

The Invisible Threat: Microplastic Biomonitoring, Systemic Translocation, and Human Health Risks

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Abstract- Microplastics are an emerging global contaminant with profound implications for human and environmental health. Defined as plastic particles 1 μm to 5 mm in size, and nanoplastics less than 1 μm (<1000 nm), these particles originate from primary manufacturing and secondary degradation of larger plastics. Their ubiquity in air, water, soil, food, and biological tissues has raised significant toxicological concern because microplastics are not inert. They act as physical particulates, chemical vectors for plasticizers, flame retardants, heavy metals, and as carriers for microbial pathogens and persistent organic pollutants. Upon inhalation, ingestion, or dermal contact, microplastics can induce oxidative stress, chronic inflammation, endocrine disruption, immune dysregulation, gut microbiome alteration, and potential genotoxicity. Evidence from in vitro, animal, and emerging human biomonitoring studies suggests associations with respiratory dysfunction, reproductive toxicity, developmental effects, and cardiovascular risk. This review consolidates existing evidence on microplastic sources, human exposure routes, mechanisms of toxicity, and associated health outcomes, while underscoring the critical need for standardized detection protocols, systematic human biomonitoring, development of safer polymers, and effective environmental remediation.

Keywords— Microplastics, Nanoplastics, Human health toxicity, Endocrine disruption, Oxidative stress, Environmental contamination, Biomonitoring

I. INTRODUCTION

Global plastic production has exceeded 400 million tons annually, with less than 10% effectively recycled, leaving the majority to fragment and persist in the environment for centuries [1,2]. Microplastics (MPs) are operationally defined as plastic particles ranging from 1 μm to 5mm, while nanoplastics (NPs) are less than 1 μm in size, with the fraction <100nm often considered nanoplastics in regulatory contexts. Their small size, high surface area to volume ratio, and environmental persistence allow them to contaminate virtually every environmental compartment [1, 2].

Unlike macroplastics, microplastics are bioavailable. They have been detected in drinking water, bottled water, table salt, seafood, fruits, vegetables, honey, indoor and outdoor air, and human tissues including placenta, lung, liver, blood, and stool [3, 4]. The toxicology of microplastics is complex because toxicity is not determined by the polymer alone, but by a triad of factors: (i) the physical particle itself, (ii)

the chemical additives leaching from it, and (iii) the environmental pollutants sorbed onto its surface [3, 4].

This makes microplastics a unique mixture toxicant. A particle inhaled from indoor air may consist of polyethylene terephthalate (PET) fiber containing antimony catalyst residue and adsorbed polycyclic aromatic hydrocarbons (PAHs). Similarly, a polystyrene fragment ingested via seafood may leach styrene monomers and carry polybrominated diphenyl ethers (PBDEs). Understanding this complexity is essential to assessing the true human health burden.

II. MAJOR SOURCES AND PHYSICOCHEMICAL CHARACTERISTICS

1. Primary vs Secondary Microplastics

Primary microplastics are intentionally manufactured in microscopic sizes. They include microbeads in personal care products, pre-production pellets (nurdles), industrial abrasives, and polymeric

nanoparticles used in drug delivery and textiles. Although regulatory bans on microbeads have reduced some sources, primary microplastics continue to enter wastewater systems [5]. Secondary microplastics result from fragmentation of larger plastics via UV radiation, mechanical abrasion, thermal degradation, and biological degradation. Major contributors include tire wear particles, synthetic textile fibers released during laundering, fragmentation of packaging films, fishing nets, and agricultural mulch films. Secondary particles are irregular in shape, more chemically weathered, and often carry higher loads of sorbed contaminants [6].

2. Polymer Types and Chemical Additives

Toxicological risk varies significantly by polymer type. The most common environmental microplastics are polyethylene (PE), polypropylene (PP), polystyrene (PS), polyethylene terephthalate (PET), polyvinyl chloride (PVC), and polyamide (PA) [7]. Plastics are never pure polymers. They contain chemical additives that may constitute up to 50% of plastic weight: plasticizers such as bisphenol A (BPA) and phthalates, flame retardants, UV stabilizers, pigments containing cadmium, lead, and chromium, and residual monomers like styrene and vinyl chloride. These additives are not covalently bound and can leach into biological systems, driving endocrine disruption and systemic toxicity. PVC and PS are of particular concern due to high additive content and inherent monomer toxicity [8].

