, will be essential to address this escalating global health challenge effectively.

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