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COMPREHENSIVE ASSESSMENT OF MICROPLASTICS TRANSFER FROM THE ENVIRONMENT INTO THE HUMAN BODY, THEIR TOXIC EFFECTS, AND HYGIENIC RISK EVALUATION

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Abstract: Microplastics (MPs) — plastic particles smaller than 5 mm in diameter — have emerged as one of the most pervasive and insidious environmental contaminants of the 21st century. Since the widespread adoption of synthetic polymers in the mid-20th century, plastic production has grown exponentially, reaching over 400 million metric tons annually. Owing to the recalcitrance of plastic polymers to biological degradation, these materials persist in ecosystems for centuries, gradually fragmenting into progressively smaller particles. Today, microplastics have been detected in virtually every environmental compartment examined — from deep ocean sediments and Arctic ice cores to agricultural soils, freshwater systems, and urban atmospheric aerosols. Alarmingly, microplastics and their nanoscale derivatives (nanoplastics, <1 μm) are now routinely detected in human biological matrices, including blood, lung tissue, placental tissue, and breast milk, signaling a transition from an environmental problem to a direct human health concern.

Keywords: Microplastics, Human Health, Toxicity, Environmental Exposure, Hygienic Risk, Food and Water Contamination

1. Introduction

The global proliferation of plastic materials represents one of the defining environmental legacies of industrial civilization. Since the commercial introduction of synthetic polymers in the 1950s, cumulative global plastic production has exceeded 9.2 billion metric tons, of which approximately 6.9 billion tons (75%) have become waste. Of this waste, only 9% has been recycled, 12% incinerated, and 79% has accumulated in landfills or the natural environment (Geyer et al., 2017). This uncontrolled accumulation drives the continuous fragmentation of macroplastics into microplastics and, ultimately, nanoplastics — a process mediated by ultraviolet radiation, mechanical abrasion, oxidative degradation, and biological activity.

The scientific and public health community's attention to microplastics has intensified dramatically since Thompson et al. (2004) first documented the accumulation of microscopic plastic fragments in marine environments. Subsequent decades of research have charted an extraordinary trajectory: from isolated oceanographic observations to the detection of microplastics in human blood (Leslie et al., 2022), placentas (Ragusa et al., 2021), and lung tissue (Amato-Lourenco et al., 2021), fundamentally repositioning microplastics from an ecological curiosity to a direct human health priority.

Microplastic contamination is now recognized as a global phenomenon with no geographic boundaries. In aquatic environments, microplastics are present in surface ocean waters at concentrations of 0.1–100,000 particles/m³, in deep-sea sediments at up to 8,000 particles/kg dry weight, and in freshwater systems including rivers, lakes, and drinking water reservoirs worldwide (Eerkes-Medrano et al., 2015; Mintenig et al., 2019). Terrestrial environments are similarly affected, with agricultural soils receiving substantial microplastic inputs via sewage sludge application, irrigation with contaminated water, and atmospheric deposition — estimated at 43,000–300,000 tonnes per year in European farmlands alone (Nizzetto et al., 2016).

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Atmospheric microplastic concentrations in urban environments range from 0.3 to 1,586 particles/m³ of air, with indoor environments frequently exceeding outdoor levels due to the off-gassing and fragmentation of synthetic textiles, upholstery, and flooring materials. The food supply chain from primary production through processing and packaging constitutes an additional major vector of microplastic introduction to the human diet.

Despite the rapidly expanding body of literature on microplastic occurrence and toxicology, critical knowledge gaps persist in the assessment of human health impacts. In particular, there is a paucity of epidemiological data directly linking microplastic exposure to specific disease outcomes in human populations. Most mechanistic toxicological data derive from in vitro cell culture studies or in vivo animal experiments, frequently at concentrations exceeding realistic environmental exposures. Dose-response relationships for chronic low-level human exposure remain poorly characterized. Furthermore, no internationally harmonized regulatory threshold values for microplastic concentrations in food, water, or air exist, rendering formal risk assessment challenging.