3. Vector Effect and Biofilm Formation

Due to their hydrophobic nature, microplastics demonstrate a high sorption affinity for persistent organic pollutants (POPs); including polychlorinated biphenyls (PCBs), dichlorodiphenyltrichloroethane (DDT), and polycyclic aromatic hydrocarbons (PAHs), and can accumulate these contaminants from surrounding water at concentrations several orders of magnitude above ambient levels. Furthermore, microplastics acquire an ecocorona composed of proteins and a plastisphere consisting of biofilms that harbor pathogenic bacteria, antibiotic resistance genes, and viruses, thereby adding a biological hazard to their chemical toxicity [9][10].

III. EXPOSURE PATHWAYS

Human exposure to microplastics is chronic, low-dose, and occurs via three major routes: ingestion, inhalation, and dermal contact [11]. Ingestion is considered the dominant pathway. Cox et al. estimated ingestion of 39,000 to 52,000 particles per person per year, rising to 74,000-121,000 when inhalation is included [3]. Contaminated seafood, particularly bivalves and small fish consumed whole, drinking water (both tap and bottled), table salt, honey, beer, and plant-based foods contaminated via sewage sludge-amended soils are significant sources [12].

Inhalation exposure is increasingly recognized as critical, especially in indoor environments where synthetic textile fibers dominate airborne fallout. Indoor air concentrations can be 5-10 times higher than outdoor air. Particles <10 μm can deposit deep in the alveoli, while fibrous microplastics show persistence with biopersistence concerns. Occupational exposure among textile workers demonstrates respiratory irritation and lung function decline [13, 14].

Dermal contact is considered a lower-risk pathway for pristine microplastics due to low skin penetration, but nanoplastics <100 nm and plastic additives in cosmetics may potentially cross compromised skin barriers, particularly when skin is damaged [15]. A unique exposure scenario is maternal-fetal transfer. Studies have detected microplastics in human placenta, meconium, breast milk, and infant stool, suggesting prenatal and early-life exposure windows of heightened vulnerability [16,17].

IV. MECHANISMS OF TOXICITY

1. Oxidative Stress and Inflammation

The primary cellular response to particle internalization is generation of reactive oxygen species (ROS), mitochondrial dysfunction, and activation of nuclear factor kappa-light-chain-enhancer of activated B cells (NF- κ B) inflammatory signaling. Persistent particles resist clearance, leading to chronic inflammation, frustrated phagocytosis, and granuloma formation, a

mechanism analogous to particle toxicology of silica and asbestos [18].

2. Chemical Toxicity and Endocrine Disruption

Leached additives such as BPA, phthalates, and nonylphenol are established endocrine disrupting chemicals (EDCs). They interfere with estrogen, androgen, and thyroid hormone receptors, alter steroidogenesis, and impair reproductive hormone signaling [19].

3. Physical Damage and Gut Barrier Dysfunction

In the gastrointestinal tract, microplastics can cause abrasion of intestinal epithelium, increased gut permeability, dysbiosis, and altered mucus secretion [20].

4. Translocation and Systemic Distribution

Particles <10 µm, and especially nanoplastics <100 nm, can cross epithelial barriers via endocytosis and paracellular transport, entering circulation and accumulating in liver, spleen, kidney, brain, and reproductive organs. Polystyrene nanoplastics have been shown to cross the blood-brain barrier and blood-placenta barrier in animal models [21].

5. Carrier Effect

Microplastics alter the bioavailability of co-contaminants. Sorbed PAHs and PCBs may desorb in gut conditions, enhancing their uptake compared to dissolved-phase exposure alone [9].

V. POTENTIAL HUMAN HEALTH OUTCOMES

1. Respiratory Outcomes

Inhaled microfibers cause dose-dependent cytotoxicity in lung epithelial cells, oxidative stress, and pro-inflammatory cytokine release. Workers exposed to synthetic fibers show higher prevalence of interstitial changes and respiratory symptoms. Experimental inhalation produces chronic lung inflammation [22].

2. Gastrointestinal and Metabolic Effects

Animal feeding studies demonstrate microplastic-induced gut microbiome dysbiosis, characterized by reduced beneficial bacteria, increased inflammatory

taxa, colonic mucosal damage, and metabolic changes including insulin resistance and lipid metabolism alteration [20,23].

3. Reproductive and Developmental Toxicity

In rodent models, oral exposure to polystyrene microplastics is associated with reduced sperm count and motility, ovarian fibrosis, and altered sex hormones. Maternal exposure during pregnancy is associated with fetal growth restriction and placental dysfunction in animal models. Human placental studies suggest association between microplastic load and reduced birth weight, though causality requires confirmation [24,25,17].

4. Cardiovascular and Systemic Effects

A landmark human study by Marfella et al. (2024) detected microplastics and nanoplastics in atheromatous plaques of carotid arteries in 58% of patients. Presence was associated with a 4.53-fold increased risk of myocardial infarction, stroke, or death over 34 months follow-up (HR 4.53). This observational study does not establish causality but highlights potential cardiovascular risk through inflammatory mechanisms [26].

5. Carcinogenic Potential

While no direct classification exists for microplastics, chronic inflammation, ROS-induced DNA damage, and leaching of carcinogenic monomers (vinyl chloride, styrene) suggest a plausible long-term risk, especially for PVC and PS particles, requiring further study [27].

VI. ENVIRONMENTAL IMPACT AND ONE HEALTH PERSPECTIVE

Environmental contamination amplifies human health risk through ecological disruption. Microplastics reduce soil fertility, water retention, and microbial diversity, affecting crop yield and food safety. In aquatic systems, they cause trophic transfer from zooplankton to marine mammals, with associated growth inhibition, reproductive effects, and mortality in aquatic organisms. Unlike biodegradable pollutants, microplastics do not degrade readily; they fragment into smaller, more

bioavailable nanoplastics that persist for centuries [28,29].

VII. PREVENTION AND REMEDIATION STRATEGIES

1. Source Reduction and Green Polymer Design

Prevention at source is most effective. This includes banning intentionally added microplastics, improving textile design to reduce fiber shedding, redesigning tires to reduce wear particles, and developing truly biodegradable, non-toxic biopolymers. The concept of safe-and-sustainable-by-design (SSbD) promotes polymers without hazardous additives [30].

2. Exposure Mitigation and Policy

Regulatory measures include improved wastewater treatment with tertiary filtration, upgrading drinking water treatment plants, regulating sewage sludge application on agricultural lands, and establishing air quality guidance for indoor microfibers. Public health measures include use of HEPA filtration indoors, reducing consumption of highly contaminated foods, and avoiding heating food in plastic containers [31].

3. Biomonitoring of Exposed Populations

Systematic human biomonitoring is critically lacking. Standardized protocols for detection in blood, urine, placenta, lung tissue, and stool using μ FTIR, Raman spectroscopy, and pyrolysis-GC/MS are urgently needed. Biomonitoring should prioritize high-exposure groups: textile workers, plastic industry workers, infants, and populations dependent on highly contaminated water sources [32,33].

4. Remediation Technologies

Wastewater and drinking water treatment using membrane bioreactors, electrocoagulation, biochar filtration, and sand filtration can remove >90% of microplastics >10 μ m, though nanoplastics remain challenging [37]. Soil bioremediation using plastic-degrading bacteria such as *Ideonella sakaiensis* (PETase), fungal degradation, and phytoremediation approaches are under investigation but remain slow and polymer-specific [38,39].

5. Integrated One Health Approach

The most effective strategy integrates environmental monitoring, human biomonitoring, toxicological research, and policy. Long-term prospective cohort studies linking microplastic exposure to health outcomes are needed [40].

VIII. CONCLUSION

Microplastics represent a pervasive, complex, and persistent challenge to human health, operating through physical, chemical, and biological mechanisms. While definitive human dose-response data are still evolving, mechanistic evidence of oxidative stress, inflammation, endocrine disruption, reproductive toxicity, and transgenerational effects warrants a precautionary approach. Early biomonitoring, exposure control, and green polymer design are far more effective than post-contamination remediation. Future research must prioritize standardized detection methods, human health cohort studies, and development of safe and sustainable polymers.

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