The chemical complexity of microplastics — which act as vectors for hundreds of plastic additives (plasticizers, stabilizers, flame retardants) and adsorbed environmental contaminants (persistent organic pollutants, heavy metals) — complicates the attribution of observed biological effects to plastic particles per se versus their associated chemical cargo.

2. Materials and Methods

Study Design

This study employed a mixed-methods approach combining a systematic literature review with critical appraisal of environmental monitoring data and toxicological experimental evidence. The literature review component followed PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. Inclusion criteria comprised: (1) peer-reviewed original research articles or systematic reviews; (2) publication between January 2010 and December 2024; (3) relevance to microplastic sources, human exposure, toxicological effects, or risk assessment. Exclusion criteria comprised: opinion pieces without primary data, studies with insufficient methodological detail, and non-peer-reviewed gray literature (with exceptions for WHO and EU regulatory documents).

The risk assessment component adopted the four-step framework established by the US National Research Council (1983) and subsequently applied in modified forms by WHO and EFSA for chemical risk assessments: hazard identification, dose-response assessment, exposure assessment, and risk characterization.

Study Objects

The objects of study encompassed: (1) environmental matrices — surface water, drinking water (tap and bottled), indoor and outdoor air, soil, and sediment; (2) food commodities — seafood, salt, sugar, honey, beverages, canned goods, and fresh produce; (3) biological matrices — human blood, lung tissue, gastrointestinal tissue, placenta, breast milk, and feces; and (4) toxicological test systems — human cell lines (Caco-2, A549, HepG2, THP-1), primary human cells, and rodent in vivo models.

Analytical Methods

1. Microscopy

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Stereomicroscopy and optical light microscopy (magnification 10–400×) are used for initial particle identification, enumeration, and morphological characterization. Fluorescence microscopy using Nile Red staining enables visualization and counting of smaller microplastic particles (down to approximately 1–5 µm) in complex matrices. Scanning Electron Microscopy (SEM) with Energy Dispersive X-ray Spectroscopy (EDS) provides high-resolution morphological imaging and elemental composition data for surface characterization and identification of inorganic additives.

2. Spectroscopic Identification

Fourier Transform Infrared Spectroscopy (FTIR) — both transmission mode and Attenuated Total Reflectance (ATR-FTIR) — is the gold standard for polymer chemical identification based on molecular fingerprint matching against reference spectral libraries. µ-FTIR imaging enables spatial mapping of microplastic distribution in complex samples. Raman Spectroscopy and µ-Raman microscopy complement FTIR, offering superior sensitivity for smaller particles (<20 µm) and the ability to analyze particles through glass or water. Pyrolysis-Gas Chromatography-Mass Spectrometry (Py-GC-MS) provides quantitative polymer mass analysis and characterization of plastic additives and degradation products.

3. Chemical Analysis of Associated Contaminants

Gas chromatography-mass spectrometry (GC-MS) and liquid chromatography-tandem mass spectrometry (LC-MS/MS) were applied for the identification and quantification of plastic additives (phthalates, bisphenols, organophosphate flame retardants) sorbed onto microplastic surfaces, and persistent organic pollutants (PCBs, PAHs, organochlorine pesticides) that have partitioned onto microplastics from contaminated environmental matrices.

4. Statistical Analysis

Descriptive statistics (means, medians, interquartile ranges) were computed for exposure estimates and toxicological endpoints. Where sufficient primary data were available, Monte Carlo simulation was applied to propagate uncertainty through the exposure assessment. Meta-analytic pooling of effect size estimates from in vitro toxicological studies was conducted using random-effects models where methodological heterogeneity permitted. All statistical analyses were performed using R (version 4.3.1) with packages metafor (meta-analysis) and ggplot2 (visualization).

3. Results

Detection Levels of Microplastics in Environmental and Human Matrices

Comprehensive analysis of published monitoring data reveals ubiquitous microplastic contamination across all examined compartments. In freshwater systems, global median concentrations of 1.0×10^3 particles/m³ were estimated, with significant variability depending on proximity to urban and industrial sources. Ocean surface water concentrations ranged from 0 to 9.7×10^5 particles/m³ (median: ~100 particles/m³). Sediment accumulation was substantially higher, with freshwater sediment concentrations of 10^2–10^6 particles/kg dry weight. Soils receiving sewage sludge contained 1,000–56,000 particles/kg dry weight.

Environmental Matrix	Concentration Range	Units	Number of Studies
Ocean surface water	0 – 9.7×10^5	particles/m³	48
Freshwater (rivers/lakes)	10 – 10^6	particles/m³	62
Treated drinking water	0 – 61	particles/L	34

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(tap)			
Bottled water	0 – 10,000	particles/L	22
Soil (agricultural)	1,000 – 56,000	particles/kg dw	29
Marine sediment	100 – 100,000	particles/kg dw	55
Indoor air	1.0 – 60	particles/m³	18
Outdoor urban air	0.3 – 150	particles/m³	21

Table 3. Summary of microplastic concentration ranges in environmental matrices compiled from published monitoring studies (2015–2024).

Regarding human biomonitoring, microplastics were detected in all human matrices examined in the reviewed studies. Human blood: 1.1–7.4 µg/mL (77% of samples positive, Leslie et al., 2022). Human lung tissue: detected in 65% of samples, 0.26–3.04 particles/g tissue. Feces: 1.9–172 particles/g, indicating significant gastrointestinal transit. Placental tissue: detected in 4/6 samples, mean 10 particles/g. These findings confirm systemic microplastic bioaccumulation and translocational capacity across biological barriers including the gut epithelium, blood-brain barrier, and placenta.

Exposure Intensity — Quantitative Estimates

Dietary exposure estimates derived from literature consolidation indicate that adult individuals in high-income countries ingest approximately 39,000–52,000 microplastic particles annually through food and water consumption alone. When inhalation is incorporated, the estimate rises to 74,000–121,000 particles per year (Cox et al., 2019). Children are estimated to have higher body weight-normalized exposures than adults due to hand-to-mouth behavior and consumption of microplastic-rich infant formula prepared in polypropylene bottles (Li et al., 2020), with estimates of 14.6–40.0 particles/kg body weight/day.

Population Group	Estimated Annual Intake	Primary Route	Uncertainty Level
Adults (food + water)	39,000 – 52,000 particles	Ingestion	Moderate
Adults (+ inhalation)	74,000 – 121,000 particles	Ingestion + Inhalation	High
Children (dietary)	Higher bw-normalized	Ingestion + Dermal	High
Infants (formula fed)	14.6 – 40.0 particles/kg bw/day	Ingestion (PP bottles)	Moderate
Occupational workers	Substantially elevated	Inhalation	High

Table 4. Estimated microplastic exposure intensities by population group and exposure route.

Oxidative Stress

Induction of reactive oxygen species (ROS) and disruption of antioxidant defense systems is among the most consistently reported effects of microplastic exposure across in vitro and in vivo systems. Polystyrene microplastics at concentrations of 10–200 µg/mL induce significant ROS generation in human intestinal Caco-2 cells, lung A549 cells, and hepatic HepG2 cells, accompanied by depletion of glutathione (GSH), reduction in superoxide dismutase (SOD) and catalase activities, and upregulation of heme oxygenase-1 (HO-1). In vivo rodent studies confirm hepatic and renal oxidative stress following oral microplastic exposure, with dose-dependent increases in malondialdehyde (MDA) as a marker of lipid peroxidation. Nanoplastics demonstrate substantially greater oxidative potential than microplastics at equivalent mass concentrations, attributed to their higher surface area-to-volume ratio.

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Systemic Inflammation

Microplastics activate innate immune responses through multiple mechanisms, including activation of the NLRP3 inflammasome, NF-κB signaling pathway, and toll-like receptor (TLR) cascades. In macrophage cell models (THP-1), polystyrene microplastics induce significant upregulation of pro-inflammatory cytokines including IL-1β, IL-6, TNF-α, and IL-8. Neutrophil extracellular trap (NET) formation has been observed in response to microplastic challenge. In vivo, systemic elevations in C-reactive protein (CRP), IL-6, and monocyte chemoattractant protein-1 (MCP-1) have been documented in rodents following 8–12 weeks of dietary microplastic exposure.

Of particular significance for human health risk assessment are recent epidemiological findings. A landmark study by Marfella et al. (2024) detected polyethylene and PVC microplastics in carotid artery atherosclerotic plaques; individuals with detectable plaque microplastics had a 4.53-fold increased risk of myocardial infarction, stroke, or all-cause death over a 34-month follow-up period (adjusted hazard ratio 4.53; 95% CI: 2.00–10.27), establishing for the first time an association between microplastic burden and cardiovascular outcomes in humans.

Genotoxicity and Mutagenicity

Microplastics induce DNA strand breaks (evidenced by comet assay), micronucleus formation, and chromosomal aberrations in multiple cell systems. DNA damage is partially mediated by oxidative stress (8-hydroxy-2'-deoxyguanosine, 8-OHdG, as a biomarker of oxidative DNA damage) and partly by direct particle-DNA interaction for nanoplastics capable of nuclear penetration. Mutagenic plastic additives, including benzo[a]pyrene and other PAHs adsorbed onto microplastic surfaces, may contribute additional genotoxic potency independent of the particle per se.

Endocrine Disruption

Plastic-associated chemicals, rather than polymer particles alone, are implicated in endocrine-disrupting effects. Phthalates (DEHP, DBP, BBP) leaching from PVC and other polymers act as anti-androgens, impairing testosterone synthesis and spermatogenesis. Bisphenol A (BPA) and structurally related bisphenols are estrogenic, binding estrogen receptors with consequent effects on reproductive development, breast tissue, and metabolic regulation. Polybrominated diphenyl ethers (PBDEs) used as flame retardants in electronics and textiles interfere with thyroid hormone signaling. Nonylphenol (NP) and octylphenol, breakdown products of nonionic surfactants used in plastic manufacturing, are weakly estrogenic.

Gut Microbiome Dysbiosis

Emerging evidence from animal studies indicates that dietary microplastic exposure dysregulates gut microbial community composition. Oral polystyrene microplastic administration in mice caused significant reductions in Lactobacillus and Bifidobacterium abundance alongside increases in potentially pathogenic taxa. Concomitant alterations in gut permeability ("leaky gut" — increased transepithelial permeability, reduced tight junction protein expression) were observed, potentially creating a positive feedback loop whereby dysbiosis amplifies systemic exposure to both microplastics and gut-derived inflammatory mediators.

4. Conclusion

This comprehensive review has synthesized evidence from environmental monitoring, human biomonitoring, experimental toxicology, and epidemiology to characterize the threat posed by microplastic contamination to human health. The principal conclusions are as follows:

Microplastic contamination is ubiquitous across all environmental compartments, food supplies, and drinking water sources examined globally. There is no population free from microplastic exposure.Human exposure occurs primarily through dietary ingestion and inhalation,

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with estimated total intakes of 74,000–121,000 particles per year for adults in high-income countries.

Microplastics and nanoplastics are absorbed into the human body, translocate across biological barriers, and accumulate in blood, organs, and tissues. Placental transfer establishes that prenatal fetal exposure occurs.

Toxicological mechanisms of harm — oxidative stress, inflammation, genotoxicity, endocrine disruption, and gut microbiome dysbiosis — are consistently demonstrated across in vitro and in vivo systems, though dose-response characterization at environmentally relevant concentrations remains incomplete.

First human epidemiological evidence links arterial microplastic accumulation to substantially elevated risk of major cardiovascular events, representing a potentially major public health burden requiring urgent confirmation and response.

The current regulatory environment is characterized by a near-complete absence of standards, thresholds, or guidance values specific to microplastic exposure, constituting a significant failure of the environmental health governance framework.

The precautionary principle strongly supports proactive risk management measures pending completion of the full evidence base.

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